

MK'S SURGERY AND CLINICAL SKILLS CLERKSHIP



MOSES KAZEVU

FOREWORD

This review book is designed for use by medical students and the junior medical doctor in the diagnosis, management and curative care of regional Adult Surgical conditions.

The book serves as a good guide and revision tool for board/shelf/licentiate examinations.

Effort has been put in to simplify the literature and make it as practical as possible while maintaining scientific accuracy.

Despite all efforts, it is possible that certain errors may have been overlooked in this book. Please inform the authors of any errors detected.

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COURSE OBJECTIVES (5th year)

KNOWLEDGE OBJECTIVES

1. Explain the physiological, anatomical and pathological basis of surgical disease.
2. Recognize symptoms and signs of the common surgical disease conditions.
3. Analyze symptoms and signs of surgical disease processes to arrive at a differential diagnosis.
4. Identify and interpret laboratory, imaging and bedside investigations required to make a diagnosis and/or monitor clinical progress.

SKILLS OBJECTIVES

1. Obtain a detailed clinical history from the surgical patient.
2. Elicit physical signs in a patient with emphasis from the surgical patient.
3. Perform basic procedures.

TOPICS

Topics	Components
Introduction to surgery	• History taking in the surgical patient • Physical examination technique by system • The operative theatre, sterility, asepsis, aseptic techniques • Infection prevention
Basic principles of surgery	• Hypovolemic and septic shock, fluid and electrolyte balance • Blood transfusion, blood and blood products • Monitoring the surgical patient • The critically injured patient-assessment and management • Surgical emergencies • Peri-operative assessment
Gastrointestinal system	• Esophageal pathologies • Stomach pathologies • Intestinal pathologies • Anal pathologies • Liver and biliary tree pathology • Pancreas • Hernias
Respiratory system	• Signs and symptoms of upper airway obstruction • Methods of airway access • Signs and symptoms of traumatic conditions ➢ Fracture ribs ➢ Flail chest ➢ Pneumothorax ➢ Hemothorax ➢ Diaphragmatic trauma • Chest drainage • Tumors of the thorax • Investigations of the respiratory system
Nervous system	• Neurological examination and examination of the cranial nerves • Assessment of head injury patient • Congenital anomalies of the nervous system • Urological, gastrointestinal, respiratory and musculoskeletal problems of the spinal injury patient • Peripheral neuropathy and nerve injuries • Investigations of the nervous system

Cardiovascular system	• Surgical anatomy of the cardiovascular system • History taking and examination of a patient with cardiovascular pathology • Signs and symptoms of pathology • Approaches to investigation of pathology
Endocrine system	• Pituitary: tumors and clinical presentation • Hyperparathyroidism • Thyroid gland: colloid goiter, thyrotoxicosis, thyroglossal cyst, tumors, symptoms, signs and investigation. Complications of thyroid disease. • The pancreas: diabetes and tumors • The adrenals: tumors and clinical manifestations, secondary hypertension in surgical practice • Breast: anatomy, functions and disorders of the breast, benign and malignant lesions of the breast, differential diagnosis and investigations.
Integumentary system	• Surgical anatomy and functions of the skin • Symptoms and signs of infections and other surgical disorders of the skin • The burnt patients ➢ Percent surface area estimation ➢ Fluid replacement ➢ Immediate management ➢ Complications in the burns patient ➢ Grafting • Common ulcers and lumps in skin and sub-dermal structures • Pigmented lesions of the skin: differential diagnosis and biopsy. • Ulcers of varying aetiology
Haemopoitic and lymphoreticular system	• Haemopoiesis • Haemolytic disease is surgery • Bleeding disorders and haemostasis • Disorders of immunity • Lymphedema • Spleen: signs and symptoms of splenic disorders as well as approaches to investigating disorders of the spleen
Head and neck	• Surgical anatomy of the head and neck • History taking and examination of the patient with head and neck pathology.

	• Signs and symptoms of head and neck disorders
Urology	• Surgical anatomy of genitourinary system • Congenital anomalies and infections of the urinary tract and genitalia • Signs and symptoms of disorder of the genitourinary tract • Physical examination including digital rectal examination • Laboratory and radiological investigations of the urinary treat • Trauma to the genitourinary tract • Common urological emergencies, conditions, malignancies. • Basic urological procedures (urethral catheterization, suprapubic cystostomy) • Basic equipment and materials used in urology • Catheters of various types: bougies and cystoscopies
Orthopedics	• Surgical anatomy of the musculoskeletal system. • Physical examination of muscles, bones and joints. • Neurological evaluations including joint motion, reflexes and muscle power • Common hand conditions and emergencies
Traumatology	• Classification and types of fractures, dislocation, ligamentous and muscular injuries • Bone healing • Spinal trauma, paraplegia, quadriplegia and physiotherapy • Outline of concomitant injuries to head, thorax abdomen and pelvis including the gastrointestinal and genitourinary system • Upper and lower limb injuries, compartment syndrome, fat embolism, pulmonary embolism • Plaster technique and complications • Amputations and rehabilitation • Infective conditions of bone and joints: septic arthritis, osteomyelitis, pyomyositis • Affections of epiphyses and soft tissue

COURSE OBJECTIVES (7th year)

KNOWLEDGE OBJECTIVES

1. Explain the physiological, anatomical and pathological basis of surgical disease.
2. Recognize symptoms and signs of the common surgical disease conditions.
3. Analyze symptoms and signs of surgical disease processes to arrive at a differential diagnosis.
4. Identify and interpret laboratory, imaging and bedside investigations required to make a diagnosis and/or monitor clinical progress.

SKILLS OBJECTIVES

1. Obtain a detailed clinical history from the surgical patient.
2. Elicit physical signs in a patient with emphasis from the surgical patient.
3. Perform basic procedures.
4. Resuscitate a critically ill surgical patient.
5. Manage common surgical conditions.
6. Communicate effectively verbally and in writing with patients, relatives and professional colleagues
7. Exhibit leadership ability

TOPICS

- Topic list essentially the same as fifth year list only addition to each section is: management of the various pathologies.

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INTRODUCTION TO SURGERY

HISTORY TAKING

HISTORY TAKING

DEMOGRAPHICS

- Make sure you know and always record the patient's:
  - Name
  - Age
  - Sex
  - Ethnic group
  - Marital status
  - Occupation
  - Address
- Always record who the informant was and record the date of the examination.
- State whether the case is a self-referral or a hospital referral.

PRESENTING COMPLAINT

- It is customary to ask the patient 'what are you complaining of?' and to record the answer in the patient's own words.
- If you ask 'What is the matter?' the patient will probably tell you their diagnosis.
- It is better not to know the diagnosis made by the patient or other doctors because none may be correct.
- These should be listed in order of severity, with a record of precisely when and how they started. (Alternatively, some clinicians advocate for them to be listed in order of chronological presentation i.e. from the longest standing complaint to the most recent).
- Whenever possible it should be noted why the patient is more concerned with one complaint than another.
- Include the duration of each symptom.

HISTORY OF PRESENTING COMPLAINT

- The full history of the main complaint or complaints must be recorded in detail, with precise dates.
- It is important to get right back to the beginning of the problem. For example, a patient may complain of a recent sudden attack of indigestion, if further questioning reveals that similar symptoms occurred some years previously, their description should be included in this section.

- It is essential to exhaust any other possible questions that involves the system of the presenting complaint.
- When assessing pain don't forget SOCRATES:
  - S-site
  - O-onset
  - C-character
  - R-radiation
  - A-alleviating factors
  - T-time course (Duration)
  - E-exacerbating factors
  - S-symptoms associated

REVIEW OF SYSTEM

- These are direct questions that every patient should be asked, because the answers may amplify your knowledge about the main complaint and will often reveal the presence of other disorders of which the patient was unaware or thought irrelevant.
- Negative answers are just as important as positive answers.
- All the answers to every question, whether they be positive or negative must be recorded.

SYSTEM	AREA OF INTEREST AND QUESTIONS
Central nervous system	• Headache • Syncopal attacks/Fainting • Convulsions • Dizziness, vertigo and lightheadedness • Tremors • Memory loss • Change in vision, smell or hearing • Muscle weakness, paralysis or sensory disturbances • Changes in sleep patterns
Respiratory system	• Cough- nature, productivity, hemoptysis • Dyspnea • Hoarseness • Wheezing

	• Chest pain • Exercise intolerance and night sweats
Cardiovascular system	• Dyspnea • Paroxysmal nocturnal dyspnea • Orthopnea • Chest pain • Palpitations
Gastrointestinal system	• Nausea and vomiting • Diarrhea or constipation • Weight loss • Dysphagia, polyphagia • Indigestion • Hematesis • Melena • Tenesmus • Bowel habits and nature of stool
Genitourinary system	• Hematuria, Dysuria • Nocturia • Polyuria • Vaginal/ penile discharge • Urine color • Urinary hesitancy • Dribbling • Urinary stream
Musculoskeletal and skin	• Aches or pains in muscles, bones or joints • Joint swellings • Limitation of movements • Locking • Weakness • Disturbances of gait • Skin rash

PAST MEDICAL HISTORY

- Record the history of those conditions which are not directly related to the presenting complaint.
- These include (Remember "D.E.A.T.H.S"):
  - Diabetes mellitus
  - Epilepsy
  - Asthma
  - Tuberculosis
  - Hypertension
  - HIV
  - Sickle cell disease
- Ask about history of similar illness in the past
- Ask also about previous hospital admissions and previous history of blood transfusion.
  - Ask about the duration of admission
  - Date of admission
  - Cause of admission/blood transfusion
  - Treatment offered

PAST SURGICAL HISTORY

- Record any history of surgical procedures performed on the patient.
  - Including date, procedure and any complications associated with the procedure.

DRUG HISTORY

- Ask if the patient is taking any drugs.
- Specifically enquire about steroids, anti-depressants, insulin, diuretics, anti-hypertensive, hormone replacement therapy and the contraceptive pill.
- Also ask for any drug related allergies.
- For children ask about the immunization history (Diphtheria, tetanus, whooping cough, measles, mumps, rubella, poliomyelitis, smallpox, typhoid and TB)

GYNAE HISTORY (WOMEN)

- Date of menarche or menopause
- Frequency, Quantity and duration of menstruation
- Last menstrual period
- The use of family planning and oral contraceptives

FAMILY HISTORY

- Enquire about the health and age, or cause of death of the patient's parents, grandparents, brothers and sisters and ask about any children who have died.
- Also ask for specific diseases in the family such as diabetes mellitus, epilepsy, asthma, Tuberculosis, Hypertension, Sickle cell disease.

SOCIAL-ECONOMIC HISTORY

- Record the marital status and type and place of dwelling.
  - Number of rooms in the house
  - Ventilation
  - Source of water
- Ask about their occupation.
- Ask about history of smoking and alcohol intake
  - Type
  - Frequency
  - Duration

SUMMARY OF HISTORY

- This is one sentence that summarizes the important parts of the history. Include identifying information, presenting complaint, a very brief description of the history of presenting complaints plus essential details from ROS, PMHx, Family and social-economic Hx.
- In the summary do not use the name (use initials) and use an estimated age range i.e.
  - <45 years: young age
  - 45-65 years: middle age
  - > 65 years: old age

IMPRESSION

- These are based on the history given.
- The impression and differentials must be accurately related to the information obtained in the history.
- Note: you may query somethings that you suspect e.g. "? malignancy".

PHYSICAL EXAMINATION

PHYSICAL EXAMINATION

- The sequence follows a specific pattern; details will be provided in the subsequent chapters of this literature.

GENERAL EXAMINTION

- Mental state- GCS, this is also assessed as you interact with the patient. (assess patients hearing- Vestibulocochlear nerve, rule out ear wax)
- Nutritional status
- Vital signs
  - Blood pressure
  - Respiratory rate
  - Pulse
  - Temperature
  - Oxygen saturation
- Important negatives- J.A.C.C.Ln. D
  - Jaundice: sclera
  - Anemia: pale conjunctiva
    - At this point you also check eye movements and pupillary light reflex

including the facial nerve (raising eyebrows)
    - Sensation on the face- Trigeminal (CN V)
- Central cyanosis: mouth
    - At this point CN IX, X and XI can be assessed- moving tongue around + saying "Ah" and note the elevation of soft palate.
    - Also assess Olfactory nerve (rule out common colds first)
- Clubbing- fingers
- Lymphadenopathy
    - Submental, submandibular, pre-auricular, subauricular, post-auricular, sub-occipital, cervical, supraclavicular and infraclavicular as well as axillary and epitrochlear
    - Check accessory by shrugging shoulders
- Dehydration- signs of dehydration

SYSTEMIC EXAMINTION

HEAD AND NECK

- Check if neck is supple (movements)- you already checked the cranial nerves because it was incorporated in the general examination

CHEST

- Inspection
- Palpation
- Percussion
- Auscultation

ABDOMEN

- Inspection
- Palpation
- Percussion
- Auscultation
- Vaginal examination (when indication), Assess hernia orifices

MUSCULOSKELETAL

- Gait
- Arms
- Limbs
- Spine
- After the examination summarize findings and offer an impression with some differentials and suggest appropriate investigations.

BASIC SURGICAL EXAMINATION AND EQUIMENT

BLOOD COLLECTION BOTTLES

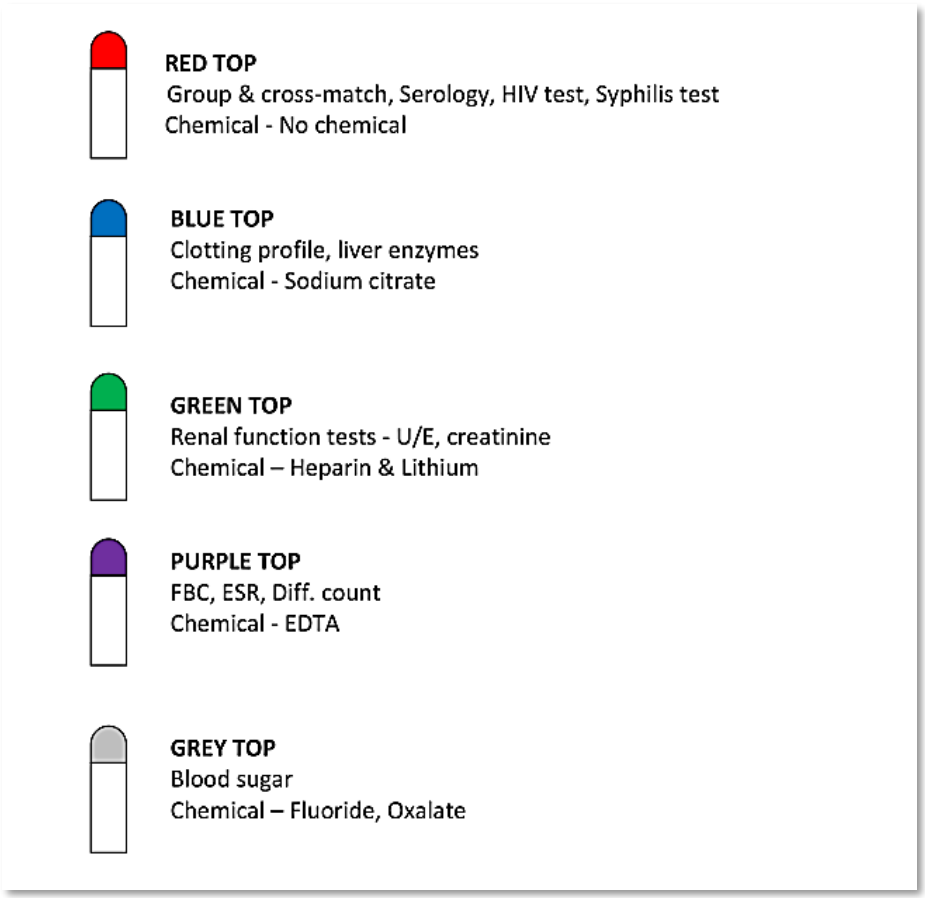


Figure 1: Blood collection bottles (Adapted from Surgery Magic notes)

LUMPS AND MASSES

FOCUSED HISTORY OF A LUMP/MASS

- Most patient with a lump/mass feel it frequently and should be able to tell you about the history of its clinical features.
- The following questions should be asked:
  - When was the lump first noticed?
    - It is important to be precise with dates and terminology.
    - Do not write '~~the lump first appeared 6 months ago~~', when you mean 'the lump was first noticed 6 months ago'.
    - Lumps may exist for months, even years, before the patient notices them.
  - What made the patient notice the lump?
    - The presence or absence of pain is important, particularly if it is the presenting feature.
    - In very general terms, pain is usually associated with inflammation, not neoplastic change.
    - Most patients expect cancer to be painful- and do themselves irreparable harm by ignoring a lump just because it does not hurt them.
  - What are the symptoms of the lump?
    - It may be painful and if it is, you must take a careful history of the pain.
    - The characteristic feature of pain associated with acute infection is its throbbing nature.
    - A lump may be disfiguring or interfere with movement, respiration or swallowing. Describe the history of each symptom carefully.
  - Has the lump changed since it was first noticed?
    - They should be able to tell you whether the lump has got bigger, smaller, or has fluctuated in size and when they noticed a change in size.
    - They may also have appreciated other changes in the nature of the lump that they can tell you about.
    - They may also have noticed tenderness, which may have altered in any of the ways that a pain may change.

- Does the lump ever disappear?
  - A lump may disappear on lying down or during exercise and yet be irreducible at the time of your examination.
  - The patient should always be asked if the lump ever goes away, because this physical characteristic is peculiar to only a few types of lumps.
- Has the patient ever had any other lumps?
  - You must ask this question because it might not have occurred to the patient that there could be any connection between their present lump and a previous lump or even a coexisting one.
- What does the patient think caused the lump?
  - Lumps occasionally follow injuries or systemic illness known only to the patient.

The history of a lump or an ulcer

- Duration: when was it first noticed?
- First symptom: what brought it to the patient's notice?
- Other symptoms: what symptoms does it cause?
- Progression: how has it changed since it was first noticed?
- Persistence: has it ever disappeared or healed?
- Multiplicity: has (or had) the patient any other lumps or ulcers?
- Cause: what does the patient think caused it?

EXAMINATION OF A LUMP

GENERAL EXAMINATION

- It is often tempting to examine only the lump about which the patient is complaining.
- This will cause you to make innumerable misdiagnoses. You must always examine the whole patient.
- Do not forget to perform a general examination and a systemic examination.

LOCAL EXAMINATION OF A LUMP

INSPECTION

- Site: this must be described in exact anatomical terms, using distances measured from bony points. Do not guess distance, use a tape measure.
- Color and texture of overlying skin: The skin over a lump may be discolored and become smooth and shiny or thick and rough.
- Shape: remember that lumps have 3 dimensions. You cannot have a circular lump because a circle is a plane figure. Many lumps are not regular spheres, or hemispheres but have an asymmetrical outline. In these circumstances, it is permissible to use descriptive terms such as pear shaped or kidney shaped.

PALPATION

- Temperature
- Tenderness
- Surface
- Edge
- Size
- Composition
- Consistence
- Fluctuation
- Compressibility
- Reducibility
- Translucence

PERCUSION

- Fluid thrill
- Resonance

AUSCULATATION

- For Bruits

EXTRAS

- Relations to surrounding structures
- State of the regional lymph glands
- State of the local tissue

- Complete your examination by performing a systemic examination.

BASIC PRINCIPLES OF SURGERY

METABOLIC RESPONSE TO TRAUMA AND SIRS

METABOLIC RESPONSE TO TRAUMA AND THE SYSTEMIC INFLAMMATORY RESPONSE SYNDROME

- Homeostasis is the maintenance of a constant internal environment.
- Complex homeostatic responses involving the brain, nerves, heart, lungs, kidneys and spleen work to maintain body constancy.
- Response to injury are in general beneficial to the host and allow healing and survival.
- Understanding of the metabolic response to injury, provides the basis to seek to reduce the need for a homeostatic response by minimizing the primary insult through elective surgical practice.
- Response to injury is graded: the more severe the injury, the greater the response.
- Following elective surgery immediate severity, there may be transient and modest rise in temperature, heart rate, respiratory rate, energy expenditure and peripheral cell count.
- Following major trauma/sepsis, these physiological functions are accentuated, resulting in a systemic inflammatory response syndrome (SIRS), hypermetabolism, marked catabolism, shock and even multiple organ dysfunction.
- The metabolic response also evolves with time. In particular the immunological sequelae of major injury evolve from a pro-inflammatory state driven primarily by the innate immune system (macrophages, neutrophils, dendritic cells) into a compensatory anti-inflammatory response (CARS) characterized by suppressed immunity and diminished resistance to infection.
- In patients who develop infective complications, CARS will drive on-going systemic inflammation, the acute phase response and continued catabolism.

MEDIATORS OF THE METABOLIC RESPONSE TO INJURY

- The classical neuroendocrine pathways of the stress response consist of afferent nociceptive neurons, the spinal cord, thalamus and pituitary gland.
- The neuroendocrine response to severe injury/critical illness is biphasic:
  - Acute phase: due to an actively secreting pituitary and elevated counter-regulatory hormones (cortisol, glucagon, adrenaline). Changes are thought to be beneficial for short-term survival.
  - Chronic phase: associated with hypothalamic suppression and low serum levels of the respective target organ hormones. Changes contribute to chronic wasting.
- Corticotrophin releasing factor (CRF) released from the hypothalamus increases adrenocorticotrophic hormone (ACTH) release from the anterior pituitary
- ACT acts on the adrenals to increase the secretion of cortisol.
- Hypothalamic activation of the sympathetic nervous system causes release of adrenaline and also stimulates the release of glucagon.
- The counter-regulatory hormones (glucagon, glucocorticoids and catecholamines) produce many aspects of the metabolic response to injury.
- There are however many other players including insulin release and sensitivity, hypersecretion of prolactin and growth hormone (GH) in the presence of low circulatory insulin-like growth factor-1 (IGF-1) and inactivation of peripheral thyroid hormones and gonadal function.
- GH has direct lipolytic, insulin-antagonizing and pro-inflammatory properties.
- The innate immune system (mainly macrophages) interact in a complex manner with the adaptive immune system (T-cells, B-cells) in co-generating the metabolic response to injury.
- Pro-inflammatory cytokines include:
  - Interleukin-1 (IL1)
  - Tumor necrosis factor alpha
  - Interleukin 6 (IL-6)
  - Interleukin 8 (IL-8)
- IL-6 and IL-8 are produced within the first 24 hours and act directly on the hypothalamus to cause pyrexia.
- Such cytokines also augment the hypothalamic stress response and act directly on skeletal muscle to induce proteolysis while inducing

acute phase protein production in the liver.

- Pro-inflammatory cytokines also play a complex role in the development of peripheral insulin resistance.
- Other important pro-inflammatory mediators:
  - Nitric oxide (produced from inducible nitric oxide synthesis)
  - Prostanoids (produced from the cyclo-oxygenase-2 enzyme)
- Within hours of the upregulation of pro-inflammatory cytokines, endogenous cytokine antagonists enter the circulation e.g. interleukin-1 receptor antagonist (IL-1Ra) and TNF soluble receptors (TNF-sR-55 and 75) act to control the pro-inflammatory response.
- A complex further series of adaptive changes includes the development of a Th2 type counter-inflammatory response (regulated by IL-4, 5, 9 and 12 and transforming growth factor beta) which if accentuated and prolonged in critical illness is characterized as the CARS and result in immunosuppression and increased susceptibility to opportunistic (nosocomial infection).
- There are many complex interactions between the neuroendocrine, cytokine and metabolic axes. E.g. Cortisol is immunosuppressive at high levels, it acts synergistically with IL-6 to promote the hepatic acute phase response. ACTH released is enhanced by proinflammatory cytokines and the noradrenergic system. The resulting rise in cortisol levels may form a weak feedback loop attempting to limit the pro-inflammatory stress response. Finally, hyperglycemia may aggravate the inflammatory response via substrate overflow in mitochondria, causing the formation of excess free oxygen radicals and also altering gene expression to enhance cytokine production
- At molecular level, the changes accompanying systemic inflammation are extremely complex and may involve gene expression/inactivation.

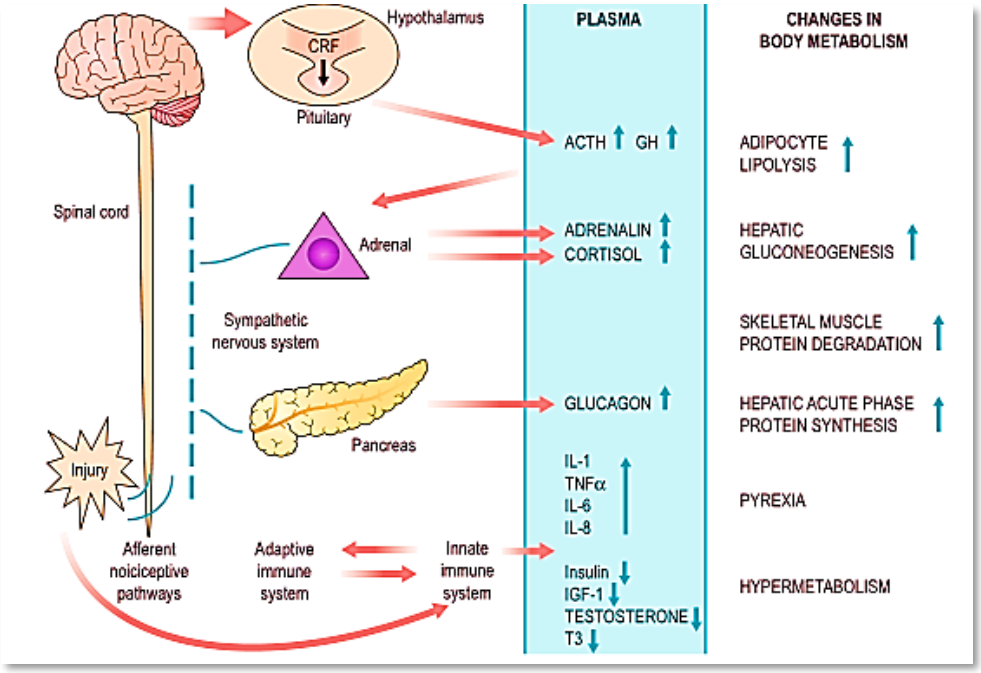


Figure 2: The integrated response to surgical injury (first 24-48h): there is a complex interplay between the neuroendocrine stress response and the pro-inflammatory cytokine response of the innate immune system (Image from Baily & Love's Short Practice of Surgery)

EBB AND FLOW MODEL

- The natural response to injury include:
  - Immobility/rest
  - Anorexia
  - Catabolism
- The changes are designed to aid survival of moderate injury in the absence of medical intervention.
- In 1930, Sir David Cuthbertson divided the metabolic response to injury in humans into the 'EBB' and the 'Flow' phase
  - 'EBB':
    - Starts at the time of injury and lasts for approximately 24-48 hours.
    - It may be attenuated by proper resuscitation but not completely abolished
    - It is characterized by hypovolemia, decreased basal metabolic rate, reduce cardiac output and lactic acidosis.
    - Predominant hormones regulating the EBB phase are catecholamines, cortisol and aldosterone (following activation of the renin-angiotensin system).
    - Magnitude of the neuroendocrine response depends on degree of blood

loss and stimulation of somatic afferent nerves at the site of injury.

- The role of the EBB phase is to conserve both circulating volume and energy stores for recovery.

➢ 'Flow' phases

- Following resuscitation, the EBB phase evolves into a hypermetabolic flow phase, which corresponds to the SIRS. This phase involves the mobilization of body energy stores for recovery and repair and the subsequent replacement of lost or damaged tissue.
- It is characterized by tissue edema (from vasodilation and increased capillary leakage), increased basal metabolic rate (hypermetabolism), increased cardiac output, raised body temperature, leukocytosis, increased oxygen consumption and increased gluconeogenesis.
- The flow phase may be divided into:
  - Catabolic phase: lasts about 3-10 days
  - Anabolic phase: lasts for weeks if extensive recovery and repair are required following serious injury
- During the catabolic phase the increased production of counter-regulatory hormones (catecholamines, cortisol, insulin and glucagon) and inflammatory cytokines (IL-1, -6 and TNF-alpha) results in significant fat and protein mobilization leading to significant weight loss and increased urinary nitrogen excretion. The increased production of insulin at this time is associated with significant insulin resistance and therefor injured patients often exhibit poor glycemic control.
- The combination of pronounced or prolonged catabolism in association with insulin resistance places patients within this phase at increased risk of complications particularly infections and cardiovascular.
- The development of complications will further aggravate the neuroendocrine and inflammatory stress

response thus creating a vicious catabolic cycle.

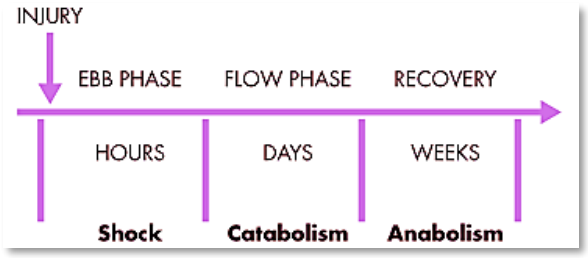


Figure 3: Phases of physiological response to injury (Image from Baily & Love's Short Practice of Surgery)

- It must be remembered that, during the response to injury, not all tissues are catabolic. Indeed, the essence of this coordinated response is to allow the body to reprioritize limited resources away from peripheral tissue (muscle, adipose tissue, skin) and towards key viscera (liver, immune system) and the wound.
- Hypermetabolism following injury is mainly caused by an acceleration of futile metabolic cycles.
- Avoidable factors that compound the response to injury include:
  - Continued hemorrhage
  - Hypothermia
  - Tissue edema
  - Tissue under-perfusion
  - Starvation
  - Immobility

SYSTEMIC INFLAMAMTORY RESPONSE SYNDROME

- Systemic inflammatory response syndrome is a systemic inflammatory response characterized by the presence of 2 or more of the following:
  - Temperature >38ºC (hyperthermia) or <36ºC
  - Tachycardia >90 beats/min
  - Tachypnea >20 min or $PaCO_2$ <4.3 kPa
  - WBC >12, 000/L or <4, 000/L (with predominant neutrophilia or neutropenia)
- Sepsis syndrome: a state of SIRS with proven infection.
- Septic shock: sepsis with systemic shock not responsive to fluids.
- Multiple organ dysfunction syndrome (MODS) is a state of derangement of physiology such that organ function cannot maintain homeostasis. Several organs may be involved:
  - Lungs (ARDS)
  - Kidneys (Acute renal failure)
  - Gastrointestinal tract (liver failure, stress ulceration)
  - Central nervous system (confusion)
  - Blood (DIC)
  - Heart (Cardiac depression)
- When two or more end-organs fail a diagnosis of MODS is made.

System	Features
Respiratory (ARDS)	Increasing minute ventilation Decreased pulmonary compliance Arterial hypoxaemia and hypercapnia Diffuse fluffy pulmonary infiltrates on chest radiograph
Renal (ARF)	Oliguria Rising urea and creatinine
Liver	Jaundice Decreased protein synthesis (hypoalbuminaemia, prolonged PT)
Gastrointestinal	Gastric stress ulceration Adynamic ileus Acalculous cholecystitis Pancreatitis
Haematology	Leukopenia Disseminated intravascular coagulation
Central nervous system	Confusion Decreasing Glasgow Coma Score

ARDS, adult respiratory distress syndrome; ARF, acute renal failure; PT, prothrombin time.

Figure 4: MODS (Image from Clinical Surgery 2nd Ed)

- Compensatory anti-inflammatory response syndrome (CARS) is an anti-inflammatory response that counteracts the systemic inflammation of SIRS. CARS is characterized by the release of potent anti-inflammatory cytokines including IL-4, IL-10 and TGF-beta. It may also be deleterious resulting in opportunistic infection such as systemic candidiasis.
- The pathophysiology of SIRS/MODS is complex. One or more of many initiating factors causes a systemic hyperinflammatory response and it is the uncontrolled activity of this response that leads to organ failure.
  - ➢ Inciting factors include:
    - Infection
      - Gram-negative bacteria: endotoxin
      - Gram-positive bacteria: exotoxin
    - Macrophage cytokine release
    - Microcirculatory injury
    - Gut barrier failure
- TNF-alpha released from activated macrophages and neutrophils is cytotoxic to endothelial cells and parenchymal cells of end-organs.
- Lipopolysaccharides (LPS) released from Gram negative bacterial cell walls activates macrophages via attachment of LPS-binding protein and activation of CD14 molecules on the cell surface.
- IL-6 and IL-1 beta cause endothelial cell activation and damage. They promote complement and chemokine release.
- High dose IV steroids have little role in established SIRS (probably because of multiple pathways of activation). Steroids for early SIRS are unproven.
- Platelet activation factor (PAF): implicated particularly in acute pancreatitis. No proven role for anti-PAF antibody.
- Inducible nitric oxide synthase synthesized by leukocytes, macrophages and Kupffer cells is active for up to 20h and produces nitric oxide (NO) a potent vasodilator and a free radical

scavenger. However, it can also form peroxynitrite ($ONOO^-$), a free radical that may cause cellular damage.

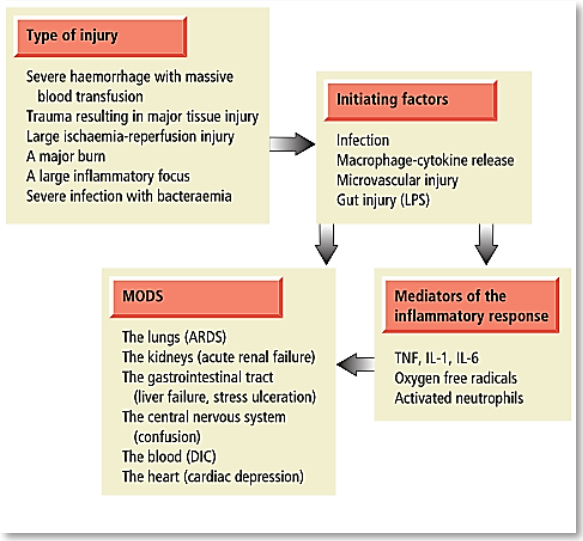


Figure 5: Pathophysiology of SIRS (Image from Clinical surgery 2nd Ed)

- Common surgical causes of SIRS include:
  - Acute pancreatitis
  - Perforated viscus with peritonitis
  - Fulminant colitis
  - Multiple trauma
  - Massive blood transfusion
  - Aspiration pneumonia
  - Ischemia-reperfusion injury

Pathophysiology

Initiating factor	→	Systemic hyperinflammatory response	→	Common end pathway	→	Clinical picture
Infection:		Release of TNF-α, IL-1, IL-6		Endothelial cell activation		Tachycardia
Gram-negative bacteria: endotoxin				Vasodilatation		Tachypnoea
Gram-positive bacteria: exotoxin				Capillary leak		Fever
Macrophage cytokine release				Intravascular coagulation		Leukocytosis
Microcirculatory injury						
Gut barrier failure						

Figure 6: Pathophysiology of SIRS (Image from Clinical surgery 2nd Ed)

- Early treatment of SIRS may reduce risk of MODS developing. The role of treatment is to eliminate any causative factor and support the cardiovascular and respiratory physiology until the patient can recover. Treatment include:
  - Treat infection: removal of necrotic tissue or drainage of abscess is essential. Nosocomial infection must be prevented by aggressive physiotherapy, pulmonary toilet and care of central lines. Wounds must be

kept clean and debrided if necessary. Antibiotic therapy is initiated on the basis of the most likely infective organisms until the result of blood culture are available when more specific antibiotic treatment can be initiated.

- Ensure adequate tissue oxygenation: adequate IV fluids with constant monitoring of urine output and pulmonary artery pressure. Diuretics may be required to maintain urinary output and should only be used when adequate hydration is achieved. Ventilatory support may be necessary with supplementary oxygen or mechanical ventilation in severe cases.
- Maintain nutritional support: enteral or total parenteral nutrition.
- Minimize systemic inflammatory response: Corticosteroids
- Use of recombinant activated protein C (still under testing)

- Overall mortality for SIRS is 7%, 14% for sepsis syndrome, 40% for established septic shock and 90% for 3-system failure.
- Factors predicting poor outcome:
  - Very young and very old
  - Severe initial insult
  - Inability to increase cardiac output
  - Inability to find septic source
  - Multiple end-organ failure

TRAUMA AND ITS MANAGEMENT

TRAUMA AND ITS MANAGEMENT

- Traumatology (Greek "*trauma*"- injury or wound), is a branch of medicine that entails the study of wounds and injuries caused by accidents or violence to a person and the surgical therapy and repair of the damage.
- Traumatology may also be known as accident surgery.
- The "Golden Hour" of Trauma is the period immediately following trauma in which rapid assessment, diagnosis, and stabilization must occur.
- The Advanced Trauma Life Support course (ATLS) has established a standardized approach to trauma assessment & management.
- The ATLS includes:
  - Primary survey- ABCDE
    - A-airway maintenance with cervical spine protection.
    - B- breathing and ventilation.

- C-circulation with hemorrhage control
- D-disability/ neurologic assessment
- E- Exposure and environmental control
- F- fluids
- G- Glucose

➢ Secondary survey
- When the primary survey is completed, resuscitation efforts are well established and the vital signs are normalizing, the secondary survey can begin.
- The secondary survey is a head-to-toe evaluation of the trauma patient, including a complete history and physical examination including the reassessment of all vital signs.
- Each region of the body must be fully examined. X-rays indicated by examination are obtained.
- If at any time during the secondary survey the patient deteriorates, another primary survey is carried out.

➢ Tertiary survey: A careful and complete examination is followed by serial assessments to help recognize missed injuries and as well as allow a definitive care management plan.

PRIMARY SURVERY AT THE SITE OF EVENT (FIRST AID)

- This uses the A, B, C approach
  - ➢ A- airway maintenance with cervical spine protection
  - ➢ B-breathing and ventilation
  - ➢ C-circulation with hemorrhagic control
- This approach is particularly important when assessing patients with multiple injuries.

AIRWAY

- Signs of an obstructed airway are cyanosis, apnea (not breathing) and stridor (a high pitched creaking noise heard especially on inspiration).
- It is essential to protect and secure an adequate airway.
- Lungs cannot be oxygenated if the airway is obstructed for example by jaw and tongue falling back, swollen soft tissues, direct damage to the upper airway, false teeth, vomitus or blood.
- The airway of unconscious patients lying on their back often becomes obstructed by their own soft tissues, but before they are rolled into the supine or semi-prone position or the neck extended always consider

and if possible exclude an associated cervical spinal injury.

- All patients found to be unconscious after an injury must be assumed to have an associated injury of their cervical spine because abrupt or careless turning may further dislocate or sublux a cervical vertebra and injure the spinal cord when no injury existed or turn partial cord damage into a complete.
- The neck should thus be immobilized in all unconscious injured patients by longitudinal manual support, the application of a hard collar and the use of stabilizing sand bags and tape before turning the patient onto their side until clinical examination and radiographs have excluded unstable fractures of the cervical spine.

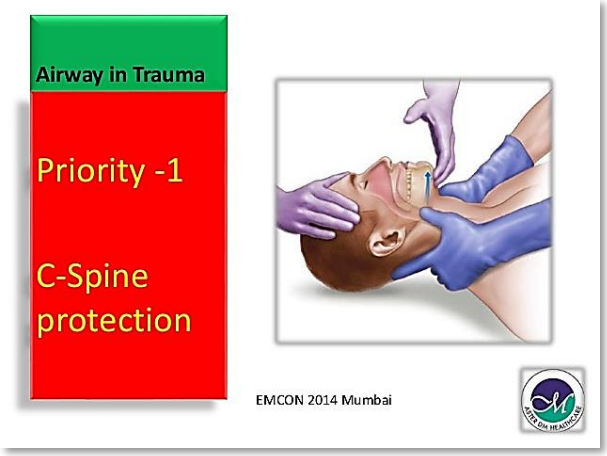


Figure 7:: Chin lift with longitudinal manual support

- Airway obstruction is often relieved by lifting the jaw upwards but may only be relieved by inserting a finger in the mouth and pulling the jaw or palate forward, especially when the obstruction is associated with a fractured maxilla or mandible.

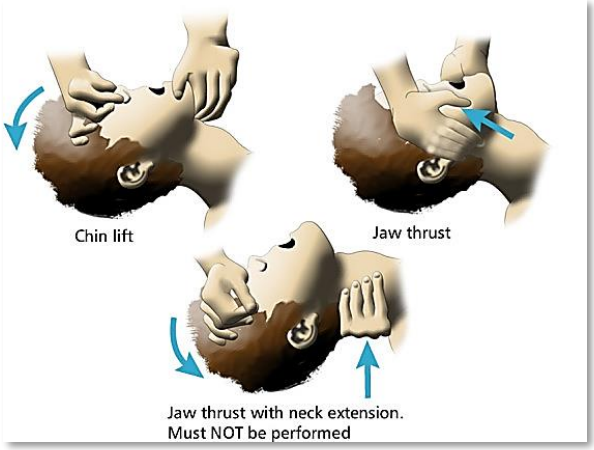


Figure 8: Jaw maneuvers to clear the airway

- An oropharyngeal or nasopharyngeal airway should be inserted as soon as possible and then in an unconscious patient, replaced by an endotracheal tube, inserted by an experienced anesthetist because the absence of a gag reflex in an unconscious patient makes aspiration of saliva, vomitus or blood into the lungs is a major hazard.

BREATHING

- If the patient remains cyanosed or apneic after the airway has been cleared, mouth-to-mouth

resuscitation should be started immediately.

- This is easier and safer (from risks of cross-infections) if a Brook's airway, a facemask, an Ambu bag and oxygen are available.

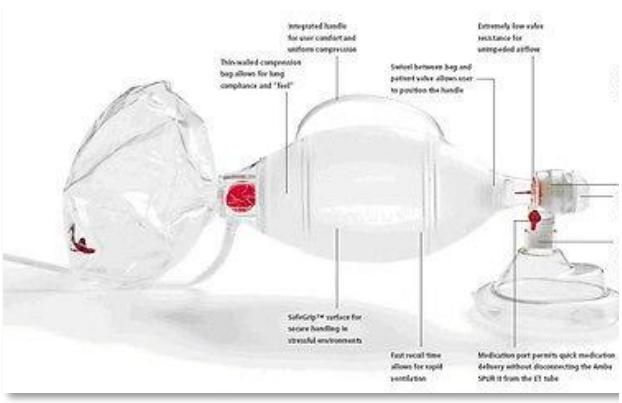


Figure 9: An Ambu bag

- Mouth to mouth ventilation combined with external cardiac massage may, however be lifesaving in patients who have undergone a short period of respiratory arrest.
- If there is no breathing or a pulse within 10 seconds begin chest compressions. Start CPR with 30 chest compressions before giving two rescue breaths (30:2) in children use a 15:2 ratio.

- There are few indications for heroic attempts at inserting home-made tracheostomy devices at the scene of the accident. The patients are probably better served by a rapid transfer to hospital by experienced ambulance staff.

CIRCULATION

- Severe external bleeding at the scene of the accident requires manual compression directly over the wound or pressure proximal to the point of bleeding, where the feeding artery can be compressed against an underlying bony point.
- Tourniquets should only be used to stop distal limb bleeding. They rarely work efficiently and often exacerbate bleeding by obstruction of the venous outflow while failing to occlude the arterial outflow.
- An effective tourniquet makes the whole limb ischemic and will cause permanent muscle and nerve damage if it is kept in place for more than 1.5 to 2 hours.
- Tourniquets theoretically cause reperfusion problems/injury when released- metabolic acidosis, myoglobinuria and hypercalcemia.
- Nevertheless, a tourniquet can sometimes be lifesaving if it is applied at the correct pressure to a mangled bleeding limb for a short period.
  - The time of application must be carefully recorded and passed on to ambulance and medical stuff.
- Direct manual pressure applied provided it is achieving its desired

effect (i.e. stopping or reducing blood loss) is usually preferable to a tourniquet.
- This can be very tiring because it must be maintained until the patient reaches the hospital.

GENERAL FIRST-AID ADVICE

- First aiders should ensure there is no immediate danger to themselves before approaching a casualty.
- Many injured patients are best served by leaving them where they are until help arrives unless they are in any kind of danger or threat e.g. inside a burning car.
- Provided the patient has a strong pulse, is breathing normally and is not overtly bleeding it is best to call for help and wait.
- As help is waited for monitor pulse, breathing and provide morale support.
- Under no circumstances should an injured patient be given anything to eat or drink.

PRIMARY SURVERY IN THE ACCIDENT AND EMERGENCY DEPARTMENT

- The first and key part of the assessment of patients presenting with trauma is called the primary survey.
- All patients who are unconscious or suspected of having multiple or serious injuries should be admitted directly to the resuscitation area of the accident and emergency department.
- The clinical assessment (history and examination) and resuscitation must occur simultaneously if lives are to be saved.
- The routine A, B, C, D, E assessment must be done despite whatever happened before the hospital admission.
  - A- airway maintenance with cervical spine protection
  - B- breathing and ventilation
  - C-circulation with hemorrhage control
  - D- disability/ neurologic assessment
  - E- exposure and environmental control
  - F- fluids
  - G-glucose

TRIAGE

- This is the assignment of degrees of urgency to wounds or illnesses to decide the order of treatment of a large number of patients or casualties.
- This should be sorted out according to their need for treatment and the resources available.
- It starts at the scene and continues at the receiving hospital.

- Priority is given to patients most likely to deteriorate clinically.
- It takes into account the vital signs, pre-hospital clinical course, mechanism of injury, age and other medical conditions.
- Multiple casualties: the number of patients and severity of injury do not exceed the capacity of treatment center- life threatening injuries and multiple system injuries are treated first.
- Mass casualties: the number of patients and severity of injury exceeds capacity of the treatment center- patients are selected for treatment according to best chance of survival with least expenditure of resources (time, personnel, equipment, supplies).

INITIAL ASSESSMENT

- This comprises of:
  - Resuscitation and primary survey
  - Secondary survey
  - Definitive treatment or transfer for definitive care
- Many mistakes are caused by not examining the patient carefully.
- The peripheral injuries are unlikely to kill the patient even if you miss them but you can easily overlook serious central ones especially injuries to the chest and abdomen.
- You will not miss a bone sticking out of a trouser leg but you can easily miss blood in his thoracic cavity or a slowly developing hemoperitoneum.
- More lives are lost from failing to care for the airway than from any other cause.

AIRWAY

- The neck must be protected by a collar and immobilized fully using sand bags and tape.
- An anesthetist should assess the need for better control of the airway.
- Look for the signs of inadequate oxygenation, airway obstruction, foreign bodies, facial, mandibular or laryngeal fractures:
  - Respiratory distress, use of accessory muscles of respiration, flaring of the nares
  - Apnea
  - Cyanosis
  - Tracheal tag
  - Loss of consciousness and presence of major facial, neck or chest injuries that might obstruct the airway may all indicate the need for endotracheal or nasogastric intubation once the neck has been stabilized.
- Look for signs of inadequate ventilation

- Chest should be uncovered and palpated to assess respiratory movements.
- Confirm the presence of air entry into the lungs with a stethoscope (though some books say the primary survey should not use instruments however do whatever it takes to save the patient).
- The neck and jaws should be palpated to check for deformity.
- Insert a finger into the mouth to extract any foreign bodies and to check for jaw fractures. Occasionally severe damage to the upper airways or trachea makes intubation impossible.
- An emergency tracheostomy is indicated if the patient is deeply cyanosed or apneic and an endotracheal tube cannot be inserted safely.

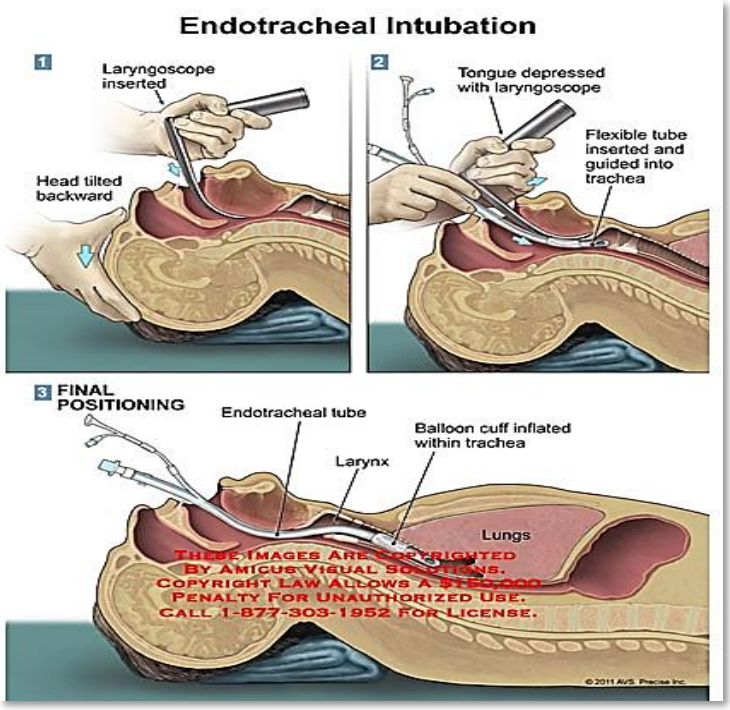


Figure 10: Endotracheal intubation

- Management may involve
  - Secretion control
  - Intubation or
  - Surgical airway e.g. cricothyroidotomy, emergency tracheostomy



Figure 11: Endotracheal tube

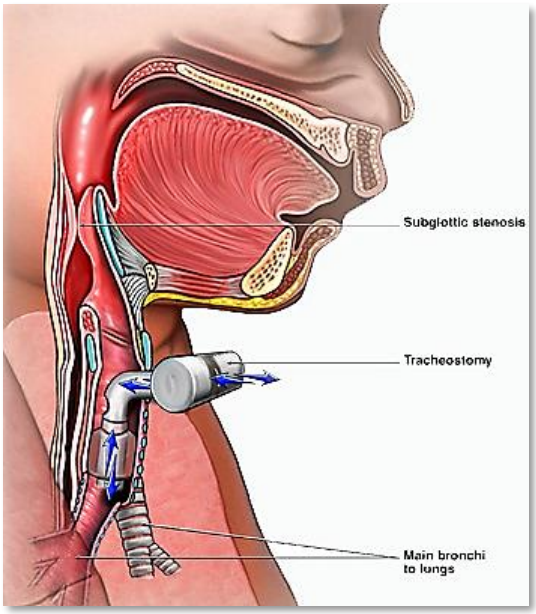


Figure 12: Tracheostomy



Figure 13: : Tracheostomy

- Establish a clear airway (chin lift or jaw thrust) but protect the cervical spine at all times.

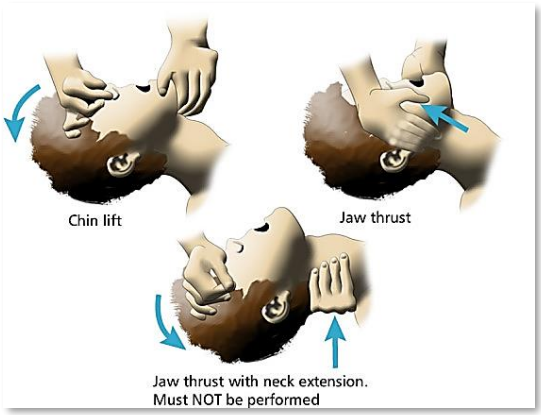


Figure 14: Jaw maneuvers to clear airway

- If the patient can talk, the airway is likely to be safe, however, remain vigilant and recheck.
- An oropharyngeal airway may be put but in an unconscious patient with no gag reflex replace with an endotracheal tube inserted by an experienced anesthetist.
- Definitive airway should be established if the patient is unable to maintain integrity of airway.

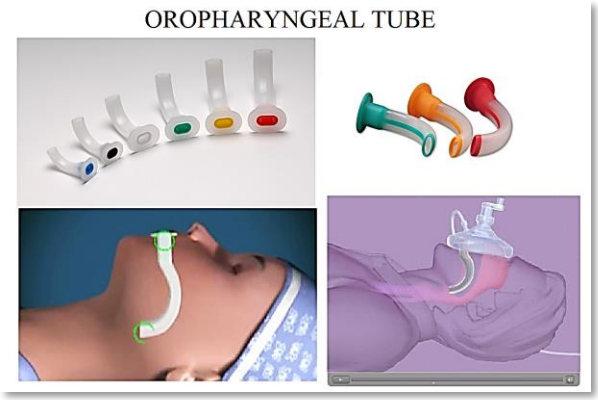


Figure 15: Oropharyngeal airway

- Cervical spine protection is critical throughout the airway management process.
- Movement of the cervical spine could cause spinal injury so movement of the cervical spine should be avoided unless absolutely necessary for maintaining an airway.

BREATHING

- Assess the condition and function of the thoracic cage.
- Once you ensure that the airway is patent, assess the adequacy of ventilation by:
  - Inspecting: for symmetry and chest movements
  - Palpating: for symmetry and chest movements
  - Percussion: for dullness
  - Auscultation: for breath sounds
- Patients with multiple injuries or chest problems causing hypoxia should be given high-flow oxygen through a closed circuit oxygen mask from the moment of arrival in the accident and emergency department.
- An oxygen saturation monitor placed on an extremity is a valuable means of assessing the effectiveness of the patient's ventilation.

INSPECTION AND PALPATION

- Open wounds or flail segments in the chest indicate the need for chest drain and positive-pressure ventilation.
  - A flail segment occurs when several ribs are fractured in two places. The flail segments sink inwards during inspiration (paradoxical breathing)
- Bruising over the chest indicates that rib fractures are likely
- Surgical emphysema (subcutaneous emphysema) suggests that the pleura has been breached.
  - It presents as a crackling sensation in the subcutaneous tissues. It is due to air being trapped in the subcutaneous tissues.
  - A 'sucking' chest wound may be present.

PERCUSION

- A tension pneumothorax must be suspected if breathing is difficult.
  - The trachea is deviated to the contralateral side and there is

decreased air entry over the affected lung.

- Although the clinical diagnosis will have to be confirmed with a chest radiograph, a chest drain should be inserted on the evidence of the clinical signs if the patient is unstable.

- Bilateral tension pneumothoraces are very rare, but cause severe cardiac and respiratory compromise.
  - They present with cyanosis, severe air hunger, a weak pulse and hypotension.
  - In bilateral pneumothoraces, the trachea remains central but air entry is poor into both lungs.
  - The rapid insertion of chest drains which are connected to under-water seal drainage bottles relieves the situation.

AUSCULTATION

- Decreasing air entry and breath sounds indicate the need for chest drainage.

LESIONS ACUTELY IMPAIRING VENTILATION

- Lesions acutely impairing ventilation:
  - Tension pneumothorax: requires needle thoracostomy followed by drainage.
  - Flail chest: management involves ventilation.
  - Hemothorax: Will usually require intercostal drain insertion
  - Pneumothorax: may require intercostal drain insertion

TENSION PNEUMOTHORAX

- The accumulation of air under pressure in the pleural space.
- An imminent danger is that the lung will collapse under the pressure.
- In tension pneumothorax, air enters the pleural cavity and is trapped there during expiration so the air pressure within the thorax mounts higher than atmospheric pressure, compresses the lung, may displace the mediastinum and its structures (including the lung) toward the opposite side and cause cardiopulmonary impairment. Also called pressure pneumothorax.

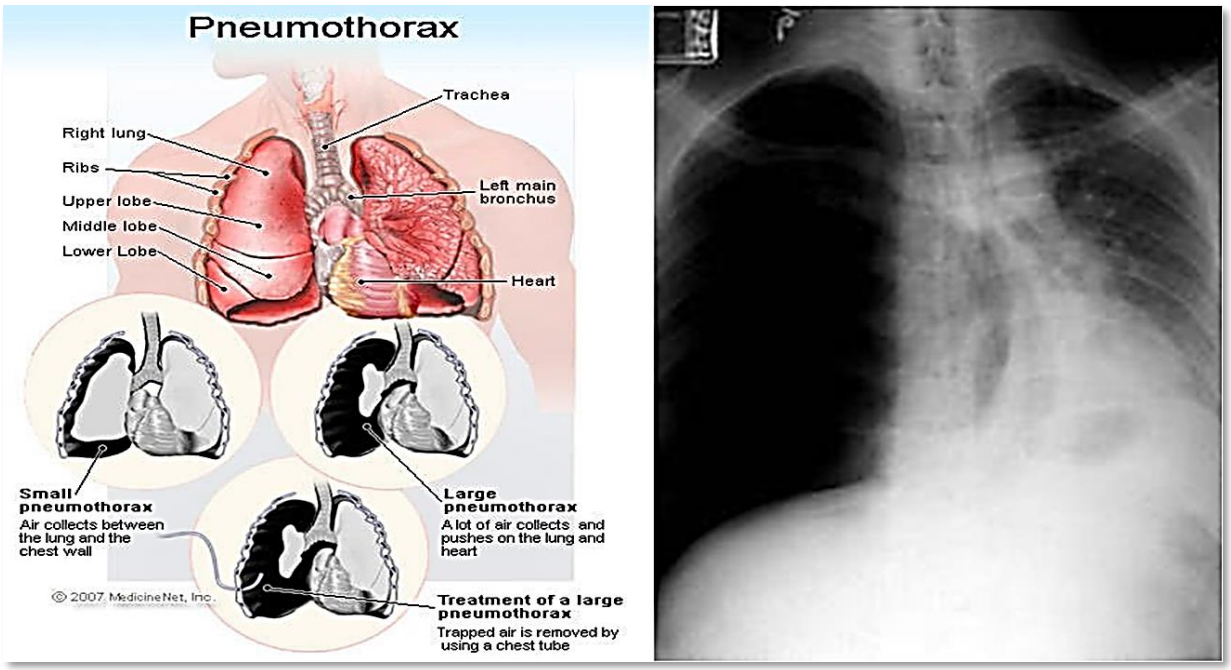


Figure 16: A pneumothorax. Note deviation of the trachea away from the affected side.

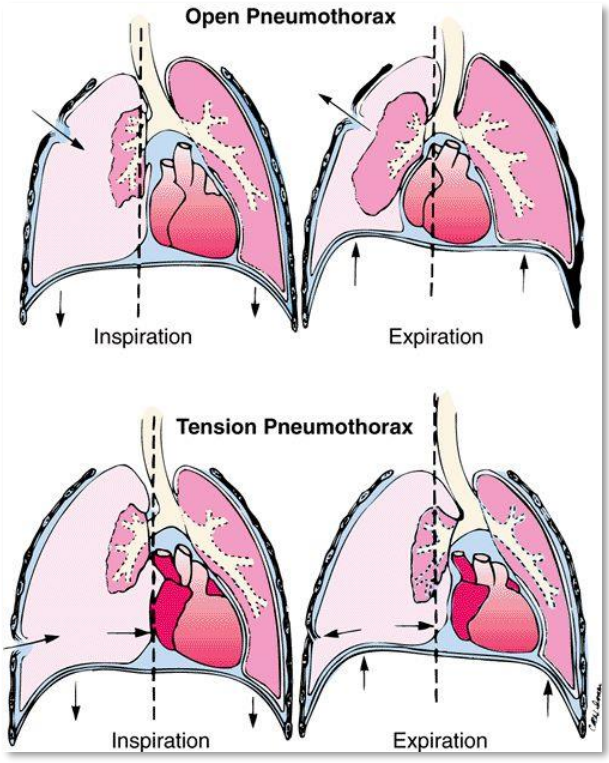


Figure 17: A pneumothorax

FLAIL CHEST

- Occurs when a segment of the rib cage breaks due to trauma and becomes detached from the rest of the chest wall.
- It occurs when multiple adjacent ribs are broken in multiple places, separating a segment, so a part of the chest wall moves independently.
- The flail segment moves in the opposite direction to the rest of the chest wall, it goes in while the rest of the chest is moving out, and vice versa.
- This so called "paradoxical breathing" is painful and increases the work involved in breathing.

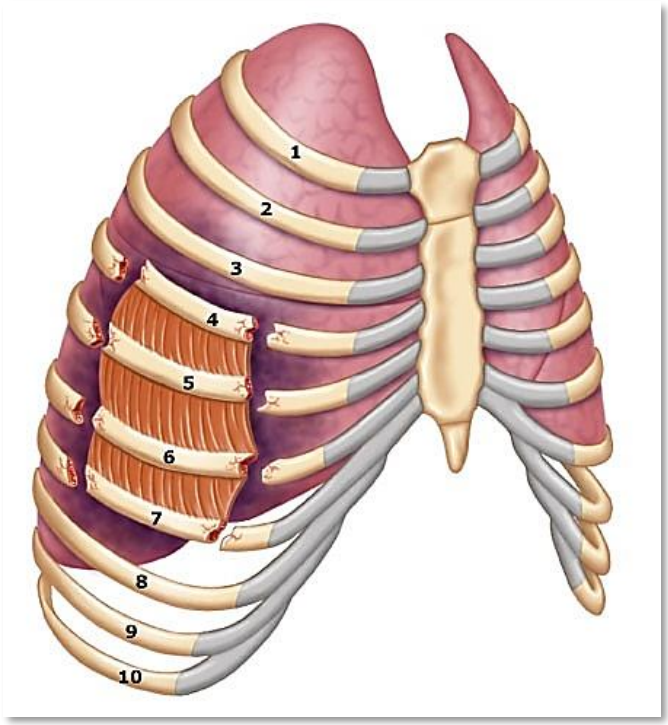


Figure 18: A flail chest

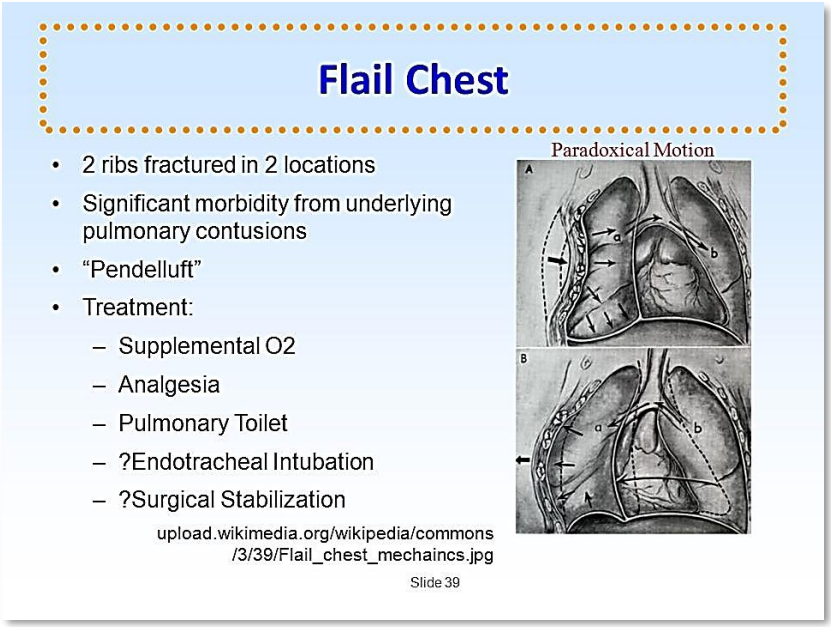


Figure 19: A flail chest

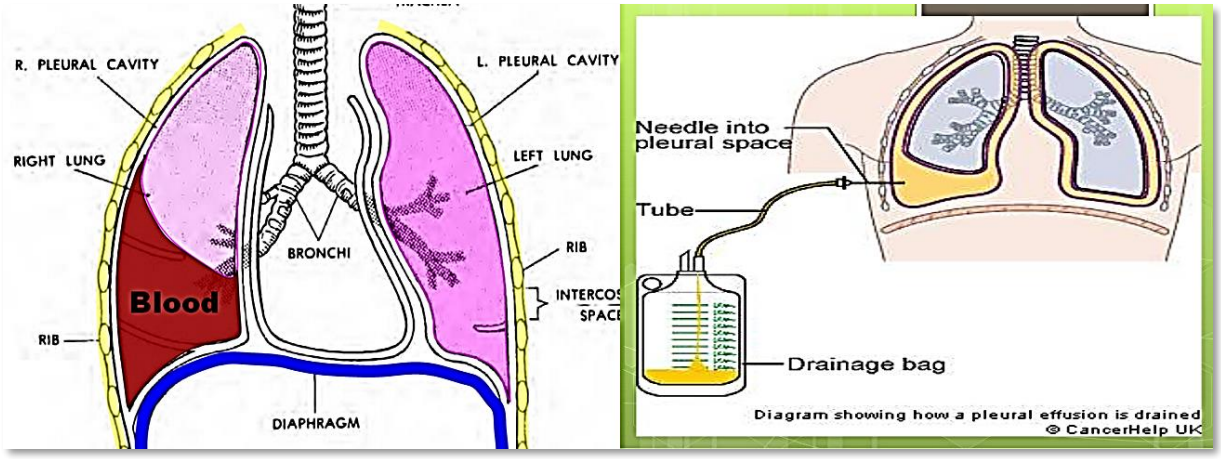


Figure 20: A hemothorax (left) and an intercostal drainage (right)

CIRCULATION

- Restoration of the circulation may take precedence over the airway and ventilation if the patient is breathing satisfactorily.
- It can be assessed simultaneously with the airway and breathing if an experienced anesthetist is available to manage ventilation.
- There are two major causes of circulatory failure: Cardiac damage/tamponade and hemorrhage.
- Cardiac tamponade is rare but life threatening and easily missed. It must therefore be briefly considered in all patients with major injuries, especially in those with penetrating injuries of the chest.
- Blood loss is the main preventable cause of death after trauma. To assess blood loss rapidly observe:
  - Level of consciousness
  - Skin color
  - Pulse
  - Bleeding- this should be assessed and controlled
    - IV access with 2 large bore cannulae in an upper limb. Access by cut down or central venous catheterization may be done according to skills available.
    - Fluid therapy should be initiated with up to 2L of an isotonic (either Ringer's lactate or Normal Saline) crystalloid solution. Pediatric patients should receive an IV bolus of 20ml/kg.
    - Use warmed fluids wherever possible

- At cannula insertion, blood should be taken for cross-match and baseline investigations.
- IV fluids will need to be given rapidly. Ringer's lactate is the preferred initial crystalloid solution.
- Direct manual pressure should be used to stem visible bleeding (not tourniquets, except for traumatic amputation, as these cause distal ischemia)
- Occult bleeding into the abdominal cavity and around long-bone or pelvic fractures is problematic but should be suspected in a patient not responding to fluid resuscitation.

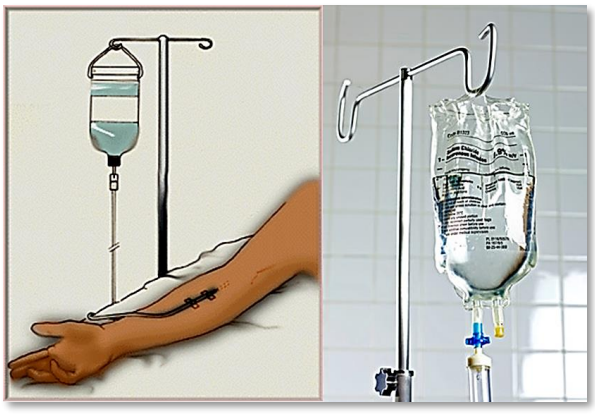


Figure 21: An IV line with fluids running

CARDIAC TAMPONADE

- Occurs when large quantities of blood collect within the pericardial cavity, around the heart and embarrass its action.
- This reduces the cardiac output, producing a weak pulse and hypotension.
- The condition should be suspected if the JVP is markedly elevated and rises rather than falls with inspiration (Kussmaul's sign), however jugular venous distention may not occur in a patient who has lost a large quantity of blood.
- Pulsus paradoxus, when the pulse volume decreases on inspiration rather than increasing, may be present.
- The heart sounds are usually muffled and poorly heard.
- Beck's triad of acute cardiac tamponade:
  - Hypotension (low arterial blood pressure)
  - Distended neck veins (raised JVP)
  - Muffled/distant heart sound
- Chest X-ray may show an enlarged cardiac shadow.
- An echocardiogram will confirm the diagnosis.
- The patient's condition may be stabilized by aspirating the blood from the pericardial sac using echocardiography and electrocardiography to ensure correct placement of the needle and

catheter before definitive surgery is undertaken.

REVEALED HEMORRHAGE

- Visible arterial bleeding presents as a pulsating stream of bright red blood coming from an open wound, whereas venous bleeding is dark and continuous.
- Arterial hemorrhage from an open wound can usually be controlled by direct digital pressure or proximal arterial compression as well as use of sterile vascular clamps before definitive surgery.
- Venous bleeding always responds to simple pressure and may be made worse by the application of a tourniquet.

CONCEALED (INTERNAL) HEMORRHAGE

- These are much harder to diagnose and therefore must be suspected in all patients with multiple or serious injuries.
- It always accompanies major fractures of long bones and fractures of the pelvis.
- They must be quickly diagnosed and treated in order to save lives.

CLINICAL SIGNS OF HEMORRHAGE

- Diagnosis based on finding signs of:
  - Hypovolemic shock- pale, anxious, sweaty patient with cold extremities, a rapid, thready pulse, tachypnea and hypotension.

DISABILITY/NEUROLOGICAL STATUS

- After A, B, and C above, rapid neurological assessment is made to establish:
  - Level of consciousness, Using Glasgow coma scale. (Alternatively the APVU scale can be used for quick assessment)
  - Pupils: size, symmetry and reaction
  - Any lateralizing sign
  - Level of any spinal cord injury (limb movements, spontaneous respiratory effort)
  - Oxygenation, ventilation, perfusion, drugs, alcohol and hypoglycemia may all also affect the level of consciousness.

Glasgow coma scale		Score
Eye opening	spontaneously	4
	to speech	3
	to pain	2
	none	1
Verbal response	orientated	5
	confused	4
	inappropriate	3
	incomprehensible	2
	none	1
Motor response	obeys commands	6
	localises to pain	5
	withdraws from pain	4
	flexion to pain	3
	extension to pain	2
	none	1
Maximum score		15

Figure 22: The Glasgow coma scale

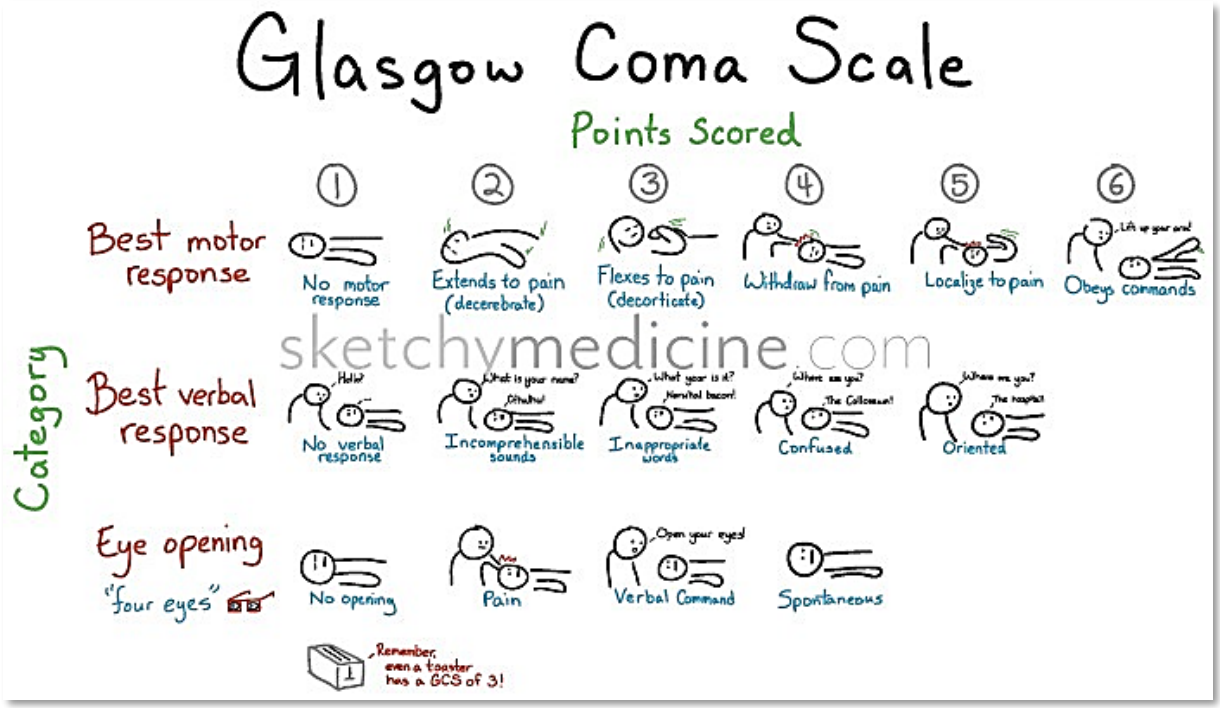


Figure 23: The Glasgow coma scale

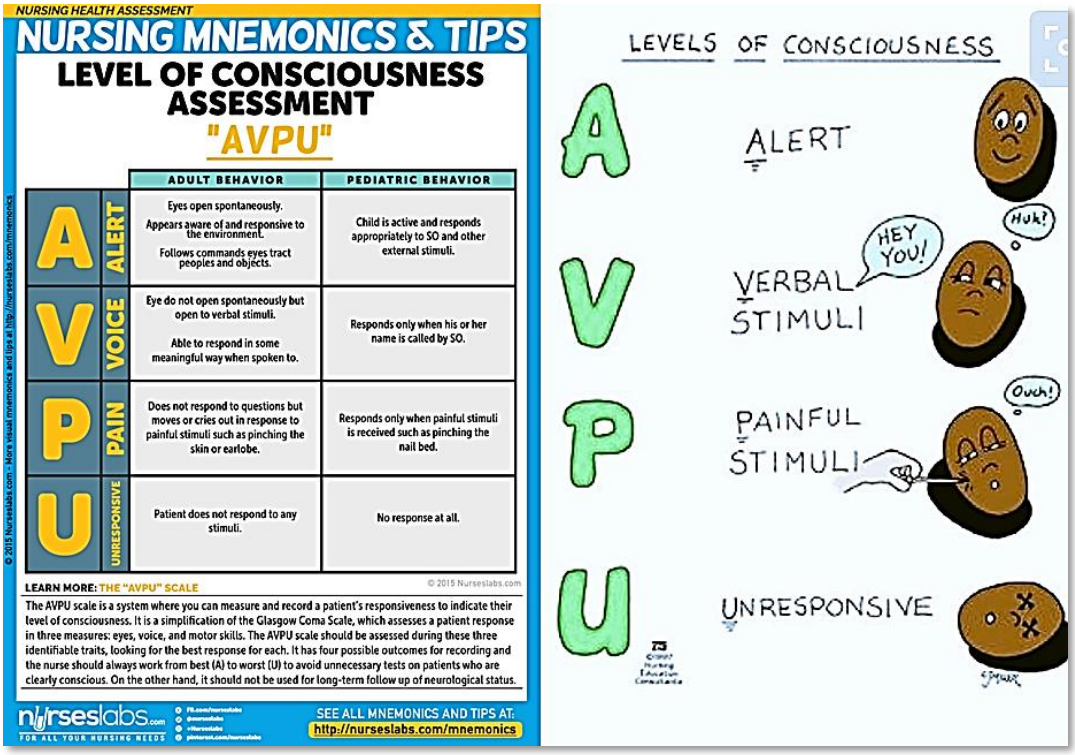


Figure 24: The AVPU scale

- Patients should be re-evaluated frequently at regular intervals.
- Deterioration can occur rapidly and often patient can be lucid following a significant head injury before worsening.
- Signs such as pupil asymmetry or dilatation, impaired or absent light reflexes, and hemiplegia/weakness all suggest an expanding intracranial mass or diffuse edema.
- This requires IV mannitol, ventilation and urgent neurosurgical opinion.

EXPOSURE/ ENVIRONMETAL CONTROL

- Undress the patient, but prevent hypothermia.
- Clothes may need to be cut off.
- After examination, attend to prevention of heat loss with warming devices, warmed blankets, etc.

FLUIDS AND GLUCOSE

- See circulation for fluids.
- Check the random blood sugar and give glucose where needed as well as insulin with potassium chloride.

ADDITIONAL MONITORING

- ECG monitoring: this can guide resuscitation by diagnosing dysrhythmias, ischemia, cardiac injury.
- Urinary catheters: Output of urine can guide fluid replacement; it reflects renal perfusion. (note: prior to catheter insertion urethral injury should be excluded- suspect if there is blood at meatus, pelvic fracture, scrotal blood, perineal bruising.) Per rectum and genital examination are mandatory prior to catheter insertion.

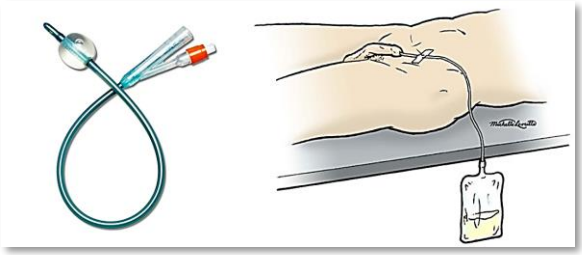


Figure 25: A urinary catheter

- Gastric catheters are inserted to reduce aspiration risk. Suction should be applied.
- Other monitoring: monitoring of resuscitation by measuring various important parameters:
  - Pulse rate, blood pressure, ventilator rate, arterial blood gases, body temperature and urinary output.

- Pulse oximetry measure oxygen saturation



Figure 26: A pulse Oximeter

- Remember: blood pressure is a poor measure of perfusion.

- In trauma centers, teamwork should ensure critically injured patients are evaluated as diagnostic procedures are performed simultaneously, thus reducing the time to treatment.
- A team approach is demanding of personnel and resources and in smaller institutions, non-hospital settings or with mass casualties, available personnel and resources can rapidly be overwhelmed.

SECONDARY SURVEY

- This begins after the "ABCDE" of the primary survey, once resuscitation is underway and the patient is responding with normalization of vital signs.
- The secondary survey is essentially a head-to-toe examination.
- Completion of the history and reassessment of progress, vital signs.
- The secondary survey thus comprises of History (AMPLE) and physical examination.
  - A- allergies
  - M- medication currently used
  - P- past illnesses/ pregnancy
  - L-last meal
  - E- events/Environment related to injury
- The physical examination will repeat some examinations already undertaken in the primary survey and will be further informed by the progress of the resuscitation.
- Examination aims to identify serious injuries, occult bleeding etc.
- A review of neurological status including GCS score is also undertaken.
- Back and spinal injuries are commonly missed and pelvis fractures cause large blood loss which is often underestimated.

PHYSICAL EXAMINTION- HEAD TO TOE

- Head- head and face fracture, CSF leaks from nose and ears.
- Eyes
  - Record the size and reaction to light (fixed and dilated pupils suggest severe brain damage)
  - Always ask the conscious patient if he can see with both eyes.
  - Look for eye injury in the unconscious patient.

- In unconscious patients check for the corneal reflex.
- Examine the conjunctiva and tongue for pallor and cyanosis. You are not looking for signs of anemia but rather the pale white tongue of massive hemorrhage or a discolored purplish tongue suggestive of hypoxia

- Mouth
  - Can a patient open and close his mouth?
  - Do the teeth occlude properly?
  - Failure to do either of these suggests a fracture or dislocation of the mandible.
  - Is the mandible stable?
- Chest
  - Is there pain on breathing?
  - Watch the breathing pattern, is there a paradoxical movement or inequality between the sides of the chest?
  - Assess the depth of breathing, does the patient use accessory muscles for breathing?
  - Are there bruises or penetrating wounds?
  - Palpate for fractures and surgical emphysema
  - Is the trachea centrally positioned or shifted?
  - Percuss the chest, are both sides equal? Is there any hyper-resonance suggestive of a pneumothorax, or dullness suggestive of hemothorax?
  - Auscultate for reduced air entry and added sounds. Coarse crepitations may suggest aspiration of blood or vomit.
- Abdomen
  - The following may suggest intra-abdominal injury.
    - Abdominal or shoulder tip pain
    - Reduced movement with respirations
    - Distension
    - Bruising
    - Rigidity
    - Tenderness
    - Absent bowel sounds
  - If there is major hemorrhage within the abdomen, signs of hypovolemic shock will be present. The abdominal organs prone to trauma are spleen and liver. These organs can rupture and cause massive hemorrhage.
- Pelvis
  - Compress the iliac bones and spring the pubis to determine whether there is a fracture.
  - Look for blood at external meatus of the urethra or if catheterized check for hematuria, which would suggest a urethral injury or bladder injury.
  - Is the bladder full, empty or ruptured?

- Limbs
  - Look for swelling, bruising, rotation or deformity.
  - Feel distal pulses e.g. dorsalis pedis and radials.
  - Test sensation
  - Palpate all bones unless there is severe pain or obvious injury
  - Move all joints unless painful
  - Always examine the distal pulses when there is a feature or soft tissue injury to a limb
  - Record all injuries and wounds carefully in the patient notes.
  - A trauma form with a two figures of a body, one front and one back view will help to record injuries and it is quicker to draw the site of a laceration than to describe it.

ADDITIONAL INVESTIGATIONS

- A range of further diagnostic tests and procedures may be required after the secondary survey. These include:
  - X-rays- Skull, lateral cervical spine, Chest, Abdominal/Pelvic
  - Focused abdominal sonogram for trauma (FAST) is used for rapid detection of intra-abdominal bleeding that often occurs early in the resuscitative process
  - Ultrasound investigations
  - Bronchoscopy
  - CT/MRI scans: only done for hemodynamically stable patients
  - Angiography

DEFINITIVE CARE

- Choosing where definitive care should continue most appropriately will depend on results of the primary and secondary surveys (what's the final diagnosis?)
- Refer to appropriate closest facilities available.

FLUIDS, ELECTROLYTE IMBALANCE AND NUTRITION

FLUIDS, ELECTROLYTES AND ELECTROLYTE IMBALANCE

- Surgical illness and operative intervention disrupt homeostasis and lead to changes in fluid, electrolyte and acid-base balance.
- Total body water constitutes 50-70% of total body weight.
- Total body water is greater in lean individuals because fat contains little water (60%).
- Total body water is greater in newborns (70%) then decreases with age to around 50%.
  - Example: an average 70kg man would have 42 litters of water since 1L of water=1kg.
- The total body water is divided amount 2 compartments:
  - Intracellular fluid (ICF): 22-30L
  - Extracellular fluid (ECF): 10-15L
- Intracellular fluid accounts for 2/3 of total body water.
- Intracellular fluid is mostly in skeletal muscle mass thus it is slightly lower in females (50%) than males (60%).
- The cell wall separates the ICF from the ECF and acts as a semipermeable membrane.
- Extracellular fluid accounts for 1/3 of total body water. ECF is made up of plasma (1/4 of ECF)-3L and Interstitial fluid (3/4 of ECF)-10-12L.
- The capillary membrane separates plasma and interstitial fluid and acts as a semipermeable membrane.

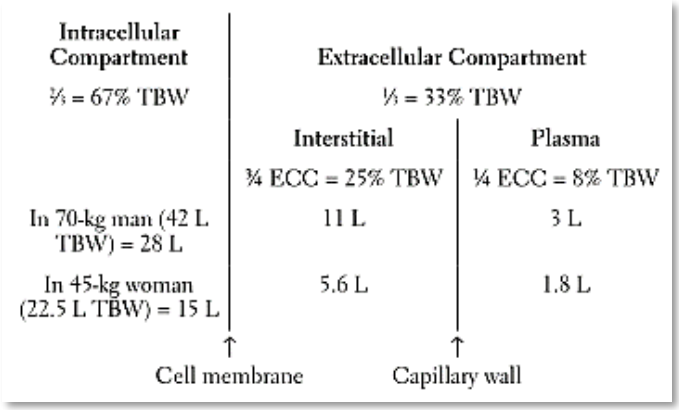


Figure 27: Body fluids

- Water flows freely between the 3 compartments, shifting compartments to maintain osmotic equilibrium between them.
- Hydrostatic pressure at the arteriolar end of the capillary and the oncotic pressure (25mmHg) of plasma at the venular extremity govern the bidirectional movement of water between the plasma and interstitial fluid across the capillary bed.
- The exchange of fluid across the capillary membranes is essential for supply of nutrients and oxygen to the tissues and the elimination of waste products of metabolism.
- Osmolality is the amount of solute dissolved in a solvent like water measured in weight (Kg) i.e. per Kg of solvent.
- Osmolarity is the amount of solute dissolved in a solvent like water measured in volume (L) i.e. per L of solution.
- The normal plasma osmolality (total particle concentration) is 280-295 mosmol/Kg H_2O.
- The main cation in plasma is sodium.
- The main anions in plasma are chloride and bicarbonate ions.
- In normal state, plasma electrolytes account for the majority of the plasma osmolality. There is a good correlation between the plasma osmolality and plasma sodium concentration i.e. a high plasma sodium concentration signifies hyperosmolality and vice versa.
- Plasma osmolality= $2[Na] + \frac{[glucose\ in\ mg/dl]}{18} + \frac{[BUN]}{2.8}$
- Note [Na] is in mmol/L and [BUN], [glucose] in mg/dl.
- Osmolar gap= measured osmolality- calculated plasma osmolality
- Normal osmolar gap <10mmol/L
- If osmolar gap> 10mmol/l suspect lactic acid, ketones, methanol, ethanol.

- As a rule of thumb:
  - Fluid input should match fluid output
  - The daily (24-h) requirements of an average subject are 100 mmol (2mmol/Kg) Sodium and 60 mmol (1mmol/Kg) Potassium.

Input	Volume (mL)	Output	Volume (mL)
Oral liquids	1500	Urine	1500
Water in food	1000	Stool	300
Water of oxidation	300	Lungs	500
		Skin	500
Total	2800		2800

Figure 28: Daily requirements of a normal 70-Kg man

INTRAVENOUS FLUID THERAPY

- Indications:
  - Preoperative resuscitation e.g. before emergency surgery, elective surgery in a jaundiced patient
  - Replacement of abnormal losses e.g. dehydration due to vomiting, diarrhea, ileostomy bags, shock due to hemorrhage or sepsis or burns
  - Provision of normal daily requirements if patient is nil by mouth (total parenteral nutrition).
  - Postoperative resuscitation
  - Electrolyte disorders
- Patients not eating or drinking must be provided with their daily requirements i.e. 2.5-3L of water, 100mmol Sodium, 60 mmol Potassium, 5mmol Calcium and 1mmol magnesium.
- Types of fluid:
  - Crystalloids: used to provide normal daily requirements and replace additional losses.
    - Normal saline (0.9% sodium chloride): 1L contains 154mmol of NaCl.
    - Dextrose saline: 1L contains 31 mmol NaCl and 40g dextrose
    - 5% Dextrose: 1L 5% dextrose contains 50g dextrose
    - Hartmann's solution (Sodium lactate solution or Ringer's lactate): replacement fluid favored by anesthetists because it is a physiological

mixture of ions and water. It should be avoided in liver failure but since it does not contain glucose it can be used in diabetics. 1L Hartmann's solution contains 3 mmol Calcium, 4mmol Potassium, 28mmol Bicarbonate ions (lactate), 109 mmol Chloride and 130mmol Sodium ions.

- Colloids:
  - Natural e.g. blood, albumin
  - Synthetic e.g. gelatin-based infusions

- Potassium can be added to Normal saline and Dextrose saline in the form of potassium chloride (KCL).
- Calculation of drop rate of IV fluids:
  - 1ml = 16 drops in usual drip set. For microdrip set 1ml=60 drops.
  - Drop rate (/min)= Quantity of fluid required in L/day x 10
    - E.g. if 2.5 liters is need per day, Drop rate= 2.5 x 10= 25 drops/minute
  - Number of drops/min= Fluid volume (ml) to be infused divided by 4
    - E.g. 100ml/hour means 25 drops/minute
  - Number of microdrop/min= Volume in ml/hour
    - E.g. 50 microdrop/min =50ml/hr
- Complications of fluid therapy:
  - Fluid overload: pulmonary edema and cardiac failure
  - Thrombophlebitis, hematoma and cellulitis in local area
  - Pyogenic reaction and bacteremia
  - Air embolism

FLUID IMBALANCES

- Fluid status is judged by examination of the patient every day:
  - Check for signs of dehydration
  - Check blood pressure and pulse
  - Examine the abdomen and chest
  - Check the ankles for edema
- The fluid balance chart should be examined to determine the patient's fluid input and output. The hourly urine output is a good guide to fluid status, urine output should not fall below 30ml/h.
- There 2 major regimens used for fluid replacement. If the patient is also losing additional fluid (e.g. vomiting, nasogastric tube, ileostomy) then this volume must also be measured and replaced over and above the normal requirements using normal saline.

- Regimen one: 1L of Normal saline (over 8 hours plus) and 2L of 5% dextrose with 20mmol KCL added to each bag (over 8 hours plus)
- Regimen two: 3L of dextrose-saline with added 20 mmol KCL to each 1L over 8 hours plus.

- Assessment of adequacy of resuscitation:
  - Clinical history and observation: Pulse, blood pressure, skin turgor
  - Urine output: oliguria is defined as <0.5 ml/kg per h
  - Central venous pressure (CVP) or pulmonary capillary wedge pressure
  - Response of urine output or CVP to fluid challenge:
    - 200-250ml bolus of colloid is administered as quickly as possible
    - Response in CVP or urine output should be seen within minutes
    - Size and duration of the CVP response rather than actual value recorded is more important

WATER EXCESS

- It can be divided into:
  - Water and salt excess: occurs in CCF, cirrhosis, nephrotic syndrome, hypoproteinemia, renal failure, excessive saline infusion.
  - Water intoxication (predominant water excess): occurs in TURP (transurethral resection of prostate) when irrigating fluid water or glycine is used, excess infusion of 5% dextrose only, SIADH section (with lobar pneumonia, empyema, oat cell carcinoma and head injury), psychogenic polydipsia
- There is an expansion of the intracellular and extracellular fluid compartments and a low serum sodium.
- Clinical features:
  - Widespread edema:
    - Gain in body weight (most sensitive and consistent sign)
    - Circulatory overload: tachycardia, pulmonary edema, hypertension, distended neck veins
    - Bilateral basal crepitations and ascites
    - Pedal edema
  - Irritability, drowsiness, weakness, convulsions and coma
  - Nausea and vomiting
  - Passing dilute urine
  - Raised central venous pressure and pulmonary capillary wedge pressure
- Investigations:

- Full blood count: hematocrit
- Urea and Electrolytes: low sodium, low potassium, low blood urea.

- Treatment is by water restriction, observation (preferably in ICU), management of electrolyte imbalance and sometimes administration of hypertonic saline.
- Administration of diuretic and hypertonic saline should be avoided as it can cause rapid changes in serum sodium water level which will lead to neuronal demyelination and fatal outcome.

WATER LOSSE (VOLUME DEFICIT)

- This is the most common fluid disorder.
- Both ECF and ICF are lost but it is usually ECF loss which is more important and assessed
- It can be:
  - Isotonic volume depletion: both salt and water are lost and this leads to hypovolemia. Fluid loss is only of the ECF and so early intravascular volume reduction occurs causing hypotension.
    - Features: hypotension and decreased tissue perfusion. Dry tongue, rapid pulse, cold clammy extremities, sunken eyes, oliguria, raised blood urea, decreased urinary sodium. Hypovolemia can be mild (<2L fluid loss), Moderate (2-3 L fluid loss) or severe (>3 L loss)
  - Loss of only water with minimal loss of electrolytes which leads into dehydration with proportionate decrease in total body water. As ECF including intravascular fluid loss is less, hypotension is less.
    - Features: severe thirst, confusion and convulsions due to hypernatremia. Blood pressure is relatively normal. Dehydration can be mild (weight loss 5%), moderate (10%) and severe (15%)
- Causes include:
  - Losses that mimic ECF
    - Hemorrhage
    - Loss of GI fluid- vomiting, NG tube suction, diarrhea, fistula drainage
    - Postoperative fluid sequestration (third spacing): intestinal obstruction
    - Intra-abdominal and retroperitoneal inflammation (e.g. pancreatitis, peritonitis)
    - Systemic inflammatory response syndrome (SIRS), burns, sepsis, pancreatitis

- Losses that are principally water
  - Fever
  - Osmotic diuresis
  - Diabetes insipidus
  - Prolonged water deprivation
  - Inadequate input during procedure

- Management:
  - Evaluation is done by doing serum sodium, urinary sodium and blood urea
  - Isotonic volume depletion: give 0.9% normal saline.
  - Pure water depletion: correct by more water intake/intravenous 5% dextrose
  - Monitoring fluid therapy by skin and tongue examination, weight gain, pulse, blood pressure, central venous pressure and pulmonary capillary wedge pressure.

ELECTROLYTE IMBALANCE

- The concentration of electrolytes is closely maintained in ECF and ICF.

Ion	*ICF*	*ECF and plasma*
Sodium	10 mmol/L	140 mmol/L
Potassium	150 mmol/L	4.5 mmol/L
Chloride	Trace only	105 mmol/L

Figure 29: Electrolyte concentrations in ECF and ICF

HYPONATREMIA

- This is the most common acute biochemical disturbance encountered in surgical practice.
- Normal sodium ECF concentration= 135-145 mEq/L
- Hyponatremia is low serum sodium (<130mmol/L) but treatment warranted with severe <100mmol/L in the acute type or <115mmol/L in the chronic type.
- Hyponatremia can be
  - Acute- presents as neurological manifestations
  - Chronic- causes pontine myelinolysis. It presents as behavioral changes, progressive weakness, and cranial nerve palsies.
- Causes:
  - Hypovolemic hyponatremia: due to hypovolemia by the following:
    - GI losses (NG tube suctioning, vomiting, diarrhea)

    - Renal losses
    - Diuretic therapies e.g. furosemide and thiazide diuretics
  - Euvolemic hyponatremia: due to
    - Hypothyroidism
    - SIADH
    - Adrenal insufficiency (Addison's disease)
  - Hypervolemic hyponatremia: rapid absorption of fluid occurs into intravascular compartment leading to pulmonary and cerebral edema.
    - Congestive heart failure
    - Cirrhosis
    - Nephrotic syndrome
- Clinical features:
  - Asymptomatic unless severe or acute
  - Irritability, disorientation/confusion, coma, seizures, Abdominal cramps, Nausea, vomiting, and pitting edema over the sternum
  - When plasma sodium falls below 120mmol/L the patient becomes confused. There is severe contraction of ECF with hypovolemia, poor venous filling, dry coated tongue, oliguria with dark scanty urine, dry skin with loss of turgor (dry wrinkled skin) and sunken eyeball.
  - If sodium levels decreases below 110mmol/L convulsion and coma may ensue.
  - In chronic hyponatremia- hypothermia, reduced tendon reflexes and pseudobulbar palsy.
- Investigations:
  - Serum sodium (low)
    - Sodium deficit= (125- Present serum sodium) x body weight in Kg x 0.6
  - Urine sodium (low)
  - Serum and Urine osmolarity

MANAGEMENT

- Treat underlying cause. Losses of sodium can be replaced by increasing sodium intake without increasing daily fluid intake (e.g. giving normal saline continuously), water retention should be treated by fluid restriction.
- CNS manifestations
  - 200ml 5% NaCl over 6 hours. Monitor serum sodium hourly.
  - Aim to increase sodium to 120mmol/l at a rate of 0.5-1.0 mmol/l per hour
  - If no CNS symptoms, do not use hypertonic saline.

- Once serum sodium is around 120 mmol/L, stop active therapy and restrict fluid intake to approximately 500ml/day.

- Asymptomatic: Can use conservative measure like fluid restriction may be enough
- SIADH
  - Restrict fluid intake to 50-60% of estimated maintenance fluid requirements (+/- 1L/day)

HYPERNATREMIA

- Normal sodium ECF concentration= 135-145 mmol/L
- This is defined as a serum sodium >150 mmol/L
- Excess infusion of normal saline cause overload in circulating salt and water. It is usually due to water deficit.
- Hypernatremia types include:
  - Euvolemic hypernatremia (pure water loss): due to failure of water intake like in comatous patients, bedridden people, postoperative patients and in patients with high fever leading into extra-renal loss of water. It can occur in diabetes insipidus or chronic renal failure as renal loss of water.
  - Hypovolemic hypernatremia (among loss of water and sodium, more water is lost than sodium): it is due to vomiting, diarrhea, more undue sweating (extra-renal), osmotic diuresis by glucose/mannitol (Renal)
  - Hypervolemic hypernatremia (both sodium and water gain but sodium is more than water gain): seen in more salt intake, excess steroids, sodium bicarbonate/hypertonic saline infusion (salt gain).
- Causes:
  - Water loss more than electrolyte
  - Cardiac failure
  - GIT losses
  - Renal losses (Renal dysfunction)
  - Diabetes insipidus
  - Diabetes mellitus
  - Increase in sodium
    - Hyperaldosteronism
    - Cushing's
    - Excessive saline or Sodium bicarbonate infusion
  - Drugs: NSAIDs, corticosteroids

- Clinical features:
  - Pitting edema
  - Puffiness of face
  - Increased urination
  - Often dilated jugular veins
  - Features of pulmonary edema
- Investigations:
  - Serum Sodium
  - Urine Sodium
  - Renal function tests
  - Hematocrit

MANAGEMENT

- Correct water deficit- rehydrate patient if dehydrated then administer half strength saline (0.45%) and later 5% dextrose i.e. gradual controlled correction is done. Otherwise cerebral edema and hyperglycemia can develop. There should be oral and nasogastric administration of water/fluids.
- Stable/asymptomatic patients
  - Take it easy (hypernatremia should be corrected slowly)
  - Replace fluids orally
- Unstable/symptomatic patients
  - IV fluids with NS, DNS
  - Avoid 5% dextrose as sodium may drop too fast
  - Rate of lowering serum sodium 0.5-1mmol/hr, aim for 12 mmol/L in 24hours and not more than this.
  - Estimate the effect of 1 liter of any infusion on serum sodium
  - Change in serum Sodium= $\frac{Infusion\ sodium - Serum\ sodium}{Total\ body\ water + 1}$
  - The answer is in mmol/L. The formula helps to calculate the infusion rate so that you do not exceed 1 mmol/min.

HYPERKALEMIA

- Normal potassium ECF concentration= 3.5-5.5 mEq/L
- Hyperkalemia is a serum potassium >5.5 mmol/L.
- Potassium excess is a dangerous condition which can cause sudden cardiac arrest.
- Causes:
  - Trauma and tissue destruction
  - Tumor necrosis
  - Crush injuries
  - Burns
  - Renal failure
  - Excessive intake of potassium rich food and supplements
  - Metabolic acidosis
  - Addison's disease
  - Diuretic therapy (potassium sparing diuretics), cyclosporine, beta blockers
  - Transfusion of stored blood
  - Insulin deficiency
  - Diabetic ketoacidosis
  - Familial hyperkalemic periodic paralysis
- Clinical features:
  - Symptoms: Muscle weakness, diarrhea, colickly abdominal pain
  - Signs:
    - Ascending paralysis with respiratory failure
    - Cardiac instability, ventricular fibrillation, cardiac arrest
    - May have signs of acute kidney injury or metabolic acidosis
- Investigations:
  - Serum potassium
    - Mild hyperkalemia: 5.5-5.9
    - Moderate hyperkalemia: 6.0-6.5
    - Severe hyperkalemia: >6.5 and ECG changes
  - ECG findings:
    - Bradycardia
    - Flat P waves
    - Increased PR interval
    - AV block
    - Wide QRS complex
    - Tall peaked symmetrical T waves (most important to remember)

MANAGEMENT

- Stop source of potassium (Oranges, bananas, ACEI, potassium sparing diuretics, septrin, heparin, NSAIDs, Beta-blockers)

Mild hyperkalemia (5.5-5.9)	Moderate hyperkalemia (6.0-6.5)	Severe hyperkalemia (>6.5)
• Push Potassium out of the body ➢ Furosemide 1mg/kg IV (hydrate patient first if dehydrated) ➢ Kayexalate/sodium Resonium 15-30g in 50-100ml 20% Sorbitol or with lactulose PO/PR ➢ Consider hemodialysis if refractory	• Protect the heart: 10mls 10% Calcium Gluconate over 10 mins. Effect is temporary but can be repeated after 15 minutes. • Push Potassium into cells ➢ 50mls 50% Dextrose with 10IU soluble insulin over 15-20 mins. May be given 2-4 hourly as required. Monitor serum glucose and potassium 2-4 hourly ➢ 10-20mg Nebulized Salbutamol (or 0.5mg in 100ml of 5% glucose over 15 min-rarely used) • Push Potassium out of the body ➢ Furosemide 1mg/kg IV (hydrate patient first if dehydrated) ➢ Kayexalate (polystyrene sulphonate) /sodium Resonium 15-30g in 50-100ml 20% Sorbitol or with lactulose PO/PR • Consider hemodialysis if refractory	• Protect the heart: 10mls 10% Calcium Gluconate over 10 mins. Effect is temporary but can be repeated after 15 minutes. • Push Potassium into cells ➢ 50mls 50% Dextrose with 10IU soluble insulin over 15-20 mins. May be given 2-4 hourly as required. Monitor serum glucose and potassium 2-4 hourly ➢ 10-20mg Nebulized Salbutamol (or 0.5mg in 100ml of 5% glucose over 15 min-rarely used) • Push Potassium out of the body ➢ Furosemide 1mg/kg IV (hydrate patient first if dehydrated) ➢ Kayexalate (polystyrene sulphonate)/sodium Resonium 15-30g in 50-100ml 20% Sorbitol or with lactulose PO/PR ➢ Consider hemodialysis if refractory

HYPOKALEMIA

- This is a serum potassium <3.5, it may be associated with hypomagnesemia and hypocalcemia.
- Causes:
  - GIT losses (NG tube suctioning, vomiting, diarrhea)
  - After trauma or surgery
  - Pyloric stenosis with gastric outlet obstruction
  - Duodenal fistula, ileostomy
  - After ureterosigmoidostomy
  - Insulin therapy
  - Poisoning
  - Drugs like beta agonists
  - Renal losses (Renal disease)
  - Metabolic alkalosis
  - Potassium wasting diuretics
  - Cushing's disease (Sodium retention leads to potassium loss)
  - Familial periodic paralysis
- Clinical features:
  - Symptoms:
    - Muscle weakness
    - Fatigue
    - Constipation
    - Muscle cramps
    - Inability to produce concentrated urine so can cause nocturia and polyuria
  - Signs:
    - Slurred speech
    - Muscle hypotonia
    - Paralytic ileus
    - Ascending paralysis
    - Reduced reflexes
    - Tetany when associated with alkalosis (Alkalosis is due to increased reabsorption of bicarbonate and excretion of hydrogen ions from the proximal convoluted tubule in the kidneys)
    - Arrhythmias

- Investigations:
  - Check potassium, other electrolytes and serum pH.
  - ECG
    - Flat or inverted T wave
    - Prominent U wave
    - QT interval prolongation
    - Depression of ST segment

MANAGEMENT

- 20mmol KCL in 250-500ml Normal saline over 1 hour. Check Potassium before repeating dose and ECG monitoring.
- Consider 6-12ml (5-10mml/l) 20% $MgSO_4$ or 2ml 50% diluted in 50ml over 1 hour.
- It cardiac arrhythmia or arrest give 2mmol KCL per minute IV for 5mins. Repeat once only if necessary
- Oral Potassium supplements if potassium >3.0.
- Hypokalemic alkalosis which occur in pyloric stenosis should be treated carefully by IV potassium as there will be severe potassium loss.

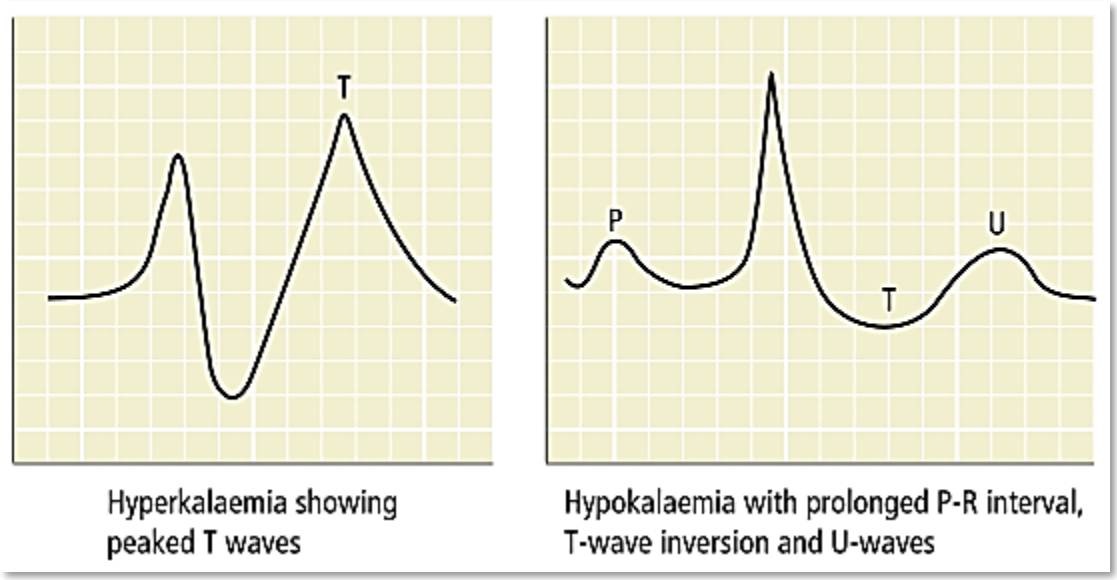


Figure 30: ECG changes in both hypo and hyperkalemia

HYPERCALCEMIA

- Normal serum calcium: 2.2-2.6mmol/L. Calcium is usually bound to albumin and the levels of both calcium and albumin should be measured together (to correct for low serum albumin, 0.025 should be added to the calcium for every 1g/L that the albumin is lower than 40g/L)
- Corrected serum Calcium >2.65mmol/L
- Causes
  - Endocrine: Hyperparathyroidism, ectopic PTH production, thyrotoxicosis, Addison's disease
  - Bone disease: secondary tumor deposits, myeloma, lymphoma and paget's disease
  - Malignancy
  - Sarcoidosis
  - Drugs e.g thiazides
  - Vitamin D intoxication especially in renal patients
  - Milk alkali syndrome
- Clinical features: usually asymptomatic until serum calcium <3.5 mmol/L but above 3.5mmol/L clinical features include
  - Muscle weakness
  - Lassitude
  - Drowsiness
  - Hyperreflexia
  - Anxiety and mania may develop with coma as a terminal event
  - Polyuria and polydipsia indicate impaired renal concentration
  - Nausea, vomiting, constipation and peptic ulceration may occur
- Investigations
  - Serum Calcium, Urea + Electrolytes, Albumin, Magnesium, phosphate, ALP, serum electrophoresis
  - PTH
  - X-rays
  - ECG
    - Short QT interval
    - Wide QRS complex
    - Flat T waves
    - AV block
    - May have fatal arrhythmias (frequent association with hypokalemia increases risk of arrhythmias)

MANAGEMENT

- Treat underlying cause
- Hydrate with normal saline 500ml/hr until urine output >200ml/hr then reduce 100-200ml/hr.
- Furosemide 1mg/kg only when patient has been hydrated or if in cardiac failure
- Hemodialysis or peritoneal dialysis with low calcium dialysate. Hemodialysis is preferred.
- Prednisolone 40mg daily especially for Vitamin D intoxication, sarcoidosis, multiple myeloma, metastasis
- Pamidronate 60-90mg in 500-100ml NS infusion.

HYPOCALCEMIA

- This is a serum calcium concentration <2mmol/L in a dramatic clinical picture characterized by tetany.
- In its mildest form there may be paresthesia and muscle cramps.
- Full-blown picture shows hyperexcitability of the nervous system expressed as carpopedal spasm (severe tonic contractions of the muscles of the hands: the fingers are bunched together and flexed at the metacarpophalageal joints and wrist is acutely flexed- main d'accoucheur, the toes are flexed and the ankle joint is also acutely plantar flexed).
  - Latent tetany may be demonstrated by Chovstek's sign (Tapping over the facial nerve induces twitching of the facial muscles)
  - Trousseau's sign (Carpal spasms induced by inflating a sphygmomanometer cuff above systolic pressure)
- Causes:
  - Hypoproteinemia
  - Vitamin D deficiency
  - Parathyroidectomy
  - Acute pancreatitis
  - Medullary carcinoma of thyroid
  - Idiopathic
- Acute hypocalcium is treated by slow infusion of calcium gluconate (10% solution).

HYPERMAGNESEMIA

- It is rare.
- Serum magnesium >2.5 mmol/L (normal serum magnesium= 1.5-2.5 mmol/L and intracellular magnesium= 26mmol/L)
- Magnesium is mainly deposited in bone (60%). It is a cofactor for many enzymes necessary in phosphorylation of glucose in the cell and ATP utilization in muscle fibers.
- Daily required dietary intake is 0.4g. it is reabsorbed well in proximal renal tubule
- Causes:
  - Advance renal failure: treated with magnesium contain anatacids
  - Ketoacidosis
  - Intentionally produced hypermagnesemia while treating pre-eclampsia.
- Clinical features:
  - Loss of tendon reflexes (most common)
  - Neuromuscular depression
  - Flaccid quadriplegia
  - Respiratory paralysis
  - Somnolence
  - Hypotension

HYPOMAGNESEMIA

- Serum magnesium <1.5 mmol/L
- Causes:
  - Malnutrition
  - Alcohol
  - Large GI fluid loss
  - Patients on total parenteral nutrition
- Clinical features:
  - Hyper-reflexia
  - Muscle spasm
  - Paresthesia
  - Tetany
  - It mimics hypocalcemia. It is often associated with hypokalemia and hypocalcemia
- Treatment: 2g (16 mEq) of magnesium sulphate slow intravenously in 10 minutes. Later maintenance dose of 1mEq/kg/day as slow continuous infusion is given/oral magnesium as needed.

ACID-BASE BALANCE

- Normal blood pH= 7.35-7.45
- Factors which control the pH:
  - Buffer systems
    - Bicarbonate buffer
    - Protein buffer
    - Phosphate buffer
  - Renal control of pH
  - Respiratory control of pH
- Acidosis is pH of blood less than 7.35
- Alkalosis is pH of blood more than 7.45
- Note: the lungs and kidneys act in concert such that in the presence of mild renal impairment, increased acid removal is achieved by the lungs and vice versa.
- Certain disease states overwhelm these homeostatic mechanisms and result in significant accumulation acid or base with corresponding deviations from normal pH.

METABOLIC ALKASOSIS

- Primary base excess i.e. bicarbonate. A standard bicarbonate above 27 mmol/L.
- Causes:
  - Repeated vomiting due to any cause. Commonly seen in pyloric stenosis (hypokalemic alkalosis)
  - Nasogastric suctioning
  - Excess alkali ingestion e.g. antacids
  - Hypokalemia and diuretics, Excess citrate
  - Cortisol excess either due to over administration or Cushing's syndrome
- Clinical features:
  - Cheyne-stokes breathing (irregular breathing- short breaths gradually increasing to deep breaths then reducing again until breathing appears to stop) with period of apnea 5-30 seconds
  - Tetany due to alkalosis. More often latent tetany which is revealed by Trousseau's sign
- Investigations:
  - Serum electrolytes
  - Arterial blood gas analysis
    - pH >7.45 (Hydrogen ion <36nmol/L)
    - $PaCO_2$ >6.0 KPa

- Bicarbonate >32mmol/L
- Base excess >+5 mmol/L

- Treatment:
  - Normal saline or double strength normal saline IV infusion with slow IV potassium chloride 40 mmol/L in saline slowly under ECG monitoring.
  - pH more than 7.7 is life threatening and requires rapid correction by infusion dilute hydrochloric acid or ammonium chloride however with care and monitoring.

RESPIRATORY ALKALOSIS

- Arterial PCO_2 is below normal
- Causes:
  - Hyperventilation during anesthesia, due to head injury/severe pain
  - High altitude
  - Hyperpyrexia
  - Encephalitis, hypothalamic tumors, drugs like salicyclates, due to cirrhosis of liver
  - Apprehension and Hysteria
- Clinical features:
  - It can be acute or chronic
  - Headache, tingling, circumoral anesthesia, tightness in chest, tetany, arrhythmias
- Investigations:
  - Serum electrolytes
  - Arterial blood gas analysis
    - pH >7.45 (Hydrogen ion <36nmol/L)
    - $PaCO_2$ <5.3 KPa
    - Bicarbonate 22mmol/L
    - Base excess +2 mmol/L
- Management:
  - Oxygen therapy
  - Treat underlying cause
  - Acetazolamide in high altitude
  - Respiratory suppression due to alkalosis is treated by carbon dioxide

METABOLIC ACIDOSIS

- It is an excess acid or base deficit. A standard bicarbonate below 21 mmol/L
- Causes:
  - Increase in fixed acid:
    - Diabetic ketoacidosis
    - Starvation
    - Hypoxia
    - Renal insufficiency
    - Cardiac arrest
    - Excessive excessive
    - Intestinal strangulation. Here anion gap is increased
  - Loss of base
    - Diarrhea
    - Ulcerative colitis
    - Gastrocolic fistula
    - Intestinal fistula
    - Ureterosigmoidostomy causes hyperchloremic hypokalemic acidosis. Here anion gap is normal
- Clinical features:
  - Rapid, deep, noisy breathing (air hunger)- Kussmaul's breathing
  - Cold clammy skin, tachycardia, right heart strain, altered level of consciousness
  - Cardiac arrhythmias, hypotension
  - Anorexia, muscle weakness, vomiting
  - pH below 7.2 is dangerous and life-threatening
  - Capillary stasis
  - Urine is strongly acidic
- Investigations:
  - Serum electrolytes
  - Arterial blood gas analysis
    - pH <7.35 (Hydrogen ion >44nmol/L)
    - $PaCO_2$ <4.7 KPa
    - Bicarbonate <18 mmol/L
    - Base excess <-5 mmol/L
- Management:
  - Correct hypoxia
  - 50mmol of 8.4% sodium bicarbonate infusion IV

- Sodium bicarbonate required in mEq/L= Body weight (Kg) x Base deficit x 0.3

➢ Correct electrolyte imbalances
➢ Treat underlying cause

RESPIRATORY ACIDOSIS

- It is a feature of respiratory failure with high arterial PCO_2 causing fall in pH.
- Causes:
  - ➢ During and after anesthesia
  - ➢ Chronic bronchitis
  - ➢ Emphysema
  - ➢ Thoracic disease
  - ➢ Upper abdominal surgeries and disease.
  - ➢ Respiratory airway obstruction
  - ➢ Myasthenia gravis
  - ➢ Poliomyelitis
  - ➢ Stroke, infection, obesity and hypoventilation
- Clinical features:
  - ➢ Features of hypercapnia: dyspnea, confusion, psychosis, hallucinations, sleep disturbances, tremor, jerks and personality changes.
  - ➢ CNS manifestations are more severe in respiratory acidosis than in metabolic acidosis as lipid soluble Carbon dioxide crosses blood brain barrier easily than bicarbonate
- Investigations:
  - ➢ Serum electrolytes
  - ➢ Arterial blood gas analysis
    - pH <7.38 (Hydrogen ion >44nmol/L)
    - $PaCO_2$ >5.7 KPa
    - Bicarbonate >22mmol/L
    - Base excess <-2 mmol/L
- Management:
  - ➢ Oxygen therapy (should not be used in chronic hypercapnia unless it is really indicted as hypoxia stimulated respiration may be suppressed), ventilator support.
  - ➢ Alkali therapy is not usually used unless acidosis is very severe (below 7.15) or there is severe bronchospasm.

Metabolic acidosis	Metabolic alkalosis
Causes	*Causes*
Hypoxic causes	*Excess H^+ loss*
Lactic acidosis	Nasogastric suction
Non-hypoxic causes	Vomiting
Ketoacidosis	Hypokalaemia
Excess HCO_3^- loss	*Excess alkali*
Renal failure	$NaHCO_3$ ingestion
Drugs (e.g. salicylates)	Diuretics
	Excess citrate
Blood gas picture	*Blood gas picture*
pH < 7.36 (H^+ > 44 nmol/L)	pH > 7.44 (H^+ < 36 nmol/L)
$Paco_2$ < 4.7 kPa	$Paco_2$ > 6.0 kPa
HCO_3^- < 18 mmol/L	HCO_3^- > 32 mmol/L
Base excess < –5 mmol/L	Base excess > +5 mmol/L
Respiratory acidosis	**Respiratory alkalosis**
Causes	*Causes*
Decreased CO_2 excretion	*Hyperventilation*
Hypoventilation	Apprehension
Ventilation–perfusion mismatch	Hysteria
	CNS injury
Airway obstruction	Mechanical ventilation
Increased CO_2 excretion	
Hypermetabolism	
Blood gas picture	*Blood gas picture*
pH < 7.38 (H^+ > 44 nmol/L)	pH > 7.42 (H^+ < 36 nmol/L)
$Paco_2$ > 5.7 kPa	$Paco_2$ < 5.3 kPa
HCO_3^- 22 mmol/L	HCO_3^- 22 mmol/L
Base excess –2 mmol/L	Base excess +2 mmol/L

Figure 31: Acidosis and alkalosis

Anion gap: This is the measurement of the interval between the sum of "routinely measured" cations minus the sum of the "routinely measured anions in the blood.

The anion gap= $(Na^+ + K^+) – (Cl^- + HCO_3^-)$

The anion gap can be normal (16 ± 4mEq/l if the calculation employs potassium and 12 ± 4mEq/L if the calculation does not employ potassium), high or low

The test is commonly performed in patients who present with altered mental status, unknown exposures, acute renal failure and acute illnesses.

A high anion gap indicates metabolic acidosis.

A low anion gap is relatively rare but may occur from the presence of abnormal positively charged proteins, as in multiple myeloma.

NUTRITION

PRINCIPLES OF NUTRITION

- Avoiding of malnutrition is the basic goal in nutrition therapy as malnutrition increases the morbidity and mortality of the disease process and prevents or delays recovery.
- Malnutrition increases the chance of sepsis, prevents wound healing, increases the respiratory complications and decreases the efficacy and tolerance to radiotherapy or chemotherapy.
- Whenever possible enteral route of nutrition should be used ideally. If that is not possible then parenteral nutrition is used.
- Overfeeding should be avoided as it leads into hyperglycemia, hepatic steatosis, raised BUN, and excess CO_2 production.
- Timing and type of nutrition is also important.
- Nutrition therapy reduces protein wasting.
- Immunomodulators like glutamine, arginine and omega 3 fatty acids are also very useful.
- Caloric requirement:
  - Neonatal: 100 kcal/kg/day
  - Adult: 40 kcal/kg/day (25-35kcal/kg)
  - Adult with catabolism: 60 kcal/kg/day
- It is given as:
  - Carbohydrates: 50%
  - Fat: 30-40%
  - Protein: 10-15%
- Caloric values:
  - Carbohydrate= 4 kcal/g
  - Fat= 9 Kcal/g
  - Protein= 4 Kcal/g

INDICATIONS FOR NUTRITIONAL SUPPORT

- Preoperative nutritional depletion
- Postoperative complications: sepsis, ileus, fistula
- Intestinal fistula: High type wherein output is more than 500ml/day. It may be duodenal, biliary, pancreatic, intestinal.
- Pancreatitis, malabsorption, ulcerative colitis, pyloric stenosis
- Anorexia nervosa and intractable vomiting
- Trauma- multiple fractures, fasciomaxillary injuries, head and neck injuries.
- Burns
- Malignant disease
- Renal and liver failure
- Massive bowel resection causing short bowel syndrome

ASSESSMENT AND NUTRITIONAL REQUIREMENTS

- Clinical findings: history and examination, regular weights, body mass index.
- Anthropometric measurements:
  - Body weight
  - Skinfold thickness (e.g. triceps, iliac crest)
  - Mid-arm circumference
  - Grip strength
- Blood tests:
  - Serum albumin
  - Serum transferrin
  - Lymphocyte count
- Nutritional requirements: carbohydrates, fats, proteins, vitamins (includes fat soluble vitamins), minerals, trace elements.

METHODS OF FEEDING

- Methods include:
  - Enteral: preferred for the patient unable to maintain an adequate oral intake but who has a functioning GIT that can be used safely. Early enteral nutrition has been advocated to persevere intestinal structure.
  - Total parenteral nutrition (TPN): can be given via a large peripheral vein or central venous channel

ENTERAL

- GIT is the best route to provide nutrition in order to preserve intestinal structure and the role of the intestine in the immune system.
- Enteral feeding can be delivered by bolus, by gravity or using mechanical pump through:
  - Mouth: needs common sense, cleanliness, compassion.
  - Nasogastric tube: confirmation of the tube in the stomach is made by injecting 5ml of air down the tube and listening through a stethoscope for its bubbling entry into the stomach. Feeding rate is 30-50ml/hours. 5 hours night time gap is given to allow gastric pH to return to normal.
  - Enterostomy: gastrostomy (surgical or endoscopic- percutaneous endoscopic gastrostomy), Jejunostomy (surgical).
- Enteral feeding can be done initially by providing supplement drinks. If this is not sufficient, then an elemental diet can be given through a fine-bore nasogastric tube directly into the stomach or small bowel. These types of feeds (e.g. osmolite) provide approximately 1912 Kcal (8000 Kj) of energy plus about 70g protein in 2-3L.
- Types of feed:
  - Polymeric (near-normal composition): contain intact proteins, starch and long-chain fatty acids. They are useful in patients with functioning stomach and normal digestive capacity.
  - Disease specific: they are tailored for requirements of specific diseases e.g. feeds for patients with liver disease are deficient in branched-chain amino acids as they can exacerbate encephalopathy.
  - Elemental: chemically defined feeds that contain simple amino acids, oligosaccharides and monosaccharides. These require minimal digestion and are useful in patients with intestinal fistulae but are far from palatable.
- Indications:
  - Long term feeding
  - Dysphagia e.g. stricture, stroke
  - Chronic disease e.g. neoplasia
  - Malnutrition
  - Sepsis
  - Burns
  - Major surgery
  - Coma/ICU

Problems with tube feeding	Complications of enteral feeding
• Blockage • Nausea and vomiting, aspiration • Hyperosmolarity • Diarrhea • Tube discomfort • Cholestasis	• Aspiration • Wound infection and leak • Diarrhea due to rapid feeding or hyperosomolarity • Hyperglycemia • Electrolyte imbalance: Hypokalemia • Refeeding syndrome due to severe hypokalemia and hypophosphataemia

ADVANTAGES OF ENTERAL NUTRITION

- Enteral nutrition preserves mucosal protein, digestive enzymes, IgA secretion, prevents mucosal atrophy and bacterial translocation.
- It is more physiological as nutrients pass through liver (first filter). Gallstone formation is prevented (unlike long term TPN) by stimulating gall bladder motility.
- It has got less serious complications.
- It is cost effective
- It supplies glutamine and short chained fatty acids to gut.

CONTRAINDICATIONS OF ENTERAL NUTRITON

- Intestinal obstruction, GI bleed, paralytic ileus, severe diarrhea, high output fistula.
- Low cardiac output, hemodynamically unstable patient.
- If safe access to enteral feeding is not present.
- Anticipated complications if thought to be present should be avoided.

GASTROSTOMY

- It is done if feeding is required for more than 1 month.
- Indications:
  - Severe malnutrition
  - Major surgeries
  - Severe sepsis
  - Trauma
  - Head and neck surgeries
- Types:
  - Based on duration of use:
    - Temporary
    - Permanent
  - Based on lining:
    - Serosal lined (temporary)
    - Mucus lined (permanent)
  - Based on technique
    - Stamm temporary gastrostomy: after opening the abdomen, anterior wall of the stomach is opened. Feeding tube (Malecot's catheter) is placed in positon. 2 layers of purse string sutures are put around the tube/ wound is closed.
    - Kader-Senn temporary gastrostomy
    - Percutaneous endoscopic gastrostomy (popular)- now becoming common method
    - Janeway's mucus lined permanent gastrostomy by creating tunnel in stomach wall.
- Problems in gastrostomy
  - Leak- gastric fistula
  - Infection
  - Aspiration and pneumonia
  - Diarrhea is common-30%
  - Bloating, abdominal cramps
  - Displacement, blockage of the tube
- Contraindications
  - Previous gastric surgeries
  - Intestinal obstruction
  - Gastric outlet obstruction

JEJUNOSTOMY

- These are becoming more popular because of:
  - Comfort
  - Easy to do
  - Can be kept for long time
  - Lesser complication than gastrostomy
- Indications are the same as gastrostomy
- Types
  - Witzel jejunostomy: site of placing jejunostomy is 30cm from duodenojejunal junction.
  - Needle jejunostomy using catheter of small gauge.

TOTAL PARENTERAL NUTRITION (TPN)

- All nutritional requirements are given only through intravenous route, not through the GIT.
- It can be through a central catheter through the subclavian/internal jugular vein where the tip of venous catheter is at distal part of superior vena cava.
- Can also be a peripheral (peripheral parenteral nutrition/ PPN) through a peripherally inserted central venous catheter (PICC) or through a femoral peripheral venous line.
- Indications:
  - Failure or contraindication for any enteral nutrition for 7-10 days.
  - High output abdominal fistulas, duodenal, biliary, pancreatic fistula.
  - Major abdominal surgeries of liver, pancreas, biliary, colonic
  - Septicemia
  - Multiple trauma
  - Short bowel syndrome
  - Severe pancreatitis, bowel ischemia, peritonitis, prolonged ileus
  - Massive GI bleeding, unstable haemodynamically
  - High risk of aspiration
  - Hyperemesis gravidarum
  - Multiorgan failure, head injury, severe burns where upper GIT is severely damaged

  - Severe malnutrition
  - Severe Crohn's disease
  - Inability to swallow e.g. esophageal tumor
- About 5% of hospital admissions require TPN.
- Contraindications:
  - Cardiac failure
  - Blood dyscrasias
  - Altered fat metabolism
- Technique:
  - Using a needle and guide wire a subclavian vein catheter is passed just below the clavicle and fixed securely to the skin.
  - TPN is given through central vein and not through a peripheral vein.
  - Peripherally inserted central catheter (PICC) is also commonly used (PPN)
- Complications:
  - Pneumothorax/hemothorax
  - Cardiac tamponade
  - Sepsis e.g. *Staphylococcus epidermidis, Candida, Pseudomonas*
  - Mineral overload
  - Reactive hypoglycemia
  - Vitamin and mineral deficiencies

REFEEDING SYNDROME

- Refeeding syndrome is occurrence of severe fluid and electrolyte imbalance in severely malnourished individuals while starting the proper feeding enteral or parenteral nutrition.
- It is common in TPN.
- It causes hypomagnesaemia, hypocalcemia and hypophosphataemia leading into myocardial dysfunction, respiratory changes, altered liver functions, altered level of consciousness, convulsions and often death.
- Gradual feeding and correction of magnesium, phosphate and calcium and other electrolytes is important.
- Condition is common in chronic starvation, severe anorexia and alcoholic patients.

BLOOD PRODUCTS AND BLOOD TRANSFUSION

- Blood is a connective tissue found in fluid form. It consists formed elements and plasma. Blood is circulated around the body in arteries and veins by the pumping action of the heart
- Plasma is the fluid component of circulating blood in which the formed elements (Erythrocytes, leukocytes and platelets) are suspended.
- Blood and plasma products are derived from voluntary non-remunerated donors aged 18-65 years.
- Donors are carefully selected and all donations are tested for known markers of disease. These include:
  - Human immunodeficiency virus (HIV)-1 and 2
  - Syphilis
  - Hepatitis B and C viruses
- Modern blood transfusion is based on the intravenous administration of those components of blood that are deficient in a particular rather than transfusion of whole blood. This practice ensures the safe economic use of blood products and increases the therapeutic scope.
- Safe blood transfusion depends on:
  - Careful selection of donors
  - Quality assurance testing of blood and blood products prepared and issued by transfusion centers
  - Blood grouping, antibody screening and cross-matching
  - Standardized blood ordering policies
  - Strict guidelines for administration and monitoring of patients receiving blood products
  - Local and central reporting of adverse effects when encountered
- Documentation of the details of any blood component infused is essential and mandatory including:
  - Indication
  - Date/time of issue
  - Donation number
  - Ordering physician

- Duration of infusion
- Who gave the infusion and who checked it prior to administration
- Time the infusion was commenced

- The patient's red cells are grouped for ABO and Rh (D). The antibody screen is carried out on all the serum samples to detect the presence in the recipient of any clinically important antibody that would hemolyze the transfused cells.
- Cross-matching entails testing the red cells from the donor units against the patient's serum.
- Before transfusing there is a 2 people verification that has to be done to ensure that the information on the transfusion request form, the labels on the unit of blood and the patient identification (from patient notes and wrist band) all agree. This remains the weakest link in the safety chain and most deaths from blood transfusion are related to misappropriation.
- Note: there is no medical or surgical condition that justifies transfusion of less than 2 units of blood.
- Type and screen (group and hold, group and save) is done for operations that do not usually require blood. The patient's serum is kept in the blood transfusion laboratory for 7 days. Should blood be required cross-matched red cell units can be available in 15 minutes.
- Patients do not require a preoperative blood transfusion unless the hemoglobin is less than 8g/dl. The exceptions to this rule are patients with cardiovascular disease or those expected to have covert cardiovascular disease (elderly patients and patients with peripheral vascular disease). These patients are likely to benefit and thus require a blood transfusion when their hemoglobin falls below 9g/dl.

BLOOD SPARING STRATEGIES

- Blood sparing strategies should be considered in all patient who might require a blood transfusion, especially:
  - Jehovah's Witnesses
  - Patients with multiple antibodies
  - Patients with serious anxieties about the transfusion of allogeneic blood.
- Blood sparing strategies include:
  - Preoperative autologous blood donation (pre-deposit)
  - Erythropoietin
  - Acute normovolemic hemodilution
  - Antifibrinolytic drugs
  - Cell salvage

PREOPERATIVE AUTOLOGOUS BLOOD DONATION

- The benefit of autologous, as opposed to allogeneic transfusion is the elimination of disease transmission and allergic and incompatible reactions (other than procedural errors).
- It should only be offered when it is possible to guarantee admission and operative dates and is targeted to patients who are to undergo elective surgery where blood will be lost and whose Hb at presentation is 11.0-14.5g/dl in males and 13.5-14.5g/dl in females.
- Pre-deposit is more expensive than allogeneic blood transfusion. In addition, the blood is wasted if the patient does not need perioperative transfusion as it cannot be administered to other patients.
- If the patient require blood in excess of the predeposited units, all possible advantage is lost thus a balance must be struck between collecting enough units to minimize allogeneic exposure and wastage.

ERYTHROPOIETIN

- This is a hormone produced and secreted by the kidneys and regulate erythropoiesis.
- Recombinant human erythropoietin (alpha and beta) are widely used in anemia associated with renal failure and as blood sparring strategy in patients undergoing major surgery.
- It is administered subcutaneously (600 units/kg) 3 times weekly and on the day of surgery and is preferably accompanied by oral/IV iron therapy.
- EPO is used to counteract blood loss in:
  - Patients scheduled for major surgery under 70 years of age with a presenting HB <13g/dl
  - Patients scheduled for major surgery who refuse allogeneic blood transfusion
  - Jehovah's Witnesses.
- The risk of EPO therapy is deep vein thrombosis, especially if the Hb exceed 13g/dl.
- A 500ml venesection is necessary whenever EPO induces the patient's hematocrit to rise above 0.5. Patients on EPO should have their hematocrit checked weekly.

ACUTE NORMOVOLEMIC HEMODILUTION

- Immediately before surgery usually following induction, 1000ml of blood are removed with replacement of blood volume by crystalloids (with monitoring of central venous pressure).
- The blood is given to the patient if and when it is needed during or after surgery.
- Acute normovolemic hemodilution is useful in fit patients if:
  - A substantial blood loss is anticipated during surgery
  - The patient's initial hemoglobin is high
  - A relatively low target hemoglobin is acceptable

ANTIFIBRINOLYTIC DRUGS

- These include aprotinin (a kallikrein inhibitor) and tranexamic acid.
- They are recommended for patients undergoing cardiac surgery that carries high risk of transfusion such as:
  - Repeat cardiac operations
  - Multiple valve replacements
  - Thoracic aortic operations
  - Patients on aspirin therapy
  - Anticipated long bypass times
- Antifibrinolytic drugs are acceptable to Jehovah's Witnesses

INTRAOPERATIVE BLOOD SALVAGE

- This include automated blood salvage using the cell saver equipment (Haemonetics) and the simpler manual systems for storage and reinfusion of red cells as exemplified by the Solcotrans autologous collection system.
- Both techniques are only applicable to clean operative sites without bacterial, bowel or tumor cell contamination.
- The Haemonetics cell saver system is a completely automated device that aspirates, anticoagulates and filters the extravasated blood from the operative field.
- The red cells are then washed before being transfused in a packed volume of 0.5. This system is used extensively



Figure 32: Cell saver

during liver transplantation and in Jehovah's Witnesses undergoing major surgery.

BLOOD AND PLASMA PRODUCTS

Blood products	Examples
Red cell preparations	• Whole blood • Red cell concentrate (Packed cells) • Leukocyte-poor red cell concentrate • Frozen red cells (not available generally) • Autologous blood
Platelets	• Random donor pooled • Single-donor apheresis • Human leukocyte antigen-matched • Cross-matched
Plasma components	• Fresh frozen plasma (plasma + all clotting factors)- ABO compatibility is essential • Cryoprecipitate (Factor VIII, von Willebrand factor, fibrinogen)- ABO compatibility is essential • Plasma protein fraction (5% albumin in buffered saline) • Salt-poor human albumin (25%) • Factor VIII concentrate • Factor VIIa • Factors II, IX, X • Factor VII • Von Willebrand factor • Antithrombin III • Intravenous IgG ➢ Human normal IgG ➢ Specific hyperimmune IgG (Rh anti-D, tetanus etc.) • Fibrin sealant (glue)

STORED WHOLE BLOOD

- This is blood from which no constituent has been removed.
- Whole blood is only indicated for treatment of acute hemorrhage, hypovolemia is accompanied by an acute reduction in red cell mass, resulting in impaired oxygen carrying capacity at a time when tissue perfusion is compromised.

- It should be reserved for those patients with substantial blood loss and a hematocrit of 0.3 or less after volume replacement with crystalloids and plasma expanders.
- Stored blood has a number of unwanted features
  - Citrate anticoagulant
  - Acid pH (6.6-6.8)
  - High level of potassium (from the stored red blood cells)
  - Ammonia (from erythrocyte adenosine)
  - Reduced red-cell 2,3 diphosphoglycerate (2,3-DPG) which leads to impaired release of oxygen from oxyhemoglobin)
- The use of stored whole blood is declining rapidly because of increasing demand for individual blood products and for economic reasons.

RED CELL CONCENTRATES

- These are solutions of erythrocytes obtained from blood after the plasma has been removed. They also contain some white cells.
- Pack red cells have the same oxygen carrying capacity of blood but a lower volume.
- They are ideal for treatment of anemic patients who invariably have a normal blood volume and are at risk from circulatory overload. Even so, unless the anemia is severe and symptomatic transfusion is no substitute for hematinic (promotion of blood production) therapy and is only indicated in patients how:
  - Do not respond to hematinic treatment (refractory anemia)
  - Are unable to adapt to the reduced oxygen-carrying capacity of the blood and exhibit signs of incipient cardiovascular failure.
  - Require urgent surgery
- Up to 10% of patients develop Alloimmunisation to leukocyte antigens, usually after repeated red cell infusions.
- The antibodies cause severe febrile reactions, which can only be prevented by the use of leukocyte-poor red cell concentrates.

FROZEN RED CELLS

- The use of cryoprotective agents such as glycerol or hydroxyethyl starch allows satisfactory storage of red cells at -80 to -196^{o}C (mechanical freezing or liquid nitrogen storage) for long periods (up to 10 years),

- This method of storage removes leukocytes, platelets and any viral particles thereby reducing the incidence of both transmission of viral disease and Alloimmunisation to leukocyte and platelet antigens.
- Red cells recovered from a frozen bank are of particular value to patients on renal dialysis programs, those with refractory anemias and those with rare cell types and complex antibody mixtures. However, frozen red cells are very expensive and impractical for most patients.

PLATELET CONCENTRATES

- These are solutions containing platelets that are frequently derived from several donors
- They can be obtained by centrifugation of blood from several donors or by platetpheresis of a single donation.
- Because of the variable but significant contamination with red cells, platelets concentrates have to be obtained from ABO/Rh-compatible donors.
- Some human leukocyte antigens (HLAs) are expressed on platelets and determine platelet survival after transfusion. These HLAs lead to Alloimmunisation in patients requiring repeated platelet transfusions.
- The antibodies generated cause rapid destruction of the transfused platelets and account for the progressive inefficacy of repeated transfusions.
- For these patients, HLA-matched platelet concentrates are needed.
- Indications of platelet:
  - Surgical
    - Bleeding and thrombocytopenia
    - Limited cover for operative interventions where platelet count <40 x 10^9/L
    - Platelet dysfunction
    - Acute disseminated intravascular coagulation (with fresh frozen plasma)
    - Massive blood transfusion (washout thrombocytopenia)
    - Post cardiac bypass platelet loss and/or dysfunction
  - Medical
    - Marrow-suppressed patients (intensive chemotherapy)
    - Aplastic anemia
- In surgical practice, platelet transfusions are most commonly used to stop bleeding in thrombocytopenic patients, to cover surgery if the platelet count is below 40 x 10^9/L and in patients with platelet dysfunction.

PLASMA PRODUCTS

- They are produced from voluntary donors and are used for a variety of purposes.
  - As coagulation factors for specific deficiency states, either to stop spontaneous bleeding or cover an operation/intervention in a patient with such a deficiency e.g. hemophilia A (congenital factor VIII deficiency).
  - To provide passive immunity to non-immunized individuals exposed to a serious infective agent (viral or bacterial) e.g. human tetanus immunoglobulin
  - In prophylaxis of hemolytic disease of the newborn due to Rhesus incompatibility and sensitization of Rh (D)-negative women
  - In management of autoimmune thrombocytopenic purpura. IgG can be used to treat idiopathic thrombocytopenic purpura requiring surgery including elective splenectomy
  - As volume replacement fluid (ALBA 4.5%) and as plasma volume expander and short-term management in hypoproteinemic patients (human Albumin solution 20%)
- The most common fraction used in surgical practice is fresh frozen plasma (FFP). Each unit is obtained from a single donation and consists of 200-300ml of plasma with 40-60ml of citrate anticoagulant nutrient mixture.
- Fresh frozen plasma is plasma prepared from the supernatant liquid obtained by centrifugation of one donation of whole blood.
- Indications of FFP:
  - Correct isolated deficiencies of plasma proteins e.g. factor II, V, VII, X, XI, XIII, pseudocholinesterase, antithrombin III and C1 esterase inhibitor
  - Reverse oral anticoagulation with warfarin/coumarin compounds if prothrombin complex concentrate is not available. Normally, reversal of anticoagulant is indicated in the presence of bleeding.
  - Provide hemostatic support and to cover operations/interventions in patients with liver disease, major hepatic resections and severe liver injuries
  - Treatment of DIC
  - In patients who develop a bleeding diathesis after large volume blood transfusion. However, platelets transfusion is more commonly needed first.
  - In treatment of thrombotic thrombocytopenic purpura where FFP us usually combined with plasma exchange
- Cryoprecipitate (factor I-fibrinogen, VII, von Willebrand factor) is used in hemophilia, von Willebrand's disease and fibrinogen deficiency. Because of the risk of transmitted infection, cryoprecipitate is now used much less in

hemophilia. Factor VIII concentrate is now used as it is safer and gives a more certain dose.

- Factor IX concentrate is indicated to arrest acute bleeding and cover operative interventions in patients with Christmas disease.
- Factor VIII and IX therapy requires expert guidance.

Blood and plasma products

	Some indications	Problems
Blood products		
Stored whole blood	Acute haemorrhage	Citrate anticoagulant Acid pH (6.6–6.8) High levels of K^+ Ammonia (from red cell adenosine) Reduced 2,3-DPG
Red cell concentrates	Refractory anaemia	10% of patients develop alloimmunization to leukocyte antigens
Frozen red cells	Patients on renal dialysis, rare cell types and complex antibody mixtures	Very expensive and impractical for most patients
Platelet concentrates	Stop bleeding in thrombocytopenic patients To cover surgery if platelet count $< 40 \times 10^9$/L	Alloimmunization leads to progressive inefficiency of repeated transfusion
Plasma products		
Fresh frozen plasma	Reversal of anticoagulant Correct isolated plasma protein deficiency Treatment of DIC Treatment of burns	Allergic reactions ARDS
Cryoprecipitate (factor VIII, von Willebrand factor, fibrinogen)	Haemophilia, von Willebrand's disease, fibrinogen deficiency	Risk of transmitted infection
Factor VIII concentrate	Haemophilia	Allergic reactions Hyperfibrinogenaemia after massive doses (now rare)
Factor IX concentrate	Acute bleeding and perioperatively in Christmas disease	Allergic reactions

Figure 33: Blood and plasma products

ADVERSE EFFECTS OF TRANSFSION

- Blood transfusion is nowadays very safe with the overall risk of an adverse outcome being 1 in 12 000.
- The mechanisms of transfusion reactions are varied, depending on the cause.
- Complications of transfusion are usually classified according to etiology; they may either be:
  - Acute
  - Delayed
- NOTE: It is recommended that every patient is monitored closely during the first 15-30 minutes of the infusion of each unit of blood. This enables early detection of the clinical manifestations of severe acute reaction due to incompatibility or bacterial infection at which point the infusion is stopped and the necessary action taken.

Acute transfusion reactions	Delated transfusion reaction

• Acute Hemolytic reaction
• Non hemolytic reactions
  ➢ Pyrogenic (febrile) reactions
  ➢ Hypersensitivity
• Metabolic, respirator and hemostatic complications
• Circulatory overload
• Septic shock (bacterially infected units)
	• Delayed hemolytic
	• Infective
	  ➢ Bacterial: brucellosis, syphilis
	  ➢ Helminthic: filariasis
	  ➢ Protozoal: babesiosis, Chagas' disease, Kala-azar, malaria, trypanosomiasis, toxoplasmosis
	  ➢ Rickettsial: relapsing fever, Rocky Mountain spotted fever
	  ➢ Viral: B19, CMV, EBV, HIV 1, 2, HTLV-1/2, hepatitis, yellow fever
	• Sensitization/Alloimmunisation
	  ➢ Hemolytic disease of the newborn
	  ➢ Immune suppression (increased infective risk)
	  ➢ Post-transfusion purpura
	  ➢ Platelet refractoriness
	• Transfusion iron overload (hemosiderosis)
	• Graft vs host disease

ACUTE REACTIONS

ACUTE HEMOLYTIC REACTION

- Hemolysis can occur intravascular or extravascular.

INTRAVASCULAR HEMOLYSIS

- This is due ABO incompatibility due to human error at the bedside (blood given to the wrong patient) or in the lab (faulty cross-matching).
- The transfused cells react with the patient's own anti-A or anti-B antibodies or other alloantibodies to red cell antigens.
- It carries a 3% mortality rate but it is higher if more than 200ml of incompatible blood are administered.
- The reaction is usually most severe if group A red cells are administered to a group O patient. The syndrome is caused by the release of polypeptide products of complement in the plasma which causes contraction of smooth muscle and degranulation of mast cells with release of vasoactive peptides (bradykinin and serotonin)
- Procoagulant substances are released from the stroma of lysed red cells. Together with antigen-antibody complexes these initiate DIC.
- Hemolytic transfusion reactions result in:
  - Hemoglobinemia, hemoglobinuria and renal failure
  - Disseminated intravascular coagulation
  - Complement-mediated cardiovascular collapse
- Hemolytic reactions occur after a small amount of blood has been transfused and almost always before the unit is transfused completely.
- Clinical features in conscious patients:
  - Pain/burning at the infusion site and along the vein
  - Facial burning
  - Chest (tightness) and back pain
  - Joint pain
  - Fever
  - Flushed facies
  - Chills
  - Rigors and vomiting
  - Apprehension, Restlessness and dyspnea
  - Hypotension
  - Oozing from vascular access sites and wounds
  - Physical examination: tachycardia, tachypnea, in severe cases hypotension

- The only manifestation of incompatibility blood transfusion in unconscious or anesthetized patients are sudden hypotension and bleeding due to DIC.
- Extensive intravascular hemolysis results in hemoglobinemia and hemoglobinuria. Urine is dark.
- Oliguria rapidly supervenes and progresses to acute renal failure.
- The differential diagnosis is between incompatibility and infusion of bacterially contaminated blood.
- Management:
  - Immediate recognition with cessation of the transfusion and replacement of the giving set. Inform medical officer on call.
  - Adequate hydration by intravenous infusion of crystalloids (normal saline)
  - Examine the blood to determine if the patient received the correct blood unit.
  - Catheterize the patient and attempt to force diuresis with IV large dose furosemide (150 mg)
    - If furosemide fails, 20% solution of mannitol (100ml) is administered.
    - If diuresis is obtained, a high urine output (30-100ml/h) is maintained by large volume crystalloid infusion. If patient progressives to acute renal failure they will require hemodialysis.
  - Low dose dopamine may be considered to increase renal blood flow.
  - Treat DIC (blood component therapy guided by clinical state and coagulation screen)
  - Treat hyperkalemia
    - 50ml of 50% dextrose + 10 IU of insulin if potassium above 6.0mmol/L
    - Followed by 10% glucose infusion containing 10 units of insulin over a period of 4 hours.
  - Add acetaminophen for fever
  - Investigate incident fully (box above)
  - Also consider endotracheal intubation if in respiratory insufficiency, in severe hemolytic reactions and patients with hypoxemia. Keep O_2 stats >92%.

When dealing with acute hemolytic transfusion episode:

1. Report incident to the blood transfusion department
2. Establish that the unit of blood has been issued to the patient who received it
3. Obtain fresh samples of patient's blood (clotted and in EDTA) for repeat cross-matching and serological testing
4. Send the unit of blood to blood transfusion department
5. Obtain further blood samples from the patient for clinical chemistry (electrolytes, urea, free hemoglobin) and coagulation screen
6. Discuss any further transfusion requirements with blood transfusion officer

EXTRAVASCULAR HEMOLYSIS

- This is due to recipients' antibodies against Rh or non-ABO antigens.
- Patients are usually exposed to antigen through prior pregnancies, transplantation or transfusion.
- Antibody titers are too low often to be detected during screening however production increase with re-exposure to the initial antigen.
- Antibodies do not activate complement (no intravascular hemolysis) instead they coat RBCs which are removed by splenic macrophages.

ACUTE NON-HEMOLYTIC REACTION

- Quality control in the manufacture of IV fluids and disposable giving sets has virtually eliminated pyrogenic reactions.
- Pyrexia following blood transfusion is nowadays as a result of Alloimmunisation to leukocyte and platelet antigens in patients requiring repeated blood transfusions. This is the commonest cause of severe febrile reaction.
- Although it is usually self-limiting and benign, the transfusion must be stopped to exclude the possibility of more serious hemolytic reactions.
- Febrile reactions in alloimunized patients can be prevented by using cell concentrate from which most of other formed elements have been removed (leukocytes, platelets, soluble histocompatibility antigens).
- Other non-hemolytic reactions include severe immediate hypersensitivity reactions and mild allergic or anaphylactoid reactions. The causes of these reactions are rarely established. The reaction results in the release of vasoactive peptides and activation of complements.
- There is profound hypotension, laryngeal spasm and/or bronchospasm and cutaneous flushing.
- Anaphylaxis is treated with:
  - Immediate termination of the transfusion
  - IV crystalloids
  - Maintenance of airway and administration of oxygen
  - Adrenaline (0.5-1.0 mg IM)
    - Adrenaline dose is repeated if necessary, every 10 min depending on the improvement in blood pressure and pulse.
  - Chlorpheniramine 10-20mg is administered by slow IV injection after the adrenaline treatment and is continued for 24-48 hours.
  - Salbutamol is administered by nebulizer.

- Severe anaphylaxis can only be predicted in patients with low serum IgA. If possible, transfusion should be avoided in these patients.

TRANSFUSION-RELATIVE ACUTE LUNG INJURY

- This is one cause of acute respiratory distress syndrome (ARDS) and it was thought to be due to pulmonary microvascular occlusion by microaggregates of platelets, leukocytes and fibrin (50-200 micrometers) which are known to be present in stored blood.
- For this reason, microaggregate filters were recommended for transfusion using in excess of 5 units of blood.
- A more definite cause is donor blood containing antibodies reacting with the patient's leukocytes (nearly always donations from multiparous women). Following transfusion, the patient develops fever, increasing breathlessness, non-productive cough and hypoxemia.
- The chest X-ray shows the typical features of ARDS with perihilar infiltrates leading to a whiteout in severe cases.
- Treatment:
  - Respiratory support as it may need ventilation with positive end-expiratory pressure (PEEP)

METABOLIC, HEMOSTATIC AND RESPIRATORY COMPLICATIONS

- These are confined to patients who because of severe hemorrhage, receive a massive blood transfusion.
- Massive blood transfusion is defined as:
  - Replacement of a volume equivalent to or exceeding the patient's own blood transfused within 12 hours.
  - Replacement of more than one half of the blood volume within 24 hours
  - Replacement of 10 units of blood over the course of few hours
- Effects of stored whole blood
  - Cold (4°C)
  - Acid pH
  - Citrate anticoagulant
  - Elevated plasma potassium and ammonia
  - Reduced 2,3-DPG
- Metabolic consequences:
  - Hypothermia: may lead to cardiac arrhythmias, including ventricular fibrillation and asystole. For this reason, blood warming is necessary if the

transfusion rate exceeds 50ml/min. Unfortunately, heating coils increase the resistance of the give circuit, nonetheless their use is essential in these patients.

- Acidosis
- Increased affinity of oxyhemoglobin for oxygen resulting in defective tissue oxygen uptake.
- Citrate intoxication is due to the chelation of ionized calcium which may result in prolongation of the QT interval. However, this does not usually affect cardiac function and ionized calcium levels rapidly return to normal after transfusion as the excess citrate is metabolized and excreted. Do not give supplemental calcium as it may give rise to arrhythmias,
- Hyperkalemia: this is rarely a problem because the excess plasma potassium enters the red cells with warming to body temperature. However, it is a consideration in patients with acidosis and renal failure when calcium is administered as the physiological antidote.
- Stored blood is deficient in platelets and labile clotting factors (V and VIII).
  - In massive transfusion of stored blood there is dilution of labile clotting factors in addition to a moderate thrombocytopenia.
  - The deficiency is corrected by transfusing 2 units of FFP for every 8 units of blood.
  - The transfusion-related thrombocytopenia is rarely significant and can usually be ignored.

- Management:
  - Hypothermia: transfuse warm blood
  - Metabolic acidosis: may need $NaHCO_3$
  - Impaired release of oxygen from RBCs: self-limiting, no therapy needed
  - Citrate intoxication causing hypocalcemia: Self-limiting, no therapy needed
  - Hyperkalemia: rarely a serious problem
  - Platelet and clotting factor deficiency: give 2 units FFP for every 8 units of blood

CIRCULATORY OVERLOAD

- Circulatory overload is encountered in the transfusion of anemic patients, particularly those with severe and longstanding anemia.
- These patients must be transfused very slowly and only with packed cells (with or without concomitant diuretic therapy).

- In some patients, an exchange transfusion has to be carried out to avoid severe congestive failure.

TRASNFUSION OF BACTERIALLY CONTAMINATED/INFECTED BLOOD

- This is fortunately rare.
- The majority have been associated with platelet transfusion.
- The pathogens are usually cold-growing strains of *Pseudomonas fluorescens* or *Yersinia enterocolitica*.
- Skin organisms such as Staphylococci can proliferate in platelet concentrates stored at 20-22^{o}C.
- The clinical picture is similar to that of ABO incompatible blood transfusion reaction.
- Despite aggressive support therapy (full resuscitation and management in ICU for septic shock), mortality is high and around 60%.

Acute reactions	Cause	Treatment
Acute non-haemolytic reactions	Alloimmunization to leukocytes commonest cause of pyrexia Immediate hypersensitivity reactions cause anaphylaxis: hypotension, laryngeal spasm ± bronchospasm, cutaneous flushing	Immediate termination of transfusion Treat anaphylaxis by: • i.v. crystalloids • maintain airway and O_2 • adrenaline (0.5–1 mg i.m.) • i.v. antihistamine (chlorpheniramine) • salbutamol nebulizer
Acute haemolytic reactions	ABO incompatibility usually caused by human error Pain at infusion site and along vein, chest and back pain, flushing, rigors and vomiting, dyspnoea, hypotension, restlessness. Evidence of DIC	• Immediate termination of transfusion • Replace giving set • i.v. crystalloids • Forced diuresis with furosemide (150 mg) ± mannitol (100 mL, 20%), haemodialysis • Treat hyperkalaemia (dextrose 50% + 10 units insulin if $K^+ > 6.0$ mmol/L) • Treat DIC • Investigate incident fully later
Transfusion-related lung injury	Donor blood antibodies reacting with patient's leukocytes lead to ARDS	Respiratory support. May need ventilation with positive end-expiratory pressure (PEEP)
Metabolic, haemostatic and respiratory complications	Massive transfusion volume (≥ patient's blood volume over 12 h)	• Hypothermia: transfuse warm blood • Metabolic acidosis: may need $NaHCO_3$ • Impaired release of O_2 from RBCs: self-limiting, no therapy • Citrate intoxication causing hypocalcaemia: self-limiting, no therapy • Hyperkalaemia: seldom a serious problem • Platelet and clotting factor deficiency: give 2 units FFP for every 8 units of blood
Circulatory overload	Seen with transfusion of anaemia	Use only RBC concentrates, diuretic therapy
Transfusion of bacterially contaminated (infected) blood	Rare. Usually after platelet transfusion. *Pseudomonas fluorescens, Yersinia enterocolitica*	Full resuscitation and management in ICU for septic shock. Despite therapy, mortality is 60%

Figure 34: Acute transfusion reactions

DELAYED REACTIONS

TRANSMISSION OF INFECTIOUS DISEASE

- A wide spectrum of infectious disease can be transmitted by the transfusion of blood and blood products, although the modern practice of screening blood donors and heat treatment of blood protein products means the risk is extremely small (1 in 10^5-10^6).
- Post-transfusion levels of HIV, human T-cell leukemia virus (HTLV), HBV and HCV are extremely low and the risk of their transmission is minimal compared with other risks.
- HCV transmission is extremely low compared with other risks.
- Hepatitis A and G can be transmitted by blood products.

IMMUNE SUPPRESION

- There is an immunosuppressive effect of blood transfusion.
- Prior to the introduction of cyclosporine, transfusion before renal transplantation was employed specifically to improve graft survival. The use of cyclosporine has made this procedure unnecessary.
- In the context of general surgery, perioperative blood transfusion has undoubted undesirable consequences because of its immunosuppressive effect (which is additive to the immunosuppression produced by operative trauma).
- Aside the risk of circulatory overload, perioperative blood transfusion enhances the risk of infective complications (proven) and may increase recurrence rate and reduce disease-free survival n patients after extirpative surgery for cancer. The latter, however remains unproven.

TRANSFUSION HEMOSIDEROSIS

- Iron overload of the monocyte-macrophage system is caused by repeated red cell transfusion over many years.
- This becomes significant after 100 units have been administered when the liver, pancreas, myocardium and endocrine glands become damaged.
- It is especially a problem in childhood anemia e.g. thalassemia and in patients with chronic refractory anemia
- Iron overload is reduced in these patients by iron chelation therapy with desferrioxamine.

GRAFT VS HOST DISEASE

- Graft vs host disease (GvHD) is a rare but usually fatal complication that occurs mainly in immunodeficient patients e.g. recipients of allogeneic marrow transplants and fetuses receiving intrauterine transfusions.
- GvHD has also been documented in immunocompetent patients after transfusion of blood from a relative.
- The disease is caused by T lymphocytes and starts some 4-30 days after the transfusion.
- The patient develops a high fever, a diffuse erythematous skin rash progressing to erythroderma and desquamation, gastrointestinal symptoms, severe hepatic dysfunction and pancytopenia.
- GvHD is prevented by administering gamma-irradiated cellular components to immunodeficient patients.
- Similarly, blood donated from relatives should be gamma-irradiated.

Delayed reactions	Comment
Transmission of infectious disease	A wide spectrum of infectious disease can be transmitted by blood (product) transfusion. Risk is very small. See text for details
Immune suppression	Perioperative blood transfusion enhances risk of infection (proven) and may adversely affect outcome in cancer (not proven)
Transfusion haemosiderosis	Iron overload of the monocyte/macrophage system occurs after 100 units of blood or red cells have been transfused over a number of years. Iron chelation with desferrioxamine may help
Graft vs. host disease	Immunologically competent transfused cells attack the host environment. May occur when immunodeficient patients are transfused. Rare but fatal

Figure 35: Delayed transfusion reactions

SHOCK AND HYPOTENSION

- Hypotension is a state of low blood pressure with systolic blood pressure (SBP) <90mmHg or mean arterial pressure <65.
  - In relative hypotension the SBP is >30mmHg below baseline
- Shock is a state of cardiovascular failure resulting in impaired tissue perfusion and disturbed cellular metabolism and function as a result of decreased oxygen supply.
- Clinically shock can be defined as a 20% sudden blood loss or shift from peripheral circulation.
- Adequate blood flow to tissues depends on:
  - Blood volume
  - Myocardial contractility
  - Peripheral resistance
- Any disturbance in the above 3 can lead to shock.
  - Pump failure leads to cardiogenic or obstructive failure
  - Peripheral circulatory failure leads to hypovolemic (decreased blood volume) or distributive shock (decreased peripheral resistance).

CLASSIFICATION OF SHOCK

- Shock can be classified into:
  - Hypovolemic (Oligemic) shock: due to diminished blood volume (loss of fluid/blood- hemorrhagic shock)

- Cardiogenic shock: due to inefficient myocardial function (decreased cardiac contractility)
- Distributive shock: due to peripheral blood pooling. Can either be
  - Septic shock: due to systemic infection
  - Neurogenic shock: due to peripheral vasodilation, reduced peripheral resistance and peripheral pooling of blood.
  - Anaphylactic shock: due to antigen-antibody reaction that also leads to peripheral pooling of blood e.g. injection of anti-tetanic serum
  - Endocrinal shock: due to acute severe hormonal imbalance e.g. in adrenal insufficiency or pituitary failure. It may present with a combination of any of the above types.
- Obstructive shock: due to decreased blood flow to the left ventricle as in cardiac tamponade, pulmonary embolism, and tension pneumothorax.

- **Patients may have more than 1 type of shock e.g. hypovolemic and septic**.

PATHOPHYSOLOGY

- This includes cellular changes, microcirculatory changes, acid-base imbalances and systemic changes.
- Cellular changes:
  - Tissue hypo-perfusion resulting in a decrease in oxygen delivery to the tissue and cells undergo anaerobic metabolism producing lactic acid (causing metabolic acidosis).
  - This continues until cellular glucose is depleted resulting in stoppage of cell metabolism release of auto-digestive enzymes and rupture of plasma and lysosomal membranes.
- Microcirculatory changes:
  - Under the effect of catecholamines the precapillary sphincters constrict, less blood enters the capillaries and the capillary circulation becomes sluggish.
  - Untreated hypovolemia thus leads to hypoxia.
  - Under normal conditions only one third of the capillary bed is open at a time.
  - Under ischemic conditions however the body reacts by opening more capillaries this results in:
    - Further slowing of the capillary circulation, the so called slugging of blood.

- Slugging encourages spontaneous coagulation of blood in these capillaries.
- If extensive, it is called disseminated intravascular coagulation (DIC).
- This depletes coagulation factors and induces bleeding in the rest of the body (consumption coagulopathy)

➢ A sluggish circulation and DIC compound tissue hypoxia and affect the function of capillary endothelium. The result of the latter is leakage of large protein molecules from the vessels into interstitial space dragging with them huge amounts of fluid. This third space loss of fluid reduces blood volume.

- Acid-base imbalance: Shock is accompanied by metabolic acidosis which is caused by accumulation of lactic acid as a result of anaerobic metabolism and renal failure as a result of prolonged ischemia, aggravates acidosis.
- Systemic changes:
  - ➢ Sympathetic response: release of catecholamines & stress hormones (Cortisol, glucagon), release of ADH and activation of renin-angiotensin-aldosterone system.
  - ➢ Cardiovascular: reduced coronary artery perfusion and direct depression of myocardium by tumor necrosis factor
  - ➢ Respiratory: metabolic acidosis results in tachypnea which causes carbon dioxide washout and compensatory respiratory alkalosis. Leakage and swelling can develop in the lungs, causing difficulty in breathing (respiratory distress- ARDS)
  - ➢ GIT: mucosal hypoperfusion causes mucosa death and stress ulcers. Mucosal death may also result in bacterial translocation, septicemia and multi-organ failure.
  - ➢ Liver: ischemic hepatic dysfunction
  - ➢ Renal: renal hypoperfusion decreases GFR, urine output and may lead to pre-renal failure and acute tubular necrosis.
  - ➢ Ischemic perfusion syndrome:
    - It is due to systemic hypoperfusion, tissue hypoxia and local activation of inflammatory mediators.
    - Further injury occurs once normal circulation is restored to tissues:
      - Myocardial depression
      - Cellular and humoral elements activated by hypoxia are flushed back into circulation causing more endothelial injury e.g. acute lung injury, acute renal injury
    - It only can be minimized by decreasing the period of hypoperfusion.

STAGES OF SHOCK

- Initial: decreased oxygen and increased lactate.
- Compensatory stage:
  - Increased respiratory rate
  - Sympathetic response: increased heart rate, fluid retention
  - Increased renin and ADH secretion
- Progressive/decompensated stage: If compensatory mechanisms and treatment are unsuccessful.
  - Worsening hypotension due to vasodilation, blood pooling in capillaries, and fluid leak from vessels.
  - Altered mental status
  - Electrolyte imbalances
- Refractory: irreversible

GENERAL CLINICAL FEATURES OF SHOCK

- Change in mental status: a continuum from agitation, confusion/delirium, obtundation/coma.
- Hypotension: absolute (systolic blood pressure <90mmHg) or relative (a drop in systolic blood pressure >40mmHg), which is why a patient may be in shock with a high or normal blood pressure.
- Increased heart rate, pulse may be thread (barely perceptible). Capillary refill >2s
- Oliguria (<30ml/hr)- from shunting of renal flow to other vital organs, intravascular volume depletion or both.
- Cool, clammy skin- compensatory peripheral vasoconstriction redirects blood from the peripheral to the vital organs (heart, brain, splanchnic). However, in early distributive shock or terminal shock the skin may be flushed or hyperemic due to failure of compensatory vasoconstriction.
- Metabolic acidosis- increased lactate production from anaerobic respiration when shock progresses to circulatory failure and tissue hypoxia along with decreased clearance of lactate by the liver, kidneys and skeletal muscle.

APPROACH TO THE PATIENT WITH SHOCK

HISTORY

- Look for underlying causes, try to classify the type of shock. Often limited due to confusion or obtundation.
- Ask for any history of:

- Bleeding? Trauma? Hematemesis? Hematochezia? Melena? (if so suspect hypovolemic shock)
- Coronary artery disease? Valvular heart disease? CCF? (if so suspect cardiogenic shock)
- History of fever? Pneumonia? Meningitis? (if so suspect septic shock)

EXAMINATION

- General examination: ill appearing, confused, lethargic, unresponsive and temperature >38ºC or <36ºC.
- Neurologic: meningism and focal neurologic deficits
- Cardiac: Tachycardic (HR> 100), hypotensive (SBP <100), Dizziness, Orthostatic hypotension, S3 heart sound, murmur, Extremities may be warm/well perfused or cold/blue, Capillary refill brisk or slow, Chest pain
- Pulmonary: Evidence of pneumonia, use of accessory muscles of respirations, Respiratory rate >30, Oxygen saturation <95%.
- Gastrointestinal: Abdominal pain, Tense/Rigid abdomen (may indicate perforated viscus, hemorrhage, peritonitis), rebound tenderness, guarding, melena or blood on rectal exam.
- Renal: Flank tenderness (pyelonephritis), Urine output <0.5ml/kg/hr or <30ml/h

INVESTIGATIONS

- Blood investigations:
  - Urea and electrolytes (Sodium, potassium, chloride and bicarbonate) + Creatinine
  - Liver enzymes
  - Serum glucose
  - Full blood count (leukocytes and bandemia in sepsis)
  - Clotting profile: Bleeding time, Prothrombin time and activated partial thromboplastin time to rule out bleeding tendencies
  - Blood culture
  - Cardiac enzymes
  - Arterial blood gas/lactate (if available)
- Imaging:
  - Chest and abdominal X-ray
  - ECG
  - Echocardiogram
  - Ultrasound- Focused assessment with sonography for trauma (FAST)

- Septic work up: CXR, blood culture, sputum microscopy culture and sensitivity, urine dipstick, microscopy, culture and sensitivity, lumbar puncture.

MANAGEMENT

- Management depends entirely on the type of shock.
- Shock is always an emergency and must be treated rapidly as it is life threatening.
- Treat the underlying cause
- Management includes:
  - Frequent monitoring of vital signs
  - 2 large bore IV cannulas- 18 gauge (green) or greater.
  - Foley catheter and monitoring of urine output.
  - Aggressive colloid (normal saline or ringers lactate) fluid resuscitation for hypotension- especially in hypovolemic + septic shock (may require >10 L)
  - Intubation if in respiratory distress
  - Packed red cells if bleeding/hypovolemic
  - Stop any antihypertensive or diuretics. Give pressors (dopamine, dobutamine, epinephrine, neosynephrine etc.) if shock is present despite aggressive fluid resuscitation.
  - Hypovolemic shock:
    - IV fluids
    - Check electrolytes
    - Fix underlying condition e.g. diabetic ketoacidosis
    - Send for X-match for urgent transfusion if hemorrhage (most common cause of hypovolemic shock)
  - Cardiogenic shock:
    - IV fluids may be harmful
    - IV Lasix and or dopamine may help
    - Fix underlying cause e.g. valve replacement
  - Septic shock:
    - Search for source and start empiric antibiotics based on likely type of infection.
    - Give IV fluids
  - Anaphylactic shock:
    - IV fluids
    - Subcutaneous epinephrine 0.3ml 1: 1000 solution if severe
    - Antihistamines and corticosteroids may help
  - Adrenal insufficiency: give hydrocortisone 100mg TDS x 5/7

- Obstructive shock
  - IV fluids
  - Urgent ECG
  - CXR to confirm
  - Thrombolysis for Pulmonary embolism
  - Chest tube for tension pneumothorax
  - Pericardiocentesis for tamponade.

HYPOVOLEMIC SHOCK

ETIOLOGY

- This is due to diminished blood volume as a result of:
  - Whole blood loss i.e. hemorrhage which can be traumatic or pathological
    - Trauma
    - Ruptured abdominal aortic aneurysm
    - Aortic dissection
    - Upper GI bleeding
    - Ruptured ectopic pregnancy
  - Plasma loss as in burns, heat exhaustion or peritonitis
  - Water and electrolyte loss (especially Sodium) as in: severe vomiting, diarrhea, intestinal obstruction and fluid sequestration (as in acute pancreatitis).

STAGES

- Compensated stage: in this stage the body employs the physiological mechanisms, in a trial to restore blood volume or at least, preserve the normal function of the vital organs (brain, kidneys, heart and lung). With loss of >15% of blood volume, the compensatory mechanisms usually fail.
- Decompensated stage (40% blood loss): if the cause of shock is not treated successfully and after failure of compensatory mechanisms there will be progressive poor perfusion and micro-circulatory changes with deterioration of functions of the brain, kidney, heart and lung.
- Refractory stage: The shock can no longer be reversed. Failure of vital organs and death occurs imminently.

CLINICAL FEATURES

- Of shock
  - Symptoms:
    - Weakness and fainting

- The patient feels cold and thirsty

➢ Signs:

- Patient is cold and clammy with a narrow pulse pressure
- Decrease in blood pressure: SBP<90 or 60 below normal
- Orthostatic hypotension can be an early sign of hypovolemia.
- Capillary refill>2s
- Oliguria: <30ml/hr
- Altered mental status (may vary from anxious to drowsy but the patient usually remains alert).
- Hypotension, tachycardia (increased heart rate, pulse may be thread-barely perceptible) and decreased pulse pressure (thready pulse)

- Of the cause
  - ➢ Sites of bleeding
  - ➢ Signs of internal hemorrhage
  - ➢ Burns
  - ➢ Intestinal obstruction
  - ➢ In hemorrhagic shock, trauma or signs of GI bleeding (melena, abdominal pain)
- Of complications:
  - ➢ Anuria
  - ➢ Acute respiratory depress syndrome

INVESTIGATIONS

- Blood investigations:
  - ➢ Full blood count (check Hemoglobin, hematocrit)
  - ➢ Cross match (for blood transfusion) as well as group and save
  - ➢ Urea and electrolytes (Sodium, potassium, chloride and bicarbonate) + creatinine
  - ➢ Clotting profile: Bleeding time, Prothrombin time and activated partial thromboplastin time to rule out bleeding tendencies
  - ➢ Liver function tests
  - ➢ Serum glucose
  - ➢ Arterial blood gasses: lactate >2 suggests hypoperfusion
- Imaging:
  - ➢ Chest X-ray and ECG: to exclude cardiogenic causes
  - ➢ Abdominal ultrasound: to rule out internal hemorrhage
  - ➢ Head CT scan: if the patient with head injury or unconscious

MANAGEMENT

RESUSCITATION

- ABCs
  - Airway should be patent by: Pulling the tongue forward, aspiration of secretions, and tracheostomy if needed
  - Breathing should be maintained by: oxygen mask and ambu bag. Administer oxygen.
  - Circulation:
    - 2 large bore IV cannula 18G (green) or bigger: one for IV fluids and the other for sampling (if no veins available/all collapsed)
    - Stop any external bleeding by compression.
  - Drugs: analgesics for any pain
- Insert an NGT to evacuate the stomach (if surgery may be needed)
- Insert a urinary catheter to monitor urine output
- Elevate legs and head down to restore cerebral blood flow (Trendelenburg's position)
- Give sufficient warmth (but avoid over warmth)

Recall:

60% of the body is fluid. It is distributed in 3 compartments:

- 2/3 is intracellular fluid
- 1/3 is extracellular fluid
  - ¾ (75%) of ECF is in the interstitium
  - ¼ (25%) of ECF is in the intravascular compartment

FLUID ADMINISTRATION

- Crystalloids: may be the only fluid given to patients with hypovolemia caused by loss of water and electrolytes.
  - Normal saline: to replenish equivalent loss of both water and electrolytes.
  - Ringer: gives potassium supply (after ensuring good urine output) in prolonged shock
  - Lactated Ringer's solution: also a buffer, in prolonged shock and acidosis
  - Note: Glucose 5% is not used in shock because glucose is rapidly redistributed this leads to an increase in the amount of ECF (without electrolytes) causing dilutional hyponatremia and increased ICF with brain edema and increased intracranial pressure.
- Fluid therapy:
  - Resuscitation:
    - 500ml crystalloid bolus over <15minutes. 250ml if old or heart failure.
    - Reassess and re-give up to 2L
    - Don't wait until SBP is under 90. A SBP of 110 in the presence of increased heart rate suggests impaired output.

- Replacement:
  - Estimate fluid losses, and replace gradually over 2-3 days i.e. in addition to maintenance fluids.
- Maintenance:
  - Basic needs:
    - 25-30ml/kg/day water. 20-25ml/kg/day if old, kidney or heart failure or malnourished. So just over 1ml/kg/hr
    - 1mmol/kg/day of sodium, potassium and chloride. In general, don't give >10mmol/hr
    - 50-100g/day of glucose.
  - Management:
    - Usually need around 2-3 liters per day so around 100ml/hr is typical e.g. 1L over 8-10 hours. 500ml over 8 hours if old or heart failure.
    - 1L of normal saline contains 154mmol of both sodium and chloride and 1 L of Hartmann's solution contains 131 mmol of sodium and 111mmol of chloride (plus 5 mmol of potassium). So 500ml of each roughly covers daily needs.
    - Typical daily regimen may be 1L 5% dextrose with 20mmol potassium, then 500ml normal saline with 20mmol potassium, then 1L 5% dextrose with 20 mmol potassium.
    - Monitor potassium to determine needs
    - Monitor increase in chloride daily if on normal saline due to risk of increased chloride ions.

- Colloids: contain large insoluble molecules increasing oncotic pressure and in theory keeping the fluid in the intravascular compartment longer. In clinical trials, however, colloids have shown no benefit (and sometimes harm) relative to crystalloids.
  - Plasma substitutes: needs matching and adequate storage
  - Dextran (polysaccharide polymers): high molecular and low molecular weight
- Blood and blood products:
  - Red blood cells
    - Indications:
      - Decreased Hb (Hb<7g/dl)
      - Hb <8g/dl in patients with ACS
      - Major hemorrhage (given in combination with FFP and platelets)
    - 1 unit of blood increases Hb by 1g/dl

- 1 unit of whole blood is around 450ml while 1 unit of packed cells is 250-300ml.
- Prescribe 1 unit at a time, checking response (symptoms and/or Hb) after each.
- If unused must return to fridge within 30 minutes for reuse.
- RBCs typically given over 2-3 hours but can be up to 4 hours most other products are given over 30 minutes.
- Assess patient at baseline, 15 minutes and at the end of transfusion.

➢ Fresh frozen plasma (FFP)
- Human plasma which contains clotting factors (II, V, VIII, IX, X, XI and antithrombin III)
- Indications
  - Major hemorrhage
  - Warfarin reversal (INR>1.5)
- Cryoprecipitate is another alternative to FFP, though less used now. It is the precipitate from human plasma, and contains fibrinogen, factor VIII (8), vWF and factor XIII (13)

➢ Platelets
- Indications:
  - Platelet <50K/μL and bleeding
  - Platelet <20 K/μL and septic
  - Platelet <10 K/μL
  - Stop any anti-platelets if giving

Transfusion reaction	Signs	Management
Mild	• <2ºC rise in temperature • Rash/Urticaria	• Pause transfusion and assess patient • Consider paracetamol or chlorphenamine • Re-start transfusion with close observation
Severe	• >2ºC rise in temperature • Rigors • Loin or back pain • Anxiety or angor animi	• Stop transfusion and replace any fluid needs with saline • Call hematology, contact lab to return product, and complete incident report

	• Unexpected increased heart rate • Infusion site pain	

- Transfusion associated circulatory overload (TACO):
  - Commonest cause of transfusion-associated death
  - Presents with 6 hours of transfusion with acute respiratory distress, pulmonary edema, increased heart rate and increased BP.
  - Risk factors: age <3 or >60, low birth weight, heart failure, kidney failure
  - Blood products are prescribed in mls in neonates to reduce TACO
- Transfusion-related acute lung injury (TRALI)
  - Very uncommon
  - Respiratory distress, decreased BP
  - Usually due to FFP or platelets
- Other complications:
  - Anaphylaxis
  - Hemolytic anemia
  - Increased potassium
  - Infection

MONITORING

- Monitor:
  - Pulse and ECG
  - Blood pressure
  - Temperature
  - Urine output (optimum urinary output= 0.5-1ml/kg/hr)
  - Arterial blood gasses (normal PO_2=80-100mmHg, normal PCO_2= 35-45mmHg)

DRUGS

- Drugs that increase BP by increasing cardiac contractility (inotropes) and/or vasoconstriction (vasopressors). Many also increase heart rate (positive chronotropy).
- Indicated in shock for which IV fluids are insufficient or inappropriate.
- Delivery is typically as an infusion through a central line.
- Many commonly used agents are synthetic versions of endogenous catecholamines

Drug	Mechanism of action	Dosage	Side-effect
Noradrenaline (norepinephrine) First line agent for shock in many ICUs	Vasopressor via alpha 1 agonism but also inotropy via Beta 1 agonism at higher doses. Reflex bradycardia from increased BP typically cancels out chronotropic effect.	0.05-1.0mcg/kg/min	Peripheral administration carries a risk of extravasation and hence tissue necrosis via vasoconstriction. However short term delivery via a large, proximal peripheral line is sometimes acceptable. Ischemia especially to the GIT, kidney and extremities Atrial and ventricular arrhythmias
Adrenaline (epinephrine) Most commonly used for anaphylaxis and cardiac arrest	Acts on all adrenergic receptors. At lower doses works primarily on beta 1 (inotropy and chronotropy) and beta 2 (airway and vessel dilation) and alpha 1 (vasoconstriction) at higher doses (including cardiac arrest dose)	0.05-1.0mcg/kg/min	
Dobutamine Used in acute heart failure	Inodilator with inotropy via beta 1 agonism and vasodilation via beta 2.	Dobutamine 5-10 microgram/kg/min IV infusion	
Dopamine	DA agonist at lower doses, alpha and beta effects at higher doses	Give dopamine 5-10 micrograms/kg/min IV infusion (dilute 400mg in 500ml of 5% dextrose infusion rate to be calculated according to body weight) • Start 5mcg/kg/min, titrate to effects.	Risk of Tachyarrhythmias Evidence suggests that dopamine inhibits secretion of prolactin and could increase lymphocytes apoptosis (impairment of immune response to sepsis)

TREAMENT OF CAUSE AND COMPLICATIONS

- Treat the underlying cause.

CARDIOGENIC SHOCK

- This is due to inadequate blood flow to vital organs due to inadequate cardiac output despite of normal blood volume.
- It is characterized by
  - SBP <90mmHg
  - Mean arterial pressure drop >30mmHg
  - Urine output <0.5ml/kg/hr
  - Heart rate >60bpm

ETIOLOGY

- Primary pump failure due to:
  - MI (commonest cause) or its complications e.g. ventricular septal rupture
  - Severe arrhythmias
  - Massive pulmonary embolism
  - Cardiac tamponade
  - Myocarditis
  - Severe valve disease
  - Trauma
  - High spinal anesthesia

CLINICAL FEATURES

- Of shock:
  - Clinical picture similar to hypovolemic shock.
  - Comatose
  - Patient is cold and clammy and pulse pressure may be narrow
  - Peripheral cyanosis
  - Poor urine output
  - Increased JVP and decreased heart rate
- Of the cause: enlarged liver, chest pain, pulsus alternans, 'gallop' rhythm, murmur, basal crackle, pulmonary edema.

INVESTIGATIONS

- Blood investigations:
  - Full blood count (check Hemoglobin, hematocrit)
  - Cross match (for blood transfusion) as well as group and save
  - Urea and electrolytes (Sodium, potassium, chloride and bicarbonate) + creatinine

- Clotting profile: Bleeding time, Prothrombin time and activated partial thromboplastin time to rule out bleeding tendencies
- Liver function tests
- Serum glucose
- Arterial blood gasses: lactate >2 suggests hypoperfusion

- Imaging:
  - Cardiorespiratory: ECG, CXR (enlarged heart, pulmonary venous congestion) and echo
  - Abdominal ultrasound: to rule out internal hemorrhage
  - Head CT scan: if the patient with head injury or unconscious

MANAGEMENT

- Management aims at:
  - Improving cardiac output:
    - Correct dysrhythmias
    - Optimize preload
    - Improve contractility
    - Reduce afterload
  - Minimizing cardiac work:
    - Maintain normal temperature
    - Sedation
    - Intubation and mechanical ventilation
    - Correct anemia
- ICU care
- Give oxygen
- Regular monitoring of temperature, heart rate, blood pressure and cardiac monitoring.
- Give dopamine 5-10 micrograms/kg/min IV infusion (dilute 400mg in 500ml of 5% dextrose infusion rate to be calculated according to body weight)
  - Start 5mcg/kg/min, titrate to effects.
  - Evidence suggests that dopamine inhibits secretion of prolactin and could increase lymphocytes apoptosis (impairment of immune response to sepsis).
- Dobutamine 5-10 microgram/kg/min IV infusion
- Prophylactic anticoagulation with ASA 75mg PO OD.
- Treat underlying cause
- If in heart failure and BP goes above 90mmHg treat as class IV.

- Captopril 25mg BD/TDS or Lisinopril 10-20mg OD or Enalapril 5-20mg OD (if patient develops dry cough Losartan 50mg OD)
- Furosemide IV 40-80mg OD or BD (monitor potassium levels)
- Digoxin 0.125mg-0.25mg daily
- Isosorbide dinitrate 5-10mg BD + hydralazine 25-50mg BD orally if patient cannot tolerate ACEi.
- Spironolactone 25-50mg OD or BD
- ASA 75mg OD

DISTRIBUTIVE SHOCK

- Vasodilation leads to a relative (though not absolute) deficiency in volume.

SEPTIC SHOCK (ENDOTOXIC SHOCK)

- Septic shock is a type of distributive shock though it may also include an element of hypovolemic shock as altered capillary permeability leads to volume loss from the vasculature.
- It is the most serious type of shock and the most difficult to treat.

ETIOLOGY

- Gram negative bacilli (commonest cause)
- *Staphylococci*
- *Candida*

PREDISPOSING FACTORS (IMMUNOSUPPRESION)

- Extremes of age
- Malignancy
- Malnutrition
- Diabetes mellitus
- Chemotherapy, corticosteroids or immunosuppressants
- HIV infection

PATHOPHYSIOLOGY

- Bacterial endotoxins are released when immune cells (i.e. macrophages) kill gram negative bacteria.
- Immune cells release cytokine, TNF and IL-2 which damage capillary endothelium leading to edema and vasodilation (rapid shifting of blood bypassing capillary) and tissue ischemia.

- Septic shock starts as maldistribution of blood followed by myocardial depression and hypovolemia.

CLINICAL FEATURES

- Hyper-dynamic (warm) septic shock (early phase): diagnosis is difficult and high index of suspicion is required to detect it at early stage.
  - Restless and confusion
  - Skin: flushed, warm and dry
  - Vitals:
    - Fever >38^{o}C + Chills
    - Mild decreased in arterial blood pressure, pulse pressure may be wide with a low DBP.
    - Tachycardia
    - Tachypnea
  - High cardiac output
- Hypo-dynamic "cold" septic shock (Late phase): picture similar to hypovolemic shock with reduced cardiac output
  - Skin: cold and clammy
  - Vitals:
    - SBP<90mmHg
    - Tachycardia
    - Tachypnea
  - Oliguria
  - Multi-organ failure starts at this stage.

DIAGNOSIS

- To diagnose septic shock, the following 2 criteria must be met
  - Evidence of infection, through a positive blood culture
  - Refractory hypotension (Despite adequate fluid resuscitation and cardiac output)
- In addition to the 2 criteria above 2 or more of the following must be met (SIRS)
  - RR >20bpm (high respiratory rate)
  - WBC <4000 cells/mm^3 or > 12 000 cells/mm^3
  - Temperature >38^{o}C or <36^{o}C
  - HR>90b/min
  - Arterial blood gasses $PaCO_2$ <32mmHg

MANAGEMENT

- Give oxygen
- Give fluids:
  - Ringer's lactate + Plasma
  - If low hematocrit packed RBCs or whole blood transfusion
- Drugs
  - Inotropes and vasopressors: dopamine and dobutamine
  - Give if patient remains hypotensive despite adequate fluid replacement.
  - Give dopamine 5-10 micrograms/kg/min IV infusion (dilute 400mg in 500ml of 5% dextrose infusion rate to be calculated according to body weight)
    - Start 5mcg/kg/min, titrate to effects.
    - Evidence suggests that dopamine inhibits secretion of prolactin and could increase lymphocytes apoptosis (impairment of immune response to sepsis).
  - Dobutamine 5-10 microgram/kg/min IV infusion
- Antibiotics: parenteral combined broad-spectrum started early without waiting for culture and sensitivity results (3^{rd} generation cephalosporin e.g. ceftriaxone + Aminoglycoside e.g. gentamicin + Metronidazole) then changed to culture and sensitivity.
- Monitor vitals and urine output regularly.
- ASA 75mg OD for DVT prophylaxis
- Proton pump inhibitors to prevent stress ulcers.
- Use of corticosteroids in septic shock remains controversial.

WOUNDS AND WOUND HEALING

- A wound is described as a break in the integrity of the skin or tissues often which may be associated with disruption of the structure and function.
- An ulcer is a disruption or break in the continuity of epithelium- may be skin, mucous membrane or others associated with microscopic death of tissue.
  - ➢ Ulcers are a type of wound.

CLASSIFICATION OF WOUNDS

CLASSIFIACTION OF SURGICAL WOUNDS

CLEAN WOUND

- This class of wounds have minimal risk of infection (less than 2%).
- They include surgically incised wounds with complete adherence of all septic conditions without any breech of any hollow viscera.
  - ➢ Thyroidectomy
  - ➢ Herniorrhaphy
  - ➢ Herniotomy
  - ➢ Lumpectomy
  - ➢ Excisions: lipoma
  - ➢ Surgeries of the brain, joints, heart, transplant
- There is no need of antibiotic use in clean wounds except in immunosuppressed patients or patients having a prosthetic heart valve.

CLEAN CONTAMINATED WOUND

- This type of wound has low risk of infection (10% risk).
- These wounds include entry into hollow organ that is not inflamed and entry into abscess cavity, minor breach in aseptic procedure, mild spillage of intra-abdominal viscous.
  - ➢ Appendectomy
  - ➢ Bowel surgeries
  - ➢ Gallbladder, biliary and pancreatic surgeries
- Prophylactic treatment with antibiotics is required.

CONTAMINATED WOUND

- These have high rate of infection (15-30% risk).
- They occur when there is major breach in aseptic condition even without entry into abscess cavity or hollow viscus.
  - ➢ Acute abdominal conditions
  - ➢ Open fresh accidental wounds
- Therapeutic antibiotics aimed at treatment and not prophylaxis are given.

DIRTY OR INFECTED WOUND

- These have very high risk of infection (40-70% risk).
- Dirty wounds appear grossly infected with pus with entry into an abscess cavity such as in gangrenous bowel.
  - ➢ Abscess drainage
  - ➢ Pyocele
  - ➢ Empyema gallbladder
  - ➢ Fecal peritonitis

Table 3.41: Wound Classification (Courtesy of SRB's Manual of Surgery 4th Ed)

Wound classifications	
• *Simple wounds:* Only skin is involved.	• Tidy wounds
• *Complex wounds:* Vessels, nerves, tendons or bones are involved.	• Untidy wounds
• *Closed wounds:*	• Clean wound
– Contusion	• Clean contaminated wound
– Abrasion	• Contaminated wound
– Haematoma	• Dirty wound
• *Open wounds:*	
– Incised wounds	
– Lacerated wounds	
– Crush injuries	
– Penetrating wounds	

WOUND HEALING

- Wound healing is a complex method to achieve anatomical and functional integrity of disrupted tissue.
- Healing is by regeneration and repair.
  - ➢ Regeneration replaces damaged tissue with natural tissue.
  - ➢ Repair replaces damaged tissue with fibrous scar. It occurs when regenerative stem cells are lost or when tissue lacks regenerative capacity.
- Recall that, based on regenerative capacity tissues are divided into:
  - ➢ Labile tissue/continuous dividers:
    - ○ Continuously in the cycle to regenerate
    - ○ Examples: small and large bowel (stem cells in mucosal crypts), skin (stem cells in basal layer), bone marrow (hematopoietic stem cells), Lung tissue (type II pneumocytes are the stem cells)
  - ➢ Stable tissue/Facultative dividers/quiescent cells:
    - ○ Are quiescent but can re-enter the cell cycle.
    - ○ Regeneration of liver by compensatory hyperplasia after partial resection.
    - ○ Each hepatocyte produces additional cells and then re-enters quiescence.
    - ○ Cells of proximal renal tubules are also stable
  - ➢ Permanent tissue
    - ○ Lacks significant regenerative potential
    - ○ Examples: myocardium, skeletal muscle, neurons.

- Healing involves neutrophils, macrophages, lymphocytes, fibroblasts and collagen in an organized staged pathway.

TYPES OF WOUND CLOSURE

PRIMARY HEALING (FIRST INTENTION)

- If staples or sutures close a wound, healing is said to be by primary intention.
- Occurs in a clean incised wound or surgical wound.
- Wound edges are approximated with sutures. There is more epithelial regeneration than fibrosis.
- Wound heals rapidly with complete closure.
- Scar will be linear, smooth and supple.

SECONDARY HEALING (SECOND INTENTION)

- Occurs in a wound with extensive soft tissue loss like in major trauma, burns and wound with sepsis.
- Edges are not approximated with sutures; granulation tissue fills the defect.
- It heals slowly with fibrosis. It leads into a wide scar often hypertrophied and contracted. It may lead into disability (contracture).
- Re-epithelialisation occurs from remaining dermal elements or wound margins.

HEALING BY THIRD INTENTION (TERTIARY WOUND HEALING OR DELAYED PRIMARY CLOSURE)

- After wound debridement and control of local infection, wound is closed with sutures or covered using skin graft.
- Primary contaminated or mixed tissue wounds heal by tertiary intention (Also known as delayed primary closure)

Note: the average wound usually achieves approximately 4-8% of its expected tensile strength after 1-2 week.

When completely healed it has 80% tensile strength as compared to uninjured tissue

STAGES OF WOUND HEALING

- There are 4 distinct stages of normal wound healing, which are not mutually exclusive and show considerable overlap.
- These stages include:
  - Stage of coagulation/hemostasis
  - Stage of inflammation

- Stage of fibroplasia/ Proliferation
- Stage of re-modeling

PHASES OF WOUND HEALING

COAGULATION/ HEMOSTASIS

- Happens almost immediately.
- Following injury, the clotting cascade is initiated, local vasoconstriction occurs and a platelet clot forms.
- Platelet-derived growth factor (PDGF) is released.
- After the transient phase of vasoconstriction there is vasodilatation to initiate the inflammatory phase.

INFLAMMATORY PHASE

- Begins immediately after vasodilatation.
- Inflammatory cells are attracted to the site of injury, initially neutrophils then macrophages, which remove tissue debris.
- Lymphocytes are recruited later and persist in chronic inflammation.
- Lasts 0 to about 4/6 days.
- Shows the features of inflammation (rubor, calor, tumor, dolor and loss of function)
- Macrophages secrete fibroblastic growth factor (FGF)
  - FGF enhances angiogenesis and skeletal development
- Polymorphonuclear leukocytes (PMN leukocytes) appear after 48 hours which secrete inflammatory mediators and bactericidal oxygen derived free radical.
  - They also remove clots, foreign bodies and bacteria.
- Chemical factors in wound healing:
  - Growth factors
    - TGF alpha- epithelial and fibroblast growth factor
    - TGF beta- important fibroblast growth factor, inhibits inflammation
    - Platelet derived growth factor- endothelium, smooth muscle, fibroblast growth factor.
    - Fibroblast growth factor- angiogenesis, skeletal development
    - Vascular endothelial derived growth factor- angiogenesis

PROLIFERATIVE PHASE (COLLAGEN/FIBROBLASTIC PHASE)

- There is formation of granulation tissue.
- It lasts from 4 days to 3 weeks (4 days to 21 days).
- Granulation tissue consists of:
  - Fibroblasts: deposits type III collagen
    - Recall Type I collagen is found in bONE, Type II

collagen is found in cartilage (car-two-lage), Type III collagen is found in blood vessels, granulation tissue, and embryonic tissue e.g. skin because of its pliability characteristic and Type IV collagen is found in the basement membrane.

- Capillaries: provide nutrients
- Myofibroblast: contract wound

REMODELLING PHASE (MATURATION PHASE)

- It is from 3weeks to 18 months.
- There is maturation of collagen by cross-linking which is responsible for tensile strength of the scar.
- There is differentiation of fibroblast into myofibroblasts which are responsible for wound contraction.
- Collagen production is not present after 42 days of wound healing.
- Type III collagen is replaced with type I collagen and blood vessels atrophy.
- The processes result in wound healing by an epithelialized white scar.
- Collagenase removes type III collagen and requires zinc as a co-factor.
- Maximal wound tensile strength is achieved at about day 60, when it is 80% of normal.

FACTORS AFFECTING WOUND HEALING

- Local factors:
  - Infection (most common cause of delayed wound healing).
  - Presence of necrotic tissue and foreign body.
  - Poor blood supply
  - Venous or lymph stasis
  - Tissue tension
  - Hematoma
  - Large defect or poor apposition
  - Recurrent trauma
  - X-ray irradiated area
  - Site of wound e.g. wounds over the joints and back heal poorly.
  - Underlying diseases like osteomyelitis and malignancy
  - Mechanism and type of wound-incised/lacerated/crush/avulsion
  - Tissue hypoxia locally reduces macrophage and fibroblast activity
- General factors:
  - Age, obesity, smoking
  - Malnutrition, zinc deficiency (required for collagenase), copper deficiency (lysly oxidase), manganese deficiency
  - Vitamin deficiency (Vitamin C-required for the hydroxylation of collagen as it is maturing, Vitamin A)
  - Hypoproteinemia
  - Anemia
  - Malignancy

- Uremia
- Jaundice
- Diabetes, metabolic disease
- HIV and immunosuppressive diseases
- Steroids and cytotoxic drugs
- Neuropathies of different causes

COMPLICATIONS OF WOUND HEALING

1. Dehiscence
   - Rupture of wound, most commonly seen after abdominal surgery.
2. Hypertrophic scars
   - Excess production of scar tissue that is localized to the wound
3. Keloid
   - Excess production of scar tissue that is out of proportion to the wound (exceeds wound margins).
   - Characterized by excess type III collagen
   - Genetic predisposition (more common in African American)
   - Classically affects ear lobes, face and upper extremities

MANAGEMENT OF WOUNDS

- Wound is inspected and classified as per the type of wounds.
- If it is in a vital area (ABCs):
  - The airway should be maintained
  - The bleeding if present should be controlled
  - Intravenous fluids are started
  - Oxygen if required may be given
  - Deeper communicating injuries and fractures should be looked for.
- If it is an incised wound then primary suturing is done after thorough cleaning.
  - Primary suturing means suturing the wound immediately within 6 hours. It is done in clean incised wounds.
  - Primary suturing should not be done if there is edema/infection/devitalized tissue/ hematoma.
  - Always associated injuries to deeper structures like vessels/nerves or tendons should be looked for before closure of the wound.
  - Wound should be widened by extending the incision whenever needed to have proper evaluation of the deeper structures-proper exploration
  - Proper cleaning, asepsis, wound excision/debridement
  - Any foreign body in the wound should be removed
  - Skin closure if it is possible without tension
  - Skin closure by graft/flap-immediate or delayed

- Untidy wound should be made tidy and clean before suturing
- Proper aseptic precautions should be undertaken
- Antibiotics/analgesics are needed
- Sutured wound should be inspected in 48 hours. (for neurovascular state)
- Sutures are removed after 7 days

- If it is a crushed or devitalized wound there will be edema and tension in the wound. So after wound debridement or wound excision by excising all devitalized tissue, the edema is allowed to subside for 2-6 days. Then delayed primary suturing is done.
  - Delayed primary suturing means suturing the wound in 48hours to 10 days. It is done in lacerated wounds. This time is allowed for the edema to subside.
- If it is deep devitalized wound, after wound debridement it is allowed to granulate completely. Later if the wound is small secondary suturing is done. If the wound is large a split skin graft (Thiersch graft) is used to cover the defect
- In a wound with tension, fasciotomy is done so as to prevent the development of compartment syndrome.
- Vascular or nerve injuries are dealt with accordingly. Vessels are sutured with 6-zero polypropylene non-absorbable suture material. If the nerves are having clean cut wounds it can be sutured primarily with polypropylene 6-zero or 7-zero suture material. If there is difficult in identifying the nerve ends or if there are crushed cut ends of nerves then marker stitches are placed using silk at the site and later secondary repair of the nerve is done.
- Internal injuries (intracranial by craniotomy, intrathoracic by intercostal tube drainage, intra-abdominal by laparotomy) has to be dealt with accordingly. Fractured bone is also identified and properly dealt with.
- Antibiotics, fluid and electrolyte balance, blood transfusion, tetanus toxoid (0.5 ml intramuscular to deltoid muscle) or antitetanus globulin (ATG) injection.
- Wound debridement (wound toilet or wound excision) is liberal excision of all devitalized tissue at regular intervals (of 48-72 hours) until healthy, bleeding, vascular tidy wound is created.
  - Wound toilet is washing the wound thoroughly using normal saline-ideal
  - Wound debridement is allowing content to come out by release incisions or fasciotomies. But

commonly debridement is used for wound excision.

- Wound excision is actually correct terminology for excision of devitalized tissues once or serially.
- Radical wound excision (pesudotumor approach) is excising entire devitalized tissues leaving tissues with visible bleeding from all layers.

COMPARTMENT SYNDROME

- This is a special entity, common in leg, forearm, thigh and arms.
- It is a syndrome due to increased intra-compartmental pressure within a limited space compromising the circulation and function of tissues within that closed space.
- Compartment syndrome in the lower limb is defined as intercompartmental pressures exceeding 30 mmHg.
- It is characterized by severe, progressive and persistent pain that poorly responds to analgesics and is aggravated by passive muscle stretching.
- Causes:
  - Narrowed space due to tight dressings/ plaster cast, lying on one limb in comatous patient
  - Increased content within the compartment due to trauma like fractures, edema, ischemia injury, hematomas, positioning after trauma, burn injury etc.
  - High pressure injuries like gun injury, oil-based material injury, extravasation of chemotherapeutic drugs, snake bites.
- Pathophysiology:
  - Edema is often a sequela of trauma. If it occurs in a limited anatomical space such as one of the four compartments of the lower limb, the tissue pressure will rise.
  - As tissue pressure rises, the pressure on and within the local venous system also rises.
  - Arterial resistance subsequently increases and inflow is reduced.
  - If untreated there is vascular compromise and the enclosed muscle becomes ischemic.
  - Prolonged ischemia results in necrosis and ultimately, fibrosis of the muscle and surrounding soft-tissue structures.
  - As pressure increases there is loss of sensation in the distribution of the involved nerves and ultimately the local pulses are lost, although the latter is not necessary to make the diagnosis.
- The “Five Ps” of compartment syndrome:

- Pain (severe, progressive, persistent, poorly localized and poorly responding to analgesia. Exacerbated on passive muscle stretch)
- Paresthesia: first sign in leg is paresthesia at the first web space (between great and second toes)
- Paralysis: Occurs after ischemia is well established
- Pulselessness: Shown to occur late at times. Pulse may be present
- Pallor: not necessary for diagnosis, may not be present
- Poikilothermia

- Tense tender regional lymph node is typical. Pulse will be usually normally felt in compartment syndrome but may become absent if there is associated arterial injury.
- It compromises circulation and function of muscles and nerves.
- It often maintains the normal color and temperature of the fingers and distal pulses may be obliterated in spite of severe muscle ischemia.
- Muscle ischemia more than 4 hours causes muscle death and myoglobinuria. Muscle necrosis releases myoglobin which is excreted in urine, damages the kidneys leading into renal failure.
- Irreversible nerve
- damage develops if ischemia persists for 8 hours.
- Compartment pressure more than 30 mmHg is an indication for fasciotomy.
- Intracompartment pressure can be measured using 3 techniques:
  - Whitesides saline/needle technique: an 18G needle connected via a 3-way stop cock to an air-filled 20ml syringe. Air filled tubing is connected to a mercury manometer and a small amount of saline sites in the tube connected to the needle. Compression of the syringe raises the pressure till saline flows into the compartment (this is indicated by meniscus moving)
  - Arterial pressure transducer: this is a device used in ICU to measure arterial blood pressure and CVP. There is no need to inject fluid. Pressure in the saline tube equalizes with compartment. The transducer is connected to a Wick or slit catheter. Slits have many longitudinal slits to equalize pressure in the tube with compartment
  - Stryker device: a variation of the arterial pressure transducer

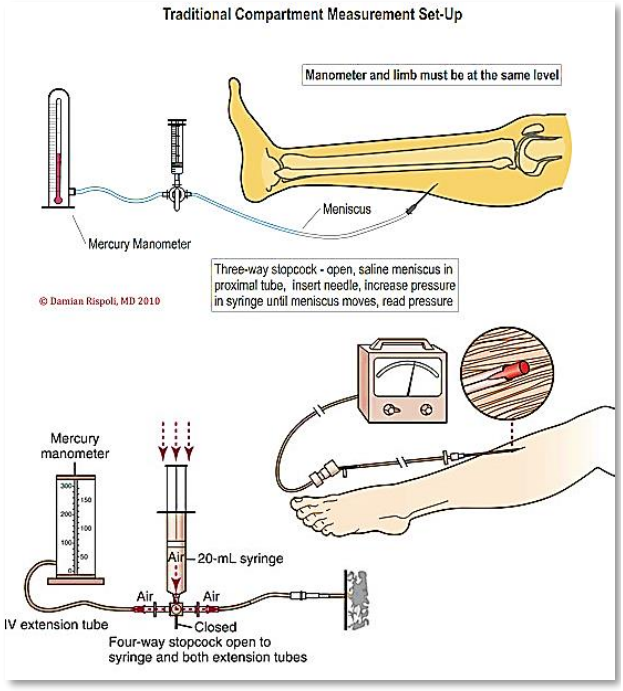


Figure 36: Whiteside saline/needle technique

- It is common in calf and forearm.
- Closed injuries cause hematoma leading to increased pressure.
- It is often associated with fracture of the underlying bone which in turn compresses the major vessels further aggravating the ischemia causing pallor, pulselessness, pain, paraesthesia, diffuse swelling and cold limb.
- If allowed to progress it may eventually lead to gangrene or chronic ischemic contracture with deformed, disabled limb.
- Treatment:
  - Fasciotomy (Under general anesthesia): when pressure exceeds 30 mmHg. Adequate length incision involving skin, fat and deep fascia should be done until underneath muscle bulges out properly. The wounds are left open, allowing swelling to occur unrestricted and dressed with appropriate sterile dressings. The wounds are closed at a later time once the acute episode has resolved. Sometimes this is not possible directly and split skin grafts are used.
  - Antibiotics
  - Catheterisation
  - Mannitol or diuretics to causes diuresis so as to flush the kidney
  - Fresh blood transfusion
  - Hyperbaric oxygen

KELOID

- Keloids ("like a claw") are common in blacks. They are much more common in females and carry a genetic predisposition often familial but can happen spontaneously.
- There is a defect in maturation and stabilization of collagen fibrils. Normal collagen bundles are absent.
- Keloids continue to grow even after 6 months, maybe for many years. They extend into adjacent normal skin and they are brownish black/ Pinkish black (due to vascularity) in color, painful, tender and

sometimes hyperaesthetic, spreads and causes itching.

- Keloids may be associated with Ehlers-Danlos syndrome or scleroderma.
- When keloids occur following an unnoticed trauma without scar formation they are called sponataneous keloids, commonly seen in Negroes.
- Some keloids occasionally become non-progressive after initial growth.
- Pathologically keloid contain proliferating immature fibroblasts, proliferating immature blood vessels and type III thick collagen stroma.
- Site: common over the sternum. Other sites are upper arm, chest wall, lower neck in front.
- Differential diagnosis: hypertrophic scar.
- Treatment: controversial
  - Steroid injection- intrakeloidal triamcinolone, is injected at regular intervals, may be once in 7-10 days, of 6-8 injections.
  - Steroid injection-excision-steroid injection
  - Methotrexate and vitamin A therapy into the keloid
  - Silicone gel sheeting, topical retinoids
  - Laser therapy
  - Vitamin E/ palm oil massage
  - Intralesional excision retaining the scar margin may prevent recurrence. It is ideal and better than just excision
  - Excision and irradiation or irradiation alone
  - Excision and skin grafting may be done.
- Note: excision and primary suturing has got high recurrence rate (more than 50%), hence it is not usually practiced.

HYPERTROPHIC SCAR

- Occurs anywhere in the body.
- Not genetically predisposed or familial.
- Growth usually limits up to 6 months.
- It is limited to the scar tissue only. It will not extend to normal skin.
- It is pale brown in color, not painful, non-tender.
- Often self-limiting also. It responds very well to steroid injection
- Recurrence is uncommon.
- It is common in wounds crossing tension lines, deep dermal burns, wounds healed by secondary intension.
- Complications:
  - Often this scar breaks repeatedly and causes infection, pain
  - After repeated breakdown it may turn into Marjolin's ulcer.

- Treatment: it is controlled by pressure garments or often revision excision of scar and closure, if required with skin graft.

SURGICAL WOUND INFECTION

ULCERS

- An ulcer is a break in the continuity of the covering epithelium (either skin/ mucous membrane) associated with microscopic/molecular death.

PARTS OF AN ULCER

- Ulcers have the following parts:
  - Margin
  - Edge
  - Floor
  - Base

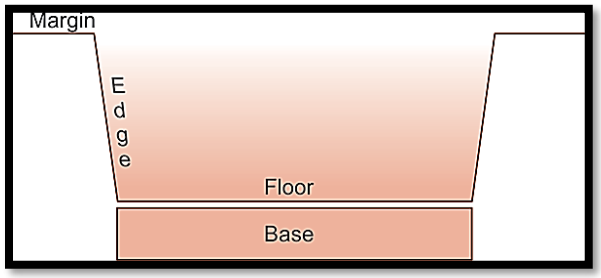


Figure 37: Parts of an Ulcer (Image Adapted from SRB's Manual of Surgery).

MARGIN

- This may be regular or irregular.
- It may be round or oval.

EDGE

- This connects the floor to the margin.
- There are different types of edges.
- The margin indicates the nature of the lesion.

SLOPING EDGE/SHELVING

- This is seen in a healing ulcer.
- Its inner part is red because of red, healthy granulation tissue.
- Its outer part is white due to scar/ fibrous part.
- Its middle part is blue due to epithelial proliferation.

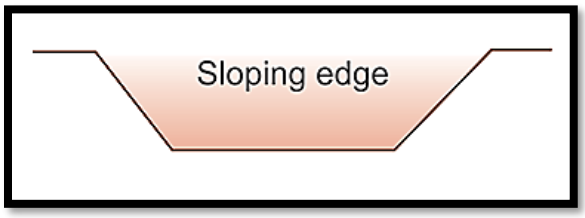


Figure 38: A healing ulcer (Image Adapted From SRB's Manual of Surgery)

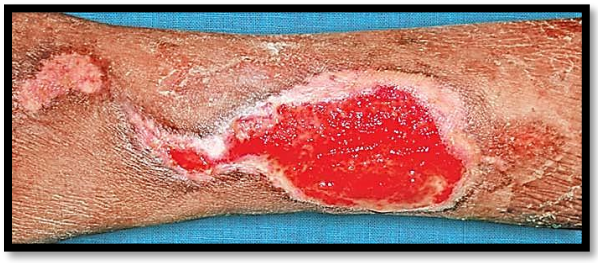


Figure 39: A healing ulcer (Image Adapted from SRB's Manual of Surgery).

UNDERMINED EDGE

- Seen in a tuberculous ulcer as well as decubitus ulcers (pressure sores).
- Disease process advances in deeper planes (in subcutaneous tissue) whereas skin proliferates inwards.

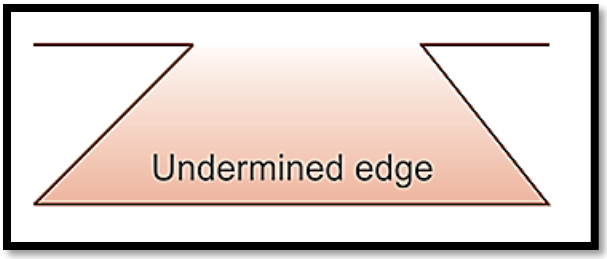


Figure 40: Undermined ulcer in TB (Image Adapted from SRB's Manual of Surgery).



Figure 41: Tuberculous ulcer on Chest wall (Image Adapted from SRB's Manual of Surgery).



Figure 42: Tuberculous ulcer around ankle (Image Adapted from SRB's Manual of Surgery).

PUNCHED-OUT EDGE

- Punched-out ulcers have sharp edges.
- Seen in a gummatous (syphilitic ulcer) and trophic ulcer (pressure sores/decubitus ulcers).
- It is due to end-arteritis.

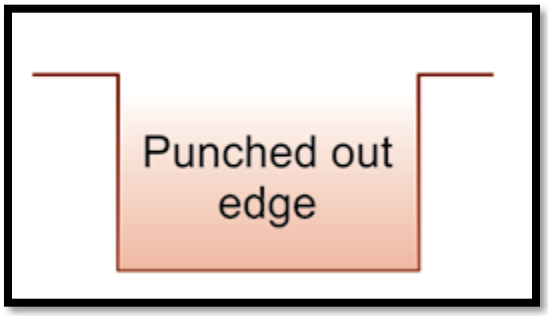


Figure 43: Punched out ulcer (Image Adapted from SRB's Manual of Surgery).

ELEVATED/EVERTED EDGE (ROLLED OUT EDGE)

- It is seen in a carcinomatous ulcer (basal cell or squamous cell carcinoma of the skin) due to spill of the proliferating malignant tissues over the normal skin.

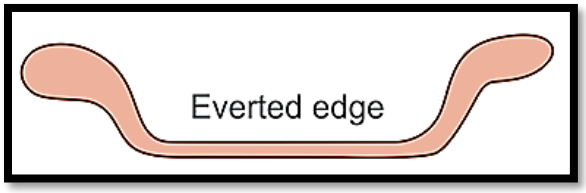


Figure 44: Elevated edge (Image Adapted from SRB's Manual of Surgery).

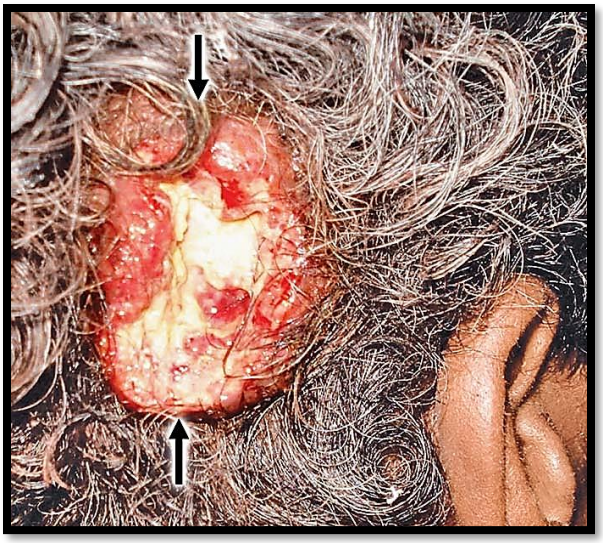


Figure 45: Squamous cell carcinoma scalp. Note the ulceroproliferative lesion with everted edges (Image Adapted from SRB's Manual of Surgery).

RAISED AND BEADED EDGE (PEARLY WHITE)

- Seen in a rodent ulcer (Basal cell carcinoma).
- Beads are due to proliferating active cells.

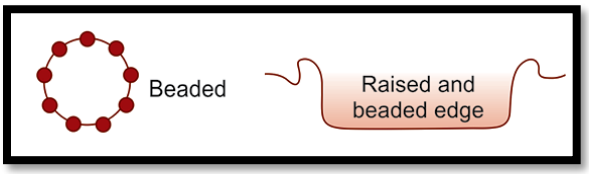


Figure 46: Raised and beaded edge (Image Adapted from SRB's Manual of Surgery).



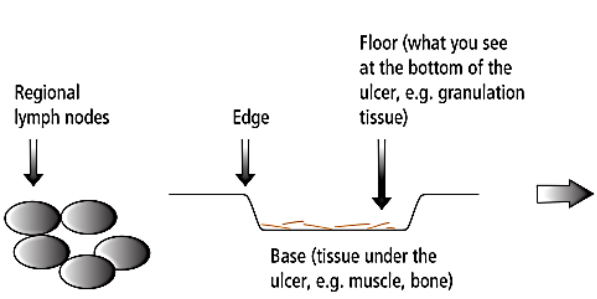
Figure 47: Basal cell carcinoma of face- Rodent ulcer. Edge is raised and beaded in appearance (Image Adapted from SRB's Manual of Surgery).

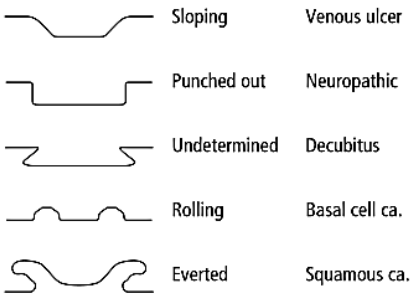
FLOOR

- It is the one that is seen.
- It may contain discharges, slough, or granulation tissue.

BASE

- This is where the ulcer rests on. It is not seen but felt.
- It may rest on bone or soft tissue.
- If there is inflammatory involvement, the surrounding tissues feel indurated and the ulcer appears fixed and is tender. Fixation and induration may be the result of neoplastic infiltration of the deeper tissue if ulcer is malignant. In this instance fixation is not accompanied by tenderness.





INDURATION OF AN ULCER

- Induration is a clinical palpatory sign which means a specific type of hardness in the diseased tissue.
- It is obvious in well-differentiated carcinomas.
- It is better felt in squamous cell carcinoma.
- It is also observed in long standing ulcer with underlying fibrosis.
- It is absent/ less in poorly differentiated carcinoma and malignant melanoma.
- Less indurated carcinoma is more aggressive.
- Specific types of induration are observed in venous diseases and chronic deep venous thrombosis.
- Brawny induration is a feature of an abscess.
- Induration is felt at edge, base and surrounding area of an ulcer.
- Induration at surrounding areas signifies the extent of disease (tumor)
- Outer most part of the induration is taken as the point from where clearance of wide excision is planned.

WAGNER'S CLASSIFICATION OF AN ULCER

- Grade 0: pre-ulcerative lesion/ healed ulcer
- Grade 1: superficial ulcer
- Grade 2: Ulcer deeper to subcutaneous tissue exposing soft tissue or bone
- Grade 3: abscess formation underneath/ osteomyelitis
- Grade 4: Gangrene of part of the tissue/ limb foot
- Grade 5: Gangrene of entire one area/foot

CLASSIFICATION OF ULCERS

- Uses two types of classification systems:
  - Clinical
  - Pathological

CLINICAL

- Ulcers classified based on:
  - Location:
    - Arterial ulcer- lateral
    - Venous- dorsum/ medial part of foot
  - Floor of ulcer: granulation
  - Discharge:
    - Serous (granulation)
    - Blood (malignant)
    - Purulent (spreading)

- Edges
  - Sloping: healing
  - Punched out: syphilis and trophic ulcers/pressure ulcers
  - Undermined- TB
  - Rolled out/ elevated- malignant
  - Raised and beaded- rodent (malignant)
- Surrounding area
  - Thick and pigmented skin (varicose ulcer)
  - Thick and dark (arterial ulcer)

PATHOLOGICAL

- Ulcers can either be:
  - Specific ulcers which are associated with a cause
    - Tuberculous ulcer, syphilitic ulcer (punched out, deep. With "wash leather" slough in the floor and with indurated base). Actinomycosis, meleney's ulcer.
  - Malignant ulcers: carcinomatous ulcer (marjolin's ulcer), rodent ulcer, melanotic ulcer
  - Non-specific ulcers not associated with a specific cause
    - Traumatic ulcer (common): may be mechanical, physical, chemical
    - Arterial ulcer: Atherosclerosis, TAO
    - Venous ulcer, Gravitational ulcer, Post-phlebitic ulcer
    - Trophic/ pressure ulcer
    - Infective ulcer: pyogenic
    - Tropical ulcers: it occurs in tropical countries. It is callous type of ulcer e.g. Vincent's ulcer.
    - Ulcers due to chilblains and frostbite (cryopathic ulcers)
    - Bazin's ulcer
    - Martonell's hypertensive ulcer
    - Diabetic ulcer
    - Ulcers due to leukemia, polycythemia, jaundice, collagen diseases, lymphedema.
    - Cortisol ulcers are due to long time application of cortisol (Steroid) creams to certain skin diseases. These ulcers last for long time and require excision and skin grafting.

GRANULATION TISSUE

HEALTHY GRANULATION TISSUE

- Occurs in a healing ulcer.
- It has a slopping edge.
- It bleeds on touch and has a serous discharge.

- 5Ps of granulation:
  - Pink
  - Punctate hemorrhage (bleeds on touch)
  - Pulsates
  - Painless
  - Pin head granulation
- Skin grafting takes up well with healthy granulation tissue.
- *Streptococci* growth in culture should be $<10^5$/ gram of tissue before skin grafting.

UNHEALTHY GRANULATION TISSUE

- It is pale with purulent discharge.
- Its floor is covered with slough, its edge is inflamed and edematous.
- It is a spreading ulcer.
- Unhealthy, pale, flat granulation tissue is seen in chronic non-healing (callous ulcer).

EXAMINATION AND ASSESMENT OF AN ULCER

HISTORY

- Mode of onset
- Duration
- Pain- its time of onset, progress, severity
- Discharge from ulcer
- History suggestive of associated disease/ treatment history
- The cause of the ulcer should be found- diabetes/ venous/ arterial/ infective

LOCAL EXAMINTION OF AN ULCER

INSPECTION

- Site of ulcer
  - Arterial ulcer: digits
    - Arterial ulcers are usually found on the lateral aspect of the foot.
  - Venous ulcer: malleoli (medial malleolus- commonly)
    - Venous ulcers are usually found on the medial aspect of the foot.
  - Trophic ulcer: heel/pressure points.
- Size of ulcer
- Shape of ulcer
- Depth of the ulcer
- Number
- Margin whether regular/irregular/ well-defined/ ill-defined
- Edge of the ulcer
  - Indurated (fibrosis): squamous cell carcinoma and chronic ulcers
- Floor of the ulcer- floor is what one sees. It rests on the base (base is not seen; it is only felt).
  - Red color in floor- healing ulcer
  - Slough with pale/purulent discharge- non-healing ulcer or tubercular

  - Wash leather slough-syphilitic ulcer
  - Proliferative and nodular floor-squamous cell carcinoma
  - Pigmented- melanoma, Pigmented basal cell carcinoma
- Discharge from ulcer bed
  - Serous: in healing ulcer
  - Purulent: in infected ulcer
    - *Staphylococci*: yellowish and creamy
    - *Streptococci*: bloody and opalescent
    - *Pseudomonas*: Greenish color due to pseudocyanin
  - Bloody: malignant ulcer, healing ulcer from healthy granulation tissue
  - Seropurulent
  - Seroanguinous: serous and blood
  - Serous with Sulphur granules: actinomycosis
- Surrounding area to be examined for inflammation, edema, eczema, scarring, pigmentation
- Inspection of the entire part/ limb

PALPATION

- Tenderness over edge, base and surrounding area.
- Warmness over surrounding area.
- Edge palpation for induration
- Palpation of base for induration/fixity
  - Fixed: malignant
  - Non-fixed: non malignant
- Depth of ulcer- trophic ulcer is deep with bone as its base- often it is measured gently in mm.
- Bleeding on palpation and touching.
- Palpation for deeper structures and its relation to ulcer.
- Surrounding skin and tibia/ calcaneum/ other related bones for thickening.
- Examination of adjacent joint for mobility.
- Examination of regional lymph nodes is essential- tenderness (Acute infection), mobility, consistency may be hard (Carcinoma metastasis)/ firm/soft and non-tender (inflammatory), fixity (malignancy), ulceration or fungation (malignancy), sinus (nonspecific, tuberculosis or carcinoma)
- Palpation also of arterial pulse, peripherally in relation to ulcer.

AUSCULATION

- Auscultation of peripheral arteries for any bruits.

SPECIFIC SYSTEM

- Examination for varicose veins in standing position.
- Examination of the abdomen for splenomegaly (sickle cell disease), hepatomegaly.
- Examination of spine (gibbus, paraspinal spasm, movements) and

neurological system like sensation and muscle power.

INVESTIGATIONS FOR AN ULCER

- Study of discharge: culture and sensitivity, AFB study, cytology.
  - For skin graft swabs and culture must yield staphylococci of $<10^5$/ gram
- Edge biopsy: biopsy taken from the edge because contains multiplying cells. Usually 2 biopsies are taken. Biopsy taken from the center may be inadequate because of central necrosis.
- FNAC of the lymph node (fine needle aspiration cytology)
- X-ray of the part to look for periostitis/ osteomyelitis
- CXR, Mantoux test is suspected in cases of a tuberculous ulcer.
- Full blood count
- NOTE: ulcer will not granulate if Hb is less than 10g/dl and serum albumin is <3gm%

MANAGEMENT OF AN ULCER

1. Investigate and treat underlying cause.
2. Correct the deficiencies like anemia, protein and vitamin deficiencies.
3. Transfuse blood if required.
4. Control pain (analgesics)
5. Control infection (antibiotics) and give rest to the part
6. Care of ulcer by debridement, ulcer cleaning and dressing
   - Ulcer cleaning
     - Done using dilute povidone iodine and normal saline (ideal)
     - It should be done daily or 2 times a day depending on the severity.
   - Debridement of ulcer
     - It is removal of devitalized tissue.
     - Small ulcers are debrided in ward.
     - Large ulcers are debrided in operation theatre under general anesthesia
     - All dead, devitalized, necrotic tissue are removed.
     - Slough should be separated adequately before debridement.
     - Often devitalized tissue separates on its own by autolysis.
     - Enzymes like collagenase are used for debridement.
     - Hydrotherapy and dressings are mechanical non-selective method of debridement.
     - Note: debridement can be surgical, mechanical, autolytic or enzymatic.

- Dressing of an ulcer aims to:
  - Keep the ulcer moist
  - Keep the surrounding skin dry
  - Reduce pain
  - Soothe the tissue
  - Protect the wound
  - Absorb any discharge
- Ulcer dressing:
  - Cotton dressing- cheap but traumatic
  - Paraffin dressing
  - Polyurethane dressing used in clean wounds.
  - Alginates (Seaweed) dressing used when there are heavy exudates
  - Type 1 collagen dressing cause hemostasis, proliferation of fibroblast and improves blood supply.
  - Foam dressing are highly absorbent, decrease the wound maceration and reduce the frequency of dressing- hydrophilic foam.
  - Hydrocolloid dressing help in separation of slough and autolysis of dead tissues.
  - Transparent film dressing are waterproof permit oxygen and water vapor across and prevent contamination.
  - Hydrogel dressings used to clean wounds.

- Causes of formation of chronic ulcers in skin: recurrent infection, trauma, absence of rest, poor blood supply, hypoxia, edema of area, loss of sensation, malignancy, specific causes like tuberculosis, fibrosis, periostitis or osteomyelitis of the underlying bone.

DIABETIC FOOT AND DIABETIC GANGRENE

- A diabetic foot is a foot that exhibits any pathology that results directly from diabetes mellitus or any long term (or chronic) complication of diabetic mellitus.
- Presence of several characteristic diabetic foot pathologies such as infection, diabetic foot ulcer and neuropathic osteoarthropathy is called diabetic foot syndrome.

PATHOGENESIS OF DIABETIC FOOT/GANGRENE

- High glucose level in tissues is a good culture media for bacteria. So infection is common.
- Diabetic microangiopathy causes blockade of microcirculation leading to hypoxia.
- Diabetic atherosclerosis: reduces blood supply and causes gangrene. Thrombosis can be precipitated by infection causing infective gangrene. Blockage occurs at plantar, tibial and dorsalis pedis vessels.
- Increased glycosylated hemoglobin: causes defective oxygen dissociation leading to more hypoxia. At tissue level there will be increased glycosylated tissue proteins, which prevent proper oxygen utilization and so aggravates hypoxia.
- Diabetic neuropathy: due to sensory neuropathy minor injuries are not noticed and so infection occurs. Due to motor neuropathy, dysfunction of muscles, arches of foot and joints occur. And loss of reflexes of foot occurs causing more prone for trauma and abscess. Due to autonomic neuropathy skin will be dry, causing defective skin barrier and so more prone to infection.

CLINICAL FEATURES

- Pain in the foot
- Ulceration
- Absence of sensation
- Absence of pulsations in the foot (posterior tibial and dorsalis pedis arteries)
- Loss of joint movements
- Abscess formation
- Change in temperature and color when gangrene sets in
- Patient may succumb to ketoacidosis, septicemia or myocardial infarction.

MEGGITT'S CLASSIFCATION OF DIABETIC FOOT

- Grade 0: foot symptoms like pain, only
- Grade 1: Superficial ulcers
- Grade 2: deep ulcers
- Grade 3: ulcers with bone involvement
- Grade 4: forefoot gangrene

- Grade 5: full foot gangrene

INVESTIGATIONS

- Blood investigations:
  - Blood sugar and ketones
  - Glycosylated hemoglobin
  - Blood urea and serum creatinine
- Urine ketones
- Imaging:
  - X-ray of part to look for osteomyelitis.
  - Doppler study of lower limb to assess arterial patency.
  - Angiogram to look for proximal blockage
  - Ultrasound of abdomen to see the status of abdominal aorta
- Pus for culture and sensitivity.

TREATMENT

- Foot can be saved only if there is good blood supply:
  - Antibiotics- decided by pus C/S
  - Regular dressing
  - Drugs: vasodilators, pentoxiphylline, dipyridamole, low dose aspirin.
  - Diabetes is controlled by insulin only.
  - Diet control, control obesity.
  - Surgical debridement of wound
  - Amputations of gangrenous area. Level of amputation has to be decided by skin changes and temperature changes or Doppler study.
- Care of feet in diabetes:
  - Any injury has to be avoided.
  - MCR foot wears must be used (microcellular rubber).
  - Feet has to be kept clean and dry especially the toes and clefts.
  - Hyperkeratosis has to be avoided.

BURNS

- Burns are a type of coagulative necrosis causing injury to the skin or other tissues as a result of flames, friction, radiation, electrical, chemical or heat.
- Recall that the skin consists of:
  - The epidermis: contains 5 histological layers (Stratum Basale, Stratum Spinosum, Stratum Granulosum, Stratum Lucidum, and Stratum Corneum)
    - The stratum Lucidum is only found in thick skin (palm of hands and soles of feet) and not in thin skin.
    - The stratum Basale and Spinosum are referred collectively as the Malpighian layer.
  - The dermis: it is divided into the upper papillary dermis and lower reticular dermis. It contains blood vessels, nerves and connective tissue.
  - Subcutaneous tissue (hypodermis): contains adipose.
- The majority of burns in children are scalds caused by accidents with kettles, pans, hot drinks and bath water.
- Among adolescents patients, the burns are usually caused by young males experimenting with matches and flammable liquids.
- In adults scalds are not uncommon but are less frequent than flame burns. Most electrical and chemical injuries occur in adults.

ETIOLOGY AND MECHANISM OF INJURY

- Heat (Thermal injury)
  - Scald- spillage of hot liquids. Common in children (70%), child abuse especially if the pattern of the burn does not fit the description of the event given by the parents e.g. burns of both buttocks typically produced by holding a small child by arms & legs and dunking them into boiling water. They also occur in adults and tend to be superficial to dermal burns.
  - Flame burns: due to fire. Common in adults (50%) and associated with inhalational injury and other concomitant trauma. They tend to be dermal or full thickness burns.
  - Contact burns- contact with hot metals/objects/materials. Commonly seen in epileptics, alcoholics and drug abusers as well as in the elderly due to loss of consciousness. They tend to be dermal or full thickness burns.
  - Flash burns due to exposure of natural gas, alcohol, combustible liquids.

- Cold (frost bite)
- Sun burns: these are usually superficial burns (they are very painful)
- Electrical injury: these are always deeper and worse than they appear. Massive debridements or amputations may be required.
  - Classification of electrical burns:
    - Low voltage (<1000 V): deep contact burns at entry and exit sites.
    - True high voltage (>1000 V): current goes through body. Extensive soft tissue and bone necrosis.
    - Flash high voltage (> 1000V): current does not pass through the body. 'Flash' burn from heat generated by current arc from high-tension source. Exposed parts of body affected (Face, hands).
  - Electrical burns may be associated with:
    - Myoglobinemia-myoglobinuria and renal failure: give plenty of fluids and osmotic diuretics like mannitol and alkalinize the urine
    - Orthopedic injuries secondary to massive muscle contractions: posterior dislocation of the shoulder, compression fractures of vertebral bodies and
    - Late development of cataracts and demyelination syndromes.
  - Electrical burns require ECG monitoring.
- Chemicals (acid/alkali): require massive irrigation to remove offending agent. Alkaline burns (Liquid Plumr, Drano) are worse than acid burns (battery acid). Alkaline burns are worse because they cause liquefactive necrosis. Irrigation must begin as soon as possible at the site where the injury occurred (tap water, shower). Do not "play chemist" and attempt to neutralize the agent. These tend to be deep burns.
- Ionizing radiation burns and Friction burns.
- Respiratory burns (inhalation injuries): Occur with flame burns in an enclosed space (e.g. burning building, car, plane).
  - They are chemical injuries caused by smoke inhalation.
  - Burns around the mouth or soot inside the throat, burnt/loss of nasal hair, change in voice and stridor are suggestive clues.
  - Diagnosis is confirmed with fiberoptic bronchoscopy however the key issue is whether respiratory support (A respirator) is needed or not, and blood gases are best to make that determination.
  - Intubation should be done if there is any concern about adequacy of the airway. Monitor carboxyhemoglobin and give 100% oxygen for 24 hours to shorten its half-life if elevated.

PATHOPHYSIOLOGY

- Burns cause damage in a number of different ways, but by far the most common organ affected is the skin.
- Burns result in local (Jackson Zones of a burn) and systemic response (affecting the cardiovascular, respiratory, metabolic, gastrointestinal and immunological system)

LOCAL RESPONSE

ZONES OF A BURN (JACKSON)

- 3 Zones of a burn were described by Jackson in 1947. These include:
  - Zone of coagulation: this is the point of maximum damage. There is irreversible tissue loss due to coagulation of constituent protein.
  - Zone of stasis: this is characterized by decreased tissue perfusion. The tissue in this zone is potentially salvageable. Additional insults, such as prolonged hypotension, infection or edema can convert this zone into an area of complete tissue loss.
  - Zone of hyperemia: tissue perfusion is increased. This zone will invariably recover.
- In a burn wound, these zones are 3-dimensional, loss of tissue in the zone of stasis will lead to the burn deepening.

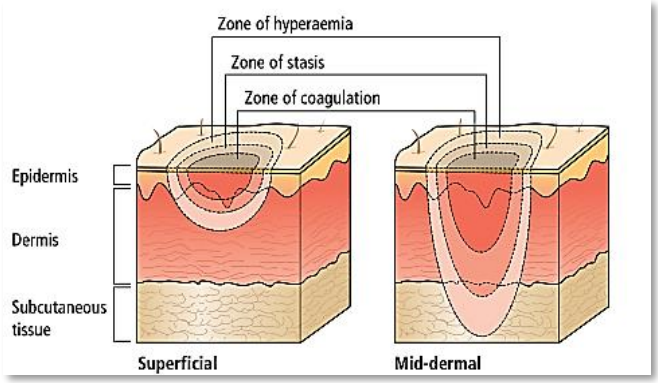


Figure 48: Jackson's burn zones (Image from Clinical Surgery 2nd Ed)

SYSTEMIC RESPONSE

- The release of cytokines and other inflammatory mediators at the site of injury has a systemic effect once the burn reaches 30%.
  - Activation and release of various complement factors, histamine and prostaglandins results in myocardial dysfunction, edema of tissues, reduced immunoglobulin synthesis.
  - Catecholamine levels are raised drastically in patients with burns. There will be lipolysis, proteolysis, increased release of glutamine and alanine from skeletal muscles.
  - Urea production is increased due to more proteolysis.

RESPIRATORY CHANGES

- Hot gasses may physically burn the nose, mouth, tongue, palate and larynx. Once injured these linings start to swell. After a few hours they may start to interfere with the larynx and may completely block the airway if action is not taken to secure the airway.
- Inhaled hot gases, steam, smoke particle, and poisons have different effects on the respiratory system:
  - Inhaled hot gases can cause supraglottic airway burns and laryngeal edema
  - Inhaled steam can cause subglottic burns and loss of respiratory epithelium
  - Inhaled smoke particles can cause chemical alveolitis and respiratory failure
  - Inhaled poisons such as carbon monoxide can cause metabolic poisoning
  - Full thickness burns to the chest can cause mechanical blockage to rib movement.
- Injury to the airways below the larynx is rare because exchange mechanisms in the supraglottic airway are usually able to safely absorb the heat from the hot air. However, steam has a large latent heat of evaporation can cause thermal damage to the lower airway. In such injuries the respiratory epithelium rapidly swells and detaches from the bronchial tree. This creates casts which can block the main upper airway.
- There are many poisonous gases that can be given off in a fire with carbon monoxide being the most common.
- Recall:
  - Carbon monoxide is a production of incomplete combustion that is often produced by fires in enclosed spaces.
  - CO is the usual cause of a person being found with altered consciousness at the scene of a fire.
  - CO binds to hemoglobin with a higher affinity than oxygen (240 times) and therefore blocks the transport of oxygen.
  - When CO binds hemoglobin it forms a stable compound carboxyhemoglobin. Carboxyhemoglobin in the bloodstream can be measured. Concentrations above 10% are dangerous and need treatment with pure oxygen for more than 24 hours. Death occurs with concentrations around 60%.
- Another metabolic toxin produced in house fires is hydrogen cyanide which causes a metabolic acidosis by interfering with mitochondrial respiration.
- Inhalational injury is caused by the minute particles within thick smoke, which because of their small size are not filtered by the upper airway and are carried

down to the lung parenchyma. They stick to the moist lining causing an intense reaction in the alveoli. This chemical pneumonitis causes edema within the alveolar sacs and decreasing exchange over the ensuing 24 hours and often gives rise to bacterial pneumonia. Its presence has a very significant effect on the mortality of any burn patient.

- Note also that burned skin is very thick and stiff and this can physically stop the ribs moving if there is a large full-thickness burn across the chest.

- Pulmonary changes are due to:
  - Massive edema due to altered pressure gradient because of the injury to basement membrane and systemic inflammatory response syndrome (SIRS).
  - Bronchoconstriction which may be seen secondary to inflammatory mediators.
  - Air way edema causing adult respiratory distress syndrome (ARDS)
    - Note: Pulmonary edema in burns can be due to burn injury, fluid overload, inhalation injury
  - Inhaled hot gases leading to supraglottic airway burns and laryngeal edema
  - Inhaled steam causing subglottic burns and loss of respiratory epithelium
  - Inhaled smoke particles causing chemical alveolitis (and superadded bacterial pneumonia) and respiratory failure
  - Inhaled poisons such as carbon monoxide causing metabolic poisoning
  - Full thickness burns to the chest can cause mechanical blockage to rib movement.

CARDIOVASCULAR RESPONSE

- Cardiovascular changes occur because burned skin activates various inflammatory processes.
- The release of neuropeptides and activation of complement are initiated by the stimulation of pain fibers and alteration of proteins by heat.
- Activation of Hageman factor initiates a number of protease-driven cascades altering the arachidonic acid, thrombin and Kallikrein pathways.
- Complement causes degranulation of mast cells and coats the proteins altered by burn. This attracts neutrophils which also degranulate and release free radicals and proteases in large amounts causing more damage to the tissue.

- Additionally, Mast cells also release primary cytokines such as tumor necrosis factor alpha (TNF-alpha). These act as chemotactic agents to inflammatory cells and cause release of many secondary cytokine.
- Inflammatory factors:
  - Alter permeability of blood vessels causing intravascular fluid escape.
  - Increase permeability such that large proteins can also escape with ease. The damaged collagen and extravasated proteins increase the oncotic pressure within the burned tissue further increasing the flow of water from the intravascular to the extravascular space.
  - Vasoconstriction raising capillary hydrostatic pressure leading into cellular aggregation.
- Note: There is a net flow of water, solutes and proteins from the intravascular to the extravascular space. This flow occurs over the first 36 hours after the injury but does not include red blood cells. Blockage of lymphatics causes poor clearance of fluid and proteins from interstitial spaces.
- In small burns, the reaction is small and localized, as the size approaches 10-15% of total body surface area (TBSA), the loss of intravascular fluid can cause a level of circulatory shock. Increase to 25% of TBSA causes an inflammatory reaction that culminates in fluid loss in vessels away from the burn injury. This is why it is important to measure the TBSA involved in any burn as it dictates the size of inflammatory reaction and the amount of fluid needed to control shock.
- In full-thickness burns, the collagen fibers are coagulated. The normal elasticity of skin is lost and therefore a circumferential full-thickness burn to a limb acts as a tourniquet as the limb swells. If untreated will progress to limb-threatening ischemia.

- Cardiovascular changes:
  - Increase in capillary permeability, leading to loss of protein and fluids from the intravascular into the interstitial compartment (hypovolemia).
  - Vasoconstriction raising capillary hydrostatic pressure leading into cellular aggregation.
  - Blockage of lymphatics causes poor clearance of fluid and proteins from interstitial spaces.
  - Release of hormones like catecholamines, vasopressin, angiotensin.
  - Peripheral and splanchnic vasoconstriction
  - Decreased myocardial contractility possibly due to tumor necrosis factor-alpha.
  - Limb-threatening ischemia in circumferential full thickness burns affecting the limbs
- These changes coupled with fluid loss from the burn wound result in systemic hypotension and end-organ damage.

GASTROINTESTINAL RESPONSE

- The inflammatory stimulus and shock can cause microvascular damage and ischemia to the gut mucosa. This reduces gut motility and can prevent absorption of food.
- Failure of enteral feeding in large burns is a life-threatening complication.
- This process also increases the translocation of gut bacteria, which can become an important source of infection in large burns.
- Gut mucosal swelling, gastric stasis and peritoneal edema can also cause abdominal compartment syndrome which splints the diaphragm and increases the airway pressures needed for respiration.

- Gastrointestinal changes:
  - Microvascular and ischemia to the gut mucosa causing GIT mucosa ulceration (Curling's ulcer)
  - Reduced gut motility and reduced absorption of food (failure to enteral feed)
  - Increased translocation of gut bacteria
  - Gut mucosal swelling, gastric stasis and peritoneal edema causing abdominal compartment syndrome which splints the diaphragm increasing the airway pressures needed for respiration.
  - Acute gastric dilation (in 2-4 days)
  - Paralytic ileus
  - Cholestasis and hepatic damage
  - Acute acalculous cholecystitis, and acute pancreatitis

RENAL AND METABOLIC RESPONSE

- Due to hypovolemia and shock there is release of ADH from posterior pituitary to cause maximum water reabsorption and release of aldosterone from adrenals to cause maximum sodium reabsorption.
- Pre-renal failure (due to hypovolemia and acute tubular necrosis).
- Toxins released from the wound along with sepsis can cause acute tubular necrosis.
- Myoglobin released from muscles (in case of electric injury or often from eschar) is most injurious to kidneys. (acute kidney injury)
- Metabolic changes:
  - Hypermetabolic rate (due to release of catecholamines, glucagon)
  - Negative nitrogen balance i.e. urinary nitrogen losses are greater than oral nitrogen intake.
  - Electrolyte imbalance
    - Hyponatremia
    - Hyperkalemia
  - Deficiencies of vitamins and essential elements
  - Metabolic acidosis due to hypoxia and lactic acid

- Renal changes:
  - Pre-renal failure (due to hypovolemia and acute tubular necrosis)
  - Acute kidney injury (due to myoglobin)
  - Release of ADH for water reabsorption
  - Release of aldosterone for sodium retention.
- Metabolic changes:
  - Hypermetabolic rate
  - Negative nitrogen balance
  - Electrolyte imbalance
    - Hyponatremia
    - Hyperkalemia
  - Deficiency of vitamins and essential elements
  - Metabolic acidosis due to hypoxia and lactic acid

IMMUNOLOGICAL RESPONSE

- Cell mediated immunity is significantly reduced in large burns, leaving an individual more susceptible to bacterial and fungal infection.
  - There is a non-specific downregulation of the immune response, affecting both cell-mediated and humoral pathways.
  - Loss of immunity, loss of proteins and immunoglobulins, loss of barrier causes sepsis. Opportunistic infection is also common.
  - Associated conditions such as DM, HIV, infections, old age, respiratory diseases worsen the sepsis in burn injury.
- Potential sources of infection include burn wound, lungs (if injured), from central venous lines, tracheostomies or urinary catheters present.
- Infection commonly due to
  - *Streptococci* (Beta hemolytic- most common)
  - *Pseudomonas*
  - *Klebsiella*
  - *Staphylococci*
  - Other gram-negative organisms
  - *Candida albicans*
- Systemic infection like pneumonia, bacteria, septicemia can occur. Sepsis is identified by fever, lethargy, leukocytosis and thrombocytopenia.

- Immunological response:
  - There is a non-specific downregulation of the immune response, affecting both cell-mediated and humoral pathways.
  - Loss of immunity, loss of proteins and immunoglobulins, loss of barrier causes sepsis. Opportunistic infection is also common.
  - Associated conditions such as DM, HIV, infections, old age, respiratory diseases worsen the sepsis in burn injury.
- Infection commonly due to
  - *Streptococci* (Beta hemolytic- most common)
  - *Pseudomonas*
  - *Klebsiella*
  - *Staphylococci*
  - Other gram-negative organisms
  - *Candida albicans*
- Systemic infection like pneumonia, bacteria, septicemia can occur.

OTHER RESPONSES

- CNS:
  - Altered mental state (electrolyte imbalance/ shock)
  - Comatose

CLINICAL FEATURES

- History of burn.
- Pain, burning, anxious status, tachycardia, tachypnea, fluid loss.
- In severe degrees features of shock. Tolerable temperature of human skin is $40^{O}C$ for brief period.

CLASSIFICATION OF BURNS

- Burns are classified according to:
  - Thickness of skin involved.
  - Percentage body surface area involved.
  - Severity

THICKNESS OF SKIN INVOLVED

- Depending on the thickness of skin involved burns can be classified as:
  - First degree burns- Also known as superficial burns
  - Second degree burns include:
    - Superficial partial thickness burns
    - Deep partial thickness burns
  - Third degree also known as full thickness burn
  - Fourth degree burns

FIRST DEGREE (SUPERFICIAL BURNS)

- First degree: Affects only the epidermis (Also known as superficial thickness burns).
- Here the epidermis looks red and painful, no blisters, heals rapidly in 5-7 days by epithelialization without scarring. Skin peels.
- Pin-prick test is positive (severe pain to pin prick).
- Example: non-blistering sun burns.



Figure 49: First degree/ Superficial thickness burns (Courtesy of SRB's Manual of Surgery 4th Ed).

SECOND DEGREE

- These are divided into:
  - Superficial partial thickness burns (Superficial dermal burns): affecting the epidermis and the papillary dermis
  - Deep partial thickness burns (deep dermal burns): affecting the epidermis, the upper dermis and the reticular dermis.
- The affected area is mottled, red, painful with blisters, and heals by epithelialization in 14-21 days (2-3 weeks) without scarring.
- Hallmark feature: Blistering.
- Superficial partial thickness (Superficial dermal):
  - Heals and causes pigmentation
  - They cause blisters.
  - Blanches with pressure.
  - They tend to be pink below blister, moist and very painful.



Figure 50: Second Degree burns (superficial partial) with blistering (Courtesy of SRB's Manual of Surgery 4th Ed).

- Deep partial thickness (Deep dermal):
  - ➢ They are yellow or white. Deep red below blister from vasodilation, or red dots (vessels) on white background.
  - ➢ Less blanching and may be blistering.
  - ➢ They may be sensory changes
  - ➢ They are also fairly dry and heal after 3-8 weeks.
  - ➢ Heal by combination of epithelialization, pigmentation, wound contracture and scarring.

Blanching: make white or by extracting color i.e. when pressure is applied to a burn area it becomes pale.

THIRD DEGREE

- Third degree (Full thickness burns): Affect the epidermis, dermis and subcutaneous tissue.
- Affected area is charred, parchment like, painless (negative pin prick sensation) and insensitive, with thrombosis of superficial vessels.
- It requires grafting. Charred, denatured, insensitive, contracted full thickness burn is called as eschar.
- These wounds must heal by re-epithelization from wound edge.
- The skin appears stiff and white/brown. There is no blanching. With a leathery texture.
- They are painless.
- Healing is prolonged (months) and incomplete.

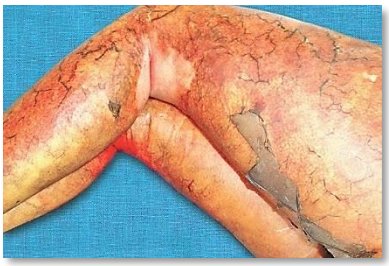


Figure 51: Third degree burns with charred skin, thrombosed veins and eschar (Courtesy of SRB's Manual of Surgery 4th Ed).

FOURTH DEGREE

- Involves the epidermis, dermis and underlying tissues- muscles, bones.
  - ➢ They are Black, charred with eschar
  - ➢ They are dry and painless



Figure 52: Face burns in an adult - severe third degree (Courtesy of SRB's Manual of Surgery 4th Ed).

Char: partially burn so as to blacken the surface.

Table 1: Classification of burns according to depth/thickness involved

Type	Layers involved	Appearance	Texture	Sensation	Healing time	Prognosis
Superficial (1st degree)	Epidermis	Red without blister	Dry	Painful	5-7 days	Heals well. Repeated sunburns increase risk for skin cancer later in life
Superficial partial thickness (2nd degree)	Extends into superficial (papillary) dermis	Redness with clear blister. Blanches with pressure	Moist	Very painful	14-21 days (2-3 weeks)	Local infection (cellulitis) but no scarring typically
Deep partial thickness (2nd degree)	Extends into deep (reticular) dermis	Yellow or white. Less blanching and may be blistering	Fairly dry	Pressure and discomfort	3-8 weeks	Scarring, contractures (may require excision and skin grafting)
Full thickness (3rd degree)	Extends through entire dermis	Stiff and white/ brown, no blanching	Leathery	Painless	Prolonged (months) and incomplete	Scarring, contractures, amputations (Early excision)
4th degree	Involves fat, muscle and bone	Black, charred with eschar	Dry	painless	Requires excision	Amputation, significant functional impairment and in some cases death

- Note: Circumferential burns of the extremities can lead to cutoff of the blood supply as edema accumulates underneath eschar. In circumferential burns of the chest, a similar mechanical problem may interfere with breathing. Escharotomies (done at the bedside, with no need for anesthesia) will provide immediate relief.
- In reality most burns will be a mixture of different depths. The assessment of depth is important for planning because the more superficial burns will tend to heal spontaneously but deeper burns will need surgical intervention.

Feature	Superficial	Superficial partial	Deep partial	Full thickness
Appearance	Red glistening wound that often blanches	Redness, with blanching and clear blisters	Paler (yellow, white), drier, less blanching and blistering	Dry, leathery, hard wound
Bleeding (use 21-G needle)	Brisk bleeding on superficial prick	Brisk bleeding on superficial prick	Delayed bleeding on deeper prick	No bleeding
Sensation (use 21-G needle	Pain	Pain	Pin can be felt but no pain	No sensation

SEVERITY

- Depending on the percentage of burns (See Table below):
  - Mild (minor)
  - Moderate
  - Severe

MILD

- Partial thickness burns <10% in adults or <5% in children.
- Full thickness burns less than 2%.
- Can be treated on outpatient basis.

MODERATE

- Second degree (Partial thickness) of 10-20% burns in adults (5-10% in children).
- Third degree (Full thickness) between 2-5% burns.
- Note: burns should not involve eyes, ears, face, hand, feet, perineum.

SEVERE

- Second degree (Partial thickness) burns more than 20% in adults, in children more than 10%.
- All third-degree burns (Full thickness) of 5% or more.
- Burns involving eyes, ears, feet, hands, perineum.
- All inhalation and electrical burns.
- Burns with fractures or major mechanical trauma.

Table 2: Classification of burns according to severity

AGE GROUP	MINOR	MODERATE	MAJOR
ADULTS	Partial thickness<10%	Second degree 10-20%	Second degree >20%
CHILDREN	Partial thickness <5%	Second degree 5-10%	Second degree >10%
Both	Full thickness <2%	2-5% full thickness High voltage injury Possible inhalation injury Circumferential burn Other health problems	Full thickness >5% High voltage Known inhalation injury Significant burn to face, joints, perineum, hands or feet Associated injuries

ASSESSMENT OF BURNS

- Note: when calculating the burn wound area, erythema should not be included. Erythema may take a few hours to fade so it is inevitable that some overestimation will occur if the burn is estimated acutely.

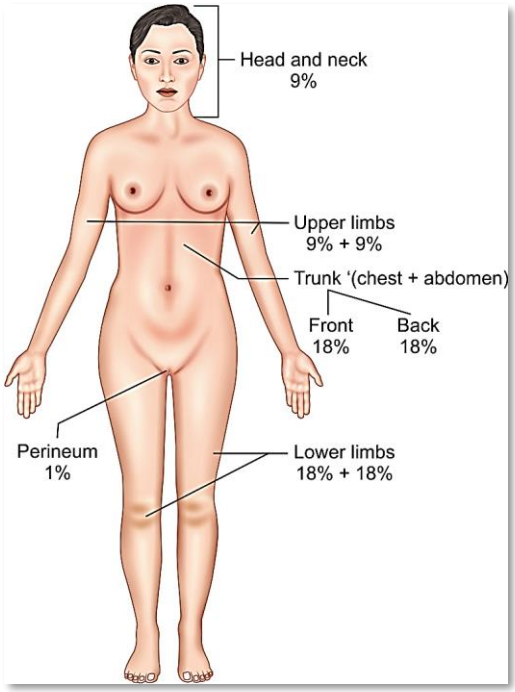


Figure 53: Wallace rule of 9s

WALLACE'S RULE OF NINE (EARLY ASSESSMENT)-ADULTS

- A percentage is assigned to each body part:
  - Head and neck- 9%
  - Arm-9% each arm
  - Trunk- 36% (9 + 9 – front chest & abdomen and 9+ 9- back chest and abdomen)
  - Leg- 18% (9% front, 9% back)
  - Perineum- 1%

RULE OF 7'S-CHILDREN

- Percentages are as follows:
  - Head and neck- 28%
  - Arms- 7% each
  - Trunk- 28% (14% front, 14% back)
  - Lower limbs-14% (7% front, 7% back) each
  - Perineum- 2%

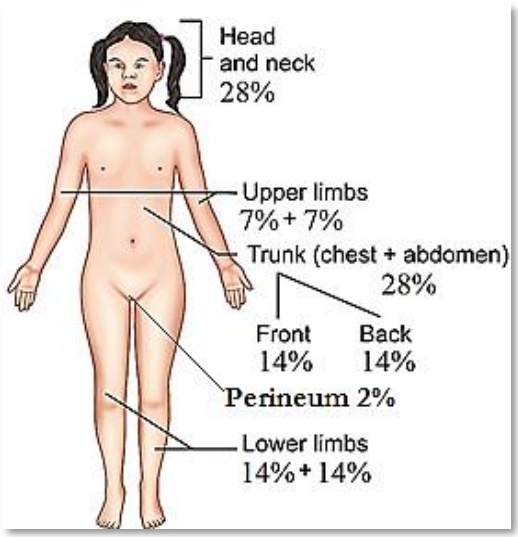


Figure 54: Rule of 7s

- This allows the emergency medical provider to obtain a quick estimate of how much body surface area is burned. For example, if an adult patient's entire back (18%) and entire left leg (18%) are burned about 36% of the patient's body surface area is affected.
- The body surface area assigned to each body part refers to the entire body part. So, if half of a patient's left leg were burned, it would be assigned a body surface area value of 9% (half of the total surface area of the leg). Thus, if a patient's entire back (18%), but only half of their left leg (9%) was burned, the amount of body surface area affected would be 27%.

- Some studies have raised concerns about the rule of nines' accuracy with obese patients, noting that the proportional contribution of various major body segments to the total body surface area changes with obesity.
- Note: Babies have bigger heads and smaller legs thus the "rule of 9s" for them assigns two 9s to the head, and both legs share a total of three 9's instead of four. In determining what is third-degree we should remember that in babies those areas look deep bright red (rather than the leathery dry, gray appearance in adults)

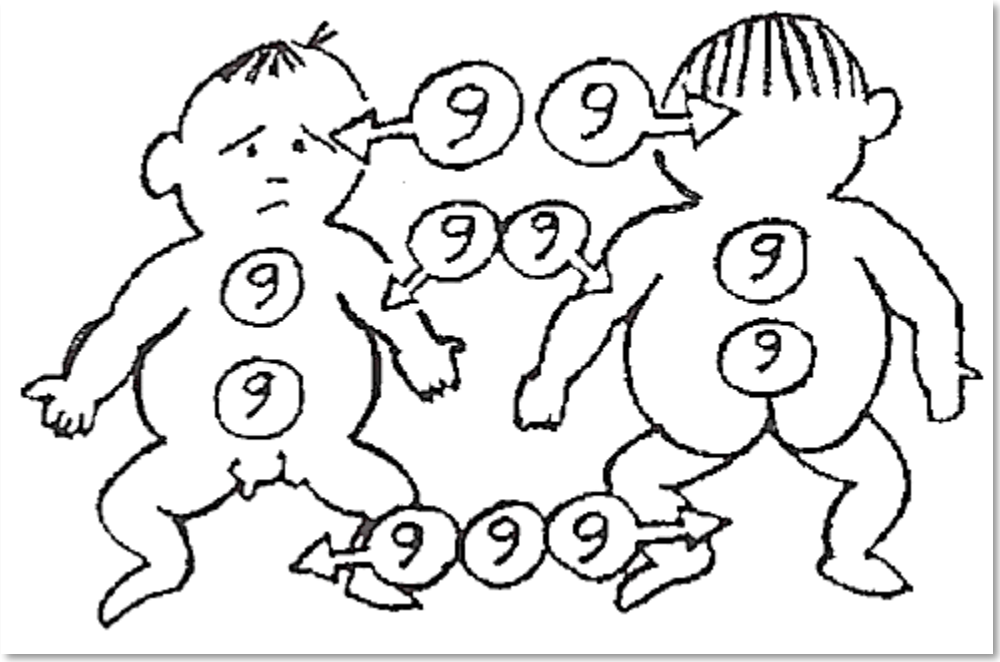


Figure 55: Wallace Rule of 9s in babies

RULE OF THE PALM

- The patient's entire palm area (i.e. not the fingers) is 1%. (0.8% in males and 0.6% in females of the total body surface area)
- The rule of palms can be used to estimate relatively small burns (<15%) or very large burns (>85% when unburnt skin is counted). For large burns it is inaccurate.
- A clean piece of paper is cut to the size of hand and through that the percentage of burns is assessed.

LUND AND BROWDER CHART

- This is a better method for assessing the burn wounds. Here each part of the body is individually assessed for involvement of burns.

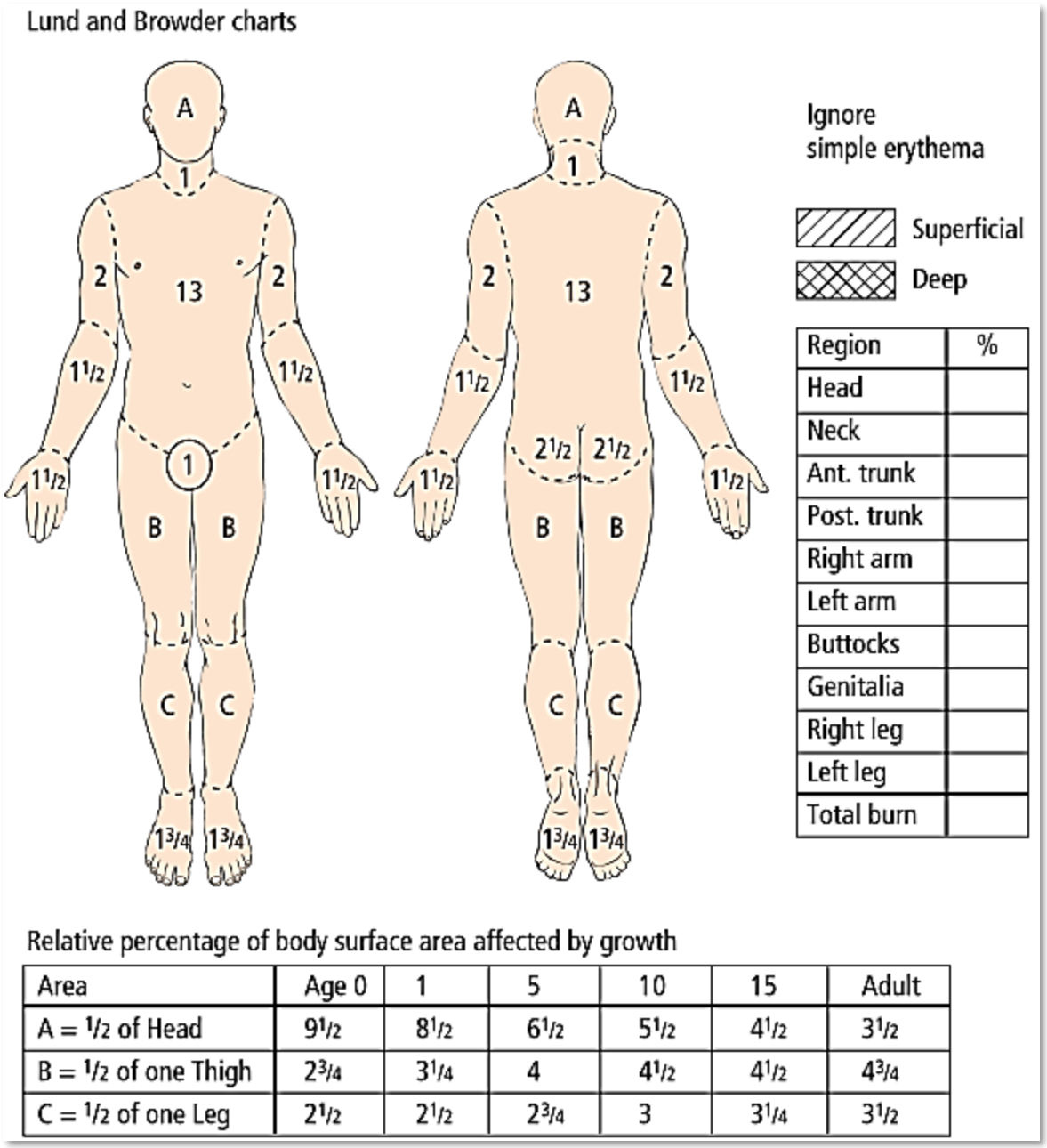


Figure 56: Lund and Browder Chart

- Note: if used correctly, this is the most accurate method of assessing burn area.

- Note: Unlike the Wallace rule of nines, the Lund and Browder chart takes into consideration the age of the person with decreasing percentage body surface area for head and increasing percentage body surface area for the legs as the child ages, making it more useful in pediatric burns.
- NOTE: 44^{O}C temperature takes 6 hours to cause deep burns. 65^{O}C takes 45-60 seconds to cause deep full thickness burn.
- When you are on call, and a patient comes with burns, the 3 things you should tell the consultant on the phone about the patient:
  - The % estimate of the burns: this helps with management of the patient (especially on admission criteria).
  - Cause of the burn- this helps tell the depth of the burns
    - Domestic burns are usually superficial.
    - Industrial burns are usually deep.
  - Time of burn: this will help with fluid replacement therapy.

DIFFERENTIAL DIAGNOSIS

- Necrotizing fasciitis

PATIENT ASSESSMENT

HISTORY

- Time and mechanism of the injury:
  - Type of burn i.e. scald, flame, electrical, chemical
    - Scald injuries
      - What was the liquid?
      - If tea/coffee, was there milk in it?
      - Was there a solute in the liquid? This will raise the boiling temperature and result in a worse injury.
    - Electrocution injuries
      - What voltage?
      - Was there a flash/arcing?
      - Contact time
    - Chemical injuries: what chemical?
  - How was the person put out? How long were they alight for?
  - What first aid was carried out? If cooling was performed, what with and for how long?
- Likelihood of concomitant injuries (i.e. fall from height, road traffic accident, explosion).

- Likelihood of inhalational injury: did the burn occur in an enclosed space?
- Any suspicion of non-accidental injury?

INVESTIGATIONS

- Blood investigations:
  - Full blood count: to pick up any cytopenias
    - Hematocrit
    - Hemoglobin
    - Platelet count
    - Differential count
  - Group and save/cross-match
  - Urea, Electrolytes and creatinine: to assess renal function and electrolyte imbalances
  - Liver function tests
- Pus swab
- For electrical burns:
  - 12 lead ECG
  - Cardiac enzymes (high-tension injury)
- For inhalation burns: chest X-ray and Arterial blood gases

MANAGEMENT

FIRST AID

- Stop the burning process and keep the patient away from the burning area.
- Cool the area with tap water by continuous irrigation for 20 minutes (not cold water as it can cause hypothermia).
- Do the ABCs.

DEFINITIVE TREATMENT

INITIAL

- Admit the patient
  - Admission criteria:
    - Any moderate and severe burns.
    - Airway burns of any type. Inhalational injury
    - Burns in extremes of age.
    - All electrical/deep chemical burns.
    - All burns affecting joints, face, perineum and all joints with fractures or multiple injuries.

- Burns likely to require fluid resuscitation and surgery.
- Patients whose psychiatric or social background
- Suspicion of non-accidental injury

- Maintain airway, breathing, circulation. (ABCs)
  - Tracheostomy/ endotracheal intubation tube may be required in impending respiratory failure or upper airway block. The airway should be secure until the swelling has subsided which is usually after 48 hours.
    - Symptoms of laryngeal edema: voice change, stridor, anxiety and respiratory difficulty are very late symptoms. Intubation at this point is often difficult or impossible owing to swelling, so acute cricothyroidotomy equipment must be at hand when intubating patients with a delayed diagnosis of airway burn.
    - The time frame from burn to airway occlusion is usually 4 and 24 hours.
  - Treat carbon monoxide poisoning with high inspired 100% oxygen for 24 hours.
- Catheterize Patient- monitor urine output (Normal is 0.5-1ml/kg/hr in adults and 1ml/kg/hr in children)
- Assess the percentage, degree and type of burn.
- Keep the patient in a clean warm and isolated environment.
- Sedation and proper analgesia.
- Labs:
  - Culture of the discharge
  - FBC at regular intervals
- Fluid resuscitation (discussed later)
- Monitoring the patient vitals hourly:
  - Pulse
  - Blood pressure
  - Respiratory rate
  - Blood gasses: PO_2, PCO_2
- Monitor:
  - Electrolyte analysis
  - Blood urea
- Give Nasal oxygen
- Patient should be in burns unit (ideally air conditioned) with barrier nursing, sterile clothes, bed sheets with all aseptic methods.

INTERMEDIATE

- Daily wound cleaning:
- Principles of dressing for burns:
  - Full-thickness and deep dermal burns need antibacterial dressings to delay colonization prior to surgery
  - Superficial burns will heal and need simple dressings
  - An optimal healing environment can make a difference to outcome in borderline depth burns.
- Methods for wound care include:
  - Open methods:
    - Application of silver sulfadiazine (Flamazine cream) without any dressing, used commonly in burns of face, head and neck.
    - For patients allergic to Sulphur drugs use Povidone cream or Chloherxidine cream
  - Closed methods:
    - Dressings done to soothe and protect the wound, to reduce pain and act as an absorbent.
    - Done for up to 10 days
  - Mixed: clean and apply wet soaks
- Sloughectomy/ Escharotomy if indicated should be performed.

LONG TERM

- Monitoring:
  - Mental status
  - Heart rate- rapid rate may also indicate infection or severe dehydration
  - Pulse rate
  - Edema
  - Temperature- spikes may indicate infection
  - Fluid replacement
  - Urine output (0.5-1ml/kg/hr for adults and 1ml/kg/hr for children)
- Check for proper wound healing: healthy granulation tissue (5Ps of granulation tissue- Pink, Punctate hemorrhages, Pulsating, Painless, Pinhead granulation)
- Nutritional status:
  - Check weight and Skin fold thickness
  - High protein diet, total parenteral nutrition

- Drugs:
  - Tetanus toxoid should be given. (0.5mls IM-single dose)
  - IV ranitidine 50 mg TDS.
  - Antibiotics: penicillins, aminoglycosides, cephalosporins, metronidazole.
  - Painkillers: IV morphine

FLUID RESUSCITATION

- Fluid needs in the first 48 hours after burn can be estimated by calculations that take into account the extent of the burn and provide either an estimated total amount needed or more simply an appropriate initial rate of infusion.
- For children, any burn which is 10% (15% adults) or above is severe and fluid has to be given IV otherwise give fluids orally in each case.
- There are 2 regimes of fluid:
  - Fluid replacement: this is given to replace the loss due to burn. Most of the fluid leaves the circulation and is in the interstitial space.
  - Maintenance fluid: this is the daily requirement due to insensible losses through sweat, breathe etc.
- Fluid resuscitation:
  - In children with burns over 10% TBSA and adults with burns over 15% TBSA, consider the need for intravenous fluid resuscitation
  - If oral fluids are to be used, salt must be added.
  - Fluids needed can be calculated from a standard formula
  - The key is to monitor urine output

FLUID AND COLLOID REPLACEMENT

- Formulas to calculate the fluid replacement:
  - Parkland regime (Baxter) this is commonly used (for crystalloids)

$$V = 4ml \times mass\ (kg) \times \%burn$$

    - The parkland formula is used to determine the amount of replacement fluid required for the first 24 hours of a burn patient.
    - Maximum percentage considered is 50%.
    - Half the volume is given in first 8 hours and the rest given in 16 hours.
    - When considering time use "time the patient was burned" not time of admission.

Expected weight for children:

For infants (<12 months) = $\frac{Age\ in\ months+9}{2}$

For children 1-5= 2 x (age in years +5)

For children 5-14= 4 x age in years

- Fluid of choice is Ringer's lactate.
- An additional 2, 000 ml of 5% dextrose in water (D5W) are usually added on the first day for the fact that the patient is typically NPO, with a nasogastric (NG) tube in place. (Colloids should not be given on day 1 as they would leak out into the edema fluid).
- On day two, half of what was given on day 1 should be given, it may include colloids if so desired though this has some controversies.
- By day 3 there should be no need for further fluid infusion and a brisk diuresis should be expected.
- The actual amount of fluid infused is eventually provided by "fine-tuning" based on the patient's response, aiming for a urine output of 1-2ml/kg/hr.

➢ Muir and Burclay formula (used for colloids)

$$\frac{\%\ Burns\ \times body\ weight\ in\ Kg}{2} = 1\ ration$$

- 3 Rations given in first 12 hours (give 4 hourly)
- 2 rations in second 12 hours (give 6 hourly)
- 1 ration in third 12 hours

- Fluids used are normal saline, ringer lactate (Hartmann fluid), plasma.
- Ringer lactate is the fluid of choice.
- Blood is transfused in later period (after 48 hours) if indicated but recently not advisable because of increased permeability so there is an increased chance of concentrating blood and making it more viscus.
- In the first 24 hours only crystalloids should be given (crystalloids can pass through capillary wall like saline either hypo, iso or hypertronic, dextrose saline, Ringer lactate).
  - ➢ Sodium is assessed by formula: 0.52mmol x kg body weight x % burns
  - ➢ Given at a rate of 4.0 to 4.4ml/kg/hour
- After 24 hours up to 30-48 hours, colloids should be given to compensate plasma loss (colloids are one which are retained in intravascular compartment).
- Plasma, haemaccel (gelatin), dextrans, hetastarch are used. Usually at a rate of 0.35-0.5ml/kg/% burns are used in 24 hours.

MAINTENANCE FLUID IN 24 HOURS

- In 24 hours Give:
  - ➢ 100ml/kg for the first 10kg
  - ➢ 50ml/kg for the next 10kg

- 20ml/kg for 24 hours for each kilogram over 20kg body weight
- 5% dextrose (Dextrose-saline) is used for maintenance

- For example, a child weighing 28kg maintenance fluid is calculated as follows:
  - First 10kg give 10kg x 100ml/kg= 1000ml
  - Next 10kg give 10kg x 50ml/kg= 500ml
  - Remaining 8kg give 8kg x 20ml/kg= 160ml
  - Give a total of 1660ml in 24hours of 5% dextrose IV

OTHER CARE

- Clean the burns thoroughly with soap (lifebuoy) and water, preferably running water (shower is preferred to tab).
- Slough removed by wet dressings. If thick slough is present in deep burns, surgical sloughectomy is recommended. Eschar requires Escharotomy.
- Specific care for burns involves the eyes, mouth, perineum and joints.
- Burns near the eyes are covered with triple antibiotic ointment (Silver sulfadiazine is irritating to the eyes).
- In the early period, all pain medication is given intravenously.
- Keep patient warm to avoid hypothermia
- Patient might need blood transfusion
- Physiotherapy of burns involving joints. Rehabilitation starts on day one.
- Nutritional support- high-calorie/high nitrogen diets, Vitamins.
- After 2 or 3 weeks of wound care and general support, the burned areas that have not regenerated are grafted.
  - The concept of early excision and grafting is used whenever possible to save costs and minimize pain, suffering and complications.
  - It implies removal in the or (on day 1) of the burned areas with immediate skin grafting.
  - This can be done only for fairly limited burns (under 20%) that are obviously third-degree, but the experts are extending the concept to highly selected patients with more extensive burns.
  - Deep dermal burns need tangential shaving and split skin grafting
  - All but the smallest full-thickness burns need surgery

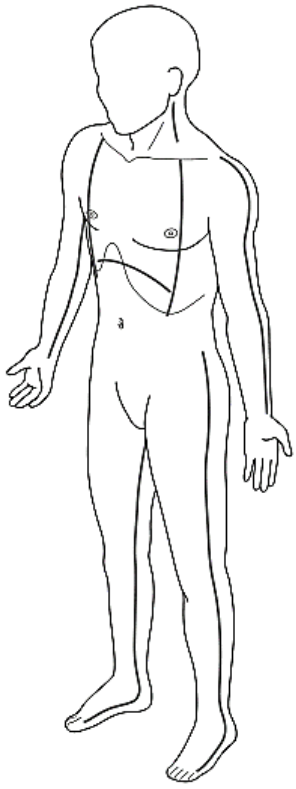


Figure 57: Incisions for Escharotomy.

- All burnt tissue needs to be excised

- Types of skin grafts:
  - Skin Autografts: burned area is covered with the patient's own skin. This is done in burn wounds less than 50% of total body surface area. Split skin grafts are taken from unburnt areas and are usually meshed (i.e. have slit perforations placed in them). This helps any exudate beneath the graft to ooze out and also allows the graft to be stretched to cover a larger area. Meshed skin grafts heal with the mesh pattern visible so tend not to be used on the face. The grafts are secured with staples or tissue adhesive and then dressed. It is important to prevent shearing forces from disrupting the graft so joints may be temporarily splinted.
  - Skin Allografts: cadaveric skin can be used as temporary biological dressing. The storage solution decreases the antigenicity of the skin and the burn patient is relatively immunosuppressed so the graft is not rejected for a few weeks. This may allow autograft donor sites time to heal for regrafting.
  - Skin Xenografts: porcine skin can also be used as a temporary biological dressing.



Figure 58: Skin grafts

- Prognosis
  - Percentage mortality= Age of patient + % burn
  - Inhalational injury increases mortality but it is arguable by how much.
  - A decision should be made about whether resuscitation should be started or whether the patient should just be kept comfortable.

COMPLICATIONS OF BURNS

EARLY

- Respiratory system:
  - Airway obstruction: due to pulmonary edema, ARDS, respiratory arrest and respiratory failure
- Cardiovascular system:

- Edema: due to release of inflammatory mediators to produce vasoconstriction and vasodilation leading to increased capillary permeability.
- Hypovolemia: due to fluid loss as a result of increased capillary permeability due to SIRS and this may lead to renal failure.
- Hypoxia: due to destruction of RBCs because thermal injury causes coagulative necrosis to the epidermis and underlying tissue.
- Shock: due to SIRS or due to an inflammatory response causing release of inflammatory mediators like cytokines which leads to vasodilatation in burnt areas.
- Toxic shock: bacterial infection and bacteremia.

- Skin
  - Hypothermia (loss of thermoregulation in skin due to damage)
    - Warm fluids that are being given to burns patient.
- Genitourinary system
  - Electrolyte imbalance and shock
- Metabolic
  - Metabolic Acidosis
  - Hypoglycemia- hyper-catabolism & mobilization of glucose as a result of stress hormones

INTERMEDIATE

- Wound infection (*Staph aureus*, *Beta hemolytic Strep*, *Pseudomonas*, *Klebsiella*)
- Septicemia
- Septic arthritis
- Stress ulcers (curling ulcer- hypovolemia, ischemia of mucosa, erosive gastritis)
- Paralytic ileus due to electrolyte imbalance
- Compartment syndrome due to eschar (slough produced)
- Hypo-proteinemia
- DVT

LATE

- Contractures: due to fibrosis
  - May lead to ectropion, microstomia, disability of different joints, defective hand functions, growth retardation causing shortening.
- Hypertrophic scar or keloids
- Marjolin's ulcer (malignant)
- Nerve compression

- Psychological effects-cosmetic effect

STOMAS

STOMAS

- A stoma is an iatrogenic hollow muco-cutaneous fistula used for enteral feeding, drainage, or to divert feces and flatus.

CLASSIFICATION OF STOMAS

- They are classified as
  - Temporary
  - Permanent

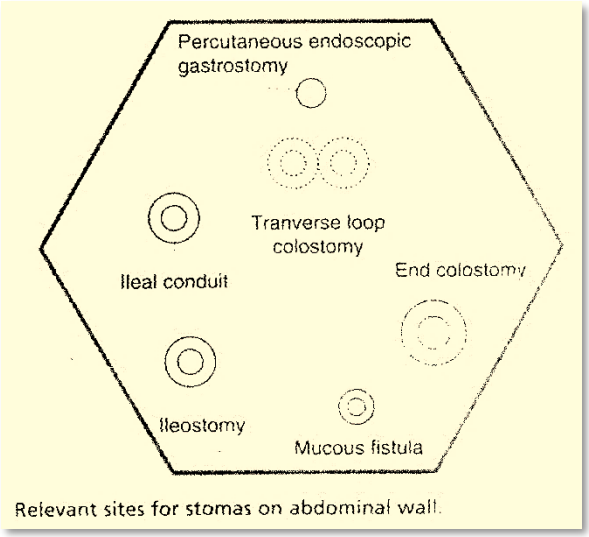


Figure 59: Stomas

TEMPORARY

- Input stomas include:
  - Gastrostomy
  - Jejunostomy
- Output stomas include:
  - Pharyngostomy
  - Esophagostomy
  - Caecostomy
  - Ileostomy
  - Colostomy

PERMANENT

- End-ileostomy
- End-colostomy

TEMPORARY INPUT STOMAS

GASTROSTOMY

- This is an opening between the skin and the stomach.
- Indicated usually for feeding following upper GIT surgery, or in caustic (NaOH) stricture of esophagus before surgery.
- Not indicated for advanced carcinoma of the esophagus.
- May be done as an open procedure or endoscopically.

JEJUNOSTOMY

- Opening between jejunum and skin.

- Indicated following surgery of the upper GIT especially stomach. This is to allow surgical wounds to heal while facilitating enteral feeding.
- A small feeding tube is inserted to allow feeds to pass through.
- Feeds should be specialized.

TEMPORARY OUTPUT STOMAS

PHARNGOSTOMY

- Used for diversion of solid and saliva to protect the bronchial tree in neonates with esophageal atresia or tracheal esophageal fistula (TOF).
- May be used as a stage operation in esophageal replacement surgery.
- It involves diverting the pharynx to the skin.
- Technically difficult operation because of the structures found in the neck.

ESOPHAGOSTOMY

- Used for diversion of solid and saliva to protect the bronchial tree in neonates with esophageal atresia or TOF.
- May be used as a stage operation in esophageal replacement surgery.
- It usually involves diverting the middle esophagus to the skin.
- Care should be taken not to damage the neck structures (nerves and vessels).

CAECOSTOMY

- Stoma between the skin and caecum.
- Used for decompression purposes of the colon following colonic surgery especially anastomosis of large bowel where the integrity of the anastomosis is not guaranteed.

ILEOSTOMY

- Diversion at the level of the ileum
- Effluent mostly fluid feces, water and electrolytes.
- These are high output and may cause electrolyte imbalances.
- May complicate into skin excoriation
- May also complicate into gallstone formation

COLOSTOMY

- This is a mucocutaneous opening between the colon and the skin.

TYPES

- They can either be temporary or permanent.

TEMPORARY

- Done in conditions wherein diversion is required to facilitate healing distally in the rectum or distal colon.
- This is closed once the purpose is over.
- Site of temporary colostomy is usually right hypochondrium and left iliac fossa.

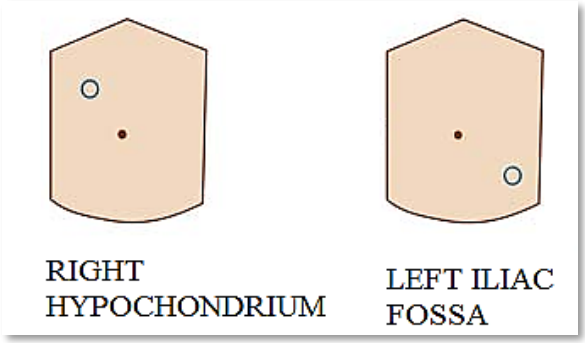
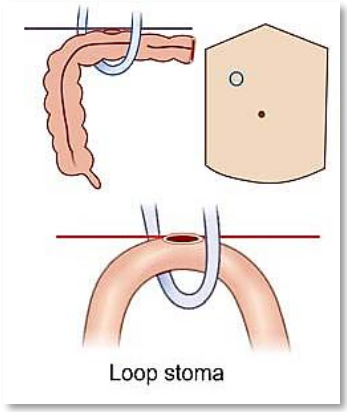


Figure 60: Sites of a colostomy

- Temporal colostomies can be
  - Loop colostomy: usually done on the transverse colon



  - Devine's double barrel colostomy: there is a gap between the 2 openings of the colostomy which prevents spillage into the loop
  - Spectacle
  - Hartmann

PERMANENT

- Permanent colostomy is always end colostomy placed in left iliac fossa, 6cm above and medial to the anterior superior iliac spine.

INDICATIONS

- Congenital
  - Congenital megacolon
  - Anorectal malformations
  - Imperforate anus
  - Hirschsprung disease
  - Anal atresia
  - Anorectal agenesis
  - Anal stenosis
- Acquired
  - Intestinal obstruction e.g. Sigmoid volvulus
  - Ischemic bowel disease/ gangrene due to strangulation
  - Penetrating trauma to the bowel (left sided)
  - Tumors: Anal Ca and Rectal Ca
  - Perforation of left sided colon
  - High anal fistula
  - After Hartmann's operation

COMPLICATIONS

- Complications can always be divided into specific to the procedure vs general for any surgical procedure and also into immediate (<24 hours), early (1 month) and later (>1 month).
- Specific:
  - Ischemic gangrene
  - Hemorrhage
  - Retraction
  - Prolapse/ intussusception
  - Parastomal hernia
  - Stenosis- leads to constipation
  - Skin excoriation
- General complications- related to underlying disease:
  - Stoma diarrhea- related to water and electrolyte imbalance, hypokalemia being the commonest and most important consequence and metabolic acidosis (due to loss of bicarbonate)
  - Nutritional disorders
  - Stones- gallstones and renal stones increase in frequency following an ileostomy.

- Psychosexual- leading to impotence in men/ loss of libido
- Residual disease e.g. Crohn's and Parastomal fistula

	Ileostomy	Colostomy
Site	Right iliac fossa	Left iliac fossa
Surface	Sprout (contents are corrosive and can damage local skin)	Flush with skin
Contents	Watery- small bowel contents	Feculent
Examples of permanent stomas	Post panprocto-colectomy	Abdomino-peroneal resection of rectum (End colostomy)
Examples of loop temporary stomas resection	Ileostomy over low anastomosis of anterior abdomen	Hartmann's procedure

PREOPERATION PREPARATION

- Psychosocial and physical preparation.
- Explanation of the indications and complications.
- Marking of the site- with the patient standing up as he or she must be able to see the stoma.
  - 5cm from the umbilicus
  - Away from scars/ skin creases
  - Away from bony points
  - At a site easily accessible to the patient
- The stoma must be within the rectus sheath.

PRINCIPLES OF SITING A STOMA

- It should be sited pre-operatively ideally.
- Should be in the most favorable position for the patient:
  - Away from bony prominences
  - Should be in a position easy for the patient to change
- An end colostomy is usually sited in the left iliac fossa.
- A transverse loop colostomy usually in the upper quadrants of the abdomen.
- Bowel mucus is usually flush with skin in the colostomy while an ileostomy usually has a sprout.

EXAMINATION OF A STOMA

- Greet the patient.
- Explain the examination and gain consent.
- Expose the patient.

INSPECTION

- Do not start at the hands as you have a specific direction to inspect the abdomen.
- Start by commenting that you seeing a stoma in Epigastric or right iliac or left iliac fossa.
  - Is it covered by a bag and in the bag there is?
    - Fecal materials
      - Formed stool or semi-formed or liquid stool.
      - Watery or urine
  - It is not covered by the bag?
    - The mucosal lining looks healthy/ unhealthy
    - There is a sprout or flush and skin
    - It has one opening (end) or loop (an afferent and efferent portion of bowel with common opening or 2 separate openings)
  - Comment on the rest of the abdomen shows:
    - A scar
    - Midline transverse incision
    - Wound dehiscence or and mass
    - Also other drains or healed
    - Stoma scar

PALPATION

- If you are only asked to inspect, then do not touch the patient at any time, even to move the stoma bag out of the way- there may be a good reason why you should not touch it.

COMPLETION

- Tell the examiner that you would like to examine the rest of the abdomen to look for reasons why the stoma may have been formed in the first place.
- Thank the patient and cover them.

REHABILITATION OF A PATIENT FOLLOWING THE PLACEMENT OF A STOMA

- Diet should be normal.
- Bag should be changed once or twice a day (needs to be emptied more frequently than this if it is urine or fluid faeces)
- Ileostomies should have the base plate under the bag changed every 5 days and the bag changed daily.
- Psychological and psychosexual support.

PREOPERATIVE AND POST-OPERATIVE MANAGEMENT

PAIN AND PAIN RELIEF

NEOPLASIA

GENERAL TERMS

- Aplasia- is an almost complete lack of development of a part, so that proper form and structure are not acquired.
- Hypoplasia- is a lack of development of a part to full or mature.
- Hyperplasia- an increase in the number of cells of a part. It merges with the process of neoplasia (tumor formation).
  - It may occur in response to chronic irritation (as in lymphoid tissue) or endocrine stimulation or it may be compensatory.
  - The liver has great powers of compensatory hyperplasia.
  - The remaining thyroid tissue may become hyperplastic when part of it is removed.
  - The bone marrow is hyperplastic when there is increased demand for blood.
  - The prostate often becomes hyperplastic in men over 45, probably due to a relative hyperestrinism.
- Hypertrophy- this is increase in the size of individual cells or fibers so that an organ is enlarged. There is no increase in the number of cells or fibers.
  - True hypertrophy occurs in response to some demand for increased function.
  - Physiological hypertrophy occurs apart from disease e.g. pregnant uterus, muscles of athletes and laborers.
  - Adaptive hypertrophy occurs in hollow viscera when the outlet is partially obstructed, the wall becoming thickened from enlargement of muscle fibers e.g. left ventricle in aortic stenosis, the stomach in pyloric stenosis, the intestine in chronic obstruction and the urinary bladder in urethral obstruction.
  - Compensatory hypertrophy is an increase in the size to make up for a loss of tissue especially in paired organs such as the kidney.
- Atrophy: is an acquired decrease in size of a portion of the body, an organ, tissue or individual cells.
  - The term is applied only when full size was once present, so that atrophy must be distinguished from hypoplasia, aplasia and agenesis.
  - In atrophy the reduction in size in part may be due to a decrease in the number of its structural units or in the size of the individual units or both.

STINGS AND BITES

SNAKE BITE

- Snakes belong to *Crotalidae*, *Elapidae* and *Viperidae* family.
- Common poisonous snakes are cobra, krait and viper.
- Common snakes are cobra, Russell's viper, saw scaled viper, pit viper, krait.
- Snake bites can cause neurological problems, cardiac complications, pulmonary edema and renal failure.
- Snake bites do not necessarily result in envenomation, even if the snake is poisonous (up to 30% of bitten patients are not envenomed).

CLINICAL FEATURES

- The most reliable signs of envenomation are severe local pain, swelling and discoloration developing within 30 minutes of the bite.

PATHOPHYSIOLOGY

- Venom is produced and stored in paired glands below the eye. It is discharged from hollow fangs located in the upper jaw. Fangs can grow to 20mm in large rattlesnakes.
- Venom dosage per bite depends on the elapsed time since the last bite,



Figure 61: Coral snake

the degree of threat the snake feels threatened and the size of the prey.

- The nostril pits respond to the heat emission of the prey, which may enable the snake to vary the amount of venom delivered.
- Venom is mostly water.
- Enzymatic proteins in venom impart its destructive properties.
  - Proteases, collagenase and arginine ester hydrolase have been identified in pit viper venom.
  - Neurotoxins comprise the majority of coral snake venom.
  - Hyaluronidase allows rapid spread of venom through subcutaneous tissue by disrupting mucopolysaccharides.
  - Phospholipase A2 plays a major role in hemolysis secondary to the esterolytic effect on red cell

membranes and promotes muscle necrosis.

- Thrombogenic enzymes promote the formation of a weak fibrin clot, which in turn, activates plasmin and results in a consumptive coagulopathy and its hemorrhagic consequences.
- Protease 1-amino acid oxidases: tissue necrosis.

- Enzyme concentrations vary among species, thereby causing dissimilar envenomation.
- Copperhead bites generally are limited to local tissue destruction. Rattlesnakes can leave impressive wounds and cause systemic toxicity.
- Coral snakes may leave small wounds that later result in respiratory failure from the typical systemic neuromuscular blockade.

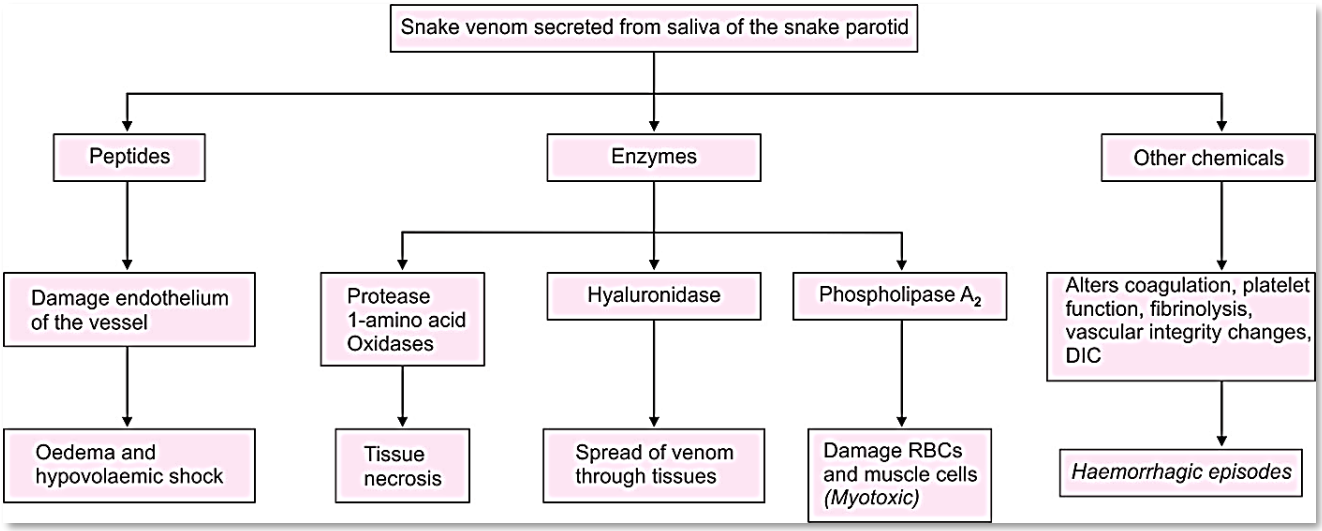


Figure 62: Effects of snake venom

LOCAL EFFECTS

- Burning pain, edema and erythema
- Swelling, ecchymoses and hemorrhagic bullae.
- Local bleeding.
- Tissue necrosis, ulceration and gangrene

SYSTEMIC

- Neurological manifestations
- Weakness, perioral paresthesia, muscle twitching
- Shock
- Bleeding tendency
- Pulmonary edema
- Renal failure
- Vipers affect multiple organs and soft tissues. Cobra and coral snakes are neurotoxic.

HISTORY

- History usually can be obtained from the patient.
- Most cases result from attempting to handle snakes, so the genus usually is known.
- Knowledge of indigenous fauna also is important.
- The time elapsed since the bite is a necessary component of the history.
- This allows assessment of the temporal effects of the bite to determine if the process is confined locally or if systemic signs have developed.
  - Obtain a description of the snake or capture it, if possible, to determine its color, pattern, or the existence of a rattle.
  - Most snakes remain within 20 feet after biting.
  - Assess the timing of events and onset of symptoms. Inquire about the time the bite occurred and details about the onset of pain. Early and intense pain implies significant envenomation.
  - Systemic symptoms include nausea, syncope and difficult swallowing or breathing.
- Determine history of prior exposure to antivenin or snakebite.
- Determine history of allergies to medicine because antibiotic may be required.
- Determine history of comorbid conditions (e.g. cardiac pulmonary, and renal disease) or medications (e.g. aspirin, beta-blockers)

PHYSICAL EXAMINATION

- Follow the established routine for a complete comprehensive examination.
  - Vital signs, airway, breathing, circulation.
  - Fang marks or scratches (determine coral snake bite pattern by expressing blood from the suspected wound)

INVESTIGATIONS

- Full Blood count
- Blood Cross match
- Coagulation studies like bleeding time, clotting time and prothrombin time.
- Blood urea and serum creatinine.
- Serum electrolytes
- Urine analysis for RBCs, albumin, myoglobin
- Liver function tests
- Creatinine phosphokinase [CPK]
- Arterial blood gasses: PO_2 and PCO_2 assessment

MANAGEMENT

FIRST AID

- Reassurance of the pain, ensure the patient is calm to preclude hysteria.
- Immobilization of the patient, keep walking to a minimum.
- Implement the ABCs.
- Monitor vital signs and establish at least 1 large bore intravenous and crystalloid infusion.
- Administer oxygen therapy. Keep a close watch on the airway at all times in case intubation becomes necessary.
- Negative-pressure suctioning devices offer some benefit if used within several minutes of envenomation. Again do not make an incision or give antivenin in the field.
  - ➢ All the first aid measures that you learned at boy scouts are wrong. Do not make cruciate cuts, suck out venom, wrap with ice or apply a tourniquet.
- Immediately transfer to definitive care.

DEFINITIVE TREATMENT

- Definitive treatment includes reviewing the ABCs and evaluating the patient for signs of shock (e.g. tachypnea, tachycardia, dry pale skin, mental status changes, hypotension).
- The site has to be incised and cleaned.
- It is thoroughly cleaned with debridement.
- Fasciotomy is not indicated in every bite, only for those patients with objective evidence of elevated compartment pressure (compartment syndrome)
- Tourniquets are used to occlude lymphatics only but not venous or arterial circulation.
- The snake has to be identified.
- The patient must be transferred to proper medical center as early as possible.
- The bite wound is identified and assessed.
- Envenomation grading determines the need for antivenin in pit viper victims. Grades are defined as mild, moderate or severe.

- Mild envenomation: local pain, edema, no signs of systemic toxicity and normal lab values.
- Moderate envenomation: severe local pain, edema larger than 30cm surrounding the wound and systemic toxicity including nausea, vomiting and alteration in lab values (e.g. fallen hematocrit or platelet value).
- Severe envenomation: generalized petechiae, ecchymosis, blood-tinged sputum, hypotension, hypo-perfusion, renal dysfunction, changes in prothrombin time and activated partial thromboplastin time, and other abnormal tests defining consumptive coagulopathy.

- Grading envenomation is a dynamic process. Over several hours, an initially mild syndrome may progress to a moderate or even severe reaction.
- Polyvalent anti-snake venom (against cobra, krait, vipers) should be given.
  - It should be given earliest within 4-24 hours.
  - Dose is 20-150ml depending on the type, severity and age of the individual. Children get the same dosages as adults.
  - It is dissolved in normal saline and given as IV infusion in 500ml saline with 20 drops/minute as flow rate. If the snake has been identified monovalent serum is better and more potent.
  - Preferred agent for crotalids is CROFAD of which several vials are usually needed
- Tetanus prophylaxis
- IV fluids, blood transfusion and plasma
- Antibiotics (ceftriaxone- third generation cephalosporin broad spectrum antibiotic more effective on gram negative)
- Urine output measurement
- Monitoring by regular checking of blood urea, serum creatinine and bleeding and clotting time.
- In cobra bite, there is neuromuscular blockade and paralysis. So neostigmine should be given 0.5 mg IV every half hourly and later repeated. It is given along with 0.6mg of atropine.
- In viper bite, DIC is common. So heparin is given as 10, 000 to 15, 000 units loading dose and later 5, 000 units as maintenance dose TDS.
- Human fibrinogen, whenever required.

COMPLICATIONS

- Local wound complications: infection, skin loss, Cellulitis and gangrene of the part.

- Compartment syndrome especially with pit viper snake bites.
- Neurological complications
- Cardiovascular and Hematological: Deep venous thrombosis (DVT), Pancytopenia, DIC and hemorrhage
- Pulmonary collapse
- Septicemia
- Renal failure
- Marjolin's ulcer
- Antivenin-associated complications: anaphylaxis (Type I) and serum sickness (Type III).

RABIES AND DOG BITES

RABIES

- Rabies is a major problem in some countries. It is an acute fatal encephalomyelitis.
- There is no predilection for age or sex even though it is observed more in children and adult males.
- Established infection is almost invariably fatal. It is caused by a genotype 1 single-stranded RNA virus of the Lyssavirus genus.
- The rabies virus is bullet-shaped and has spike-like structures arising from its surface containing glycoproteins that cause the host to produce neutralizing, hemagglutination-inhibiting antibodies.
- The virus has a marked affinity for nervous tissue and the salivary glands.
- 2 major epidemiological types of rabies exist:
  - Urban rabies (95%): most frequently transmitted to humans through rabid dogs, and less frequently cats.
  - Sylvan (wild) rabies: this is maintained in the wild by a host of animal reservoirs such as foxes, skunks, jackals, mongooses and bats.
- Transmission is usually through the bite of an infected animal. However, the percentage of rabid bites leading to clinical disease ranges from 10% (on the legs) to 80% (on the head). Other forms of transmission if they occur are rare.
- The virus replicates in the muscle cells near the entry wound. It penetrates the nerve endings and travels in the axoplasm to the spinal cord and the brain.
- In the CNS the virus again proliferates before spreading to the salivary glands, lungs, kidneys and other organs via the autonomic nerves.
- In the brain it causes encephalitis and in the spinal cord it causes transverse myelitis.
- Natural occurring rabies virus is called as street virus which shows

long incubation period of 20-60 days. Serial passage of the virus to brain of rabbits creates fixed virus which has got a short incubation period of 4-6 days which does not show Negri bodies. This fixed virus which cannot multiply in extraneural tissues is inactivated to prepare vaccine.

CLINICAL FEATURES

- Incubation period is variable and ranges from few weeks to several years (average 1-3 months).
- Bites on the head, face and neck have a shorter incubation period than those elsewhere.
- In humans, 2 distinct clinical varieties are recognized:
  - Classic variety (Furious rabies): the dogs are aggressive like a mad dog. Dog changes its behavior with loss of fear of humans, bites objects, eat, mud etc. running amok, voice change, inability to bark properly, excessive salivation and foaming paralysis and death. In humans:
    - Prodromal period only shows pain and tingling at the site of the initial wound.
    - 10 days later, marked anxiety and agitation or depressive features develop.
    - Hallucinations, bizzare behavior, increased salivation and paralysis may occur.
    - Hyperexcitability (Hallmark of this form of rabies) is precipitated by auditory or visual stimuli.
    - Hydrophobia (fear of water) is present in 50% of patients and is due to severe pharyngeal spasms on attempting to eat or drink.
    - Aerophobia (Fear of air) is considered pathognomonic of rabies.
    - Examination reveals hyperreflexia, spasticity and evidence of sympathetic overactivity indicated by pupillary dilation and diaphoresis.
    - The patient goes on to develop convulsions, respiratory paralysis and cardiac arrhythmias.
    - Death usually occurs in 10-14 days.
  - Paralytic variety (Dumb rabies): Dog is paralytic and sleepy. There is no aggressiveness at all. Dog sleeps for 3 days and dies. Once symptoms of rabies develop in a dog it rarely survives more than a week. In humans:
    - Presents with a symmetrical ascending paralysis

resembling the Guillain-Barre syndrome.
    - This variety of rabies commonly occurs after bites from rabid bats.

DIAGNOSIS

- Clinical
    - Class I: touching or feeding the diseased animal, lick over intact skin or scratches without oozing of blood
    - Class II: licks on broken skin, scratches with blood ooze and all bites except over head, face, palms and fingers. Minor wounds less than five in number
    - Class III: all bites over head, face, palms and fingers, lacerated wounds, wounds more than five in number, wild animal bites, and contamination of mucous membrane with saliva.
- Skin punch biopsy can be used to detect antigen with an immunofluorescent antibody test on frozen section.
- Viral RNA can be isolated using the reverse transcription polymerase chain reaction (RT-PCR).
- Isolation of viruses from saliva or the presence of antibodies in blood or CSF may establish the diagnosis.
- Corneal smear test is not recommended as it is unreliable. The classic Negri bodies are detected at post-morterm in 90% of all patients with rabies, these are esosinophilic, cytoplasmic, ovoid bodies, 2-10 nm in diameter, seen in greatest numbers in the neurons of the hippocampus and cerebellum.
- Diagnosis should be made pathologically on the biting animal using RT-PCR, immunofluorescence assay (IFA) or tissue culture of the brain

MANAGEMENT

- Once CNS disease is established, therapy is symptomatic and death is virtually inevitable.
    - Patient should be nursed in a quiet, darkened room.
    - Nutritional, respiratory and cardiovascular support may be necessary.
    - Drugs such as morphine, diazepam and chlorpromazine should be used liberally in patients who are excitable.
- Prevention
    - Pre-exposure prophylaxis: given to individuals at high risk of contracting rabies e.g. laboratory workers, animal handlers and veterinarians
        - 3 doses (1.0 ml) of human diploid (HDCV) or chick embryo cell vaccine given by deep subcutaneous or intramuscular injection on

days 0, 7 and 28 to provide effective immunity.
    - A reinforcing dose is given after 12 months and additional reinforcing doses are given every 3-5 years depending on the risk of exposure.
- Post-exposure prophylaxis:
    - Wound should be cleaned carefully and thoroughly with soap and water and left open.
    - Povidone-iodine or alcohol is then recommended to reduce the virus further.
    - Human rabies immunoglobulin should be given immediately (20 IU/Kg).
    - Half should be injected around the wound and the other half should be given intramuscularly.
    - People that have been previous immunized against rabies do not need to receive the immunoglobulin just the post-exposure vaccination at days 0 and 3.
    - Five 1.0 ml doses of HDCV should be given intramuscularly as follows on days 0, 3, 7, 14 and 28.
    - Injection should be given over the deltoid muscle as abdominal-based large needle method is being phased out as it is expensive.
    - Prophylaxis is effective when given within 10 days of exposure.
    - Reaction to the vaccine is uncommon.

- Domestic animals should be vaccinated if there is any risk of rabies in the country otherwise, they should be put down if they cannot be vaccinated.
    - BPL inactivated nervous tissue vaccine (20 % infected sheep brain suspension): single dose 5ml to dogs, 3 ml to cats. Second dose after 6 months. Then once a year regularly.
    - Modified live virus vaccine: 33% infected chick embryo suspension. Dose-3ml single dose which is repeated once in 3 years.
    - Oral vaccines: used successfully to control wild foxes in Canada by placing vaccine in food through baits.

DOG BITES

- For all patients presenting, a thorough history and clinical examination is a must.
- It is vital that serious or major injuries are excluded such as to the airway and cervical spine, major vessels with life or limb threat or

specialized structures especially of the head and neck.

- Assessment of these patients should be in accordance with the principles of Advanced Trauma Life Support (ATLS)-ABCs.
- Any penetrating bite injury can lead to considerable damage of both hard and soft tissues.
- History assessment:
  - Where and when the bite occurred.
  - How did the bite happen and (if a child) was there supervision of the child and/or animal?
  - Is there any associated relevant medical history: DM (increased risk of wound infection), Chronic liver or lung disease and Immunosuppressive states (HIV/AIDS, chemotherapy)- can reduce healing rate and increase risk of infection.
  - What is the patient's tetanus status?
  - History of alcohol dependence, smoking and/or substance abuse can also affect wound healing and infection risk and may have been a precipitant to the bite injury.
  - Consideration should also be given to current medications, nutritional states and extremes of age.
  - The timing of the injury to the time of presentation is also vital. Delay in presentation and treatment has been identified as a significant risk factor for a resulting wound infection.
- Examination: Local
  - Wound often comprises of tissue tears and lacerations, punctures, crush injuries with or without resulting tissue loss.
  - Consideration should be given to the presence of hard tissue, soft tissue or a combination of both.
  - Typical dog bite results in a 'hole and tear effect' with a combination of a deep puncture and tissue tearing.
  - Dog bite wounds at the 'minor end' of the scale of severity can be a simple abrasion or superficial penetration of the skin layers and these are the wounds likely to present to the GP for treatment.
  - Damage to muscle, vessels, nerves, bone or joints constitutes a serious injury and their management requires a thorough examination, assessment and will involve referring the patient to hospital to the relevant surgical specialty.

MANAGEMENT

- Wound must be thoroughly cleaned with saline.

- Irrigation and debridement is important in preventing subsequent infection and should be instigated at the earliest opportunity.
- Local anesthetic should be administered through uninvolved adjacent skin and away from the damaged tissue and if a distal periphery is involved, instructing the patient to elevate the affected limb can reduce subsequent tissue edema and swelling.
- Tetanus immunization as well as rabies vaccine plus rabies immunoglobins are given.
  - Anti-tetanus toxoid (0.5 ml)
  - 20IU/kg of Human rabies immunoglobulin. Half should be injected around the wound and the other half should be given intramuscularly
  - Five 1.0 ml doses of human diploid (HDCV) should be given intramuscularly as follows on days 0, 3, 7, 14 and 28.
- Puncture wounds are at higher risk of infection, as they harbor microorganisms deep within the wound, and have a narrow entry point making thorough cleaning difficult.
- Crush injuries can precipitate infections because of the resulting ischemic damaged tissue. This devitalized tissue predisposes to bacterial colonization and growth and also leads to infection.
- Closure of wound:
  - Primary closure: wound closure in the primary care setting is rarely recommended and therefore majority of injuries are left open to heal by secondary intention. The concern has always been that closure of the wound could lead to tissue necrosis, abscess formation and systemic sepsis.
  - In cases of minor wounds (superficial lacerations, abrasions) through cleaning and wound 'toilet' should be undertaken with irrigation. These wounds are deemed low risk. Exceptions are to those wounds deemed high risk cases for infection or clinically the wound appears infected.
- The use of prophylactic antibiotics in high risk wounds (location on the hand, deep puncture wounds or immunocompromised host) should be strongly considered, together with those cases presenting late (>9 hours since injury).
  - Antibiotic of choice is co-amoxiclav (amoxicillin and clavulanic acid)
  - For patients allergic to penicillin doxycycline combined with metronidazole is recommended.

COMPLICATIONS

- Infection: *Pasteurella spp*.
- Lymphangitis
- Local abscess
- Septic arthritis
- Tenosynovitis
- Osteomyelitis
- Bite wounds can be complicated by serous disseminated infection leading to septic shock, meningitis and brain abscess, endocarditis and other severe sequel including DIC (especially in immunocompromised)

MAMMALIAN BITE

- It also includes human bites also.
- Infection rate is more in mammalian bites.
- Human bites are bacteriologically the dirtiest bite one can get. They require extensive irrigation and debridement (in the OR).
- Proper wound toileting is very important.
- Within 12 hours, incised wound is closed primarily.
- All lacerated wounds and wound which is seen after 12 hours is left open. Wound is closed secondarily.
- Antibiotics are must in all mammalian bites.
- Human bite is very dangerous bite.

SPIDER BITE

- Types of spider bites include:
  - Black widow spider bite
  - Brown recluse spider bite

BLACK WIDOW SPIDER BITE

- Black widow spiders are black with a red hourglass on their belly.
- Their bite causes neurotoxicity.
- It causes muscle spasms, pain and abdominal rigidity.
- Nausea, vomiting, Hypertension, tachycardia and diaphoresis are the other features.
- Its effects last for 2-3 days.
- Cleaning the wound, antibiotics, antihistamines, specific antivenims are the line of treatment.
- The antidote is IV calcium gluconate. Muscle relaxants also help.



Figure 63: Black widow spider

BROWN SPIDER BITE

- These are often not recognized at the time, by the next day a skin ulcer develops with necrotic center

and a surrounding halo of erythema.

- It releases sphingomyelinase-D which causes necrosis of the skin and hemolysis.
- Local effects: skin rash, blister formation, necrosis of the skin and extensive ulceration.
- Systemic effects: anaphylaxis, arthralgia, thrombocytopenia, hemolysis and renal failure.
- Often it can be life-threatening especially in children and elderly.
- The effects last for 2-3 weeks.
- Wound debridement (surgical excision may be needed but should be delayed until the full extent of the damage is evident-as much as 1 week), antibiotics, antihistamines, steroid therapy, antivenins and management of systemic complications.
- Dapsone is helpful.



Figure 64: Brown spider

BEE STING

- Honey bee has got barbed stinger with two lancets.
  - ➢ These lancets get attached to human skin to release the venom.
  - ➢ Bees die after sting.
- Yellow jacket wasps are more aggressive.
- Bee venom contains dopamine, histamine, neurotoxin and toxic peptides.

CLINICAL FEATURES

- Allergic reactions
- Anaphylaxis: wheezing and rash
- Pain in the local region, edema, pruritus, flushing
- Hypotension (vasomotor shock-'pink and warm' shock), laryngeal edema, bronchospasm
- Muscle spasm, convulsion
- Renal failure in severe cases

MANAGEMENT

- The stingers should be removed without squeezing them.
- Soda bicarbonate is used to neutralize the bee venom.
- Antibiotics and antihistamines are also given.
- Epinephrine is the drug of choice (0.3 to 0.5ml of 1: 1 000 solution).

SCORPION STING

- Scorpion stings are painful but rarely life-threatening.
- Young children and older adults are most at risk of serious complications.

- World-wide, only about 30 of estimated 1500 species of scorpions produce venom toxic enough to be fatal.
- A scorpion sting is caused by the stinger in a scorpion's tail. When a scorpion stings, its stinger can release venom. The venom contains a complex mix of neurotoxins.

CLINICAL FEATURES

- Symptoms:
  - Pain which can be intense
  - Numbness and tingling
  - Slight swelling
  - Warmth
- Signs:
  - Difficult breathing
  - Muscle twitching or thrashing
  - Unusual head, neck and eye movements
  - Drooling
  - Sweating
  - Nausea and vomiting
  - Hypertension
  - Tachycardia
  - Restlessness or excitability or inconsolable crying in children
  - Allergic reactions and anaphylaxis: hives, difficult breathing, nausea and vomiting

MANAGEMENT

- Analgesia
- Scorpion antivenin

CHEST SURGICAL CONDITIONS

BASIC SURGICAL PROCEDURES

BASIC SURGICAL PROCEDURES

INTERCOSTAL CHEST DRAIN

INTERCOSTAL CHEST DRAIN (ICD)

- Chest drain (also called chest tube, thoracic catheter, tube thoracostomy or intercostal drain) is a conduit inserted through the chest wall and into the pleural space to remove air (pneumothorax) or fluid from the pleural cavity.
- The fluid can be blood (hemothorax), pus (empyema), chyle (chylothorax) or a pleural effusion.
- For a chest drain to be efficient it must allow re-expansion of the underlying lung and must prevent entry of air or drained fluid back into the chest.
- A chest drain must therefore have 3 components:
  - An unobstructed chest drain
  - A collecting container below chest level
  - A one-way mechanism such as an underwater seal or Heimlich valve

INDICATIONS

1. Recurrent pleural effusion or massive pleural effusion
   - Chylothorax (lymphatic fluid)
   - Empyema thoracis (pus)
   - Hemothorax (blood)
   - Hydrothorax (Serous fluid)
2. Pneumothorax
3. In any hypo-ventilated patient
4. Post-operation:
   - Thoracotomy
   - Esophagectomy
   - Cardiac surgery

CONTRAINDICATIONS

1. Refractory coagulopathy
2. Diaphragmatic hernia
3. Hepatic hydrothorax
4. Scarring in the pleural space (adhesions)

MECHANISM OF ACTION

- Drainage occurs during expiration when pleural pressure is positive.
  - Recall when relaxed pleural pressure is sub-atmospheric (-2.5 mmHg). Normal atmospheric pressure 760mmHg

- Fluid within pleural cavity drains into an underwater seal.
- Air bubbles through water seal to outside world.
- The length of drain below fluid level is important. If greater than 2-3 centimeters increases resistance to air drainage.

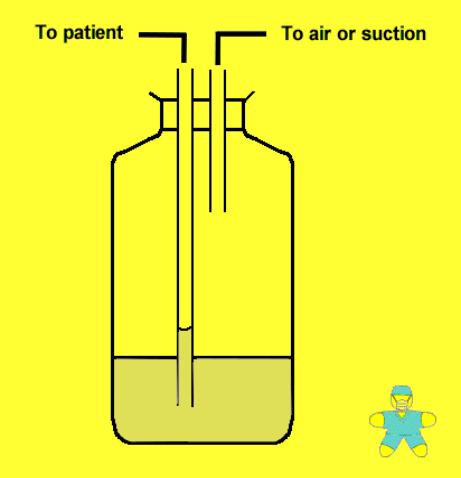


Figure 65: Basic concept of an intercostal chest drain

PRINCIPLES OF CHEST DRAINS

INSERTION

- Unless emergency situation then pre-procedure chest X-ray should be performed.
- Drain usually inserted under local anesthesia using aseptic technique.
- Inserted in 5th intercostal space in mid-axillary line
- Inserted over upper border of rib to avoid intercostal vessels and nerves.
- Blunt dissection and insertion of finger should ensure that pleura cavity is entered.
- Previous it used to be taught that:
  - To drain fluid, it should be inserted to base of pleural cavity
  - To drain air, it should be inserted towards the apex of the lung
- Probably the position of the tube in the pleural cavity does not matter provided there is no loculation of fluid within pleural cavity.
- A large drain (28 Fr or above) should be used to drain blood or pus.
- Drain should be anchored and purse-string or Z-stitch inserted in anticipation of removal



Figure 66: An Intercostal chest drain inserted with a Z-string

EQUIPEMENT

- Bottles:
  - Under water vacuum

- Under water seal
- One-way valve apparatus

ASSESING FUNCTIONALITY OF CHEST DRAIN

- Ask the patient to cough and check out bubbles in the bottle
- Check the moving menisci

TRANSFERRING THE PATIENT

- Clump the tube with artery forceps
- Patient should carry it not raised above insertion
- When you reach the place, put it down and unclamp

DO'S AND DON'TS OF CHEST DRAINS

- Avoid clamping of drain as it can result in a tension pneumothorax
- Drain should only be clamped when changing the bottle or transferring patient.
- Always keep drain below the level of the patient.
- If lifted above chest level contents of drain can siphon back into chest.
- If disconnection occurs reconnect and ask patient to cough.
- If persistent air leak consider low pressure suction.
- Observe for post-expansion pulmonary edema.

REMOVAL

- Remove drain as soon as it has served its purpose.
- For a simple pneumothorax it can often be removed within 24 hours.
- To remove drain, ask patient to perform a Valsalva maneuver.
- Remove drain at the height of expiration.
- Tie to pre-inserted purse-string or Z-stitch.
- Perform a post-procedure chest X-ray to exclude a pneumothorax

COMPLICATIONS

- "There is no organ in the thoracic or abdominal cavity that has not been pierced by a chest drain"

EARLY

- Misplacement: Tube displaced, tube inserted too far, tube placed subcutaneously.
- Subcutaneous surgical emphysema
- Injury to organs and other structures:
  - Diaphragm causing injury to liver or spleen,
  - Parenchymal or hilar injuries, lung lacerations
  - Injury to the heart
  - Phrenic nerve injury
  - Neurovascular bundle injury
  - Esophageal perforation-hemorrhage
  - Bowel injury in the presence of unrecognized diaphragmatic hernia
- Contralateral pneumothorax
- Hemorrhage and hemothorax

INTERMEDIATE

- Surgical emphysema
- Re-expansion pulmonary edema
- Tube blockage
- Infection along the chest tube
- Pain

LATE

- Retained hemothorax
- Pneumothorax after removal
- Empyema
- Abscess
- Fistula-formation

CHEST TRAUMA

CHEST TRAUMA

- When considering trauma to the chest it is important to remember the anatomical structures present and how they could be damaged.
- In general trauma to the chest can be considered as:
  - Blunt
  - Penetrating
- Chest injuries may occur secondary to other traumatic processes and may significantly impair breathing for example severe burns to the chest can cause encircling eschar that impairs the respiratory excursions of the thoracic cage.
- In both blunt and penetrating types of chest trauma the forces causing the trauma can either be:
  - Low velocity
  - High velocity.
- The mechanism of injury is important as it determines the extent and type of damage sustained.
- The mechanisms that cause injury to the chest are identical to those that cause injuries to the abdomen and frequently patients with thoracic injuries also have abdominal involvement. An important example of this is a rib fracture on the left side that leads to splenic laceration.
- The problems caused by thoracic trauma relate to the disruption of adequate gas exchange, leading to hypoxia, hypercapnia and acidosis.
- Other problems relate to shock secondary to disruption of major viscera in the chest.

TYPES OF CHEST INJURY

- These include:
  - Chest wall trauma
    - Fractured ribs
    - Flail chest
    - Fractured sternum
  - Lungs and pleura
    - Simple pneumothorax
    - Tension pneumothorax
    - Hemothorax
    - Pulmonary laceration
    - Tracheobronchial disruption
  - Cardiovascular injuries
    - Cardiac trauma

    - Aortic disruption
  - Diaphragmatic injuries
- These injuries can be detected on either primary survey or secondary survey.
- Primary survey injuries:
  - Fractured ribs
  - Flail chest
  - Fractured sternum
  - Pneumothorax
  - Tension pneumothorax
  - Hemothorax
  - Cardiac tamponade
- Secondary survey injuries:
  - Pulmonary contusion
  - Aortic disruption
  - Diaphragmatic injury
  - Tracheobronchial disruption
  - Esophageal disruption

PRIMARY SURVEY INJURIES

FRACTURED RIBS

- The most common fractures are those of the lower ribs usually the 5th to the 9th.
- Fractures of the lower ribs have significance because they can lacerate upper intra-abdominal viscera (especially the spleen).
- It is also important to note that fractures of the upper 4 ribs particularly the 1st rib are indicative of extremely high-velocity trauma as these ribs do not usually fracture because of their short, stout anatomy.
- Fractured ribs can also puncture the pleura and even the lung substance and cause a pneumothorax.
- In more severe thoracic cage trauma 2 or more consecutive ribs can be fractured in 2 or more places this is defined as a flail segment.
- The consequences of rib fractures are treated individually. A simple rib fracture itself requires no specific treatment other than analgesia.

FLAIL SEGMENT

- This happens when 2 or more consecutive ribs are fractured in 2 or more places.
- A flail segment compromises a patient's breathing because the fractured segment undergoes paradoxical movement on inspiration. The segment moves inwards rather than outwards and hence impedes ventilation of the ipsilateral lung.

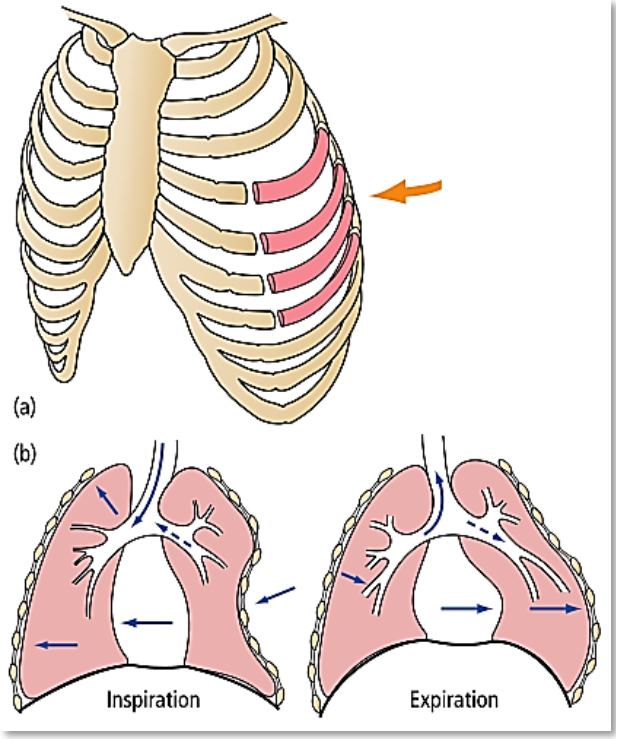


Figure 67: Rib fractures leading to a flail segment with paradoxical movement. The paradoxical respiration can lead to embarrassment of gaseous exchange.

- The degree of trauma causing a flail segment is likely to lead to underlying lung contusion and even laceration.
- A flail segment requires strapping of the segments and even positive pressure ventilation to compensate for poor oxygenation and to aid healing of the chest.

FRACTURED STERNUM

- The mechanism of injury is usually a severe impact on a solid structure, most common example is where a driver of a motor vehicle is thrown onto the steering wheel.
- A fractured sternum produces intense, deep pain.
- The risk of injury to the mediastinum cause the greatest concern.
- Patients with sternal fractures should always be admitted and observed and ECG performed to look for electrical abnormalities
- Cardiac enzymes should also be measured to ensure myocardium has not sustained significant damage.
- Management also involves adequate analgesia.

PNEUMOTHORAX

- This is air in the pleural cavity secondary to trauma.

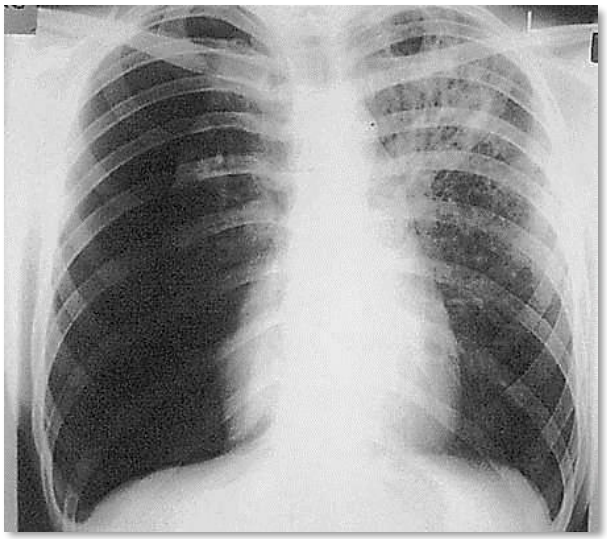


Figure 68: Chest X-ray showing a simple pneumothorax on the right and a lung contusion on the left

- It is the most common pleural-related problem that any doctor is likely to encounter in practice.
- Despite most pneumothoraces occurring with trauma, they may be associated with other diseases e.g. bullous disease of the lung secondary to emphysema.
- Pneumothoraces may be caused by fractures of the ribs secondary to blunt trauma or by a penetrating injury (e.g. a knife).
- A pneumothorax can either be a:
  - Tension pneumothorax
  - Simple pneumothorax
- Tension pneumothorax develops when a penetrating injury of the chest allows air from the external environment to enter the pleural cavity but not leave the wound acting as a one-way valve.
- Eventually the accumulation of air in the pleural cavity displaces the mediastinum to the opposite side, thereby decreasing venous return and leading to cardiac arrest that shows signs of electromechanical dissociation.

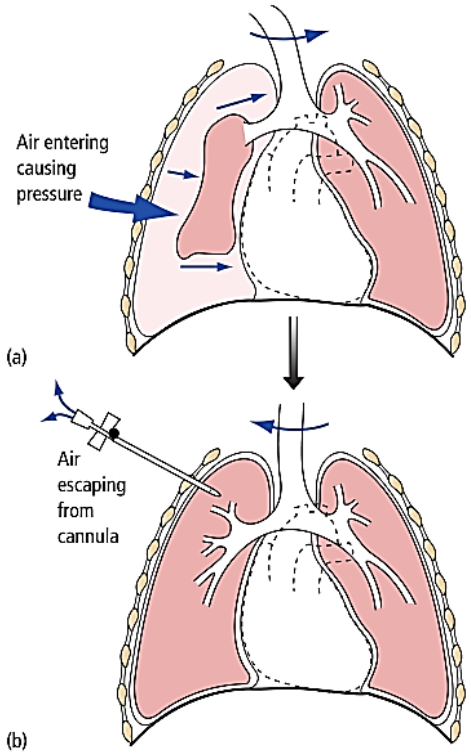


Figure 69: The mediastinal shift that occurs due to a tension pneumothorax. Release of tension pneumothorax after insertion of a cannula into the right second intercostal space.

- A tension pneumothorax should be recognized at the time of the primary survey.
- The patient is typically short of breath and the trachea is displaced away from the side injury.
- The chest on the injured side also fails to expand and is Hyperresonant to percussion.
- If, together with the above findings, breath sounds are absent on the injured side, the patient should not proceed to chest X-ray.
- Immediate management is to relieve the build-up air in the

pleural cavity by needle decompression.

- A large bore needle is inserted into the 2nd intercostal space in the midclavicular line on the affected side.
- Successful decompression is heralded by a rush of air through the needle after insertion. This converts a tension pneumothorax into a simple pneumothorax.
- Simple pneumothoraces are those where there is a defect in the chest wall that allows air to enter and leave the pleural cavity causing the lung to collapse.
- Air does not accumulate in the pleural cavity there is no deviation of the mediastinum as seen in a tension pneumothorax.
- The initial management of an open pneumothorax is prompt closure of the defect with a sterile occlusive dressing large enough to overlap the edges of the wound and taped securely on 3 sides. This works as a flutter valve preventing air from entering the pleural cavity when the patient inhales but allows air to leave when the patient exhale.
- Definitively both pneumothoraces are managed by insertion of a chest tube to drain the air from the pleural cavity.
- The majority of simple pneumothoraces resolve in days when the re-expanded lung adheres to the chest wall due to the secretion of fibrin.
- A drain tube should only be removed if there is cessation of bubbling in the drainage bottle even with suction.
- When the lung is fully expanded, this lack of bubbling needs to have been present for at least a day.
- It is important to tie a second purse-string suture to allow adequate closure of the drain site. After drain removal, a chest X-ray should be taken to confirm continued lung expansion.

HEMOTHORAX

- This is accumulation of blood in the pleural space.
- Hemothoraces are most commonly associated with laceration or disruption of the lung parenchyma and are obviously more common in penetrating than blunt injuries.
- Hemothoraces can either be:
  - Small: these are less than 1L of blood
  - Massive: rapid accumulation of more than 1L of blood in the chest cavity. This is most commonly caused by disruption of the hilar vessels of the lung and hence the problems experienced by the patient would be not only those of

hypoxia but also those of hypovolemia.

- Clinically, a massive hemothorax is characterized by hypoxia, hypotension and a chest where there may be obvious signs of trauma and on the ipsilateral side decreased expansion, stony dull percussion note and decreased breath sounds.
- Initial management involves insertion of a large bore chest tube into the 6th intercostal space in the midaxillary line.
- If a massive hemothorax is suspected, the blood from the chest can be used for auto-transfusion.
- The subsequent management of the patient depends on the volume of blood loss.
  - If over 1L is immediately drained from the thoracic cavity, the patient requires an early thoracotomy
  - If less than 1L is drained, the patient can be managed expectantly with decision for surgery based on the rate of continuing blood loss.
  - If the rate of blood loss is 200ml/h over the subsequent 2-4 hours, the patient should be considered for surgery.

CARDIAC TAMPONADE

- Cardiac tamponade is the accumulation of fluid in the pericardial sac which restricts the filling and contraction of the heart and leads to shock.
- If this fluid is blood it can come from disruption of the great vessels or the heart itself, most commonly as a result of penetrating injuries, although severe blunt trauma can also be a cause.
- As little as 20ml of blood can cause symptoms.
- Beck's triad of cardiac tamponade:
  - Elevated JVP
  - Decreasing in blood pressure
  - Muffled heart sounds
- Additionally, the patient may demonstrate pulsus paradoxus and a rise in venous pressure with inspiration (Kussmaul's sign).
- Transthoracic echocardiography can help in confirming the diagnosis. However, the diagnosis is often made only when Pericardiocentesis is performed in a last-ditch effort to relieve the cardiogenic shock.
- In this procedure, a long spinal needle is inserted in the subxiphoid region and blood is evacuated. This procedure should always be performed under ECG monitoring.
- In very extreme situations the thorax is opened and the pericardium evacuated.

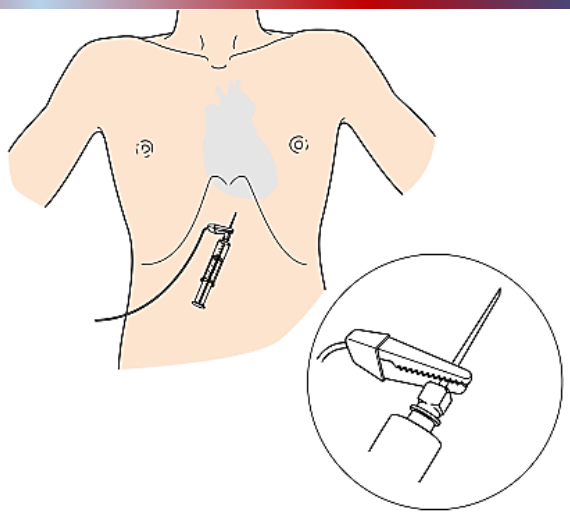


Figure 70: Site of insertion of a Pericardiocentesis needle to decompress a cardiac tamponade

SECONDARY SURVERY CHEST INJURIES

- Hemothoraces and pneumothoraces can also cause death during the secondary survey however, the occurrence and discovery of visceral injuries that were not obvious initially tend to dominant the secondary survey.

PULMONARY CONTUSION

- This can often display subtle signs, where significant portions of the lung fail to function and cause the patient to become markedly hypoxic.
- The patient's injuries may often be sufficiently severe to require assisted ventilation and may also contribute to the development of adult respiratory distress syndrome (ARDS).
- More severe pulmonary injuries can lead to pulmonary laceration. However, this is most commonly observed with an associated fractured rib or secondary to penetrating trauma.

AORTIC DISRUPTION

- Traumatic disruption of the aorta is a common cause of death after trauma that involves very rapid deceleration from great speeds such as high-velocity road traffic accidents and falls from a great height.
- These patients are initially stable but continue to be hypotensive despite resuscitation due to failure of hematoma to contain the bleeding.
- There are usually few specific signs and symptoms.
- A high index of suspicion and a history of decelerating force will lead to inspection of chest X-ray for specific signs of mediastinal widening.
- If the patient is stable, angiography and even CT scanning will help confirm the diagnosis.
- Immediate referral to a cardiothoracic surgeon is imperative if the patient is to survive.

DIAPHRAGMATIC INJURY

- Huge compressional forces can cause the diaphragm to be torn from its attachment.
- Diaphragmatic injuries can also be due to penetrating trauma where there is associated abdominal injury.
- Diaphragmatic trauma is often missed or diagnosed late.
- Some patients may present later with herniation through previous diaphragmatic defects caused by trauma.
- On chest X-ray these injuries may be recognized by an elevated diaphragm and confirmed by inserting contrast into the esophagus and observing a high stomach.

TRACHEOBRONCHIAL DISRUPTION

- This is uncommon and fatal if overlooked.
- Patients who suffer severe tracheobronchial disruption usually die at the scene of the accident.
- Those with minor degree of disruption are frequently recognized by the fact that they have a pneumothorax with associated emphysema, the pneumothorax persists despite chest tube drainage.
- Frequently, definitive surgery is required to correct the abnormality.

ESOPHAGEAL DISRUPTION

- The esophagus is most commonly traumatized by penetrating trauma.
- The esophagus can also tear due to forceful expulsion of contents from the stomach during hug impacts.
- This causes gastric contents and air to leak into the mediastinum, resulting in mediastinitis and air in the subcutaneous tissue of the neck, recognized as surgical emphysema.
- Patients may also develop a pneumothorax usually more common on the left than the right.
- The diagnosis is confirmed when particulate gastric matter is recovered from the chest tube.
- Prompt diagnosis and repair produce the best results in these patients.

Anatomical region	Clinical features	Management
CHEST WALL		
Fractured ribs	• Usually 6th to 9th ribs. • Fracture of 1st rib or upper 4 ribs indicates high velocity trauma	• Adequate analgesia • Breathing exercises
Flail chest	• Paradoxical chest wall movement on respiration	• Adequate analgesia • Strapping of flail segment • Positive pressure ventilation
Fractured sternum	• Intense deep pain • Look for cardiac injury	• Adequate analgesia • Cardiac monitor • Check cardiac enzymes
LUNG AND PLEURA		
Simple pneumothorax	• Dyspnea • Chest wall Hyperresonant	• Chest X-ray • Chest drain if >5-10% lung collapse (5th-6th intercostal space midaxillary line)
Tension pneumothorax	• Dyspnea • Trachea deviated to opposite side • Chest wall Hyperresonant • Cardiac arrest	• Do not perform chest X-ray to confirm diagnosis • Insert large-bore needle into the chest (2nd intercostal space midclavicular line) then chest X-ray and chest drain
Open (sucking) pneumothorax	• Open defect in chest wall	• Cover defect with sterile occlusive dressing tapped down on 3 sides.
Hemothorax	• Penetrating injury • Decreased expansion, stony dull percussion, reduced breath sounds • + hypoxia and shock for massive hemothorax (>1L) • + dyspnea for small hemothorax (<1L)	• Insert large-bore chest drain • If >1L blood discharges immediately patient needs thoracotomy • If <1L blood discharges immediately, observe • If continuing blood loss of 200ml/h patient needs thoracotomy
Pulmonary contusion or laceration	• Hypoxia • ARDS	• May need ventilation

Tracheobronchial disruption	• Pneumothorax plus subcutaneous emphysema • Persistent air leak from chest drain	• Surgical repair
CARDIOVASCULAR		
Cardiac tamponade (cardiac trauma)	• Beck's triad: Elevated JVP, Decreased blood pressure and muffled heart sounds • Pulsus paradoxus • Rise in JVP on inspiration (Kussmaul's sign)	• Pericardiocentesis • Sternotomy and drainage of pericardium (open repair of heart injury)
Aortic disruption	• History of rapid deceleration in high-velocity injury • Hypotensive despite resuscitation	• Chest X-ray shows widened mediastinum • Diagnosis confirmed with contrast-enhanced CT • Open surgical repair
Venous injury	• Rare • History of penetrating injury	• Surgical repair
MISCELLANEOUS		
Esophagus	• Mediastinitis • Subcutaneous emphysema in neck sometimes hydropneumothorax (L>R)	• Urgent surgical repair • Poor prognosis with mediastinitis
Diaphragm (rupture or penetration)	• History of compression force on abdomen • History of penetrating trauma	• Often present late with herniation of stomach into chest • Reduction of stomach into abdomen and open repair of diaphragm

ESOPHAGEAL PATHOLOGY

RELEVANT SURGICAL ANATOMY

- The esophagus is a hollow muscular tube that transports food from the pharynx to the stomach.
- It begins at the lower edge of the cricoid cartilage (lower border of C6 vertebra) and ends at the esophagogastric junction (T12 vertebra).
- It is 25cm long.
- The esophagus lies anterior to the vertebral column and posterior to the trachea it travels through the thorax in the posterior mediastinum and empties into the cardia of the stomach. It lacks serosal layer but is surrounded by a layer of loose fibroareolar adventitia. Because the esophagus does not have a serosal layer, esophageal anastomoses are prone to leaks.
- The esophageal mucosa is lined by squamous epithelium that becomes columnar near the gastroesophageal junction. The next layer encountered moving radially outward is the submucosa which has the submucous (Meissner) plexus. Next are 2 muscular layers, the inner circular muscle layer and the outer longitudinal muscle. Sandwiched in between these two muscular layers is the myenteric (Auerbach) plexus.
- The esophagus is divided into 3 parts:
  - Upper (cervical esophagus):
    - Extends from the cricophayngeus (horizontal part of the inferior constrictor muscle i.e. lower cricoid border and 6th cervical verterba) to the level of the carina.
    - This part lies in the midline. It descends anterior to the vertebral column through the superior and posterior mediastinum.
    - In the groove between the esophagus and trachea is the recurrent laryngeal nerve on each side.
    - Lateral are common carotid arteries and posterior part of the thyroid gland
    - In the lower neck the esophagus deviates to the left.
    - The thoracic duct ascends on the left side.

- Middle (thoracic esophagus):
  - Extends from the level of the carina to halfway between the carina and esophagogastric junction
  - In the superior mediastinum it is between the trachea and the vertebral column
  - It deviates from the left to the midline and then to the right behind the aortic arch (pulsating constriction) 25cm from incisors.
  - It enters the posterior mediastinum and descends on the right of the thoracic aorta.
  - Below the pulmonary roots the vagus nerves descend in contact with the esophagus. The left trunk lies on the anterior side and right trunk lies on the posterior side.
  - It inclines to the left, crosses the midline to the right as it enters the diaphragm at 10^{th} thoracic vertebra to enter the abdomen.
- Lower (abdominal esophagus):
  - This is the remaining segment that joints the stomach
  - It emerges from the right diaphragmatic crus slightly left to the midline and level with 10^{th} thoracic vertebra
  - It grooves its posterior surface to left lobe of the liver and curves sharply to the left ending at the gastric cardiac orifice.
  - This part is the shortest and is 1.25cm long.

Recall the vertebral levels at which the following structures traverse the diaphragm:

T8- inferior vena cava (IVC)

T10- esophagus

T12- aorta

"I (IVC) ate (T8) ten (T10) eggs (esophagus) at (aorta) noon (T120"

- Initially it is striated muscle and progresses to smooth muscle in the lower half. It is lined by squamous epithelium. The proximal 1/3 of the esophagus contains striated muscle only, the middle 1/3 contains both striated and smooth muscle whilst the distal 1/3 contains smooth muscle only. Clinically significant motility disorders involve smooth muscle in the lower 2/3 only.

- The cardia is the junction between the esophagus and stomach. It contains the squamocolumnar junction which forms the serrated Z-line that marks an abrupt change from the tough pale squamous epithelium of the esophagus to the columnar epithelium of the stomach.
- The lower esophagus is the most important site of communication between the portal and systemic venous systems in view of the development of varices in patients with portal hypertension.
- Three areas of narrowing (evident on barium swallow are seen), the so-called Esophageal constrictions:
  - Cricopharyngeal constriction- is at the beginning of the esophagus, it is 15cm from the incisor teeth
  - Aortic/Bronchial constriction- is where the left mainstem bronchus and aortic arch cross. It is 25cm from incisor teeth
  - Diaphragmatic constriction: this is at the hiatus. It is 40cm from the incisor teeth. This distance serves as an important landmark in upper gastrointestinal endoscopy.

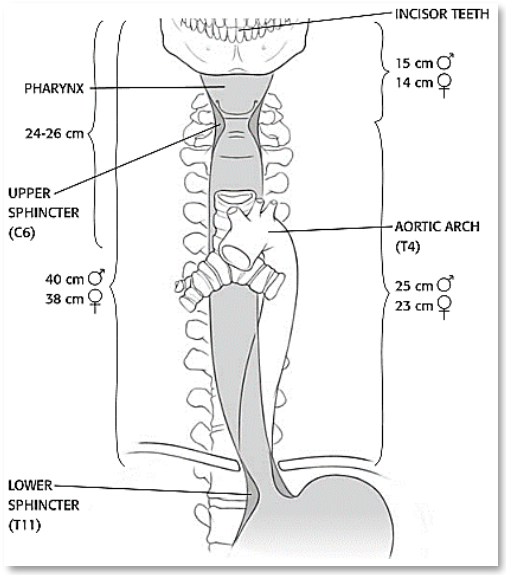


Figure 71: Important clinical endoscopic measurement of the esophagus in adults (Image adapted from First Aid for the Surgery Clerkship 2nd Edition)

- The esophagus receives its artery blood supply by the inferior thyroid artery, esophageal branches of the aorta, gastric arteries and inferior phrenic arteries
  - The superior and inferior thyroid arteries supply the upper esophagus.
  - The intercostals, left gastric and phrenic arteries supply the lower esophagus.
- Its venomous drainage is by the inferior thyroid vein, brachiocephalic vein, left hemiazygos vein, azygos vein, coronary vein, splenic vein and inferior phrenic vein. Veins are longitudinal and they lie in the submucosal plane in the lower third and in the muscular plane above.
  - Venous drainage of the upper esophagus is into the inferior thyroid and vertebral veins, whereas the mid and lower esophagus drains into the azygos, hemiazygos and left gastric veins.
- The lymphatics forms extensive plexuses such that lymph flows long distances in the large submucosal plexus before passing through the muscular coat to draining lymph nodes.
- These are grouped into 3 main tiers:
  - Para-esophageal (lie alongside the esophagus)
  - Mediastinal lymph nodes

- Deep cervical, supraclavicular, tracheobronchial and coeliac nodes (from above downwards)

- In general lymph drainage from the upper 2 thirds of the esophagus proceeds in a proximal direction whereas the lower third distally to the subdiaphragmatic region and coeliac lymph nodes.
- The esophagus receives parasympathatic nerve branches of the vagus nerve.
- 2 nerve plexuses in the esophageal wall (Meissner's plexus in the submucosa and Auerbach's plexus in the muscularis) form networks of ganglion cells which receive axons from the vagus.
- Two sphincters are present which function as control points:
  - Upper esophageal sphincter (UES, Cricopharyngeus muscle): prevents the passage of excess air into the stomach during breathing.
  - Lower esophageal sphincter (LES):
    - It is a physiological sphincter with a high physiological pressure zone located in the lower end of the esophagus in the terminal 4cm with a resting pressure of 10-25mmHg.
    - It relaxes with initiation of pharyngeal phase of swallowing and prevents reflex of gastric contents when swallowing is not occurring.
    - Esophageal peristalsis accompanying swallowing is termed primary peristalsis. Secondary peristalsis can be initiated by the esophageal musculature without the pharyngeal phase to clear the esophagus of any substance left behind from primary peristalsis.
    - This LES prevents reflux of gastric and duodenal contents and is influenced by food, gastric distension, gastric pathology, smoking, GI hormones and alcohol.

RELEVANT PHYSIOLOGY

- Swallowing or deglutition refers to the passage of food from the oral cavity into the stomach.
- Swallowing is divided into 3 phases:
  - Voluntary (oral) phase: initiates swallowing process
  - Pharyngeal phase: is involuntary and constitutes passage of food through the pharynx into the esophagus.
  - Esophageal phase: involuntary phase that transports food from the pharynx to the stomach.
- During the voluntary (oral) stage the food bolus is voluntarily squeezed or rolled posteriorly into the pharynx by pressure of the tongue upwards and backwards

against the palate. From here on swallowing becomes entirely or almost entirely automatic and ordinarily cannot be stopped.

- The pharyngeal stage of swallowing is caused by a swallowing reflex
  - Receptors present around the opening of the pharynx (especially over the tonsillar pillars) are stimulated when the bolus moves from the mouth into the pharynx and initiate the reflex activity.
  - Afferents impulses are carried through the trigeminal nerve, glossopharyngeal and vagus nerve to deglutition centers in the medulla oblongata and lower pons (i.e. the nucleus of the tractus solitaries and nucleus ambiguous)
  - Efferent impulses through the trigeminal, glossopharyngeal, vagus and hypoglossal cause muscular contraction.
  - The soft palate is pulled upwards to close the posterior nares preventing reflux of food into the nasal cavities.
  - The palatopharyngeal folds on each side of the pharynx are pulled medially to form a sagittal slit through which the food must pass into the posterior pharynx (the slit performs a selective action allowing food that has been masticated sufficiently to pass with ease. Because this stage of swallowing lasts less than 1 second, any large object is usually impeded too much to pass into the esophagus.)
  - The vocal cords of the larynx are strongly approximated and the larynx is pulled upwards and anterior by the neck muscles. These actions combined with the presence of ligaments prevent upward movement of the epiglottis causing the epiglottis to swing backward over the opening of the larynx. All these effects acting together prevent passage of food into the nose and trachea.
  - The upward movement of the larynx also pulls up and enlarges the opening of the esophagus, at the same time the upper esophageal (pharyngoesophageal sphincter) relaxing allowing food into the esophagus. Between swallows, the sphincter remains strongly contracted, thereby preventing air from going into the esophagus during respiration.
- The pharyngeal phase occurs in less than 6 seconds during which respiration is inhibited.
- During the esophageal stage food is propelled by 2 types peristalsis:
  - Primary peristalsis: this is a continuation of the peristaltic wave that begins in the pharynx and spreads into the esophagus during the pharyngeal phase. This wave passes all the way from the pharynx to the stomach in 8 to 10 seconds.
  - Secondary peristalsis: this is a result of distention of the esophagus by retained food (as primary peristalsis fails to move all the food in the esophagus). It is

initiated by the intrinsic circuits in the myenteric plexus and partly by reflexes that begin in the pharynx and are then transmitted upward through the vagal afferent fibers to the medulla and back to the esophagus via the glossopharyngeal and vagus.

  - The musculature of the pharyngeal wall and upper third of the esophagus is striated and thus peristaltic waves in this region are controlled by skeletal nerve impulses from glossopharyngeal and vagus nerves.
  - In the lower third of the esophagus the musculature is smooth muscle but this portion of the esophagus is also strongly controlled by the vagus through connections within the esophageal myenteric plexus.
  - When the esophageal peristaltic wave approaches the stomach, a wave of relaxation transmitted through myenteric inhibitory neurons precedes the peristalsis causing the stomach to relax and opening the lower esophageal (Cardiac) sphincter (receptive relaxation)
  - After food enters the stomach the cardiac sphincter contracts to prevent regurgitation of food in the esophagus. With this the esophageal phase of deglutition is completed.

- Tertiary esophageal contractions can occur spontaneously and are non-propulsive. They are encountered in various esophageal motility disorders but also occur in healthy individual.

DYSPHAGIA

- This is difficulty in swallowing.
- Painful swallowing is known as odynophagia.
- Dysphagia with associated hoarseness of voice may present in advanced pharyngeal or post cricoid carcinomas. At late stage, laryngeal carcinoma also causes dysphagia along with hoarseness of voice.
- Dysphagia for solids implies significant disease, whereas dysphagia for liquids only is more likely to be of functional origin (esophageal motility disorder) in which case it may be intermittent.
- Persistent and progressive dysphagia indicates mechanical narrowing of the esophageal lumen and is usually associated with regurgitation. Eventually, the patient is unable to swallow saliva and exhibits constant drooling.
- Obstructive dysphagia is first experienced when 20-30% of esophageal lumen is lost, most patients usually present when 50% of the esophageal lumen is compromised.

- When there is a problem with swallowing in the voluntary (oral or pharyngeal) phases, patients will usually say that they cannot swallow properly but they do not characteristically describe 'food sticking'. Instead, when they try to initiate a conscious swallow, food fails to enter the esophagus, stays in the mouth or enters the airways causing coughing or spluttering.
- Esophageal dysphagia occurs in the involuntary phase and is characterized by a sensation of food sticking.

CLASSIFICATION

- Dysphagia may be classified by
  - Onset
    - Acute: due to foreign body impaction or acute infection
    - Chronic: stricture or carcinoma etc.
  - Duration
    - Intermittent
    - Progressive
  - Location
    - Oropharyngeal: Pathology in voluntary/pharyngeal phase of the swallowing wherein patient also develops cough while swallowing.
    - Esophageal: Pathology in esophageal involuntary phase of swallowing characterized by food getting stuck in the pathway. But site of "food getting stuck" feeling is not relevant.

CAUSES

- They can be divided into
  - Extraluminal causes
  - Intramural causes
  - Luminal

EXTRALUMINAL CAUSES

- Mediastinal nodes- secondaries/ lymphoma/ TB
- Mediastinitis/ mass
- Aortic aneurysm, Thoracic artery aneurysm
- Rolling hiatus hernia
- Thyroid enlargement- malignant: it is uncommon to develop dysphagia in a thyroid swelling. There will always be dyspnea when dysphagia develops. Large

malignant thyroid or anaplastic thyroid can cause dysphagia with dyspnea or stridor. Retrosternal goiters may also present with dysphagia.

- Dysphagia lusoria: the word Lusoria means "sport of nature" in Latin. Dysphagia lusoria is a congenital vascular anomaly of aortic root.
  - Aortic arch anormalies are:
    - Double arch (40%)
    - Right and left ligamentum arteriosum (25%)
    - Anomalous innominate or common carotid artery
    - Aberrant right subclavian artery (10%)
  - All patient having this anomaly (dysphagia lusoria) have got an aberrant right subclavian artery in a transposed position arising from descending aorta that courses posterior to esophagus. Often there will be complete vascular ring around trachea and esophagus.
  - Commonly they are asymptomatic but may present with dysphagia, chest pain, stridor, wheeze, recurrent respiratory infection (usually presents after the age of 40).
  - Investigation: Chest X-ray, barium swallow (T4 level diagonal impression), endoscopy (shows pulsating extraluminal compressive mass), MRI, CT chest
  - Treatment is reconstruction or ligation of aberrant right subclavian artery by sternotomy/ by neck approach.
- Congenital anomalies
- Pharyngeal pouch (Zenker's diverticulum)
- Malignancy

INTRAMURAL CAUSES (ESOPHAGUS AND OTHER AREAS)

- Carcinoma esophagus: here dysphagia is of short duration and progressive. 2/3 of the lumen should be blocked by tumor to develop dysphagia. It is also associated with weight loss, anorexia and anemia.
- Corrosive/TB/inflammatory/congenital stricture esophagus
- Benign or malignant strictures, GERD (gastroesophageal reflux disease)
- Achalasia cardia: failure of the lower esophageal sphincter to relax. Dysphagia is more with liquids than solids. There is frequent regurgitation, recurrent chest infections due to aspiration. It is usually has a long history.

- Plummer-Vinson syndrome (esophageal web): iron deficiency anemia, glossitis, cheilosis and esophageal webs. Iron deficiency anemia leads to desquamated epithelium. It is common in middle-aged women.
- Esophageal diverticulum
- Carcinoma posterior 1/3rd of tongue/pharynx
- Diffuse esophageal spasm: they are in-coordinated contractions of the esophagus causing chest pain or dysphagia. It is common in distal 2/3rd of the esophagus. Hypertrophy of circular muscle fibers with very high persistent pressure of 400-500mmHg is specific. Treatment is calcium channel blockers, vasodilators, endoscopic dilation and extended esophageal surgical myotomy up to the aortic arch (very useful especially for dysphagia, not much for chest pain)
- Esophageal ring (Schatzki ring): ring of mucosal tissue
- Systemic sclerosis
- Congenital anomalies
- Retropharyngeal abscess/ peritonsillar abscess (Quinsy)/ acute tonsillitis/ pharyngitis

LUMINAL CAUSES

- Foreign body in esophagus- coin/ dentures/ fish or meat bone
- Infection: candidiasis, pharyngitis, retropharyngeal abscess
- Esophagitis
- Polyps
- Other causes: Cranial causes (neurological)
  - Bulbar palsy/pseudobulbar palsy, Infarction/hemiplegia
  - Vertebro basilar insufficiency

COMMON CAUSES OF DYSPAGIA	RARE CAUSES OF DYSPHAGIA
• GERD • Carcinoma esophagus • Plummer-Vinson syndrome • Mediastinal swellings-Primary tumors/nodal mass either lymphoma or secondaries or tuberculosis	• Diffuse esophageal spasm • Esophageal diverticula • Chagas' disease • Dysphagia lusoria • Thyroid swelling • Boerhaave's syndrome: vertical full thickness tear of lower esophagus due to vomiting with closed glottis. It is often life threatening and emergency • Neurological (stroke, bulbar palsy, motor neuron disease, Parkinson's disease etc.)

	• Congenital anomalies of esophagus • Drug induce: KCL, quinine, NSAIDs • Mediastinal fibrosis

EVALUATION OF THE PATIENT

HISTORY

- Demographics: Name, age, sex, address, occupation, religion
- Symptoms:
  - When did it start?
  - Is it new or long standing?
  - Is it rapidly worsening or relatively constant? (Intermittent- spasms, constant./worsening- malignant stricture or both-achalasia)
  - Was there difficult swallowing solids or liquids from the start? (if it started with liquids, think motility disorders-systemic sclerosis, myasthenia gravis, bulbar palsy or achalasia or pharyngeal problems. If it started with solids think mechanical problems e.g. benign or malignant stricture)
  - Is it worse on solids from the start?
  - Is there any difficulty in making swallowing movement? (bulbar/pseudobulbar palsy)
  - At which level does food stick?
  - Is swallowing painful? (cancer, ulcer, esophageal spasm- relieved by glyceryl trinitrate)
  - Is dysphagia intermittent or is it constant and getting worse?
  - Can it be relieved by anything e.g. warm drinks?
  - Is it associated with coughing (tracho-esophageal fistula)?
  - Does the neck bulge or gurgle on drinking? (Pharyngeal pouch)
  - Any chest pain? Heart burn? (GERD)
  - Coughing on lying? (achalasia, pharyngeal pouch)
  - Regurgitation or substance cramp?
  - Any significant weight loss? (Consequence of poor intake or sign of malignancy)
- Predisposing factors:
  - History of alcohol or smoking
  - Diet- history of hot foods (chili)
  - Any gastro-esophageal reflux disease
  - History of taking corrosive substance (Accidental or intentional suicidal)
  - History of surgery (strictures)

- History of radiotherapy (Ca esophagus)
- Immune suppression

INVESTIGATIONS

- Diagnostic:
  - Chest X-ray: shows mediastinal mass lesion/foreign body
  - Barium swallow: may show irregular filling defect or extrinsic compression.
  - Esophageal manometry: in achalasia cardia/GERD
  - 24 hours pH monitoring: ideal and most accurate for GERD. Small pH probe (transnasal catheter) is passed into the distal esophagus 5cm proximal to upper margin of the LOS under manometry guidance. The probe is connected to a digital recorder worn by the patient for 2 hours. Record is analysed using a computer. A pH less than 4 for more than 4% of total 24 hours period (more than neart to one hour in total in 24 hours) is pathological reflux. It is often assessed by scoring system. Radio-telemetry pH probes are used not without any nasal tube. It is passed and placed on the esophageal wall using endoscope.
  - Esophagoscopy: once lesion is detected, it is treated accordingly. Biopsy from lesions, endotherapy if needed should be carried out (like F/B removal, stricture dilatation, sclerotherapy)
  - Endosonography: useful in many conditions. It can assess the site, layers of esophagus, nodes, spread etc. properly. Different layers are seen as alternating hyperechoic and hypoechoic bands.
  - Ultrasound: to see abdominal nodes, liver/ascites
  - CT scan: identifies the anatomical location of the cause (nodes/tumors/aorta/cardiac causes/congenital). Extent, spread, nodal status, size and operability of the tumor also well-assessed
  - MRI
- Full blood count: hematocrit, anemia is common with tumors than reflux
- Liver function test: to exclude hepatic disease

TREATMENT

- Depends on cause- modified Heller's myotomy, esophageal resection, dilatation, F/B removal etc.

GASTROESOPHAGEAL REFLUX DISEASE

ESOPHAGITIS

ESOPHAGEAL STRICTURES

- This is local, stenotic regions within the lumen of the esophagus.
- They are usually as a result of an inflammatory or neoplastic process.
- Benign strictures arise most commonly secondary to long-standing reflux disease and less commonly esophageal/gastric resections or ingestion of corrosive agents.

ETIOLOGY AND RISK FACTORS

- Corrosive/caustic esophagitis (most common):
  - Mainly due to ingestion of alkali (Lye Stricture-Lye is a strong alkali sodium hydroxide) sodium hydroxide, occasionally due to acid (sulphuric acid, nitric acid). Acid commonly damages the stomach.
  - Ingestion of corrosive substances can be accidental (children) or suicidal (adults)
  - It causes extensive inflammation of the mucosa with periesophagitis which if not treated leads to multiple strictures in the esophagus.
  - Sometimes it causes severe life-threatening necrotizing lesion which requires immediate surgical intervention.
  - Alkali is odorless and tasteless and so large volume is ingested. Alkali causes liquefaction, saponification and thrombosis of vessels and later leading to fibrosis and stricture.
  - Acid causes intense pylorospasm, antral pooling of acid, coagulation necrosis and eschar formation.
  - Severity depends on type of agent, its concentration and volume.
- Long standing GERD (peptic stricture/esophagitis induced)
- Radiation esophagitis
- Infectious esophagitis e.g. Tuberculosis
- Foreign body
- Post-surgical
- Congenital
- Drugs like tetracycline, vitamin C
- Sclerotherapy for bleeding varices

CLINICAL FEATURES

- Small strictures may remain asymptomatic however some may obstruct the esophageal lumen significantly causing progressive dysphagia for solids.

- Odynophagia may or may not be present.
- Differential diagnosis being esophagitis due to candidiasis especially in HIV positive patients.
- Features of esophageal corrosive lesion:
  - Acute/ immediate: lasts for 3 weeks
    - Severe pain, shock, laryngeal edema
    - Mediastinitis, septicemia, hemorrhage, perforation
  - Late/chronic
    - Dysphagia
    - Stricture-50%
    - Severe malnutrition
    - Recurrent respiratory infection
    - Esophageal shortening
    - Malignant changes
    - Tracheo-esophageal fistula formation
- Corrosive strictures can be multiple. Damage is more in lower 1/3rd of the esophagus.
- Phases of tissue injury in corrosive ingestion:
  - Phase 1: Acute necrosis (1-4 days)
  - Phase 2: Ulceration-Granulation (4-12 days)
  - Phase 3: Cicatrisation and scarring (3weeks-6 months)
- Degrees of burns after corrosive esophageal and gastric burns:
  - 1st degree: mucosal hyperemia and edema
  - 2nd degree: small bleeding, exudates, ulcers, pseudomembrane
  - 3rd degree: mucosal slough, deep ulcers, massive bleed, complete obstruction, charring, perforation

DIAGNOSIS

- Initial evaluation via barium swallow may reveal the presence of stricture. It establishes the length and width of the stricture. It shows a Cork-screw esophagus (differential diagnosis: esophageal spasm).
- Esophagoscopy is necessary in all cases, since the stricture should be evaluate for malignancy, also useful to determine the appropriate treatment. It should be done with caution as the danger of perforation is high.



Figure 72: Cork-Screw esophagus

MANAGEMENT

- The esophagus is visualized endoscopically and bougie dilators are carefully passed through the stricture, each successful dilation is done with progressively larger dilator.
- Dysphagia is relieved in most cases following adequate dilatation of the esophageal lumen.
- The principal aim of dilatation is to stretch the stricture gradually to achieve an esophageal lumen of 10-15mm in diameter.
- The most feared complication of dilatation is esophageal rupture. Other complications include hemorrhage and bacteremia.
- Management of corrosive esophageal strictures:
  - Acute phase management:
    - Neutralize with vinegar or citrus food if it is alkali ingestion (if pH of solution <11.5 then damage is less). With acids give antacids, milk, egg whites. Early endoscopy is needed to assess the severity and extent.
    - Emetics and sodium bicarbonate are avoided as they may precipitate perforation.
    - 1^{st} degree burns:
      - 48 hours observation
      - Oral feeds are started once patients swallows saliva painlessly
      - Regular follow-up endoscopy at 1^{st}, 2^{nd} and 8^{th} month. Stricture if formed can be identified by this time
    - 2^{nd} and 3^{rd} degree:
      - Fluid therapy, Antibiotics, nutrition, resuscitation, PPIs, aerosolized steroirds
      - Fiberoptic guided airway intubation if needed/tracheostomy, endoscopic esophageal stenting, feeding jejunostomy, laparoscopy or evaluation
      - Unstable patients have high mortality. Laparotomy is done in such patients. If esophagus and stomach shows full thickness necrosis, resection of these parts is done and end cervical esophagostomy with jejunostomy is done. When in doubt re-exploration for second look is done after 36-58 hours to assess the stomach.
  - Careful early gentle repeated endoscopy is mandatory
  - Though advocated often for 2-3 weeks, use of steroids is controversial and under debate.

- Antibiotics if there are chances of aspiration or perforation
- Regular esophageal dilatation is done for strictures. The stricture is dilated endoscopically using guidewire. Dilators are solid type with gradual increase in diameters. Often radiologic C-ARM guidance is needed to pass the guide wire into the stomach. Dilatation should be done up to a minimum 16 mm diameter. Pneumatic or balloon dilatation is also practiced. Gum elastic dilators, Savary-Gilliard dilators, balloon dilators are other dilators used. Earlier, blind dilatation using esophageal bougies of increased diameters was the practice which is followed even now in many places but changes of perforation are higher.
- Esophageal resection in corrosive strictures is technically difficult and may be hazardous. Esophageal bypass is better and easier and following later by regular endoscopic surveillance for malignant transformation (5%). Colon is used as replacing conduit as stomach itself may be diseased in corrosive pathology.
- In multiple strictures esophageal resection and colonic transposition may be advocated if risk of malignancy is considered.

- Malignance can develop in corrosive strictures (5%)

BARRETT'S ESOPHAGUS

ESOPHAGEAL HERNIA

ESOPHAGEAL DIVERTICULAE

PLUMMER VINSON

ACHALASIA CARDIA (CARDIOSPASM)

- Achalasia (From the Greek working meaning failure to relax) is a motility disorder characterized by failure of the lower portion of the esophagus to relax during swallowing.
- It is the most common primary esophageal motility disorder caused by inflammation of the myenteric plexus of Auerbach leading to fibrosis with decrease or loss of the myenteric ganglion cells and selective destruction of non-cholinergic, non-adrenergic inhibitory neurons. There is reduce nitric oxide and VIP (mediators of LES relaxation)
- The resulting dysphagia is due to 3 mechanisms:
  - Complete absence of peristalsis in the esophageal body
  - Incomplete/impaired relaxation of the LES after swallowing
  - Increased resting tone of the LES (>25mmHg)
- There is elevation of intraluminal esophageal pressure, esophageal dilation and subsequent progressive loss of normal swallowing mechanisms- a functional holdup of ingested material.
- Achalasia is due to loss of the ganglion cells in the myenteric (Auerbach's) plexus, the cause of which is unknown however in South America, chronic infection with the parasite *Trypanosoma cruzi* causes Chagas' disease which has marked clinical similarities to achalasia.

ETIOLOGY

- There is absence or less numbered ganglions in myenteric plexus
  - Stress
  - Vitamin B1 deficiency
  - Chaga's disease (*T. cruzi*)
  - Diffuse esophageal spasm (corkscrew esophagus)
  - Most commonly it is idiopathic and there is degermation of Auerbach's myenteric plexus along the entire length of the esophagus

PATHOLOGY

- The dilated esophagus contains few ganglion cells (contrast with Hirschsprung's disease of the colon where the dilated colon contains normal ganglion cells proximal to a constricted aganglionic segment).
- Histology of muscle specimens shows a reduction in the number of ganglion cells (and mainly inhibitory neurons) with a variable degree of chronic inflammation.

- The mucosa of the esophagus often shows esophagitis with mucosal ulceration secondary to bacterial proliferation.
- In 'vigorous achalasia' which may be an early stage of the disease there is inflammation and neural fibrosis but normal number of ganglion cells.
- In the early stages the esophagus is of normal caliber and still exhibits contractile (although non-peristaltic) activity. In some patients these uncoordinated contractions result in pain as much as a sense of food sticking.
- With time the esophagus dilates and contractions disappear, so that the esophagus empties mainly by the hydrostatic pressure of its contents. This is nearly always incomplete, leaving residual food and fluid. The gas bubble in the stomach is frequently absent as no bolus with its accompanying normal gas passes through the sphincter.
- The 'megaesophagus' becomes tortuous with a persistent retention esophagitis due to fermentation of food residues and this may account for the increased incidence of carcinoma of the esophagus.
- 'Pseudoachalasia' is an achalasia-like disorder that is usually produced by adenocarcinoma of the cardia but has also been described in relation to benign tumors at this level. It has been presumed that the inability of the LES to relax is linked to the loss of body peristalsis but other cancers outside the esophagus (bronchus, pancreas) have also been associated with pseudoachalasia.

CLINICAL FEATURES

- Most common in middle life (20-50 years) but can occur at any age.
- Typically presents as generally ill patient with dysphagia, although pain (often mistaken for reflux) is common in the early stages. Patients often present late and having had relatively mild symptoms remain untreated for many years.
- Dysphagia for both solids and liquids (there is dysphagia to liquids first). Initially it is intermittent, of variable severity and may be aggravated by emotional stress and cold liquids.
- Regurgitation of food is frequent and there may be overspill into the trachea, especially at night.
- Severe halitosis (due to the decomposition of stagnant food within the esophagus)
- Chest pain occurs in early stage.
- Classic triad of achalasia:
  - Dysphagia
  - Regurgitation

- Weight loss

- Leucoplakia is commonly encountered in advanced cases.
- Achalasia with diffuse esophageal spasm is called as 'vigorous achalasia'.
- GERD produces a sour taste and a gush of saliva due to the presence of hydrochloride from the gastric acid (water brash) while achalasia does not. Heartburn is common in 50% of patient.
- Walking while eating, chin thrusting, neck and shoulder extension, Valsalva maneuver facilitates emptying of food from the esophagus.
- Malnutrition, lung abscess formation, odynophagia and weight loss may also be present.

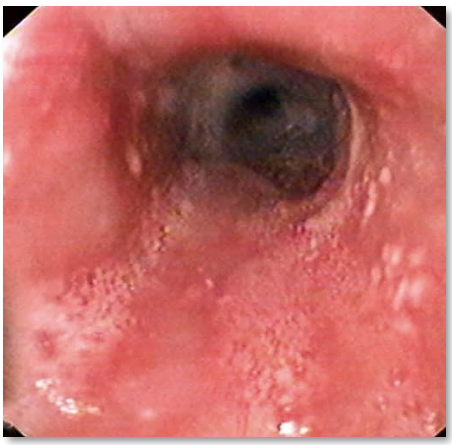


Figure 73: Endoscopic view of achalasia

STAGING

- I-proximal dilatation <4 cm
- II- dilatation between 4-6 cm
- III- dilation >6cm
- IV-sigmoid dilatation

INVESTIGATIONS

- Lateral upright chest X-ray: may reveal a dilated esophagus and the presence of air-fluid levels in the posterior mediastinum (double mediastinal strip of dilated esophagus). PA CXR may show patches of pneumonia.
- Endoscopy may show a tight cardia and food residue in the esophagus.
- Barium swallow is diagnostic and will show:
  - Pencil like smooth narrowing of the lower esophagus- bird's beak sign (rat tail sign/Steeple sign) due to the collection of contrast material in the proximal dilated segment and the passage of a small amount of contrast through the narrowed LES.
  - Grossly distended esophagus proximal to a tapering stricture at the distal end of the esophagus.
  - Absence of fundic gas bubble
  - Sigmoid esophagus or megaesophagus

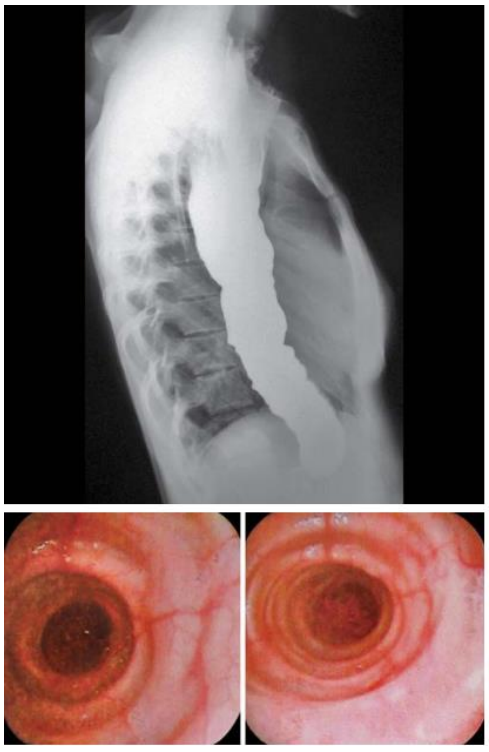


Figure 74: Sigmoid esophagus in achalasia cardia- Barium X-ray and endoscopic view (Image adapted from SRB's Manual of Surgery 4ed)

- The typical features of well-developed achalasia are often absent and endoscopy and radiology can be normal
- Esophageal motility study will confirm non-peristaltic contractions, incomplete LES relaxation and increased LES tone.
  - Manometry (Esophageal manometry): there is no peristalsis. There is raised resting pressure in the esophagus and the lower end does not relax on swallowing. This establishes a firm diagnosis.

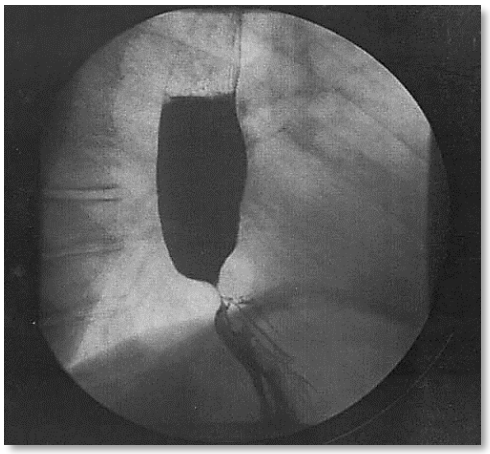


Figure 75: Barium study demonstrating a dilated esophagus and smooth, tapered gastroesophageal junction, typical of achalasia (Adapted from First Aid for the surgery Clerkship 2nd Ed)

- Esophagoscopy is indicated to rule out mass lesions or strictures and to obtain specimens for biopsy. Malignant tumors of the gastroesophageal junction can mimic the finding of achalasia (pseudoachalasia)

DIFFERENTIAL DIAGNOSIS

- Carcinoma esophagus
- Stricture esophagus
- Scleroderma



Figure 76: Barium swallow X-ray showing features of achalasia cardia (image adapted from SRB's Manual of Surgery 4th Ed)

MANAGEMENT

- Achalasia responds well to treatment.
- Medical management:
  - Drugs that relax the LES- nitrates, calcium channel blockers (nifedipine, verapamil) and antispasmodics. Calcium channel blockers are ineffective for long-term use however, sublingual nifedipine may be useful for transient relief of symptoms if definitive treatment is postponed.
  - Botulinum toxin (Botox): this is done by endoscopic injection into the LES. It acts by interfering with cholinergic excitatory neural activity at the LES. It binds to presynaptic receptors and irreversibly inhibits axonal acetylcholine release. Symptomatic improvement is obtained in 50% but the effect is not lasting. The effect is not permanent, and the injection usually has to be

repeated after a few months. For this reason, its use is restricted to elderly patients with other comorbidities.

- Surgical management: Esophagomyotomy (Heller's myotomy) with or without fundoplication is the treatment of choice for achalasia.
  - This involves cutting the muscle of the LES and cardia. The major complication is GERD and most surgeons therefore add a partial anterior fundoplication (Heller-Dor's operation). The procedure is ideally suited to minimal access laparoscopic approach and most surgeons use intraoperative endoscopy to judge the extent of the myotomy and to ensure that the narrow segment is abolished. It is successful in more than 90% of cases and may be used after failed dilatation.
  - Indications for Heller's cardiomyotomy:
    - Children and young adults
    - After failure of pneumatic dilation or botulinum toxin injection therapy
    - Patients with co-existent pathology requiring surgical intervention
    - Patient's preference
- Esophagomyotomy: Esopphagus is exposed via a transthoracic (left thoracotomy), transabdominal, thorascopic or laparoscopic technique. The tunica muscularis of the esophagus is incised distally with extension to the LES. Complete division of the LES necessitates the addition of an antireflux procedure such as Nissen 360^{o} fundoplication or partial fundoplication
- Endoscopic dilatation: has a lower success rate and a higher complication rate. It involves inserting a balloon or progressively larger sized dilators through the narrowed lumen which causes tearing of the esophageal smooth muscle and decreases the competency of the LES.
  - Note: Esophageal perforation is four times more likely following dilatation compared to esophagomyotomy.
  - With a 30-mm balloon, the incidence of perforation should be less than 0.5%. The risk of perforation increases with bigger balloons and they should be used cautiously for progressive dilatation over a period of weeks.
  - Forceful dilation is curative in 75-85% of cases.

COMPLICATIONS

- Risk of squamous cell carcinoma is as high as 10% in patients with long-standing achalasia (duration 15-25 years).
- Patients may also develop pulmonary complications: Aspiration pneumonia, Bronchiectasis, Asthma due to reflux and aspiration

ESOPHAGEAL PERFORATIONS

ESOPHAGEAL CARCINOMA

- Esophageal neoplasms are almost always malignant.
- Benign tumors account for fewer than 1% of cases and are usually leiomyomas or congenital cysts.
  - The only important benign tumor of the esophagus is leiomyoma, which has a predilection for the lower esophagus.
- The majority of benign tumors are small and asymptomatic but larger ones can project into the lumen of the esophagus and cause dysphagia. Symptomatic benign esophageal neoplasms are treated with local resection or enucleation.
- Esophageal carcinoma is the 6th most common cancer in the world accounting for 7% of all GI malignancies.
- Most malignant esophageal neoplasms seen worldwide are of squamous cell histologic types (90%) whereas adenocarcinoma histology predominates in industrialized nations.
  - The increasing prevalence of adenocarcinomas (due to Barrett's) as compared to what was mostly squamous cell carcinoma is shifting the epidemiology of esophageal cancer.
  - Other tumors of the esophagus are less common (leiomyomas, melanomas, carcinoids, lymphomas)
- Most cases of adenocarcinoma arise in the distal third or gastroesophageal junction (80%).
- Esophageal carcinoma is common in
  - Middle third (50%)
  - Lower third (33%)
  - Upper third (17%)
- Metastases are usually to liver, lung, and bones with at least 35% of patients with distant metastases at the time of diagnosis.
- Early diagnosis is only incidental or discovered on screening studies.
- Prognosis is poor with an overall 5-year survival rate of only 5%.
- In places with high soil nitrosamine content, the prevalence of esophageal cancer is almost 1% of adults. Esophageal cancer is more common in men (5:1) between 50 and 70 years of age. Whites are affected more.

RISK FACTORS

- Environmental
  - Tobacco
  - Alcohol
  - Food additives (nitrates in smoked and pickled meats)
  - Diet deficiency (Vitamin A, C, riboflavin)
  - Mycotoxin
- Esophageal disorders
  - Predisposition to squamous cell cancer
    - Achalasia
    - Plummer-Vinson syndrome
    - Esophageal webs
    - Strictures associated with lye ingestion and human papilloma virus infection
    - Chronic esophagitis
  - Predisposition to adenocarcinoma
    - GERD/Barrett's esophagus
- History of radiation therapy to the mediastinum
- Tylosis/Palmoplantar keratoderma (this is an inherited disease with thickening of the skin of palm and sole)
- Note: Vitamins (A, B12, C, E, folic acid and riboflavin) and trace elements (iron, zinc, selenium, and molybdenum) are thought to be protective.

PATHOGENESIS

- Mucosal insult seems to be a common pathway toward the genesis of esophageal cancer.
- Chronic ingestion of extremely hot liquids, esophageal burns from acid or base ingestions, radiation-induced esophagitis and reflex esophagitis are all implicated in causing esophageal cancer.
- Alcohol, smoking, nitrosamines, and malnutrition/vitamin deficiency also play a role in cancer development.
- Barrett esophagus which occurs when the normal squamous epithelium becomes columnar in response to injury from acid reflux, is considered a premalignant lesion.

- About 10% of patients with Barrett esophagus will develop adenocarcinoma. In patients with achalasia about 5% will develop squamous cell carcinoma of the esophagus.
- Because there is no serosal layer covering the esophagus, disease is often locally advanced or metastatic on presentation, leading to poor overall survival rates.

PATHOLOGY

- Squamous cell carcinoma is the common histological type world-wide.
- Adenocarcinoma especially of the lower esophagus and gastro-esophageal junction is commoner in the West
- Macroscopically the disease presents as:
  - Stenosing
  - Polypoid (fungating, protruded)
  - Ulcerative (excavated)
- Growth of esophageal cancer (squamous cell carcinoma or adenocarcinoma) occurs by intra-esophageal spread, direct extension and lymphatic or hematogenous metastases.
- Squamous cell carcinoma more typically invades adjacent structures compared with adenocarcinoma.
- Distant metastases are present in 25-30% of patients at the time of diagnosis.
- The liver, lungs and bones are the most frequent sites. Early cancer of the esophagus (confined to mucosa/submucosa) is only detected by screening. Both types spread to regional lymph nodes once the muscle coat is involved.
- Spread:
  - Direct:
    - Lack of serosal layer in esophagus favors local extension. In upper third it spreads through muscular layer and get adherent to left main bronchus, trachea, and left recurrent laryngeal nerve (Causes hoarseness), aorta or its branches (causes fatal hemorrhage but rare)
    - It may perforate and cause mediastinitis
  - Lymphatic:
    - It spreads both by lymphatic permeation and lymphatic embolization
    - It can cause satellite nodules elsewhere in the esophagus away from the main tumor
    - Above in the neck, it spreads to supravlavicular lymph nodes
    - In thorax, it spreads to paraesophageal, tracheobronchial lymph nodes to subdiaphragmatic lymph nodes

- In abdomen it spreads to coeliac lymph nodes

➢ Blood: spreads to liver, lungs, brain and bones

CLINICAL FEATURES

- At time of diagnosis most patients have advanced disease and are not surgical candidates for curative resection.
- The lack of serosal lining and rich submucosal lymphatic network of the esophagus allows early extension of tumor into adjacent mediastinal structures as well as early local lymph node spread.
- About 75% of patients have mediastinal or Extrathoracic lymph node metastases at the time of diagnosis
- Classic presentation of distal esophageal adenocarcinoma:
  - ➢ Old man with a history of GERD
  - ➢ Progressive dysphagia to solids initially and then to liquids (in 74% of patients, it is due to invasion of serosal layer). Note: Dysphagia does not usually develop until 60% of the esophageal lumen is obstructed.
    - Progressive dysphagia starts with meat, then other solids, then soft foods, eventually liquids and finally (in several months) saliva.
    - Esophageal obstruction results in malnutrition, weight loss, regurgitation and occasionally aspiration.
- Typically, patients feel well and have no other symptoms. Some may have
  - ➢ Non-specific GI complaints
  - ➢ Mild weight loss, easy fatigability and weakness (often related to diminished caloric intake resulting from obstructive symptoms than to cachexia from metastatic disease).
  - ➢ Chest pain
  - ➢ Odynophagia
  - ➢ Invasion of a recurrent laryngeal nerve causing hoarseness from vocal cord paralysis
  - ➢ Features of broncho-esophageal fistula in carcinoma of upper third of the esophagus (30%)
  - ➢ Substernal/abdominal pain
- Patients with esophageal squamous cell carcinoma often have a history of heavy alcohol and tobacco use and present with more pronounced symptoms owing to more advanced disease.

- Physical examination is non-specific and patients appear well unless if significant metastatic disease is present. Supraclavicular lymphadenopathy at presentation is rare.
- With advanced disease, the patient will appear cachectic, supraclavicular lymphadenopathy may be present as may signs of distant metastasis. May develop symptoms depending on local invasion (stridor, coughing, aspiration pneumonia, hemoptysis, vocal cord/recurrent laryngeal nerve paralysis).
  - Hoarseness of voice- recurrent laryngeal nerve palsy
  - Hiccough- phrenic nerve involvement
  - Horner's syndrome- sympathetic fibers involved
  - Supraclavicular lymph node enlargement
  - Distant mets e.g. spinal pain

DIAGNOSIS

- Diagnostic investigations:
  - Barium swallow: detects lesion in 96% of patients.
    - Barium swallow must precede the endoscopy to help prevent inadvertent perforation.
    - Findings range from small mucosal defects to "apple core" lesion to complete obstruction (Shouldering signs and irregular filling defect)
    - Irregular stricture with shouldering and moderate proximal dilation
  - Flexible Endoscopy and biopsy for definitive diagnosis. If Barrett esophagitis alone is noted, the extent of disease can be determined and biopsies performed to look for dysplasia or carcinoma in situ.
    - ¾ of adenocarcinoma is found in the distal esophagus, most Squamous cell carcinoma are found in the middle and lower third. The cervical esophagus is an uncommon site for disease.
  - Endoscopic ultrasound is used for primary tumor staging and regional lymph node status. Ultrasound depicts the normal five layers of the esophagus and can accurately determine the T status in 85% to 90% of patients. Fine needle aspiration biopsy of abnormal appearance lymph nodes under ultrasound guidance may be performed.
  - CT scan and PET scan is best at evaluation of regional and distant disease. Furthermore, CT scan assess operability but most cases can only get palliative (rather than curative) surgery.

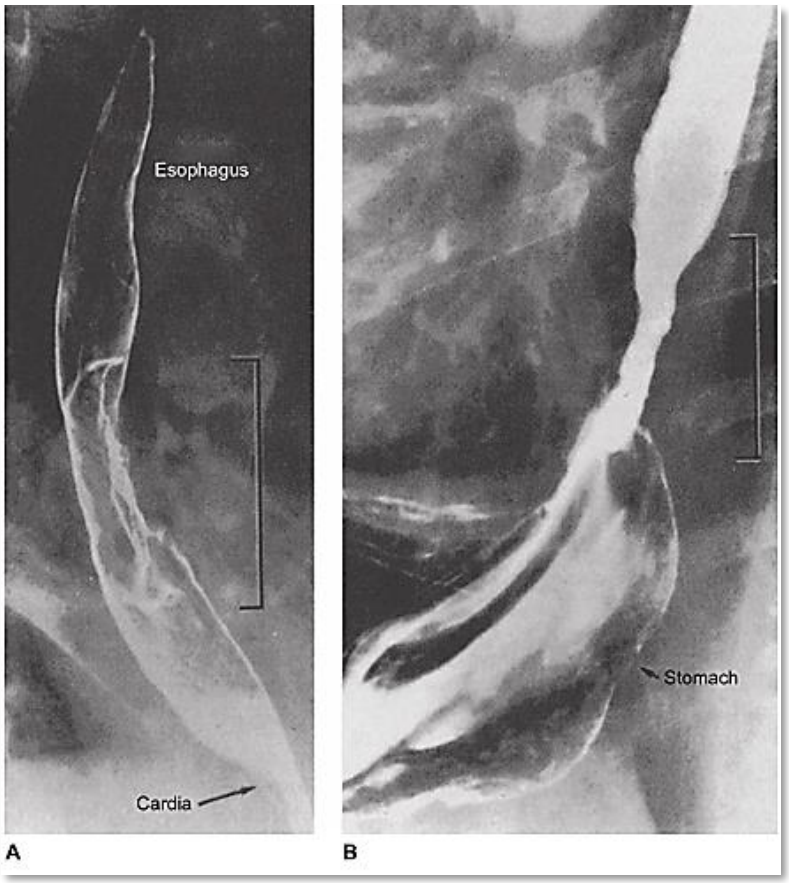


Figure 77: A: contrast radiograph showing the typical "Apple core" lesion of carcinoma of the middle 1/3 of esophagus. B: Ragged edge seen in carcinoma of the distal esophagus (Image from Blueprints Surgery)

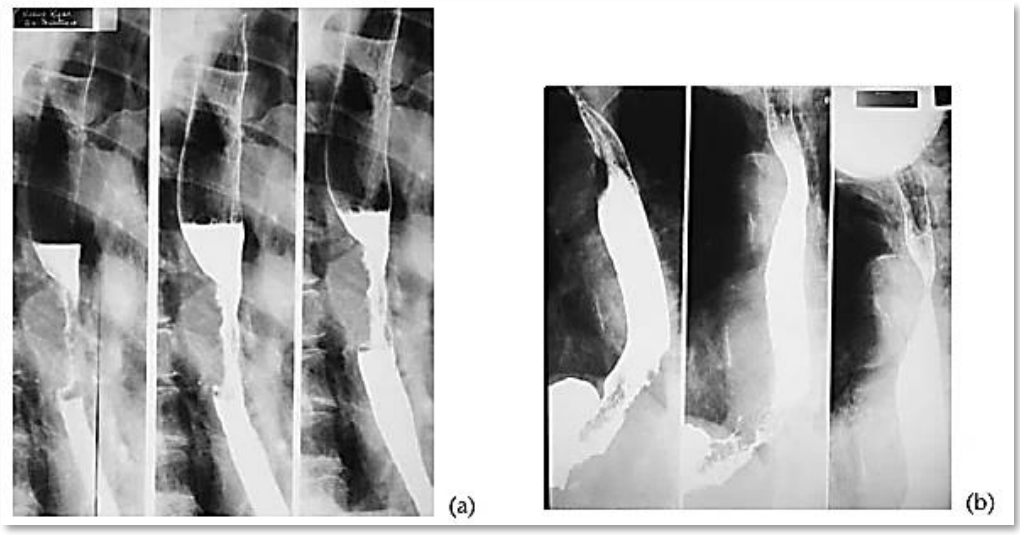


Figure 78: (A) Squamous carcinoma of the middle third of the esophagus. (B) adenocarcinoma of lower third of the esophagus

- Supportive: for general fitness and for procedures
  - Full blood count
  - Chest X-rays: to check for aspiration pneumonia
  - Abdominal ultrasound: to look for liver and lymph nodes status in abdomen
  - Liver function tests
  - Urea, electrolytes and creatinine
  - Blood group and cross match
  - Bronchoscopy is performed in proximal esophageal tumors to exclude bronchial involvement. This, together with vocal cord and phrenic nerve paralysis, involvement of mediastinal structures (especially aorta) and distant metastases, indicate inoperability.

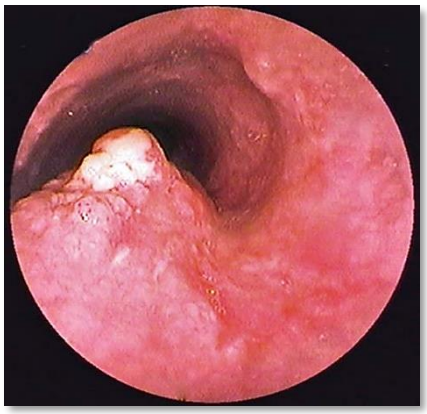


Figure 79: Carcinoma esophagus (Endoscopic view)

STAGING: TMN STAGING

Primar tumour (T)			
TX	Primary tumour cannot be assessed		
T0	No evidence of primary tumour		
Tis	Carcinoma *in situ*		
T1a	Tumour invades lamina propria		
T1b	Tumour invades submucosa		
T2	Tumour invades muscularis propria		
T3	Tumour invades adventitia		
T4	Tumour invades adjacent structures		
Regional lymph nodes (N)			
NX	Regional lymph nodes cannot be assessed		
N0	No regional lymph node metastasis		
N1	Regional lymph node metastasis		
Distant metastasis (M)			
MX	Presence of distant metastasis cannot be assessed		
M0	No distant metastasis		
M1	Distant metastasis		
Stage grouping			
Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage IIA	T2	N0	M0
	T3	N0	M0
Stage IIB	T1	N1	M0
	T2	N1	M0
Stage III	T3	N1	M0
	T4	Any N	M0
Stage IV	Any T	Any N	M1

MANAGEMENT

- Treatment depends on the stage of the disease and the condition of the patient.
- Some patients with resectable lesions are unfit for surgery by virtue of significant comorbid disease. The nutritional state of the patient must also be considered and assessed. In malnourished patients, a period of enteral nutrition is undertaken for a few weeks before surgery.
- The goals of surgical treatment are removal of the tumor and regional lymph nodes followed by re-establishment of gastrointestinal tract continuity.
- Treatment of patients who are unfit or have locally advanced disease is by chemoradiation. This is followed by restaging. Surgery is considered in fit patients whose disease is downstaged by the treatment.
- Management of localized disease/early stage disease
  - Surgical resection, especially for symptoms control (right thoracic or transhiatal approach, gastric pull-up or colonic interposition can be used to reconstruct the GI tract). Note: essophagectomy provides the possibility of cure.
    - Proximal extent of resection should be 10cm above macroscopic tumor and distal extent of resection is 5cm from macroscopic distal end of tumor. Proximal stomach has to be removed commonly especially in lower 1/3rd of tumor. Sufficient removal of contiguous structures may be needed in curative resection.
  - Radiotherapy for avoidance of perioperative morbidity and mortality (can shrink tumor but may predispose to local complications and not palliate dysphagia and odynophagia)
  - Pre/postoperative chemotherapy to downstage patients with resectable bulky tumors.
  - Combination therapy of these three modalities is becoming increasingly common. Studies have shown that they can improve long term survival however there remains controversy.
  - Various operations with varying degrees of invasiveness have been developed for treating esophageal cancer. In deciding which procedure to perform the surgeon must take into consideration the location of the tumor, the proposed esophageal substitute and the physical condition and body habitus of the patient.

- The most common surgical approaches are transthoracic (thoracotomy) and transhiatal without thoracotomy. Incisions may be required in the neck, chest and abdomen.
- The most common transthoracic esophagectomy is one called the Ivor Lewis Esophagectomy (named after Welsh surgeon that developed the procedure) that involves upper midline laparotomy with mobilization of the stomach and is followed by right thoracotomy with esophageal resection and anastomosis. It is useful for patients with tumors in the middle and lower esophagus.
- For tumors of the upper esophagus, many surgeons use a modified approach called McKeown modification which involves additional intrathoracic esophageal mobilization and anastomosis in the right neck through a separate cervical incision (three hole technique). The tubularized stomach is used as the esophageal substitute in most cases. Other possible substitutes are the colon and small bowel.
- Esophagectomy without thoracotomy is termed the transhiatal approach. This technique uses laparotomy for gastric and esophageal mobilization, followed by left cervical incision for the anastomosis. The major benefit of this approach is avoiding the complications that arise from thoracotomy.
- With advances in medicine, Esophagectomy can now be performed using laparoscopic and thoracoscopic techniques. A cervical incision is also used. This is complex and challenging procedure that has a steep learning curve. Early results of laparoscopic esophagectomy seem to compare favorably with open surgery though long-term data isn't available at the moment.
- Post-operative management
  - Fluid and electrolyte management
  - Antibiotics and proper analgesia
  - Respiratory care, ICT care, physiotherapy
  - Prevention of DVT-elevation, exercises, heparin
  - Monitoring for bleeding, sepsis, leak, oxygen saturation
  - Total parenteral nutrition during initial postoperative period and early jejunostomy feeding for nutrition

- Treatment for advanced stage IV disease
  - Chemotherapy to promote tumor shrinkage
  - Palliate care for symptoms as survival rate is poor nonetheless
- Postoperative complications
  - Fistulae
  - Abscesses

- Respiratory complications

- Other options include endoscopic laser therapy, endoscopic dilation and stent placement or placement of a gastrotomy or jejunostomy.
- Non-operative treatment
  - Radiotherapy:
    - This is done by super-voltage external beam radiotherapy may be curative (radical) or palliative to relieve dysphagia and metastatic bone pain.
    - It can also be given as an adjunct to surgical treatment either in the form of multimodality treatment or after esophagectomy to improve locoregional control.
    - Contraindications to radical radiotherapy:
      - Large tumors (>9.0 cm)
      - Presence of tracheal broncho-esophageal fistula
    - The min disadvantages of radical radiotherapy are the development of a fibrous stricture in half the patients treated.
    - Brachytherapy (intracavity irradiation) with caesium or iridium pellets loaded into an applicatr and placed in the lumen of the esophagus is another technique used for palliation of dysphagia.
  - Chemotherapy:
    - Several agents are used in combination treatment regimens for esophageal cancer.
    - These include: 5-fluorouracil (5-FU), Cisplatin, Vindesine, mitomycin C, paclitaxel and etoposide.
    - Paclitaxel achieves a high response rate in metastatic esophageal cancer and also acts as a radiation sensitizer. Most combination regimens have 5-FU with or without leucovorin. The second most common agent used in combination regimens is cisplatin.
- Palliation of advanced esophageal cancer:
  - It is directed to relief dysphagia
  - Several methods are used:
    - Recanalization:
      - Photocoagulation using Nd:YAG laser together with dilatation.
      - Photodynamic ablation using hematoporphyrin derivative followed by irradiation with red light (wavelength 630 nm): this combination causes necrosis of tumor by highly reactive species such as singlet oxygen.

- Endoscopic guided fulguration of advanced esophageal neoplasm with the BICAP probe
- Intratumor injection of absolute alcohol under endoscopic control: used as adjunct to stenting to deal with tumor overgrowth/ingrowth

○ Intubation:

- Most widely used method for palliation of dysphagia in inoperable esophageal caner.
- Main disadvantage: treatment is performed in a single session, with dramatic restoration of swallowing.
- Plastic tubes have been replaced by self-expanding metallic stents as these provide excellent palliation and are relatively easy to insert.
- Several types are available. Some patients develop a malignant tracheo-esophageal fistula (spontaneously or after radiotherapy) that can be sealed with covered self-expanding metallic stents.

➢ Surgical bypass is seldom used nowadays.
➢ Gastrotomy should not be done as a palliative procedure.

- Indications for palliative therapy
  - ➢ Patient not fit for major surgery
  - ➢ Metastasis via blood, to adjacent organ or peritoneal/liver
  - ➢ Relieve pain
  - ➢ Relieve dysphagia
  - ➢ Prevent bleeding
  - ➢ Prevent aspiration

Complications of oesophagectomy

- 5-10% mortality
- Haemorrhage
- Respiratory infection, often severe
- Septicaemia
- Chylothorax, injury to thoracic duct
- Anastomotic leak—thoracic leak is most dangerous (5-10%)
- Hoarseness due to recurrent laryngeal nerve palsy
- Stricture formation (40%)
- Gastro-oesophageal *reflux* in gastric transfer
- *Conduit necrosis* due to ischaemia either stomach or colon
- *Colonic dysmotility* causing partial obstruction in colon transfer

ABDOMINAL SURGICAL CONDITIONS

ABDOMINAL MASSES

HISTORY

- Most patients with a lump feel it frequently and should be able to tell you about the history of the clinical features such as:
  - When the lump was first noticed?
    - It is important to be precise with dates and terminology. Do not write "the lump first appeared 6 months ago", when in fact you mean "the lump was first noticed 6 months ago." Many lumps may exist for months, even years before being noticed.
  - What made the patient notice the lump?
    - The presence or absence of pain is important particularly if it is the presenting feature. In general terms, pain is usually associated with inflammation, not neoplastic change.
  - What are the symptoms of the lump?
    - Describe the history of each symptom carefully.
  - Has the lump changed since it was first noticed?
  - Does the lump ever disappear?
    - A lump may disappear on lying down or during exercise and yet be irreducible at the time of your examination.
  - Has the patient ever had any other lumps?
  - What does the patient think caused the lump?
    - Lumps occasionally follow injuries or systemic illnesses known only to the patient.
- In a patient presenting with an abdominal mass, the following features should also be assessed carefully:
  - Pain: site, nature, aggravating or relieving factors, duration of pain, referred pain.
  - Vomiting: type, content, hematemesis, relation to food frequency.
  - Bowel habits: constipation, diarrhea, bloody diarrhea, furious diarrhea, tenesmus (straining to defecate without the ability to do so).
  - Decreased appetite and weight

- Jaundice: it is an important factor in relation to liver, gallbladder or pancreatic masses.

PHYSICAL EXAMINATION

INSPECTION

- Anatomical location: describe in exact anatomical terms using distance measured from bony points.
- Size: measure dimensions using tape, asymmetrical lumps will need more measurements to describe them accurately, sometimes a diagram will clarify your written description.
- Shape: use descriptive terms such as 'pear-shaped or kidney shaped'
- Surface and margins: is it smooth, bumpy, regular or irregular
- Movement with respiration

PALPATION

- Extent and borders: the edge of a lump may be clearly defined or indistinct
- Surface: it may be discolored and become smooth and shiny or thick and rough.
- Temperature
- Tenderness: Is the lump tender? Which parts are tender? Always try to feel the non-tender part before feeling the tender area, and watch the patient's face for signs of discomfort as you palpate
- Composition:
  - Consistency:
    - Stony hard (not indentable)- usually bone or calcification
    - Firm: hard but not as hard as bone
    - Rubbery: slightly squashable similar to a rubber ball.
    - Spongy: soft and very squashable but still with some resilience
    - Soft: squashable and no resilience
  - Fluctuation: pressure on one side of a fluid filled cavity makes all the other surfaces protrude. This is elicited by feeling at least 2 other areas of the lump whilst pressing on a third.
- Translucence (transillumination):
  - Transilluminate: contains water, serum, lymph or plasma or highly refractile fat
  - Opaque: blood and other fluids
- Mobility
- Pulsatility, compressibility and reducibility

- Plane of swelling (by leg raising test)
- Presence of other masses
- Often mass needs to be examined for change of position- in sitting, in standing, in side position, after a brisk walk, in knee elbow position from retroperitoneal mass and for puddle sign (but difficult to keep patient in this position)

PERCUSSION

- Generally:
  - Solid and fluid filled lumps sound dull
  - Gas filled lumps sound hollow and resonant
- Percussion over an abdominal mass however is important in predicting the anatomical location of the mass.
  - If mass is dull then it is in the anterior abdominal wall or in front of the bowel intra-abdominally like liver, spleen, gallbladder.
  - If the mass is with an impaired resonant note, then the mass is arising from the bowel like stomach, colon, small bowel.
  - If the mass is resonant on percussion, then the mass is probably in the retroperitoneal region.
- Check for fluid thrill over the lump as well as in the abdomen.



Percussion over the mass is absolute need to find out the plane of the mass. Anterior masses are dull on percussion; posterior masses are resonant and mass from bowel has impaired resonance.

AUSCULATATION

- Listen for any bruits and for bowel sounds

VAGINAL AND RECTAL EXAMINATION

- Per vaginal examination is done to assess pelvic masses.

- Per rectal examination is done to look for any secondaries in rectovesical pouch, primary tumor or relation of lower abdomen masses (pelvic masses)
- Do not forget to do a general examination and assessment of the lymph nodes (Especially regional).

ABDOMINAL TRAUMA

GASTRITIS AND PEPTIC ULCER DISEASE

- The stomach is divided into:
  - Fundus
  - Body
  - Pyloric region (antrum and pyloric canal)
- The stomach acts as a receptacle for ingested food which is partially digested and hydrolyzed by acid and pepsin into semifluid chyme and then delivered into the small intestine.
- The stomach possesses the property of adaptive relaxation i.e. increase in capacity without a significant rise in intraluminal pressure.
  - Adaptive relaxation is mediated by stretch reflexes and vagal afferents and is therefore greatly impaired after gastric surgery.
- The mucosa lining the proximal stomach contains the parietal and chief cells whereas the mucosa lining the more muscular antropyloric segment secretes an alkaline mucus but contains specialized endocrine (G) cells that release gastrin under the influence of gastrin releasing peptide.
  - Parietal cells secrete acid and intrinsic factor
  - Chief cells produce pepsinogens that are then converted to active pepsin by HCL.

GASTROINTESTINAL BLEEDING

GASTRIC TUMORS

ACUTE ABDOMEN

- Acute abdomen is defined as an abrupt onset of abdominal pain usually accompanied by 1 or more peritoneal signs:
  - Rigidity
  - Tenderness (with or without rebound)
  - Involuntary guarding
- Most causes are surgical and considered as EMERGENCIES.
- When emergency surgical intervention is required it is also termed surgical abdomen.
- The pain may be due to inflammation, perforation, obstruction, ischemia, infarction or rupture of an abdominal organ.
- Recall when assessing pain use SOCRATES: Site, Onset, Character, radiation, alleviating factors, Timing course, Exacerbating factors and Associated symptoms

HISTORY

- Should include demographic details, occupation, recent travel, history of recent abdominal trauma
- Pain is the most common presenting feature.

SITE

- Visceral pain:
  - Poorly localized, usually dull, achy pain arising from distension or spasm in hollow organs e.g. crampy pain felt during early intestinal obstruction.
  - Mid-epigastrium: Stomach, duodenum, hepatobiliary system, pancreas
  - Mid-abdomen: jejunum, ileum

- Lower abdomen: colon, internal reproductive system

- Parietal pain:
  - Sharp, well-localized, somatic pain arising from irritation (usually by pus, bile, urine or gastrointestinal secretions) of the parietal peritoneum. E.g. inflamed appendix causing sharp right lower quadrant pain due to irritation of nearby peritoneum.

ONSET

- Is usually abrupt.

CHARACTER

Character of pain	Causes
Gradual steady pain	• Acute cholecystitis • Acute cholangitis • Hepatic abscess • Diverticulitis
Intermittent colicky pain	• Small bowel obstruction • Inflammatory bowel disease • Biliary colic
Abrupt excruciating pain	• Perforated ulcer • Ruptured aneurysm • Ureteral colic
Rapid onset severe constant pain	• Acute pancreatitis • Ectopic pregnancy • Mesenteric ischemia • Strangulated bowel • Acute appendicitis

RADIATION

- Biliary tract pain may radiate to the right shoulder or right scapula (due to right hemidiaphragmatic irritation).
- Splenic rupture pain may radiate to left shoulder.
  - Kehr's sign: pain referred to the left shoulder due to irritation of the left hemidiaphragm. Often seen with splenic rupture.
- Kidney pain may radiate from flank to groin and genitalia (loin to groin)
- Pancreas pain may radiate to the back.

ALLEVIATING AND AGGRAVATING FACTORS

- Include:
  - Change in position
  - Association with food (better or worse)
  - Pain that wakes one from sleep (significant)
- If worsened by movement/coughing suspect active peritonitis, pancreatitis is relieved by sitting forward

ASSOCIATED SYMPTOMS

- Vomiting: pain relieved by vomiting is indicative of small bowel obstruction, afferent loop syndrome. Vomiting undigested food or bile suggests upper GI pathology or obstruction while feculent vomiting suggests lower GI obstruction
- Hematemesis or melena
- Bowel habits: constipation or obstipation (no stool or flatus) is suggestive of bowel obstruction. Stool/urine color
- Mucoid diarrhea with blood (red currant jelly stool) is seen in intussusception.
- Anorexia: very non-specific symptom, however most patients with acute appendicitis will have anorexia.
- Fever: seen in appendicitis, acute cholecystitis and acute pancreatitis.

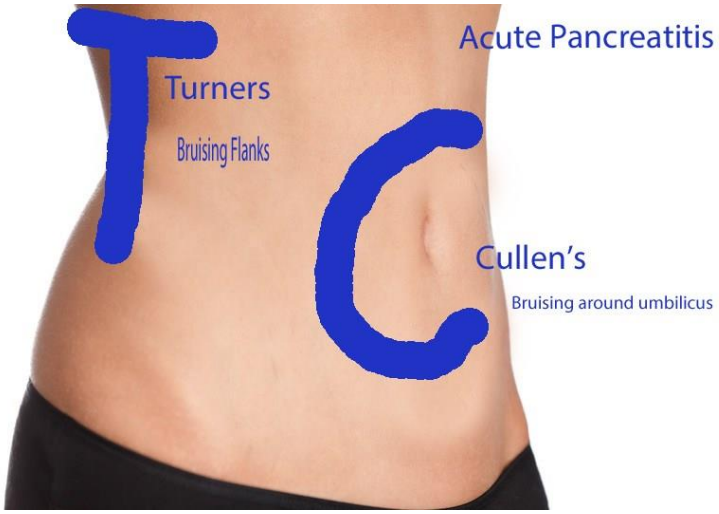
MEDICAL AND SURGICAL HISTORY

- History of past abdominal surgery (Small bowel obstruction due to adhesions)
- Atrial fibrillation: increased risk for mesenteric ischemia (embolism) because of emboli to mesenteric arteries.
- Menstrual and sexual history: Acute salpingitis vs Pelvic inflammatory disease Vs rupture ectopic
  - Contraception (including IUCD use)
  - Last menstrual period
  - Previous gynecological or tubal surgery
  - Previous ectopic pregnancy
  - Vaginal bleeding

PHYSICAL EXAMINATION

- Steps in physical examination of acute abdomen:

1. Inspection
2. Auscultation
3. Cough tenderness
4. Percussion
5. Guarding or rigidity
6. Palpation:
    - One-finger
    - Rebound tenderness
    - Deep palpation
7. Punch tenderness
    - Costal area
    - Costovertebral area
8. Special signs e.g. Murphy's sign in cholecystitis
9. External hernia and male genitalia
10. Rectal and pelvic examination



- Look out for:
    - Vital signs: most patients with a surgical abdomen will have vital sign abnormalities secondary to pain, inflammation, fluid and electrolyte derangements, and anxiety
    - General: hydration status, mentation, nutritional status
    - Chest: auscultation
    - Abdominal examination (as above)

Definitions

- **Cullen's Sign** – Irregular hemorrhagic patches around the umbilicus
- **Grey Turner Sign** – Bilateral flank bruising or ecchymosis. A classic finding of bleeding into the retroperitoneum around the kidneys and pancreas.
- **Kehr's Sign** – Referred pain in the L shoulder r/t irritation of the adjacent diaphragm
- **FAST** – Focused Assessment with Sonography in Trauma - Identify free fluid (usually blood) in the peritoneal, pericardial, or pleural spaces









Condition	Helpful signs
Perforated viscus	• Scaphoid tense abdomen • Diminished bowel sounds (late) • Loss of liver dullness • Guarding or rigidity
Peritonitis	• Motionless • Absent bowel sounds (late) • Cough and rebound tenderness • Guarding or rigidity
Inflamed mass or abscess	• Tender mass (abdominal, rectal or pelvic) • Punch tenderness • Special signs (Murphy's, psoas or obturator)
Intestinal obstruction	• Distended abdomen • Visible peristalsis (late) • Hyperperistalsis (early) or quiet abdomen (late) • Diffuse pain without rebound tenderness • Hernia or rectal mass (some)
Paralytic ileus	• Distention • Minimal bowel sounds • No localized tenderness

Ischemic or strangulated bowel	• Not distended (until late) • Bowel sounds variable • Severe pain but little tenderness • Rectal bleeding (some)
Bleeding	• Pallor • Shock • Distention • Pulsatile (aneurysm) • Tender (e.g. ectopic pregnancy), mass • Rectal bleeding (some)

- Note: Serial abdominal exams and observation may be necessary in cases in which etiology of abdominal pain is initially unclear.

INVESTIGATIONS

- Blood:
  - Full blood count
  - Urea, creatinine and Electrolytes
  - Serum amylase and lipase
  - Liver function tests for right upper quadrant pain
  - Serum glucose
  - Clotting panel
  - Blood culture
  - Malaria slide
  - Group and save/Cross match
- Imaging
  - Electrocardiography (ECG): to rule out myocardial infarction (also do cardiac enzymes if ST-T changes seen) and also as a pre-operative baseline cardiac assessment
  - Chest X-ray and Abdominal X-ray to look for free air (can detect as little as 1-2ml): easier to see under right hemidiaphragm. Presence of stomach bubble obscures it on the left.
  - Abdominal ultrasound

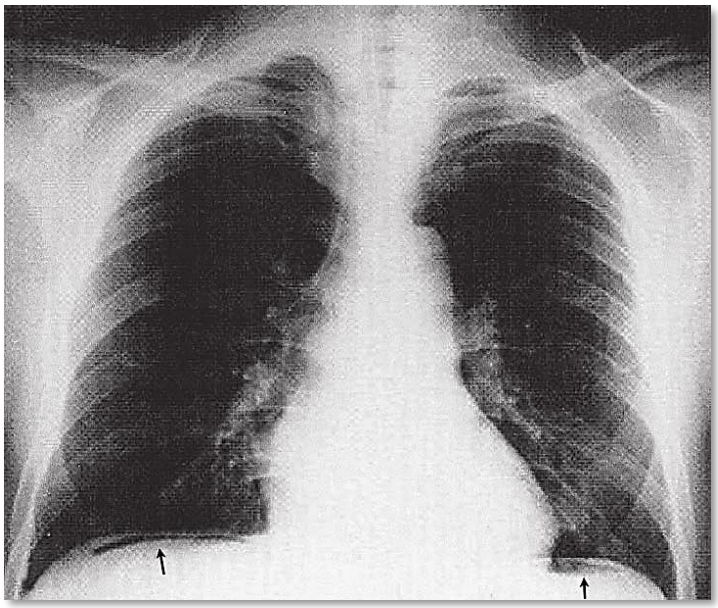


Figure 80: Erect CXR showing free air under both hemidiaphragms (Arrows)

- Abdominal computed tomography (CT) should be used after the above assessment is complete and the diagnosis remains elusive (e.g. young male with clinical signs and symptoms of appendicitis should not undergo CT).
  - Contrast for abdominal CT: for the most optimal imaging both oral and IV contrast is used. In some cases, (such as impaired renal function or allergy to IV contrast) this is not feasible.
  - Non-contrast CT although suboptimal for most cases (Except nephrolithiasis) still provides a lot of information.
- Urine: Gravindex for all women of childbearing age

MANAGEMENT

- Dependent on the cause.
- Early diagnosis improves outcome.
- Key is deciding whether surgical intervention is needed (Exploratory laparotomy)
- Indications for urgent surgical interventions:
  - Physical findings
    - Involuntary guarding or rigidity especially if spreading
    - Increasing or severe localized tenderness
    - Tense or progressive distention
    - Tender abdominal or rectal mass with high fever or hypotension
    - Rectal bleeding with shock or acidosis
    - Equivocal abdominal findings with
      - Septicemia (high fever, marked or rising leukocytosis, mental changes or increasing glucose intolerance in a diabetic patient)
      - Bleeding (unexplained shock or acidosis, falling hematocrit)
      - Suspected ischemia (acidosis, fever, tachycardia)
      - Deteriorating on conservative treatment
  - Radiological findings
    - Pneumoperitoneum
    - Gross or progressive bowel distention

- Free extravasation of contrast material
- Space-occupying lesion on scan with fever
- Mesenteric occlusion on angiography

➢ Endoscopic findings
- Perforated or uncontrollably bleeding lesion

➢ Paracentesis findings
- Blood, bile, pus, bowel contents or urine

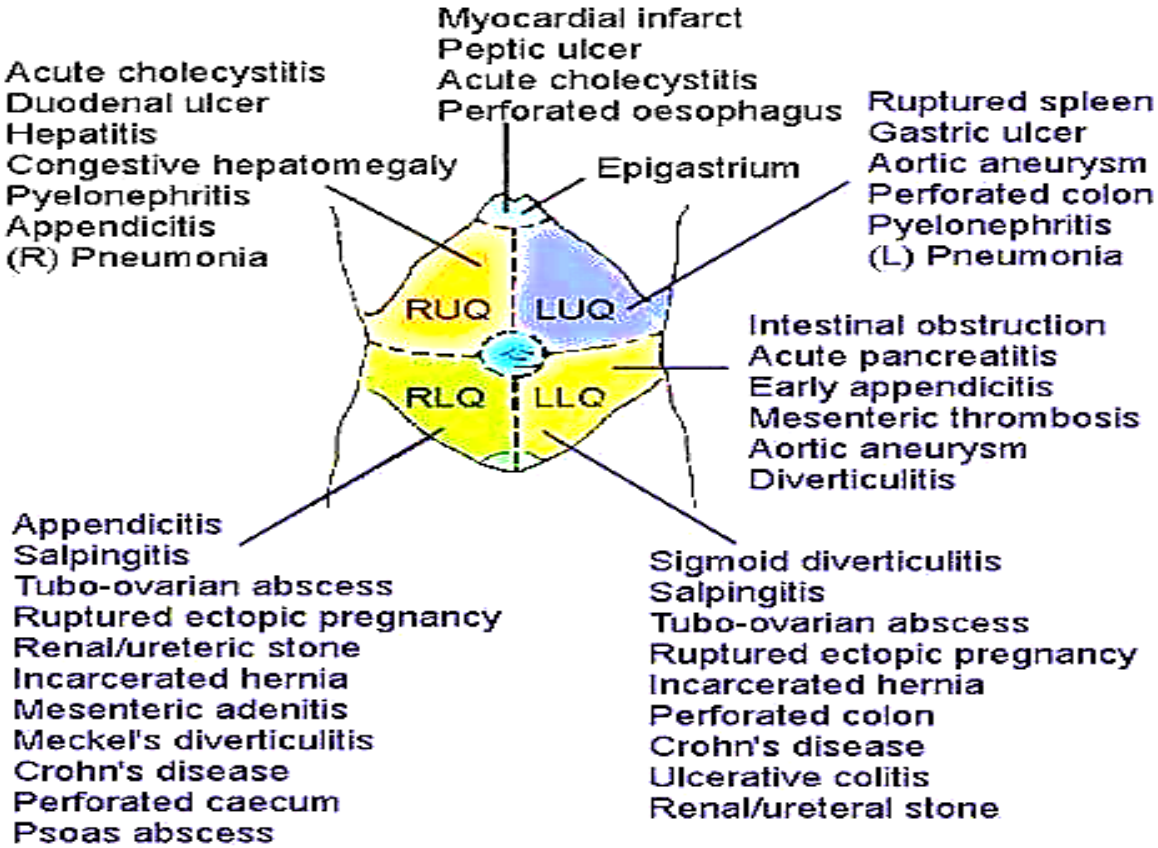
SURGICAL CAUSES OF ABDOMINAL PAIN

- Can be divided into infectious, inflammatory, intoxication, ischemic, malignant, metabolic, obstetric and gynecological, traumatic.
- It may also be classified according to the site.

Site	Examples
Right upper quadrant	• Perforated duodenal ulcer • Acute cholecystitis • Hepatic abscess • Retrocecal appendicitis • Appendicitis in a pregnant woman (commonest surgical emergency in a pregnant woman)
Right lower quadrant	• Appendicitis • Cecal diverticulitis • Meckel's diverticulitis • Intussusception
Left lower quadrant	• Sigmoid diverticulitis • Volvulus
Left upper quadrant	• Splenic rupture • Splenic abscess
Diffuse	• Bowel obstruction • Leaking aneurysm • Mesenteric ischemia
Periumbilical	• Early appendicitis • Pain from small bowl obstruction
Suprapubic	• Ectopic pregnancy

	• Ovarian torsion • Tubo-ovarian abscess • Psoas abscess • Incarcerated groin hernia

- Some important non-surgical causes of abdominal pain include:
  - Myocardial infarction
  - Mittelschmerz (mid-cycle pain due to ovulation)
  - Poisoning (lead, black widow spider)
  - Herpes zoster
  - Lower lobe pneumonia
  - Endocrine: Addisonian crisis, diabetic ketoacidosis
  - Sickle cell crisis
  - Porphyria
  - Psychological (hysteria)



INTESTINAL OBSTRUCTION

- The small intestine is divided into:
  - Duodenum: the first part
  - Jejunum: the second part
  - Ileum: the terminal part
- The lining mucosa is folded into villi to increase absorptive surface and these are covered by tall columnar absorptive cells (enterocytes) that have microvilli at their luminal surfaces (brush border).
- Goblet cells found interspersed among the enterocytes, synthesize the mucinous glycoprotein essential for maintaining the surface glycocalyx.
- At their bases, the villi are surrounded by intestinal crypts that contain stem cells from which the surface epithelium is constantly replaced by a process of mitosis and migration.
- The primary function of the small intestine is the absorption of nutrients but it is also involved in digestion by brush-border enzymes that are essential for the final hydrolysis of peptides and carbohydrates.
- The digestion and absorption of fluid, electrolytes, iron, folate, carbohydrate, fat and protein is completed in the jejunum.

- The absorption of bile salts and vitamin B12 only occurs in the terminal ileum which contains specific transport sites.
- The critical length of small bowel required for maintenance of oral nutrition is 100cm. Below this length some form of parenteral nutritional support is needed.
- Another function of the small intestine is the synthesis of peptide and amine intestinal hormones. These are located within the enteroendocrine cells and in the neurons of the myenteric plexus. They influence intestinal secretion and transport, growth and differentiation, splanchnic hemodynamics and the release of insulin.
- The small bowl microflora plays an important role in intraluminal metabolism of various substances. Obstruction and stasis increases bacterial population substantially. Even in the absence of obstruction this bacterial overgrowth may produce adverse consequences including malabsorption.
- The normal intestinal mucosa is able to resist invasion by pathogenic bacteria but this antimicrobial barrier can break down under pathological states with translocation of pathogens into blood and lymph causing a systemic inflammatory response syndrome.

INTESTINAL OBSTRUCTION

- Intestinal obstruction happens when there is restriction to the normal passage of intestinal contents. The obstruction may be complete (total blockage of the lumen) or incomplete (partial blockage)
- It may be divided into 2 main groups:
  - Paralytic (adynamic)
  - Mechanical (dynamic)
- The most common causes of intestinal obstruction are adhesions and hernias.

CLASSIFICATION

- Depends on:
  - Etiology (Cause):
    - Dynamic
    - Adynamic

- Speed of onset:
  - Acute: common in small bowel. It has a Rapid onset with severe symptoms
  - Chronic: Insidious (gradual, subtle presentation over weeks) E.g. Most cases of carcinoma of large bowel
  - Acute on chronic: common in large bowel. It is a chronic obstruction that suddenly becomes complete

- Site of obstruction
  - High- small bowel obstruction
  - Low- large bowel obstruction

- Nature:
  - Simple- bowel is occluded without damage to its blood supply
  - Strangulated- blood supply of the involved segment of intestine is cut off e.g. strangulated hernia, volvulus, intussusception or adhesive band. Gangrene may occur if untreated.

- Whether they are congenital or acquired
  - Congenital:
    - Anorectal malformation e.g. imperforate anus
    - Volvulus neonatorum
    - Meconium ileus
    - Hirschsprung disease
    - Mal-rotation
    - Bands and adhesions
    - Intestinal atresia
    - Duodenal atresia
    - Adhesions
    - Congenital megacolon
  - Acquired:
    - Hernia (most common)
    - Postoperative
    - Intussusception
    - Roundworm
    - Gallstones
    - Tuberculosis

- Malignancy
- Internal hernias

ETIOLOGIC CLASSIFICATION

- These are either:
  - Dynamic: due to a mechanical obstruction (peristalsis against a mechanical obstruction). Can have acute or chronic form. They various causes may be classified as:
    - Extramural
    - Intramural
    - Intraluminal
  - Adynamic: peristalsis may be absent e.g. paralytic ileus or it may be present in a non-propulsive form e.g. mesenteric vascular occlusion or pseudo-obstruction.

Causes of intestinal obstruction	
Dynamic	Adynamic
Intraluminal • Fecal Impaction • Foreign bodies • Food bolus • Parasites • Pedunculated tumor • Bezoars (bolus composed of indigestible material-orange pith or hair bezoar, particularly in gastrectomized patients) • Gallstones 'ileus': Large gallstones in a chronically inflamed gallbladder that has become adherent to the duodenum erodes through the 2 organs by a process of pressure necrosis, thus entering the duodenum and becoming impacted lower down the small intestine usually in the small terminal ileum.	• Paralytic ileus • Mesenteric vascular occlusion • Pseudo-obstruction
Intramural	

• Strictures (TB strictures) • Tumors: carcinomas, lymphomas etc. • Congenital atresia • Diverticulitis • Inflammatory disease: Crohn's disease	
Extramural • Bands/ adhesions (commonest cause) • Hernia: External and internal • Volvulus • Intussusception • Compression by tumors (nodal tumor deposits)	

DYNAMIC OBSTRUCTION

- These are due to mechanical blockage of normal propulsion and passage of intestinal contents.
- It may occur in an acute or chronic form.
- Causes of dynamic intestinal obstruction are classified:
  - In the lumen (intraluminal)

    - Fecal impaction
    - Foreign bodies
    - Food bolus
    - Gallstones
    - Roundworm (parasites)
    - Meconium ileus
    - Pedunculated tumors
  - In the wall (intramural):
    - Tuberculous stricture
    - Crohn's disease
    - Malignancy
    - Congenital atresia
  - Outside the wall (Extramural):
    - Adhesions-40%
    - Hernia-25%
    - Volvulus
    - Intussusception

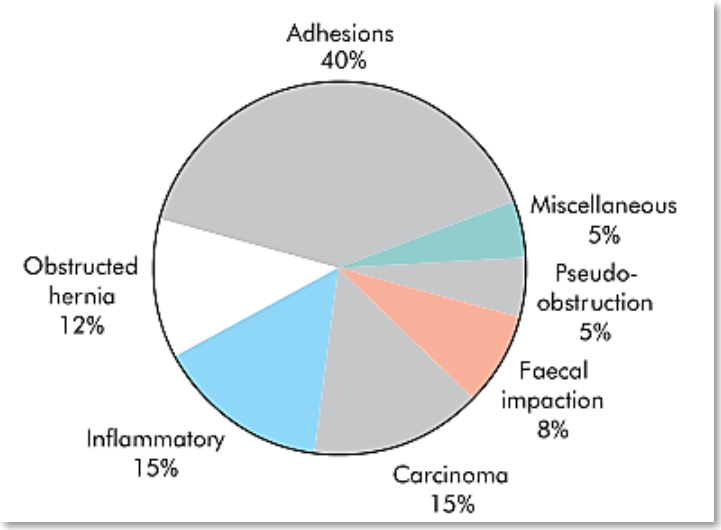


Figure 81: Different causes of intestinal obstruction

- Adhesions commonly cause small bowel obstruction than large bowel obstruction.
  - 80% of intestinal obstruction occurs in small bowel.
  - 20% occur in the colon.
    - 70% of colonic obstruction is due to malignancy.
    - 30% is due to volvulus, diverticulitis, inflammatory cause like tuberculosis, etc.
- Mortality is 3% in obstruction without strangulation, 30% in obstruction with strangulation.
- Recurrent obstruction is more common in adhesions.

PATHOLOGY AND PATHOGENESIS

- There are changes that occur:
  - Proximal to the bowel obstruction
  - At the site of obstruction
  - Distal to the obstruction

PROXIMAL TO THE BOWEL OBSTRUCTION (BEFORE THE OBSTRUCTION)

- Intestinal obstruction leads to increased peristalsis (hyperactivity) which may eventually become vigorous in an attempt to overcome the obstruction and if obstruction is not relieved peristalsis ceases and there is flaccid paralysis and dilatation of the bowel.

Intestinal obstruction
↓
Increased peristalsis
↓
Becomes vigorous
↓
Obstruction not relieved
↓
Peristalsis ceases.
↓
Flaccid, paralysed, dilated bowel

- The vigorous peristaltic contractions are clusters contractions that causes severe colicky abdominal pain experienced by the patient.
- Fluid from saliva (1000ml/day), stomach (2000ml/day), pancreas (1500ml/day), bile (1000ml/day) and intestine (3000ml/day) collects just proximal to the obstruction.
- Accumulation of swallowed air and fluids causes the proximal bowl to dilate. The interface between air and fluid in the dilated loops accounts for the fluid levels seen in the erect abdominal film of these patients.
- There is increased transudation across the capillary membrane as venous drainage of the affected segments is impaired.
- The wall of the obstructed gut becomes edematous and because of edema and inflammation, absorption decreases.
- The fluid and electrolytes that accumulate in the lumen of the obstructed bowel and within its wall are effectively lost (sequestrated third-space losses) and contribute (together with vomiting) to the fluid and electrolyte deficit in these patients.
  - In the first 12 hours of obstruction there is only decreased absorption which causes accumulation of fluid and electrolytes in the lumen.
  - After 12 hours, there is also increased intestinal secretion causing further accumulation of the fluid.

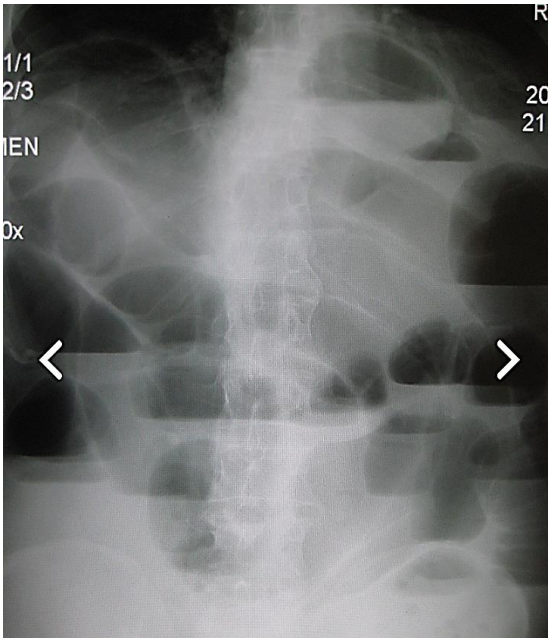
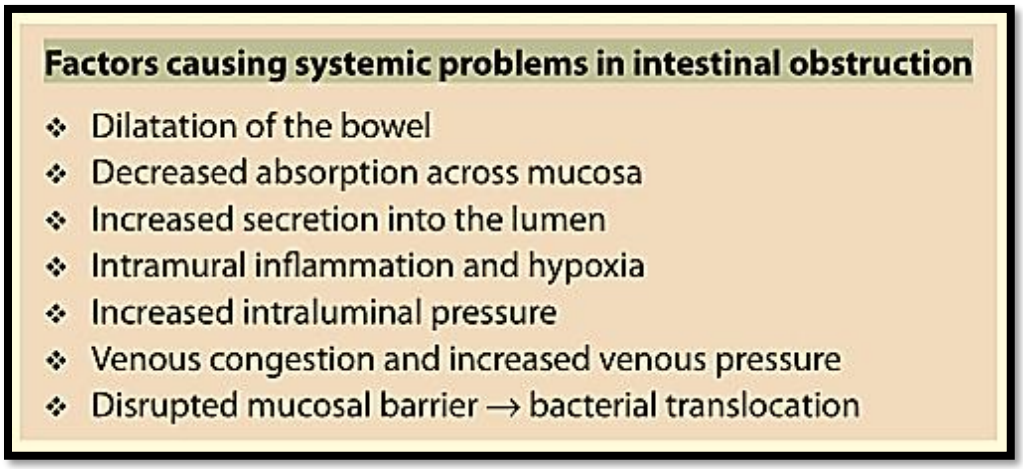


Figure 82: Upright abdominal X-ray demonstrating a small bowel obstruction. Note multiple air fluid levels. You more than 2to make a diagnosis of obstruction

- Bacteria overgrowth occurs within the obstructed loops of intestine (*E. coli*, *Klebsiella*, anaerobes, Bacteroides and other organisms). Bacteria multiply and toxins are released causing toxemia occurs.

- Unless the distention is relieved, there is progressive occlusion (by stretching) of intestinal intramural vessels such that, untreated, a mechanical intestinal obstruction leads to ischemia and eventually necrosis with perforation of the bowel.
  - Inflammatory response in the bowel wall (intramural inflammation) causes accumulation of activated neutrophils and macrophages in the muscle wall which release reactive enzymes and cytokines which damage secretory and motor process of muscle leading to dilatation of the bowel.
  - Increased release of nitric oxide in muscle wall and production of intramural reactive oxygen metabolites alter gut motility and permeability.
  - Intestinal hypoxia also causes dilatation.
  - Dilatation of bowel wall increases intraluminal pressure which exceeds the bowel wall venous pressure causing ischemia which causes further dilatation and ischemic injury. This leads into eventual blockage of arterial perfusion causing bowel necrosis/gangrene.



AT THE SITE OF OBSTRUCTION

- Initially venous return is impaired, this leads to congestion, edema of bowel wall which turns purple.
- Later this jeopardizes the arterial supply and the bowel loses its shininess & peristalsis and becomes blackish in color.
- This may lead to gangrene which can cause perforation leading to bacteria and toxins migrating into the peritoneum causing peritonitis.

Initially venous return is impaired.
↓
Congestion, oedema of bowel wall occurs which turns purple.
↓
Later this jeopardizes the arterial supply.
↓
Loss of shineness, blackish discolouration, loss of peristalsis.
↓
Gangrene.
↓
Perforation occurs.
↓
Bacteria and toxins migrate into the peritoneum.
↓
Peritonitis.

BOWEL DISTAL TO OBSTRUCTION (AFTER OBSTRUCTION)

- The distal bowel empties to a collapsed state. It becomes Inactive and collapsed



Figure 83: Pathology of Intestinal obstruction

CLINICAL FEATURES

- Four cardinal symptoms of intestinal obstruction:
  - Vomiting
  - Colicky abdominal pain
  - Abdominal distension
  - Absolute constipation
- NOTE not all may be present.

HISTORY

- Vomiting: early sign in high obstruction, late or absent in chronic or low obstruction.
  - Jejunal obstruction: early and persistent
  - Ileal obstruction: recurrent occurring at an interval, initially bilious later feculent. This is caused by bacterial overgrowth in the obstructed small intestine
    - True fecal vomiting only happens in gastrocolic fistula.
  - Large bowel obstruction: vomiting is a late feature
  - Note: Vomiting together with sequestration of fluid in the dilated loops, rapidly leads to dehydration with significant water and electrolyte deficits particularly sodium and chloride. The dehydration leads to raised PCV and prerenal azotemia with elevation of blood urea and reduced urine output.
- Abdominal pain:
  - Pain is first to develop and it is sudden and severe.
  - Initially it is colicky and intermittent.
  - Later continuous and severe. Constant severe pain is ominous and indicates with infarction of the bowel or the onset of peritonitis.
  - Pain begins usually around umbilicus in small bowel obstruction. In small bowel obstruction, it is crampy, recurrent paroxysms occurring as short crescendo/decrescendo episodes (of 30 seconds).
  - In distal obstruction pain is suprapubic. In large bowel obstruction it is of longer episodes of minutes (in paralytic/ adynamic ileus pain is diffuse and mild).
  - Post op obstruction- may be disguised by general discomfort and by opiates given to the patient.
- Abdominal distension: becomes progressively more marked the lower the obstruction is situated and may reach extreme degrees in low colonic obstruction and paralytic ileus. It is due to accumulation of gas and fluid in obstructed bowel.

- Jejunal obstruction: absent or minimal
- Ileal obstruction: obvious visible intestinal peristalsis (VIP) and borborygmi sounds (rumbling noise in the abdomen caused by gas in the intestine)- step ladder peristalsis.
- Large bowel obstruction: enormous distention



Figure 84: Step ladder peristalsis

- Constipation: it is absolute i.e. neither feces nor flatus is passed
  - It is an early feature of large bowel obstruction but late feature of small bowel obstruction.
  - Exceptions: Richter's hernia obstruction, Gall stone obstruction, mesenteric vascular occlusion, intestinal obstruction with a pelvic abscess

EXAMINATION

- Dehydration: leads to oliguria and renal failure. There is loss of skin turgor, hypotension and persistent tachycardia.
- Features of toxemia and septicemia: tachycardia, tachypnea, fever, sunken eyes, cold periphery.
- In pain, may be rolling about with colic.
- Temperature:
  - Fever- inflammation in bowel wall/ ischemia/ perforation
  - Hypothermia can occur when septicemia develops due to lack of pyrogenic response. It suggests poor prognosis.
- During inspection look for:
  - Strangulated external hernia- inguinal or femoral (difficult in fat people).
  - Abdominal scar-previous operation may suggest adhesions or bands.
  - Abdominal distention, may have visible peristalsis.
- On palpation:
  - Abdominal tenderness
    - It is initially localized but later becomes diffuse- is a feature of intestinal obstruction.
  - Rebound tenderness and guarding will not be present in simple obstructions which are features of strangulation.
  - A mass may be present (intussusception or carcinoma)
- Features of strangulation:

- Continuous severe pain, shock, tenderness, rebound tenderness (Blumberg's sign)
- Guarding and rigidity, absence of bowel sounds
- In case of strangulated hernia, a swelling which is tense, tender, rigid, irreducible, no expansible impulse on coughing and history of recent increase in size is seen.

- On percussion the distended abdomen is resonant to percussion on the anterior aspect but is dull towards the flanks.
- On auscultation Bowel sounds: they are increased- high pitched metallic (rushes and groans) sounds followed by metallic tinkling sounds of dilated bowel. Eventually once fatigue occurs or gangrene develops bowel sounds are not heard- silent abdomen of peritonitis develops (in paralytic ileus, there are only continuous metallic sounds of dilated bowel).
- Per-rectum examination: shows empty, dilated rectum, often with tenderness. If rectal growth is the cause for obstruction, it may be palpable.
  - May sometimes reveal a mass in the pouch of Douglas, apex of an intussusception or fecal impaction.
- Digital vagina examination in a female patient is mandatory. This is done before the rectal examination with the same gloved finger.

INVESTIGATIONS

- Imaging:
  - Plain Abdominal X-ray
    - Supine abdominal X-ray shows distention of the intestine. It distinguishes small bowel from large bowel obstruction (see table below)
    - Erect abdominal X-ray is taken if perforation is suspected.
      - Multiple central air-fluid levels in a step-ladder fashion in small bowel. In large bowl they are less numerous and located in the flanks and suprapubic regions
      - Proximal to obstruction: lesser the air fluid level

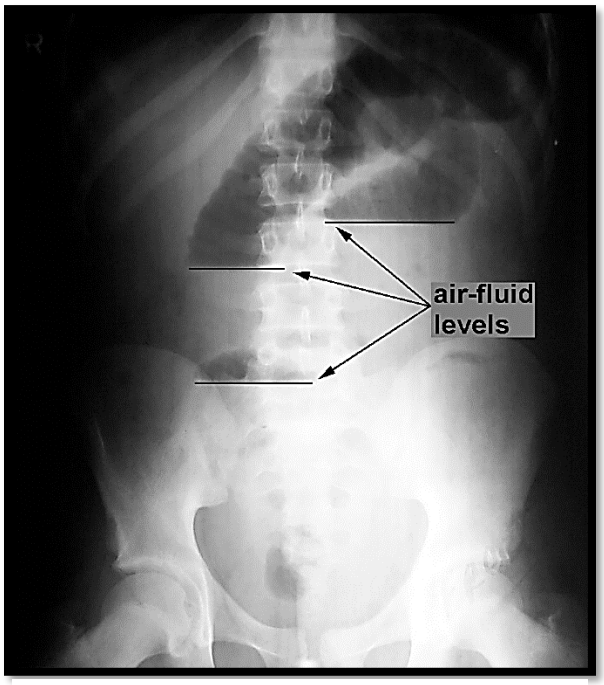


Figure 85: AXR showing air-fluid levels

- Distal to obstruction: more the air fluid level.

- Normally, 3 fluid levels can be seen in plain X-ray film- at fundus of stomach, at duodenum and often at cecum.
- Jejunum- show valvulae conniventes (Herring bone pattern)/valves of Kerckring (plica circularis/ valvulae conniventes)
- Ileum is smooth and characterless (by Wangensteen)
- Large bowel shows haustrations of tenia coli.
- Pneumobilia (gas in biliary tree) may be due to gallstone ileus.

➢ Triad of small bowel obstruction in plain X-ray

- Dilated small bowel loops> 3cm
- Multiple air fluid levels in erect X-ray
- Paucity of air in the colon



Figure 87: Small bowel obstruction. Note the valvulae conniventes, the central position of the bowel and the coiled-spring appearance

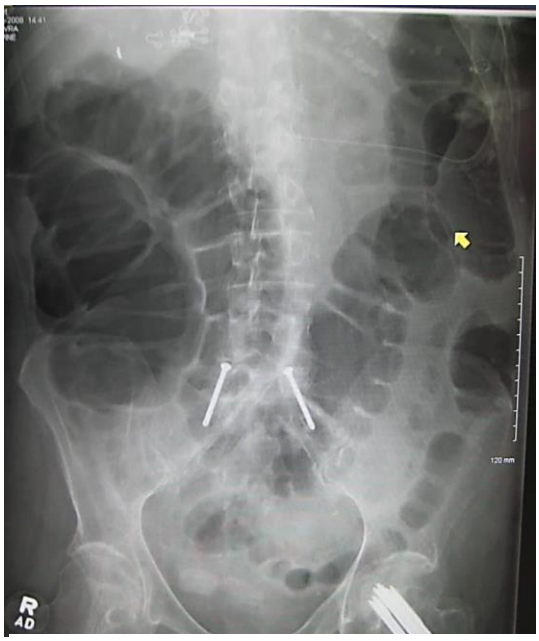
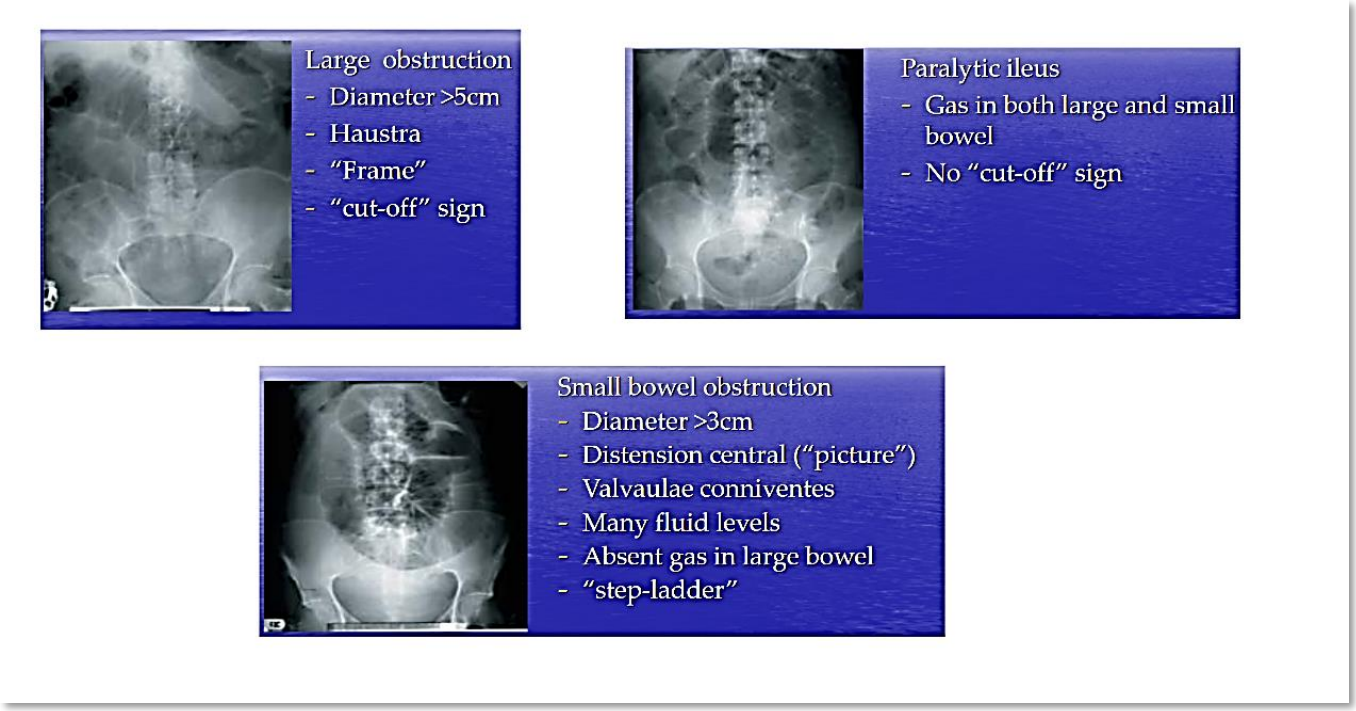


Figure 86: Large bowel obstruction. Note the haustra which do not traverse the entire diameter of the bowel. The bowel is in the periphery

SMALL BOWEL ON X-RAY	LARGE BOWEL ON X-RAY
• Less than 3cm wide • Tends to be central • It is only seen if it contains gas • Mucosal folds (valvulae conniventes/valves of Kerckring) • Obstruction on X-ray shows a "coiled-spring appearance"	• Less than 6cm wide, Cecum and sigmoid up to 9cm. • Tends to be peripheral • Ascending and descending colon in fixed positions laterally while transverse colon and sigmoid have a variable position on the mesentery • Haustral folds do not go all the way across the lumen • Contains feces-mottled appearance



- Chest X-ray: shows elevation of the diaphragm which is secondary to the abdominal distention. It may also show free air underneath the right diaphragm in patients with infective paralytic ileus (secondary to intestinal perforation).
- Abdominal ultrasound:
  - Dilated bowel and fluid in the peritoneal cavity.
  - Better than X-ray but not as good as CT scan.
  - Doppler US is useful in detecting strangulation
- Barium enema X-ray

- Useful in intussusception
- Barium meal is usually contraindicated in acute intestinal obstruction.
- Barium follow through X-ray may be done with caution in suspected/partial intestinal obstruction under fluoroscopy, otherwise it may precipitate complete obstruction or may cause perforation and barium peritonitis which is very dangerous.

➢ CT scan
- Very reliable investigation for intestinal obstruction.
- Gives idea on changes in bowel wall, ischemia, strangulation, mesenteric edema and thickening. Also shows bowel wall gas, portal venous gas and mass lesion.

- Blood:

➢ Full blood count: Hemoglobin and Hematocrit (PCV) are raised. The white cell count is usually normal or slightly elevated unless there is bowel infarction and/or peritonitis.

➢ Electrolytes, Blood urea and serum creatinine: these are raised as a result of an element of pre-renal failure due to hypovolemia. Serum sodium and chloride are low. Hyperkalemia may be observed in patients with infarcted intestines.

➢ LFTs

➢ Arterial blood gas: acidosis is common

	Proximal small bowel	***Distal small bowel***	***Large bowel***
Site of obstruction	*Duodenum and jejunum*	*Ileum*	*Any where in large intestine*
Causes:	Congenital Lipomas Leiomyomas Malignancy Bands and adhesions	Tuberculosis strictures Malignancy Crohn's Gallstones Hernias—common cause Roundworm Congenital	Malignancy Tuberculosis stricture Anorectal malformation Volvulus Congenital megacolon Bands
Clinical features:	Severe vomiting, dehydration, no or less distension, colicky pain	Central distension, vomiting, dehydration central abdominal pain	Constipation, distension—early; Late vomiting less pain
Special features: Plain X-ray	Valvulae conniventes	Characterless Central fluid level	Dilatation and haustration

COMPLCIATIONS OF INTESTINAL OBSTRUCTION

- Hypovolemic and septic shock
- Perforation
- Peritonitis
- Intra-abdominal abscess formation
- ARDS
- Renal failure

DIFFERENTIAL DIAGNOSIS

- Paralytic obstruction
- Pseudo-obstruction
- Ascites

MANAGEMENT

- Resuscitation- ABCs
- Depends on cause.
- Principles of management:
  - Decompression of obstructed gut
  - Replacement of fluid and electrical losses
  - Special conservative measures in certain situations
  - Surgical intervention
- Acute obstruction of sudden onset, complete and with risk of strangulation is an urgent problem requiring emergency surgical intervention.
- Chronic large bowel obstruction, slowly progressing obstruction can be investigated at some leisure with sigmoidoscopy, colonoscopy and barium enema and treated electively.

DECOMPRESSION

- This is done by insertion of a nasogastric sump suction tube (Salem). This is aspirated at least every hour and left draining into a bag in the intervening periods.
- The daily aspirate is measured and the amount used in calculating the daily fluid and electrolyte requirements.
- If the intestinal obstruction responds to conservative management, the daily amount of aspirate gradually reduces and its nature changes to clear often bile-stained fluid.
- Nasogastric aspiration: reduce toxic effects, bowel distension and improve pulmonary ventilation as well as reduce possibility of aspiration pneumonia.

FLUID AND ELECTROLYTE THERAPY

- Fluids and electrolytes should be given intravenously.
- Fluids used: Hartmann's solution, normal saline and 5% dextrose.
- Initially large amounts are administered (1L every 3-4 hours) to replace the losses. Thereafter, maintenance IV fluid therapy is continued until return of normal bowel function.
- The usual daily requirements of potassium (40-120 mmol) are met by infusing 60-80mmol of KCL in divided doses over 24 hours.
- If hypokalemia is severe, up to 40mmol may be infused over 1h in 400ml of fluid. It is important to remember that potassium must always be administered slowly and never given as a bolus injection because of the risk of cardiac arrhythmias and arrest.
- Catheterize patient and Monitor urine output.

SPECIAL CONSERVATIVE MEASURES

- For certain types of intestinal obstruction, additional specific measures may result in rapid relief of the obstruction thus avoiding surgical intervention.
- Examples:
  - Passage of a rectal tube or flexible sigmoidoscope to deflate sigmoid volvulus
  - Barium contrast enema to reduce an early intussusception in an infant
  - Manual removal of feces and/or oil retention enema to deal with obstruction caused by fecal impaction
- It is important to assess the patient frequently to establish progress on conservative management. This is confirmed by relief of symptoms (vomiting and pain), improvement of general condition and vital signs (pulse rate, temperature and blood pressure) and observations such as reduction in amount of aspirate and abdominal girth and return of normal bowel sounds.

SURGICAL INTERVENTION

- This is undertaken if:
  - The underlying disease needs surgical treatment (obstructed hernia, obstructing carcinoma etc.) in this respect, intestinal obstruction due to adhesions often settles with conservative management and therefore is not initially treated surgically unless there is clinical evidence of strangulation.
  - The patient does not improve with conservative treatment
  - There are signs of strangulation or peritonitis.
- Surgery:

- Immediate laparotomy to relief obstruction.
- Warm saline soaked mop is placed over the doubtful area with 100% oxygen inhalation for 20 minutes if colour becomes normal with peristalsis then bowel is viable.
- If bowel is not viable resection and anastomosis is done. A good peritoneal wash is given and the abdominal cavity is drained.
- Abdomen is closed in layers using non-absorbable sutures. Often tension sutures are required.
- Laparoscopic approach may be useful in partial obstruction, proximal obstruction due to band. Conversion when needed should be done without hesitation.
- When checking for viability of bowel, look for:
  - Peristalsis
  - Pulsations
  - Colour (black/pink)- dull and lusterless serosa is seen in ischemia
  - Serosal shining
  - Bleeding in mesentery and bowel wall
  - Friability- friable, flabby muscle is seen in ischemia

- A defunctioning loop colostomy or ileostomy may be performed to minimize the complications of anastomotic leak.
- Post-surgical complications:
  - Pelvic abscess
  - Subphrenic abscess
  - Biliary or fecal fistulas
  - Burst abdomen
  - Bands and adhesions
  - Incisional hernias

ADDITIONAL MEASURES

- ICU critical care.
- Antibiotics: ampicillin, gentamycin, metronidazole, cephalosporins
- Analgesics
- Blood transfusion: FFP or platelet in critical patients.
- CVP for fluid and monitoring- pulmonary capillary wedge pressure monitoring are often needed in hemodynamically unstable patients.

INTUSSUSCEPTION (ISS)



Figure 88: Intussusception

- Intussusception is telescoping or invagination of 1 portion (Segment) of bowel into the adjacent segment of bowel.
- Types:
  - Antegrade: most common
  - Retrograde: rare (jejunogastric in gastrojejunostomy stoma)
- It can be single or multiple (Rare)
- It can be ileo-colic (most common type, 75%), ilieoileal (ileum inside ileum), colocolic (colon inside colon), ileoileocolic (Ileum inside cecum or ascending colon).
- Intussusception is common in weaning period of a child (common in males) between 6-9 months.
- It is the commonest cause of intestinal obstruction in children of 3-18 months age.
- In elderly intussusception:
  - Colocolic is most common type
  - Apex is formed usually by growth
  - No role of hydrostatic reduction
- Causes:
  - Idiopathic ISS (common in children and occurs in terminal 50cm of ileum)
  - During weaning, change in diet causes inflammation and edema of Peyer's patches- may stimulate ISS.
  - Upper respiratory tract viral infection which causes edema of Peyer's patches is also thought as an etiology for intussusception in children.

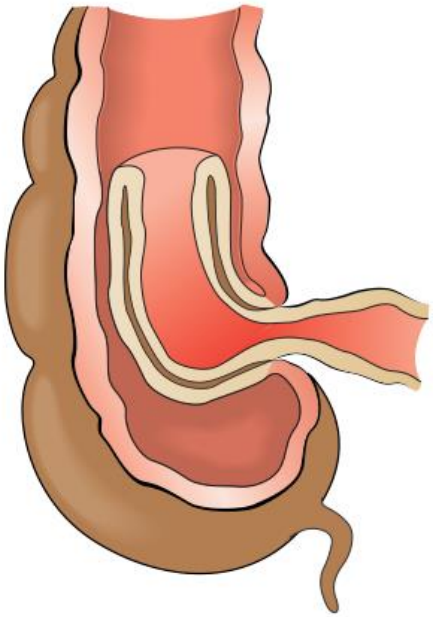


Figure 89: Ileocolic intussusception

- Other causes in adolescents and adults are submucosa lipoma, leiomyoma, polys in jejunum (Peutz-Jegher syndrome), other polyps and carcinomas with papillary projections.

PATHOLOGY

- Apex (leading point): the one which advances
- Intussucipiens: the one which receives (outer sheath)
- Intussisceptum: tubes which advances (middle and inner sheath)
  - Apex and inner tubes will have compromised blood supply and can become gangrenous.
  - Because of ischemia, apex sloughs off and bleeds, which mixes with the mucous to produce the classic red-currant jelly that is passed per anum.
  - Gangrene which sets in leads to perforation and peritonitis.
  - Red currant jelly is not commonly observed in ISS in adult but it can occur.

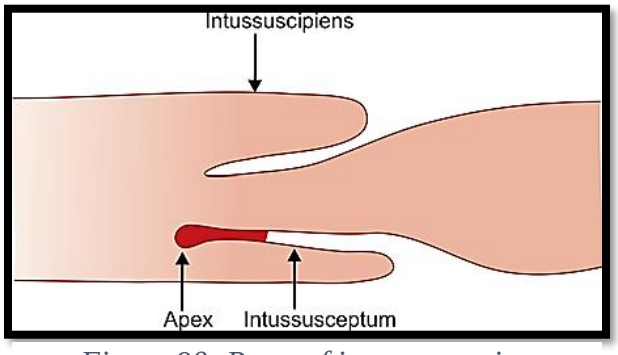


Figure 90: Parts of intussusception

CLINICAL FEATURES

HISTORY

- Common in males (3:2)
- Common in 6-9 months. But can also occur at later age
- Common in spring and winter, coinciding the gastroenteritis and respiratory infections in respective periods.
- Commonest cause of intestinal obstruction in infancy.
- Initial colicky abdominal pain (75%) which eventually becomes severe and persistent.
- Sudden onset of pain in a male child with progressive distension of the abdomen, vomiting, with passage of "red-currant-jelly" stool. It is usually not found in adult ISS.
- Often ISS is recurrent when it gets reduced, child automatically becomes asymptomatic (Mother often complains that the child cries during an episode and sleeps peacefully once it gets reduced).

EXAMINATION

- A mass is felt either on the left or right of the umbilicus which is sausage shaped with concavity towards umbilicus, smooth, firm, resonant, not moving with respiration, mobile, contracts under the palpating fingers.
- Often mass appears and disappears.
- Right iliac fossa is empty (Sign of Dance).
- After 24-48 hours, abdominal distension appears and increases progressively with features of intestinal obstruction.
- Features of intestinal obstruction with step-ladder peristalsis.
- Blood stained stool is often obvious on digital examination of the rectum.
- Occasionally ISS can be seen per anally and felt with a long mesentery.
- Eventually, gangrene and perforation occurs with features of peritonitis.

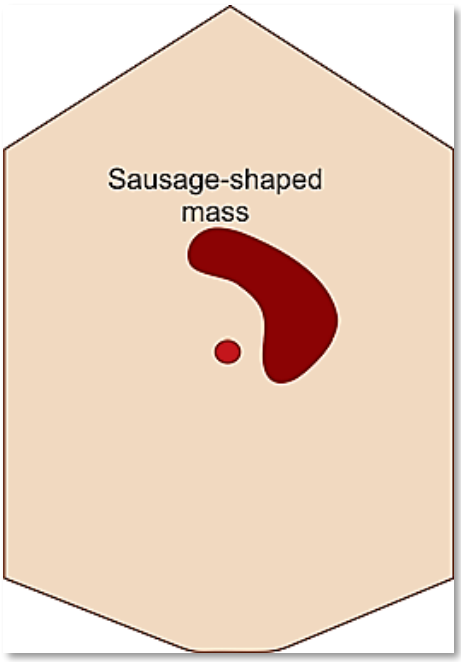


Figure 91: Sausage-Shaped mass of intussusception

Abdominal palpatory findings in ISS

- Palpable mass (85%)
  - Sausage shaped smooth, firm mass
  - Mass does not move with respiration
  - Mobile in all directions
  - Resonant
  - Mass contracts under the palpating fingers
  - Mass appears and disappears
- Empty right iliac fossa
- Features of intestinal obstruction/peritonitis—later

COMPLICATIONS

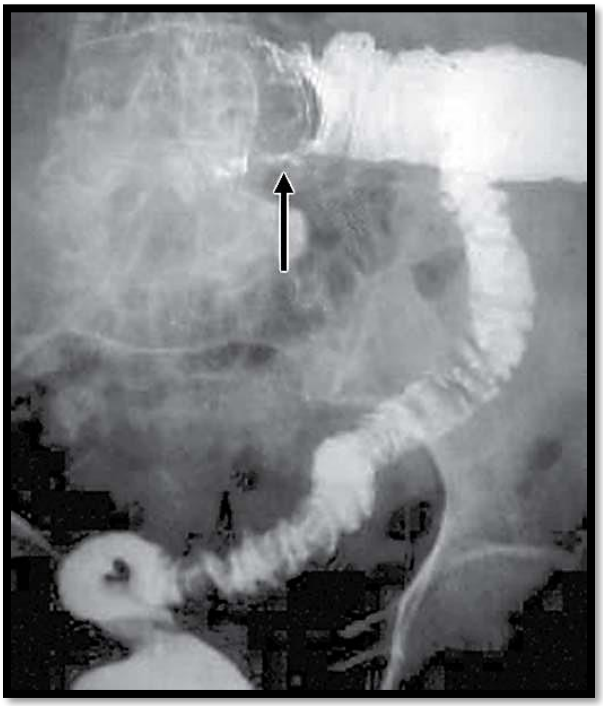
- Intestinal obstruction
- Perforation
- Peritonitis

DIFFERENTIAL DIAGNOSIS

- In children:
  - Acute gastroenteritis
  - Purpura with intestinal symptoms
- In adults:
  - Carcinoma colon
  - Mesenteric mass

INVESTIGATIONS

- Imaging:
  - Barium enema shows typical claw sign or coiled spring sign (Pincer end).





  - Ultrasound shows target sign or pseudokidney sign or bull's eye sign which is diagnostic.

- Doppler may show mass with doughnut sign and is useful to check blood supply of bowel.
- Plain X-ray abdomen shows multiple air fluid levels.

MANAGEMENT

INITIAL MANAGEMENT

- Ryle's tube aspiration (Wide bore nasogastric aspiration)
- IV fluids
- Antibiotics
- Catheterisation

LATER MANAGEMENT: NON-OPERATIVE MANAGEMENT

- Reduction by hydrostatic pressure using either warm saline or micro-barium sulphate solution or air.
- Barium or saline is infused into the rectum through a catheter (Foley's catheter).
- Under fluoroscopy, reduction can be observed.
- Child will pass large quantity of air and feces, distension reduces, child shows recovery and stops crying.
- Air or contrast enters the proximal bowel freely.
- Palpable abdominal mass disappears.
- Rare complication is perforation of colon. It is done in early stage within 24 hours of presentation.
- 70% cases of ISS will respond to non-operative method. It is contraindicated in complete obstruction, perforation and peritonitis.

SURGERY

- Cope's method: if reduction does not occur, laparotomy is done under general anesthesia. By gently milking out the intussusception with warm packs, it is reduced. After reduction, viability of the bowel is checked carefully. If manual reduction is not possible it is likely bowel is gangrenous and so resection and anastomosis is done. In case of viable bowel, often terminal ileum is anchored to the ascending colon and Jackson veil band is cut. Patient also require nasogastric tube aspiration, IV fluids and antibiotics.
- Laparoscopic approach may be used to reduce the intussusception
- If intussusception persists for more than 48 hours or intussusception in adult requires resection. Ileocolic resection is sufficient.

VOLVULUS

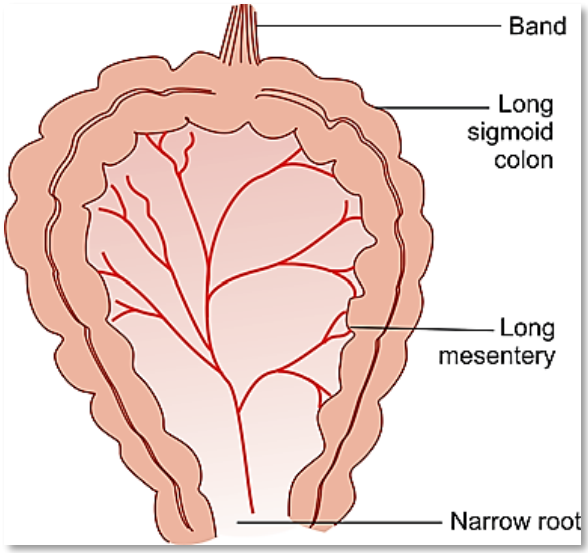
- Volvulus is the twist (rotation) in the axis of the loop of the bowel either clockwise or anticlockwise.
- 15% of large bowel obstruction is due to volvulus.
- Sigmoid colon is the commonest site (Anticlockwise)-65%.
- The rotation causes early obstruction of the vascular pedicle supplying the affected portion.
- Unless recognized early, volvulus leads to intestinal infarction which often involves large segments of the gut.
- Risk factors for small bowel volvulus (which may affect the whole of the mid-gut):
  - Adhesions
  - Bands between the anti-mesenteric aspect of the bowel
  - Anterior abdominal wall
  - Congenital mal-rotation of the gut
- Risk factors of sigmoid volvulus:
  - Chronic constipation
  - Redundant pelvic mesocolon

CAECAL VOLVULUS

- Caecum is the second common site (clockwise) (C for C)-30%.
- Its common in females, presents as intestinal obstruction.
- Caecal bascule is the presence of constricting band across the ascending colon (Bascule-French-See-saw and balance).
- Caecum will be markedly distended and found in the center of the abdomen.
- It is due to lack of fixation of the caecum- mobile caecum.
- Occasionally it is associated with mal-rotation.
- Caecal volvulus is the commonest cause of large bowel obstruction in pregnancy.
- X-ray shows round gas shadow in right iliac region. CT scan is very useful. Barium enema is also helpful.
- Resection and anastomosis (surgery) is the only treatment.
- Volvulus of small intestine (mid-gut) volvulus neonatorum, gastric volvulus are other volvulus which can occur.

SIGMOID VOLVULUS (VOLVULUS OF PELVIC COLON)

- Common in Asia especially South India because of high fiber diet.
- Common cause of large bowel obstruction in Peru and Bolivia due to high altitude.
- More common in males and old age.
- It is common in patients with chronic constipation with laxative abuse.
- Predisposing factors include:
  - Adhesions
  - Peridiverticulitis
  - Overloaded redundant pelvic colon
  - Long pelvic mesocolon
  - Narrow attachment of sigmoid mesocolon.
- Sigmoid volvulus is always anticlockwise.
- It requires one and half turn of rotation to cause vascular obstruction and gangrene which eventually leads to perforation either at the root or at the summit of the sigmoid loop.
- Enormous distension of the colon occurs. Sometimes ileum comes to the root of the sigmoid volvulus and encircles it causing compound volvulus in which case knotted small bowel also becomes gangrenous- ileosigmoid knotting.
- Sigmoid volvulus is common in:
  - Ogilvie's syndrome
  - Mentally retarded individuals



  - Chagas's disease
  - Hypothyroidism
  - Anticholinergic drugs
  - Multiple sclerosis
  - Scleroderma
  - Parkinson's disease

TYPES

- Acute
- Recurrent

CLINICAL FEATURES

- Pain in the abdomen- initially left sided, eventually all over.
- Absolute constipation (Obstipation- no feces, no flatus)
- Enormous distension of abdomen, starting from left iliac fossa

extending to the whole of the abdomen (tympanic abdomen)

- Late vomiting and eventually dehydration
- Features of peritonitis.
- Hiccough and retching can occur.
- Tyre like feel of the abdomen is diagnostic.

DIFFERENTIAL DIAGNOSIS

- Ogilvie's syndrome: it is acute colonic pseudo obstruction. It is due to malfunctioning sacral parasympathetic nerves. Splenic flexure is the junction of collapsed and dilated large bowel. Descending colon is atonic causing acute functional obstruction. It may be due to trauma, retroperitoneal irritation, antidepressants, uremia, diabetes, myxedema, hypokalemia etc. prokinetic drugs, colonoscopic decompression, tube Caecostomy are the treatment
- Fecal impaction
- Carcinoma recto-sigmoid region
- Idiopathic megacolon

INVESTIGATIONS

- Plain X-ray (Diagnostic in 70-80%)
  - ➢ Omega sign- single, grossly distended loop of colon arising out of the pelvis and extending towards the diaphragm.
  - ➢ Coffee- bean sign or Bent-inner tube sign.
- Contrast enema (dilute barium/ water soluble contrast media is used)
  - ➢ Birds beak sign (Ace of spades appearance)- upper end of barium column tapers into the spirally twisted distal sigmoid colon.
- CT scan (for difficult cases): shows characteristic whirl pattern.
- Blood- full blood count, renal function tests, serum electrolyte.

TREATMENT

- Ryle tube aspiration
- IV fluids
- Catheterisation
- Antibiotics
- A flatus tube or sigmoidoscope is passed in operation theatre (proper care is taken otherwise it leads to perforation). If it de-rotates, patient will pass flatus and feces and distention reduces.
- If de-rotation does not occur, laparotomy through midline incision should done. Dilated sigmoid colon is de-rotated manually and checked for viability. If viable, it can be fixed to the lateral wall of abdomen or pelvis- sigmoidpexy.
- If sigmoid colon is gangrenous, it is resected and proximal cut end is brought out as colostomy and distal end is brought out as mucous fistula, from the rectum (Paul-

Mikulicz operation). Later after 6 weeks, continuity is maintained after proper preparation.

- Resection of the gangrenous sigmoid done; proximal cut is brought out as end colostomy, distal end closed- Hartmann's operation. Later in 6-12 weeks colorectal anastomosis is maintained.
- Primary resection and anastomosis is not advisable because of the contamination and anastomotic leak.
- Mortality in sigmoid volvulus is due to perforation, peritonitis and septicemia.

PARALYTIC ILEUS

PARALYTIC ILEUS (ADYNAMIC INTESTINAL OBSTRUCTION)

- Paralytic ileus is a state in which the intestines fail to transmit peristalsis due to failure of neuromuscular mechanism i.e. Auerbach's and Meissner's plexus.
- It may be localized or generalized.
- Causes include:
  - Electrolyte imbalance: hypokalemia
  - Postoperative, intestinal ischemia
  - Infective- pus, blood (sepsis), bile, toxins, enteritis
  - Uremia
  - Spinal injury and surgery
  - Retroperitoneal hemorrhage
  - Plaster jacket
- The small bowel is distended throughout its length.
- Absorption of fluid, electrolytes and nutrients is impaired.
- Significant amounts of fluid may be lost from the extracellular compartment.

CLINICAL FEATURES

- Usually history of recent operation or trauma.

- No passage of flatus
- No bowel sounds
- Marked abdominal distention
- Vomiting of large volumes of fluid
- Tachycardia
- Respiratory distress due to pressure over diaphragm
- High pitched tinkling note 'like bells at evening pealing'
- Dull abdominal pain (not colicky)
- Features of fluid/ protein/ electrolyte imbalance

INVESTIGTIONS

- Serum electrolytes: especially potassium.
- ECG
- X-ray abdomen: dilated loops of small bowel. Gas may be present in the colon.
- Ultrasound abdomen to find out the possible cause of ileus e.g. sepsis

TREATMENT

- Nasogastric aspiration
- Primary cause is treated
- IV fluids
- Electrolyte management
- Catheterization and urine output measurement
- Do not stimulate peristalsis ("Don't flog a tired horse")
  - Measure abdominal girth to see if patient is recovering or not.
  - Decompression of large bowel can be tried by inserting a flatus tube per anally into the rectum and keeping in place for few hours.
  - Most often, patient recovers in 3-6 days by conservative treatment.
  - In prolonged life threatening paralytic ileus, laparotomy is done and bowel is decompressed with Savage's decompressor and closed with tension sutures
- Catchpole regime: adrenergic blocking agent along with cholinergic stimulant (neostigmine) is used rarely only in resistant cases as medical therapy.

ADHESIONS AND BANDS

- This is the most common cause of intestinal obstruction in Western countries.
- Causes:
  - Infection due to peritonitis, appendicitis, post-laparotomy and other acute infective abdominal conditions.
  - Materials used during surgery cause dense inflammatory reactions- silk, thread, foreign body, mop and gauze, talc powder, drugs like sulphonamides and penicillins
  - Ischemia of bowel due to poor blood supply, sepsis.
  - Gynae conditions, bowel injury, radiation induced enteritis,

Crohn's disease, other inflammatory bowel diseases.
  - Specific conditions like TB, malignancy
- Types of adhesions
  - Type 1- Fibrinous adhesions (It is avascular and flimsy)
  - Type II= Fibrous adhesions (due to lack/poor blood supply bowel gets attached to part of peritoneum or omentum or other parts of the bowel with dense vascular adhesions to maintain blood supply. It will persist and precipitate intestinal obstruction, it is often subacute and recurrent)
- Clinical features of intestinal obstruction.
- Investigations:
  - Plain X-ray abdomen shows dilated bowel loops
  - Oral contrast with CT scan is very useful
  - In partial obstruction only enterocylsis with water-soluble iodine dyes can be done.
- Treatment:
  - Initial:
    - IV fluids
    - Nasogastric aspiration
    - Antibiotics
    - Often per anal flatus tube insertion
    - Commonly patient responds in 4-5 days.
  - Later treatment:
    - Open surgical adhesiolysis using fingers gently.
    - Laparoscopic adhesiolysis is becoming popular, safer, ideal with less recurrent adhesion rate and gives good results.
    - Noble's plication of intestines- wherein adjacent coils of small bowel are sutured to prevent in going for further recurrent adhesions.
    - Childs-Phillips mesenteric plication is similar way of plication of intestinal mesentery which will prevent crumpling of bowel and adhesion formation.

Prevention of adhesions

- Instilling drugs like hyaluronidase, steroids, de polyvinyls, silicone, fibrinolysins, chondroitin
- Gentle handling of the bowel
- Prevention of spillage of contents like bile, fa
- Ideal peritoneal wash using 8-10 liters of salin the abdomen
- Careful placing of the drains
- *Laparoscopic procedures has got lesser chance sions than open method*

OBSTRUCTION DUE TO BANDS

- Dense fibrous strings attached from one portion of the abdomen to another area or bowel causing

entrapment of intestines leading into obstruction and often dangerous strangulation.

- Common causes are vitelointestinal duct, Ladd's band, omental band, postsurgical fibrous band, tuberculous band.
- Clinical features are like of intestinal obstruction.
- Management is release of the band either through laparoscopy or through laparotomy.

INTERNAL HERNIA

- Is a rare entity.
- A portion of small bowel gets entrapped in one of the retroperitoneal fossae or congenital internal defect e.g. mesenteric defect.

SITES

- Mesenteric defect, defect in transverse mesocolon.
- Foramen Winslow
- Diaphragmatic hernia
- Hernia through duodenal/ Caecal/ appendiceal/ intersigmoid retroperitoneal fossae
- Hernia through broad ligament

FEATURES

- Herniation occurs in specific site whenever there are adhesions.
- Features of intestinal obstruction- acute/ subacute/ recurrent
- Diagnosis is usually on table. But enterocylsis, contrast CT scan are useful.

TREATMENT

- Reduction of hernia, release of adhesion, if bowel is not viable resect and anastomose.
- Often major vessels may be present adjacent to the constriction ring which may get injured while releasing causing torrential hemorrhage.
- Decompression is first done prior to reduction.

APPENDICITIS

- The appendix begins to bud off from the cecum at around the 6th week of embryological development.
- The vermiform appendix is narrow, of variable length (2-20cm) with its base attached to the posteromedial surface of the cecum close to the junction of the ileum and cecum.
  - Location: terminal end of cecum where 3 taeniae join, about 2cm below the ileocecal orifice.
  - Length: 2-20 cm but can be variable.
  - Diameter of appendix: 3-8 mm
    Size of lumen: 1-3 mm (size of a matchstick).
  - Luminal capacity <1ml
- The mesoappendix an extension of the mesentery contains appendicular artery of Seshachalam (a branch of ileocolic artery which is derived from the superior mesenteric artery).
  - Recall that the appendicular artery is an end-artery (it does not have collaterals)
  - Thrombosis of these vessels thus leads to gangrenous appendicitis.
- The base of the appendix remains in a fixed position with respect to the cecum, whereas the tip can end up in various positions. The different anatomical locations of the appendix include:
  1. Retrocecal appendix (most common)
  2. Periileal appendix
  3. Postileal appendix
  4. Pelvic appendix
  5. Subcecal appendix
  6. Paracecal appendix
- The appendix is composed of the same layers as the colon wall: Mucosa, submucosa, inner circular muscle, outer longitudinal muscle and serosa

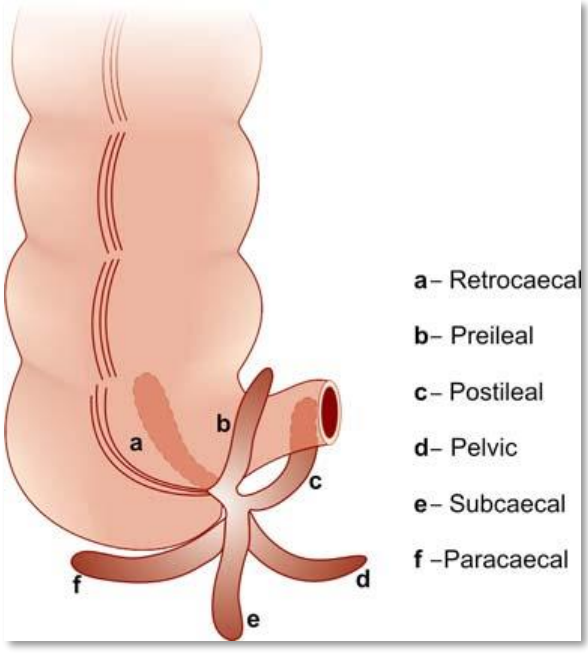


Figure 92: Positions of the appendix (adapted from SRB's Manual of Surgery)

- The submucosal layer of the appendix has lymph follicles. When these enlarge in response to infection, the swollen submucosal layer can block the lumen of the appendix.
- Although many claim that the appendix is merely a vestigial organ, it is actually an immunological organ and secretes IgA. However, it is not an essential organ and can be removed without immunological compromise.
- Acute appendicitis is the inflammation of the appendix.
- It is one of the most common acute surgical diseases.
- It is common in young white males at the peak of lymphoid tissue growth. A second peak in the incidence of appendicitis occurs in the elderly.
- Fiber rich diet prevents appendicitis while less fiber increases chances of appendicitis.
- It is common in May and August (seasonal variation) and is often called as epidemic appendicitis.

ETIOLOGY

- Etiology remains unclear. Diet may be involved (Reduced fiber content) as the disease is very common in West but rare in Africa.
- Obstruction of lumen (obstructive appendicitis)
  - Fecalith (small, firm ball of stool most common cause in adults)- it is associated with gangrene or perforation
  - Strictures
  - Tumors
  - Gallstones
  - Foreign body
  - Round/ thread worm/Pinworm (*Oxyuris vermicularis*)
  - Adhesions and kinking-carcinoma cecum near the base, ileocecal Crohn's disease
- Viral infections: associated with mucosal edema and inflammation (lymphoid hyperplasia) with a bacterial infection.
  - *E. coli* (80%)
  - *Enterococci* (30%)
  - *Streptococci* and Anaerobic *streptococci*
  - *Clostridium Welchii*
  - *Bacteroides*

- Pseudoappendicitis is appendicitis due to acute ileitis following *Yersinia infection*. It is often due to Crohn's disease.

PATHOGENESIS

- Arise from either:
  - Obstructive causes
  - Non-obstructive causes (Catarrhal)

OBSTRUCTIVE

- Luminal obstruction (due to either lymphoid obstruction in young patients or fecalith commonly seen in older patients) leads to increased mucus production and inflammation.
  - Recall: the appendiceal mucosa continues to secrete normally despite being obstructed
- As inflammatory fluid collects inside the lumen there is distention and increase in intraluminal pressure leading to blockage of venous & lymphatic drainage. The resident bacteria also multiply rapidly further increasing intraluminal pressure.
- Intraluminal pressure eventually exceeds capillary and venule pressures, however Arteriolar blood continues to flow in causing vascular congestion and engorgement.
- Impaired blood supply results in mucosal ischemia and ulceration with ultimate bacterial translocation/invasion. Bacterial spread through submucosa and muscularis propria resulting in acute obstructive appendicitis.
- Inflammation and ischemia progress to involve the serosal surface of the appendix.
- The appendicular artery can thrombose and cause ischemic necrosis which can cause gangrene, perforation of the tip/base and peritonitis
- After perforation there is localization by greater omentum and dilated ileum. This can suppurate forming an appendicular abscess, if pus is not present inside it is referred to as an appendicular mass.
- Acute appendicitis with blockage at the opening of the lumen results in mucus collecting inside the lumen of the appendix resulting in its enlargement (Mucocele of the appendix)

NON-OBSTRUCTIVE

- Acute inflammation of mucus membrane with secondary infection without obstruction causes acute non-obstructive appendicitis.
- It may lead to:

- Resolution
- Fibrosis
- Recurrent appendicitis
- Obstructive appendicitis
- Gangrene (rare)
- Peritonitis
- Suppuration

TYPES

TYPE	COMMENT
ACUTE OBSTRUCTIVE APPENDICITS	• Pus collects in the blocked lumen of the appendix which is blackish, gangrenous, edematous and rapidly progresses leading to perforation either at the tip or at the base of the appendix • This leads to peritonitis, formation of appendicular abscess or pelvic abscess • Most often, there will be thrombosis of the appendicular artery
ACUTE NON-OBSTRUCTIVE APPENDICITIS (CATARRHAL)	• Inflammation of mucous membrane occurs with redness, edema and hemorrhages which may result in resolution, ulceration, fibrosis, suppuration, recurrent appendicitis, gangrene (rare) and peritonitis
RECURRENT APPENDICITIS	• Repeated attacks of non-obstructive appendicitis leads to fibrosis, adhesion causing recurrent appendicitis
SUBACUTE APPENDICITIS	• Is the milder form of acute appendicitis
STUMP APPENDICITIS	• Is retained long stump of appendix after commonly laparoscopic appendicectomy

CLINICAL FEATURES

- Rare before age of 2 (common in other age groups & children i.e. 5 to 35 years)
- Murphy's triad:
  - Pain
  - Vomiting
  - Temperature

SYMPTOMS

- Pain: earliest symptom.
  - Visceral pain starts around the umbilicus-T10 distribution (due to obstruction and distension of appendix and stimulation of visceral afferent stretch fibers). Pain is a dull ache or colic (presumably from obstruction of the appendiceal lumen)
  - After a few hours somatic pain occurs in right iliac fossa (due to irritation of parietal peritoneum because of inflamed appendix)
  - Pain is severe and diffuse-spread of infection into general peritoneal cavity.
  - Not all patients experience this shift of pain and the presentation may start with discomfort in the right lower quadrant. Retrocecal appendicitis may cause pain higher in the right abdomen whereas appendicitis located in the pelvis may cause vague pelvic discomfort.
  - Patients often report that movement and coughing induces sharp exacerbation of the pain.

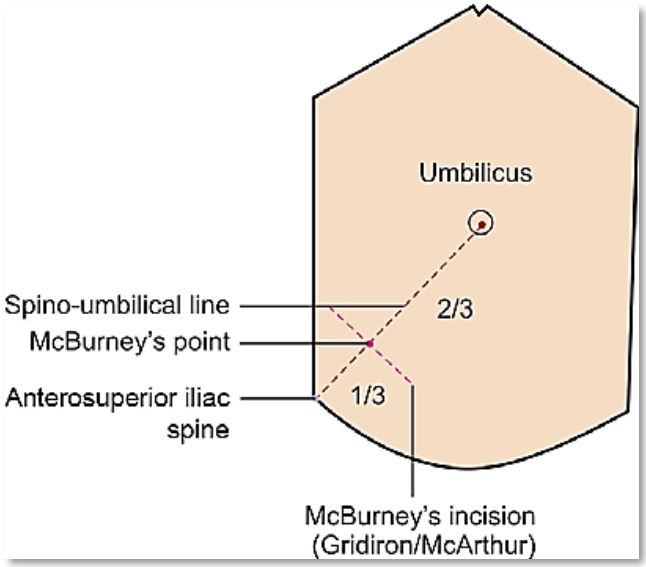


Figure 93: McBurney's point is the junction of lateral 1/3rd and medial 2/3rd of spinoumbilical line. McBurney's/Gridiron's/McArthur's incision is perpendicular to this line in this point. (Image from SRB's Manual of Surgery)

- Vomiting and nausea: due to reflex pylorospasm
- Anorexia
- Constipation (usual feature)/ diarrhea (20%) (diarrhea common in postileal or pelvic appendix)
- Fever (low grade)
- Tachycardia
- Urinary frequency: Bladder irritation from contact with inflamed appendix
- Note:
  - In children it is important to ask about a sore throat or flu-like symptoms as these often accompany mesenteric adenitis.

- Female patients in particular should be asked about dysuria, frequency and cloudy/strong smelling urine because UTI can often cause lower abdominal pain. Likewise, a menstrual history is essential. A missed period may point to an ectopic pregnancy.
- Pain at mid-cycle may indicate that the pain is due to ovulation (Mittelschmerz) and vaginal discharge may indicate pelvic inflammatory disease.

SIGNS

- Tachycardia
- Mild pyrexia. A high temperature (>39°C) indicates probable abscess formation or some other diagnosis such as a viral illness.
- The tongue is also usually furred.
- An appendicular abscess or mass (phlegmon) if present may be palpated in the right iliac fossa
- Important signs:
  - Abdominal Tenderness and rebound tenderness in right iliac fossa (Blumberg sign/Release sign)
    - Ask the patient to cough while watching the facial expression. If coughing produces obvious pain, the patient should be asked to indicate the site of maximum pain. In acute appendicitis the patient points to the right iliac fossa.
    - Rebound tenderness may be elicited by pressing gently into the right lower quadrant of the abdomen and then suddenly releasing the hand, watching the patient's face for signs of discomfort (Another approach is to percuss the area, this elicits the same response and is kinder).
  - Pointing sign- at Mc Burney's point (a point one third the distance along a line drawn from the right anterior superior iliac spine to the umbilicus)
  - Signs of peritoneal irritation
    - Rovsing sign- on pressing left iliac fossa, pain occurs in RIF due to shift of bowel loops which irritate the peritoneum.
    - Psoas test (Cope's)- for Retrocecal appendix, hyperextension of hip causes pain in the RIF. The patient will get relief by flexing the right thigh at the hip which relaxes the psoas muscle.
    - Obturator test- for pelvic appendix, internal rotation of right hip causes pain in RIF due to irritation of obturator internus muscle.

- Baldwing test- positive in Retrocecal appendix- when legs are lifted off bed with knee extended, the patient complains of pain while pressing on abdomen (Ribs-ilium)
- Bastede sign-this is an obsolete sign in chronic appendicitis pain and tenderness is felt in the right iliac fossa on inflation of the colon with air.

- Hyperesthesia in Sherren's triangle (this triangle is formed by anterosuperior iliac spine, umbilicus and pubic symphysis).
- RECTAL EXAMINATION SHOULD ALWAYS BE DONE. It elicits tenderness on the right.
- Note: in elderly patients presentation tends to be atypical, leading to delayed diagnosis. The pain is less and it may present as small bowel obstruction. There is delayed leukocytosis. There is a higher risk of perforation and mortality than in young patients.
- Perforation at the time of surgery is more often seen in very young children (<5 years) and in older adults (>60 years) as a result of delayed diagnosis.

INVESTIGATIONS

1. Bloods: Full blood count with differential: Leukocytosis (>10, 000 in 90% of cases), usually with concomitant left shift (polymorphonuclear neutrophil predominance)
2. Urinalysis: helpful in ruling out genitourinary causes of symptoms. RBCs and WBCs may be present secondary to extension of appendiceal inflammation to the ureter. Significant hematuria or pyuria and bacteriuria from a catheterized specimen should suggest underlying urinary tract pathology.
3. Imaging
   - Plain X-ray
     - Abdominal X-ray: not particularly useful in most cases i.e. in clear-cut features of acute appendicitis. May reveal appendicolith/fecalith (<15% of cases). It is indicated if there is some clinical suspicion of intestinal obstruction or ureteric colic.

- Chest X-ray: air under the right side of the diaphragm signifies perforation.

➢ Ultrasound:

- Sensitivity of 85% and specificity of 92% for diagnosing appendicitis
- Positive findings: enlarged (> 6mm), hyperechoic thickened appendix wall (>2mm)-target sign i.e. appendiceal wall thickening, appendicolith, interruption of submucosal continuity, periappendicular fluid and non-compressible appendix (Note: the appendix will become compressible after perforation, this is a source of false negatives)
- Especially useful in ruling out gynecological pathology. It may rule out ureteric stones, pancreatitis, ovarian cysts, ectopic pregnancy and also confirm appendicular mass or abscess.

➢ Abdominal CT with contrast:

- Very sensitive (95-98%) and Somewhat specific (83-90%)
- Useful in identifying several other inflammatory processes that may present similarly to appendicitis
- Positive findings:
  - Dilation of appendix to >6mm in diameter
  - Thickening of appendiceal wall (representing edema)
  - Non-filling of the lumen by contrast or air.
  - Periappendiceal streaking (densities within perimesenteric fat)
  - Periappendicular fluid collection

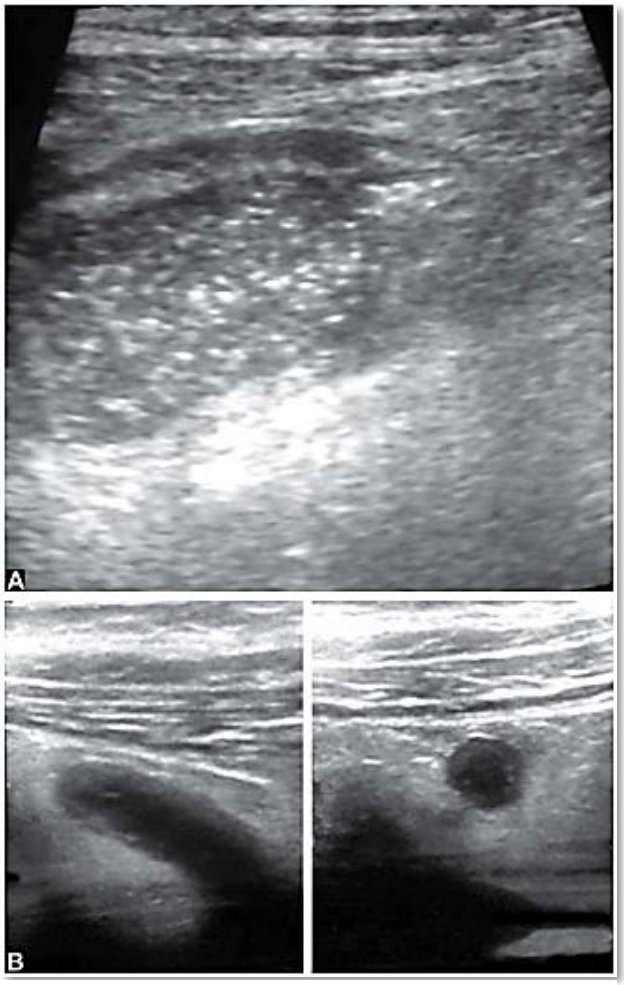


Figure 94: Ultrasound showing dilated non-compressible appendix in two different patients

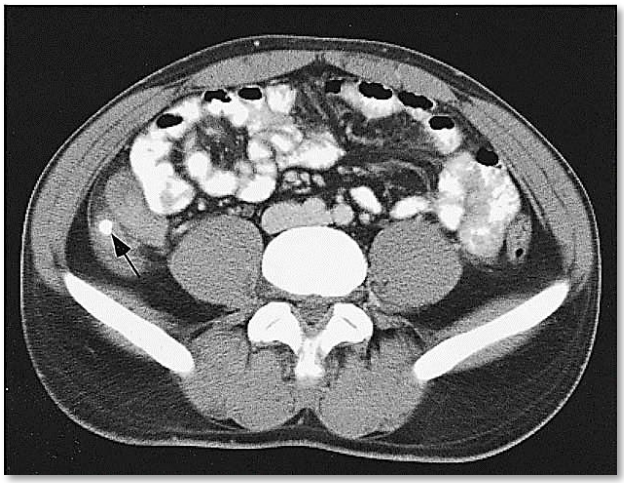


Figure 95: Abdominal CT scan demonstrating appendicolith and acute appendicitis

- Presence of appendicolith
- Presence of mass/abscess

4. Laparoscopy: most useful in female patients in the reproductive age
5. Aspiration cytology: peritoneal fluid is aspirated using a fine catheter introduced immediately below the umbilicus, the test is positive if more than 50% of the cells are neutrophils. This indicates the presence of infection.
6. Gravindex: to rule out ectopic pregnancy for female patients.

DIAGNOSIS

- Based on the Alvarado Score (MANTRELS Score)
  - M- migration of pain to right iliac fossa-1
  - A- anorexia- 1
  - N-nausea/vomiting-1
  - T- tenderness in right iliac fossa (Blumberg sign)-2
  - R-rebound tenderness-1
  - E- Elevated temperature (fever 37.3°C)-1
  - L-Leukocytosis (>10, 000)-2
  - S-shift towards neutrophils (neutrophilia)-1
- Outcomes:
  - <5: not sure
  - 5-6: compatible
  - 6-9: probable
  - >9: confirmed

Alvarado scoring for appendicitis (1986):	**Score**
Migrating pain	1
Anorexia	1
Nausea and vomiting	1
Tenderness in right iliac fossa	2
Rebound tenderness	1
Elevated temperature	1
Leucocytosis with count more than 10,000	2
Shift to left with neutrophilia in peripheral smear	1
Total score	**10**
Score less than 5: Not sure.	
Score between 5-6: Compatible.	
Score between 6-9: Probable.	
Score more than 9: Confirmed.	

TREATMENT

- Resuscitation (ABCs)
- Preoperative: IV fluids plus antibiotics (Ciprofloxacin + Metronidazole)
- For uncomplicated appendicitis, Appendicectomy can be done
  - Approaches:
    - Gridiron incision: perpendicular to McBurney's point
    - Lanz crease incision: centering at McBurney's point (cosmetically better)
    - Rutherford Morison's muscle: cutting incision (Muscles are cut upwards and laterally)
    - Right lower paramedian incision/lower midline incision: when in doubt or when there is diffuse peritonitis
    - Laparoscopic approach: becoming popular and better
    - Foweler-Weir approach: by cutting muscle medially over the rectus
  - Both open and laparoscopic techniques are appropriate. Laparoscopic appendectomy is associated with less postoperative pain, a shorter hospital course, better cosmesis and faster return to work
  - Postoperative antibiotics are given for 5 days (metronidazole and cefuroxime). IV fluids are also given. Once bowel sounds are heard, oral diet is started.
- Drugs (for 5 days)
  - Ciprofloxacin
  - Benzyl penicillin
  - Metronidazole
  - Gentamycin
- Analgesics
- Selected advanced cases with appendiceal abscess may initially be managed nonoperatively with antibiotics and

Troubles in Appendicectomy

- During surgery if appendix is found normal, other cause for symptoms should always be looked for e.g. Meckel's diverticulum, Crohn's disease, ovarian/pelvic causes in females, malignancy
- Appendicular tumor may be found. If it is the tip, appendicectomy is sufficient. It could be carcinoid tumor. If it is in the base right hemicolectomy is done.
- Absence of appendix (rare). Caecum and taeniae should be traced properly before finalizing it.
- Appendicular abscess/pelvic abscess formation
- Malignancy in the cecum is identified on table- right hemicolectomy should be done.
- If Crohn's disease is identified during surgery, appendicectomy can be done with care if the base of the appendix is normal but in rare occasions where the appendix is involve by Crohn's disease appendicectomy should not be done but treat only with antibiotics and steroids otherwise fistula can develop

percutaneous CT-guided abscess drainage. This may be followed by the development of a fecal fistula but this normally heals spontaneously.

- For perforation: Peritoneal washout and parenteral antibiotics.

DIFFERENTIAL DIAGNOSIS

GASTROINTESTINAL CONDITIONS

- Gastroenteritis
- Mesenteric adenitis
- Meckel's diverticulum
- Intussusception
- Typhoid fever
- Primary peritonitis

GENITOURINARY CONDITIONS

- Ectopic pregnancy
- Pelvic inflammatory disease
- Ovarian torsion/cyst/tumor
- Urinary tract infection/pyelonephritis
- Ureteral stone

CHILDREN

- Meckel's diverticulitis
- Acute colitis
- Round worm colic
- Acute iliac lymphadenitis
- Intussuception
- Lobar penumonia

ELDERLY

- Acute diverticulitis
- CA caecum
- Mesenteric ischemia
- Intestinal obstruction
- Aortic aneurysm leak
- Crohn's disease

FEMALES

- Ruptured ectopic pregnancy

- Ovarian cyst
- Ovarian abscess
- Oophritis
- Salpingitis
- Mittelschmerz- rupture of ovarian follicle during mid-menstrual period

COMPLICATIONS OF ACUTE APPENDICITIS

1. Relapse and recurrent appendicitis
2. Appendicular mass
   - Localization of infection occurring 3 to 5 days after an attack of acute appendicitis. Inflamed appendix, greater omentum, edematous cecum, parietal peritoneum and dilated ileum (ileus) forms a mass in the right iliac fossa.
   - The mass is tender, smooth, firm, well localized, not moving with respiration, not mobile, all borders well made out (well localized) and resonant on percussion. Patient may have fever and features of toxicity.
   - Investigation: Ultrasound confirms the mass
   - Treatment: Conservative as nature has already localized the infection. If disturbed it will cause a fecal fistula
     - Observation (temperature, BP, pulse)
     - Marking the mass to identify progression/regression
     - Antibiotics (Ampicillin, metronidazole, gentamicin or other drugs given depending on severity and requirement)
     - IV fluids
     - Analgesics
     - Initial nasogastric aspiration
       - Patient usually show response by 48 to 72 hours and mass reduces in size, temperature and pulse becomes normal. Appetite is regained. 90% of patients respond to conservative treatment
     - Patient is discharged and advised to come for interval appendicectomy after 6 weeks
   - Contraindication to conservative treatment:
     - When diagnosis is in doubt
     - Acute appendicitis in children and elderly
     - Burst, gangrenous appendicitis
     - In patients in whom diffuse peritonitis sets in

Criteria to discontinue *Ochsner-Sherren* regimen

- Patient becomes more toxic (tachycardia, high fever)
- Persistent vomiting
- Increase or spread of pain abdomen (means onset of diffuse peritonitis)
- Increased size of the mass
- Suppuration (abscess formation) in the mass

In these patients the regimen is discontinued.The patient is taken for immediate surgery, either through laparotomy or through classic approaches

3. Appendicular abscess:
   - Signs and symptoms:
     - High fever, features of toxicity, tender smooth, dull (to percuss), soft welling in right iliac fossa which lies towards right lateral and lower side with clear upper margin but indistinct lower margin
     - Increasing right lower quadrant pain, tender, fluctuant right lower quadrant mass palpable on rectal examination, anorexia, fever, localizing peritonitis, leukocytosis
   - Diagnosis: confirmed by CT scan
   - Treatment:
     - Antibiotics
     - Percutaneous or operative drainage, Pelvic abscess is drained per-rectally or through posterior colpotomy (in females)
   - Interval appendicectomy is done after 3 months
4. Perforation- 20% mortality
5. Generalized peritonitis, septicemia, Overwhelming sepsis and death
6. Portal pyaemia (Pylephlebitis)
   - Is rare nowadays. It is septic portal system thrombosis. It is commonly seen in immunosuppressed individuals. Infection spreads to liver through portal vein causing rapid multiplication of virulent organisms leading to septicemia (toxemia with hypotension, tachycardia), jaundice, tender palpable liver. Patient with be drowsy.
   - Treatment- antibiotics like cefoperazone, amikacin, metronidazole, meropenem, fluid management, ventilator support
   - It carries a poor prognosis.
7. Intestinal obstruction due to obstructive ileus, inflammatory adhesion, formation of band between appendix and omentum or between appendix and small bowel
8. Intraperitoneal abscess formation, either subphrenic or multiple small intraloop abscesses

COMPLICATIONS AFTER APPENDICECTOMY

1. Paralytic ileus
2. Reactionary hemorrhage due to slipping of ligature of the appendicular artery.
3. Residual abscess (pelvic, paracolic, local, subdiaphragmatic)
4. Portal pyemia (Pylephlebitis)
5. Recurrent intestinal obstruction due to formation of adhesions and kinking
6. Right inguinal hernia (direct)- due to injury to illoinguinal nerve

7. Wound infection and sepsis- 10%
8. Fecal fistula usually following drainage of an abscess:
   - Conservative treatment- antibiotics, IV fluids, dressing, zinc oxide cream over the skin, observation
   - Most of the time fistula subsides provided there is no distal obstruction by adhesion or kinking or specific causes like carcinoma or TB
   - If persists even after 6 weeks, resection of ileocecal segment and anastomosis is done
9. Respiratory problems and DVT

DIVERTICULAR DISEASE

CHRONIC INFLAMMATORY BOWEL DISEASE

COLON POLYPS

CARCINOMA OF THE COLON AND RECTUM

CARCINOMA OF THE ANUS

LIVER AND SPLEEN

LIVER AND SPLEEN INJURY

LIVER TUMORS

BENIGN TUMORS

- Only 5% of liver tumors are benign, these include:
  - Hepatocellular adenoma
  - Focal nodular hyperplasia
  - Hemangioma (most common)
    - Capillary (no clinical consequence)
    - Cavernous-7% (capable of attaining large size and rupturing)
  - Lipoma
- The incidence of adenoma is 1 per million in women with a history of oral contraceptive use. These medicines increase the risk by a factor of 40. This lesion most commonly occurs in women between 30 and 50 years.
- Adenoma and focal nodular hyperplasia are 5 times more common in female patients.

CLINICAL FEATURES

- Symptoms:
  - Patients with adenomas and hemangiomas can be asymptomatic or present with dull pain, rupture can produce sudden onset of severe abdominal pain. These lesions can also become large enough to cause jaundice or symptoms of gastric outlet obstruction (nausea and vomiting)
  - Focal nodular hyperplasia is rarely symptomatic.
- Signs:
  - Large lesions can be palpated
  - Jaundice may occur in patients if the tumor causes bile obstruction

DIAGNOSIS AND INVESTIGATIONS

- The lesions are most often found incidentally at laparotomy or on imaging studies requested for other reasons.

- Lab evaluation is often unremarkable although hemorrhage in an adenoma can lead to hepatocellular necrosis and a rise in transaminase levels.
- Hemangioma can cause a consumptive coagulopathy.
- Ultrasound differentiates cystic from solid lesions.
- Triple-phase computed tomography (CT) is the best study for distinguishing between various types of benign and malignant lesions but in certain cases, this determination is not possible.
  - Adenomas are low density lesions
  - Focal nodular hyperplasias may appear with a filling defect or central scar
  - Hemangiomas have early peripheral enhancement after contrast administration. Hemangiomas should not be biopsied because of the risk of bleeding.

MANAGEMENT

- Patients with adenoma who are using oral contraceptive should stop.
- If the lesion does not regress resection should be considered in otherwise healthy individuals because of the risk of malignant degeneration or hemorrhage.
- Relative contraindications to resection:
  - Tumor that is technically difficult to resect
  - Tumor of large size in which a large portion of the liver would need to be removed
- Symptomatic hemangiomas should be resected, if possible. Because focal nodular hyperplasia is not malignant and rarely causes symptoms it should not be resected unless it is found incidentally at laparotomy and is small and peripheral enough to be wedged out easily.

MALIGNANT TUMORS

- Malignant tumors of the liver may either be primary or secondary.
- The most common hepatic malignancy is as a result of metastases (Secondary tumor), the primary tumor is usually from colon, breast, lung (especially bronchogenic carcinoma), pancreas and stomach.

HEPATOCELLULAR CARCINOMA (HEPATOMA)

- This is a malignant tumor derived from hepatocytes frequently found in association with chronic liver disease particularly cirrhosis.
  - 80-90% of patients with hepatocellular carcinoma have underlying cirrhosis with alcoholic cirrhosis being the predominant type in western countries. In the Far east, post-hepatitis cirrhosis is more common.

- Hepatocellular carcinoma accounts for 80% of liver cancers and <2% of all cases.
- It is more common in males (3:1) and is usually diagnosed in the fifth or sixth decade.

ETIOLOGY AND RISK FACTORS

- Infectious:
  - Hepatitis B
  - Hepatitis C
  - Liver flukes
- Non-infectious
  - Cirrhosis (most commonly due to alcoholism)
  - Aflatoxins (Produced from fungi found in peanuts)
  - Hemochromatosis
  - Alpha-1 antitrypsin deficiency
  - Anabolic steroid use
  - Smoking, Vinyl chloride and oral contraceptives

CLINICAL FEATURES

- Symptoms:
  - Weight loss
  - Weakness
  - Dull pain in the right upper quadrant or epigastrium or shoulder
  - Nausea and vomiting
- Signs:
  - Signs of portal hypertension including splenomegaly and ascites
    - Splenomegaly (33%)
    - Ascites (50%)- due to hypoalbuminemia and decreased oncotic pressure
  - Jaundice
  - Non-tender hepatomegaly
  - A bruit can commonly be heard over a hepatocellular carcinoma due to its abundant vascularity

DIAGNOSIS AND INVESTIGATIONS

- Bloods:
  - Non-specific: ALP, AST, ALT, Gamma-glutamyl transferase (GGT), and des-gamma-carboxy prothrombin (DCP) are raised
  - Specific blood marker: alpha fetoprotein (AFP)

- Hepatitis panel because most cancers occur in the setting of liver disease and cirrhosis.

- Imaging: differentiate benign and malignant lesions
  - Contrast CT and Ultrasound can visualize the tumor. Ultrasound can distinguish cystic from solid lesions, whereas CT or MRI can reveal multiple lesions and clarify anatomic relationships. They can also demonstrate nodularity of the liver, hypersplenism, and portal hypertension indicative of underlying disease.
  - CT or ultrasound-guided needle biopsy will give the definitive diagnosis.

MANAGEMENT

- Before resection, assess underlying health of the liver using Child-Turcotte-Pugh scoring system.
  - Patients with Child Class B disease may tolerate a limited resection
  - Patients with Child class C disease will generally not tolerate a resection
- A decision to resect the tumor depends on comorbid disease and the location and size of the tumor.
- Surgical resection is the only cure, consisting of either:
  - Lobectomy
  - Segmental/wedge resection
- A 1-cm margin is required
- Transplant is also a possibility (Especially for patient with small tumors who are not candidates for resection because of tumor location or concomitant cirrhosis) however, there often is a high recurrence ate due to the continued presence of the underlying risk factors e.g. hepatitis B, C etc.
  - Milan criteria (tumor <5cm or no more than 3 tumors the largest of which is <3cm) will receive extra points on the liver transplant list.
- Local chemotherapy infusion into the hepatic artery, hepatic artery embolization and liposomal chemotherapy are the newer treatment options being explored.

Clinical Variable	1 Point	2 Points	3 Points
Encephalopathy	None	Stage 1–2	Stage 3–4
Ascites	Absent	Slight	Moderate
Bilirubin (mg/dL)	<2	2–3	>3
Bilirubin in PBC or PSC (mg/dL)	<4	4–10	10
Albumin (g/dL)	>3.5	2.8–3.5	<2.8
Prothrombin time (seconds prolonged or INR)	<4 sec or INR <1.7	4–6 sec or INR 1.7–2.3	>6 sec or INR >2.3

Child class A = 5–6 points; Child class B = 7–9 points; Child class C = 10–15 points.
PBC, primary biliary cirrhosis; PSC, primary sclerosing cholangitis; INR, international normalized ratio.

Figure 96: Child-Turcotte-Pugh Scoring system for Cirrhosis

PROGNOSIS

- Most patients die within the first 4 months if the tumor is not resection.

- After resection or transplant the 5-year survival rate is about 25%.

METASTATIC NEOPLASMS

- Arise most commonly from colon, breast, or lung (especially bronchogenic carcinoma), pancreas and stomach.

CLINICAL FEATURES

- Patients are usually asymptomatic until the disease is advanced and the liver fails
- Symptoms:
  - Fatigue
  - Weight loss
  - Epigastric fullness
  - Dull right upper quadrant pain
  - Ascites
  - Jaundice
  - Fever

DIAGNOSIS AND INVESTIGATIONS

- Bloods:
  - Increased ALP, GGT, lactic dehydrogenase (LDH), AST and ALT (though nonspecific)
  - Raised carcinoembryonic antigen (CEA) is seen in those with colonic cancer
- Imaging:
  - Mets will enhance on contrast CT
  - Intraoperative ultrasound with liver palpation is the most sensitive diagnostic tool

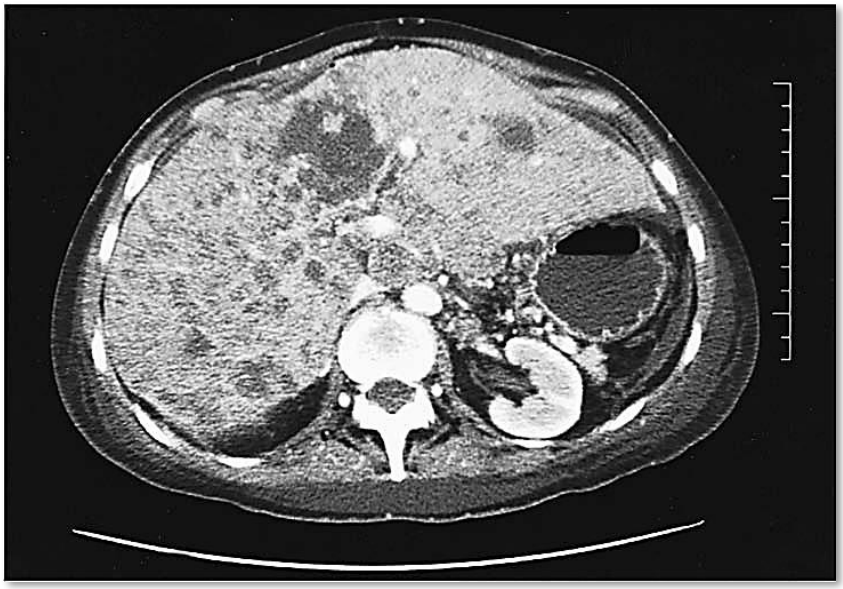


Figure 97: Abdominal CT in hepatic mets (Note slit like appearance of intrahepatic IVC secondary to compression by mets. Also note presence of ascites and bilateral pleural effusions, left greater than right) Image from First Aid for the Surgery Clerkship 2nd Ed..

MANAGEMENT

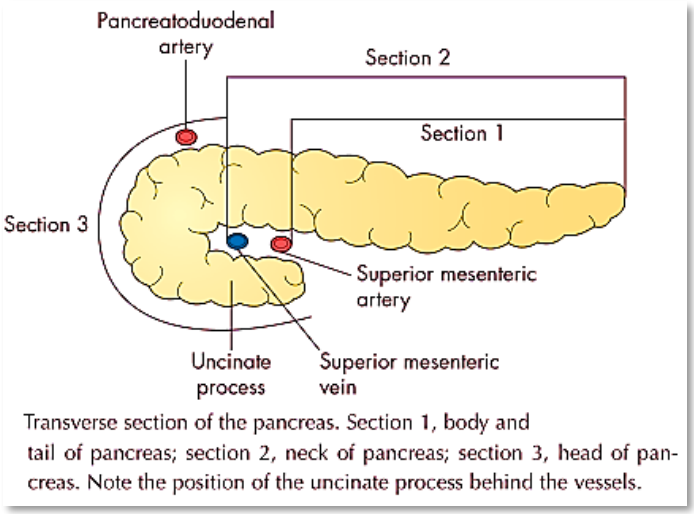
- Resection, if possible is the treatment of choice

- When colon cancer metastasizes to the liver, resection of up to 3 lesions has been shown to improve survival and should be attempted as long as the operative risk is not prohibitive.
- In general, liver metastases from other tumors should not be resected.

- Radiofrequency ablation (RFA) is now extensively done even for multiple lesions, and the short-term results are promising.

PANCREAS

- The pancreas also known as 'abdominal tiger'. Previously it was thought to act as a cushion for the stomach.
- The pancreas is a large central elongated retroperitoneal gland overlying the vertebral column (L1-L2) in the supracolic compartment of the abdomen.
- It lies posterior to the stomach, separated by the lesser sac.
- The pancreas is divided into:
  - Head (30% of the gland mass): lies in the concavity of duodenum, overlying the body of the L2 vertebra and the vena cava. Coming off the side of the pancreatic head and passing to the left and behind the superior mesenteric vein is the uncinate process of the pancreas.
  - Neck: the aorta and superior mesenteric vessels lie behind the neck of the gland. Behind the neck of the pancreas near its upper border the superior mesenteric vein joins the splenic vein to form the portal vein.
  - Body
  - Tail: reaches the hilum of the spleen
- The pancreas weighs about 80g and is about 15-20cm in length.
- The pancreas is a key regulator of digestion and metabolism through both endocrine (concerned with the production of hormones) and exocrine (concerned with the production of enzymes needed for digestion) function.
- 80-90% of the pancreas is composed of exocrine acinar tissue which are organized into lobules.



Transverse section of the pancreas. Section 1, body and tail of pancreas; section 2, neck of pancreas; section 3, head of pancreas. Note the position of the uncinate process behind the vessels.

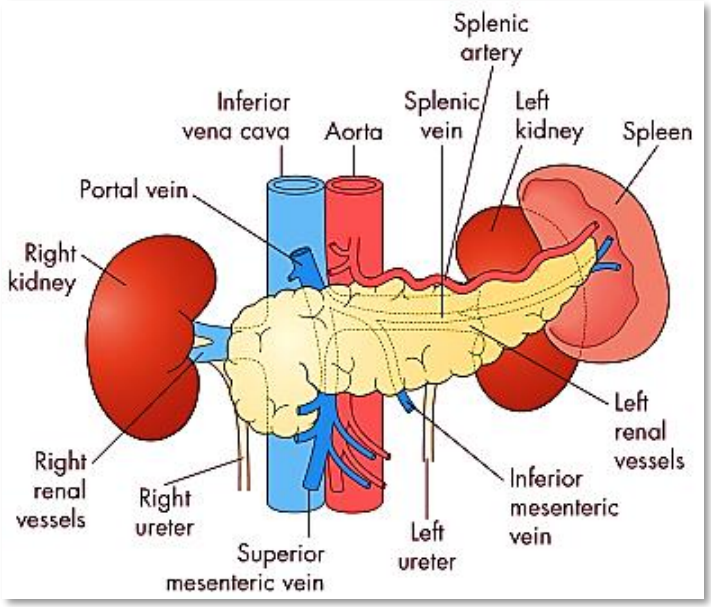


Figure 98: Posterior relation of the pancreas (Image from Bailey and Love Short Practice of Surgery)

- The main pancreatic duct branches into interlobular and intralobular ducts, ductules and finally acini.
  - The main duct is lined by columnar epithelium which becomes cuboidal in the ductules.
  - Acinar cells are clumped around a central lumen which communicates with the duct system.
  - The main pancreatic duct (duct of Wirsung) begins in the tail of the pancreas and runs on the posterior surface of the body and head of the pancreas receiving numerous tributaries at right angle along its length (Herring bone pattern).
  - The main pancreatic duct is joined by the common bile duct (a combination of the common hepatic duct and the cystic duct) in the wall of the second part of the duodenum to form hepatopancreatic ampulla of Vater and opens on the summit of major duodenal papilla (8-10 cm from the pylorus)
- The accessory pancreatic duct (Duct of Santorini) begins in the lower part of the head and opens into the duodenum at minor duodenal papilla (6-8cm from the pylorus)
- The endocrine pancreas consists of islets of Langerhans which comprise of 4 recognized cell types:
  - A cells (α-cells)-20% which produce glucagon
  - B cells (β-cells)-75% which produce insulin, islet amyloid-associated polypeptide and pancreastatin
  - D cells which secrete somatostatin and probably gastrin
  - F cells which secrete pancreatic polypeptide
- Within an islet, the B cells form an inner core surrounded by other cells. Capillaries draining the islets cells drain into the portal vein, forming a pancreatic portal system.
- The pancreas has a complex vascular supply and venous/lymphatic drainage and is surrounded by the duodenum and upper jejunum.

- Arterial blood supply is from:
  - Pancreatic branches of splenic artery
  - Superior pancreaticoduodenal artery (a branch of the gastro-duodenal artery which arises from the hepatic artery a branch of the coeliac trunk)
  - Inferior pancreaticoduodenal artery (a branch of the superior mesenteric artery)
- Venous drainage follows a similar pattern to the arteries and is by the superior and inferior pancreaticoduodenal veins into the portal vein.
- The pancreas receives its parasympathetic supply from the vagus and sympathetic innervation from the splanchnic nerves.
- During the embryonic development of the pancreas, there are 9 key processes that occur.
  - Day 26: Dorsal pancreatic duct arises from the dorsal side of the duodenum.
  - Day 32: Ventral bud arises from the base of the hepatic diverticulum
  - Day 37: Contact occurs between the buds. Fusion by the end of week 6
  - Week 6: Ventral bud produces the head and uncinate process
  - Week 6: Ducts fuse
  - Week 6: Ventral duct and distal portion of the dorsal duct form the main duct (Duct of Wirsung)
  - Week 6: Proximal dorsal duct forms the duct of Santorini
  - Month 3: Acini appear
  - Month 3-4: Islets of Langerhans appear and become biologically active
- Malrotation of the ventral bud in the 5th week results in an annular pancreas while the mode of ductule fusion in the 17th week produces the various possible ductular patterns.
- Between 12th and 40th weeks of fetal life, the pancreas differentiates into exocrine and endocrine elements.
- The primitive ducts and their ductules are responsible for the lobular arrangement of the pancreas.
- The pancreas is associated with both endocrine and exocrine function.

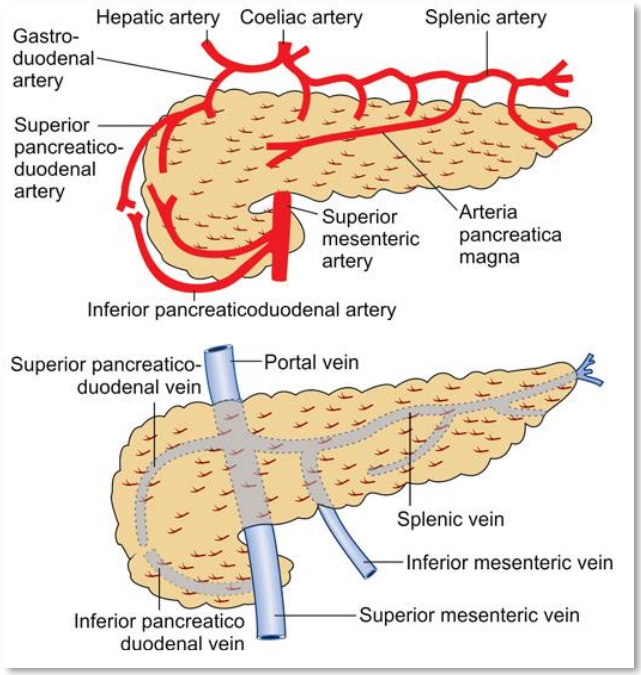


Figure 99: Arterial and venous blood supply of the pancreas (Images from SRB's Manual of Surgery 2nd Ed)

- The exocrine secretion contains water and bicarbonate ions and digestive enzymes secreted as inactive precursors (zymogens) into the duodenum in response to neural and hormonal influences. Enzyme activation begins after the zymogens enter the duodenum, where mucosal enterokinase cleaves trypsinogen into trypsin which activates the other enzymes.
  - In response to a meal, the pancreas secretes digestive enzymes in an alkaline (pH 8.4) bicarbonate rich fluid. Spontaneous secretion is minimal
  - The hormone secretin (produced from the S cell of the duodenal mucosa) evokes a bicarbonate rich fluid (via cAMP cascade).
  - Cholecystokinin (CCK) also known as pancreozymin (produced from the I cells of the duodenal mucosa) is produced in response to food. CCK is responsible for enzyme release (Through phospholipase C, and calcium mechanism).
  - The protein part of the pancreatic juice is secreted by acinar cells, duct cells secrete fluid and electrolytes.
  - Vagal stimulation increases the volume of secretion.
  - About 6-20g of digestive enzymes enter the duodenum each day.
  - During eating, cephalic phase stimulates 10% of pancreatic secretion through peripherally mediated acetylcholine, gastric phase stimulates further 15% of pancreatic secretion through gastrin release and vagal stimulation and finally in the main intestinal phase 75% of the pancreatic juice is secreted by release of secretin due to duodenal acidification and by release of bile and CCK following entry of fat and proteins in the duodenal.
- The main stimulus for insulin secretion is an increase in blood glucose level (hyperglycemia)
- Other stimuli for insulin release include: amino acids (arginine and lysin) and other hormones (glucagon, growth hormone, cortisol, placental lactogen, estrogens). Insulin release is also enhanced by free fatty acids in order to prevent ketoacidosis which would otherwise occur during fasting in normal individuals.
- Glucagon secretion is stimulated by low blood glucose levels (hypoglycemia). It has catabolic actions and initiates glycogenolysis, gluconeogenesis and break down of fat into free fatty acids and glycerol.
- Glucagon inhibits protein synthesis.
- Somatostatin is an inhibitory hormone. It inhibits growth hormone, insulin and glucagon secretion, gastric acid and pepsin secretion, the release of pancreatic enzymes and intestinal motility. It reduces splanchnic blood flow.

- The physiological function of pancreatic polypeptide remains unknown.
- Disorders of surgical importance include:
  - Acute pancreatitis
  - Chronic pancreatitis
  - Pancreatic cancer

PANCREATITIS

PANCREATITIS

- This is inflammation of the gland parenchyma of the pancreas.
- For clinical purposes it is useful to divide pancreatitis into:
  - Acute Pancreatitis: presents as an **emergency** and is usually **reversible**.
    - Acute pancreatitis
    - Acute relapsing pancreatitis
  - Chronic Pancreatitis: **prolonged** and frequently **lifelong disorder** resulting from the development of fibrosis within the pancreas. Continuous inflammatory changes result in irreversible morphological changes.
    - Chronic pancreatitis
    - Chronic relapsing pancreatitis

Marseilles' Classification of pancreatitis

1. Acute pancreatitis
2. Acute relapsing pancreatitis
3. Chronic relapsing pancreatitis
4. Chronic pancreatitis

ACUTE PANCREATITIS

- Acute pancreatitis is an acute condition presenting with abdominal pain and is usually associated with raised pancreatic enzyme levels in the blood or urine as a result of pancreatic inflammation and hemorrhage.
- Acute pancreatitis may recur but is usually reversible.
- The disease may occur at any age, with a peak in young men and older women.
- It can be classified as (Atlanta Classification):
  - Mild acute pancreatitis (edematous): characterized by interstitial edema of the gland and minimal organ dysfunction. 80% of patients have this. It carries a mortality of 1%.

Acute pancreatitis should be suspected in the alcoholic who develops an "upper" acute abdomen. The classic picture has a rapid onset (a couple of hours). The pain is constant, epigastric, radiating straight through to the back with nausea, vomiting and retching. Physical findings are relatively modest, found in the upper abdomen.

- Severe acute pancreatitis (necrotizing): characterized by pancreatic necrosis, a severe systemic inflammatory response and often multi-organ failure. This carries a mortality between 20-50% (1/3 of patients die due to multiple organ failure in the early stages while others die after the first week of onset due to septic complication).

- Pancreatitis can alternatively also be classified as
  - Acute edematous pancreatitis: occurs in alcoholics or patients with gallstones. Presents as epigastric and mid-abdominal pain that starts after a heavy meal or bout of alcoholic intake. Pain is constant, radiates straight through to the back and is accompanied by nausea, vomiting and (after the stomach is empty) continued retching. There is tenderness and mild rebound tenderness in the upper abdomen. During the early stages serum lipase and amylase are elevated and after a couple of days their levels increase in urine. A key finding to establish the edematous nature is an elevated hematocrit. Resolution follows a few days of pancreatic rest (NPO, NG suction, IV fluids).
  - Acute hemorrhagic pancreatitis: This is a much deadlier disease. It starts as the edematous form but an early lab clue is lower hematocrit (the degree of amylase elevation does not correlate with the severity of the disease). Other findings have been catalogued by the Ranson's criteria (See assessment of severity). Intensive care is needed as a common final pathway for death is the development of multiple pancreatic abscesses that necessitate daily CT scans and drainage as well as antibiotics.
  - Acute suppurative pancreatitis (Pancreatic abscess): may become evident in someone who was not getting CT scans, because persistent fever and leukocytosis develop about 10 days after the onset of pancreatitis. Imaging studies done at that time will reveal collection (or collections) of pus. Percutaneous drainage and antibiotics will be indicated.

PATHOPHYSIOLOGY

- The underlying mechanism of injury in pancreatitis is thought to be premature activation of pancreatic enzymes within the pancreas, leading to a process of auto-digestion.
- Multiple etiologies have been identified with alcoholism and gallstone disease accounting for 80-90% of cases.
  - Pancreatic ductal obstruction (by gall stones, duodenal ulcers, duodenal crohn's, periampullary diverticulm/tumor, trauma, pancreatitic duct stricture,

pancreatic divisum, ascaris, or Clonorchis Sinensis) leads to ductal hypertension.

- Alcohol causes direct toxicity, hypersecretion of gastric and pancreatic juices, reflux, plugging of pancreatic proteins, injury by release of free radicals like superoxide and hydroxyl, spasms of Oddi and stimulates trypsinogen.

- Anything that injures the acinar cell and impairs the secretion of zymogen granules or damages the duct epithelium and thus delays enzymatic secretion can trigger acute pancreatitis.
  - Trypsinogen is first activated and premature activation of trypsin leads to activation of other enzymes.
  - Proelastase is activated to elastase and it causes capillary rupture. Prolipase is activated to lipase and causes metabolism of triglycerides to glycerol and fatty acids that combine with calcium forming saponified fat. These effects result in sequestered fluid, saponified fat, blood, and toxins all together forming a chicken broth fluid.
- Once cellular injury has been initiated, the inflammatory process can lead to pancreatic edema, extensive hemorrhage and eventually necrosis.
  - 2 types of necrosis are seen in acute pancreatitis:
    - Liquefactive necrosis: due to denaturation of enzymes
    - Fat necrosis: due to saponification of peri-pancreatic fat
- Lecithinase, amylase and factors like lysolecithinase, prostaglandins, bradykinins, kallikrein, myocardial depressant factor, free radicals, cytokine, tumor necrosis factor and platelet activation factor are also released to have local and systemic effects.
- As inflammatory mediators are released into circulation systemic complications can arise such as hemodynamic instability (hypovolemic shock), bacteremia (Due to translocation of gut flora), acute respiratory distress syndrome and pleural effusions, gastrointestinal hemorrhage, renal failure and disseminated intravascular coagulation (DIC).
  - Toxins released may lead to acute tubular necrosis and so acute renal failure
  - Left sided diaphragm gets elevated and left sided pleural effusion occurs
  - Lecithinase reduces the surfactant in the alveoli of lung and infection leads to pulmonary insufficiency, ARDS and respiratory failure
  - Because calcium is utilized for saponification hypocalcemia sets in
  - Diffuse oozing in pancreatic bed occurs which utilizes platelets and causes DIC

- In severe cases extensive necrosis with hemorrhage occurs causing acute hemorrhagic necrotizing pancreatitis (Fulminant pancreatitis) that carries a high mortality.

ETIOLOGY (TRAPNELL'S ETIOLOGICAL CLASSIFICATION)

- Most common causes:
  - Alcohol (40--50%): alcohol causes contraction of the sphincter of Oddi at the ampulla of vater, this decreases the drainage of pancreatic enzymes in duodenum thus increasing the risk of premature activation of pancreatic enzymes. It is common in men
    - Other implicated mechanisms include: the effect of alcohol on diet, malnutrition, direct toxicity of alcohol and hyperlipidemia.
  - Gallstones (40%): Passage of gallstones down the common bile duct causes obstruction of ampulla leading to premature activation of pancreatic enzymes. Patients who have small gallstones and a wide cystic duct may be at a higher risk of passing stones. Gallstones are common in patients above 60 years. It is common in women.
- Other causes include:
  - Abdominal trauma (e.g. RTA in which a person has the seatbelt around abdomen and not pelvis)
  - Hyperparathyroidism and Hypercalcemia (Calcium is required for activation of enzymes)
  - Hyperlipidemia, Diabetes, Porphyria, Autoimmune diseases (associated with primary sclerosing cholangitis, Sjogren's syndrome, biliary cirrhosis)

Causes of pancreatitis

P- posterior perforation of peptic ulcer

A-Alcohol

N-neoplasm

C- Cholelithiasis (gall stones)

R- Renal disease (end stage)

E- ERCP

A- Anorexia (malnutrition)

T- Trauma

I- Infections

T-Toxins (Drugs)

I-incineration (burns)

S- Surgery/Scorpion bite

- Drugs (corticosteroids, valproic acid, thiazides, estrogens, isoniazid, cimetidine, salicylic acid, tetracycline)
- Scorpion stings, Vascular diseases
- Viral infection (mumps, coxsackie B, cytomegalovirus, EBV-infectious mononucleosis), *Mycoplasma pneumoniae* (usually mild),
- Rupture of a posterior duodenal ulcer
- Autoimmune pancreatitis
- Malnutrition
- Post Endoscopic retrograde cholangiopancreatography (in 1-3% patients): due to disruption of the duct and enzyme extravasation.
- Following biliary, upper gastrointestinal or cardiothoracic surgery
- Fulminant liver failure
- Hereditary pancreatitis: this is a rare familial condition associated with mutations of the cationic trypsinogen gene. In normal individuals, small amount of spontaneous trypsinogen activation into trypsin occurs however further activation is prevented by trypsin inhibitors. In hereditary pancreatitis the gene mutation causes defective trypsin inhibitors and trypsin becomes resistant to its inhibitor leading to accumulation of active trypsin in the pancreas. Patients often suffer acute pancreatitis in their teens which progresses to chronic pancreatitis in the next 2 decades. They have a 40% risk of developing pancreatic by the age of 70 years.
- Pancreatic duct obstruction: tumor (Ampullary tumor), pancreatic divisum, Ascaris infestation
- Idiopathic (10-20%): always check for serum calcium level, fasting lipid profile, autoimmune markers and viral titers in patients with idiopathic acute pancreatitis.

Effects of alcohol on pancreas

- Hypertriglyceridaemia, fatty acids and ethyl ester metabolites → pancreatic injury
- Formation of pancreatic juice which contains high enzymes but low enzyme inhibitors → enzyme activation within pancreas; precipitation of proteins, intraductal plug formation, ductal hypertension and obstruction → pancreatic injury
- Formation of O_2 free radicals inside the pancreas → pancreatic injury
- Direct pancreatic acinar injury
- Sphincter of Oddi spasm by alcohol leading into ductal obstruction
- Repeated subclinical acute pancreatitis causes fibrosis and chronic pancreatitis

- Note: it is essential to establish the etiology of pancreatitis before labelling it as idiopathic as the etiology needs to be addressed as part of the management.

CLINICAL FEATURES

- Pain is the cardinal symptom
  - It develops quickly reaching maximum intensity within minutes rather than hours and persists for hours or even days.
  - The pain is severe, constant and refractory to usual doses of analgesics.
  - Pain is usually experienced first in the epigastric region (with radiation to the back in 50% of patients) but may be localized to either upper quadrant or felt diffusely throughout the abdomen.
  - Some patients may gain relief by sitting or leaning forwards.
  - Acute pancreatitis can mimic most causes of the acute abdomen and should rarely be discounted in differential diagnosis.
  - Differential diagnosis:
    - Sudden onset: perforated ulcer,
    - Right upper quadrant pain: biliary colic and acute cholecystitis
    - Radiation to the chest: Myocardial infarction, pneumonia or pleuritic pain
- Nausea, vomiting and retching. Retching may persist despite the stomach being kept empty by nasogastric aspiration.
- Hiccoughs can be troublesome and may be due to gastric distension or irritation of the diaphragm.
- Bleeding into the fascial planes can produce bluish discoloration of the flanks (Grey Turner's sign) or umbilicus (Cullen's sign).
  - Neither sign is pathognomonic of acute pancreatitis.
  - Cullen's sign was first described in association with rupture of an ectopic pregnancy.
- Subcutaneous fat necrosis may produce small, red, tender nodules on the skin of the legs.
- Occasionally hematesis or melena can occur due to duodenal necrosis, gastric erosions, decreased coagulability/DIC
- Neurological derangements due to toxemia, fat embolism, hypoxia, respiratory distress can occur. It may be mild psychosis to coma.

DIAGNOSIS

HISTORY

- Age and sex: incidence peaks between 40-50 years but can occur at any age, equal incidence in men and women.
- Symptoms:
  - Sudden epigastric pain which may be relieved by leaning forward

- Pain radiates to the back
- Drugs don't relief pain
- Frequent vomiting, nausea and retching
- Low grade fever
- Many patients have eaten an unusually large meal or drunk some alcohol before the pain began

- Previous history of biliary tract disease (gallstone) as well as indigestion or illness with mumps (in children).
- Previous history of pancreatitis.
- History of taking corticosteroids, Isoniazide, cimetidine, thiazide diuretics or estrogen based therapies.
- Social history:
  - Alcohol intake

EXAMINATION

- General examination:
  - Patient may be well or gravely ill with profound shock, toxicity and confusion.
  - Patients lie still because pain is severe and if they are pale and sweating it means they may have become hypovolemic. If the patient has become hypovolemic the JVP and blood pressure may be low.
  - May become grey, apprehensive, dyspneic and cyanosed if respiration is impaired.
  - Tachypnea is common, tachycardia is usual and hypotension may be present
  - There may be a slight tinge of jaundice if pancreatitis has been caused by a stone lodged in the lower end of the bile duct. Mild jaundice may also appear on the second or third day of the illness if edema in the head of the pancreas is causing compression of the bile duct.
  - Pyrexia may be present but temperature is rarely greatly elevated (low grade fever). If acute swinging pyrexia is present suspect cholangitis.
- Abdominal examination:
  - Inspection
    - If pain is severe and the tone of the abdominal muscles is increased the abdomen will not move with respiration.
    - A paralytic ileus may develop causing mild abdominal distention.
    - Bruising and discoloration in the left flank (Grey Turner's sign) and around the umbilicus (Turner's sign): these are rare and late signs seen in patients with very severe hemorrhage.

- There may be bluish discoloration of the inguinal ligament (Fox's sign)

➢ Palpation: Tenderness and guarding in the upper abdomen. These signs may be mild. A mass can develop in the epigastrium due to inflammation.

➢ Percussion: may cause pain if there is peritonitis and may be dull over any pseudocyts that are developing.

➢ Auscultation: bowel sounds are usually present in first 12 to 24 hours but fade away if a paralytic ileus develops.

- Note: a pleural effusion is present in 10-20% of patients. Pulmonary edema and pneumonitis are also described and may give rise to the differential diagnosis of pneumonia or myocardial infarction.
- **Remember:**
  - ➢ **Bruising and discoloration in the left flank and umbilicus can occur after a large intraperitoneal or retroperitoneal hemorrhage from another cause such as a leaking abdominal aortic aneurysm.**
  - ➢ **A number of patients develop a collection of inflammatory exudates in the lesser sac. This is initially suggested by fullness in the epigastrium which may become a more prominent mass if a pseudocyt or abscess develops**
  - ➢ **Acute pancreatitis can be extremely difficult to diagnose and as it has no distinctive features, it is often forgotten and missed thus should always be considered when dealing with an acute abdomen.**

INVESTIGATIONS

DIAGNOSTIC

BLOOD INVESTIGATIONS

- Serum amylase (increases 3 to 4 times normal- >1000 somogyi units) and serum lipase
  - ➢ Serum lipase is more specific because serum amylase is raised when there is injury to salivary gland.
    - Serum lipase is only found in gastric and intestinal mucosa and liver.
    - Serum amylase is found in the salivary glands, small bowel, ovaries, testes and skeletal muscle
  - ➢ A normal serum amylase does not exclude acute pancreatitis particularly if the patient has presented a few days later.
  - ➢ Hypocalcemia is a poor prognostic indicator (calcium is used in saponification in fat necrosis)

- Note Serum amylase or lipase are seen 12 to 48 hours however, amylase and lipase are present in urine on the 3rd to 6th day.

- Serum calcium, Fasting lipid profile, autoimmune markers and viral titers especially in idiopathic pancreatitis.

IMAGING

- Abdominal ultrasound- It is thus done to rule out tumors, gall stones etc. It does not establish the diagnosis of acute pancreatitis, the swollen pancreas may be seen but ultrasonography should be performed within 24 hours in all patients to detect gallstones as a potential cause, rule out acute cholecystitis as a differential diagnosis and determine whether the common bile duct is dilated.
- Plain X-ray- plain erect CXR and AXR are not diagnostic of acute pancreatitis but are useful in the differential diagnosis.
  - Suggestive features on AXR:
    - Generalized or local ileus ('Sentinel loop' of dilated proximal small bowel i.e. the mid to distal duodenum or proximal jejunum located in the left upper quadrant. A sentinel loop is characterized as distention and/or air-fluid level near a site of inflammation, in pancreatitis it is secondary to pancreatitis associated ileus)
    - Distension of transverse colon with collapse of descending colon (colon cut off sign)
    - Air fluid level in the duodenum (signifying perforation)
    - Renal halo sign
    - Obliteration of psoas shadow
    - Calcified gallstones or pancreatic calcification may sometimes be seen
    - Localized ground glass appearance (Haziness due to retroperitoneal accumulation of fluid)
  - CXR may show pleural effusion and in severe cases a diffuse alveolar interstitial shadowing suggesting acute respiratory distress.
    - Basal atelectasis
    - Sub-diaphragmatic fluid collection
    - Pleural effusion
- Contrast-enhanced CT: not necessary for all patients, particularly those deemed to have a mild attack on prognostic criteria. It is done when diagnosis is uncertain, in severe pancreatitis and in patients with suspected fluid collection, pseudocyst or a pseudoaneurysm as well as organ failure with signs of sepsis or progressive clinical deterioration.

- Cross-sectional MRI may yield similar information as CT.
- Endoscopic retrograde cholangiopancreatography (ERCP) and Endoscopic ultrasound (EUS) can help in detecting stones in the common bile duct and directly assessing the pancreatic parenchyma. ERCP allows identification and removal of stones in the common bile duct in gallstone pancreatitis. In patients with severe acute gallstone pancreatitis and signs of ongoing biliary obstruction and cholangitis an urgent ERCP should be done.
- Note: occasionally the diagnosis is only made at laparotomy (as shown on the image)
- Urinary trypsin and activated peptide (TAP), serum trypsinogen-2 and C-reactive protein (CRP) are also useful in differentiation of mild from severe disease. In this respect CRP is only reliable as an index of severity after the 2nd or 3rd day of the disease. CRP levels in excess of 100mg/dl usually indicates necrotizing pancreatitis

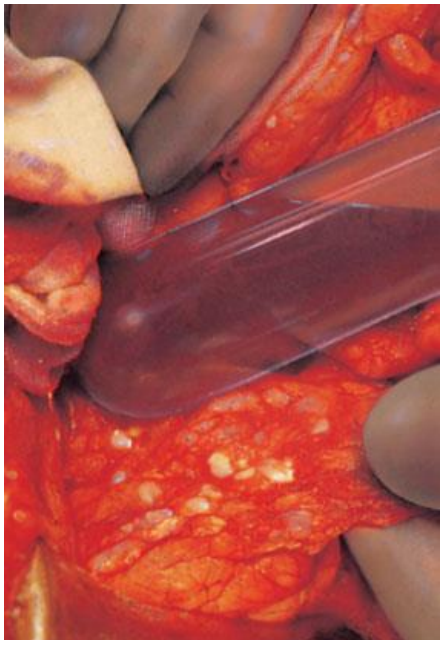


Figure 100: Widespread fat necrosis of the omentum. A test tube filled with blood-stained peritoneal fluid. This specimen was rich in amylase. Fat necroses are dull, opaque, yellow-white areas suggestive of drops of wax. They are most abundant in the vicinity of the pancreas but are widespread in the greater omentum and the mesentery. At necropsy, they can sometimes be demonstrated beneath the pleura and pericardium and even the subsynovial fat of the knee joint. Fat necroses consist of small islands of saponification caused by liberation of lipase which splits fats into glycerol and fatty acid. Free fatty acids combine with calcium to form soaps (fatty necrosis)

SUPPORTIVE

- FBC, U&E, Creatinine, Liver enzymes
- Chest X-ray- for effusion and ARDS.

DIFFERENTIAL DIAGNOSIS

- Gastritis/Perforated duodenal ulcer
- Perforated esophagus/peptic ulcer
- Acute cholecystitis and acute cholangitis (both may coexist with acute pancreatitis)
- Acute appendicitis
- Acute upper small bowel obstruction
- Acute mesenteric ischemia
- Rupture abdominal aortic aneurysm
- Ectopic pregnancy
- Salpingitis
- Intestinal obstruction
- Diabetic ketoacidosis
- Myocardial infarction
- Pneumonia

ASSESSING SEVERTIY

- The Ranson criteria are well-known prognostic signs used for predicting the severity of disease on the basis of clinical and laboratory results.
- The ability to predict a patient's risk of infectious complications and mortality at the time of admission and during the initial 48 hours allows institution early therapy during admission.
- In patients with less than 3 of the 11 prognostic factors, the mortality rate is low (1%), with 3 or 4 factors, 18%, with 5 to 6 factors, 50% and with more than 6 factors, 90%.
- The Glasgow grading is also a similar scoring system

Scoring systems to predict the severity of acute pancreatitis: in both systems, disease is classified as severe when three or more factors are present

Ranson score	Glasgow scale
On admission	**On admission**
Age > 55 years	Age > 55 years
White blood cell count > 16×10^9 l^{-1}	White blood cell count > 15×10^9 l^{-1}
Blood glucose > 10 mmol l^{-1}	Blood glucose > 10 mmol l^{-1} (no history of diabetes)
LDH > 700 units l^{-1}	Serum urea > 16 mmol l^{-1} (no response to intravenous fluids)
AST > 250 Sigma Frankel units per cent	Arterial oxygen saturation (PaO_2) < 8 kPa (60 mmHg)
Within 48 hours	**Within 48 hours**
Blood urea nitrogen rise > 5 mg%	Serum calcium < 2.0 mmol l^{-1}
Arterial oxygen saturation (PaO_2) < 8 kPa (60 mmHg)	Serum albumin < 32 g l^{-1}
Serum calcium < 2.0 mmol l^{-1}	LDH > 600 units l^{-1}
Base deficit > 4 mmol l^{-1}	AST/ALT > 600 units l^{-1}
Fluid sequestration > 6 litres	

AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; PaO_2, arterial oxygen tension.

- Both scoring systems have been replaced by the Acute Physiology and Chronic health evaluation (APACHE II) scoring system which gives better differentiation and has an added advantage that it can be used at any time in the course of the disease.
- The APACHE II system allocates 3 sets of points: A, B and C
  - A: assessment of clinical parameters e.g. Vital signs, electrolytes, arterial blood gasses etc.
  - B: points allocated in accordance with age
  - C: points added for comorbid disease or chronic health of patient.
- The APACHE II score is the sum of A, B and C, if this exceeds 9 the patient has severe acute pancreatitis. Mortality is very high if the score increases after admission.

MANAGEMENT

- Management is dependent on cause but is usually
  - Conservative (medical 70-90%):
  - Surgical (10-30%)
- Note:
  - For mild pancreatitis a brief period of fasting may be sensible in a patient who is nauseated and in pain, but there is little physiological justification for this. Antibiotics are not always indicated. Apart from analgesics and anti-emetics, no drugs or interventions are warranted unless there is evidence of deterioration.
  - For severe pancreatitis a more aggressive approach is needed. The patient should be admitted to a high-dependency or an intensive care unit and monitored invasively. Aggressive fluid resuscitation guided by frequent measurement of vital signs, urine output and central venous pressure. Supplemental oxygen should be given and serial arterial blood gas analysis should be done. Blood should be sent for hematocrit, clotting profile, blood glucose, liver enzymes and renal function tests, serum levels of calcium and magnesium.

- Conservative management involves the mnemonic (PANCREAS):
  - P- pain relief, Protease inhibitors, plasma
    - For pain relief use pethidine and other analgesics, morphine should not be used as it causes spasms of the sphincter of Oddi.
    - Protease inhibitors: aprotinin, antisnake venoma, or EACA can be given though they have not been shown to improve outcome.
    - Plasma: fresh frozen plasma and platelets may be required in anticipation of DIC and hemorrhage.
  - A- Antibiotics and anticholinergics
    - Antibiotics- 3rd generation cephalosporins (cefotaxine, cefuroxime, cefoperazone, ceftazidime), Ciprofloxacin plus metronidazole, and imipenem. To reduce the anticipated sepsis. Should not be given beyond 14 days and should be guided by microbiological cultures
    - Anticholinergics to reduce sphincter pressure
  - N- nasogastric aspiration, Nasal Oxygen and Nutritional support (total parental nutrition though there is no data to support a practice of 'resting' the pancreas and feeding only by parenteral or nasojejunal routes. If nutritional support is felt to be necessary, enteral nutrition via NGT should be used)

ACUTE PANCREATITIS MANAGEMENT BOX

- Admit to ICU
- P- Pain management-(Pethidine), Plasma products (FFP, Platelets if in DIC or hemorrhage)
- A-antibiotics: 3rd generation cephalosporins (cefotaxine, cefuroxime, cefoperazone, ceftazidime), Ciprofloxacin plus metronidazole, and Imipenem
- N- Insert NGT, Cannulate, Catheterize and Consider Endotracheal intubation when in distress. Give Nasal Oxygen. Consider nutrition (TPN or NGT feeds).
- C- Calcium gluconate: 10ml of 10% 8 hourly
- R- Rehydrate: 250-400ml/hour ringer lactate, normal saline, dextrose saline, plasma and fresh blood transfusion/packed cells.
- E- electrolyte management and endotracheal intubation
- A- Antacids: IV ranitidine 50mg 6 hourly or IV omeprazole 40mg BD
- S- somatostatin analogue (octreotide 50µg as loading dose IV, 50 µg in 1 hour in 5% dextrose for maintenance dose)
- Blood should be sent for hematocrit, electrolytes, clotting profile, blood glucose, Liver and renal function, serum levels of calcium and magnesium
- Monitor vitals and urine output
- Surgery if patient deteriorate

- C- calcium gluconate 10ml 10% 8 hourly (for hypocalcemia), Calcitonin, CVP line (to monitor, for fluid therapy and total parental nutrition), Catheterization (monitor urine output 0.5-1ml/kg/hr) and cannula
- R- rehydration, ranitidine, respiratory support (ventilator), resuscitation when required
  - Rehydration with 250-400ml/hour ringer lactate, normal saline, dextrose saline, plasma and fresh blood transfusion/packed cells as there is a lot of fluid sequestration and third spacing.
  - IV ranitidine 50mg 6 hourly or IV omeprazole 40mg BD to prevent stress ulcers
- E- endotracheal intubation, Electrolyte management
  - Monitor electrolytes and manage in ICU where endotracheal intubation, ventilatory support, tracheostomy can be done if needed
- A- antacids
- S-somatostatin and its analogue (Octreotide-50µg as loading dose IV, 50 µg in 1 hour in 5% dextrose for maintenance dose) to reduce pancreatic secretions though their use has not shown any improvement in outcome.

- There should also be management of complications as they arise
- Surgery removes intra and extrapancreatic necrotic materials, pancreatic fluid and toxins. It permits preservation of viable pancreatic tissue. Open surgery is the gold standard for infected pancreatic necrosis.
- If gallstones are the cause of an attack of predicted or proven severe pancreatitis, or if the patient has jaundice, cholangitis or dilated common bile duct, urgent ERCP should be done within 72 hours of the onset of symmptoms. Sphincterotomy and clearance of the bile duct can reduce the incidence of infective complications in these patients.
- In patients with cholangitis, sphincterotomy should be carried out or a biliary stent placed to drain the duct. ERCP is an invasive produce and carries a small risk of worsening the pancreatitis.
- Indications for surgery
  - When diagnosis is in doubt
  - Severe pancreatitis without improvement with medical treatment
  - Infected necrosis of pancreas
  - Correction of associated biliary tract disease
  - Gallstones pancreatitis (treat with early interval cholecystectomy only after acute pancreatic inflammation has resolved. Acutely, ERCP with endoscopic sphincterotomy may be used to relieve biliary obstruction).

- Complications (pseudocysts >6cm, pancreatic abscess)

COMPLICATIONS

SYSTEMIC COMPLICATIONS

- Systemic complications are more common in the first week. They include:

1. Neurological
   - Visual disturbances
   - Confusion, irritability and encephalopathy
2. Cardiovascular
   - Shock
     - Hypovolemic shock due to sequestration of fluid as well as extensive hemorrhage
     - Septic shock from perforation and translocation of gut microbiota
   - Arrhythmias
3. Respiratory
   - Acute respiratory distress and respiratory failure due to enzymes damaging the alveolar capillary interface.
   - Pancreatic pleural effusion (left sided 20%): this is an encapsulated collected of fluid in the pleural cavity arising from acute pancreatitis. It may coexist with ascites or there may be a communication with an intra-abdominal collection. Percutaneous drainage under imaging guidance is necessary
4. Gastrointestinal: Ileus
5. Genitourinary: Renal failure
6. Hematological: DIC (due to activation of coagulation factors by pancreatic enzymes) and septicemia (common after 7 days)
7. Metabolic
   - Hypocalcemia- calcium used up in activation of enzymes
   - Hyperglycemia
   - Hyperlipidemia
8. Miscellaneous: Subcutaneous fat necrosis and arthralgia

- Note:
  - Management of systemic complications is multidisciplinary. When there is organ failure, appropriate supportive therapies including inotropic support for hemodynamic instability, hemofiltration in the event of renal failure, ventilator support for respiratory failure and correction of coagulopathies (including DIC).

- There is no role for surgery during intial period of resuscitation and stabilization, surgical intervention is contemplated only in the patient who deteriorate as a result of local complications following successful stabilization.

LOCAL COMPLICATIONS

- Usually develop after the first week and include:

1. Acute fluid collection:
   - Fluid is located in or near the pancreas. The wall encompassing the collection is ill defined.
   - The fluid is sterile and most such collections resolve.
   - No intervention is needed unless if there is a large collection causing symptoms or pressure effects in which percutaneous aspiration under ultrasound or CT guidance can be done.
   - Transgastric drainage under endoscopic ultrasound guidance is an alternative option. If acute fluid collection does not resolve it evolves into a pseudocyst or an abscess if infected.
2. Sterile and infected pancreatic necrosis:
   - Sterile necrotic material should not be drained or interfed with however if the patient shows signs of sepsis it should be determined if the necrotic pancreas or the peripancreatic fluid is infected.
   - A CT scan should be performed and a needle passed into the area under CT guidance, chossing a path that does not traverse hollow viscera.
   - If the aspirate is purulent, percutaneous drainage of the infected fluid should be carried out.
   - The tube drain inserted should have the widest bore possible and the aspirate should be sent for microbiological assessment and antibiotic sensitivity report.
   - The fluid may be viscous with particulate matter and the drain may need regular flushing with full aseptic precautions. If sepsis worsens despite this, pancreatic necrosectomy should be considered.
3. Pseudocyst of the pancreas:
   - Fibrous tissue surrounding the liquefactive necrosis and enzymes. There is persistence of amylase enzyme levels.
   - Pancreatitic pseudocysts can be a late sequela of acute pancreatitis or of pancreatic (upper abdominal) trauma. The pseudocyst can rupture and cause hemorrhage into the abdominal cavity.

- Formation of a pseudocyst requires 4 weeks or more from the onset of acute pancreatitis.
- They can be multiple or single (common). Over 50% communicate with the main pancreatic duct most commonly in the lesser sac.
- They may cause pressure symptoms (early satiety, vague discomfort, a deep palpable mass).
- Endoscopic ultrasound and aspiration of the cyst fluid is useful. The fluid should be sent for carcinoembryonic antigen (CEA) levels, amylase levels and cytology.
- Fluid in a pseudocyst has low CEA levels and levels above 400ng/ml are suggestive of mucinous neoplasm. Cytology typically reveals inflammatory cells in the pseudocyst fluid.
- Percutaneous FNA can be done if EUS is not present. Pseudocysts resolve spontaneously in most cases but can complicate.
- Treatment is dependent on the size and age of the pseudocyst. Drainage is done either percutaneously to the outside, endoscopically into the stomach or surgcically into the gastrointestinal tract.
- Cysts 6cm or smaller, or those that have been present for less than 6 weeks are most likely to have complications and can be observed for spontaneous resolution. Bigger (>6cm) or older cysts (>6 week) are more likely to rupture or bleed and they need to be treated.

4. Pancreatic abscess- due to *E. coli (35%), Klebsiella (25%), Enterococcus (25%) or Other (Staphylococci, Pseudomonas, Proteus, Enterobacter, Anaerobes, Candida-10%)*. Patient complains of abdominal pain, high fever, persistently elevated amylase in the fact of a bout of acute pancreatitis. Percutaneous drainage with the widest possible drains placed under imaging guidance is the treatment, along with appropriate antibiotics and supportive care. Repeated scans may be required depending on the progress of the patient, and drains may need to be flushed, repositioned or reinserted. Very ocassionally, open drainage of the abscess may be necessary.
5. Chronic pancreatitis- recurrent pancreatitis leads to fibrosis of pancreatic
6. Hemorrhage: may occur into the gut, retroperitoenum or peritoneal cavity. Possible causes include bleeding into a pseudocyst cavity, diffuse bleeding from a large raw surface or pseudoaneurysm. Recurrent bleeding is common, often culminating in fatal hemorrhage. CT, angiograpy or MR angiography help to make diagnosis. Treatment involves embolization or surgery.

7. Pancreatic pseudo aneurysm
8. Pancreatic ascites- This is a chronic, generalized, peritoneal, enzyme-rich effusion usually associated with pancreatic duct disruption. Paracentesis reveals turbid fluid with a high amylase level. Adequate drainage with wide-bore drains placed under imaging guidance is essential. Measures that can be taken to suppress pancreatic secretion include parenteral or Nasojejunal feeding and administration of octreotide. An ERCP may allow demonstration of the duct disruption and placement of a pancreatic stent.
9. Colonic stricture
10. Portal/Splenic vein thrombosis: may develop silently and be identified on CT. A raised platelet count should raise suspicion. In context of pancreatitis treatment is conservative. Screen patient for pro-coagulant tendencies. If varices or other manifestations of portal hypertension develop treat accordingly by endoscopic injection or banding, beta-blockers etc. Thrombocytosis may mandate use of aspirin or other antiplatet drug for a period. Systemic anticoagulation if instituted early in the process may achieve recanalization of the vein but is not routinely used as it carries a risk of on-going pancreatitis.

CLINICAL BOX: A case of epigastric pain and vomiting

History

A 50-year-old man presents to the ER with vomiting and epigastric pain which radiates through to the back. The pain was of gradual onset, coming on over the last 2 days. He denies any previous episodes. He is not on any regular medication but admits to drinking in excess of 8 cans of lager a day. He is a heavy smoker but denies any recreational drug use. He is homeless and relates his heavy drinking to depression.

Examination

The patient is sweaty and agitated. He says he is unable to lie flat for the examination and vomits persistently. His blood pressure is 150/80mmHg and he has a pulse rate of 120/min. Palpitation of his abdomen reveals tenderness in the epigastrium. The abdomen is not distended and he has normal bowel sounds. Rectal examination is unremarkable.

MCV shows 102 fL (normal 76-96fL)
Serum amylase shows 4672 IU/dl (Normal 0-100IU/dl)
Gamma-glutamyl transferase (GGT) shows 212 IU/L (normal 11-51 IU/L)
Albumin shows 25g/l (normal= 35-50g/l)

Questions

1. What is the most likely diagnosis?
2. Which important differential diagnosis should be excluded?
3. How will you grade the severity of the condition?
4. What are its causes?
5. What are the other causes of the elevated serum markers of this condition?
6. How will you manage this condition?
7. Give four potential complications.

CHRONIC PANCREATITIS

- A percentage of patients with acute pancreatitis development of chronic pancreatitis.
- The chronic form of disease is characterized by persistent inflammation that causes destructive fibrosis of the gland.
- Chronic pancreatitis is thus defined as a continuing inflammatory disease of the pancreas characterized by irreversible morphological change typically causing pain and/or permanent loss of function.
- Many patients have exacerbations, but the condition may be completely painless.
- The clinical picture is of recurring or persistent:
  - Upper abdominal pain with evidence of malabsorption
  - Exocrine pancreatic insufficiency: Steatorrhea
  - Endocrine pancreatic insufficiency: Diabetes
- The disease occurs more frequently in men (male to female ratio is 4:1) and the mean age of onset is about 40 years.
- Chronic pancreatitis can be:
  - Chronic relapsing pancreatitis
  - Chronic pancreatitis (persistent)
    - Chronic non-calcifying pancreatitis
    - Calcifying pancreatitis: it can be in the duct (stone in the pancreatic) and parenchyma

ETIOLOGY AND PATHOLOGY

- 60-70% is caused by high alcohol consumption but only 5-10% of people with alcoholism develop chronic pancreatitis.
  - The underlying mechanism is unclear however, some genetic and metabolic factors have been implicated.
  - Alcohol reduces pancreatic blood flow, alter cell viability releases the free radicals creates pancreatic ischemia and activates the pancreatic stellate cells which produce abundant extracellular matrix and collagen.

TIGAR-O Risk Factor Classification 2001

T – Toxic - alcohol/tobacco/dietary/drug.
Metabolic-hypercalcemia/lipidemia/lipoprotein lipase deficiency.
I – Idiopathic—early/late onset/tropical.
G – Genetic mutations—CFTR/SPINK 1.
A – Autoimmune primary/with Sjogren/Crohn's disease.
R – Recurrent and severe acute/ischemic.
O – Obstructive—pancreas divisum//annular pancreas/stenotic papilla/duodenal obstruction/trauma/pancreatic ductal stones/ choledochocele.

Figure 101: TIGAR-O Risk Factor Classification 2001

- Other causes:
  - Pancreatic duct obstruction from stricture formation after trauma, acute pancreatitis or due to pancreatic cancer
  - Congenital abnormalities: pancreas divisum and annular pancreas if associated with papillary stenosis are rare causes of chronic pancreatitis
  - Hereditary pancreatitis: this is an autosomal dominant disorder with an 80% penetrance, associated with mutations in the cationic trypsinogen gene on chromosome 7. Carries an increased risk of pancreatic cancer especially if present at a young age and if the patient smokes.
  - Cystic fibrosis
  - Infantile malnutrition
  - Idiopathic chronic pancreatitis (30%): can be early or late onset
  - Hyperparathyroidism, Hyperlipidemia and hypercalcemia
  - Autoimmune pancreatitis: diffuse enlargement of pancreas, diffuse and irregular narrowing of the main pancreatic duct and a possible association with other autoimmune diseases. The changes may be confused with neoplasia. Autoantibodies may be present and the immunoglobulin (Ig)G4 concentrations are elevated.
- At onset of disease when symptoms develop the pancreas may appear normal, later the pancreas enlarges and becomes hard as a result of fibrosis.
- The ducts become distorted and dilated with areas of both stricture formation and ectasia.
- Calcified stones weighing from a few mg to 200mg may form within the ducts.
- The ducts may become occluded with a gelatinous proteinaceous fluid and debris, and inflammatory cysts may form.
- Histologically, the lesions affect the lobules producing ductular metaplasia and atrophy of acini, hyperplasia of duct epithelium and interlobular fibrosis.

PATHOGENESIS

- Chronic pancreatitis can be categorized into 2 forms:
  - Calcific pancreatitis: usually associated with persistent alcohol abuse.
  - Obstructive pancreatitis: secondary to pancreatic duct obstruction.
- Alcohol induced calcific pancreatitis is the most common form of disease.
  - Proposed mechanisms of disease include ductal plugging and occlusion by protein and mineral precipitates.
  - The resulting inflammation and patchy fibrosis subsequently leads to parenchymal destruction and eventual atrophy of the gland.

- Obstructive chronic pancreatitis is due to ductal blockage secondary to scarring from acute pancreatitis or trauma, papillary stenosis, pseudocyst or tumor which results in upstream duct dilation and inflammation.

CLINICAL FEATURES

- Abdominal pain is the principal presenting and most frequent indication for surgery.
- The pain is often dull and gnawing. Severe flare-ups of pain may occur superimposed on background discomfort.
- The pain is upper abdominal, is either intermittent or persistent and frequently radiates to the back.
  - If the disease is mainly in the head of the pancreas the pain is epigastric and right subcostal
  - If the disease is limited to the left side of the pancreas, left subcostal and back pain are seen.
  - In some patients the pain may be diffuse.
  - Radiation to the left shoulder usually occurs.
- Patients are often addicted to narcotic pain relievers.
- The pain prevents sleep and time off work is frequent. The number of hospital admissions for acute exacerbations is a pointer towards the severity of disease.
- Nausea is common during attaching and vomiting may occur.
- Weight loss is common because the patient does not feel like eating.
- Other symptoms result from exocrine insufficiency (malabsorption- foul smelling steatorrhea in more than 30% of patients) and endocrine insufficiency (diabetes mellitus)
- All the complications of acute pancreatitis can occur with chronic pancreatitis.

DIAGNOSTIC EVALUATION

- The diagnosis of chronic pancreatitis is best made using imaging techniques that detect pancreatic morphologic changes rather than test glandular function, given the functional reserve of the pancreas.
- Only in the early stages of the disease will there be a rise in serum amylase. Tests of pancreatic function merely confirm the presence of pancreatic insufficiency or that more than 70% of the gland has been destroyed.
- Pancreatic calcifications may be seen on AXR.
- Ultrasound and CT are useful in the initial imaging process however endoscopic retrograde cholangiopancreatography (ECRP) is the most accurate means of

diagnosing chronic pancreatitis because it clearly defines the pathologic changes of the pancreatic ductal system and the biliary tree.

- ERCP can help determine the type of operation required, if operative intervention is indicated.
- Radiological signs of chronic pancreatitis:
  - Heterogeneously inflamed or atrophied gland
  - Dilated and strictured pancreatic duct
  - Presence of calculi

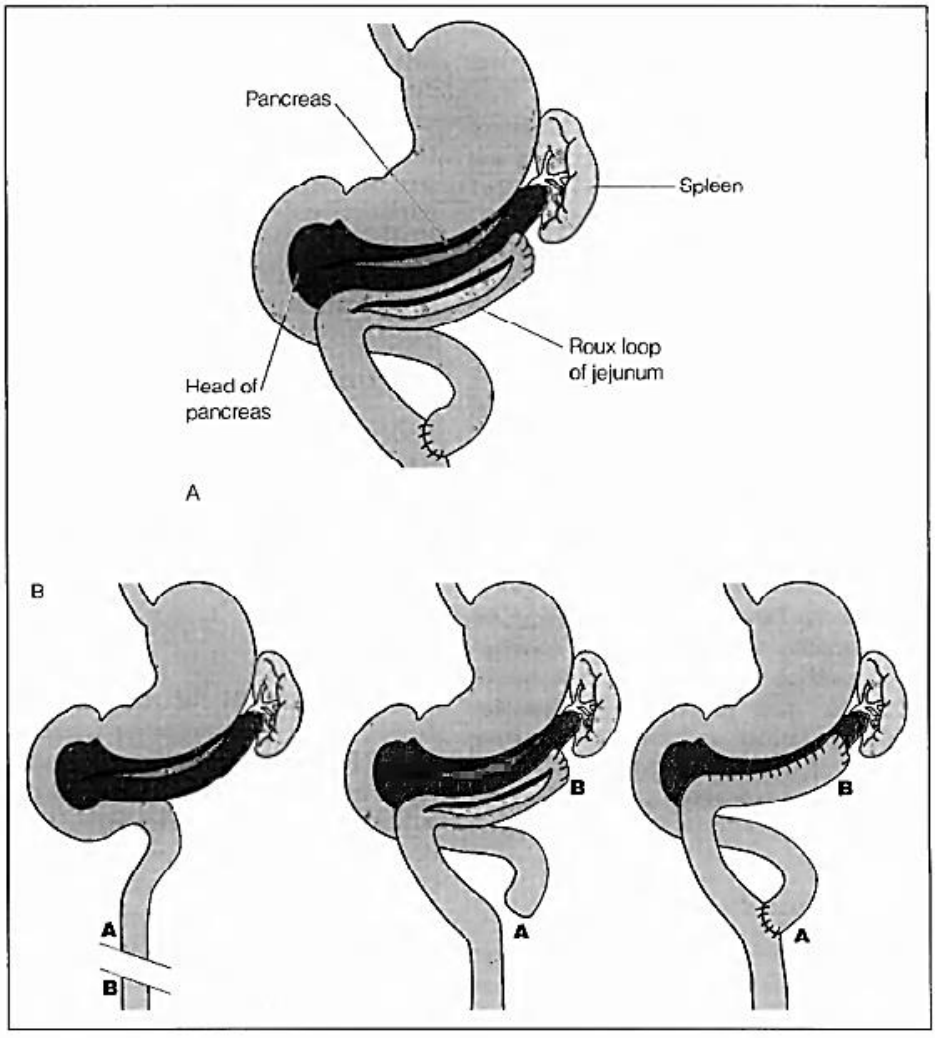
TREATMENT

- Medical:
  - Treat the addition: Stop alcohol intake and involve a psychologist or counsellor
  - Alleviate abdominal pain
    - Eliminate obstructive factors (duodenum, bile duct, pancreatic duct)
    - Escalate analgesia in a stepwise fashion
    - Refer to pain management specialist
    - For intractable pain consider CT/EUS guided coeliac axis block
  - Nutritional and digestive measures:
    - Diet: low in fat and high in protein and carbohydrates
    - Pancreatic exocrine insufficiency is treated with oral pancreatic enzymes
    - Correct malabsorption of fat-soluble vitamins (A, D, E and K) and Vitamin B12
    - Medium chain triglycerides in patients with severe fat malabsorption (they are directly absorbed by the small intestine without need for digestion)
    - Reducing gastric secretions may help
  - Insulin is used to treat diabetes mellitus.
- Surgical: only undertaken if medical therapy has failed in relieving chronic intractable pain
  - Indications for surgery:
    - Persistent pain
    - GI or biliary obstruction
    - Pseudocyst infection, hemorrhage or rupture
    - Enlarging pseudocysts
  - Functional drainage of the pancreatic duct and resection of disease tissue are the goals of any procedure

- Based on ERCP and CT findings, the correct operation can be planned.
- Endoscopic pancreatic sphincterotomy might be beneficial in patients with papillary stenosis and high sphincter pressure and pancreatic ductal pressure.
- Patients with a dominant pancreatic duct stricture and upstream dilation may benefit by placement of a stent across the stricture. The stent should be left in for no more than 4-6 weeks as it will block.
- Pancreatic duct stones may be extracted at ERCP and this may sometimes be combined with extracorporal shock wave lithotripsy.
- Pseudocysts may be drained internally under EUS guidance. Percutaneous or transgastric drainage of pseudocysts under US or CT guidance can also be done.
- The role of surgery is to overcome obstruction and remove mass lesion
- For patients with a "chain of lakes"- appearing pancreatic duct caused by sequential ductal scarring and dilation, a longitudinal pancreaticojejunostomy (Puestow procedure) is indicated to achieve adequate drainage.
- A Roux-en-Y segment of proximal jejunum is anastomosed side to side with the opened pancreatic duct, facilitating drainage.
- Distal pancreatic duct obstruction causing localized distal parenchymal disease is best treated by performing a distal pancreatectomy.
- Total pancraetectomy and islet autotransplantation has been reported in selected patients.

- Ampullary procedures: ERCP with endoscopic sphincterotomy.
- Ductal decompression procedures:
  - Puestow procedure (longitudinal pancreaticojejunostomy) for segmental ductal dilation.
  - Duval procedure (retrograde drainage with distal resection and end-to-end pancreaticojejunostomy).
- Ablative procedures (resection of portions of pancreas):
  - Frey procedure (longitudinal pancreaticojejunostomy with partial resection of the pancreatic head).
  - Whipple procedure (pancreaticoduodenectomy with choledochojejunostomy, pancreaticojejunostomy, and gastrojejunostomy).
  - Near-total pancreatectomy.
- Pseudocysts: Nonepithelialized, encapsulated pancreatic fluid collections. Up to 30% of pseudocysts resolve on their own with bowel rest (TPN and NPO). If after 6 weeks they have not resolved and are > 6 cm in size, internal drainage of the mature cyst is indicated via cyst gastrostomy or Roux-en-Y cyst jejunostomy.

Figure 102: Surgical procedures in Chronic pancreatitis



Longitudinal pancreaticojejunostomy used in the treatment of chronic pancreatitis.

PANCREATIC TUMORS

- Pancreatic tumors are classified as follows:
  - Exocrine tumors
    - Benign: benign cystadenoma (Rare)
    - Malignant:
      - Adenocarcinoma: in ampulla, periampullary or head of pancreas. Periampullary carcinoma may arise from any component- duodenal mucosa or CBD or pancreatic duct component or all. Occasionally squamous cell carcinoma or combination of adenosquamous can occur.
      - Cystadenocarcinoma: commonly found in body and tail of pancreas, usually attains a large size (5%).
  - Endocrine tumors
    - Insulinoma (beta cells)- Whipple's triad
    - Gastrinoma (G cells)- Peptic ulcer
    - Glucagonoma (alpha cells)- Diabetes, necrolytic migratory erythema
    - VIPoma (Pancreatic cholera-Verner-Morrison syndrome)- Watery-diarrhea, hypokalemia, Achlorhydia (WDHA syndrome)
    - Somatostatinoma (S/delta cells)- Diabetes, steatorrhea, gallstones
  - Lymphomas

PANCREATIC ADENOCARCINOMA

- Pancreatic adenocarcinoma is the leading cause of cancer death, trailing other cancers such as lung and colon.
- Pancreatic adenocarcinoma originates in the exocrine pancreas (ductal cells) and 2/3 occur in the head of the pancreas
- Men are affected more than women by 2:1 ratio.
- The peak incidence is 50-60.
- African-American are affected more (2:1 Black to white ratio)
- Ductal adenocarcinoma accounts for 80% of the cancer types and is usually found in the head of the gland.
- Local spread to contiguous structures occurs early, and metastases to regional lymph nodes and liver follows.
- Risk factors:
  - Increasing age
  - Race: African-American

- Cigarette smoking (Tobacco use doubles risk)
- High energy diet rich in fat
- Diabetes mellitus, alcohol abuse, chronic pancreatitis, familial pancreatitis
- Hemochromatosis with pancreatic calcification, cirrhosis , obesity
- Occupational exposure to carcinogens like DDT, benzidine
- Previous cholecystectomy

CLINICAL FEATURES

- Signs and symptoms of carcinoma of the head of the pancreas are intrinsically related to the regional anatomy of the gland.
- Patients classically complain of:
  - Obstructive jaundice- dark urine and pale stools (due to obstruction in head),
  - Weight loss and anorexia
  - Constant deep abdominal pain (posterior epigastric pain radiating to the back) due to peripancreatic tumor infiltration.
- Nausea, epigastric bloating, change in bowel habit and vomiting are occasionally present. In advanced disease, hematemesis and melena may result from direct invasion of the duodenal mucosa by tumor and duodenal obstruction may supervene.
- Patients may present with jaundice and a palpable non-tender gallbladder indicating tumor obstruction of the distal common bile duct (Courvoisier's sign).
  - Courvoisier's law/sign: in the presence of a palpably enlarged gallbladder which is non-tender and accompanied with mild painless jaundice the cause is unlikely to be gallstones.
- Pruritus often accompanies the development of jaundice.
- Fever and rigors may accompany the jaundice (bacterial cholangitis) and tend to occur in long-standing biliary obstruction.
- Migratory thrombophlebitis: Trousseau's syndrome is seen especially in tumors of the body or tail though this is not specific for pancreatic cancer.
- On rectal examination a rectal shel may be evident in the rectovesical or rectovaginal pouch (Blumer's shelf). There may be evidence of ascites, and distant metastases may be present in the supraclavicular fossa (Troisier's sign).

DIFFERENTIAL DIAGNOSIS

- Carcinomas of the ampulla of vater
- Carcinoma head of pancreas
- Carcinoma of Distal common bile duct

- Carcinoma of duodenum

DIAGNOSTIC EVALUATION

- Most common lab abnormalities are:
  - Elevated carcinoembryonic antigen (CEA) or CA 19-9 (tumor markers)
  - Elevated alkaline phosphatase
  - Direct bilirubin levels indicating obstructive jaundice. The average bilirubin level in neoplastic obstruction is typically higher than that seen in bile duct obstruction from gallstone disease.
  - CT and ERCP are the modalities of choice for the evaluation of pancreatic cancer.
    - CT reveals the location of the mass, extent of tumor invasion or metastasis and degree of ductal dilation. CT also shows the relationship of the cancer to the superior mesenteric artery and vein and the coeliac axis, patency of the portal vein and liver metastasis.
    - ERCP defines the ductal anatomy and extent of ductal obstruction and provides biopsy specimens for tissue diagnosis.
    - Drainage stents can be placed into the common bile duct during ERCP for biliary tree decompression.
    - A double duct sign may be seen in periampullary lesions.

TREATMENT

- Tumor head of pancreas: The operation for resectable tumors in the head of pancreas is pancreaticoduodenectomy (Whipple procedure).
  - Removal of gallbladder, common bile duct, antrum of stomach, duodenum, proximal jejunum and head of pancreas (en bloc)
  - Reconstruction with pancreaticojejunostomy, choledochojejunostomy and gastrojejunostomy.
  - An alternative is the modification known as the pylorus-preserving pancreaticoduodenectomy in which the stomach and duodenum are preserved to eliminate post-gastrectomy symptoms.
- Tumor of the body/tail: Distal “near-total” pancraetectomy
- Tumors of the pancreatic tail are usually unresectable because they are asymptomatic during growth and thus present at a late stage. Tumors in the head of the pancreas however present earlier because they cause biliary obstruction.
- If unresectable (due to liver/peritoneal metastases, nodal metastases beyond the zone of resection, or tumor invasion of the superior mesenteric artery), palliative care is considered:

- Relieve biliary obstruction (jaundice and pruritus)
  - Endoscopic or transhepatic (radiological) stenting using self-expanding metallic stents, which have largely replaced plastic stents for this purpose.
  - Endoscopic/radiological stenting has replaced surgical biliary bypass, cholecytojejunostomy or choledochojejunostomy unless duodenal obstruction is also present when a double bypass (biliary bypass and gastrojejunostomy) is needed.
- Relieve duodenal obstruction (vomiting)
- Splanchnic nerve bloc (pain control)
  - Percutaneous coeliac plexus block with 50ml of 50% alcohol or 20ml of 6% phenol
  - Bilateral thoracoscopic splanchnicectomy
  - Opioid analgesia

- Post-operative chemoradiation therapy is controversial but typically includes 5-fluorouracil (5-FU) and external-beam radiation.

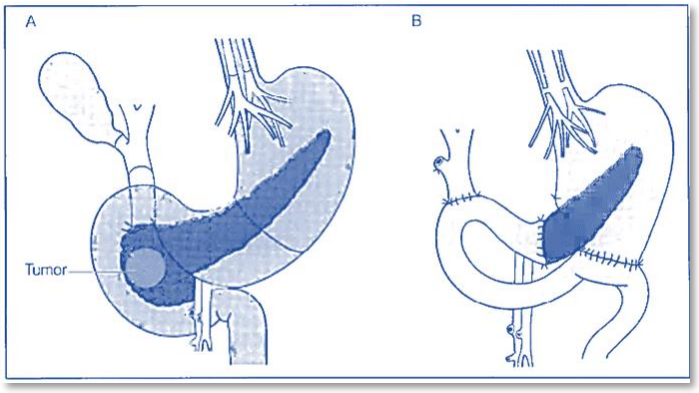


Figure 103: Pancreaticoduodenectomy (Whipple procedure). Preoperative anatomic relationships (A) and postoperative reconstruction (B)

PROGNOSIS

- Long term survival for pancreatic cancer remains dismal, and most patients die within 1 year of diagnosis.
- The 5-year survival for all patient with tumors of the head of pancreas is approximately 3%.
- For individuals with tumors amenable to Whipple resection, the 5-year survival rate is only 10-20%.
- Tumors of the body and tail are invariably fatal because diagnosis is usually made at a more advanced stage due to the lack of early obstructive findings.

OTHER PANCREATIC TUMORS/NEOPLASMS

- About 20% of all pancreatic cystic lesions will not be pseudocyst. These lesions may be benign or malignant but even the malignant ones carry better prognosis than adenocarcinoma.
- Simple cysts are usually congenital. When they appear in children, no treatment is required.
- In patients with polycystic kidney disease, 10% will have pancreatic cysts, they also do not require treatment.
- More complicated cystic disease of the pancreas include serous cystadenoma and mucinous cystic neoplasms.
- Solid and cystic papillary neoplasms tend to present as large lesions in young women. Resection is often curative.
- Intraductal papillary mucinous neoplasms are distinguished from other cystic lesions of the pancreas in that the pancreatic duct is enlarged because of mucin deposition. ERCP demonstrates mucin in the duct. These lesions have malignant potential and require resection. Long-term prognosis is excellent.
- Pancreatic endocrine tumors may be benign or malignant.
  - ➢ Compared with pancreatic adenocarcinoma, endocrine tumors, even when malignant are slow-growing and many metastasize only to regional lymph nodes. Hence curative surgical excision is possible in a significant proportion of patients.
  - ➢ Normal islet cells also synthesize the protein chromogranin along with specific peptide hormones. Elevated plasma levels of chromogranin or neuron-specific enolase are thus useful markers for pancreatic endocrine tumors.

- MEN 1: Inherited autosomal dominant disorder involving
  - Pancreas: diffuse islet cell disease consisting of micronodular and macronodular hyperplasia and often with multiple tumors secreting multiple peptide hormones, non-B-cells tumors (especially Gastrinoma)
  - Parathyroid gland: hyperparathyroidism with hyperplasia of the glands
  - Pituitary gland: Pituitary adenoma (prolactinoma)
- MEN 2: inherited autosomal dominant disorder involving
  - MEN 2a: Medullary carcinoma of the thyroid, Pheochormocytoma and Parathyroid adenomas
  - MEN 2b: Medullary carcinoma of the thyroid, Pheochormocytoma and ganglioneuromas of the oral mucosa

CYSTADENOCARCINOMA

- Common in females age 40-60 years.
- Common in the body and tail
- Accounts for less than 2% of all pancreatic exocrine tumors.
- Prognosis better than adenocarcinoma
- Treatment: Surgical resection-distal/total pancreatectomy.

CYSTADENOMA

- Common in older and middle-aged women.
- Two types are recognized:
  - Serous: benign
    - Occurs between 30 and 50 years.
    - They are generally asymptomatic
    - Ultrasound reveals a complex low-density mass with fine septae. The fluid is generally clear
    - Resection is generally curative.
  - Mucinous: generally benign but has potential to be malignant.
    - Usually require formal partial pancreatectomy.
- Treatment: Surgical resection.

ENDOCRINE TUMORS (ISLET CELL TUMORS)

INSULINOMA

- This is a beta cell neoplasm with overproduction of insulin.
- It is the most common islet cell tumor.
- 90% are benign.
- It is common in women than men and commonly affects middle-aged patients.
- Most are solitary lesion with even distribution in the head, body and tail of the pancreas.
- It associated with multiple endocrine neoplasia I (MEN I)

syndrome (<10% of cases) then multiple insulinomas may be present.

- Clinical features related to hypoglycemia triggered catecholamine release. Patients can lose consciousness or experience seizures.
- Insulinoma is characterized by Whipple's triad:
  - Symptoms of hypoglycemia with fasting (palpitations, tachycardia, and shaking)
  - Fasting glucose <2.8 mmol/l (50mg/dl)
  - Relief of symptoms after eating
- Differential diagnosis: Surreptitious (Sneaky) insulin administration
- Diagnosis:
  - Fasting serum insulin level >10 uU/ml (Normal: <6uU/ml)
  - Fasting insulin to glucose ratio> 0.3
  - Proinsulin or C-peptide levels should be measured to rule out surreptitious exogenous insulin administration
  - CT or MRI to localize the lesion and determine whether metastases are present.
- Treatment:
  - Surgical enucleation or resection is usually curative (90% of patients)
  - Note: even if the lesion is not localized, surgical exploration is indicated to try to locate and resect the tumor. Intraoperative ultrasound should be used in this case. Even when metastatic disease is present, resection may be indicated for symptomatic relief.
  - Diazoxide can improve hypoglycemic symptoms by inhibiting pancreatic insulin release.

GASTRINOMA (ZOLLINGER-ELLISON SYNDROME)

- This is a neoplasm associated with overproduction of gastrin.
- This is the second most frequent islet cell tumor.
- The syndrome is slightly more common in men than in women and generally affects patients in middle age.
- 25% of gastrinomas are associated with MEN-1.
- The lesions most commonly occur in the pancreas or duodenum but can also occur in other areas.
- 90% are located in the "Gastrinoma triangle" bordered by
  - Junction of second and third part of the duodenum
  - Cystic duct
  - Superior mesenteric artery under the neck of the pancreas

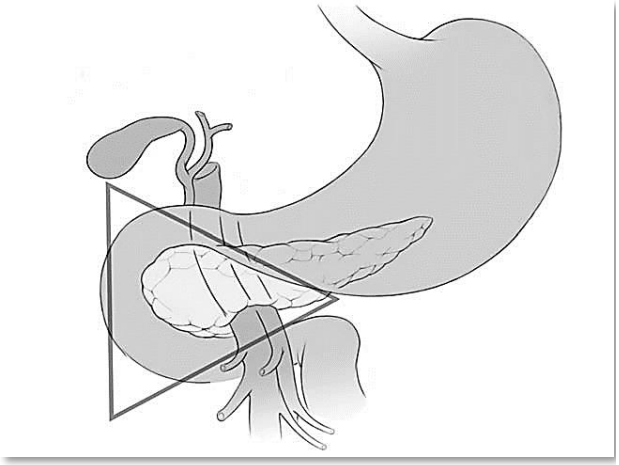


Figure 104: Gastrinoma triangle- the anatomic triangle in which approximately 90% of gastrinomas are found (Image from First Aid for the Surgery Clerkship 2nd Ed)

- The tumor is small, slow-growing, multiple and 60% of them are malignant.
- Clinical features:
  - Signs of peptic ulcer disease (Especially in patients with recurrent or unusually located ulcers)
  - Epigastric pain most prominent after eating
  - Profuse watery diarrhea (Secretory diarrhea)
- Diagnosis:
  - Fasting serum gastrin level >500 pg/ml (normal <100pg/ml)
  - Secretin stimulation test will cause paradoxical increase in gastrin in patients with Z-E syndrome (double fasting level or increase of 200 pg/dl over the fasting level)
  - Ulcers in unusual locations (e.g. third part of duodenum or jejunum) is highly suggestive.
  - Octreotide scan to localize tumor
  - CT, MRI and U/S have some utility in localizing the tumor.
- Treatment:
  - Proton pump inhibitor to alleviate symptoms
  - Surgical resection (curative or debulking)
  - Chemotherapy

VIPOMA

- This is a tumor associated with the overproduction of vasoactive intestinal peptide (VIP).
- It is also known as Verner-Morrison syndrome or WDHA syndrome (Watery Diarrhea, Hypokalemia, Achlorhydia)
- Most are malignant, majority have metastasized to lymph nodes and the liver at the time of diagnosis.
- 10% are extrapancraetic.
- Clinical features:
  - Severe watery diarrhea
  - Signs of hypokalamia: palpitations/arrhythmias, muscle fasiculations/tetany, paresthesias
- Diagnosis: fasting serum VIP level >500 pg/ml (normal <200 pg/ml) with exclusion of other causes of diarrhea
- Treatment:

- Surgical resection, chemotherapy
- Octreotide (somatostatin analogue)- diarrhea control

GLUCAGONOMA

- This is a rare alpha cell neoplasm resulting in overproduction of glucagon.
- Most are malignant, large primary tumors that have usually metastasized to lymph nodes and liver at the time of diagnosis.
- Clinical features:
  - Mild diabetes (hyperglycemia)
  - Anemia
  - Mucositis
  - Weight loss due to low amino acid level
  - Severe dermatitis: often a red psoriatic-like rash with serpiginous borders over trunk and lower limbs
- Diagnosis
  - Fasting serum glucagon level >1000pg/ml (normal <200 pg/ml)
  - Skin biopsy to confirm presence of necrolytic migratory erythema
- Treatment:
  - Surgery and chemotherapy
  - Octreotide to inhibit the release of glucagon

SOMATOSTATINOMA

- Very rare tumor
- Malignant
- Treated by surgery and chemotherapy

GALLBLADDER

- The gallbladder is a pear-shaped (size=5-12cm) reservoir located in a fossa on the inferior surface of the liver.
- It consists of:
  - Fundus
  - Body
  - Infundibulum
  - Neck
- Hartmann's pouch is a pathological one located in the infundibular region created by gallstones.
- The Gallbladder drains through the cystic duct into the common hepatic duct to form the common bile duct.
  - The left hepatic duct is formed by the ducts draining II, III, IV segments of the liver.
  - The right hepatic duct is formed by the ducts draining V, VI, VII, VIII segments of the liver.
  - The right and left hepatic ducts join at the porta hepatis to form the common hepatic duct.
  - The common hepatic duct joints the cystic duct to form the common bile duct.
  - The common bile duct is normally 10-12cm in length and 6-8mm in diameter. It joins the major pancreatic duct in the wall of the second part of duodenum to form the ampulla of Vater.
  - The intraduodenal part of the common bile duct is surrounded by smooth muscle fibers called as sphincter of Oddi.

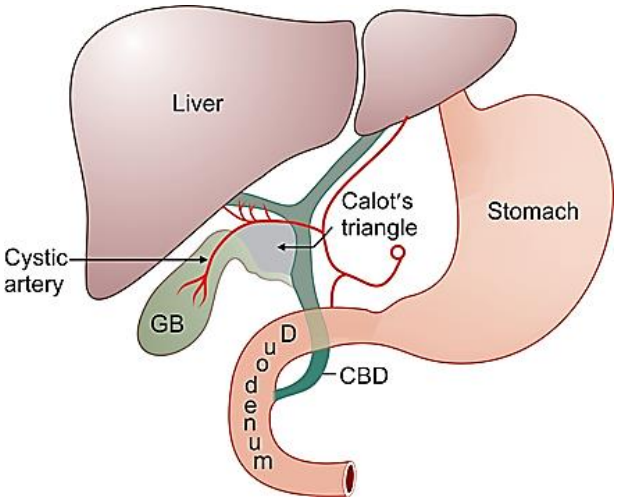


Figure 105: Anatomy of the gallbladder

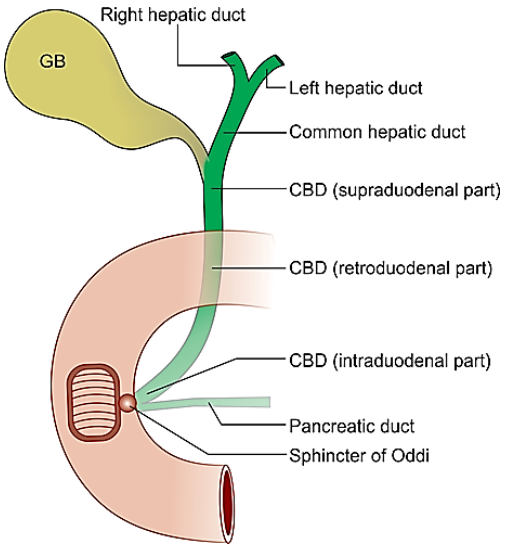


Figure 106: Common bile duct parts

- The extrahepatic biliary tree receives blood from gastroduodenal, retroduodenal, posterosuperior pancreaticoduodenal arteries.

- The gallbladder is supplied by the cystic artery (A branch of the right hepatic artery).
- Calot's triangle is formed by:
  - Common hepatic duct to the left,
  - Cystic duct below
  - Inferior surface of the liver above.
- The cystic artery originating from the right hepatic artery passes behind the common hepatic artery, and enters the Calot's triangle to reach the gallbladder.
- Calot's triangle contains the lymph node of 'Lund' (Fred Bates Lund).
- Often the cystic artery, hepatic artery, and cystic duct have anomalous positions and anomalous origins. Both gallbladder neck and cystic duct contain mucosal folds called valves of Heister.
- About 1000ml of bile is secreted from the liver which contains water (98%), bile salts, bile pigments, fatty acids, lecithin, cholesterol and electrolytes (sodium, potassium, chloride, bicarbonate, calcium, magnesium) with a pH more than 7.0.
- The main function of the gallbladder is to concentrate and store the bile (capacity of the gallbladder is 40-50ml)
- Bile salts form micelle which make cholesterol soluble.
- Bilirubin conjugated in the liver is secreted into the bile which in the bowel is converted into urobilinogen by bacteria. Urobilinogen gets absorbed in the bowel, enters the liver for re-secretion again and part of it is excreted in the urine. Absence of urobilinogen in urine signifies obstructive jaundice.
- In the absence of gallstones or any other disease, bile is sterile. Symptomatic gallstone disease shows positive culture for bacteria commonest being *E. coli* and *Klebsiella*.
- Ultrasound is the first line for investigating gallbladder and biliary tree pathologies.
- Further investigations with computed tomography (CT) or Magnetic resonance cholangiopancreatography (MRCP)/Endoscopic retrograde cholangiopancreatography (ERCP) can be done.

GALLSTONES/ CHOLELITHIASIS

- Cholelithiasis is the presence of gallstones within the gallbladder. Choledocholithiasis refers to stones in the common bile duct.
- Gallstones are the most common biliary pathology.
- Gallstones are round, oval or faceted concretions found in the biliary tract containing cholesterol, calcium carbonate, calcium bilirubinate or a mixture of these elements.
- 80% of stones are composed primarily of cholesterol while the remaining 15% are pigmented.
- Gallstones are more common in females (2:1) and people above the age of 40 years.
- These are classified based on chemical composition into:
  - Cholesterol stones: Cholesterol solitaire (Radiating crystalline appearance) are common and are often solitary. These stones contain 51-99% pure cholesterol plus an admixture of calcium salts, bile acids, bile pigments and phospholipids. Cholesterol which is insoluble in water is secreted from the canalicular membrane in phospholipid vesicles. Whether cholesterol remains in solution depends on the concentration of phospholipids and bile acids. Micelles formed by the phospholipid hold cholesterol in a stable thermodynamic state. When bile is supersaturated with cholesterol or bile acid concentrations are low, unstable unilamellar phospholipid vesicle form, from which cholesterol crystals may nucleate and stones may form.
  - Pigmented stones: are small and often multiple. They contain less than 30% cholesterol.
    - Black or greenish black pigmented stones (20-30%): composed of an insoluble bilirubin pigment polymer mixed with calcium phosphate and calcium bicarbonate. Black stones accompany hemolysis, usually hereditary spherocytosis or sickle cell disease. For unclear reasons, patients with cirrhosis have a higher instance of pigmented stones.
    - Brown pigmented stones: contain calcium bilirubinate, calcium palmitate and calcium stearate as well as cholesterol. They are rare in the gall bladder. They form in the bile duct and are related to bile stasis and infected bile. Stone formation is related to the deconjugation of bilirubin diglucuronide by bacterial beta-glucoronidase. Insoluble unconjugated bilirubinate precipitates. Brown pigment stones are associated with the

presence of foreign bodies within the bile ducts such as endoprosthesis (stents) or parasites (*Clonorchis sinesis* and *Ascaris lumbricoides*)

- Mixed stones: 90% common. They contain cholesterol, calcium salts or phosphate, carbonate, palmitate, proteins and are multiple faceted.

- Gallstones are common in the following populations "5Fs"
  - Female
  - Fat
  - Fertile
  - Forty
  - Flatulent

ETIOLOGY/RISK FACTORS FOR GALLSTONE FORMATION

- Super-saturation of bile
  - Female
  - Fertile
  - Fat, High calorie
  - Forty
- Altered Gallbladder function
  - Stasis
  - Poor emptying
  - Poor absorption
  - Infection
- Altered enterohepatic circulation
  - Ileal resection
  - Ileal diseases
  - Altered bowel transit time
  - Altered bowel flora
  - Cholestyramine
  - Deoxycholate
- Hemolytic diseases
- Pregnancy, oral contraceptives, inflammatory bowel disease, hyperlipidemia, and total parenteral nutrition (TPN).
- Cholesterol nucleation factors
  - Mucus and Glycoprotein
  - Infection

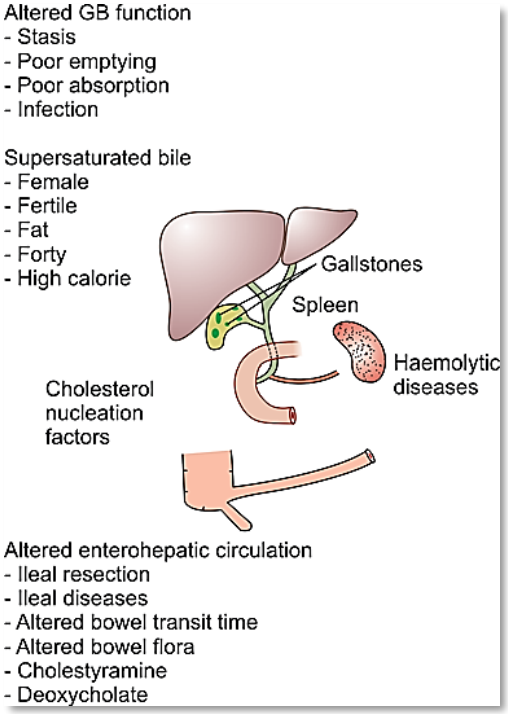
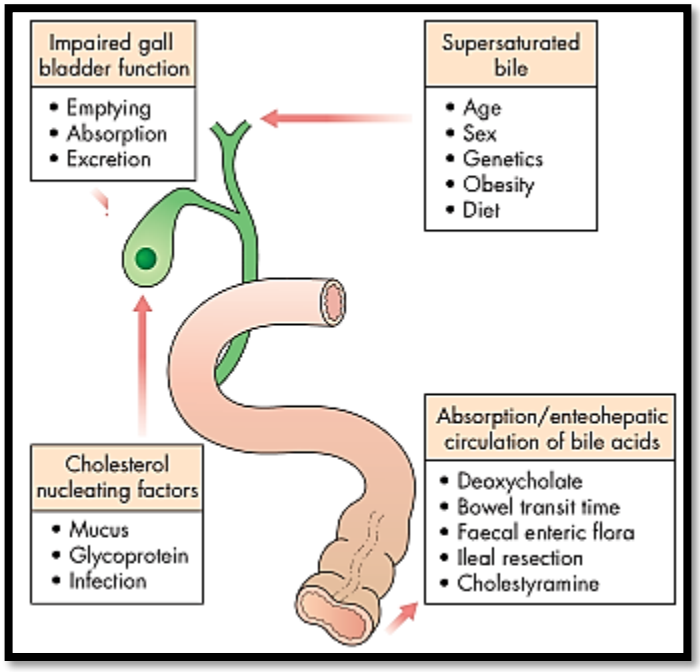


Figure 107: Causes of gallstone formation



Risk factors for gallstone prevalence and symptomatic gallstone disease.

Increased prevalence
Female sex*
Obesity*
Age*
Genetic and ethnic factors*
Diet depleted in fibre and high in animal fats*
Diabetes mellitus*
Ileal disease and resection
Haemolytic states†
Infections of the biliary tract†
Parasitic infestations†
Cirrhosis†
Cystic fibrosis
Precipitation of symptomatic disease
Pregnancy
Clofibrate
Thiazide diuretics
?Oral contraception

* Increased prevalence of cholesterol stones.
† Increased prevalence of pigment stones.

PATHOGENESIS

CHOLESTEROL STONES

- Cholesterol stones are preceded by the formation of biliary sludge and account for the vast majority of gallstones encountered in western countries.
- Cholesterol stones have a protein matrix but are composed predominantly of cholesterol with varying amounts of bile pigments and calcium salts deposited on the periphery.
- Cholesterol gallstones do not commonly harbor bacteria and are not usually associated with infected bile.
- They are often radiolucent but cast acoustic shadows on ultrasound.
- They are often multiple and medium-sized but when solitary attain a large size and have a radiating crystalline cross-sectional appearance.
- The formation of cholesterol stones involves 7 processes:
  - Super-saturation of bile with cholesterol
  - Incomplete transfer of cholesterol from biliary vesicles to bile salt micelles
  - Formation of abnormal high-cholesterol containing biliary vesicles
  - Aggregation and fusion of unstable vesicles
  - Cholesterol crystallization (nucleating and antinucleating factors)
  - Biliary sludge formation
  - Stone growth

- Cholesterol which is insoluble in water is secreted from the canalicular membrane in phospholipid vesicles. Whether cholesterol remains in solution depends on the concentration of phospholipids and bile acids. Micelles formed by the phospholipid hold cholesterol in a stable thermodynamic state. When bile is supersaturated with cholesterol or bile acid concentrations are low, unstable unilamellar phospholipid vesicle form, from which cholesterol crystals may nucleate and stones may form.
- There is an imbalance in bile between cholesterol, bile salts and phospholipids producing lithogenic bile. Cholesterol stones may be associated with inflammatory bowel disease.

PIGMENTED STONE

BLACK STONES

- Black stones account for 25% of gallstones in the West though their prevalence is higher in the Far east.
- They are composed of bilirubin polymers without calcium palmitate, small amounts of cholesterol (<30%) and a matrix of organic material.
- They are associated with infection in less than 20% of patients.
- Black pigmented stones are usually multiple, small, irregular and dark-green to black in color.
- The pathophysiology is largely unknown. Although hemolytic states predispose to formation of black pigmented stone, most occur in patients without detectable chronic hemolysis.



Figure 108: Pigmented stones showing faceted look

- They are common in cirrhotic patients who have an elevated concentration of monoconjugated bilirubin and lower bile salt concentration than normal.
- Mucins may be involved in the development of pigmented stones especially those forming in the intrahepatic ducts.



Figure 109: Multiple gallstones collected during cholecystectomy

BROWN PIGEMENT STONES

- Brown pigment stones form in the bile ducts (primary ductal calculi) and are associated with infection of the biliary tract.
- They are brownish yellow, soft and mushy.
- Brown pigment stones contain calcium bilirubinate, calcium stearate, calcium palmitate and only small amounts of cholesterol bound in a matrix of organic material.
- Brown pigment stones are caused by infection with gram negative bacteria (*Escherichia coli* and *Bacteroides fragilis-98%*) which elaborate and release β-glucoronidase which causes hydrolysis of soluble conjugated bilirubin to insoluble calcium bilirubinate.
  - They form in the bile duct and are related to bile stasis and infected bile. Stone formation is related to the deconjugation of bilirubin diglucuronide by bacterial beta-glucoronidase. Insoluble unconjugated bilirubinate precipitates. Brown pigment stones are associated with the presence of foreign bodies within the bile ducts such as endoprosthesis (stents) or parasites (*Clonorchis sinesis* and *Ascaris lumbricoides*)
- Brown pigments stones are encountered in biliary tract conditions associated with stasis and infection and chronic obstructive disease, indwelling biliary endoprosthesis and around non-absorbable suture material or metal clips used in biliary tract surgery.

MIXED STONES

- Are associated with anatomical abnormalities, stasis, previous surgery, and previous infections.

CLINICAL FEATURES

- Most patients are asymptomatic (>80%).
- Symptomatic patients present with
  - Severe right upper quadrant pain radiating to the back (right shoulder blade), epigastrium or left upper quadrant that tends to be worse after eating (especially after fatty foods)
  - The pain is more often persistent than intermittent. Onset is usually at night. It is described as colicky but more often is dull and constant
  - Dyspepsia, flatulence, Nausea and vomiting may also be present
- The symptom complex is called biliary colic and typically resolves over a few hours (or it may be aborted by anticholinergics). It occurs when a stone

temporarily occludes the cystic duct. There are no signs of peritoneal irritation or systemic signs of inflammatory process.

- Jaundice and dark urine may follow an attack of biliary coli and indicate common bile duct obstruction by a calculus. Jaundice often subsides after a few days but may persist as a major presenting symptom.
- Mirrizzi's syndrome is external compression of the common hepatic duct by a gallstone impacted in the cystic duct.
- Hydrops of the gallbladder occurs when there is complete obstruction of the cystic duct by a gallstone, causing the gallbladder to fill with fluid from the gallbladder mucosa. The fluid is often milky white.
- Spectrum of symptomatic gallstone disease includes:
  - Acute biliary colic/acute cholecystitis and Chronic cholecystitis due to stone obstruction at the gallbladder neck with superimposed infection
  - Cholangitis/septicemia: due to obstruction of common bile duct with superimposed infection
  - Acute gallstone pancreatitis: due to gallstones passing through biliary system
  - Biliary fistulous disease
  - Gallstone ileus: due to migration of a large stone into the gut causing intestinal obstruction

DIAGNOSIS AND INVESTIGATIONS

- Often incidental, as most patients are asymptomatic
- Blood investigations:
  - Liver function tests
  - Full blood count
- Imaging:
  - Abdominal plain films picks up 15% of gallstones
  - Ultrasound: Procedure of choice, classic findings include an acoustic shadow ("headlight") and gravity-dependent movement of gallstones with patient repositioning

Differential diagnosis of radio-opaque shadow in X-ray

- Kidney stone
- Gall stone
- Calcified 12th rib tip
- Phlebolith
- Pancreatic stone
- Radio-opaque foreign body
- Faecolith
- Calcified lymph node
- Calcified renal tuberculosis
- Renal cell carcinoma—calcifications
- Adrenal tumour—calcification

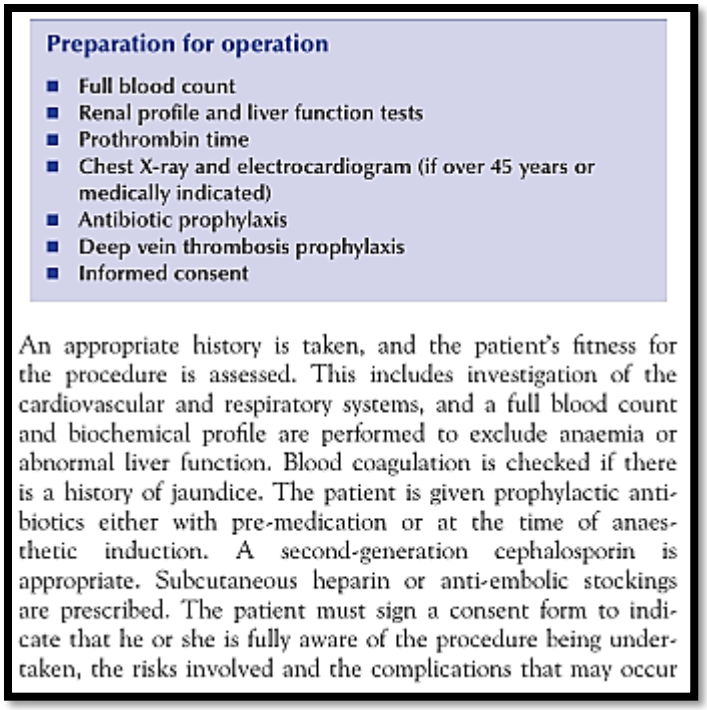


Figure 110: Sonogram demonstrating classic "headlight" appearance of cholelithiasis

TREATMENT

- Asymptomatic patients: do not require cholecystectomy unless the patient:
  - Has a porcelain gallbladder (which has an increased incidence of carcinoma)
  - Has sickle cell anemia
  - Has a stone >2-3 cm
  - Is a pediatric patient
  - Is immunocompromised or has diabetes
- Symptomatic patients: require cholecystectomy.
  - A laparoscopic cholecystectomy is ideal and can be performed on 95% of patients.
  - Open cholecystectomy is done through right subcostal Kocher's incision. Open approach is used if patient is not fit for laparoscopic surgery (anesthersia), in suspected CBD stones, Mirizzi syndrome, suspected carcinoma gallbladder.
  - During laparoscopic cholecystectomy, if there is on table difficulty in dissection at Calot's triangle, then conversion into open approach may be required.

- If an early operation is not indicated one should wait approximately 6 weeks for the inflammation to subside before proceeding to operate.



Preparation for operation

- Full blood count
- Renal profile and liver function tests
- Prothrombin time
- Chest X-ray and electrocardiogram (if over 45 years or medically indicated)
- Antibiotic prophylaxis
- Deep vein thrombosis prophylaxis
- Informed consent

An appropriate history is taken, and the patient's fitness for the procedure is assessed. This includes investigation of the cardiovascular and respiratory systems, and a full blood count and biochemical profile are performed to exclude anaemia or abnormal liver function. Blood coagulation is checked if there is a history of jaundice. The patient is given prophylactic antibiotics either with pre-medication or at the time of anaesthetic induction. A second-generation cephalosporin is appropriate. Subcutaneous heparin or anti-embolic stockings are prescribed. The patient must sign a consent form to indicate that he or she is fully aware of the procedure being undertaken, the risks involved and the complications that may occur

- An attack of acute pancreatitis, cholangitis or obstructive jaundice is usually an indication for prophylactic cholecystectomy.
- Conservative treatment followed by cholecystectomy:
  - Over 90% of cases subside with conservative measures.
  - Conservative treatment is based on 4 principles
    - Nil per oral (NPO) and intravenous fluid administration
    - Administration of analgesics
    - Administration of antibiotics. As the cystic duct is blocked in most cases, the concentration of antibiotics in the serum is more important than its concentration in bile. A broad-spectrum antibiotic effective against Gram-negative aerobes is most appropriate (e.g. cefazolin, cefuroxime or gentamicin)
    - Subsequent management: When temperature, pulse and other signs of inflammation begin to subside, oral fluids are reinstated followed by regular diet. Ultrasound is performed to ensure that no local complications have developed (normal bile duct size without any stone in the bile duct). Cholecystectomy may be performed on the next available list, or patients may be allowed home to return when the inflammation has completely resolved.
- Note: conservative treatment must be abandoned if pain and tenderness increase, depending on the status of the patient, operative intervention and cholecystectomy should be performed. If the patient has serious comorbid conditions a percutaneous cholecystotomy can be performed under ultrasound control which will rapidly relieve symptoms. A subsequent cholecystectomy is usually required.
- Medical treatment to dissolve gallstones involves chenodeoxycholic acid or urosodeoxycholic acid drugs though it is not very successful.

- Complications of cholecystectomy
  - Leakage of bile from cystic duct or gallbladder bed
  - Jaundice due to retained ductal stones. Retained stones can be treated by ERCP or if a T-tube is in place by extraction with a Dormia basket down the T-tube track (Burhenne maneuver)
  - Injury to the common bile duct

EFFECTS AND COMPLICATIONS OF GALLSTONES

- In gallbladder:
  - Biliary colic
  - Acute cholecystitis
  - Chronic cholecystitis
  - Empyema of the gall bladder: this is pus in the gall bladder. It may be a sequel of acute cholecystitis or a result of a mucocele becoming infected. Treatment is drainage and later cholecystectomy
  - Mucocele
  - Perforation
- In the bile ducts
  - Bile obstruction
  - Acute cholangitis
  - Acute pancreatitis
- In the intestine
  - Intestinal obstruction (gallstone ileus)

CHOLEDOCHOLITHAISIS

- Choledocholithiasis is the presence of stones in the common bile duct.
- Choledocholithiasis can result in transient or complete blockage of the common bile duct.
- Patients may relate episodes of passing dark urine or light-colored stools caused by the inability of bile pigments to reach the gastrointestinal tract and form subsequent renal clearance.
- Choledocholithiasis can also lead to ascending cholangitis, demonstrated by right upper quadrant abdominal pain, fever and chills.
- Pancreatitis owing to choledocholithiasis (gallstone pancreatitis) typically manifests with epigastric pain radiating to the back.
- Choledocholithiasis may be associated with jaundice in addition to signs of biliary colic. It may present as obstructive jaundice, acute cholangitis or acute pancreatitis.
- Choledocholithiasis is classically associated with increased serum bilirubin and alkaline phosphatase.
- Choledocholiathisis can be diagnosed by intraoperative cholangiography at the time of surgery or preoperatively or postoperatively by endoscopic retrograde cholangiopancreatography (ERCP).
- Management involves cholecystectomy.

CHOLECYSTITIS

- This is inflammation of the gallbladder wall.
- It can be classified as:
  - Acute cholecystitis
    - Acute calculous cholecystitis
    - Acute acalculous cholecystitis
  - Chronic cholecystitis

ACUTE CALCULOUS CHOLECYSTITIS

- This is inflammation of the gallbladder wall.
- Commonly it occurs in a patient with pre-existing chronic cholecystitis however it can occur as a first presentation.
- It is usually caused by impacted gallstones in the Hartmann's pouch obstructing the cystic duct.
- Causative bacteria include:
  - *E. coli* (most common)
  - *Klebsiella, pseudomonas, proteus*
  - *Salmonella*
  - *Strep. faecalis*
  - *Clostridium Welchii*
- Infection is through either
  - Hematogenous spread via the hepatic artery-cystic artery
  - Portal vein
  - Through bile after filtering in the liver via portal circulation

PATHOGENESIS AND PATHOLOGY OF CHOLECYSTITIS

- Stones cause obstruction at Hartmann's pouch or in the cystic duct precipitating stasis, edema of the wall, bacterial infection, and the various effects seen in acute cholecystitis.
- Impacted stones may also cause mucosal erosion allowing bile salts to act over the submucosal tissue as bile is toxic to these tissue. It leads into necrosis further infection and often perforation of the gallbladder usually at Hartmann's pouch.

- Pathology of acute cholecystitis:
  - Gallbladder is distended with edematous friable wall. The wall contains dilated vessels
  - There are areas of necrosis and patchy gangrene in severe cases
  - Mucosa shows ulceration and necrosis
  - Lumen contains infection fluid/infected bile or frank pus
  - Histology shows
    - Acute inflammation with neutrophils
    - Edema
    - Areas of necrosis and cell death

CLINICAL FEATURES

- Sudden onset of pain in the right hypochondrium with tenderness, guarding and rigidity usually for >3 hour.
  - The pain may radiate to right scapula area.
- Palpable, tender, smooth, soft gallbladder
- Murphy's sign: pain on deep inspiration resulting in inspiratory arrest (positive in 1/3 of patients).
- Sonographic Murphy's: Pain over RUQ when palpated with ultrasound probe (87% sensitivity).
- Area of hyperesthesia between 9^{th} and 11^{th} ribs posteriorly on the right (Boas's sign)
- Jaundice may be present
- Nonspecific and variable features: Fever, nausea, vomiting, and anorexia
- Palpable tender mass in Gall bladder region (25%)
- Tachycardia, toxic features, ileus and mild abdominal distension may be seen in severe cases.

INVESTIGATIONS

DIAGNOSTIC

IMAGING

- Ultrasound of abdomen: very useful, reveals presence or abscess of gallstones, pericholecystic fluid, and thickening of gallbladder wall (>4mm). Ultrasound will also show a dilation of the common bile duct if the stone has passed.
- Plain Abdominal X-ray: 10% of gallstones are radio-opaque, also rules out other causes of acute pain abdomen (duodenal ulcer perforation, peritonitis). A gas shadow is seen in the region of the gall bladder in emphysematous gallbladder. Porcelain gallbladder may be seen as opacified area in gallbladder region.
- HIDA scan (Most sensitive): Non-filling of the gallbladder even when the small bowel is visualized is characteristic of acute cholecystitis

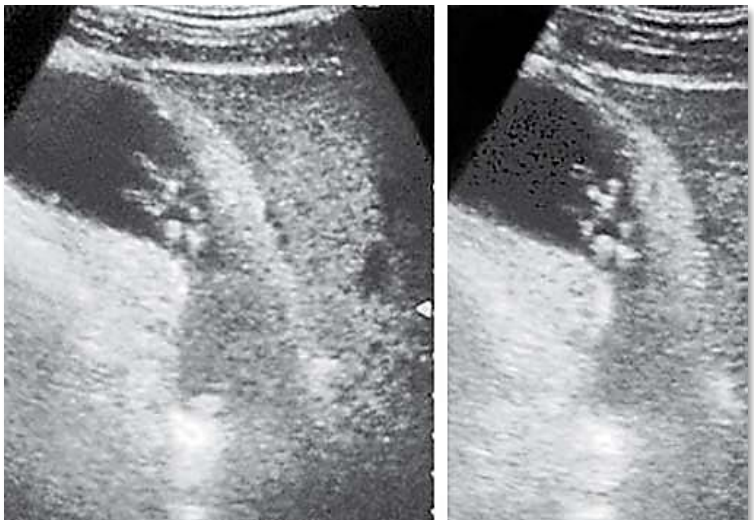


Figure 111: Ultrasound picture of visualization of multiple gallstones (Image Courtesy of SRB's Manual of Surgery)

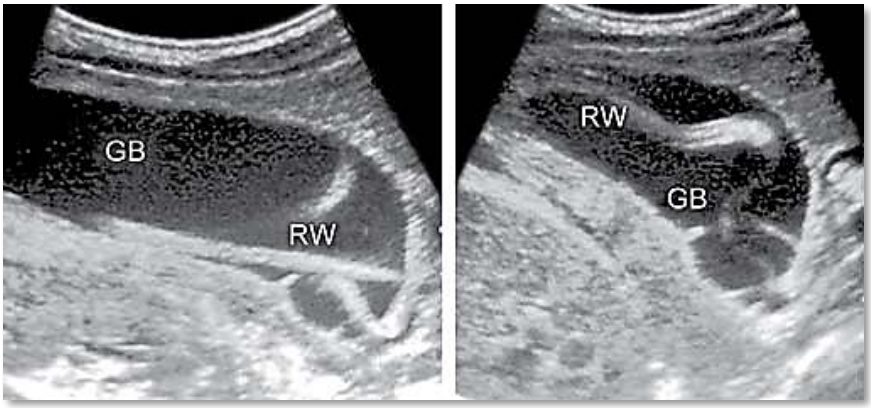


Figure 112: Ultrasound showing roundworm in gallbladder (Image Courtesy of SRB's Manual of Surgery)

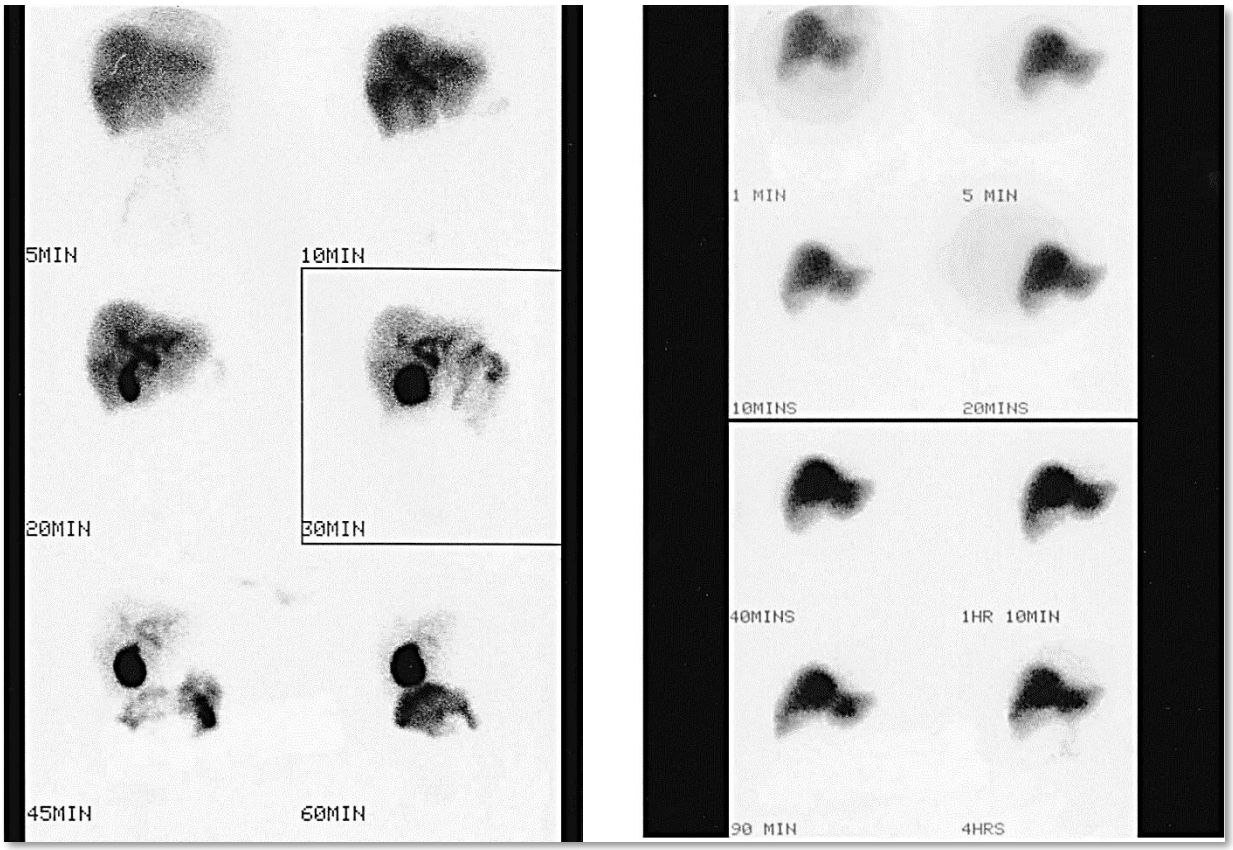


Figure 113: Normal HIDA scan on the left demonstrating uptake of contrast in intrahepatic bile duct and gallbladder at 20 minutes and excretion into small bowel at 30 minutes. On the Right image note the gallbladder is not visualized even at 4 hours even though the small bowel is, this is consistent with acute cholecystitis (Images from First Aid for Surgery Clerkship)

SUPPORTIVE

BLOOD

- Full blood count: leukocytosis with predominant neutrophilia (>10 x 10^9/L)
- Live enzymes (may increase) to check functioning of the liver
- Alkaline phosphatase (may be increased)
- Serum amylase (increases but below diagnostic threshold for acute pancreatitis)
- Total bilirubin (increases)
- Urea, creatinine and electrolytes

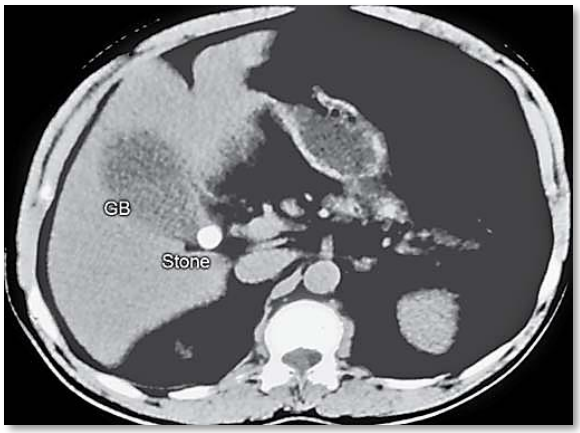


Figure 114: CT scan showing stone in the neck of the gallbladder (Image Courtesy of SRB's Manual of Surgery)

DIFFERENTIAL DIAGNOSIS

- Duodenal ulcer perforation
- Acute pancreatitis
- Retrocecal Acute appendicitis
- Right sided Acute pyelonephritis
- Lobar pneumonia, myocardial infarction
- Ruptured ectopic pregnancy
- Viral hepatitis

TREATMENT

- Admit patient
- Initial conservative management (non-operative)-95%
  - Nasogastric aspiration
  - IV fluids
  - Analgesics and antispasmodics
  - Broad spectrum antibiotics (especially Third generation cephalosporins)
    - Cefoperazone,
    - Ceftazidime,
    - Ceftriaxone,
    - Cefotaxime + amikacin,
    - Tobramycin + Metronidazole
  - Observation
  - Follow up ultrasound
- Later after 3-6 weeks, elective cholecystectomy either by open method through right subcosatal (Kocher's incision) or through laparoscopy is done
- Cholecystotomy is done immediate in patients with:
  - Empyema gallbladder
  - Persisting symptoms
  - Progressing symptoms
- During Cholecystotomy the gallbladder is opened and all stones and pus are

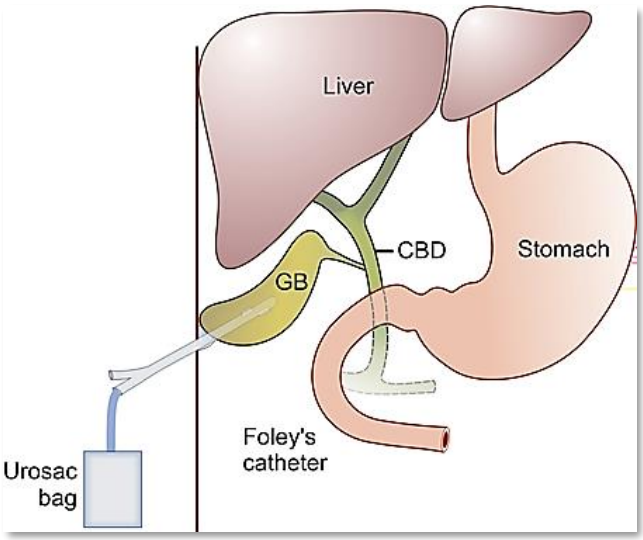


Figure 115: A Foley's or Malecot's catheter is placed in severely ill patients with acute cholecystitis to drain pus initially and later elective cholecystectomy is done. The procedure is done in elderly, severely ill, empyema GB, Emphysematous GB, perforation of GB (cholecystotomy) (Image Courtesy of SRB's Manual of Surgery)

removed. Either a Foley's or Malecot's catheter is placed in the gallbladder and is exteriorized.

- After 3 weeks, elective cholecystectomy is done
- Indications for cholecystotomy or emergency cholecystectomy (5%)
  - Empyema gall bladder
  - Persisting symptoms/failure of medication
  - Emphysematous cholecystitis
  - Perforation/peritonitis
  - Elderly

COMPLICATIONS

- Perforation: this usually occurs in the fundus or in the neck (Hartmann's). It can cause cholecystoduodenal, cholecystointestinal or cholecystobiliary fistula.
- Peritonitis
- Pericholecystitic abscess
- Cholangitis and septicemia
- Empyema gallbladder
  - This is a type of acute cholecystitis carrying high mortality wherein the gallbladder is filled with pus. In 30% of cases pus may be sterile. It also occurs in a pre-existing mucocele of the gallbladder where it gets infected.
  - It is commonly observed in impacted stone, diabetic individuals, immunosuppressed people like HIV or long time steroid therapy.
  - It can perforate, form an abscess or causes peritonitis-biliary and bacterial
  - Clinical features: Fever, toxicity, pain and tenderness in right hypochondrium, tender, smooth, globular, gallbladder is palpable in right hypochondrium to the right of the right rectus muscle.
  - Investigations: Ultrasound of the abdomen, Full blood count (total count is raised), and radioisotope scan
  - Treatment:
    - Antibiotics: Cefotaxime, Quinolones, Ceftriaxone
    - Cholecystectomy- an emergency procedure
    - Often initially cholecystotomy is done with either Foley's or Malecot's catheter kept in situ later after 3-6 week cholecystectomy is done.
  - Complications: septicemia, rupture and peritonitis
- Gangrenous gall bladder.

ACUTE ACALCULOUS CHOLECYSTITIS

- Acute cholecystitis without evidence of gallstones is thought to be due to biliary stasis.
- It is not an uncommon entity (5-10%) but can be commonly missed.
- Risk factors: ICU patients (due to bile stasis and stasis) with multi-organ system failure, trauma (especially after major surgery), burns, sepsis, TPN or any other stress or in cases of cholecystoses.
- The exact cause is not known. Gallbladder distension, release of activation factor VII may be the cause
- Pathology is edema and necrosis of the gallbladder wall with features of acute inflammation
- Presentation is usually acute
- Investigations:
  - Isotope study (HIDA) to confirm diagnosis,
  - Ultrasound of the abdomen: biliary sludge and inflammation is seen. Complications such as gangrene, empyema or perforation of the gallbladder can be seen
  - Labs: leukocytosis with increased ALP, LFTs, amylase and total bilirubin
- Treatment:
  - Urgent Cholecystectomy may be done by open method using Kocher's incision (right subcostal) or via laparoscopy by 4-port technique (umbilical 10-mm, Subxiphoid 10-mm and two 5-mm ports)
  - Percutaneous Ultrasound guided/CT guided or open cholecystostomy initially, later cholecystectomy is the treatment of choice.
  - Safe dissection of the Calot's triangle is key
  - If the whole GB cannot be safely removed partial cholecystectomy should be done.

EMPHYSEMATOUS CHOLECYSTITIS

- This is seen in elderly male patients
- It is common in diabetic and immunosuppressed individuals
- Clostridium Welchii is the causative agent
- Gas is seen in the gallbladder
- It results in life-threatening septicemia
- It causes severe fulminant cholecystitis
- Gangrene, perforation and peritonitis are common
- Emergency cholecystectomy is needed
- Absences of stones is observed in more than 50% of cases

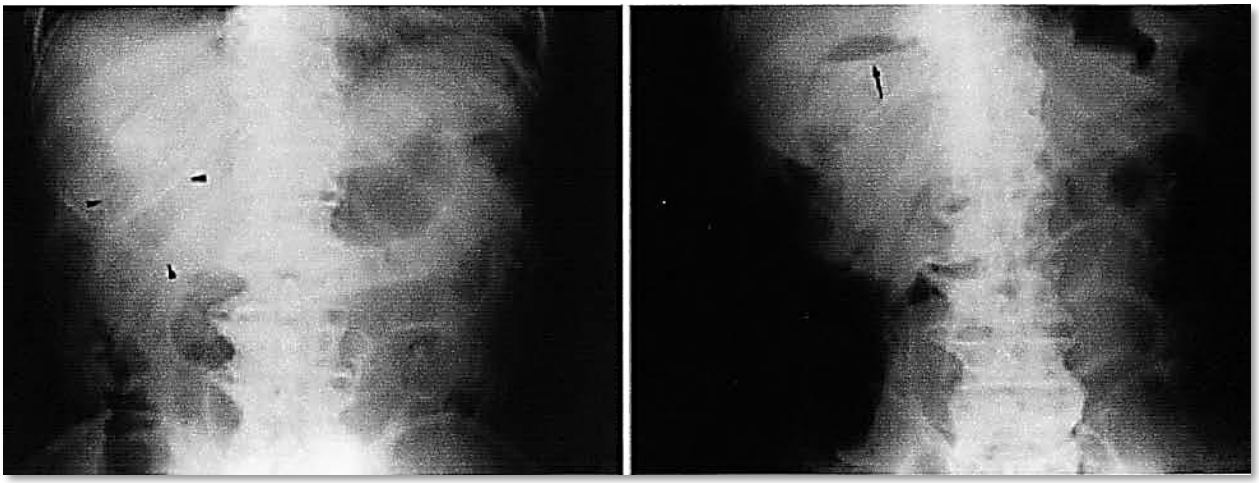


Figure 116: AXR demonstrating rim-like gas within gallbladder (Image from First Aid for the surgery clerkship)

CHRONIC CHOLECYSTITIS

- The is chronic inflammation and thickening of the gallbladder.
- The gallbladder is non-functioning and non-distending

ETIOLOGY

- Gallstones
- Chronic acalculous cholecystitis
- Cholecystoses
  - These are chronic inflammatory conditions of the gallbladder associated with cholesterol deposits.
  - They are due to defective transport of absorbed cholesterol which accumulates in the mucosa. There is also increased absorption of cholesterol by gallbladder epithelium.
  - Cholecystoses gallbladder is more prone to infection
  - It may precipitate stone formation and is a premalignant condition
  - Types:
    - Type 1: Aggregrations of cholesterol crystals in the mucosa or submucosa- cholesterosis (strawberry gallbladder). Lipoid contents are present in large foamy cells which has phagocytosed cholesterol. Here cystic duct is normal. Disease occurs only in gallbladder. It is a premalignant condition
    - Type 2: Cholesterol laden polypoid projections in the mucosa (Cholesterol polyposis/ Gallbladder polyp)
    - Type 3: Granulomatous thickening and hyperplasia of the gall bladder- cholecystitis glandularis proliferans
    - Type 4: Diverticular formation in the wall of the gallbladder (diverticulosis of gallbladder)
    - Type 5: Gallbladder wall fistula
  - Clinical features:
    - Features of acalculus cholecystitis like dyspepsia
    - Positive Murphy's sign
  - Investigations:
    - Ultrasound of the abdomen
    - OCG
    - Isotope study
  - Treatment: Cholecystectomy- always should be done whether it is symptomatic or not as it is potentially malignant.

- Organisms associated with chronic cholecystitis:
  - *Klebsiella*
  - *Streptococci*
  - *Salmonella*

PATHOLOGY

- Gall bladder is shrunken, contracted, small, non-functioning and fibrotic with a thickened wall.
- Mucosa proliferates into the lumen creating deep clefts in the wall projecting into the muscular wall of the gallbladder being lined by epithelium. It is called as Rokitansky-Ashcoff's sinuses.
- Muscular wall is atrophied and is often replaced by fibrous tissue.

CLINICAL FEATURES

- Pain in right hypochondrium, may be colicky or persistent
- Positive Murphy's sign: in the sitting position during deep inspiration, while palpating in the right hypochondrium the patient winces with pain at the summit of the inspiration.
- Moynihan's sign: same as the murphy's sign however elicited when patient is lying down.
- Flatulent dyspepsia
- Intolerance to fatty meals
- Biliary dyspepsia

DIFFERENTIAL DIAGNOSIS

- Peptic ulcer
- Pancreatitis
- Hiatus hernia

INVESTIGATIONS

- Ultrasound of the abdomen: may show stone with posterior acoustic shadowing. Gallbladder wall will be thickened.
- Isotope study may help confirm the infection
- Liver function tests
- Total count may be raised if there is an acute recurrent infection

TREATMENT

- Cholecystectomy (laparoscopic or open method)

COMPLICATIONS

- Common bile duct stones
- Cholangitis
- Pancreatitis
- Mirizzi's syndrome

MUCOCELE OF THE GALLBLADDER

- This is due to obstruction of the cystic duct by a stone in the neck (Hartmann's pouch) of the gallbladder without any infection or inflammation in the gallbladder. This causes absorption of all bile and secretion of mucous into the gall bladder allowing gallbladder to distend causing mucocele of the gallbladder (Hydrops of the gallbladder)
- Cholangiocarcinoma occluding the cystic duct can rarely also cause mucocele of the gallbladder.
- The content is usually sterile and milky white.

CLINICAL FEATURES

- Painless swelling in the right hypochondrium
- Non-tender, smooth, soft, globular palpable gallbladder (content is sterile)
- Features of dyspepsia

INVESTIGATIONS

- Ultrasound of the abdomen
- Liver function tests

TREATMENT

- Cholecystectomy, either laparoscopic or open method.

CHOLANGITIS

TUMORS OF THE BILIARY TRACT

OBSTRUCTIVE JAUNDICE

- This is jaundice that develops due to biliary obstruction which may be partial, complete or intermittent.
- It is also known as surgical or cholestatic jaundice.
- It causes conjugated hyperbilirubinemia (Serum bilirubin> 2.5mg/dl).
- Normal serum bilirubin level is 0.2-0.8mg/dl.
- Sclera icterus is visible when serum bilirubin levels exceeds 2.5mg/dl (35-40 µmol/L)
  - ➢ Jaundice is first seen in the frenulum (underneath the tongue)

To convert mg/dl to µmol/L multiply mg/dl by 17.1 for bilirubin)

METABOLISM OF BILIRUBIN

- Bilirubin is a product of red blood cell breakdown in the spleen. After about 120 days senescent red blood cells are destroyed by the tissue macrophages of the spleen (in the red pulp of the spleen).
- Hemoglobin is split into heme (iron + porphyrin) and the globin part.
- The globin part is broken into amino acids and is utilized for the re-synthesis of hemoglobin.
- The iron from heme is reused for hemoglobin synthesis, it is bound to transferrin and transported to be stored as ferritin in the cells of the liver and bone marrow. Some iron deposits in tissue as hemosiderin.
- The heme is broken down by heme oxygenase to oxy-heme. Oxy-heme is further converted by heme reductase to biliverdin a green pigment.
- Biliverdin is converted into unconjugated bilirubin. The enzyme involved is biliverdin reductase.
- Unconjugated bilirubin is bound to albumin and transported to the liver for conjugation via the splenic artery that joins the superior mesenteric to form the portal vein.
- In the liver bilirubin is bound to cytoplastic proteins ligandins and Z- proteins. Bilirubin is conjugated with 2 glucuronic acid moieties to form conjugated bilirubin. The enzyme involved is uridine 5'diphosphate (UDP) glucoronyltransferase.

- Conjugated bilirubin is then secreted into bile which is stored and concentrated in the gall bladder. Bile is secreted into the duodenum (in response to CCK from I cells in the mucosa of the duodenal cells) from the common bile duct at the major duodenal papilla that is guarded by the sphincter of Oddi at the ampulla of Vater.
- In the intestines bilirubin is deconjugated by bacterial/intestinal enzyme beta-glucorinidase to form free bilirubin which can be reabsorbed in the terminal ileum and taken back to the liver via the portal vein system (the so called enterohepatic circulation)
- Some bilirubin is converted by bacterial dehydrogenase to urobilinogen (a colorless substance). Some of the urobilinogen is excreted in the urine and is converted to urobilin (orange-yellow pigment) by dehydrogenase giving urine its characteristic color.
- The bulk of urobilinogen, bilirubin and urobilin is excreted in feces by conversion of stercobilinogen and stercobilin which gives feces their characteristic orange-yellow color.

CAUSES

- Causes can be classified as:
  - ➢ Intraluminal
    - Common bile duct stones
    - Biliary strictures
    - Pancreatic stricture (due to chronic pancreatitis)
    - Parasitic infestations
    - Biliary atresia
    - Klatskin tumor (carcinoma at the confluence of hepatic ducts above the level of the cystic duct and so will cause hydrohepatosis without GB enlargement)
  - ➢ Mural
    - Cholangiocarcinoma
    - Ascending cholangitis
    - Sclerosing cholangitis: Fibrous obliteration of biliary tract leading to jaundice cirrhosis and portal Hypertension. Associated with IBD (especially ulcerative colitis)
    - Choledochal cyst

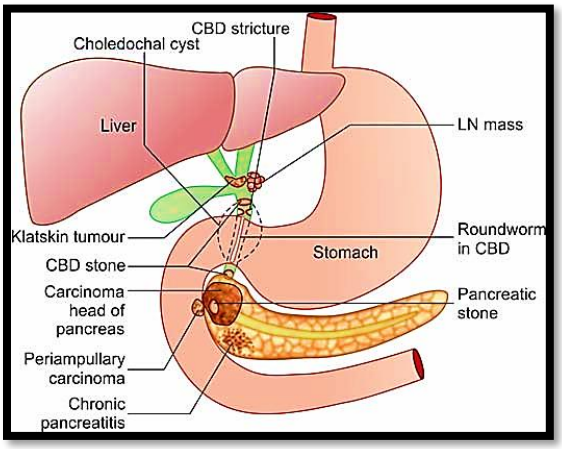


Figure 117: Causes of obstructive jaundice (Image adapted from SRB surgery)

- Extramural
  - Carcinoma of head and periampullary region (pancreas, ampulla, duodenum, distal common bile duct) of the pancreas
  - Extrinsic compression of CBD by lymph nodes or tumors

BENJAMIN'S (1983) CLASSIFICATION OF BILIARY OBSTRUCTION

- Type 1: complete obstruction
  - Tumors: pancreatic, cholangiocarcinoma
  - Common bile duct ligation-iatrogenic
  - Primary/secondary liver tumors
- Type 2: intermittent obstruction
  - Choledocholithiasis
  - Periampullary tumor
  - Choledochal cyst
  - Bile duct papilloma
  - Hemobilia
  - Duodenal diverticula
- Type 3: chronic complete obstruction
  - Bile duct stricture
  - Congenital
  - Traumatic
  - Post-radiotherapy
  - Chronic pancreatitis
  - Cystic fibrosis
- Type 4: Segmental obstruction
  - Traumatic
  - Sclerosing cholangitis
  - Cholangiocarcinoma, intrahepatic biliary stone (hepatolithiasis)

CLINICAL FEATURES

- Features of Obstructive jaundice:
  - Severe jaundice
  - Pruritus (due to deposition of bile salts in the skin), more on the back and forearms
  - Dark urine (due to the presence of conjugated bilirubin in urine)
  - Steatorrhea (fatty stool) due to improper absorption of fats (Recall: bile is required for absorption of fat, it participates in emulsification of fat).
- Fever, may or may not be present
- Loss of weight and appetite
- Pain in right hypochondrium, palpable gallbladder, hydro-hepatotic palpable, smooth, soft, non-tender liver are other features.
- Courvoisier's law suggests inflammatory/neoplastic cause.
  - Courvoisier's law: A palpable gallbladder in the presence of jaundice is unlikely to be due to gallstones.
  - Usually caused by a neoplastic stricture obstructing the distal common bile duct.
  - Rule may not be useful in:
    - Absence of gallbladder
    - Intrahepatic gallbladder
    - Previous cholecystectomy
    - Large stone in Hartmann's pouch
    - Empyema gallbladder with CBD stone
    - Double impacted stone-one in CBD and one in cystic duct
- Complications of vitamin deficiency:
  - Vitamin A: night blindness
  - Vitamin D: Osteomalacia and pathological fractures (poor absorption of calcium)
  - Vitamin E: Poor wound healing, recall vitamin E is an antioxidant
  - Vitamin K: bleeding disorders (Recall factors II, VII, IX and X are vitamin K dependent clotting factors)
- Charcot's triad/Reynold's pentad as presentation in cholangitis.

INVESTIGATIONS

- Blood investigations:
  - Serum bilirubin: normal value <1.0 mg/dl (17.1µmol/l).
    - Both direct (conjugated) and indirect (unconjugated) bilirubin are assessed
    - Direct is increased in obstructive jaundice i.e. conjugated hyperbilirubinemia
    - Van den Bergh's test is done
  - Serum albumin, globulin and A:G ratio
    - Normal Serum albumin is more than 3.5 g/L
  - Prothrombin time: normal value is 12-16 seconds. It is significant if it is more than 4 from the control or more than 1 and half times the control. It is corrected by injection of Vitamin K, 10mg IM OD for 5 days or by FFP-5-10 units.
  - Serum alkaline phosphatase, gamma glutamyltransferase (GGT), AST, ALT, 5' nucleotidase (this is the most reliable since its level is not influenced by bone disease and the enzyme is not induced by alcohol)
    - ALP, 5' nucleotidase and gamma glutamyltransferase are elevated
    - ALT and AST are minimal or sometimes not elevated
  - Full blood count: Shows neutrophilia in inflammatory conditions.
  - Tumor markers:
    - CA 19/9 is useful for carcinoma pancreas (more than 70 units/L) with 70% sensitivity and 90% specificity. But it may also increase in other causes of biliary obstruction and cystadenoma.
    - Alpha-fetoprotein is raised in hepatocellular carcinoma.

		Hepatocellular		
Type of jaundice	**Haemolytic**	**Early**	**Late**	**Obstructive**
Serum bilirubin				
Unconjugated	Elevated	Normal/elevated	Normal/elevated	Normal
Conjugated	Normal	Normal	Elevated	Very elevated
Urinary bilirubin	Normal/elevated	Elevated	Elevated	Very elevated
Urinary urobilinogen	Normal/elevated	Normal	Decreased	Very decreased
Liver function tests				
Alkaline phosphatase	Normal	Normal	Elevated	Very elevated
γ-GT	Normal	Elevated	Elevated	Very elevated
Transaminases	Normal	Elevated	Very elevated	Normal/elevated
Lactate dehydrogenase	Normal	Elevated	Very elevated	Normal/elevated
Reticulocytes	> 2%	Normal	Normal	Normal

- Imaging:
  - Abdominal ultrasound: may show gallstone.
  - Endoscopic retrograde cholangiopancreatography (ERCP): to visualize the site of obstruction, brush biopsy, bile sample for analysis.
  - Percutaneous transhepatic cholangiography (PTC): to decompress, assess proximal dilated obstructed biliary system if ERCP fails, dine polythene catheter can be kept in situ to have biliary drainage. PTC-stenting across the obstruction can be done under image (C arm) guidance
  - Magnetic resonance cholangiopancreatography (MRCP): Non-invasive diagnostic tool. It shows 96% sensitivity and 99% specificity.
  - CT scan in case of tumors to assess operability.
  - Endoscopic ultrasound (EUS): it is done through endoscope. It is more accurate in assessing pancreatic mass, staging disease, to identify involvement of portal venous system, common bile duct stones. It is also useful in endoscopic ultrasound guided Fine needle aspiration and cytology, celiac axis neurolysis and EUS guided immunotherapy.
  - Intraductal ultrasound (IDUS): it is very useful in assessing tumor stage, tumor margin in bile duct cancer. It is also used in assessing pancreatic duct to differentiate pancreatic cancer and chronic pancreatitis.
  - CT/MR angiogram or venogram: in assess vascularity and portal venous system in malignancy.
- Urine tests: Fouchet's test for bile pigments, Hay's test for bile salts and test for urobilinogen in urine. Conjugated bilirubin is water soluble and is therefore filtered in the glomerulus and present in urine.
  - Fouchet's test: 10ml of urine + 5ml of $BaCl_2$ + Pinch of $MgSO_4$ causes formation of $BaSO_4$ which is filtered over a filter paper and few drops of Fouchet's reagent is added. Green or blue color signifies presence of bile pigments in the urine.
  - Hay's test for bile salts: Sprinkle Sulphur to 2ml of urine. In presence of bile salts Sulphur sinks to the bottom.
  - Ehrlich's test: 5 ml of freshly voided urine + 1ml of Ehrlich reagent (p-dimethyl amino benzaldehyde) and wait for 5 minutes. Formation of red color signifies presence of urobilinogen in urine. Normally it is present in traces, in obstructive jaundice it is absent and in hemolytic jaundice it is in excess.

MANAGEMENT

- Treat underlying cause
- Management involves:
  - Establishing the cause of the jaundice
  - Assessment of the general condition of the patient
  - Staging in patients with tumors
  - Appropriate treatment (which may be surgical, endoscopic or radiological)

PREOPERATIVE

- Proper diagnosis and assessment (history and physical examination)
- Correction of metabolic abnormalities, improvement of the general condition and institution of specific measures designed to minimize the incidence of complications associated with prolonged or severe cholestasis such as:
  - Infections (cholangitis, septicemia, wound infection)
  - Disorder of clotting mechanism
  - Renal failure
  - Liver failure
  - Fluid and electrolyte abnormalities
- Hypokalemia is frequently present and should be corrected.
- Perform a viral hepatitis panel.
- Vitamin K IM 10mg-20mg for 3-5 days.
- Fresh frozen plasma- often requires 6 bottles or more
- Adequate hydration is most important 5/10% dextrose for 12-24 hours before surgery followed by an osmotic diuretic (mannitol 100-200ml BD IV) or a loop diuretic administered intravenously at the time of induction.
- Catheterize and monitor urine output hourly
- Blood transfusion in case of anemia
- Oral neomycin, lactulose for hepatic encephalopathy. As well as correction of hypokalemia, restriction of sedative, hypnotics and potent analgesics with prompt treatment of infection.
- Repeat monitoring by doing prothrombin time, electrolytes
- Prophylactic antibiotics like third generation cephalosporins especially for:
  - All jaundiced patients
  - Patients with rigors and pyrexia
  - Patients undergoing emergency biliary procedures/operations
  - Elderly patients

  - Patients with common duct stones even if not jaundiced
  - Patients undergoing secondary biliary intervention
- Calcium supplements e.g. calcium chloride IV
- Preoperative decompression: indicated if bilirubin is >12mg/dl (>150 μmol/L), impending liver failure, sepsis, Hepatorenal syndrome, severe malnutrition or cardiopulmonary disease
  - Preoperative decompression is achieved by insertion of a plastic endoprosthesis or by endoscopic sphincterotomy in patients with periampullary cancer.
- Correction of coagulopathy, prevention of renal failure, infection, hepatic encephalopathy, and electrolyte imbalance (correction of hypoglycemia and dilutional hyponatremia due to water retention, avoiding isotonic saline infusion)

OPERATIVE

- Depends on the cause
- Common bile duct stones- ERCP stone removal, choledocholithotomy, transduodenal sphincteroplast, choledochojejunostomy or choledochoduodenostomy.
- Carcinoma periampullary or head of pancreas- Whipple's operation or triple bypass or ERCP stenting.
- Biliary stricture- Stenting, choledochojejunostomy, Roux-en-Y hepaticojejunostomy
- Klatskin tumor- radical resection or palliative stenting
- Biliary atresia- Kasai's operation or liver transplantation
- Choledochal cyst- excision, hepaticojejunostomy, mucosal resection
- Management of pruritus: pruritus may be due to retention of bile salts which activates the release of histamine in skin, central mechanism or by release of endogenous opioids. It is often difficult to treat. Once cause is treated and obstruction is relieved pruritus will regress.
  - Drugs and therapies used: Cholestyramine (ion exchange resin binds bile salts in intestine inhibiting their absorption), rifampin, ondansetron, gabapentin, sertratine, urosodeoxycholic acid, antioxidants, phototherapy, plasmaphereis.

POST OPERATIVE

- Monitoring with prothrombin time, bilirubin, albumin, creatinine, electrolyte estimation
- FFP or blood transfusion

- Antibiotics
- Observation for septicemia, hemorrhage, pneumonia, pleural effusion, bile leak
- Care of T-tubes and drains
- T-tube cholangiogram in 10-14 days.
- Total parenteral nutrition, Central venous pressure line, nasogastric tube, urinary catheter.

CLINICAL BOX: A case of fever, pain and jaundice

History:

As the junior doctor on call, you are asked to review the blood results of an 87-year-old man, who was admitted that morning with possible appendicitis. He is confused and unable to give an accurate history. He had been spiking temperature during the afternoon and had increasing right-sided abdominal pain.

Examination:

The observation chart shows he has a temperature of 38ºC and tachycardia of 120/min. You notice he has a yellow discoloration of the skin and sclera and abdominal examination reveals that the maximal tenderness is in the right upper quadrant. There are no palpable masses or abdominal herniae. Rectal examination demonstrates normal stool stool with no palpable rectal mass. A plain abdominal radiograph, done that morning, was normal.

Investigations done: FBC, Serum amylase, Urea & Electrolytes, Creatinine, AST, ALT, GGT, Bilirubin, Albumin, Blood glucose, C-reactive protein (CRP)

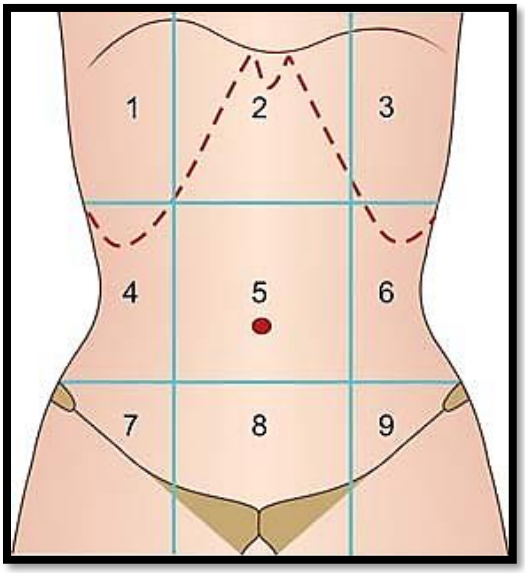
		Normal
Haemoglobin	15 g/dL	11.5–16.0 g/dL
Mean cell volume	82 fL	76–96 fL
White cell count	21 × 10^9/L	4.0–11.0 × 10^9/L
Platelets	344 × 10^9/L	150–400 × 10^9/L
Sodium	136 mmol/L	135–145 mmol/L
Potassium	4.5 mmol/L	3.5–5.0 mmol/L
Urea	6 mmol/L	2.5–6.7 mmmol/L
Creatinine	72 µmol/L	44–80 µmol/L
Amylase	69 IU/dL	0–100 IU/dL
Aspartate transaminase (AST)	68 IU/L	5–35 IU/L
Alkaline phosphatase (ALP)	442 IU/L	35–110 IU/L
Gamma-glutamyl transferase (GGT)	121 IU/L	11–51 IU/L
Bilirubin	92 mmol/L	3–17 mmol/L
Albumin	42 g/L	35–50 g/L
Blood glucose	4.0 mmol/L	3.5–5.5 mmol/L
C-reactive protein (CRP)	212 mg/L	0–6 mg/L

Questions:

1. What is the likely diagnosis?
2. What are the classical characteristics to indicate this?
3. What are the most common causes?
4. Which are the most common organisms?
5. What investigations should be performed?

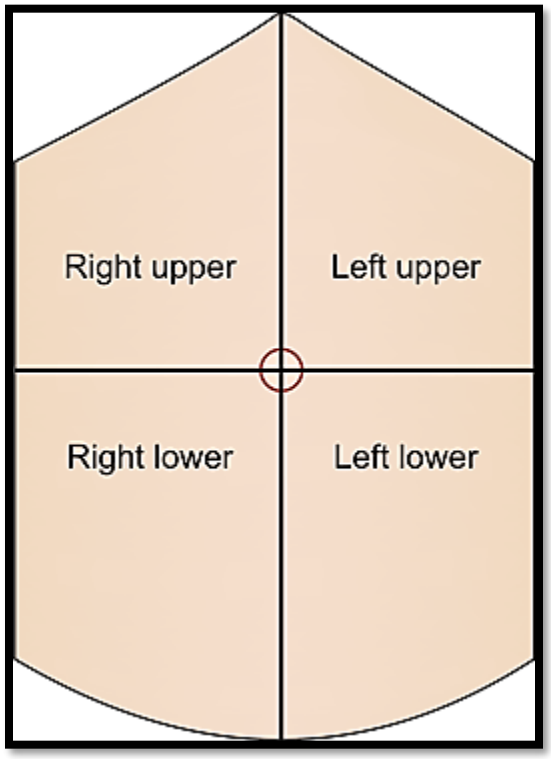
DIVSIONS OF THE ABDOMEN

- Recall that the abdomen is divided by 4 lines into 9 regions.
  - Transpyloric line: a line midway between the umbilicus and xiphisternum
  - Transtubercular line: as the levels of two tubercles on the iliac crest
  - Right vertical line: through the midpoint of right anterosuperior iliac spine and pubic symphysis
  - Left vertical line: through the midpoint of left anterosuperior iliac spine and pubic symphysis



- The 9 regions are thus:
  1. Right hypochondrium
  2. Epigastic region
  3. Left hypochondrium
  4. Right lumbar region
  5. Umbilical region
  6. Left lumbar region
  7. Right iliac fossa
  8. Hypogastrium
  9. Left iliac fossa

- The abdomen can also be divided into 4 quadrants by two perpendicular lines through the umbilicus.



- The 4 quadrants are thus:
  - Right upper quadrant
  - Left upper quadrant
  - Right lower quadrant
  - Left lower quadrant

PAINFUL ANAL CONDITIONS

- The rectum is 15cm long and extends from the rectosigmoid to the anorectal line. It curves with the sacrum and contains 3 folds.
- The rectal wall consists of the mucosa, submucosa and 2 complete muscle layers: the inner circular and the outer longitudinal.
- The anal canal is 3.5cm long and it extends from the anorectal line to the anus.
- The dentate line (pectineal line) divides the anal canal to an upper part that contains columnar epithelium and autonomic innervation, from an endoderm origin as well as a lower part that contains squamous epithelium (continuous with perianal skin) and pudendal somatic innervation with ectoderm origin.
- The columns of Morgagni begin at this line and extend cephalad.
- Pain from above the dentate line is visceral, diffuse and poorly localized while pain from below the dentate line is somatic and well localized.
- Within the anal canal lie anal sphincters
  - The internal sphincter is a circular muscle continuous with the inner circular smooth muscle layer and is under parasympathetic control
  - The External sphincter blends with the outer longitudinal skeletal muscle layer with 3 parts subcutaneous, superficial and deep muscle and is under voluntary control via the pudendal nerve (S4). The superior border of the external sphincter fuses with the puborecalis muscle which forms a sling originating at the pubis and joining behind the rectum.
- The space between the internal and external sphincter (intersphincteric plane) is a fibrous continuation of the longitudinal smooth muscle of the rectum. Normally 6 to 10 anal glands lie in this space. Each gland has a duct and discharges into the anal crypt of the dentate line. Infection originating in these glands is the primary cause of perirectal abscess and fistula in ano.

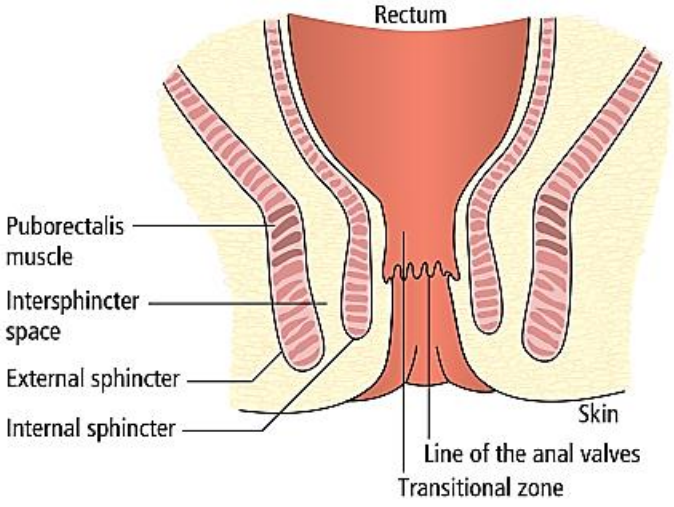


Figure 118: Cross-section through the anal canal and lower rectum showing the important anatomical relations of the regions

HEMORRHOIDS/PILES

- The word 'Hemorrhoid' is derived from Greek words Haima (bleed) + Rhoos (flowing), mean bleeding.
- The pile is derived from the Latin word 'Pila' means ball.
- Piles- a ball or mass, Hemorrhoids= blood to ooze.
- The anal canal is lined by the anal cushions, which consist of 3 thick vascular submucosal bundles that always lie in the left lateral (3 O'clock), right posterior lateral (7 O'clock) and right anterior lateral positions (11 O'clock) in lithotomy position.

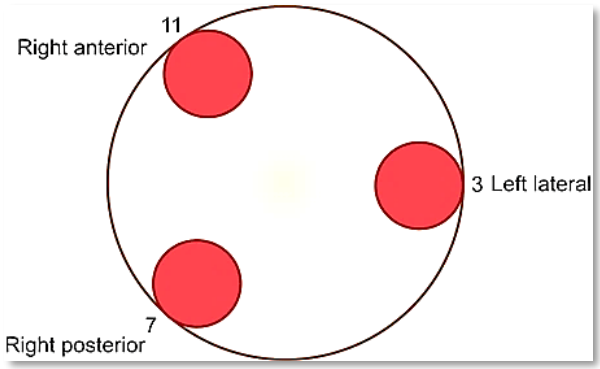


Figure 119: Anal cushions

- The functions of these cushions is not entirely clear but they aid continence (anal tone) and engorge during defecation to protect the anal canal from abrasions.
- Hemorrhoids are the downward sliding of the anal cushions abnormally due to straining or other causes.
  - Anal cushions are aggregations of blood vessels (Arterioles, venules), smooth muscles and elastic connective tissue in the submucosa that normally reside in left lateral, right posterolateral and right anterolateral anal canal.
  - These positions can be found at 3, 7 and 11 o'clock.
- Hemorrhoids consist of dilated venous plexus, a small artery and areolar tissue.
- Piles can be mucosal or vascular.
  - Vascular type is seen in young.
  - Mucosal is seen in old.
- Present concept is weakening of Park's ligament which is the lower end of the external sphincter.

TYPES

- Hemorrhoids can either be:
  - Internal- above the dentate (pectineal) line, covered with mucous membrane
  - External- below the dentate (pectineal) line, covered with skin
  - Interno-external- together occur.

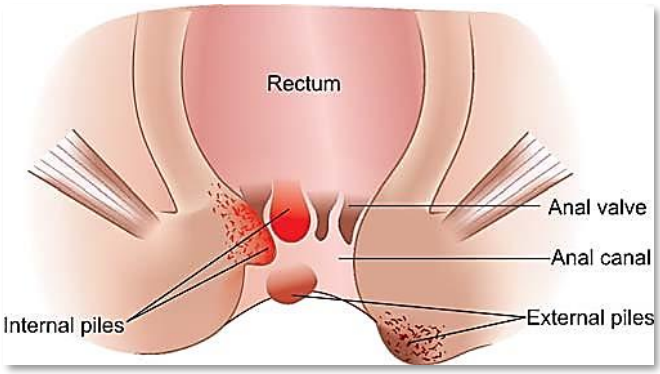


Figure 120: Types of hemorrhoids

CLASSIFICATION

- Hemorrhoids can be classified according:
  - Location
  - Severity

ACCORDING TO LOCATION

- Primary hemorrhoids: Located at 3, 7, 11 o'clock positions related to the branches of the superior hemorrhoidal vessels which divides on the right side into 2, and on the left side it continues as 1.

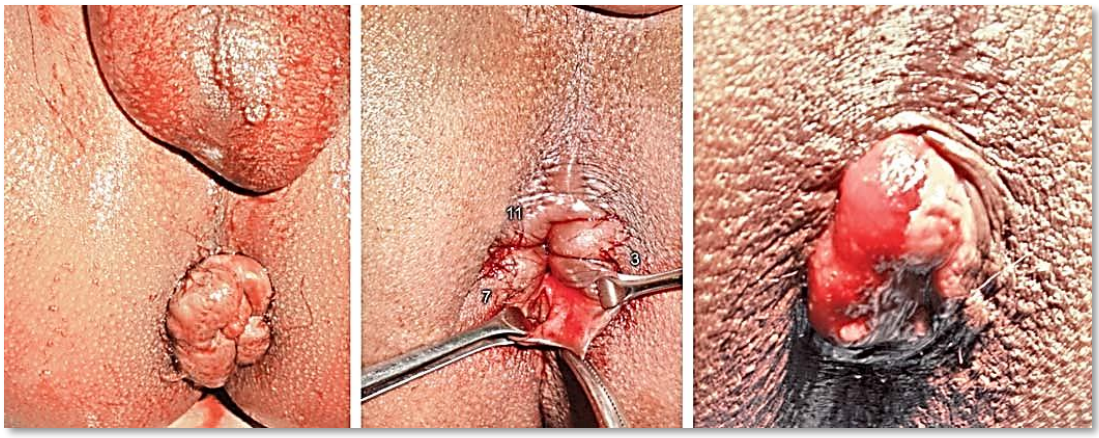


Figure 121: Hemorrhoids

- Secondary hemorrhoids: One which occur between the primary sites.

ACCORDING TO SEVERTIY

- First degree: piles within that may bleed but does not come out
- Second degree: piles that prolapse during defecation but returns back spontaneously
- Third degree: piles prolapsed during defecation, can be replaced back only by manual help

- Fourth degree: pile that are permanently prolapsed.

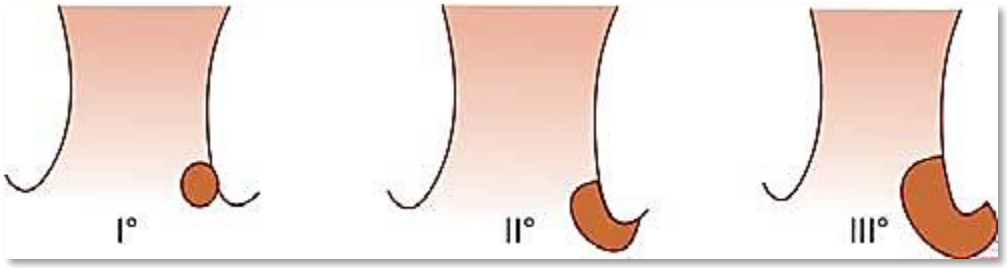


Figure 122: Classification of piles

- Piles begin as pedicle and it is located at the origin of the internal pile i.e. at the level of anorectum.

ETIOLOGY

- Hereditary
- Idiopathic
- Morphologic: weight of the blood column without valves causes high pressure. Veins in the lower rectum are in loose submucosal plane but the veins above enter the muscular layer, which on contraction increases the venous congestion below (more prevalent in patients with constipation). Superior rectal veins have no valves (as they are tributaries of portal vein) and so more congestion.
- Other causes:
  - Straining, diarrhea, constipation, hard stool, low fiber diet, over-purgation
  - Carcinoma rectum, portal hypertension (rare cause)
  - Pregnancy: during pregnancy raised progesterone relaxes the venous wall and reduces its tone, enlarged uterus compresses the pelvic vein and constipation is a common problem.

CLINICAL FEATURES

- It occurs at any age but mostly between 30 to 65 years.
- Incidence is equal in both sexes.
- Painless Bleeding- 1st symptom- 'splash in the pan'- 'bright red and fresh'- occurs during defecation
  - Note: mucus or fecal soiling may occur, though in general changes in bowel habit are uncommon and should alert to other cause
- Mass per anum
- Discharge- a mucoid discharge

- Pruritus
- Pain- may be due to prolapse, infection or spasm
- Prolapse of internal hemorrhoids may produce moisture in the anal region or mucus discharge that causing itching.
- Anemia- secondary

EXAMINATION

- On inspection, prolapsed piles will be visualized
- On P/R examination, only thrombosed piles can be felt.

DIFFERENTIAL DIAGNOSIS

- Carcinoma
- Rectal prolapse
- Perianal warts
- Causes of bleeding per anum
  - Piles
  - Fissure in ano
  - Fistula in ano
  - Polyps
  - Ulcerative colitis
  - Amoebic colitis
  - Carcinoma rectum and carcinoma colon
  - Diverticulitis
  - Intussusception
  - Vascular anomaly of the colorectum
  - Mesenteric ischemia

INVESTIGATIONS

- Proctoscopy: note number, degree, size, surface and appearance of piles as well as features of chronicity of the prolapse.
- Sigmoidoscopy or colonoscopy: malignancy
- Endoscopy to rule out other sources of rectal bleeding e.g. cancer and inflammatory bowel disease.
- Barium enema: malignancy
- Full blood count-check for anemia (hematocrit) and platelet count

COMPLICATIONS

- Profuse hemorrhage which may require blood transfusion
- Strangulation- by anal sphincter
- Thrombosis- piles appear dark purple/black, feels solid and tender
- Ulceration
- Gangrene
- Fibrosis
- Stenosis
- Suppuration leads to perianal or submucosal abscess
- Pylephlebitis (portal pyaemia) is rare but can occur in 3rd degree piles after surgery.

TREATMENT

- External hemorrhoids are best treated with analgesia and warm soaks. If pain is severe, the thrombosed hemorrhoids must be excised.
- Mild bleeding and protrusion of external hemorrhoids can be controlled with conservative treatment including avoidance of constipating foods, increasing the fiber content of the diet and using bulk-forming stool agents.
- The above measures along can resolve all symptoms.
- Rubber-band ligation a simple outpatient procedure can be used to treat 1st and 2nd degree hemorrhoids. Injection with phenol and almond oil could play a similar role.
- 3rd and 4th degree hemorrhoids and mixed internal/external hemorrhoids require hemorrhoidectomy.

Preventive	Therapeutic/curative
Diet- more fiber/ liquid, laxatives	Medical ➢ Sitz bath- immersing anal region for 15-20 minutes, 2-3 times a day. To reduce edema, pain and promote healing ➢ Analgesics, local anesthetic cream ➢ Antihistamines ➢ Antibiotics ➢ Laxatives ➢ Local anesthetics and anti-inflammatory drugs e.g. annomax
	Parasurgical ➢ Sclerotherapy ➢ Banding ➢ Cryotherapy ➢ Infrared coagulation (IRC) ➢ Laser therapy ➢ Doppler guided haemorrhoidal artery ligation (DGHAL): good for pregnant and diabetic patients
	Surgical ➢ Open haemorrhoidectomy (Milligan-Morgan)-Britain ➢ Closed haemorrhoidectomy (Hill-Fergusson)- USA ➢ Stapled haemorrhoidopexy (Antonio Longo) ➢ Anal stretching- Recamier, Lord's

Severity	Medical	Parasurgical	Surgical
1st degree	• Medical interventions are usually enough	• Sclerotherapy can be done • DGHAL can be done	
2nd degree	• Medical interventions are usually enough	• Sclerotherapy can be done • Barron's banding is done • DGHAL can be done	
3rd degree	• Medical interventions can help patients especially before surgery	• Laser therapy can be done • DGHAL can be done	• Hemorrhoidectomy is gold standard
4th degree		• DGHAL can be done	• Hemorrhoidectomy is gold standard

ANAL FISTULA (FISTULA IN ANO)

- A fistula is an abnormal communication between two epithelial/endothelial lined surfaces surrounded by granulation tissue.
- A fistula in ano has its external opening in the peri-rectal skin and its internal opening in the anal canal at the dentate line.
- Pain is rare with anal fistula. Patients often complain of peri-rectal itching, irritation and discharge.
- It is believed that fistula in ano can be:
  - Crytoglandular (90%) and associated with infection of the intersphincteric glands
  - Non-cryptoglandular (other causes)-10%

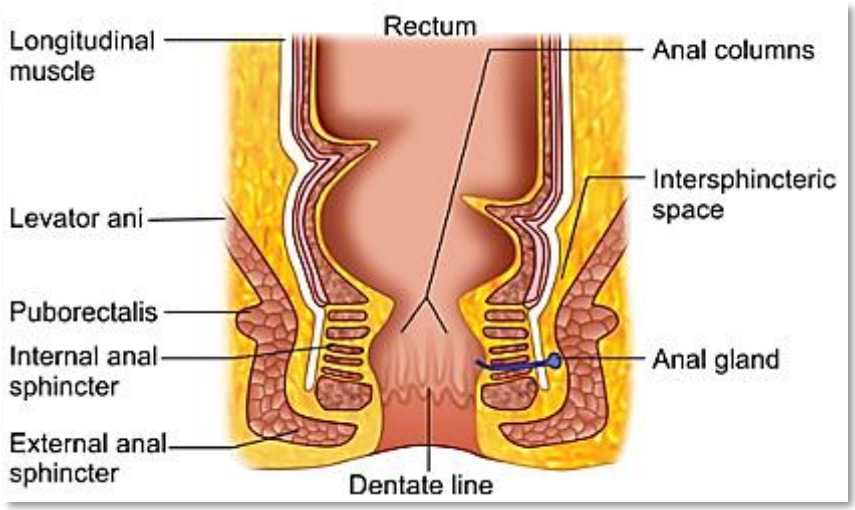


Figure 123: Anatomy of anal glands (Cryptoglands of Morgagni)

ETIOLOGY

- Drainage (40%) or rupture of an anorectal abscess (i.e. anal gland infection) (most common cause)
  - Anal fistula forms during the chronic stage of an acute inflammatory process that begins in the intersphincteric anal glands.
  - Extension of the acute inflammation can result in a supralevator, ischiorectal or perianal abscess.
  - With chronic inflammation the abscess communicates with the external surface forming supralevator trans-sphincteric or intersphincteric fistulae.
- Crohn's disease
- Diverticulitis with perforation and fistula to the perineum
- Infection: HIV, TB, actinomycosis, lymphogranuloma venereum
- Malignancies of the distal rectum, anal canal or perianal skin
- Trauma: foreign body, iatrogenic, anal fissure
- Hydradenitits suppurative

CLASSIFICATION

- Fistulae can be either:
  - Low level fistulas: these open into the anal canal below the internal ring
  - High level fistulas: these open into the anal canal at or above the internal ring
- Standard classification (Milligan Morgan-1934 and Goligher-1975)
  - Pelvi-rectal fistula
  - Submucosal fistula
  - Subcutaneous fistula (most common)
  - High anal fistula
  - Low anal fistula (common)

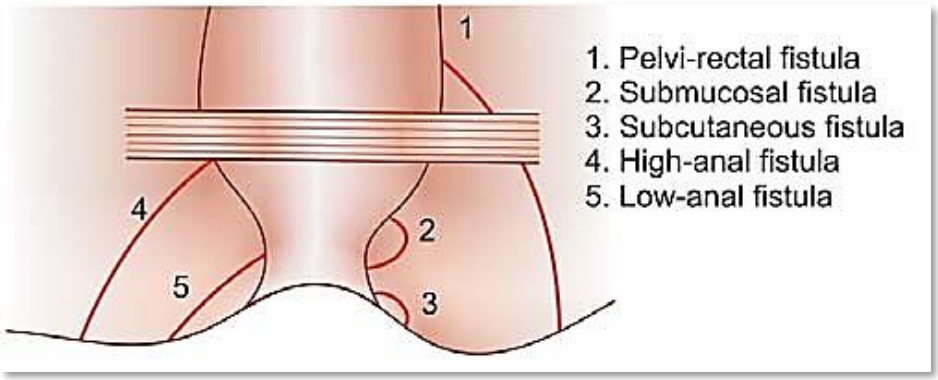


Figure 124:Standard classification

- Park's classification (1976)
  - Intersphincteric fistula (70%-commonest)
  - Transsphincteric fistula (25%)
  - Supralevator/Suprasphincteric fistula (4%)
  - Extrasphincteric fistula (1%)

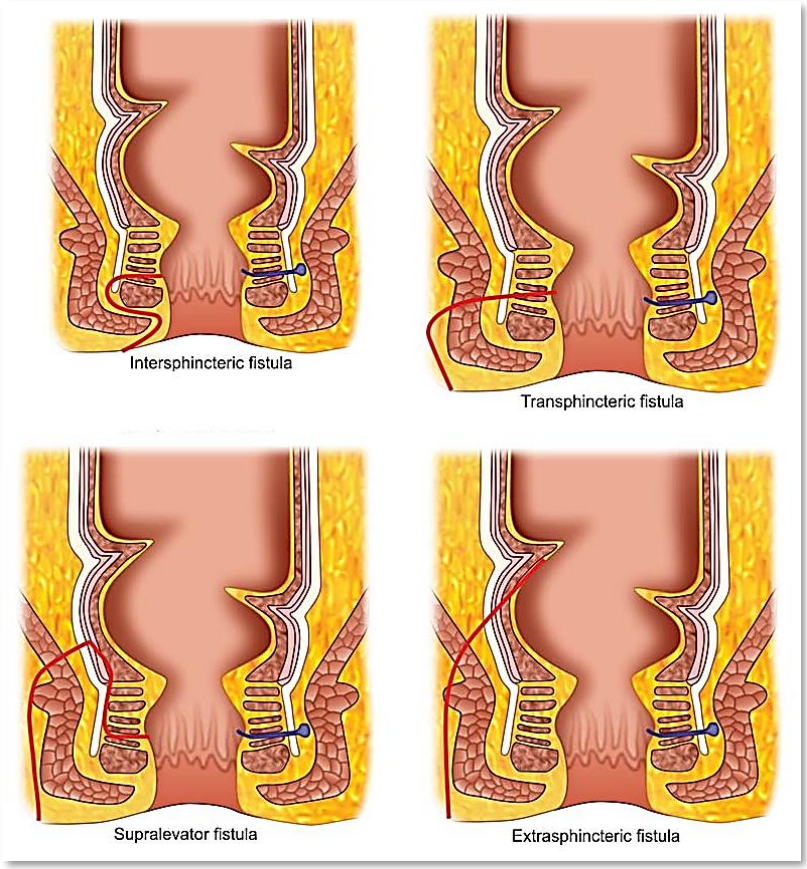


Figure 125: Park's Classification

CLINICAL FEATURES

- Persistent symptoms or 'non-healing' following abscess drainage
- Discharge (continuous or intermittent) and soiling
- Pruritus
- Rectal pain which may be worse on sitting and defecation
- Untreated can cause sepsis and anatomical changes leading to incontinence
- On examination:
  - Goodsall's rule:

- Relates to the location of the internal and external openings of a fistula in ano.
- Fistulas with an external opening in relation to the anterior half of the anus is of a direct type.
- If the anus is bisected by a line in the frontal plane, an external opening inferior to that line connects the internal opening via a short direct tract.
- If the external opening is posterior to the imaginary line, the fistula tract follows a curved route to the internal opening in the posterior midline, it may present with multiple external openings all connected to a single internal opening.
- An exception is an external opening that is anterior to this imaginary line and more than 3cm from the anus, in which case the tract may be curve posterior and end in the posterior midline.

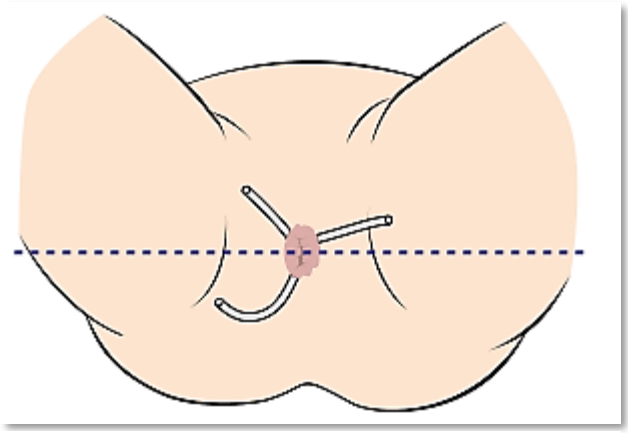


Figure 126: Goodsall's rule

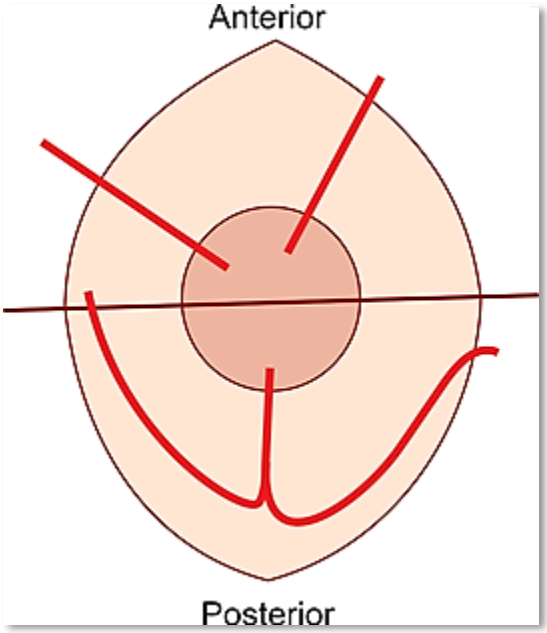


Figure 127: Goodsall's rule. Anterior fistulae are having straight track. Posterior fistulae are having curved tracks with internal opening in the posterior midline

- ➢ Examination under anesthesia if there is significant discomfort
- ➢ P/R examination shows indurated internal opening usually in the midline posteriorly.
- ➢ Most of the fistulae are on posterior half of the anus.

INVESTIGATIONS

- Simple fistulas are diagnosed clinically
- Chest X-ray, ESR and barium enema X-ray.
- If required fistulogram is done only under anesthesia
- MRI/MRI fistulogram is ideal
- Endorectal U/S (US perineum) is useful to assess deeper plane.
- Dscharge study, methylene blue dye study, biopsy
- Colonoscopy often when ulcerative colitis/Crohn's is suspected

- Specific blood test

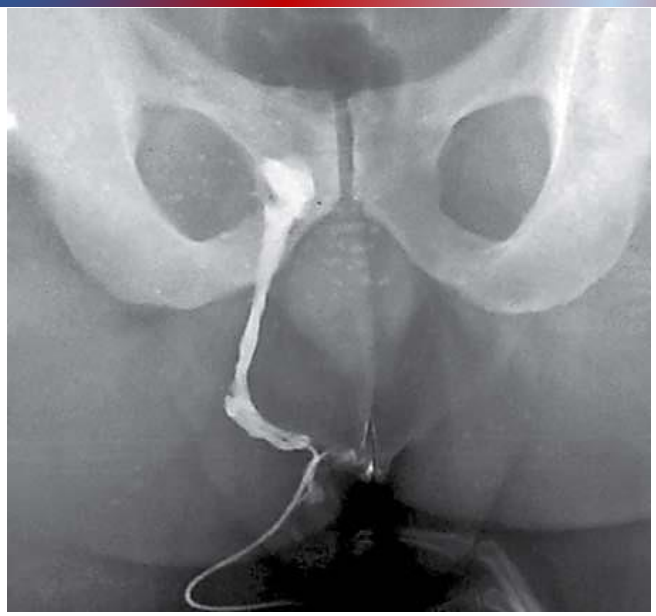


Figure 128: Fistulogram X-ray showing track

MANAGEMENT

- The mainstay of treatment is to lay the fistula tract open. Procedure depends on the type of fistula
- Low fistula tract (superficial, intersphincteric, low transphincteric) options:
  - Fistulotomy in which the fistula is opened and allowed to heal by secondary intention
  - Injection of fibrin glue/sealant
  - Internal sphincterotomy is the treatment for an intersphincteric fistula
- High fistula tract options:
  - Fistulotomy and placement of seton cord which is left in fistula to allow drainage during healing.
    - In the first stage, a seton suture is placed around the sphincter to stimulate fibrosis adjacent to the sphincter muscle
    - In the second stage performed 6-8 weeks later, the intersphincteric portion of the fistula tract is laid open
    - This is done to prevent incontinence.
  - Advancement flaps: adjacent mucosa used to seal internal opening.
  - Fistula plug
  - Ligation of intersphincteric fistula tract (LIFT)

ANAL FISSURE

- An anal fissure is a tear in the anal canal, frequently associated with sudden pain or bleeding during defecation.
- The longitudinal tear (ulcer) in the anal canal can occur anywhere from below the dentate line to the anal margin.
- 90% of anal fissures are in the 6 O'clock position. It may be associated with skin tags or a sentinel pile.
- Causes:
  - Straining in constipation
  - Childbirth
  - Trauma
  - IBD
- Management:
  - Initial advice:
    - Avoid staining
    - Minimize constipation: increase fiber and fluid intake, consider laxative
    - Immerse anus in warm water with shallow bath or Sitz bath kit, 2-3 times daily.
  - Topical medical treatment:
    - 0.4% Glyceryl trinitrate ointment is first line
    - Other options: diltiazem 2%, Botox
  - Surgical:
    - Lateral anal sphincterectomy in which internal sphincter fibers are divided over the length of the fissure. This may be required for chronic anal fissures.
    - Further option is excision and anal advancement of flap.

PILONIDAL SINUS



Figure 129: Pilonidal sinus and abscess

- A pilonidal sinus is a hair-containing sinus or abscess that usually involves the skin and adjacent tissue in the intergluteal region.
- Most patients have pain, swelling and drainage when these sinuses become infected.
- Most investigators believe that this condition is caused by ingrowth of hair, although whether it is acquired or congenital is not known.
- Pilonidal disease can occur at any age but is most prevalent between adolescence and the third decade of life.
- Recurrent infections are common.
- The treatment depends on the phase of the disease at presentation.
- Pilonidal abscess is best treated by incision and drainage. However, for a pilonidal cyst, results of treatment remain imperfect although numerous methods have been reported. The simplest methods of treatment are incision and drainage, and curettage with secondary healing or cyst excision and closure.

- Anorectal abscess is due to obstruction of anal glands/crypts at the dentate line with subsequent infection by gut bacteria.
- Locations include:
  - Perianal (60%): limited to skin of anal canal. It results when pus spreads downwards between the 2 sphincters. It manifests as a tender swelling of the anal verge
  - Ischiorectal (20%): crosses the external anal sphincter. It is formed if a growing intersphincteric abscess penetrates the external sphincter below the puborectalis. Infection can spread into the fat of the ischiorectal fossa and the abscess can become quite large
  - Intersphincteric: between the internal and external sphincters. It results from infection in the anal glands. Fecal coliforms are typically the offending organisms. As the abscess enlarges within the intersphincteric plane, it can spread in any several directions.
  - Supralevator (Rare): superior extension above levator. It develops when an intersphincteric abscess expands upwards between the internal and external sphincters
- Abscess rupture, spontaneous or surgical leads to fistula formation in 50% of patients

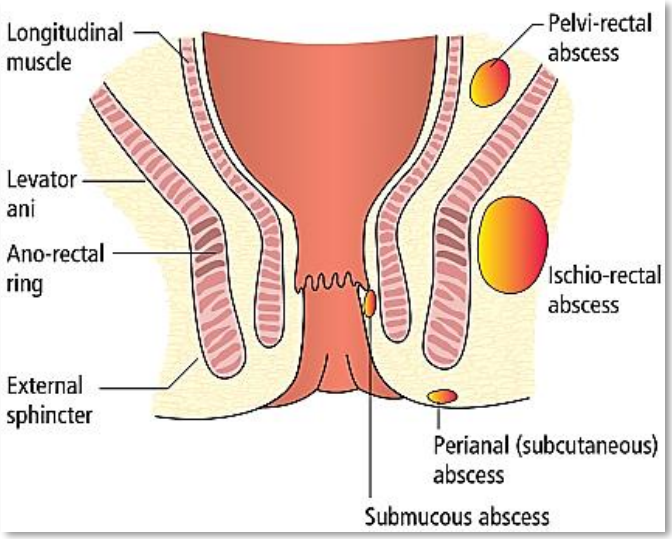


Figure 130: Common sites of anorectal suppuration

RISK FACTORS

- These may cause abscess formation via anal gland infection and/or separate mechanisms:
  - Bowel inflammation: IBD (especially Crohn's), diverticulitis, TB, Hidradenitis suppurativa
  - Immunosuppression: diabetes, HIV
  - Trauma: rectal foreign bodies, receptive anal sex

- Demographic and social: male, aged 20-60

CLINCAL FEATURES

- Perianal pain and pruritus. Pain may be worse on siting and defecation
- Constipation due to painful defecation
- Deeper abscesses may cause systemic symptoms
- On Examination:
  - Perianal swelling (external or felt by P/R)
  - Purulent/bloody discharge
  - Examination under anesthesia if there is significant discomfort

INVESTIGATIONS

- Can often be diagnosed clinically.
- Deeper abscesses may require endoanal US or MRI

DIFFERENTIAL DIAGNOSIS

- Pilonidal abscess
- Hydradenitis suppurativa
- Folliculitis
- Periprostatic abscess
- Bartholin gland abscess
- Inflammatory bowel disease
- Actinomycosis
- Tuberculosis

MANAGEMENT

- Incision and drainage:
  - Can be done in ward/ED for simple perianal abscesses otherwise will require theater.
  - The primary opening of the anal crypt is rarely identified.
  - Most wounds can be left open, without packing. Intersphincteric abscesses may require post-op drain.
  - Intersphincteric abscess is treated by using the proctoscope to observe the bulging area and then performing an internal sphincterotomy over the abscess.
  - Supralevator abscesses are drained through the appropriate ischiorectal space or through the rectum.

- Horsehoe abscess (those that surround the rectum) are drained through the rectum with corner drains placed in each ischiorectal abscess.
- Post-procedure, advise frequent sitz baths (water to hip height) and consider laxative to reduce pain defecation.

- Antibiotics (in addition to Incision and drainage) only if there is evidence of sepsis, extensive cellulitis or immunosuppression.

HERNIA

- A hernia is a protrusion of a viscus or part of a viscus through the wall of its containing cavity.
- A hernia comprises of a hernial sac and neck.
- The abnormal opening may be artificial or natural (area of weakness or disruption of the fibromuscular tissues of the body wall).
- Herniae may occur in the brain (e.g. after head injury), in the muscle through its fascial covering, internally in the abdomen (internal hernia) or externally through a weak normal opening (inguinal and femoral herniae) or an abnormal opening (e.g. incisional hernia) in the abdominal wall.
- 75-85% of abdominal wall hernias are groin hernias.
  - 15% of males will develop groin hernia.
  - 5% of females will develop groin hernia.
- Inguinal hernias are the most common types of hernias (73%) because the muscular anatomy in the inguinal region is weak and also due to the presence of natural weakness like deep ring and cord structures.
- Femoral hernias account for 17%, umbilical hernias account for 8.5% and others account for about 1.5% (excluding incisional hernia).
- In general, incisional hernia is next to inguinal hernia in occurrence.

Common herniae

- Inguinal hernia (73%)
- Incisional hernia
- Femoral hernia (17%)
- Umbilical hernia (8.5%)
- Epigastric
- Other herniae (1.5%)

- Risk factors:
  - "Increased intra-abdominal pressure"
    - Straining, lifting of heavy weights
    - Chronic constipation (habitual, rectal stricture),
    - Chronic cough (tuberculosis, chronic bronchitis, bronchial asthma, emphysema),
    - Urinary causes: old age (BPH, carcinoma prostate), Young age (stricture urethra), very young age- Phimosis, meatal stenosis
    - Ascites
  - Wall weakness

- Obesity, pregnancy and pelvic anatomy (especially in femoral hernia in females),
- Smoking
- Previous surgery: Appendectomy through McBurney's incision may injure the ilioinguinal nerve causes right sided direct inguinal hernia.
- Familial collagen disorder- Prune Belly syndrome
- Acquired herniation is also probably due to collagen deficiency called as metastatic emphysema of Read.
- An indirect inguinal hernia occurs in a congenital, preformed sac i.e. the remains of processus vaginalis. Chances of presence of bilateral preformed sac is 60%.

PARTS OF A HERNIA

- The hernia comprises of:
  - Covering
  - Sac
  - Content

COVERING

- These are the layers of the abdominal wall through which the sac passes.

SAC

- This is a diverticulum of peritoneum with mouth, neck, body and fundus.
- The neck is narrow in indirect sac but wide in direct sac.
- The body of the sac is thin in infants.

CONTENTS

- Omentum- Omentocele (epiplocele). Difficult to reduce the sac later, initially it can be reduced easily.
- Intestine- Enterocele- commonly small bowel, but sometimes even large bowel. Difficult to reduce the sac initially.
- Richter's hernia: a portion of circumference of bowel is the content.
- Urinary bladder may be the content or part of the posterior wall of the sac- cystocele.
- Ovary, often with fallopian tube.
- Meckel's diverticulum- Littre's hernia.
- Amyand hernia: this is an inguinal hernia containing the vermiform appendix. It should not be confused with a De Garengeot hernia in which a femoral hernia contains the appendix.

CLASSIFICATION OF HERNIA

- Hernias can be classified:
  - Clinically
  - According to etiology
  - According to the contents
  - Anatomical classification

CLINICAL CLASSIFICATION

- Hernias can be classified as:
  - Reducible hernia: hernia gets reduced on its own or by the patient or by the surgeon.
    - Intestine reduces with gurgling and it is difficult to reduce the first portion.
    - Omentum is doughy, and it is difficult to reduce the last portion.
    - Exanpsile impulse on coughing present.
  - Irreducible (incarcerated) hernia: contents cannot be returned to the abdomen due to narrow neck, adhesions, overcrowding. Irreducibility predisposes to strangulation.
    - Obstructed hernia: it is an irreducible hernia with bowel inside that is obstruction, but blood supply to the bowel is not interfered.
    - Strangulated hernia: it is an irreducible hernia with obstruction to blood flow. The swelling is tense, tender, with no impulse on coughing and with features of intestinal obstruction. Features of intestinal obstruction may be absent in case of Omentocele, Richter's hernia, Littre's hernia.
  - Inflamed hernia: it is due to inflammation of the contents of the sac e.g. appendicitis, salpingitis. Here hernia is tender but not tense, overlying skin is red and edematous.

ETIOLOGICAL CLASSIFICATION

- Hernias can be classified as either
  - Congenital- common
    - Occur in a preformed sac/defect. Clinically may present at a later period due to any of the precipitating causes like in indirect inguinal hernia.
  - Acquired
    - It is secondary to any causes which raise the intra-abdominal pressure leading into weakening of the area like in direct inguinal hernia.

CLASSIFICATION ACCORDING TO CONTENTS

- Omentocele- omentum
- Enterocele- intestine
- Cystocele- urinary bladder
- Littre's hernia- Meckel's diverticulum
  - Note: Littre described Meckel's diverticulum in a hernia sac 81 years before Meckel was born.
- Richter's hernia- part of the bowel wall. Only part of the lumen protrudes, it can cause strangulation without obstruction.
- Maydl's hernia (hernia-en-W)- W-shaped loop (two adjacent loops) in hernial sac with tight, strangulated neck. This is a rare form of inguinal hernia; 2 loops of intestine are incarcerated in the sac. The intervening loop of small intestine remains in the abdominal cavity becomes strangulated by compression of its mesenteric vessels at the neck of the sac.
- Sliding hernia- contains partly extra-peritoneal structures (e.g. cecum or sigmoid colon) such that the hernial sac does not totally surround the contents (or in other words a viscus forms part of the wall)

ANATOMICAL CLASSIFICATION

- Depend on the site of the hernia including:
  - Abdominal wall herniae
    - Inguinal hernia
    - Femoral hernia
    - Epigastric hernia
    - Umbilical hernia
    - Paraumbilical hernia
    - Incisional hernia
    - Spigelian hernia: this is an interstitial hernia of abdominal wall. The defect occurs at the lateral border of the rectus abdominis, emerging through a defect in the transversus and internal oblique fascia halfway between the umbilicus and pubic symphysis.
      - The swelling is diffuse and difficult to palpate as it is covered by the external oblique.
      - It may be identified by its position above and medial to the location of an inguinal hernia.
  - Internal:
    - Diaphragmatic (hiatus or congenital)
    - Mesenteric
    - Omental

- Others
  - Obturator hernia
  - Lumbar hernia

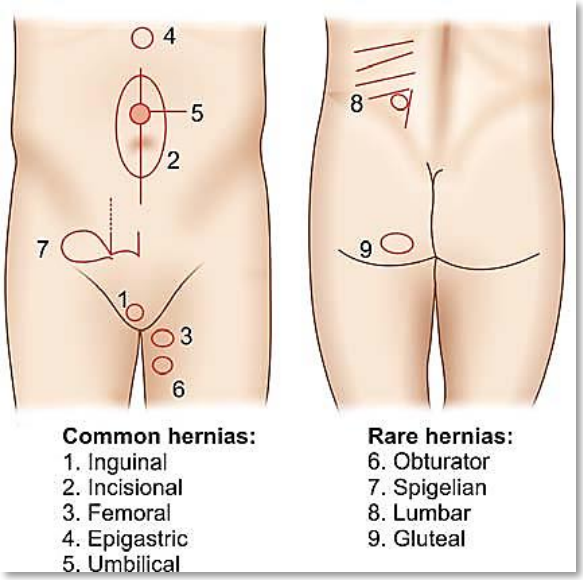


Figure 131: Herniae (Image adapted from SRB's Manual of Surgery 2nd Ed)

- Herniography (by Gullmo): injection of contrast material into the peritoneal cavity and taking films in supine and prone positions to diagnose protrusions of peritoneal sac is called as herniography.
- It was earlier also used for diagnosing undescended testis.

MANAGEMENT OF HERNIA

- Assess the hernia for:
  - Severity of symptoms
  - Risk of complications (type, size of neck)
  - Ease of repair (size, location)
  - Likelihood of success (size, 'loss of right of abode')
- Assess the patient for fitness for surgery, impact of hernia on lifestyle (job, hobbies)
- Principles of surgery:
  - Herniotomy: Excision of the hernial sac.
  - Herniorrhaphy: repair of the defect
  - Hernioplasty (ideal)

INGUINAL HERNIA

- Inguinal hernia is a common complaint, occurring mostly in men. It is twice as frequent as femoral hernia and in males more than 90% of herniae are inguinal.
- They may occur at any age.
  - In children they are generally associated with developmental disorder e.g. persistent processus vaginalis or testicular maldescent.
  - In young adult males they are also related to congenital defects such as a persistent processus, which may be precipitated from potential to actual existence by physical effort.

SURGICAL ANATOMY OF INGUINAL CANAL

- The inguinal canal is an oblique passage in lower part of abdominal wall, 3.5-4cm long, situated above the medial half of inguinal ligament, extending from deep inguinal ring to superficial inguinal ring.
  - In infants both superficial and deep rings are superimposed without any obliquity of the inguinal canal.
  - Inguinal canal in female is called as "Canal of NUCK"
- The Superficial inguinal ring is a triangular opening in the external oblique aponeurosis and is 1.25cm above the pubic tubercle.
  - The ring is bounded by a superomedial and inferolateral crus.
  - Normally the ring does not admit the tip of little finger.
  - It corresponds to the midpoint of the inguinal ligament (halfway the point between the anterior superior iliac spine and the pubic tubercle)
- The Deep inguinal ring is a U-shaped condensation of the transversalis fascia that lies 1.25cm above the inguinal ligament midway between the symphysis pubis and the anterosuperior iliac spine (mid-inguinal point).
- The Inguinal (Poupart's) ligament is formed by the lower border of the external oblique

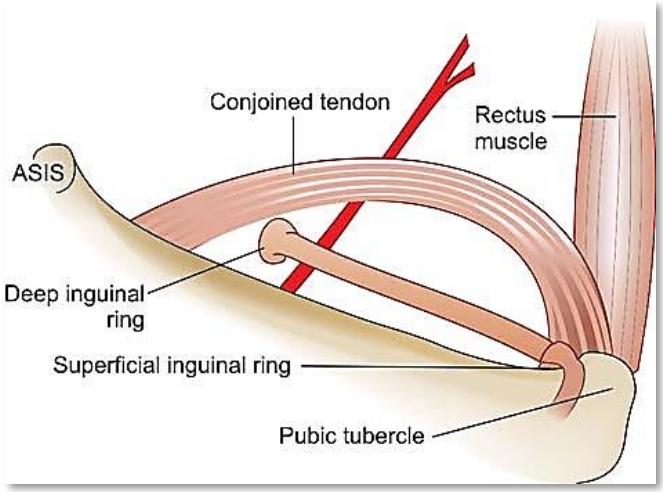


Figure 132: Anatomy of the inguinal canal (Image adapted from SRB's Manual of Surgery 2nd Ed)

aponeurosis which is thickened and folded backwards on itself, extending from anterosuperior iliac spine to pubic tubercle.

- Contents of the inguinal canal:
  - Ilioinguinal nerve (L1)
  - Spermatic cord in male or round ligament in females
- Contents of the spermatic cord:
  - Vas deferens
  - Artery to vas
  - Testicular and cremasteric artery
  - Genital branch of genitofemoral nerve (L1, L2)
  - Pampiniform plexus of veins
  - Remains of processus vaginalis
  - Sympathetic plexus around the artery to vas
- Coverings of the spermatic cord:
  - Internal spermatic fascia from fascia transversalis
  - Cremasteric fascia
  - External spermatic fascia from external oblique aponeurosis is seen below the external ring in the scrotum

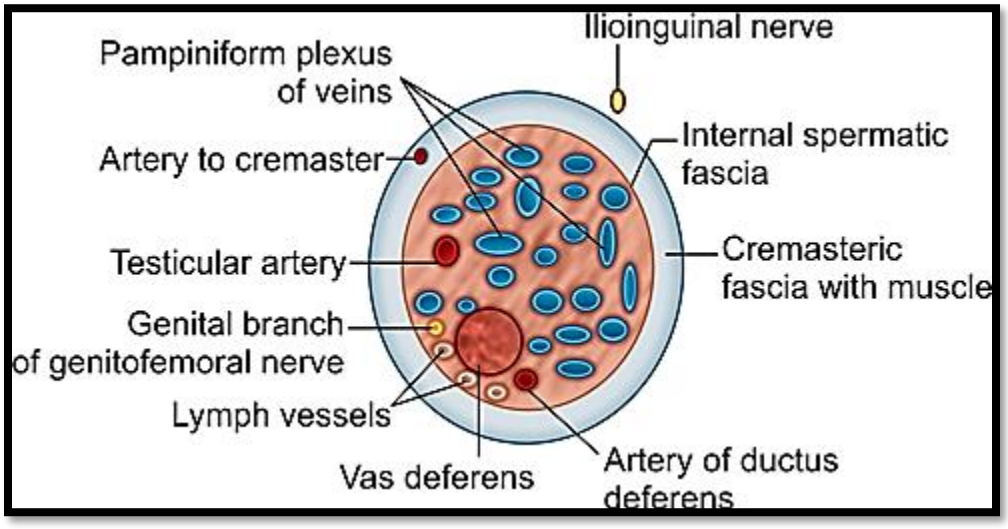


Figure 133: Coverings and Contents of the spermatic cord (Image adapted from SRB's Manual of Surgery 2nd Ed)

- Boundaries of the inguinal ligament:
  - Anteriorly: External oblique aponeurosis (medial) and conjoined muscle (internal oblique and transversus abdominis) laterally.

- Posteriorly: inferior epigastric artery, transversalis fascia and insertion of the conjoined tendon medially.
- Superiorly (roof): conjoined tendon (Arched fibers of internal oblique and transversalis abdominis)
- Inferiorly (floor): inguinal ligament

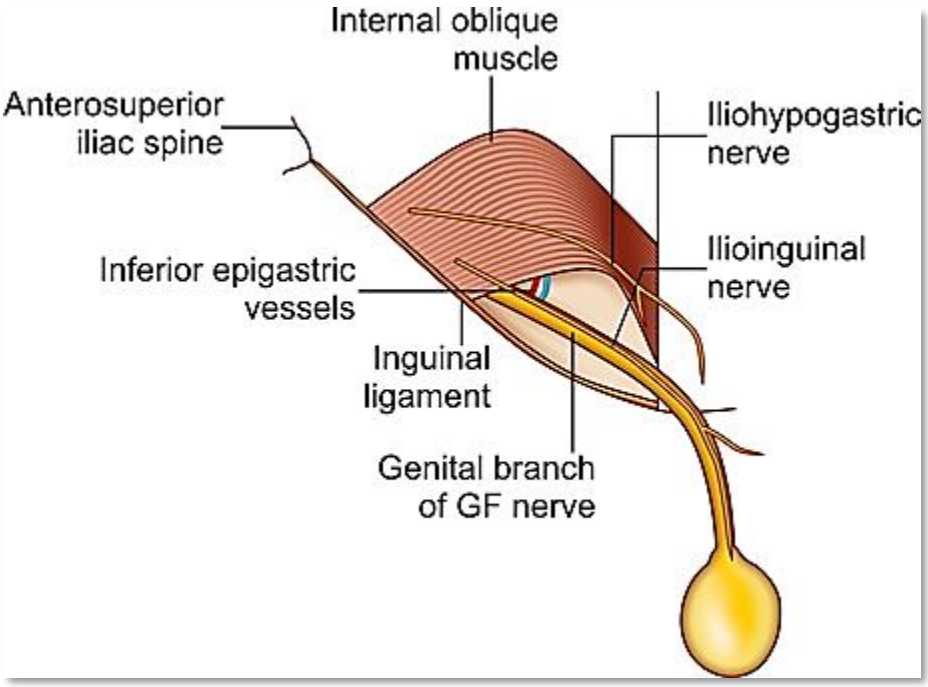


Figure 134: The inguinal canal (Image adapted from SRB's Manual of Surgery 2nd Ed)

- Defence mechanism of inguinal:
  - Obliquity of inguinal canal
  - Arching of conjoint tendon
  - "Shutter mechanism" of internal oblique
  - "Ball value mechanism" due to contraction of cremaster muscle which plugs to superficial ring
  - When external oblique muscle contracts, intercrural fibers of superficial ring appose causing "slit valve mechanism"
  - Hormones

CLASSIFICATION OF INGUINAL HERNIA (EARLIER)

- This includes:
  - Anatomical classification
  - Classification according to the extent

ANATOMICAL CLASSIFICATION (IN INGUINAL HERNIA)

- These can be classified as either indirect hernia or direct hernia.
- Indirect hernia: it comes out through internal (Deep) ring along with the cord. It is lateral to the inferior epigastric artery. The inguinal ligament is inferior to the hernia
- Direct hernia: it occurs through the posterior wall of the inguinal canal through "Hesselbach's triangle" (bounded medially by lateral border of rectus muscle, laterally by inferior epigastric artery, below by inguinal ligament). Sac is medial to the inferior epigastric artery.

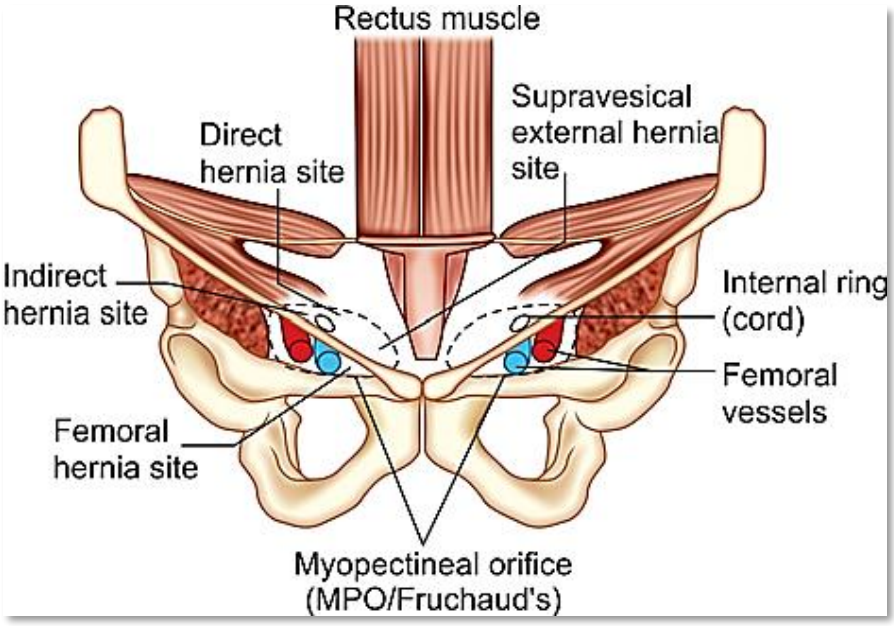


Figure 135: Herniae (Image adapted from SRB's Manual of Surgery 2nd Ed)

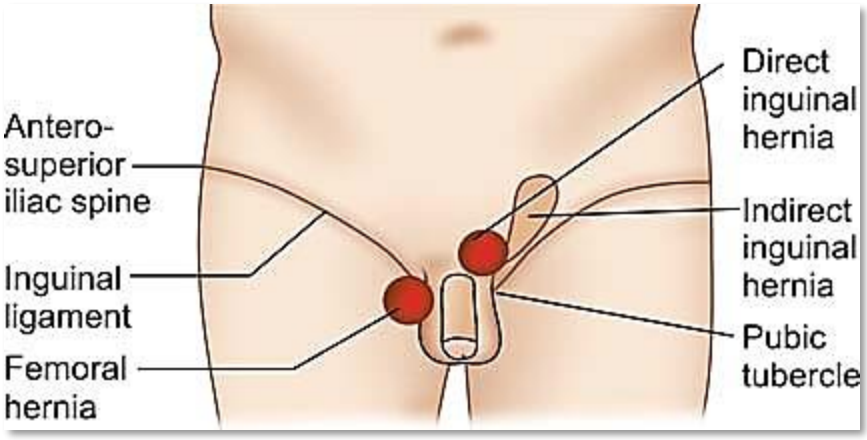


Figure 136: Herniae (Image adapted from SRB's Manual of Surgery 2nd Ed)

CLASSIFICATION TO EXTENT

- Types:
  - Incomplete:
    - Bubonocele: sac is confined to the inguinal canal
    - Funicular: here sac crosses the superficial inguinal ring, but does not reach the bottom of the scrotum.
  - Complete: here sac descends to the bottom of the scrotum.
  - Saddle-bag or pantaloons hernia sac has got both medial and lateral component.
- Note: inguinal hernia is above and medial to the pubic tubercle. Femoral hernia is below and lateral to pubic tubercle.



Figure 137: Bubonocele (Image adapted from SRB's Manual of Surgery 2nd Ed)

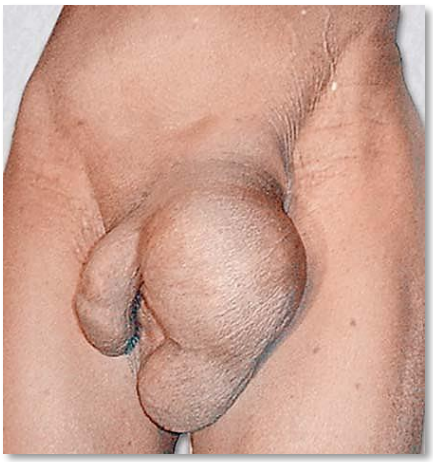


Figure 138: Funicular (Image adapted from SRB's Manual of Surgery 2nd Ed)

INDIRECT (OBLIQUE) INGUINAL HERNIA

- This is the most common type of hernia (65%).
- It is more common in younger age group as compared to direct inguinal hernia which is more common in elderly.

- It is more common on right side in 1st decade but in 2nd decade the incidence is equal on both sides.
- Hernia is bilateral in 30% of cases.
- An indirect hernia occurs when a sac develops by emerging through the deep inguinal ring and passing through the inguinal canal in the spermatic cord adjacent to the vas deferens and surrounded by the coverings of the cord.
- Sac is thin in indirect type. Neck is narrow and lies lateral to inferior epigastric vessels.
- The sac, if sufficiently large, may emerge through the external inguinal ring (funicular inguinal hernia) or descend into the scrotum (complete inguinal scrotal hernia)



Figure 139: Complete inguinal (Image adapted from SRB's Manual of Surgery 2nd Ed)

- Coverings of indirect hernia (from inside out)
  - Extra-peritoneal tissue
  - Internal spermatic fascia
  - Cremasteric fascia
  - External spermatic fascia
  - Skin
- It may contain omentum or small bowel and has the potential to become irreducible and strangulated.
- Precipitating cause: smoking, obesity, respiratory causes (bronchial asthma, TB, bronchitis), ascites, previous surgery e.g. appendectomy which can cause direct inguinal hernia, urinary problems like BPH, urethral stricture, straining, multiple pregnancies, chronic constipation (due to anorectal strictures), rectal stricture due to chronic proctitis (amoebic), TB of anorectum, previous anorectal surgery, rectal carcinoma or stricture due to lymphogranuloma venereum.
- Types:
  - Incomplete
    - Bubonocele: where the hernia is limited to the inguinal canal
    - Funicular: Processus vaginalis is closed just above the epididymis. Contents of the sac can be felt separately from testis which lie below the hernia.
  - Complete (Scrotal): Testis appears to lie within the lower part of hernia. It can occur in any age group. It occurs in a congenital preformed sac (processus

vaginalis). More commonly contents descend into the pre-existing sac, only when there are precipitating cause which force the content down.

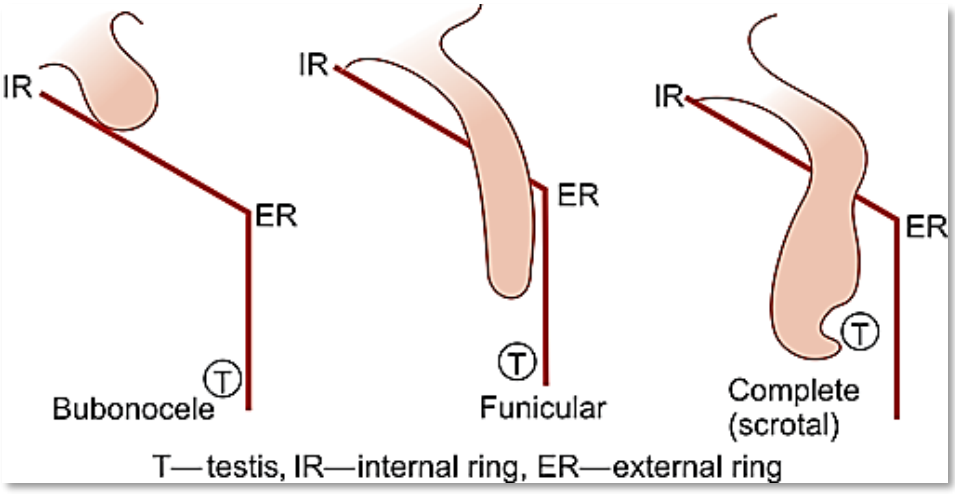


Figure 140: Types of indirect inguinal herniae (Image adapted from SRB's Manual of Surgery 2nd Ed)

CLINICAL FEATURES

HISTORY

- Prevalence of inguinal hernia is 25% in males, 2% in females.
- It is more common in males (20:1).
- Patient presents with dragging pain and swelling in the groin which is better seen while coughing and standing and felt together with an expansile impulse.
- In complete type, the content descends down to the scrotum completely.
- Contents are either small bowel, large bowel, omentum or combination of all these. In females, sometimes ovary and tubes may be the content. In infants, swelling appears when the child cries and is often translucent.
- It is usually reducible but can go for irreducibility, inflammation, obstruction, strangulation.

PHYSICAL EXAMINATION

- Internal ring occlusion test:
  - Internal ring is located half inch (1.25cm) above the mid-inguinal point (center point between anteriosuperior iliac spine and pubic symphysis). After reducing the contents, in lying down position, internal ring is occluded using the thumb.
  - Patient is asked to cough.
  - If a swelling appears medial to the thumb, then it is a direct hernia.

- If swelling does not appear and on releasing the thumb swelling appears during coughing, then it is an indirect hernia confirmed in standing position.
    - It is seen above and medial to the pubic tubercle

- Ring invagination test: After reduction of hernia, the little finger/ index finger of the examiner is invaginated from the bottom of the scrotum, gradually pushed up and rotated to enter the superficial inguinal ring. The impulse on coughing is felt at the tip of the invaginated finger. This test is done only in males.
- Zieman's test: The examiner places his index finger on the deep inguinal ring and middle finger on the superficial inguinal ring, ring finger over saphenous opening. The patient is asked to cough or to hold the nose and blow. It the impulse is felt on the index finger (covering the deep ring), it is indirect hernia.



Figure 141: Zieman's test (Image adapted from SRB's Manual of Surgery 2nd Ed)

- Head or leg rising test: this is done to look for abdominal wall muscle tone and Malgaigne bulging. Valsalva manoeuver is also used to check the tone of abdominal wall muscles.



Figure 142: Head rising test (Image adapted from SRB's Manual of Surgery

- Abdominal, respiratory, urological examination is done to look for any precipitating factors like chronic bronchitis, ascites, stricture urethra, BPH
- Per rectal examination is a must
- Inguinal hernia in females:
  - Increased thickness of labium majus on palpation, when compared to contralateral side.



Figure 143: Left inguinal hernia in females (Image adapted from SRB's Manual of Surgery 2nd Ed)

- Silk glove sign: index finger is invaginated across scrotum towards the external ring. When patient coughs, inguinal hernia is felt as a slit like sensation.
- Palpation of bulbar urethra for stricture (thickening/crescent like feel/ button like depression).

USE THE FIVE FINGERS OF THE HAND TO COMPLETE ALL TESTS FOR HERNIA

Thumb- for deep ring occlusion test

Index, middle and ring finger for Zieman's test

Little finger for superficial ring invagination test

- Rules of hernia examination
  - Never forget to examine opposite side
  - Never forget to do Per rectal examination
  - Never forget to examine urethra
  - Never forget to check abdominal muscle tone
- Differential diagnosis:
  - Hydrocele- infantile/ encysted/large vaginal/ bilocular
  - Undescended testis
  - Femoral hernia
  - Lipoma of the cord
  - Hydrocele of the canal of neck (in females)
  - Inguinal lymph node enlargement
  - Groin abscess

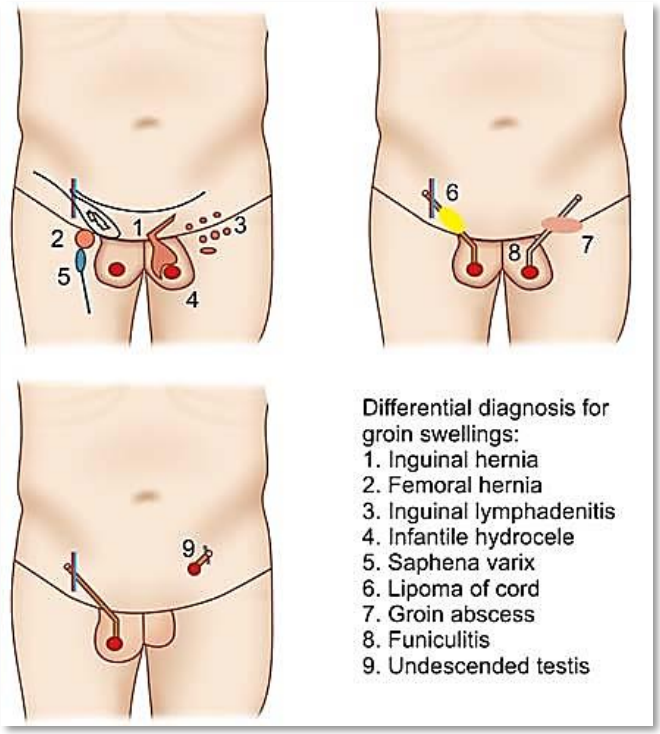


Figure 144: Differential diagnosis for groin swelling (Image adapted from SRB's Manual of Surgery 2nd Ed)

INVESTIGATIONS

- Ultrasound of abdomen
- Chest X-ray to rule out chronic bronchitis
- Tests relevant for precipitating causes

DIRECT HERNIA

- Direct hernia occurs through Hesselbach's triangle which is bounded by inferior epigastric artery laterally, lateral border of rectus medially and inguinal ligament below.
  - The hernia is medial to the inferior epigastric artery with a wide neck. The sac is thick and often the medial wall or content may be bladder.
  - Hesselbach's triangle is divided into medial and lateral halves by obliterated umbilical artery (medial umbilical ligament).
  - Direct hernia is classified as medial or lateral depending on which part of the Hesselbach's triangle it is arising from.
- Direct hernia accounts for 10-15% of hernias.

- 50% of direct hernias occur bilateral.
- 35% of inguinal hernias are direct.
- It is uncommon in females and children.
- It is always acquired due to weakening of posterior wall of inguinal canal.
- Coverings of direct hernia (From inside out):
  - Extraperitoneal tissue
  - Transversalis fascia
  - Conjoined tendon
  - External spermatic fascia
  - Skin
- Predisposing factors: chronic cough, smoking, straining, BPH, constipation, heavy work, previous appendectomy.
- A direct hernia usually appears as a diffuse bulge that cannot be controlled by pressure over the internal ring. It is above and lateral to the pubic tubercle.
- Malgaigne bulgings are often seen in these patients on examination more often than in indirect hernia. They are protrusions of abdominal wall muscle during leg raising test as weak, soft, supple, swellings which signifies poor abdominal muscle tone.



Figure 145: Bilateral direct inguinal hernia (Image adapted from SRB's Manual of Surgery 2nd Ed)

- Direct hernia rarely descends into the scrotum and strangulation is not common as in indirect hernia. But in longstanding cases, it can descend down to the scrotum and strangulation can occur.

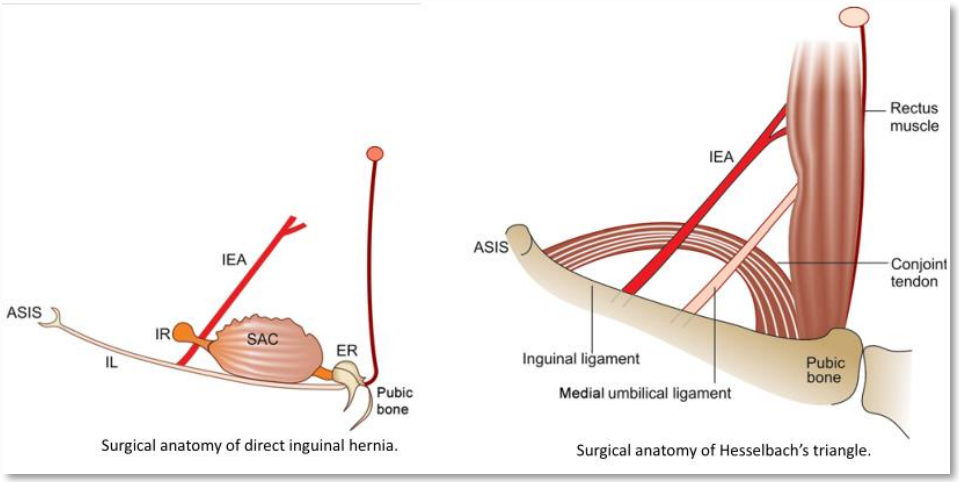


Figure 146: Direct inguinal hernia (Image adapted from SRB's Manual of Surgery 2nd Ed)

- Investigations same as indirect hernia.

INDIRECT INGUINAL HERNIA	DIRECT INGUINAL HERNIA
• Can occur in any age (child to adult) • Occurs in a pre-existing sac • Protrusion through the deep ring, herniation occurs later • Pyriform/oval shape, descends obliquely and downwards • Can be complete by descent into scrotum • Neck of sac is narrow and lateral to inferior epigastric artery • Sac is anterolateral to the cord • Ring occlusion test does not show any impulse after deep ring occlusion • Invagination test shows impulse on the tip of the little finger • Zieman's test shows impulse on the index finger • Commonly unilateral but can be bilateral • Obstruction and strangulation are common • Sac should be opened during surgery	• Common in elderly • Always acquired • Herniation through posterior wall of the inguinal canal • Globular/round shape, descends directly forward • Descent down into the scrotum is rare • Neck of the sac is wide and medial to inferior epigastric artery • Sac is posterior to the cord • Test shows impulse even after occluding the deep ring • Invagination test: Impulse is felt over the pulp of the little finger • Zieman's test shows impulse on the middle finger • Commonly bilateral • Obstruction and strangulation are rare but can occur • Sac is not necessarily opened unless obstruction is present

STRANGULATION

- Strangulation of an inguinal hernia develops when constriction occurs at the neck of the sac, cutting off the blood supply of the contents.
- Initially the venous blood supply is obstruction. This causes swelling of the contents (omentum or bowel) and eventually a combination of edema and constriction interrupts the arterial blood supply and gangrene supervenes in the strangulated loop.
- These changes are accompanied by:
  - Severe local pain
  - Irreducibility of hernia
  - Tenderness
- The symptoms of small bowel obstruction are also evident with colicky abdominal pain, nausea and vomiting.
- When the hernia first becomes obstructed it is possible to reduce it by manipulation. It is important however, to ensure that the hernia is not reduced en masse into the abdominal cavity with contents still strangulated by the constricting neck of the sac.
- An irreducible hernia per se does not indicate strangulation and may be incarcerated (obstructed) with an adequate blood supply but completely irreducible.
- Management:
  - Admit patient
  - Ryle's tube aspiration
  - Intravenous fluids to correct dehydration and electrolyte imbalance
  - Antibiotics
  - Catheterisation to maintain adequate urine output
  - Emergency surgery

MANAGEMENT OF INGUINAL HERNIA

- Ideally all inguinal herniae should be repaired by elective surgical operation.
- Surgery is usually performed under general anesthesia but local or regional anesthesia is also used.
- Surgical repair classically consists of 2 elements:
  - Excision of the hernia sac (herniotomy) and repair or buttressing of the weakness in the posterior inguinal canal (herniorrhaphy)

- Note: in direct hernia the sac is not opened and care should be taken at the medial aspect due to presence of the bladder (which should be emptied before surgery

- In infants (always indirect herniae), the internal and external rings are superimposed and only a herniotomy is required for effective treatment.
- Laparoscopic repair of adult inguinal herniae is also performed by surgeons.
- Sometimes a truss may be used to control an inguinal hernia if the patient is unfit for or refuses surgery. However, trusses are unsatisfactory, do not treat the hernia and the patient is still at risk of incarceration (obstruction) and strangulation.
- Hernioplasty is the present choice (ideal) for all inguinal and groin hernias. Mesh is placed either onlay/underlay (over conjoint tendon to inguinal ligament) or inlay (in preperitoneal space).
  - A polypropylene mesh is used to strengthen the posterior wall of the inguinal canal.
  - Herniotomy is done prior to mesh placement

COMPLICATIONS OF HERNIA REPAIR

- Hemorrhage
- Hematoma: may be in the wound or scrotum
- Acute urinary retention: this frequently follows bilateral repair
- Wound infection: this should be rare as hernia repair is a clean operation however, it occurs in 5-8%.
- Hematocele
- Post-herniorrhaphy hydrocele, lymphocele
- Chronic pain: trapping of the Ilioinguinal nerve
- Hyperesthesia over the medial side of inguinal canal due to injury to iliohypogastrc nerve-neuralgia (15%)
- Testicular pain and swelling followed by atrophy usually means that the repair is too tight and testicular artery is compromised. Testicular atrophy will occur when the swelling subsides.
- Recurrence of hernia (5%): if it occurs within 3 years it is called early and late if it occurs after.
  - Preoperative predisposing factors: smoking, chronic cough, constipation, old age, anemia, hypoproteinemia, ascites and straining (BPH, carcinoma prostate, stricture urethra)
  - Operative: tension in the sutures and weak anterior abdominal wall.

➢ Postoperative: infection (50%), hematoma formation during early surgery, retained sac in pantaloons hernia, straining

FEMORAL HERNIA

SURGICAL ANATOMY OF FEMORAL CANAL

- The femoral canal is a the medial most compartment of the femoral sheath, which extends from the femoral ring above to saphenous opening below.
- The femoral canal is bounded anteriorly by the inguinal ligament, posteriorly by the fascia over the pectineus muscle (iliopectineal ligament of Cooper, pubic bone and fascia of the pectineus are all posterior), medially by the lacunar ligament (Gimbernat's ligament) and laterally by the femoral vein.
- The canal is 1.25cm long and 1.25cm wide at the base. Below it is closed by cribriform fascia.
- The canal contains fat, lymphatics and the lymph node of Cloquet.

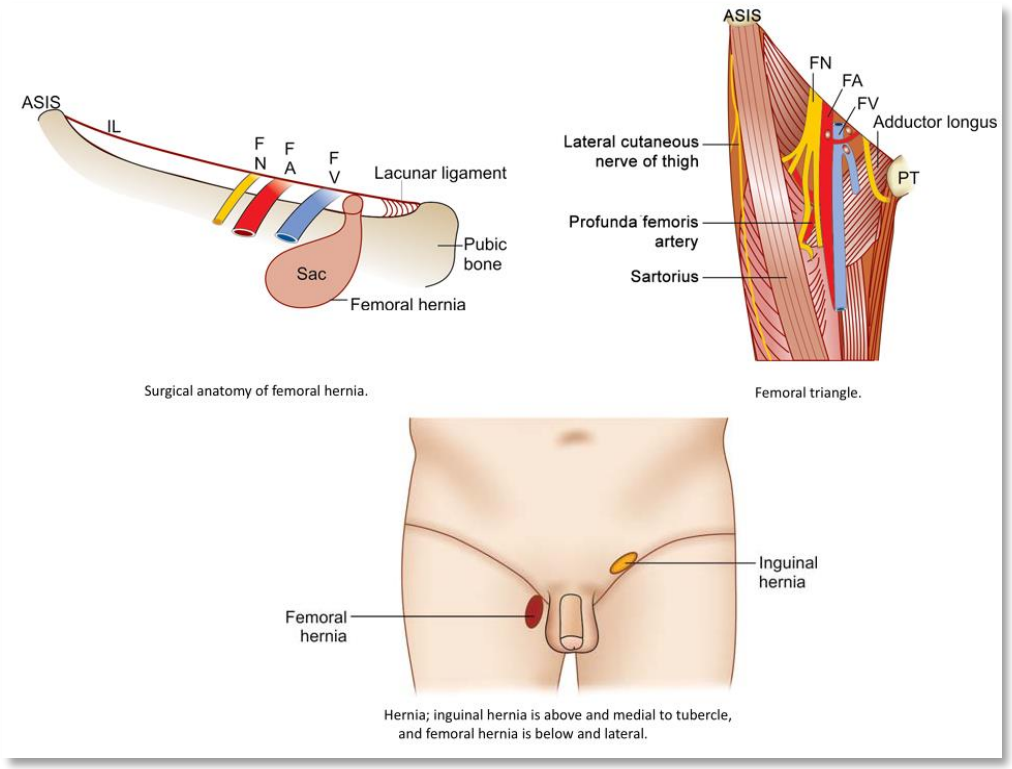


Figure 147: The Femoral canal and femoral hernia (Image adapted from SRB's Manual of Surgery 2nd Ed)

- A Femoral hernia emerges through the femoral canal and may be felt as a soft swelling below and lateral to the pubic tubercle.
- It is a protrusion of peritoneum through the femoral canal, below which it emerges subcutaneously.
  - It protrudes through the femoral canal, the hernial sac descends down vertically up to the saphenous opening and then escapes out into the loose areolar tissue to expand out like a retort.
  - Because of its irregular pathway and narrow neck, it is more prone for obstruction and strangulation. During surgery, precaution should be taken about the femoral vein and pubic branch of obturator artery (or accessory obturator artery) which often may get injured leading to torrential hemorrhage.
- It is usually a small sac and may contain omentum or small bowel. Because of its position below the inguinal ligament it must be distinguished from a saphena varix which disappears on pressure or on lying down and has a cough impulse.
- It must also be distinguished from femoral artery aneurysm, enlarged lymph nodes or on very rare occasions, a psoas abscess.
- Etiology of femoral hernia:
  - Wide femoral canal
  - Multiple pregnancies

CLINICAL FEATURES

- It is common in females (2:1), common in multipara.
- Rare before puberty.
- 20% occur bilateral however more common on right side.
- Presents as:
  - Swelling in the groin that is below and lateral to the pubic tubercle (inguinal hernia is above and medial to the pubic tubercle)
  - Swelling, impulse on coughing, reducibility, gurgling sound during reduction, dragging pain are the usual features.
  - When obstruction and strangulation occur (40%):
    - Painful, tender, inflamed, irreducible swelling without any impulse is seen.
    - Abdominal distension, vomiting and features of toxicity may be present.
- Strangulation and Richter's hernia are common in femoral hernia.

- Often on medial side, a portion of bladder forms the wall of the femoral hernial sac (Sliding-femoral hernia)
- Often femoral hernia can be associated with inguinal hernia also.
- Gaur's sign: in femoral hernia, distension of superficial epigastric and/or circumflex iliac veins occurs due to the pressure by the hernial sac.
- Hydrocele of femoral hernia occurs when adherent omentum which is the content secretes fluid into the sac.
- Herniation through a gap in the lacunar ligament (medial) is always strangulated and is called as Laugier's femoral hernia (L for L)
- In congenital dislocation of hip, femoral hernia occurs behind the femoral vessels (Narath's femoral hernia)
- If sac lies under the pectineal fascia it is called a Cloquet's hernia

DIFFERENTIAL DIAGNOSIS

- Inguinal hernia
- Enlarged Cloquet lymph node of any cause
- Psoas abscess- psoas spasms with flexed hip but difficulty in extension
- Lipoma
- Femoral aneurysm
- Distended psoas bursa (disappears on hip flexion)
- Saphena varix- this is an enlarged terminal portion of the long saphenous vein. It is soft, disappears on lying down, fluid thrill, impulse on coughing and venous hum on auscultation are present. There is associated varicose veins on leg.
- Hematoma in the region.

MANAGEMENT

- Because femoral herniae are most likely to lead to strangulation than inguinal hernia, they should always be repaired without delay.
- A truss should not be used.
- Surgically the hernia may be approached from:
  - Below (femoral approach)
  - Above via the inguinal canal (Lotheissen approach)
  - Rectus abdominis muscle (McEvedy approach)
- The contents of the sac are emptied, the sac is excised and the femoral canal is obliterated with 3 interrupted non-absorbable sutures.
- Polypropylene mesh can be buttressed over the femoral canal to close the defect.

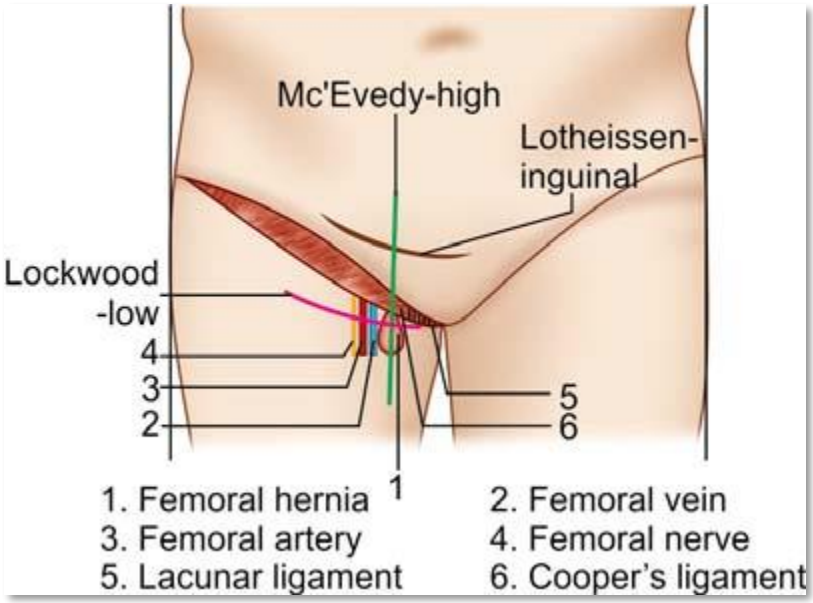


Figure 148: Different surgical approaches for femoral hernia (Image adapted from SRB's Manual of Surgery 2nd Ed)

UMBILICAL HERNIA

UMBILICAL HERNIA

- True umbilical hernia is common in infants (Especially in Africans) and is due to a persistent defect in the abdominal wall at the umbilicus.
- The majority close spontaneously and surgical closure is rarely necessary.
- Intervention is required in the unlikely event of strangulation or incarceration.



Figure 149: Congenital umbilical hernia (Image from Clinical Surgery 2nd Ed)

PARAUMBILICAL HERNIA

- These herniae occur in obese adult women and are prone to strangulate.
- The defect occurs through the midline just above the umbilicus.
- The sac may contain omentum or small intestine or both and because of the narrow neck, strangulation is relatively common.
- With long-standing herniae, adhesions occur between the contents and the wall of the sac so that the hernia becomes irreducible.
- Strangulated herniae are repaired as an emergency.
- Long-standing herniae should be repaired electively. The Mayo repair is commonly used.
  - With this technique the contents of the sac are freed from its wall and reduced.
  - The sac is excised and the fascial defect is repaired transversely with the upper flap overlapping the lower, thereby doubling the strength of the repair.



Figure 150: Paraumbilical hernia (Image from Clinical Surgery 2nd Ed)

EPIGASTRIC HERNIA

- These usually small but often quite painful swellings occur in the midline between xiphisternum and umbilicus.
- The swelling most frequently consists of herniation of Extraperitoneal fat through a small defect in the linea alba.
- Sometimes it carries a peritoneal sac with it that may contain omentum but this is rare.
- Pain is localized to the site with tenderness on pressure but it may also simulate the symptoms of peptic ulcer.

- Clinical examination reveals a tender swelling in the midline. Sometimes incarcerated fat becomes devascularized and necrotic.
- Treatment is surgical and may be carried out under local anesthesia, enlarging the defect, excising the fat and suturing the defect with non-absorbable sutures.

INCISIONAL HERNIA

- This is a hernia that protrudes through a defect in an old abdominal wound.
- It is common in old age and obese individuals.
- It occurs in 10% of abdominal surgeries, 70% occurs in first 5 years and 30% occurs in 5-10 years.
- Types of defects in incisional hernia:
  - Small defect <2cm
  - Large and wide defect >2cm
  - Very large defect
  - Massive/diffuse
  - Multiple defects- Swiss cheese pattern
- Predisposing factors:
  - Vertical incision has got high chances of incisional hernia than horizontal scars.
  - Layered closure of the abdomen has got higher chance than single layer.
  - Continuous closure has got higher chances than interrupted closure
  - Using absorbable suture material has got higher chances of hernia than non-absorbable sutures
  - Emergency surgical wound has higher chances than elective surgical wounds.
  - Laparotomy for peritonitis, acute abdomen and trauma can commonly cause incisional hernia
  - Drainage through the main laparotomy wound may precipitate formation of incisional hernia
  - Chronic cough, smoking, obstructive uropathy, constipation can precipitate incisional hernia
  - Diabetes, old age, malnutrition, malignancy, anemia, hypoproteinemia, jaundice, ascites, liver disease, uremia, steroid therapy, immunosuppressive disease are other precipitating factors.

- The margins of the defect in the abdominal wall under the old incision can be felt and the hernia is easily demonstrated by asking the supine patient to raise his or her head off the pillow (thus tensing the abdominal muscles).
- An incisional hernia often contains bowel that is adherent to the peritoneal sac.
- Clinical features:
  - Swelling in the scar region
  - Pain
  - Impulse on coughing
  - Gurgling sound
  - Often bowel peristalsis may be visible under the skin
  - Eventually features of irreducibility, obstruction, strangulation is seen.
  - Hernia is common in lower abdomen
  - It may be small or large, huge or massive (diffuse)
  - Scar, its extent and location whether healed primarily or secondarily, skin over the scar and swelling is noted. Details of the swelling with expansile impulse on coughing and examination both in lying down and standing are done.
  - Gap cannot be assessed in an irreducible hernia.
- Surgical repair requires excision of the sac and identification and apposition of the margins of the hernia.
- Occasionally, with a very large incisional hernia, it is not possible to bring the muscle edges together and a polypropylene mash (Marlex) has to be inserted to close the abdominal wall defect.
- Complications of incisional hernia:
  - Irreducibility
  - Obstruction
  - Strangulation
  - Incarceration

ORTHOPEDICS

GENERAL PRINCIPLES

GENERAL PRINCIPLES OF FRACTURES

- A fracture (sometimes abbreviated FRX or Fx or F_x or #) is a partial or complete break in the structural continuity of a bone which may or may not be involved with soft tissue injury.
- If the overlying skin remains intact it is a closed (or simple) fracture.
- If the skin or one of the body cavities is breached it is an open (or compound) fracture, liable to contamination and infection.
- Bone is the only tissue in the human body other than liver that heals by regeneration instead of by scarring.
- For regeneration to occur the bone must be immobilized to allow uninterrupted formation of new bone.

CLASSIFICATION

- Classification aims to have a standard interpretation of a fracture for research and clinical management, tell you the severity of a fracture and how it could have been caused as well as better management.
- Fractures can be classified according to:
  - Clinical classification
  - Anatomical site
  - Etiology (mechanism of injury)
  - Radiological appearance (Radiological pattern of fracture)
  - Eponyms

CLINICAL CLASSIFICATION

- Fractures are either classified as:

- Open fractures (Compound fractures)
- Closed fractures (simple fractures)

OPEN FRACTURES (COMPOUND FRACTURES)

- There is a wound on the skin surface leading to the site of the fracture i.e. a direct communication exists between the body surface and the fracture.
- Open fractures are liable to contamination by organisms and are thus true orthopedic emergencies.
- The amount of communication can vary from a small puncture wound in the skin to a large avulsion of soft tissue that leaves the bone exposed.
- Prognosis depends on extent of soft tissue injury and by type/level of bacterial contamination.
- Treatment plan: prevent infection (Broad spectrum antibiotics and Tetanus prophylaxis), restore soft tissues (Wound cleaning and debridement), achieve bone union (reduction, immobilization), avoid malunion and institute early joint motion and muscle rehabilitation.

CLOSED FRACTURES (SIMPLE FRACTURES)

- There is no communication between the site of the fracture and the exterior of the body.



Figure 151: Open fracture (Left) vs Closed Fracture (Right)

ANATOMICAL SITE

- Fractures are classified by their anatomical location in relation to a specific bone identifying a fracture by location does not indicate whether the fracture is open or closed nor does it indicate the type of fracture (transverse, oblique, spiral etc.).
- Fractures may affect the proximal, middle or distal end of a bone.

ETIOLOGY (MECHANISM OF INJURY)

- According to etiology fractures are divided into 3 groups:

- Traumatic fractures
- Stress/Fatigue fractures
- Pathological fractures

TRAUMATIC FRACTURES

- These are the most common types. They occur in bones previously free of disease.
- Fracture caused by sudden and excessive force which may be direct or indirect.
- They can either be due to:
  - Direct force: the bone breaks at the point of impact; the soft tissues also are damaged. A direct blow usually splits the bone transversely or may bend it over a fulcrum so as to create a break with a 'butterfly' fragment. Damage to the overlying skin is common, if crushing occurs the fracture pattern will be comminuted with extensive soft-tissue damage.
  - Indirect force: bone breaks at a distance from where the force is applied, soft tissue damage at fracture site is not inevitable. Most fractures due to a combination of forces (twisting, bending, compressing or tension), the x-ray patterns reveal:
    - Twisting: causes a spiral fracture- (A)
    - Compression causes a short oblique fracture- (B)
    - Bending results in fracture with a triangular 'butterfly' fragment- (C)
    - Tension tends to break the bone transversely- (D); in some situations, it may simply avulse a small fragment of bone at the points of ligament or tendon insertion.

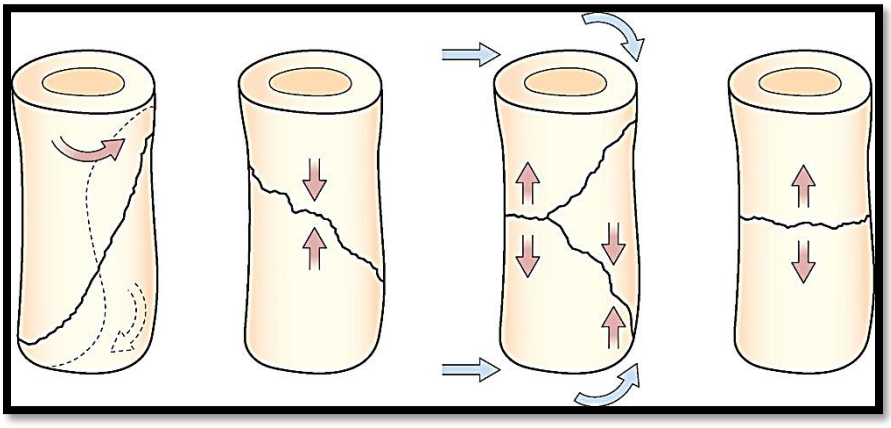


Figure 152: From left to right, (A) Spiral fracture due to twisting (B) Short Oblique fracture due to compression (C) Triangular butterfly fragment due to bending (D) Transverse fracture due to tension

- NOTE: the above description applies mainly to the long bones. A cancellous bone, such as a vertebra or the calcaneum when subjected to sufficient force will split or be crushed into an abnormal shape.

STRESS/FATIGUE FRACTURE

- These occur in normal bone which is subject to repeated heavy loading typically in athletes, dancer or military personnel who have grueling exercise programs.
- These high loads create minute deformations that initiate the normal process of remodeling- a combination of bone resorption and new bone formation in accordance with Wolff's law.
- When exposure to stress and deformation is repeated and prolonged, resorption occurs faster than replacement and leaves the area liable to fracture. A similar problem is seen in people on drugs that alter normal balance of bone resorption and replacement. Stress fractures are increasingly seen in patient with chronic inflammatory diseases who are on treatment with steroids or methotrexate.
- These types of fractured are confined to bones of lower limb: metatarsal bone (>50%), calcaneus (25%), shaft of fibula and tibia (20%), neck of femur.
- Onset of pain in the affected bone is gradual, pain increased by continuous activity and relieved by rest.
- O/E: marked tenderness over affected bone and swelling.
- Radiographic changes occur only 2-4 weeks and may show only a faint hairline. Rarely is there displacement of fragments.
- Treatment: decreased physical activity

PATHOLOGICAL FRACTURE

- These fractures occur even with normal stresses because the bone has been weakened by a change in its structure (e.g. in osteoporosis, osteogenesis imperfect or Paget's disease) or through a lytic lesion (e.g. a bone cyst or a metastasis)

- Some common tumors which metastasize to bone are thyroid cancer, breast cancer, prostate cancer, renal cancer.
- Primary bone tumors causing fractures: Giant cell bone tumor, Ewing's tumor
- Causes: local or generalized
  - Infections: pyogenic osteomyelitis
  - Benign tumors: chrondroma, giant cell tumor
  - Malignant tumors: osteosarcoma, Ewing's, metastatic carcinoma especially the above-mentioned tumors that metastasize to bone, multiple myeloma, diffuse metastatic carcinoma.
  - Miscellaneous: simple bone cyst, eosinophilic granuloma, tabes dorsalis, osteonecrosis after irradiation, paget's disease, gaucher's disease.
  - Congenital disorders: osteogenesis imperfect
  - Diffuse rarefaction of the bone: senile osteoporosis, parathyroid osteodystrophy, infantile rickets, Cushing's syndrome, nutritional osteomalacia.

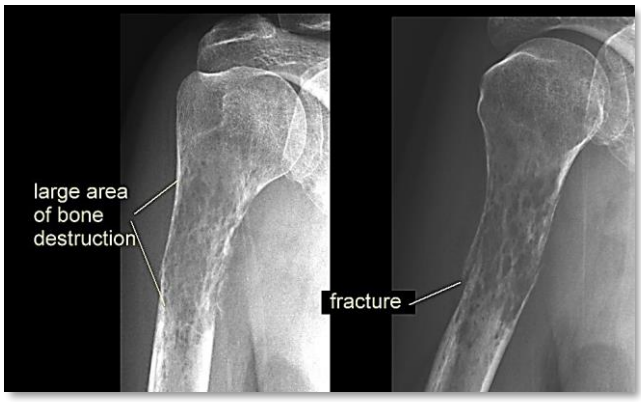


Figure 153: Osteolytic bone



Figure 154: Osteolytic bone

- Osteoporosis is the most common pathologic condition association with pathologic fractures.
- When treating the broken bone, the underlying condition must be treated as well.

RADIOLOGICAL PATTERN

- This classification is based on the X-ray view of the fracture.
- It is the description of shape and pattern as seen on radiographs.
- Pattern indicates:
  - Nature of causative violence

- Clue to easiest method of reduction
- The likely stability of the fragments

- Common patterns could be:
  - Transverse
  - Oblique
  - Spiral
  - Comminuted (more than 2 fragments)
  - Compression or crush
  - Impacted
  - Greenstick (incomplete break occurring in resilient bones in children)
  - Burst
  - Avulsion
- These patterns fall into 2 well defined groups:
  - Complete fractures: Fracture split into two or more fragments. X-ray can help predict behavior after reduction.
    - Transverse fractures: fragments usually remain in place after reduction.
    - Oblique fractures: tend to shorten and re-displace even if the bone is splinted
    - Spiral fractures: tend to shorten and re-displace even if the bone is splinted
    - Impacted fractures: Fragments are jammed tightly together and the fracture line is indistinct
    - Comminuted fractures: more than two fragments are present, because there is poor interlocking of the fracture surfaces, these are often unstable.

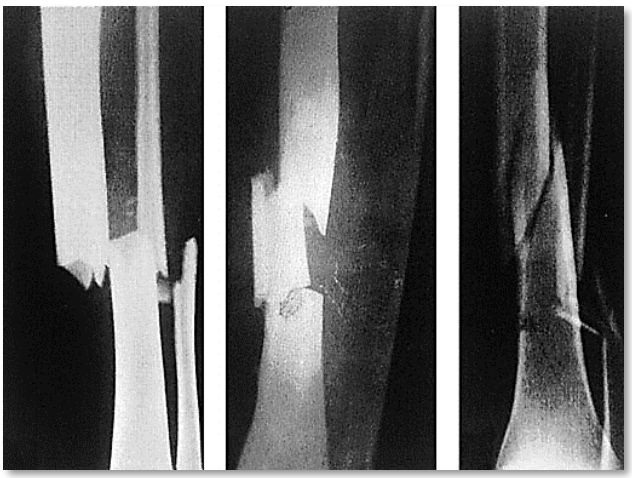


Figure 155: From left to right (A) Transverse (B) Segmental (C) Spiral

  - Incomplete fractures: Bone is incompletely divided and the periosteum remains in continuity

- Greenstick fracture: bone is buckled or bent (like snapping a green twig), this is seen in children, whose bones are springier than those of adults. Children can also sustain injuries where the bone is plastically deformed (misshapen) without there being any crack visible on X-ray.
- Compression fracture: Occur when cancellous bone is crumpled. This happens in adults and typically where this type of bone structure is present e.g. in the vertebral bodies, calcaneum and tibial plateau.



Figure 156: Greenstick fracture, note the Buckle or torus

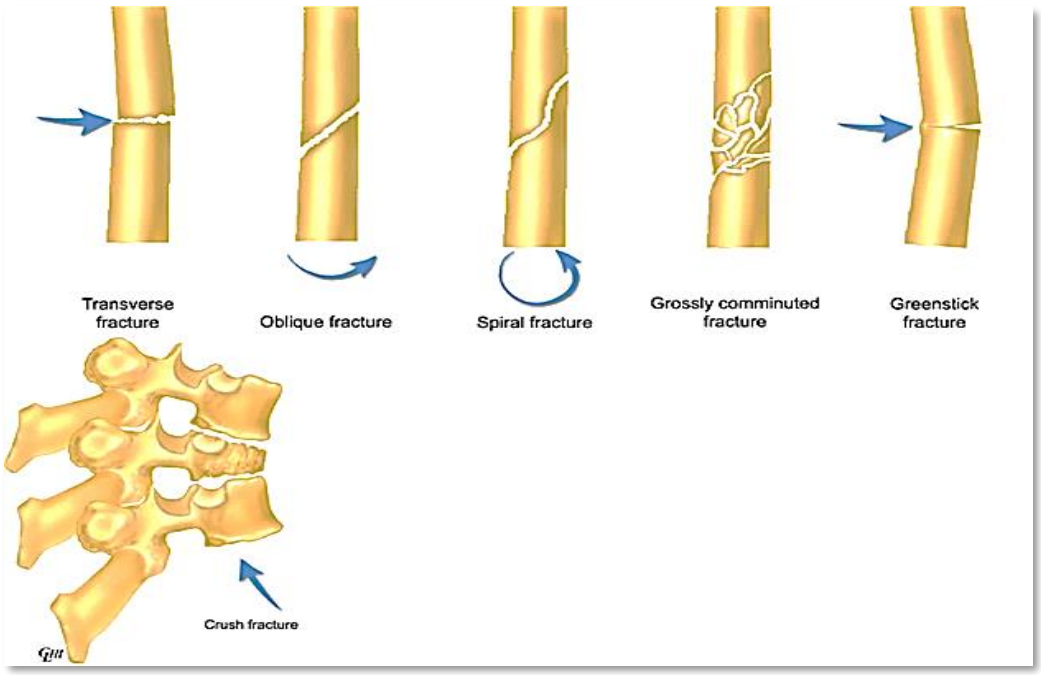


Figure 157: Fracture patterns

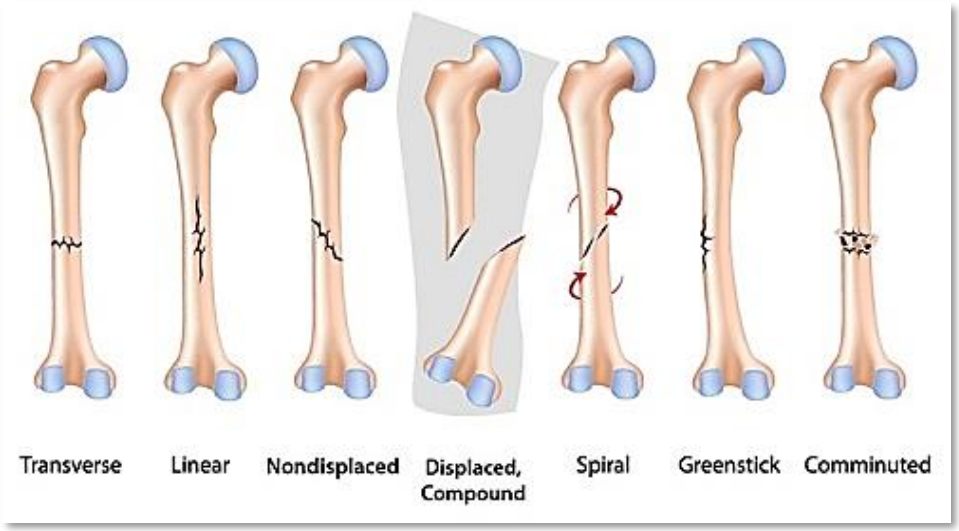


Figure 158: Fracture patterns

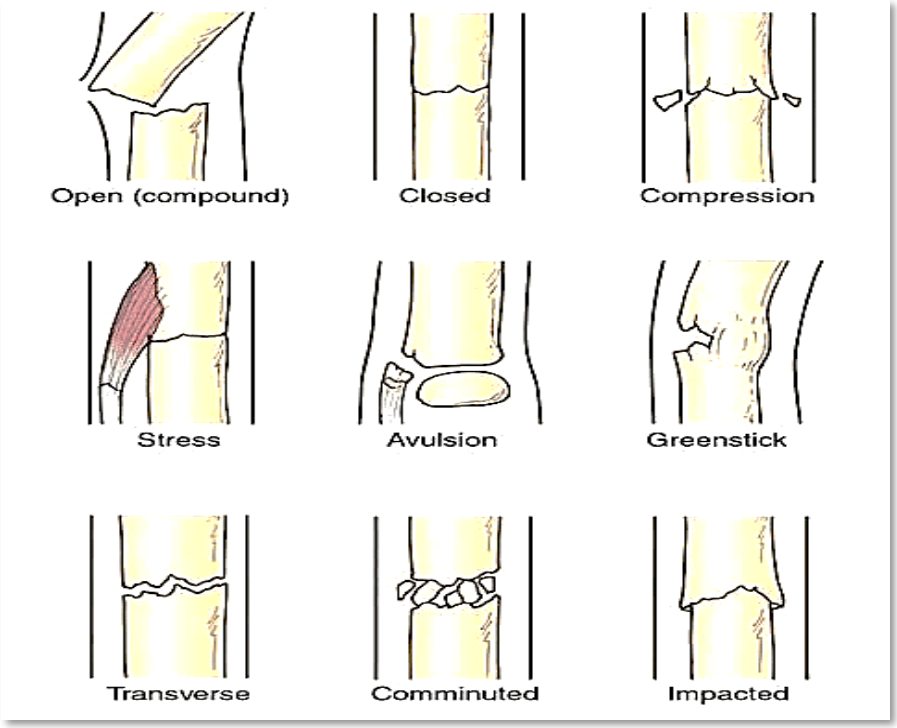


Figure 159: Fracture patterns

EPONYMS

- Traditional classifications often bear the originator's name.
- This type of classification can only be used to specific type of injuries however, even then the term is often inaccurately applied, famously in the case of Pott's fracture, which is often applied to any fracture around the ankle though that is not what sir Percival Pott implied when he described the injury in 1765.
- These are based on the people that first described the fractures:
  - Gastilo & Anderson- open tibial fracture
  - Garden's classification for fracture neck of femur
  - Neer classification of proximal humeral fractures
  - Hansen classification for ankle fracture
  - Muller AO classification of fractures: this is an alphanumeric classification of fractures developed by Muller and colleagues

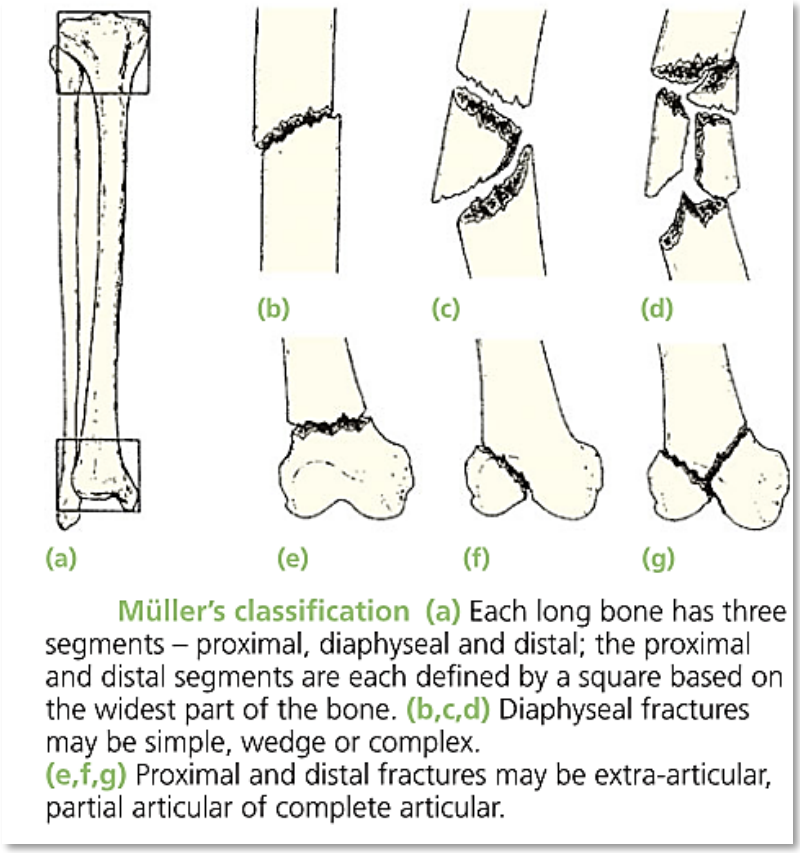


Figure 160: Muller's classification

DISPLACEMENT

- After a complete fracture the fragments usually become displaced, partly by the force of the injury, partly by gravity and partly by the pull of muscles attached to them.
- Displacement is usually described in terms of translation (shift), alignment, rotation and altered length.

TRANSLATION (SHIFT)

- The fragments may be shifted sideways, backward or forward in relation to each other such that the fracture surfaces lose contact.
- The fracture will usually unite as long as sufficient contact between surfaces is achieved, this may occur even if reduction is imperfect or indeed even if the fracture ends are off-ended but the bone segments come to lie side by side.

ANGULATION (TILT)

- The fragments may be tilted or angulated in relation to each other. Mal-alignment, if uncorrected may lead to deformity of the limb.

ROTATION (TWIST)

- One of the fragments may be twisted on its longitudinal axis, the bone looks straight but the limb ends up with a rotational deformity.

LENGTH

- The fragments may be distracted and separated, they may overlap, due to muscle spasm, causing shortening of the bone.

- Types of displacement include: translation (shift), Angulation (tilt), Rotation, impaction and overlap.
- NOTE: all types can be corrected with closed manipulation except rotational displacement (must be surgically corrected as it can lead to malunion and deformities).

DESCRIBING FRACTURES

- When describing, the description must include:
  - Clinical classification: is the fracture open or closed? (only comment on this when there is a patient)
  - Anatomical classification:
    - Which bone is broken?
    - Has it involved a joint surface?
  - Radiological pattern: Only comment on this if there is an X-ray.
    - Transverse: slow to join because the area of contact is small, if the broken surface is accurately apposed, however the fracture is stable on compression.
    - Spiral: joints more rapidly (because contact area is large) but is not stable on compression.
    - Comminuted: slow to join because (1) they are associated with more severe soft tissue damage and (2) they are likely to be unstable.
  - Displacement: assess
    - Shift or translation- backwards, forwards, sideways, or longitudinally with impaction or overlap.
    - Tilt or angulation- sideways, backwards or forwards.
    - Twist or rotation- in any direction.
- Example: Closed, transverse fracture of the shaft of the radius with no displacement.
- A problem often arises in the description of angulation. 'Anterior angulation' could mean that the apex of the angle points anteriorly or that the distal fragment is tilted anteriorly: in this text it is always the latter meaning that is intended ('anterior tilt of the distal fragment' is probably clearer).

CLINICAL ASSESSMENT OF A FRACTURE

- Involves:
  - History
  - General examination (general signs)
  - Local examination

HISTORY

- History of injury, followed by inability to use the injured limb- but beware! The fracture is not always at the site of the injury e.g. a blow to the knee may fracture the patella, femoral condyles, shaft of the femur or even acetabulum.
  - What happened? Was there a lot of violence involved or a simple fall? (get both direct history from victim and collateral history from witnesses)
  - How did it happen? Can likely injuries be predicted from the patient's description of the incident?
  - Where and when did it happen? Did it happen recently or has the patient come late?
  - What was the patient like before it happened? Does the patient have any other medical problems?
  - Also ask about patient's last meal (in case they may need to go to theater)
- The patient's age and mechanism of injury are important, if a fracture occurs with trivial trauma suspect a pathological lesion.
- Pain, bruising, swelling are common symptoms but they do not distinguish a fracture from a soft-tissue injury.
- Deformity is much more suggestive.
- Always enquire about Symptoms of associated injuries: pain and swelling elsewhere (it is a common mistake to get distracted by the main injury, particularly if it is severe), numbness or loss of movement, skin pallor or cyanosis, blood in urine, abdominal pain, difficulty with breathing or transient loss of consciousness.
- Previous injuries: once the acute emergency has been dealt with, ask about previous injuries or any other musculoskeletal abnormality that might cause confusion when the X-ray is seen.
- General medical history: important in preparation for anesthesia or operation.

- Note: Due to the medicolegal implications associated with accidents, fractures and injuries it is important to keep meticulous notes at the time or as soon after the event as possible.

GENERAL EXAMINATION

- Unless it is obvious from the history that the patient has sustained a localized and fairly modest injury, priority must be given to dealing with the general effects of trauma:
  - Follow the ABCs: Airway obstruction, Breathing problems, Circulatory problems and Cervical spine injury (neck collar).
  - Ask a simple question such as 'What is your name?' if you get a sensible answer then the patient is conscious with a good airway. However, for most patients with a single or few injuries, the examination will concentrate on the injured part and the fitness of the patient for anesthetics.
- During the secondary survey it will also be necessary to exclude other previously unsuspected injuries and to be alert to any possible predisposing cause (such as Paget's disease or a metastasis).
- General signs (a broken bone is part of a patient). Look for evidence of:
  - Shock or hemorrhage
  - Associated damage to brain, spinal cord or viscera
  - Predisposing causes
- Local signs:
  - Injured tissues must be handled gently. To elicit crepitus (grating or grinding of the broken bone ends) or abnormal movement is unnecessarily painful, X-ray diagnosis is more reliable.
  - Examine the most obviously injured part.
  - Test for artery and nerve damage.
  - Look for associated injuries in the region.
  - Look for associated injuries in distant parts.

INSPECTION (LOOK)

- Look for the following signs:
  - Swelling, bruising, deformity
  - Discoloration and loss of function
  - Deformity; measure and record any:
    - Angulation
    - Rotation
    - Translation

- Shortening
- These may be better assessed on X-ray

➢ Any compound wounds (i.e. any wounds that reach to the site of the fracture) These should be assessed accurately and measured and recorded using the Gustillo classification (of an open tibial fracture):

- Grade 1:
  - Wounds <1cm,
  - Simple fracture pattern,
  - No skin crushing, clean wounds (mild contamination)
- Grade 2:
  - Wounds measuring 1-10cm but without significant soft tissue crushing, including no flaps, degloving or contusion.
  - # pattern may be more complex.
  - Moderate contamination
- Grade 3:
  - Wounds >10cm, also includes injuries older than 8 hours,
  - Severe contamination plus:
  - A: no periosteal stripping
  - B: Periosteal stripping
  - C: neurovascular damage
  - Tip: to remember A, B and C the mnemonic "AA"-Apparent apposition (i.e. no periosteal stripping), "BB"- bone bare (periosteal stripping), "CC"- Circulatory compromise (i.e. neurovascular damage)

➢ Note also the posture of the distal extremity and the color of the skin (for tell-tale signs of nerve or vessel damage).

PALPATION (FEEL)

- Bony tenderness
- Warmth
- Distal pulses
- Move joints for crepitus (a scratching sound made by a broken bone or rough joint)
- Soft tissue damage, this is often graded as follows:
  - ➢ Grade 0: little or no soft tissue injury
  - ➢ Grade 1: superficial abrasion with moderate swelling and bruising
  - ➢ Grade 2: deep abrasion, tense swelling with bruising and blistering

- Grade 3: extensive contusion, tense swelling, compartment syndrome and/or vascular damage

MOVEMENT (MOVE)

- Abnormal/painful movement: do not cause the patient extra discomfort just to confirm abnormal movement. Furthermore, such abnormal movement may cause further soft tissue damage and further periosteal stripping.
- Simply do an X-ray.

INVESTIGATIONS

IMAGING

X-RAYS

- Follow the "rules of twos"
  - TWO views: usually an anterior/posterior view (AP) and a lateral view. This gives a better view of a fracture or dislocation. For some injuries e.g. the hand oblique views are better.
  - TWO joints: include the joint above and the joint below the bone under consideration. In the forearm or leg, one bone may be fracture and angulated. Angulation however is impossible unless the other bone is also broken or a joint dislocated.
  - TWO sides: useful for comparison, particularly in children because it allows comparison of the epiphyseal lines in immature bones and distinguishes them from the fracture line.
  - TWO injuries: Severe force often causes injuries at more than one level. Thus with fractures of the calcaneum or femur it is also important to also X-ray the pelvis and spine.
  - TWO occasions: repeat X-ray 10-14 days (1-2 weeks) later
- All x-rays should be centered on the area of maximal tenderness.
- Radiographic features of a fracture:
  - Lucencies at the site of the fracture
  - Discontinuity in the cortex or surface of a bone or joint.
- MRI can be used to view extent of soft tissue injury.

ULTRASOUND

- May help detect joint effusions.

RADIOISOTOPE BONE SCAN

- When there is doubt, radioisotope bone scanning can help determine whether a bone is fractured. This is most useful about 2 weeks after the injury. It is a highly sensitive test but does not provide any information about the fracture except that it is there.
- A useful example is the scaphoid fracture, which may be seen on a second X-ray at 2 weeks but there can still be doubt. A negative scan is very positive reassurance in such a situation and may help to allay lingering clinical doubt.

BLOODS

- Full blood count: For Hemoglobin, hematocrit and detection of infection
- Urea, Electrolytes and creatinine: may point towards dehydration and renal failure secondary i.e. during hemorrhagic shock
- Liver function tests

HEALING OF FRACTURE

- Healing begins as soon as the bone is broken, provided the conditions are favorable and proceeds through several stages.
- The repair of tubular bone has been classified into 5 stages for simplicity:
  - Stage of inflammation and hematoma formation: Tissue damage and bleeding. (immediate response)
  - Stage of cellular proliferation (subperiosteal and endosteal cells): Inflammatory cells appear.
  - Status of callus formation: Osteoblasts and osteoclasts appear. Takes 2-4 weeks
  - Stage of consolidation: Woven bone replaced by lamellar bone and fracture united.
  - Remodeling: New formed bone remodeled to resemble normal structure.

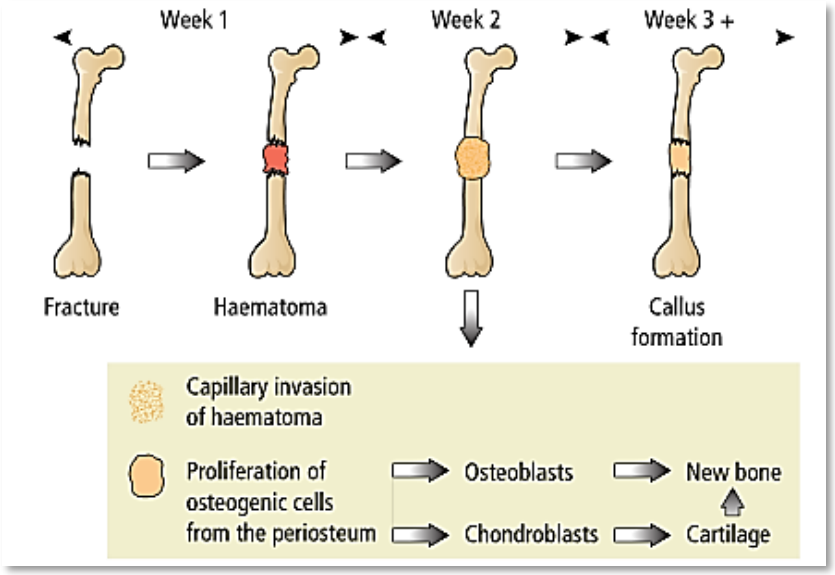


Figure 161: Stages of Fracture healing



Figure 162: Stages of wound healing

MANAGEMENT OF FRACTURE

- Since bones are dynamic living tissues with a very rich blood and nerve supply. The immediate consequences of fractures therefore are severe pain and blood loss and the strategy of early management is to minimize pain with splints and analgesics and to anticipate the effects of blood loss i.e. hypovolemic shock.
- General treatment is the first consideration: treat the patient, not only the fracture.
  - First Aid- ABCs
  - Resuscitation of patient and treatment of shock
  - Pain relief
  - Treatment of the fracture
- When fractures occur with other injuries save life first (resuscitate! Primary and secondary survey), Save the limb, save the joint and then restore function.
- All fractures unite: somehow, whenever. Be aware of this difference you create make use of it rationally (P. Demmer)
- Bone is the only tissue in the human body other than liver that heals by regeneration instead of by scarring.
- For regeneration to occur the bone must be immobilized to allow uninterrupted formation of new bone.

IMMEDIATE MANAGEMENT

- Follow the ABCs: Airway obstruction, Breathing problems, Circulatory problems and Cervical spine injury (neck collar).
- Ask a simple question such as 'What is your name?' if you get a sensible answer then the patient is conscious with a good airway. However, for most patients with a single or few injuries, the examination will concentrate on the injured part and the fitness of the patient for anesthetics.

RESUSCITATION AND BLOOD LOSS

- For most upper limb and peripheral lower limb fractures blood loss is small and is tolerated even by the elderly.
- Blood loss may be significant with major pelvic fractures (6+ units) and long bone fractures particularly the femur (2-3 units) and the tibia (1 unit).
- In general, all patient with long bone injuries should be cross-matched for blood.
- Gain venous access with 2 large bore cannulae.
- Start running fluids (normal saline) as you wait for blood (See chapter on shock).
- Monitor vital signs.

PAIN RELIEF

- Systemic pain relief with opiates given in adequate doses in combination with an antiemetic to offset the adverse effects opiates. In injured patients, intravenous rather than intramuscular opiates should be used. Clinicians, particularly when new are often afraid to give adequate doses of opiates in fear of affecting consciousness and clinical signs. In general, however, when serious head or visceral injury has been excluded, they may be used generously and early.
- Local anesthetic nerve blocks: are frequently effective. Femoral nerve block for example can remove the need for systemic drugs following femoral fracture although the anxiolytic benefits of CNS agents should not be underestimated.
- Splintage: can be applied to most fractures before arrival to hospital. This alone can relieve most intolerable pain. In general, a splint should encompass the joint above and below the injury. Simple expedients such as binding the arm to the chest, with or without a sling, or simply binding the legs together is often sufficient.

TREATMENT OF THE FRACTURE

- Debridement is required for open fractures as they are surgical emergencies. Formal surgical wound debridement (removal of all dead tissue) should be performed as soon as possible and preferably within 6 hours of injury.
- Wounds must be left open and closed either as a secondary procedure after a few days or left to heal spontaneously. Such patients all need supplementary broad-spectrum antibiotics and some form of tetanus prophylaxis i.e. tetanus toxoid booster to those with a previous immunization record or human antitetanus globulin for those with no previous active immunity.
- Treatment of the fracture consists of: Reduction, immobilization and rehabilitation.

REDUCTION

- This is restoration of the displaced fragments to their anatomical positions.

- It aims at restoring the anatomy:
  - Correct axial alignment
  - Correct rotational deformity
  - Restore length
  - Joint alignment
- Methods include: (1) closed manipulation (2) open reduction (3) traction
  - Closed manipulation: is not necessary when there is no displacement, displacement does not matter or reduction is unlikely to succeed. It may be done under general anesthesia (manipulation under anesthesia-MUA)
  - Open reduction: often accompanied by internal fixation (ORIF)
  - Traction (discussed later)

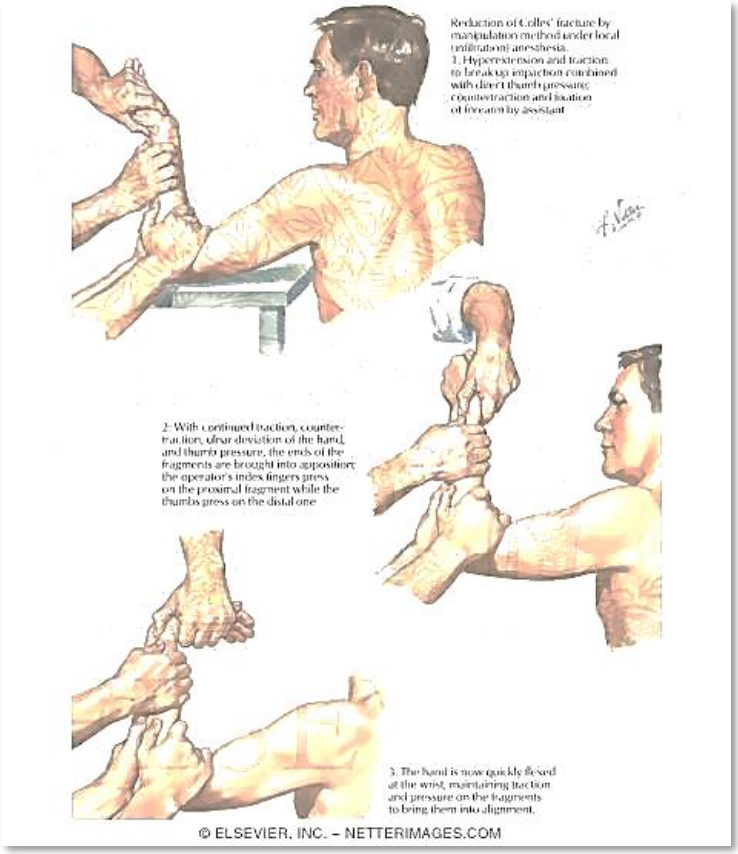


Figure 163: Closed reduction

IMMOBILIZATION

- After reduction (whether closed or open) next is to hold (immobilize)
- Immobilization (or Stabilization) keeps the bony fragments in their position until union occurs.
- Fixation can be open or closed
- Methods of immobilization include: (1) Casting and Splintage (2) functional bracing (3) Internal fixation (4) External fixation

CASTING AND SPLINTAGE

- After reduction the fracture can be held in place be a plaster of Paris cast until union occur.
- The fracture must be held in a position that maintains it in 3 dimensions (tilt, twist and



Figure 164: Plaster of Paris used to treat a forearm mid-shaft fracture of the radius and ulnar.

shift) and the cast must be the proper length.

- In order to ensure complete control of all dimensions of the fracture, it is necessary to control the joint above and below the fracture otherwise joint movement may result in distortion in one or more dimensions.
- Plaster of Paris is relatively brittle, messy and very difficult to apply well. It is heavy and awkward, particularly in the elderly and takes up to 3 days to dry fully. For these reasons, strong and light glass fiber and polyurethane resin material have been developed.
- These are not as versatile as plaster of Paris and are better for secondary casts, which are applied after a week or two when the fracture swelling has settled.

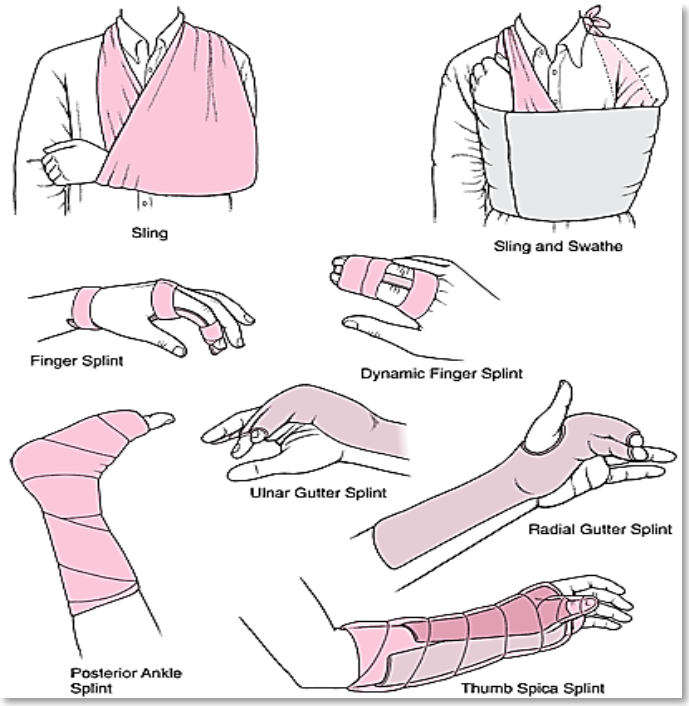


Figure 165: Splintage

- Splintage can also be done to hold them together until they unite until they unite, meanwhile joint movement and function must be preserved.

FUNCTIONAL BRACING

- Casts have many disadvantages such as:
  - They are heavy
  - They immobilize the joint and prevent access to the fracture. The immobility can result in muscle wasting and joint stiffness.
- Functional bracing overcomes the above disadvantages i.e. there is freeing of the joints while maintaining alignment at the fracture site.
- Functional bracing is highly dependent on a very accurate fit and can be used only after a few weeks when the pain and swelling have settled.
- In practice, functional bracing is used in the management of fractures of the tibia and fibula.

INTERNAL FIXATION

- Internal fixation is technically very demanding and has many complications and most important of all, prevents natural healing. If fixation is to be used it can be achieved by a number of ways:

- Apposition: once fractures are realigned, they may only be held in apposition for healing to proceed satisfactorily. This is particularly true in children. Apposition is done by using semi-flexible wires known as 'K' (for Kirschner) wire. They hold position within producing immobility and so healing occurs by natural callus formation. They can be left standing proud of the bone and can easily be pulled out once union is established and before consolidation.

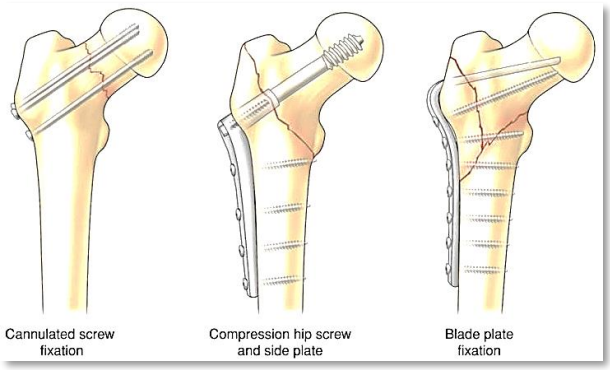


Figure 166: Internal fixation

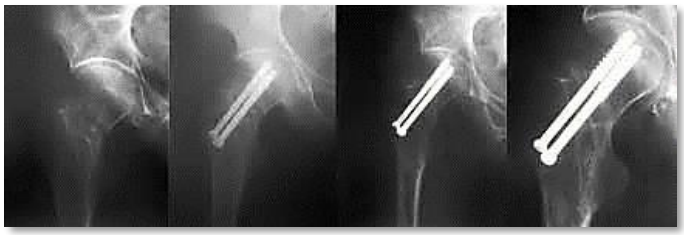


Figure 167: Internal fixation

- Interfragmentary compression: usually achieved by screws or sometimes by tension band wires. These achieve great accuracy and are particularly valuable in cancellous bone around joints. They are also useful in long bones, particularly in the upper limb but in these situations extra support is required from an onlay device.
- Onlay device: are metal plates that are used to buttress weak structure around joints and to fix long bones in the upper limb. These very rigid systems inhibit natural bone union and although they permit early movement, they ultimately delay healing and full load-bearing.
- Intramedullary or inlay devices: are the most satisfactory method of fixation. They achieve alignment without unduly disturbing natural bone healing. They are a relatively inaccurate method of restoring anatomical position and so are not useful around joints. Their great strength makes them ideal devices for treating long-bone fractures particularly in the lower limb.

EXTERNAL FIXATION

- When fractures are open and associated with extensive soft-tissue damage and contamination, neither plaster casting nor internal fixation is appropriate.
- Plaster splints are unsuitable because the wounds become inaccessible for inspection and dressing while internal fixation is hazardous because of the very high risk of wound infection.

- A compromise solution is to apply an external fixation device which consists of a strong metal rod (or series of rods) that runs parallel to the fractured bone and is attached to the bone by a series of pins.
- Such a device stabilizes the fracture and gives access to the soft tissues for dressings and secondary surgery such as skin grafting. A disadvantage of external fixators is the risk of infection at the pin sites.



Figure 168: External fixation

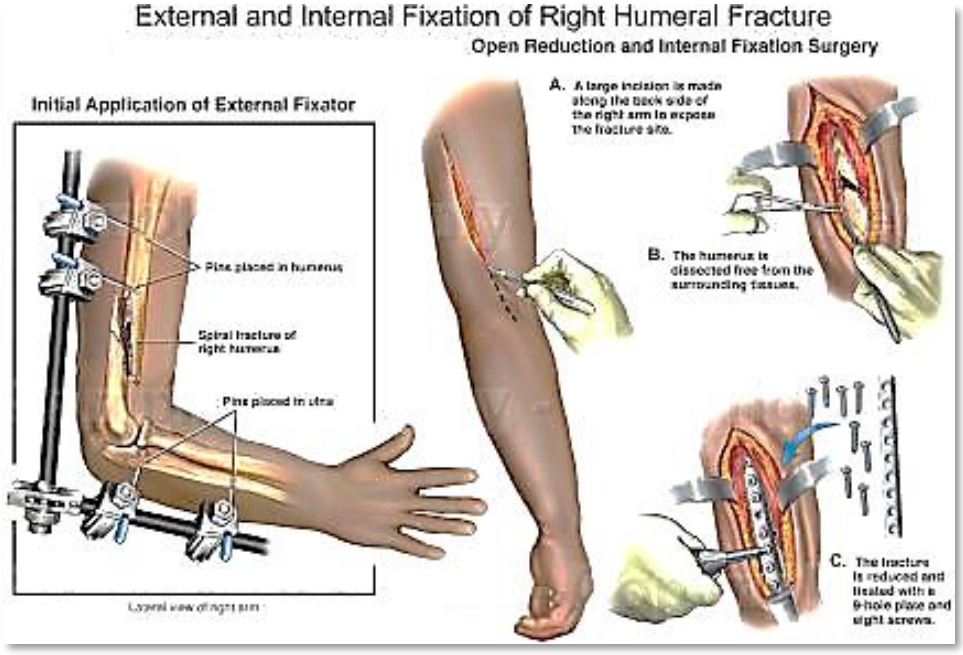


Figure 169: External and internal fixation of right humeral fracture

TRACTION

- Done using weights and can either be skin or skeletal. (Details discussed in next chapter)

REHABILITATION

- Rehabilitation: should start as soon as possible after injury, it aims at maintaining functions of the uninjured parts, and once fracture is united restoring functions of the injured parts.
- Physiological loading of the bone, so muscle activity and early weight-bearing are encouraged.
- Rehabilitation by exercise:
  - Prevents edema
  - Prevents joint stiffness
  - Prevents muscle wasting and atrophy
  - Prevent DVT
  - Enhance fracture healing
  - Prevent contractures

Treatment of fractures at a glance

- Treat the pain and exclude the risk of serious blood loss. Most low-energy fractures are not associated with blood loss, but pelvic and long-bone fractures are, so calculate the potential blood loss
- Reduce the fracture: do this as simply and safely as possible. Restore function
- Hold the fracture in a functional position. This may mean using:
  (a) A cast
  (b) Doing an operation and inserting fixation devices such as nails, plates or screws
  (c) Applying external fixation where there is extensive soft-tissue damage
  (d) Using weights to apply gentle traction
- Rehabilitate the patient: work with the rest of the team of healthcare professionals including nurses, physiotherapists and occupational therapists

COMPLICATIONS

- Early complications may be either due to direct consequence of injury or in association with the treatment.
- Late complications are generally related to the fracture but a few are unfortunately precipitated by treatment (or lack of it).

EARLY COMPLICATIONS

- Hemorrhage- may lead to shock. Internal or external e.g. # femur-2L, Tibia-1/2L, Pelvis 3-5L
- Hematoma
- Nerve or vascular injury
- Soft tissue injury e.g. visceral organs, urethra etc.
- Tendon injury

INTERMEDIATE

- Infection
- Fat embolism
- Osteomyelitis
- Septic wound/ septicemia in compound #s
- Avascular necrosis
- Compartment syndrome caused by localized swelling
- Joint stiffness
- Volkmann ischemic contractures
- Thromboembolism

LATE

- Malunion (may lead to late neuropathies e.g. valgus deformity at elbow-an ulcer): due to poor supervision of healing
- Non-union: usually associated with
  - Excess movement
  - Insufficient movement
  - Poor local blood supply due to either anatomy (e.g. tibia) or a lot of trauma damage
- Delayed union
- Growth arrest in children due to epiphyseal damage
- Osteoarthrosis- common and particular #s especially displaced
- Joint instability/deformity

- Post-traumatic atrophy
- Osteochiorosis-harden brittle
- Osteoarthritis
- Shortening of bone
- Intra-articular and peri-articular adhesion
- Hypostatic pneumonia
- DVT
- Pressure sores

CAUSES OF NON-UNION

LOCAL

- Local infection (sepsis)
- Interposition of soft tissue
- Inadequate or poor local blood supply e.g. # neck of femur (ischemia)
- Inadequate immobilization
- Bone loss or crushing
- Over traction- loss of apposition in between
- Iatrogenic- wrong open reduction and internal fixation
- Local malignancy- bone destruction
- Severe communication
- Extensive opening
- Dissolution of # hematoma by synovial fluid bathing the joint

SYSTEMIC

- Anemia
- Poor general health
- Mineral and vitamin deficiency especially calcium and vitamin D
- Metabolic disease e.g. uncontrolled DM
- Hyperparathyroidism acidosis help destroy the bone
- Lack of androgen, estrogen hormones

TRACTION

- Traction is applied to the limb distal to the fracture so as to exert a continuous pull in the long axis of the bone, with a counterforce in the opposite direction (to prevent the patient being merely dragged along the bed).
- Traction is safe enough, provided it is not excessive and care is taken when inserting the traction pin.
- Traction includes:
  - Skin traction
  - Skeletal traction

SKIN TRACTION

- Skin traction will sustain a pull of no more than 4 or 5kg (has a limit).
- Holland strapping or one-way stretch Elastoplast is stuck to the shaved skin and held on with a bandage.
- The malleoli are protected by Gamgee tissue and cords or tapes are used for traction.
- It is used for individuals between ages of 3 and 18 years.
- Indications include:
  - Fractures:
    - Shaft of femur (main indication in children)
    - Other indications in adults include:
      - Intertrochanteric fractures
      - Fracture of pelvis
      - Unstable Hip dislocation
      - Intervertebral disc prolapses
      - Upper femoral epiphysis separation
  - Extremes of age:
    - Children between 3 to 18 years (longitudinal bone growth may be arrested)
    - Old people-fragile bones
  - Those reacting to pin
  - Fixed flexion deformity

TYPES OF SKIN TRACTION

- These include:
  - Gallows
  - Natural
  - Boot
  - Lateral
  - Dunlop (for supracondylar fractures)

ASSESSING A PATIENT ON SKIN TRACTION

- Make general comments:
  - A patient lying supine in bed, not in any obvious pain and in skin traction.
- On assessing adequacy of skin traction one has to take into account:
  - Patient
    - Alignment: comment on whether the big toe/web space between the big toe and other big toe are in line with patella & anterior superior iliac spine.
    - Leg length: compare the length of both legs from medial malleolus to anterior superior iliac spine (Discrepancy< 1.5)
    - Compartment syndrome: rule out compartment syndrome which is characterized by severe progressive pain that responds poorly to analgesia.
    - Assess peripheral pulses and passively flex patients toes and there should not be any pain
  - Traction system
    - Traction kit: bandage must be below the knee generally and distal to fracture. Bandage should not be too tight so check for signs of edema in the feet.
    - Protection/ cushioning of malleoli must be present
    - Traction strings should pass through the pulley and should not shift sideways
    - The weight must not sit on anything and must be suspended in air.
    - The contents of the weight must be **1/10 of the body weight** for effective traction.
    - Sand bags should be placed under the femur to maintain the slight flexion of the knee.
  - Bed Elevation= 4cm x every kilogram of the weights placed.

ADVANTAGES AND DISADVANTAGES OF SKIN TRACTION

- Advantages:
  - Non invasive
  - Easy to apply
  - Cheap
- Disadvantages:
  - Has a weight limit: 4-5 kg
  - Ischemia if the bandage is too tight
  - Allergic reactions to strapping
  - Stiffness and contracture formation

LENGTH OF TRACTION

AGE	UNION	CONSOLIDATION
Child	3 weeks	6 weeks (3 x 2)
Adult	6 weeks	12 weeks (6 x 2)

CHILD

- At 3th week review patient:
  - Clinically:
    - Absence of pain
    - Mobility- child playing around in bed
    - Callous formation
  - X-ray
- At 6th week review patient and check x-ray

ADULT

- At 4-6th week review patient:
  - Clinically:
    - Absence of pain
    - Mobility
    - Callous formation
  - X-ray
- At 6th week review patient and check x-ray
- At 12th week review patient and check x-ray

COMPLICATION OF SKIN TRACTION

EARLY

- Compartment syndrome: Severe progressive pain that responds poorly to analgesics.
- Skin avulsion
- Allergic reaction

INTERMEDIATE

- Gangrene or ischemia

LATE

- Joint stiffness
- Hypostatic pneumonia
- DVT and embolism
- Mal-union, non-union and delayed union with traction that is not properly applied.

GALLOWS TRACTION (BRYANT)

- This is a type of skin traction used in fractures of the shaft of femur in children <3 years and weighing <15kg.
- Indications:
  - Orthopedic: fracture of the femur (main indication)
  - Non-orthopedic:
    - Perineal or gluteal burn/ abscess
    - Rectal prolapse
    - Hernias- obturator and umbilical
    - Non-thrombosed hemorrhoids
    - Spinal bifida
- Complications:
  - Skin avulsion
  - Allergic reaction to strapping
  - Compartment syndrome
  - Joint stiffness
  - Gangrene or ischemia of distal limb
  - Hypostatic pneumonia
- Signs due to incorrect application e.g. Hand can't sweep under buttocks.

- Advantages:
  - Easy to apply
  - Non invasive
  - Prevents decubitus ulcers
  - Children don't find it distressing
  - Cheap
- Disadvantages:
  - Weight limitation
  - Ischemia if too tight
  - Stiffness and contractures formation

DUNLOP TRACTION

- The main indication of Dunlop is a supracondylar fracture of the humerus in children.
- Skin traction is placed on the forearm and a special frame used on the side of the bed.
- Traction is placed along the axis of the forearm as well as at right angles to the humerus by means of a broad sling placed around the upper arm.
- Bed blocks are required on the lateral side (Fracture side up) of the bed.

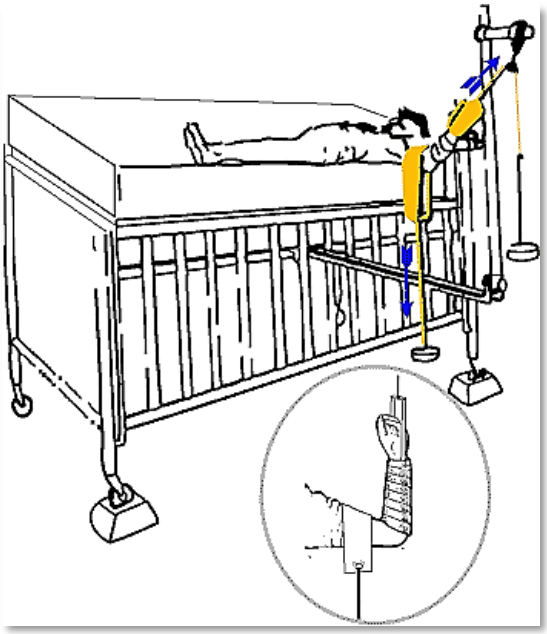


Figure 170: Dunlop traction

SKELETAL TRACTION (PERKINS TRACTION)

- This is a type of skeletal traction with a patient on a Perkins bed (from under bed should have springs half way and hallow on the other half to aid in exercises) and doing Perkins exercises.
- What can you see?
  - A patient lying supine in bed with leg abducted/adducted
  - He/she has a # femur
  - He/she is on skeletal traction with weights
  - Bed is raised with hard board

STATE OF LEG DURING PERKIN'S TRACTION

- Abducted= # of proximal 1/3 of femur (to avoid angulation after healing)
- Adducted/ straight= all other femur #s

APPLICATION OF PERKINS TRACTION

- Insert pin 2cm distal and 2cm lateral to the tibial tuberosity.
- Apply 1/7 of body weight (Adults) and for each kilogram of weight raise foot end of bed by 4cm.
  - ➢ This prevents over-traction & hence non-unions.
- Measure both patient's legs from the anterior superior iliac spine to the tip of medial malleoli to make sure they are the same length.
- If necessary, adjust the traction weight and elevation of the foot end of the end so as to let the bone fragments overlap by about 1 cm.
- Check leg length every day for the next 2 weeks and adjust the weight appropriately. Then check length every 2 weeks.
- Note when assessing patient on skeletal traction, follow a similar plan to that described under skin traction.

ADVANTAGES

- Perkins exercise helps to:
  - ➢ Increases blood supply
  - ➢ Promote healing
  - ➢ Controlled movement and compression of the bone ends encourages union
  - ➢ Prevents muscles atrophy- quadriceps
  - ➢ Prevents knee joint stiffness/ contractures
  - ➢ Prevents DVT, Decubitus ulcers and hypostatic pneumonia
  - ➢ Encourages positive psychological effect on patient

DISADVANTAGES

- Injury to common peroneal nerve (foot drop)
- Fracture of tibia at the site of insertion of the pin
- Pin tract infection (Pain, swelling, tenderness, pus), pin traction infection is prevented by:
  - ➢ Daily wound cleaning with proper technique (follow the 3 "S"):
    - S- spread the skin from the pin
    - S- squeeze towards the pin
    - S- swab around the pin making rotatory movements (use spirit swabs)
    - Edges of pins are then covered with spirit swabs
  - ➢ Types of pins used: Denham pin (has teeth in the middle), Steinmann (Smooth).

INDICATIONS

- Indications:
  - Undisplaced incomplete fracture of neck of the femur
  - All intertronchanteric fractures
  - Subtrochanteric fractures in which the contraction of the iliopsoas has not flexed the upper fragment so much as to bring it seriously out line.
  - All # of the shaft of the femur in patients over 18, including overlapped, double, spiral, comminuted and open #s and # with severe STI.
  - Supracondylar # in which the lower fragment has not been too severely flexed by the contraction of gastrocnemius.
  - All condyle # of the femur, except those in which a condyle has rotated completely

CONTRAINDICATONS

- All complete # of the neck of the femur
- Displacement of the proximal femoral epiphysis
- Subtrochanteric # with severe flexion of the proximal fragment
- Supracondylar # with marked flexion of the distal fragment
- Displacement of the distal femoral epiphysis
- # of the condyles in which a fragment has rotated completely.
- All patients under 18. Their epiphyses will not have united and pin may damage the epiphyseal plate.
- Arthritis of the knee, or a stiff knee from any cause which will make exercise impossible without moving the fragments too much
- Non-union in # treated by other methods

COMPLICATIONS

EARLY (DURING INSERTION)

- Damage to common peroneal nerve causing foot drop
- Fat embolism
- # of Tibia at the site of insertion of the pin
- Soft tissue injury
- Hemorrhage
- Hematoma

LATE (DURING TRACTION)

- Decubitus ulcer (bed sores)

- Pin site infection- osteomyelitis
- Pin may become loose
- Osteoporosis- bone resorption because patient is bed bound
- Over traction- causing non-union
- Malunion
- Delayed union
- Joint stiffness (ankyloses)
- Muscle disuse atrophy
- DVT
- Hypostatic pneumonia
- Contractures

PREVENTION

- Some complications can be prevented by careful pin insertion and Perkins exercise.
- Carful pin insertion:
  - Trans-tibial pin should be inserted 2cm distal and 2 cm lateral to the tibial tuberosity (this avoids injury to the common peroneal nerve)
  - The pin should be inserted from lateral to medial so as to avoid injury to common peroneal nerve
  - Can also be pushed into calcaneum or distal femur
- Types of pins
  - Denham pin: has some teeth in the middle
  - Steinmann: pin smooth
- Do daily inspection of pin site for possible infection

MANAGEMENT

- Start periods of 10-30 min of active exercise several times a day from the 3rd day.
- Callus in the first 10 days is what determines the outcome
- Testing the femur for clinical union:
  - No tenderness at the # site
  - Bone cannot be angulate at the # site
  - No tenderness on trying to angulate
  - Callus can be felt
- X-ray should be done on the 4th, 6th and 12th week.
  - If no callus on the X-ray by the 4th week suspect that there will be a delay in the union

- When there are definite signs of clinical union usually in the 6th to 10th week remove the weights and continue exercise- range of movement improves progressively
- Transverse and oblique #s take longer to heal than spiral #s.

- If during the first 2-3 days after a patient has fractured his femur he becomes disoriented, drowsy or comatose or he has a cough, shortness of breath and hemoptysis suspect fat embolism.
  - This is a result of globules of fat escaping from the injured marrow of the femur and entering the capillaries of the lungs or brain.
  - Look for petechia over the chest, mouth or in the conjunctiva.
  - Fat in urine is usually confirmatory.
  - Management: Restrict fluids, give oxygen and give him diuretics.

FRACTURES IN CHILDREN

GREENSTICK FRACTURES

- This is an incomplete fracture.
- Fracture breaks like a greenstick.
- On X-ray buckle is seen.



Figure 171: Greenstick fracture- needs only correction of angulation

EPIPHYSEAL FRACTURES

- A Salter-Harris fracture/Epiphyseal fracture is a fracture involving the epiphyseal plate or growth plate (also called the physis)
- This injury is common in children especially in long bones, this is because the physis is relatively a weak part of the bone.
- Joint strains that might cause ligament injuries in adults are liable to result in separation of the physis in children.
- Over 10% of fractures in children affect the physis.
- Fractures involving the epiphyseal plate were named after Robert B Salter and William H Harris who created and published the classification system for these types of fractures.
- Recall the structure of a bone:

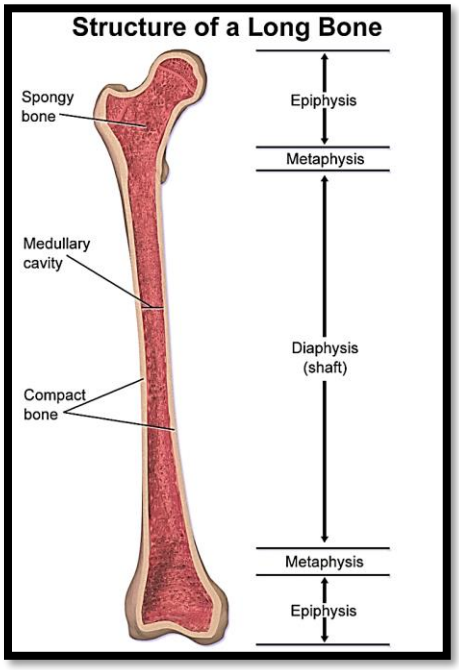


Figure 172: Anatomy of a long bone

CLASSIFICATION

- There are 5 main types described by Salter and Harris (1963)

TYPE I

- Involves a transverse fracture through the hypertrophic or calcified zone of the growth plate.
- Even if the fracture is quite alarmingly displaced, the growing zone of the physis is usually not injured and growth disturbance is uncommon.
- Incidence is about 5%.

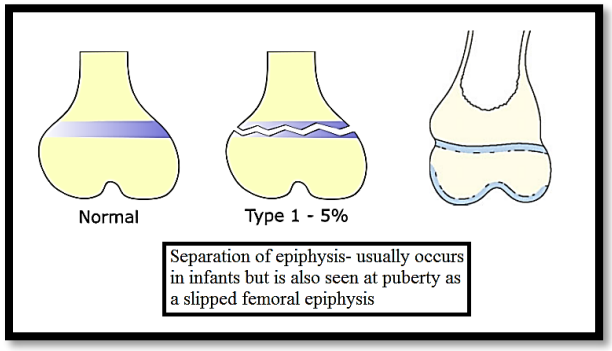


Figure 173: Salter-Harris Type I- "Straight through"



Figure 174: Salter-Harris I fracture of distal radius "Straight through"

TYPE II

- Entails a fracture through the growth plate and metaphysis sparring the epiphysis.
- This fracture is essentially similar to type I but towards the edge the fracture deviates away from the growth plate and metaphysis.
- The fracture splits off a triangular metaphyseal fragment of bone (sometimes referred to as Thurston-Holland fragment)
- It accounts for the commonest type of epiphyseal fracture (incidence of 75%) and takes approximately 2-3 weeks or more in the spine to heal.

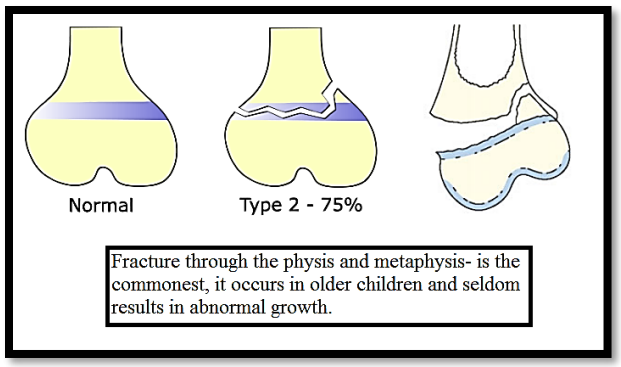


Figure 175: Salter-Harris Type II with Thurston Holland fragment - "Above"



Figure 176: Salter-Harris Type II fracture of ring finger proximal phalanx

TYPE III

- A fracture through growth plate and epiphysis sparing the metaphysis.
- The fracture splits the epiphysis and then veers off transversely to one or the other side, through the hypertrophic layers of the physis (as these layers are closer to the epiphysis than the metaphysis) and may result in growth disturbance.
- Incidence= 10%.

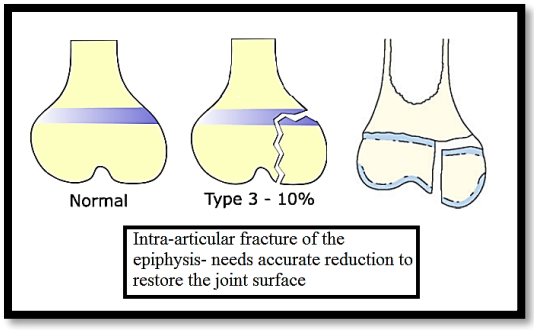


Figure 177: Salter-Harris Type III- "Lower"



Figure 178: Salter-Harris Type III fracture of big toe proximal phalanx

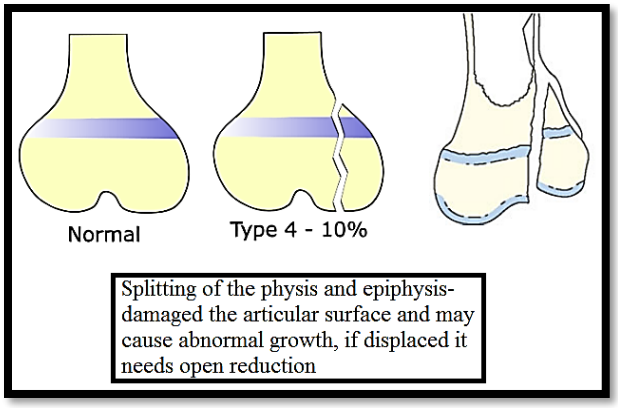


Figure 179: Salter-Harris Type IV- "Through Everything"



Figure 180: Salter-Harris IV fracture of big toe proximal phalanx

TYPE IV

- Fracture through all 3 elements: growth plate (physis), metaphysis and epiphysis.
- These fractures are liable to displacement and a consequent misfit between the separated parts of the physis, resulting in asymmetrical growth.
- Incidence= 10%.

TYPE V

- A longitudinal compression injury of the physis.
- There is no visible fracture but the growth plate is crushed and this may result in growth arrest.
- Incidence- 1%

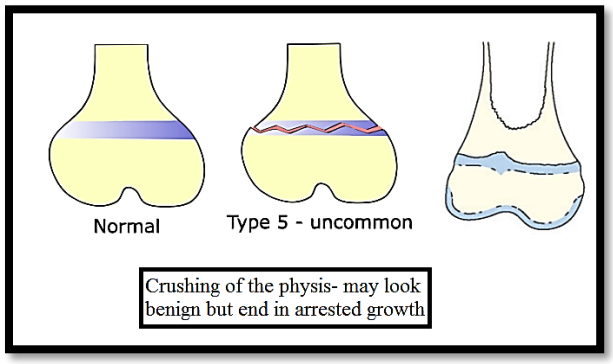


Figure 181: Salter-Harris Type V- "Rammed"

- Rang (Rang 1969) had added a type VI an injury to the perichondrial ring (the peripheral zone of Ranvier) which carries a significant risk of growth disturbance. The diagnosis is made usually in retrospect after development of deformity.
- The mnemonic "SALTER" can be used to help remember the first 5 types:
  - Type I- S= Slip/ Separated or straight across- fracture of cartilage of physis
  - Type II-A= Above- the fracture lies above the physis or Away from the joint.
  - Type III-L= Lower- fracture is below the physis in the epiphysis.
  - Type IV- TE= Through Everything- fracture through metaphysis, physis and epiphysis.
  - Type V-R= Rammed (crushed)- the physis has been crushed.
- Alternatively, "SALTER" can be used for the first 6 types as above but adding Type V- "E" for "Everything" or "Epiphysis" and Type VI- "R" for "Ring".
- Injuries to the physis usually run transversely through the hypertrophic or calcified layers of the growth plate, often veering off into the metaphysis at one of the edges to include a triangular lip of bone.
- This has little effect on longitudinal growth, which takes place in the germinal proliferating layers of the physis.
- If the fracture traverses the cellular 'reproductive' layers of the physis it may result in premature ossification of the injured part & serious disturbance of bone growth.

MECHANISM OF INJURY

- Physeal fractures usually result from falls or traction injuries.
- They occur mostly in road accidents and during sporting activities or playground tumbles.

CLINICAL FEATURES

- These fractures are more common in boys than girls and are usually seen either in infancy or between the ages of 10 and 12.
- Deformity is usually minimal but any injury in a child followed by pain and tenderness near the joint should arouse suspicion and X-ray examination is essential.

X-RAYS

- Recall that the physis is made of cartilage that is radiolucent and epiphysis may be incompletely ossified thus making it hard to tell whether the bone is damaged or deformed.
- The younger the child the smaller the "visible" part of the epiphysis and thus the more difficult to make diagnosis, comparison with the normal side is a great help.
- Telltale features:
  - Widening of the 'Physeal gap'
  - Incongruity of joint or tilting of the epiphyseal axis
- If there is marked displacement the diagnosis is obvious, but even a type 4 fracture may at first be so little displaced that the fracture line is hard to see.
- If there is the faintest suspicion of a Physeal fracture a repeat X-ray after 4 or 5 days is essential.
- Type 5 and 6 injuries are usually diagnosed only in retrospect.

TREATMENT

UNDISPALCED

- Splinting the part in a cast or a close-fitting plaster slab for 2-4 weeks (depending on the site of injury and the age of child).
- Type 3 and 4 fracture- check X ray after 4 days and again at about 10 days is mandatory in order not to miss late displacement.

DISPLACED

- Reduce as soon as possible.
- Type 1 and 2- closed reduction, the part is then splinted securely for 3-6 weeks.
- Type 3 and 4 demand perfect anatomical reduction
  - This is done by gentle manipulation under general anesthesia and if successful the limb is held in a cast for 4-8 weeks (longer periods for type 4 injuries).
  - If they cannot be reduced accurately by closed manipulation, immediate open reduction and internal fixation (ORIF) with smooth K-wires is essential. The limb is then splinted for 4-6 weeks but it takes that long again before the child is ready to resume unrestricted activities.

PROGNOSIS

- Fractures in children generally heal relatively fast but may take several weeks to heal.
- Most growth plate fractures heal without any lasting effects.
- Rarely bridging bone may form across the fracture, causing stunted growth and/or curving. In such cases the bridging bone may need to be surgically removed.
- A growth plate fracture may also stimulate growth causing a longer bone than the corresponding bone on the other side.

COMPLICATIONS

- Type 1 and 2- if properly reduced have an excellent prognosis and bone growth is not adversely affected.
  - Exception: injuries around the knee involving the distal femoral or proximal tibial physis, both growth plates are undulating in shape, so a transverse fracture plane may actually pass through more than just the hypertrophic zone but also damages the proliferative zone.
  - Complications such as malunion or non-union may occur if diagnosis is missed and fracture remains unreduced (e.g. fracture separation of the medial humeral epicondyle).
- Type 3 and 4: result in premature fusion of part of the growth plate or asymmetrical growth of the bone end.
- Type 5 and 6: premature fusion and retardation of growth. Can also result in bridging.

- Establish deformity whether from asymmetrical growth or from malunion of a displaced fracture (e.g. a valgus elbow due to proximal displacement of a lateral condylar fracture) should be treated by corrective osteotomy. If further growth is abnormal the osteotomy may have to be reported.

SUPRACONDYLAR FRACTURE OF THE HUMERUS

- These are among the commonest fractures in children.
- The distal fragment may be displaced either posteriorly or anteriorly.

MECHANISM OF INJURY

- Posterior angulation or displacement (95% of all cases) suggests a hyperextension injury, usually due to a fall on the outstretched hand.
  - The humerus breaks just above the condyles. The distal fragment is pushed backwards and (because the forearm is usually in pronation) twisted inwards.
  - The jagged end of the proximal fragment pokes into the soft tissue anteriorly, sometimes injuring the brachial artery or median nerve.
- Anterior displacement is rare; it is thought to be due to direct violence (e.g. a fall on the point of the elbow with the joint in flexion.)

CLASSIFICATION (GATLAND CLASSIFICATION)

TYPE I

- An undisplaced fracture



Figure 182: Undisplaced supracondylar fracture (Gatland Type I)

- Treatment: collar and cuff or backslab.

TYPE II

- An angulated fracture with the posterior cortex still in continuity (periosteal hinge).

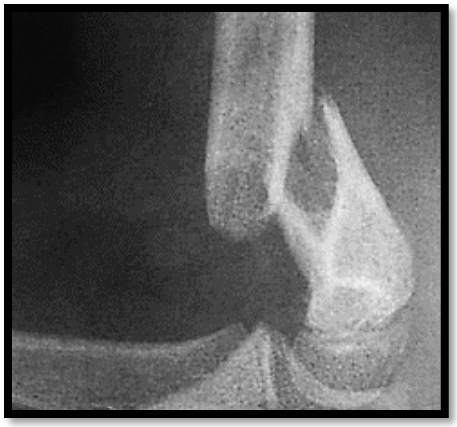


Figure 183: Angulated fracture with periosteal hinge (Gatland type II)

- Type IIA- a less severe injury with the distal fragment angulated.
- Type IIB- a severe injury, the fragment is both angulated and malrotated.

- Treatment: Manipulation under anesthesia (MUA) + collar cuff or backslab. (convert to type I)

TYPE III

- Completely displaced fracture (Although the posterior periosteum is usually still preserved, which will assist surgical reduction)
- Treatment:
  - Manipulation under anesthesia (to convert to type 1)
  - K-wire (Kirschna wires) fixation
  - After 3 weeks take the patient to theatre to remove K-wire under general anesthesia and to put a slab.

FRACTURES IN ADULTS

UPPER LIMB FRACTURES

GALEAZZI FRACTURE-DISLOCATION OF THE RADIUS

MECHANISM OF INJURY

- This injury was first described in 1934 by Galeazzi. The usual cause is a fall on an outstretched hand, probably with superimposed rotation force.
- It may also be caused by direct trauma or blow to the radius.
- The radius fractures in its lower third shaft and the distal radio-ulnar joint subluxates or dislocates.
- Remember "**Gale-radius**" to help recall that the Galeazzi fracture is of the distal shaft of radius.
- To also remember the site of displacement note the joint usually displaces where the one the fractured bone is bigger.

CLINICAL FEATURES

- The Galeazzi fracture is much more common than the Monteggia. Always assess for distal radial-ulnar joint disruption in all patients with radial shaft fractures.
- Prominence or tenderness over the lower end of the ulna is the striking fracture.
- Swelling and often bruising is also seen in the area.
- Patients also have wrist pain, instability or both
- It may be possible to demonstrate the instability of the radio-ulnar joint by 'balloting' the distal end of the ulna (the 'piano-key sign' or by rotating the wrist).
- It is important to also test for an ulnar nerve lesion, which may occur.
- X-ray:
  - A transverse or short oblique fracture seen in the lower third of the shaft of radius, with angulation or overlap
  - The distal radio-ulnar joint is subluxated or dislocated



Figure 184: Galeazzi fracture

TREATMENT

- Restore the length of the fractured bone.
- In children closed reduction is often successful, in adults open reduction and compression plating (ORIF) of the radius is successful.
- An X-ray is taken to ensure that the distal radio-ulnar joint is reduced, there are 3 possibilities:
  - ➢ The distal radio-ulnar joint is reduced and stable:
    - No further action needed
    - Arm rested for few days then gentle active movements are encouraged.
    - Radio-ulnar joint checked clinically and radiologically during the next 6 weeks.
  - ➢ The distal radio-ulnar joint is reduced and unstable:
    - Forearm should be immobilized in the position of stability (usually supination) supplemented if required by a transverse K-wire.
    - Forearm is splinted in an above-elbow cast for 6 weeks.
    - If there is a large ulnar styloid fragment it should be reduced and fixed.
  - ➢ The distal radio-ulnar joint is irreducible:
    - Unusual
    - Open reduction is needed to remove the interposed soft tissue.
    - Triangular fibrocartilage complex (TFCC) and dorsal capsule are then carefully repaired and the forearm immobilized in the position of stability (Again usually supination, supported by a wire if needed) for 6 weeks.
- Note: non-anatomic reduction of the radial shaft fractures will compromise pronation and supination.

MONTEGGIA FRACTURE-DISLOCATION OF THE ULNA

- Previously this described a fracture of the shaft of the ulna associated with dislocation of the proximal radio-ulnar joint (radial head). The radiocapitellar joint is inevitably dislocated or subluxated as well.
- Recently it also entails any fracture of ulna associated with dislocation of the radio-capitellar joint, including trans-olecranon fractures in which the promixal radio-ulnar joint remains intact.
- If the ulnar shaft fracture is angulated with the apex anterior (most common) then the radial head is displaced anterior.
- If the fracture apex is posterior, the radial dislocation is posterior.

- If the fracture apex is lateral, then the radial head will be laterally displaced.
- In children the ulnar injury may be an incomplete fracture (greenstick or plastic deformation of the shaft).

MECHANISM OF INJURY

- Usually the cause is a fall on the hand, it at the moment of impact the body is twisting, its momentum may forcibly pronate the forearm.
- The radial head usually dislocates forwards and the upper third of the ulna fractures and bows forwards. Sometime the causal force is hyperextension.
- Remember '**Monte-ulnar**' to recall that this is a fracture of the shaft of ulnar with proximal radio-ulnar dislocation.

CLINICAL FEATURES

- The ulnar deformity is usually obvious but the dislocated head of radius is masked by swelling.
- A useful clue is pain and tenderness on the lateral side of the elbow.
- The wrist and hand should be examined for signs of injury to the radial nerve.
- X-ray:
  - Usual case: head of the radius (which normally points directly to the capitulum) is dislocated forward and there is a fracture of the upper third of the ulna with forward bowing.
  - Backward or lateral bowing of the ulna (which is much less common) is likely to be associated with, respectively, posterior or lateral displacement of the radial head. Trans-olecranon fractures, also are often associated with radial head dislocation.

TREATMENT

- Restore length of fractured ulna, only then can the dislocated joint be fully reduced and remain stable.
- In adults: the ulnar fracture must be accurately reduced, with the bone restored to full length and then fixed with plate and screws, bone grafts may be added for safety
- The radial head usually reduces once the ulna has been fixed. Stability must be tested through a full range of flexion and extension. If the radial head does not reduce or is not stable, open reduction should be performed.
- If the elbow is completely stable, then flexion-extension and rotation can be started after very soon after surgery. If there is doubt, then the arm should be immobilized in plaster with the elbow flexed for 6 weeks.



Figure 185: Monteggia fracture-dislocation

COLLES FRACTURE

- This was an injury described by Abraham Colles (1814) characterized by a transverse fracture of the radius just above the wrist, with dorsal displacement of the distal fragment.
- It is the most common of all fractures in older people, the high incidence being related to the onset of postmenopausal osteoporosis.
- Thus, the patient is usually an older women who gives a history of falling on her outstretched hand.

MECHANISMS OF INJURY

- Force is applied in the length of the forearm with the wrist in extension.
- The bone fractures at the cortico-cancellous junction and the distal fragment collapses into extension, dorsal displacement, radial tilt and shortening.

CLINICAL FEATURES

- 'Dinner-fork' deformity with prominence on the back of the wrist and a depression in front.
- In patients with less deformity there may only be local tenderness and pain on wrist movements.
- X-rays:
  - Transverse fracture of the radius at the corticocancellous junction (2.5cm above the wrist through the epiphysis) and often the ulnar styloid process is broken off.
  - Displacement:
    - Dorsal shift
    - Dorsal tilt (proximal tilt)
    - Supination
    - Impaction
    - Lateral shift (Radial shift)
    - Lateral tilt
    - Rotation
- To remember the displacement in colles' fracture recall: College Rotating Doors Shut I'm late
  - College- colles
  - Rotating- Rotation
  - Doors
    - Dorsal shift
    - Dorsal tilt (proximal tilt)
  - Shut
    - Supination
  - I'm

- Impaction
- Late
  - Lateral tilt
  - Lateral shift (radial shift)

- Note: Colle's fracture is described in Elderly in children fractures similar to this are known as Colle's like

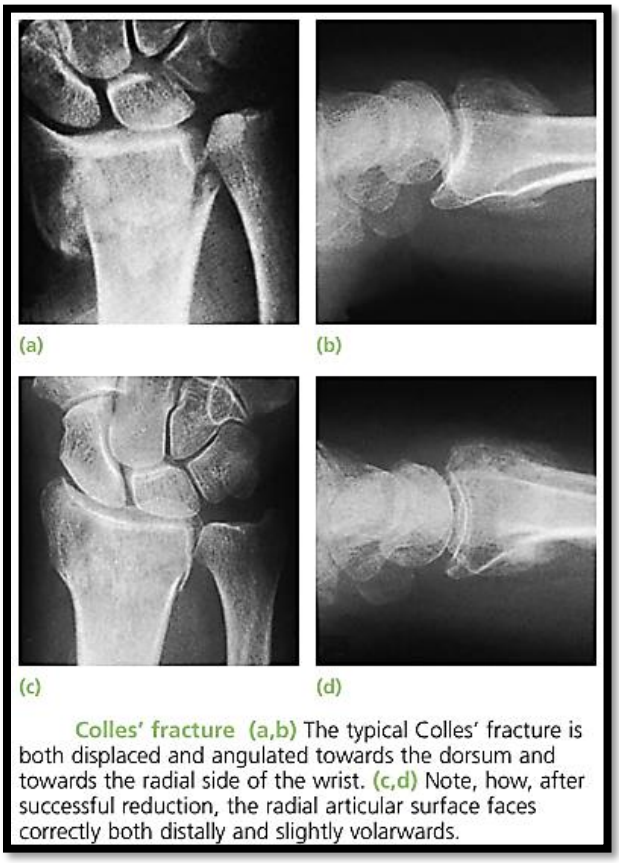


Figure 186: Colles fracture

TREATMENT

- Closed manipulation under general anesthesia:
  - Correct the impaction first
  - Center it (correct the radial displacement)
  - Correct proximal tilt by dorsiflexing the wrist.
  - Lock the arm towards the little finger using a slab (also appears like a "dinner fork" but it is a corrected one)
- Request X-ray of the wrist (AP and lateral view)-to check if fracture has been reduced
- Review after 24 hours for neurovascular state
- Review after 7 days and repeat X-ray of the wrist to rule out displacement
- Review after 7 days and repeat X-ray.
- Review after 3 weeks
- Review after 8 weekS
- If the fracture is displaced in any of the reviews the patient goes back to theatre for reduction and the procedure is repeated.

SMITH'S FRACTURE

- In this injury the distal fragment is displaced anterior (which is why it is sometimes called a 'reversed Colles'). It is caused by a fall on the back of the hand.

CLINICAL FEATURES

- Presentation:
  - Wrist injury
  - No dinner fork deformity instead a 'garden spade' deformity
- X-ray:

- Fracture through the distal radial metaphysis
- Lateral view: distal fragment is displaced and tilted anteriorly- the opposite of a Colles' fracture.
- The entire metaphysis can be fractured, or there can be an oblique fracture exiting at the dorsal or volar rim of the radius.

- Treatment:
  - Reduced by traction, supination and extension of the wrist and forearm is immobilized in a cast for 6 weeks.
  - X-rays should be taken at 7-10 days to ensure the fracture has not slipped.
  - Unstable fractures should be fixed with percutaneous wires or a plate.

LOWER LIMB FRACTURES

FRACTURE OF THE FEMUR

- Fractures of the femur are classified according to:
  - Site of fracture
  - Displacement (Garden's classification)

SITE OF FRACTURE

- Fractures can be classified as:
  - Neck of Femur #
    - Subcapital
    - Transcervical
    - Basal
  - Intertrochanteric #
  - Subtrochanteric #
  - # of the shaft- proximal, mid or distal

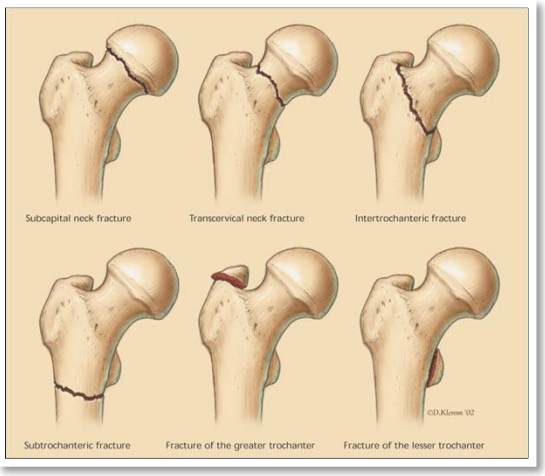


Figure 187: Anatomical fractures of the femur

GARDEN'S CLASSIFICATION OF FEMORAL NECK FRACTURES

TYPE I

- Incomplete and minimally displaced

TYPE II

- Complete break without displacement

TYPE III

- Complete break and partially displaced

TYPE IV

- Complete break with the displacement
- Carries a risk of avascular necrosis

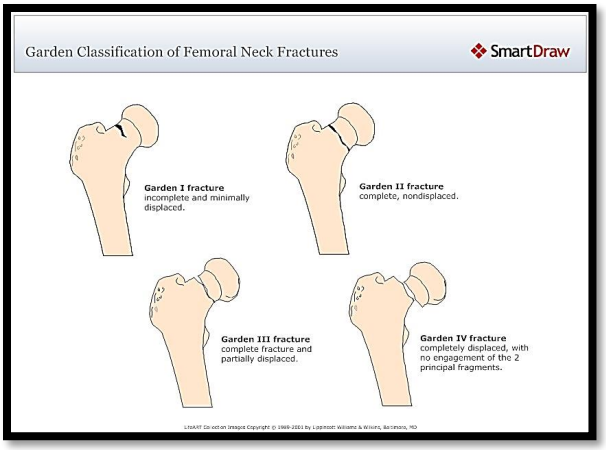


Figure 188: Garden classification of femoral neck fractures

ANKLE FRACTURES

- Any fracture involving the ankle was termed a Potts' fracture.
- Classification of ankle fractures are varied and generally complex.
- To understand these types of fractures it is important to recall the ankle mortise which is composed of:
  - Medial malleolus
  - Distal articular surface of tibia
  - Tibio-fibula syndesmosis
  - Lateral malleolus

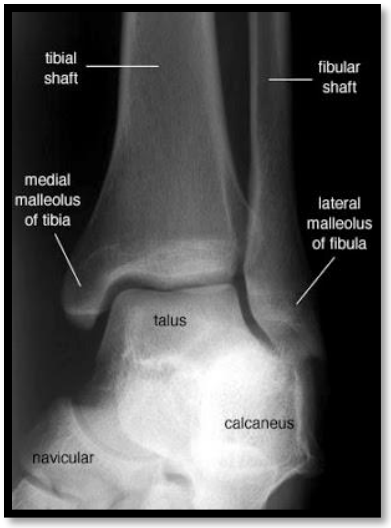


Figure 189: Anatomy of the Ankle Mortise

- Note: the talus is not a component of the ankle mortise.

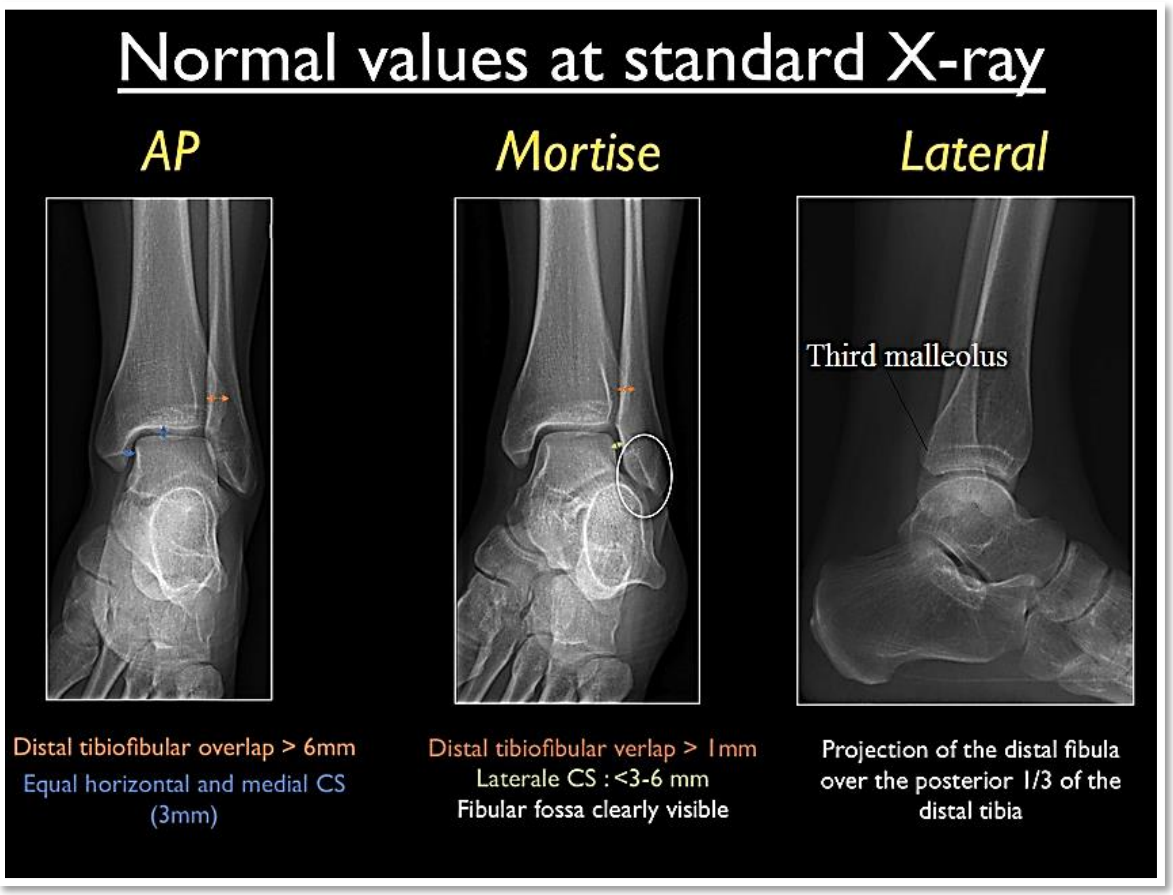


Figure 190: Anatomy of the mortise

- Ankle fractures often carry a history of a twisting injury, acute pain and inability to bear partial or full weight.
- Presence of swelling and tenderness at one malleolus or over the entire ankle may be seen as well as pain with motion.
- Fracture of the ankle are classified according to:
  - Anatomical site
  - Mechanism of action
  - Eponym (Weber's classification- in relation to the fibula)

ANATOMICAL

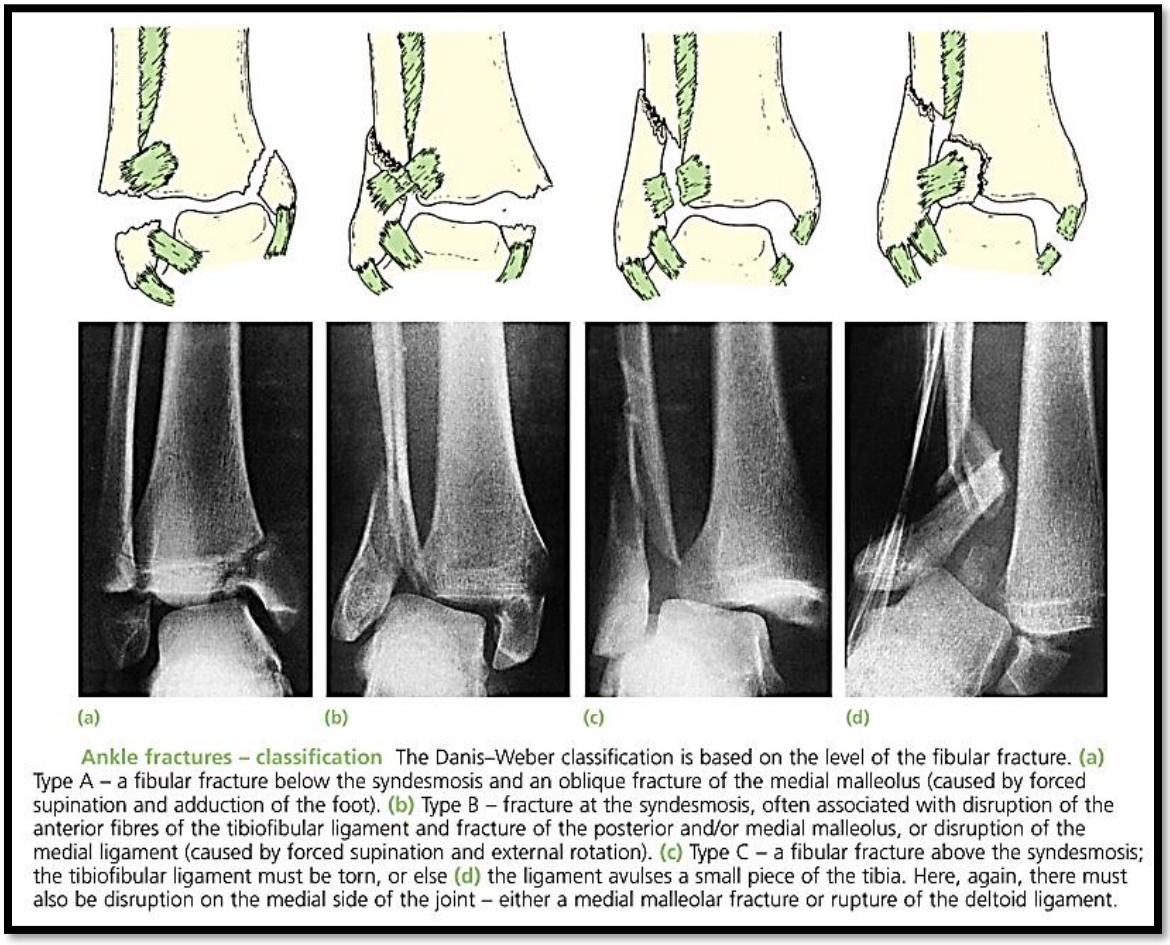
- These include:
  - Unimalleolar (medial or lateral)
  - Bimalleolar (medial and lateral)
  - Trimalleolar (medial, lateral and posterior)

MECHANISM OF INJURY (LAUGE-HENSEN CLASSIFICATION)

- Include:
  - Supination with external rotation
  - Supination with adduction
  - Pronation with abduction
  - Pronation with external rotation

WEBER'S CLASSIFICATION

- This is relation to the fibula and tibio-fibular syndesmosis
  - Type A: below tibio-fibula syndesmosis
  - Type B: At the level of the tibio-fibula syndesmosis
  - Type C: Above the Tibio-fibula syndesmosis



Ankle fractures – classification The Danis–Weber classification is based on the level of the fibular fracture. (a) Type A – a fibular fracture below the syndesmosis and an oblique fracture of the medial malleolus (caused by forced supination and adduction of the foot). (b) Type B – fracture at the syndesmosis, often associated with disruption of the anterior fibres of the tibiofibular ligament and fracture of the posterior and/or medial malleolus, or disruption of the medial ligament (caused by forced supination and external rotation). (c) Type C – a fibular fracture above the syndesmosis; the tibiofibular ligament must be torn, or else (d) the ligament avulses a small piece of the tibia. Here, again, there must also be disruption on the medial side of the joint – either a medial malleolar fracture or rupture of the deltoid ligament.

Figure 191: Ankle fractures

MANAGEMENT

- Stable fractures (lateral malleolar fractures with stable mortise and no medial swelling or tenderness) can be treated with Cam walkers or short-leg walking casts.
- Unstable fractures (lateral malleolar fractures with either mortise widening or medial swelling or tenderness) are treated with plating of the fibula (for spiral or oblique fractures) and two screws (for most medial malleolar fractures).
- Syndesmostic disruption requires fixation with screws
- Posterior malleolus fractures should be fixed if the fragments comprise >25% of the plateau joint surface or if there is displacement after reduction.
- Dislocations should be reduced urgently even if surgery will be performed later to ensure viable soft tissue over the ankle
- Note: The lateral malleolus should be fixed first, which will bring the ankle out to length, reduction and fixation of the medial malleolus can then be accomplished.

JOINT DISLOCATIONS

- This is an abnormal separation in the joint.
- It is also known as luxation.
- A partial dislocation is referred to as subluxation.
- Joint dislocations are caused by sudden trauma on the joint like an impact or fall.
- They cause damage to the surrounding ligaments, tendons, muscles and nerves.
- Dislocations can occur in any joint major (shoulder, knees etc.) or minor (toes, fingers, etc.)
- The most common joint dislocation is a shoulder dislocation.
  - Dislocated shoulder- 45% of all dislocations

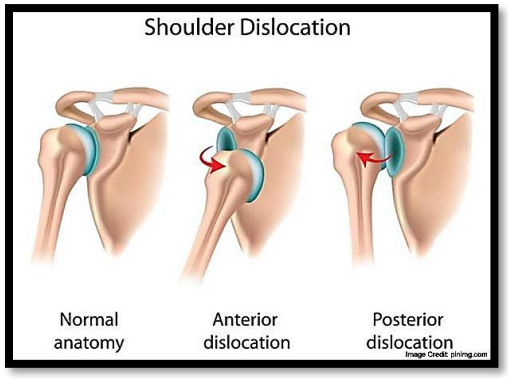


Figure 192: Shoulder dislocation

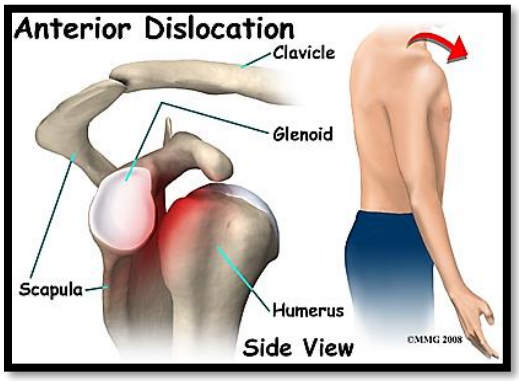


Figure 193: Anterior shoulder dislocation

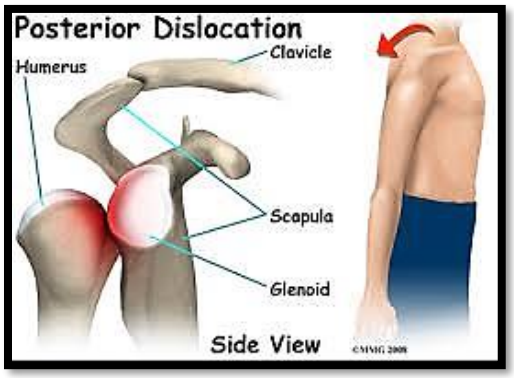


Figure 194: Posterior shoulder dislocation

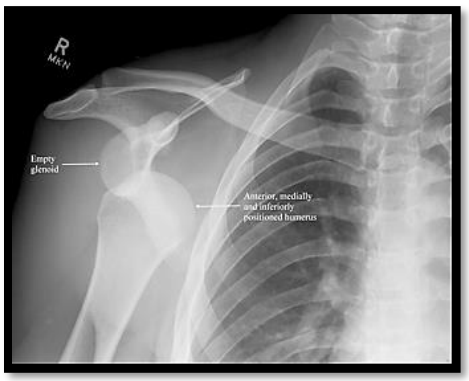


Figure 195: Shoulder dislocation

- Knee- patellar dislocation
- Elbow: posterior dislocation, 90% of all elbow dislocations

- Wrist: Lunate and Perilunate dislocation most common
- Finger: interphalangeal (IP) or metacarpophalangeal (MCP) joint dislocations
- Hip: posterior and anterior dislocation of hip
- Foot and ankle

- The following symptoms are common with any type of dislocation:
  - Pain
  - Joint instability
  - Deformity, Bruising
  - Reduced muscle strength
  - Difficult moving joint and stiffness



Figure 196: Shoulder dislocation

- Plain radiographs usually a minimum of 2 views
- A dislocated joint usually can be successfully reduce (i.e. manipulated) back into its normal position.

AMPUTATIONS

AMPUTATIONS

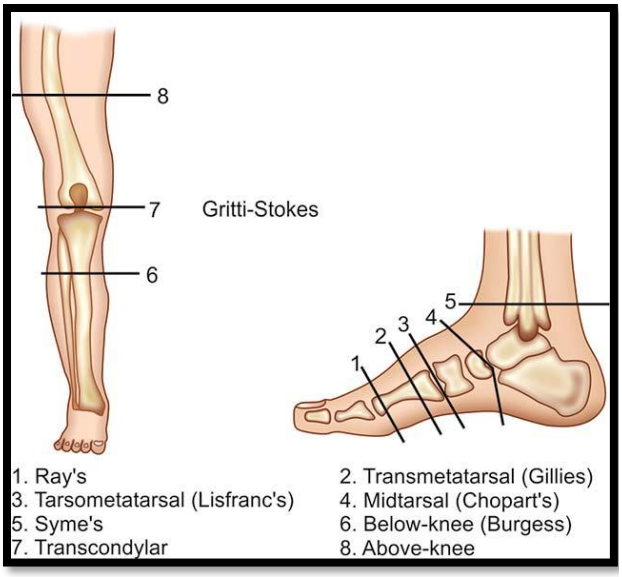
- This is removal of a limb or other appendage of the body.

INDICATIONS

- Indicated when part of the limb is Dead, Deadly/Dangerous, Dying and Damn nuisance.
- Dead (Vitality of limp is destroyed):
  - The limb may be dead when arterial occlusion or stenosis causes tissue infarction.
  - Dry gangrene due to arterial occlusion or stenosis.
  - Arterial occlusion:
    - Major vessels- atherosclerotic/ embolic occlusions
    - Small vessel- DM, Buegers disease, Raynaud's disease, egotism
- Deadly/ Dangerous (life saving)
  - Wet gangrene with putrefaction infection.
  - Gas gangrene due to C. perfringes
  - Necrotizing fasciitis
  - Neoplasm: osteogenic sarcoma, extensive melanoma, Marjolin's ulcer
  - Arteriovenous fistula: life of the patient is threatened by spread of a local condition.
  - Spreading cellulitis
  - Uncontrolled sepsis (diabetic foot) systemic infection.
- Dying
  - Severe laceration
  - Severe fractures
  - Partial amputation due to trauma or burns
- Damn nuisance (deformed/ neuropathy)
  - Polydactyly
  - Severely impaired gait

TYPES OF AMPUTATIONS

1. Rays amputation
   - Amputation of toe with head of metatarsal or metacarpals.
2. Tarsometatarsal (Lisfranc's)
3. Transmetatarsal (Gillies)
4. Midtarsal (Chopart's)
5. Syme's
6. Below-knee (Burgees)
7. Transcondylar (through knee)
8. Above-knee
9. Hip-disarticulation



COMPLICATIONS

EARLY

- Hemorrhage and hypovolemic shock
- Hematoma:
  - Identified by pain, swelling over the stump underneath the flap.
  - May delay healing, precipitate infection or flap necrosis due to pressure
  - It is aspirated using a wide bore needle and after aspiration, pressure dressing is needed. If hematoma reforms after 2-3 aspirations it should be drained by opening the wound on one corner and inserting hemostat into the wound.
- Infection

LATE

- Phantom limb- typical awareness of sensation that as if amputated part is still present partly or in toto. It is often painful, disturbing or hyerasthetic.
  - Exact cause is not known but probably due to presence of severe pain at the amputated part just prior to amputation making brain area for that part in alert situation causing phantom limb. (Cortical plasticity).
  - Reassurance, prosthesis, analgesics help to control the condition.
  - Telescoping of phantom limb: feeling of the amputated limb getting “shorter”
- Ulceration of stump:
- Flap necrosis: due to poor blood
- Stump neuroma:
  - Occurs due to proliferation of the nerve fibrils beyond the point of nerve division and is usually due to failure of cutting of the nerve more proximal to the level of division of the bone.
  - It causes pain and tenderness over the stump. It is usually relieved by analgesics, reassurance and prosthesis. Occasionally it may require reexploration of the wound, excision of end neuroma and also cutting nerve more proximally.

- Painful scar
- Stump pain: due to infection, poor blood supply, causalgia, stump neuroma, phamtom limb, DVT, adherent scar, formation of spurs and osteophytes at amputated bone end.
- Sequestrum formation
- Contracture of joint proximal to the amputated stump
- Deformities
- Keloids and hypertrophic scars

OSTEOARTHRITIS

- This is a degenerative bone and joint condition initially involving thinning of the hyaline cartilage until the underlying bone is exposed and becomes damaged due to direct stress.
- It can be:
  - Primary (idiopathic) which is localized or generalized (rare) or
  - Secondary which is usually localized.
- Nodal osteoarthritis is a primary osteoarthritis involving the hands.

RISK FACTORS

- Age>50 years
- Nodal OA:
  - Post-menopausal women
  - Family history
- Secondary:
  - Mechanical strain from obesity, trauma (fracture into a joint, cartilage tear) or employment
  - Joint disease: inflammatory arthritides
  - Congenital conditions: congenital dislocation of the hip
  - Metabolic: gout, crystal arthropathy, Hemochromatosis
  - Infection: tuberculosis
  - Chronic inflammation: rheumatoid arthritis
  - Osteoporosis reduces the risk

CLINICAL FEATURES

Primary nodal OA	Secondary OA
• Tender, swollen joints with reduce movement • Typically affects the distal interphalangeal joints (DIP), thumb carpometacarpal (CMC) joint, and knees • Muscle wasting • Squaring of hand due to thumb adduction and bone swelling at CMC	• Typically affects the knee, hip or spine • Painful joint exacerbated by movement and worse in the evening • Joint gelling- stiffness after resting and instability • Hip OA typically radiates to the groin and buttocks.

• Heberden's nodes at the DIP and Bouchard's nodes at the PIP	• Spin OA may compress a nerve and radiate down the leg

INVESTIGATIONS

- Diagnosis can be clinical- especially if patient >45 years old- and does not require radiological evidence.
- X-ray (anteroposterior and lateral views) can be useful to exclude other conditions and are needed in younger patients or atypical histories. In osteoarthritis, may see 'LOSS':
  - L-loss of joint space (asymmetrical), reflecting thinning of the cartilage
  - O-osteophytes, reflecting proliferation and ossification of cartilage in unstressed areas
  - S-subchondral sclerosis (thickening)
  - S-Subchondral cysts, reflecting fluid-filled microfractures
- Bloods:
  - CRP/ESR to rule out inflammatory cause.

MANAGEMENT

- Lifestyle and physical therapy:
  - Exercise to improve strength and joint stability
  - Reduce weight
  - Walking aids
  - Physio
- Pharmacological:
  - Regular paracetamol +/- topical NSAID gel (knee or hands only), then move up pain ladder if needed. Paracetamol is traditionally preferred for long-term use over NSAIDs as it is safer but recent research question its efficacy.
  - If adding/switching to NSAIDs PO (standard or coxib) stop topical NSAID and give PPI. Unlike paracetamol, there is good evidence of efficacy.
  - Further option: intra-articular steroid injections, topical capsaicin for hand or knee OA.
  - Glucosamine and chondroitin are not recommended
- Surgical: joint replacement

RHEUMATOID ARTHRITIS

SEPTIC ARTHRITIS

OSTEOMYELITIS

- This is infection of the bone
- It may be:
  - Acute osteomyelitis: <2 weeks time duration
  - Chronic osteomyelitis: >2 weeks (typically presents 6 weeks or more after infection)

ACUTE OSTEOMYELITIS

RISK FACTORS

- Portal to pathogen entry:
  - Trauma: open fracture or orthopedic surgery
  - Surgical prosthesis
  - Intravenous drug use
- Disease
  - TB
  - Diabetes
  - PVD
  - Immunosuppression
  - Alcoholism
  - Sickle cell disease

PATHOPHYSIOLOGY

- Route of infection is mainly hematogenous (common in children, patients with urinary catheters or TB), rarely by direct/contiguous spread (e.g. trauma, cellulitis, abscess, surgery or prosthesis).
- Most cases of acute hematogenous osteomyelitis are caused by *Staphylococcus aureus*. Other less common pathogens include:
  - *Streptococcus pyogenes* and *Hemophilus influenzae* (in children)
  - Gram negative bacilli (in adults)
  - *Pseudomonas aeruginosa* (in IV drug users)
  - *Salmonella (in sickle cell patients)*
- Bacteria lodge in end artery of metaphysis and multiply.
  - Most common site for acute osteomyelitis is the metaphyseal end of a single long bone (especially around the knee)
- There is local increase in serum and white blood cells leading to decrease in blood flow and pressure necrosis. WBCs also release enzymes that cause bone lysis and leave necrotic areas (sequestra) with new bone forming around this (involucrum)
- Pus moves to haversian and medullary canals and eventually beneath the periosteum.
- Chronic osteomyelitis ensues after 6 months of infection.

CLINICAL FEATURES

- History of infection (e.g. skin or throat) or trauma
- Local inflammation, Significant pain in the affected area,
- Systemic symptoms: anorexia, fever, irritability, nausea, malaise and rapid pulse
- Limited joint motion, slight effusion in neighboring joints, tenderness, swelling of soft tissue and guarding apparent on physical exam.
- Diabetic patients may be asymptomatic due to neuropathy.
- Vertebral osteomyelitis:
  - Localized spine inflammation. 'Cold' in TB
  - Chronic back pain which is worse at night and at rest
  - Associated discitis

DIAGNOSIS

- Full blood count: Increased WBC, +/- anemia
- ESR and C-reactive protein are increased
- Blood culture is the gold standard. Also, culture blood, pus, and local joint effusion.

- Perform urine urinalysis, microscopy culture and sensitivity to look for cause.
- X-ray may show dark area in bone, soft-tissue swelling however signs may be minimal in acute infection. MRI provides clearer picture if diagnosis is uncertain.
- Deep circumferential soft-tissue swelling with obliteration of muscular planes

DIFFERENTIAL DIAGNOSIS

- Septic arthritis: Swelling and tenderness directly on the joint with intense pain on joint movement, high WBC and positive culture.
- Rheumatic fever: more insidious onset, less local and constitutional symptoms
- Ewing's sarcoma: Early symptoms are more insidious and less intense and present with bone destruction

MANAGEMENT

- Medical:
  - Infection must be diagnosed early
  - Intravenous antibiotics (usually oxacillin or cloxacillin 8-16g adult) started soon after obtaining specimen for culture. Antibiotics are given for 6 weeks initially as IV then oral.
  - Monitor temperature, swelling, pain, WBC and joint mobility
- Surgical:
  - Open drainage of abscess if antibiotics fail or signs of abscess appear.
  - After surgical drainage, wound is left open to heal by secondary intention

CHORNIC OSTEOMYELITIS

- This is often seen in lower extremities of a diabetic patient.

PATHOPHYSIOLOGY

- Usually an end result of untreated or treatment failed acute osteomyelitis. Occasionally it can occur due to trauma or surgery.
- The cause is usually polymicrobial (hence difficult to eradicate).
- Untreated acute osteomyelitis results in a cavity walled off by an involucrum (new bone) containing granulation tissue, sequestrum (dead bone) and bacterium.
- Drainage of pus into surrounding soft tissue and skin occurs via sinus tracts. Persistent drainage can lead to carcinoma.
- Bone fragments and exudates are usually unreachable by antibiotics.
- It results in severely deformed bones and pathological fractures.

CLINICAL FEATURES

- Persistent drainage following an episode of acute osteomyelitis or onset of inflammation and cellulitis following an open fracture.
- Fever, pain, mild systemic symptoms, tenderness
- Easy to diagnose when drainage is present and X-ray shows bone destruction and deformity. In cases with abscess of drainage, radionuclide imaging studies are very helpful.
- Radiological findings:
  - Areas of radiolucency within an irregular sclerotic bone
  - Irregular areas of destruction present. Often, periosteal thickening can be seen.

DIFFERENTIAL DIAGNOSIS

- Acute suppurative arthritis
- Rheumatic fever: examine synovial fluid
- Cellulitis: absence of soft tissue swelling on radiographs

MANAGEMENT

- Varies from open drainage of abscess or sequestrectomy to amputation
- Most effective: extensive debridement of all necrotic and granulation tissue along with reconstruction of bone and soft tissue defects with concomitant antibiotics (usually given for 12 weeks).
- Excellent adjunct: temporary placement of polymethylmethacrylate beads in the wound for a depot administration of antibiotics.

COMPLICATIONS

- Soft tissue abscess
- Septic arthritis due to extension to adjacent joint
- Metastatic infections to other areas
- Pathologic fractures
- If significant spinal involvement, paraplegia

BONE TUMORS

- Occur due to uncontrolled cellular proliferation of a single clone of cells whose regulatory mechanisms are defective.
- Benign tumors are 200 times more likely than malignant ones.
- History and physical examination are crucial in diagnosis as they will reveal the duration of the mass, onset of pain, other associated symptoms and the chronological sequence of these symptoms.
- The mass should be noted for size, location, consistency, mobility, tenderness, local temperature, and change with position. Also note any muscular atrophy.
- Radiography is important, it is important to note that bone tumor should not be diagnosed without an X-ray.
- Bone reacts to a benign or malignant tumor by bone production or destruction. An X-ray appearance shows a combination of bone production and bone destruction.
- 3 patterns of X-ray appearance:
  - Permeative: rapidly spreading intramedullary tumor, tumor replaces marrow and fat.
  - Moth eaten: a poorly circumscribed, slow-growing malignant tumor
  - Geographic: well circumscribed slow-growing tumor, therefore bone has time to react and results in sclerotic margins
- Bone tumors may be:
  - Primary
    - Benign: Osteoid osteoma, osteoblastoma, osteochondroma, enchondroma, and Giant cell tumor
    - Malignant: Osteosarcoma, chondrosarcoma, Ewing's tumor and multiple myeloma
  - Secondary: more common, always malignant.

BENIGN BONE TUMORS

MALIGNANT BONE TUMORS

- These include:
  - Osteosarcoma
  - Chondrosarcoma
  - Ewing's tumor
  - Multiple myeloma

OSTEOSARCOMA

- This is a tumor made of a malignant spindle cells in the stroma producing osteoid.
- It is overall the commonest primary bone tumor.
- Peak incidence is between the age of 10-30 years and is more common in males than females.
- There are many subtypes of osteoid forming sarcomas.
- Most common sites:
  - Around the knee (distal femur, proximal tibia)
  - Proximal humerus
  - Mandible (rare)
- Note: Osteosarcoma can occur secondary to Paget's disease, this is seen in elderly patients alongside patient with prior exposure to radiotherapy.

CLINICAL FEATURES

- Pain associated with a tender mass
- Dilated veins may be visible on the skin over the mass
- Pathological fractures
- Constitutional symptoms e.g. fever, weight loss
- Features of metastasis to the lungs

INVESTIGATIONS

- Imaging:
  - Bone X-ray
    - Often seen first on X-ray but diagnosis usually involves CT or MRI
    - X-ray shows a poorly defined lesion in the metaphysis with areas of bone destruction and formation.
    - Codman's triangle: due to new bone formation under the corners of the raised periosteum
    - Sun-ray appearance: occurs when bone spicules are formed perpendicular to the surface of the bone

  - Technitium bone scan
  - CT or MRI
  - CXR, CT chest, whole body MRI or FDG-PET scan for staging of cancer
- Blood investigations:
  - Full blood count: shows cytopenia with bone marrow involvement
  - ALP: increased in osteosarcoma (this is a good indicator for prognosis)
  - LDH: increased
- Biopsy for histology: Spindle-shaped tumor cells with odd, hyperchromatic nuclei showing a high mitotic rate. Giant cells may be present.

TREATMENT

- High dose methotrexate, doxorubicin, cisplastin and ifosfamide along with surgical intervention (usually amputation)
- Tumors hematogenously metastasize to the lung.
- Surgery plus chemotherapy gives a 5-year survival of about 60%. Prognosis is better if tumor is in a small bone.

ENDOCRINE

THYROID GLAND

THYROID DEVELOPMENT

- The thyroid gland is the first endocrine gland to develop at the 24th day of gestation.
- It develops from a median down growth of a column of cells from the pharyngeal floor between the first and second pharyngeal pouches (subsequently marked by the foramen caecum of the tongue).
- The canalized column becomes the thyroglossal duct which is displaces forward by the developing hyoid bone and then below the hyoid (C3), lies slightly to one side, more commonly to the left.
- The duct bifurcates to form the thyroid lobes and a portion of the duct forms the pyramidal lobe.

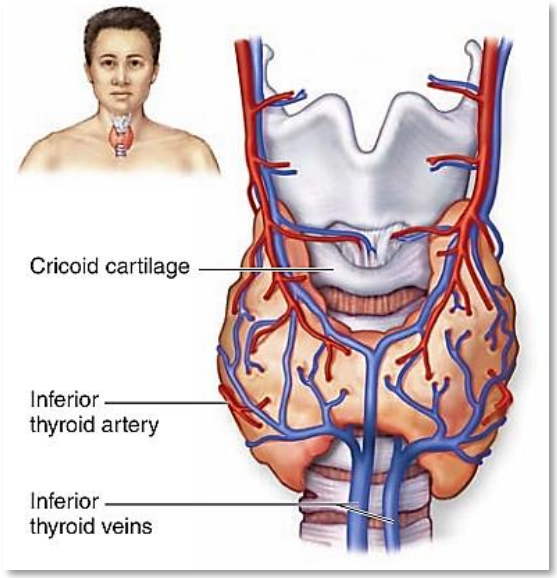


Figure 197: The thyroid gland

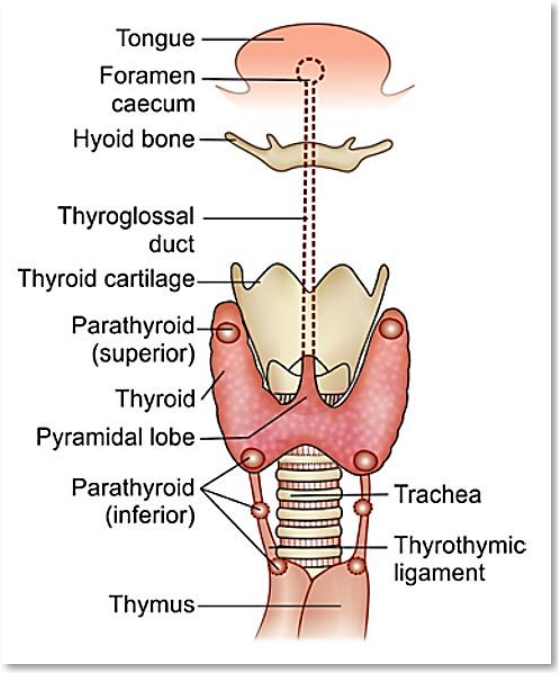


Figure 198: Development of the thyroid gland

ANATOMY

- The gland weighs about 15-20g and is located in the anterior triangle of the neck (muscular triangle).
- It originates in early embryonic life from the foregut endoderm.
- The parafollicular cell or C-cells are derived from the neural crest (ectoderm).
- It consists of a right and left lobe that are connected by an isthmus which extends from the 2nd to the 4th tracheal rings.
- Each lobe is 5 x 3 x 1.5 cm in size and extends from the middle of the thyroid cartilage to the 6th tracheal ring.
- The thyroid also has a pyramidal lobe which is an upward extension as fibrous strands or muscular strands from the junction of the isthmus and left lateral lobe.
- The gland is covered by pretracheal fascia which is attached to the larynx thus causing the thyroid to move on swallowing. This important clinical finding helps to discriminate thyroid swellings from other swellings in the neck.
- Berry's ligament is a strong condensed vascular connective tissue between the lateral lobe and cricoid cartilage on each side.
- Blood supply:
  - Arterial
    - Superior thyroid artery: 1st branch of external carotid artery, enters at the superior pole of the gland and bifurcates into a larger anterior superficial branch and a smaller posterior branch.
    - Inferior thyroid artery: a branch of thyrocervical trunk of subclavian artery. It passes behind the carotid sheath running medially reaching the posterolateral aspect of the gland.
    - Thyroidea ima artery: a branch of aorta or brachiocephalic artery enters the isthmus or lower pole of one of the lateral lobes (10%).
    - Tracheal and esophageal branches serve blood supply to retained thyroid gland after thyroidectomy.
  - Venous
    - Superior thyroid vein: drains in the internal jugular.
    - Middle thyroid vein: short and drains into the internal jugular. It is first to be ligated in thyroidectomy.
    - Inferior thyroid veins are many in number. Drain into brachiocephalic vein.

- Kocher's vein may be present which drains lower or middle thyroid.

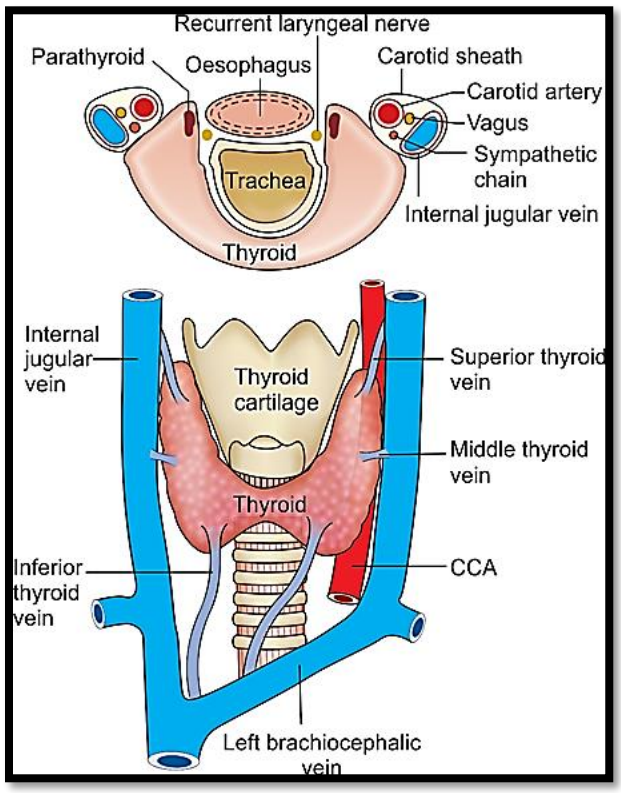


Figure 199: Venous drainage of the thyroid

- Lymphatic drainage
  - Primary:
    - Tracheao-esophageal nodes.
    - Pre-laryngeal nodes (Delphian nodes). Formerly purpose of these lymph nodes were uncertain hence the name.
      - Delphi is a place in Greece where Pythia, snake women after sulphurous fume inhalation uttered meaningless jargon purpose of it was unclear.
    - Mediastinal nodes.
  - Secondary:
    - Deep cervical nodes
    - Supraclavicular nodes
    - Occipital nodes

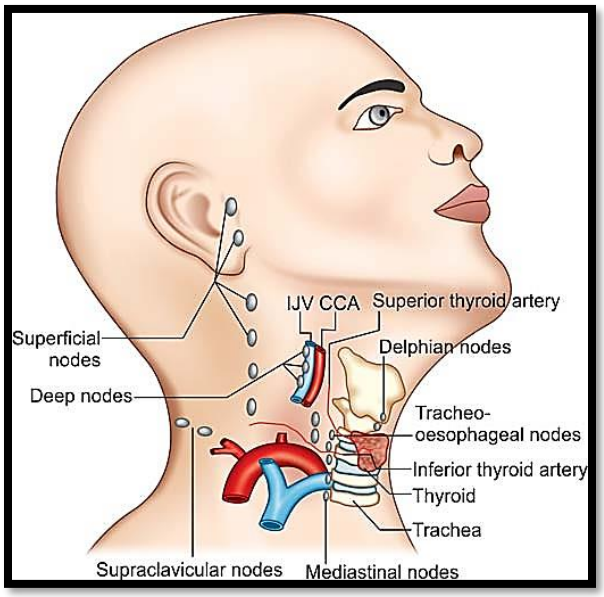


Figure 200: Lymphatic drainage of the thyroid

- Important Relations
  - Recurrent laryngeal nerve lies in the tracheooesophageal groove in relation to Berry's ligament. It supplies the intrinsic muscles of the larynx and vocal cords
  - Superior laryngeal nerve which gives a branch, external laryngeal nerve supplies cricothyroid muscle (A tensor/adductor of vocal cords). It accompanies superior thyroid artery.

- Parathyroid glands- four in number, 2 on each side embedded in thyroid.

PHYSIOLOGY

- Thyroid gland contains 2 secretory cells:
  - Follicular cells: secrete thyroxine (T4) and tri-iodothyronine (T3)
  - Parafollicular cells ("C" cells)- secrete calcitonin
- 90% of body iodide uptake is in the thyroid gland, whose uptake into the follicular cells is regulated by TSH and follicular iodide content.
- Iodothyronins (MIT, DIT) are formed in follicular cells by coupling of inorganic iodide with tyrosine under the control of the enzyme thyroid peroxidase.
- These are biologically inert molecules. T4 is formed by coupling 2 DIT molecules and T3 by coupling 1 MIT and 1 DIT molecule. Both are bound to thyroglobin which is the primary component of colloid matrix.
- The hypothalamus-pituitary-thyroid axis regulates thyroid hormone production and releases in a classic feedback system.
- TRH is a regulatory hormone from hypothalamus and TSH is a regulatory hormone from the anterior pituitary.

ASSESSMENT OF A PATIENT WITH A THYROID SWELLING

HISTORY OF A PATIENT WITH A THYROID SWELLING

- When was the swelling first noticed?
- Has it changed over time? Is it growing or the same size?
- Does it cause pain?
- Any of the associated symptoms:
  - Dyspnea
  - Dysphagia
  - Dysphonia and hoarseness of the voice
- Signs and Symptoms of hyper/hypothyroidism
- Medical and surgical history

EXAMINTION OF THE THYROID GLAND

- Greet the patient, explain the examination to the patient and gain consent.
- Position the patient to sit with both hands down on their side.
- The examiner should be standing in front and looking at the whole patient.
- Do not forget to perform a General examination.

INSPECTION

- Comment on the general condition of the patient: distress, restless, sluggish.
- Comment on appearance of hair and their dressing: scanty, warm clothes
- Comment on the eyes- opthalmoplagia, proptosis (do this behind the patient), chemosis (edema of the conjunctiva), lid lag/ retraction.
- Ask the patient to swallow some water- check for movement.
- Ask the patient to protrude their tongue- check for movement.
- Ask the patient to extend their arms and place a paper on their hands to check for tremors.
- Comment on obvious swelling on the neck- size, site, shape.

PALPATION

- Feel the hands for sweatiness
- Feel the pulse- irregularly irregular, bradycardia
- Check blood pressure and palpate for axillary and cervical lymphadenopathy
- Go behind the patient
  - ➢ Palpate the thyroid gland with the fingers using the thumb to slightly flex the neck anteriorly
  - ➢ Push on one side and use the other hand to feel.
  - ➢ Comment on the consistency, mobility, extent
  - ➢ Palpate the supraclavicular lymph nodes.

AUSCULTATION

- Auscultate for bruits over the gland
- Auscultate for heart rate and sound
- Auscultate for breath sounds

- Thank your patient for cooperation.

- Summarize your findings and offer a diagnosis.
  - ➢ Differentials:
    - ○ Thyroglossal cyst
    - ○ Thymoma
    - ○ Lymph node enlargement
    - ○ Lymphoma
    - ○ Lipoma
    - ○ Thyroid cyst
    - ○ Anterior cervical abscess
    - ○ Pharyngeal pouch

INVESTIGATIONS

1. Blood investigations
   - ➢ Serum T3 (Serum tri-iodothyronine), T4 (thyroxine) and TSH (thyroid stimulating hormone)
   - ➢ Free T3 and T4.
2. Imaging
   - ➢ Neck X-ray: AP view- tracheal deviation, Gland calcification- bleed easily
   - ➢ Ultrasound of the thyroid: cyst or solid mass.
   - ➢ Chest X-ray: tracheal deviation, lung mets, retrosternal shadow

- ECG- arrhythmias and ECHO
- Radioisotope I^{123} scan: shows either cold, hot or warm nodule.
- CT scan
- MRI

3. Fine needle aspiration cytology: shows benign or malignant cells
4. Indirect laryngoscopy: check for vocal cords (recurrent laryngeal)
5. Baseline:
   - Urea and electrolytes
   - Creatinine
   - Liver function tests
   - Full blood count

THYROGLOSSAL CYST

- The tract left behind (the thyroglossal tract) usually closes, although a portion of the epithelial lining may persist and form a cyst (thyrogossal cyst) usually in the late adolescent.

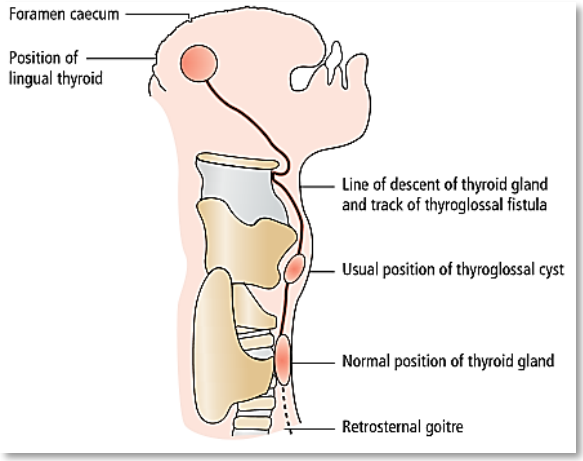


Figure 201: Thyroglossal tract

- The connection to the tongue means that the cyst will move on protrusion of the tongue.



Figure 202: Patient with a thyroglossal cyst

- The cyst may become infected occasionally leading to a cutaneous fistula (thyroglossal fistula).
- The causes of midline swellings in the neck:
  - Thyroid (most common): moves on swallowing
  - Thyroglossal cyst: moves on swallowing and protrusion of the tongue
  - Dermoid cyst (embryological skin cyst) does not move on swallowing or protrusion of the tone.
- The causes of midline swellings of the neck can therefore be differentiated by first asking the patient to swallow and then to stick out the tongue.

GOITER

- A goiter is an enlargement of the thyroid gland.
- The normal thyroid gland is impalpable.
- Goiters are described by:
  - Toxicity:
    - Hyperthyroid (Toxic)
    - Euthyroid
    - Hypothyroid (non-toxic)
  - Nodularity
    - Solitary nodule
    - Multinodular
  - Neoplastic
    - Benign
    - Malignant
- Diagnosis must have all 3 entities.

CLASSIFICATION OF THYROID SWELLINGS

- To remember the classification of thyroid conditions, remember "S.T.I.N"
- Goiters are classified under:
  - Simple (nontoxic)
  - Toxic
  - Inflammatory (thyroiditis)
  - Neoplastic

SIMPLE NONTOXIC GOITRE (EUTHYROID)

- Simple Diffuse hyperplastic: physiological, pubertal, pregnancy
- Colloid goiter
- Non-toxic Multinodular goiter
- Solitary non-toxic nodule
- Recurrent non-toxic nodule

TOXIC (HYPERTHYROID)

- Diffuse toxic hyperplasia (primary): Grave's disease
- Toxic Multinodular goiter (Secondary)- Plummer's disease
- Toxic adenoma (solitary toxic nodule)
- Recurrent toxicosis

INFLAMMATORY

- Autoimmune: Chronic lymphocytic thyroiditis (Hashimoto's disease)
- Subacute Granulomatous (De Quervain's) thyroiditis
- Fibrosing: Riedel's thyroiditis
- Infective: Acute (bacterial thyroiditis, viral thyroiditis, 'sub-acute thyroiditis') and chronic (Tuberculous, syphilitic)
- Other: Amyloid

NEOPLASTIC

- Benign- adenomas (follicular, Hurthle cell)
- Malignant
  - Primary
    - Papillary carcinoma
    - Follicular carcinoma
    - Medullary carcinoma
    - Anaplastic carcinoma
    - Lymphoma
  - Secondary: metastatic deposits

SOLITARY THYROID NODULE

- This is a common conditions affecting 5% of the female population but only 5% of solitary thyroid nodules are malignant.
- Investigations are therefore needed to discriminate between a benign and malignant thyroid nodule, as usually surgery is not required for benign solitary thyroid nodules.
- Causes:
  - Thyroid cysts
  - Degenerative thyroid nodule
  - Benign follicular adenoma (including solitary toxic adenoma)
  - Differentiated thyroid carcinoma
- History:
  - Features suggestive of malignancy:
    - Previous history of neck irradiation e.g. as a child
    - Hoarseness (potentially caused by infiltration of the recurrent laryngeal nerve)
    - Family history of papillary thyroid carcinoma)
  - Hyperthyroid symptoms
  - Pressure symptoms
- Investigations: 2 most important investigations are TSH and FNA
  - Blood TSH, T3 and T4
  - Fine needle aspiration (FNA)
    - A 21 G needle attached to a syringe is flushed with saline, is passed several times through the nodule while suction is maintained on the syringe.
    - The aspirated cells are then smeared on the slide and wet and/or dry fixed.
    - Results of cytology show benign cells, suspicious cell, malignant cells or the specimen is inadequate and consists of red cells only (seen in about 10-20% of thyroid FNAs).
    - If FNA reports suspicious cells, then the nodule should be removed as 30% are malignant.
  - Thyroid ultrasound: distinguishes solid nodules and cysts and whether it is a dominant nodule within a multinodular goiter
  - Technetium 99m scan gives an idea of the function of the nodule.
    - If the nodule is hot (and if TSH is suppressed the nodule is a solitary toxic adenoma)
    - If the nodule is cold (Decreased uptake) there is a slight increased risk of malignancy (10%).

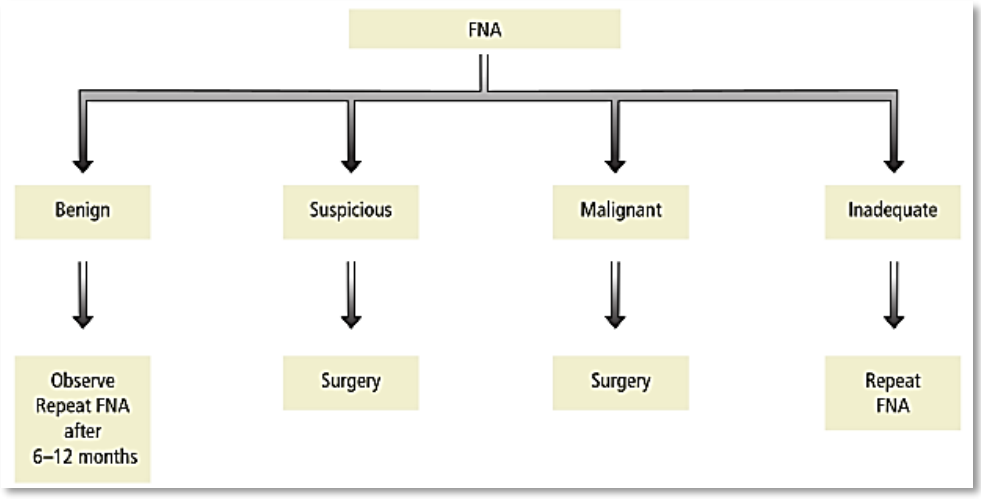


Figure 203: Treatment algorithm for FNA results

- Treatment:
  - A benign thyroid nodule does not require treatment unless symptomatic or cosmetically unacceptable
  - Suppression of TSH by thyroxine therapy is generally unhelpful in trying to reduce the size of a benign nodule.
  - FNA should be repeated after 1 year and if this remains benign then no further treatment is required.
  - Patients with suspicious nodules on FNA should undergo thyroid lobectomy as 30% of these are malignant
  - Frozen section is of little value at surgery as the degree of capsular invasion required to differentiate between benign follicular adenoma and carcinoma cannot be assed.
  - Patients with malignant nodules should undergo surgery.

INFLAMMATORY THYROID CONDITIONS

- Include:
  - Hashimoto's thyroiditis
  - Sub-acute De Quervain's Granulomatous thyroiditis
  - Riedel fibrosing thyroiditis

HASHIMOTO'S THYROIDITIS

- This is a chronic autoimmune disease characterized by immune destruction of the thyroid and hypothyroidism.
- It most commonly causes painless goiter (firm, rubbery, tender and smooth) in premenopausal females more than in males and has a peak age of 40 to 65 years.

- Usually bilateral but occasionally one lobe is involved.
- It is associated with HLA-DR5.
- It is associated with other autoimmune disease (SLE, rheumatoid arthritis, Sjogren syndrome)
- Clinically it presents with:
  - Initial inflammation that may cause transient hyperthyroidism (hashitoxicosis)
  - Hyperplasia causes hyperthyroid (Hashitoxicosis), as destruction progresses the patient is Euthyroid at a point but eventually Fibrosis leads to hypothyroid.
  - Signs of hypothyroidism due to autoimmune destruction of thyroid tissue. (decreased T4 and increased TSH)
  - Hepatosplenomegaly
- Pathology:
  - Gross: the gland appears pale and enlarge.
  - Microscopic:
    - Chronic inflammation: Lymphocytic inflammation with germinal centers (increased risk of Non-hodgkin B-cell lymphoma) and epithelial "Hurthle cell" changes.
    - Hurthle cell changes: some cells around follicles become more pink.
- Antithyroglobulin, antimicrosomal, and anticolloid antibodies are present (however these do not mediate the disease)
- Investigations
  - FNAC
  - Serum T3, T4, TSH
  - Thyroid antibodies assay
  - ESR is very high (over 90mm/hour)
- Treatment:
  - L-thyroxine therapy.
  - Steroid therapy often is helpful.
  - If goiter is large and causing discomfort, then subtotal thyroidectomy is done.

SUBACUTE DE QUERVAIN GRANULOMATOUS THYROIDITIS

- This is the second most common form of thyroiditis.
- It affects females more than males and has peak age 30 to 50 years.
- Granulomatous thyroiditis follows a viral infection (mumps or coxsackie virus).
- It presents as a tender, firm, enlarged thyroid gland which may be associated with transient hyperthyroidism.
- Microscopically there is granulomatous inflammation.
- The disease typically follows a self-limited course (does not progress to hypothyroidism).
- FNAC is useful

- Prednisolone 20mg for 7 days helps.

RIEDEL FIBROSING THYROIDITIS

- This is a rare disease of unknown etiology.
- It is characterized by destruction of the thyroid gland by dense fibrosis and fibrosis of surrounding structures (Trachea and esophagus).
  - There is chronic inflammation with extensive fibrosis of the thyroid gland.
- Females are affected more frequently than males, with most patients being middle-aged.
- It presents as hypothyroidism with 'hard wood', non-tender thyroid gland.
- The condition may clinically mimic carcinoma and present with stridor, dyspnea or dysphagia the only difference is anaplastic carcinoma is seen in the elderly while Riedel fibrosing thyroiditis is seen in the middle-aged.
- Riedel thyroiditis is associated with retroperitoneal and mediastinal fibrosis.
- Investigations
  - T3 and T4 may be low due to hypothyroidism.
  - Radioisotope scan will not show any uptake
  - FNAC to rule out carcinoma (anaplastic)
- Treatment:
  - Isthmectomy is done to relived compression on the airway.
  - They require L-thyroxine replacement later as hypothyroidism is common
  - High dose of steroid is often used.
  - Thyroidectomy is not necessary

THYROID NEOPLASMS

- The thyroid contains both benign and malignant tumors.
- Benign tumors include:
  - Adenomas (follicular adenoma, Hurthle cell adenoma)
- Malignant tumors include:
  - Papillary carcinoma (60%)
  - Follicular carcinoma (20%)
  - Anaplastic carcinoma (10%)
  - Medullary carcinoma (5%)
  - Malignant lymphoma (<5%)- not a primary thyroid tumor

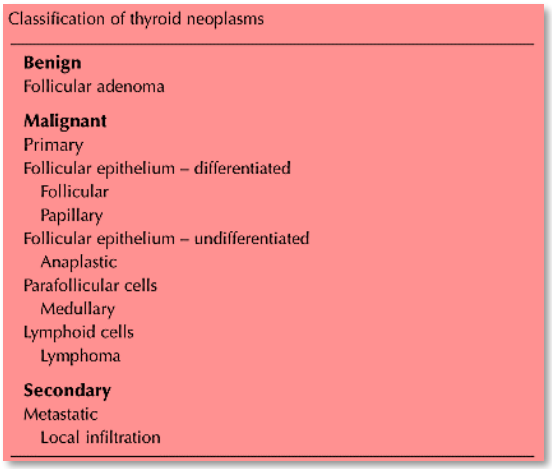


Figure 204: Classification of thyroid neoplasms

- Secondaries in thyroid are rare but can come from colon, kidney, melanoma, breast.
- Thyroid cancers are common in females (3:1)
- Papillary carcinoma spreads through lymphatics, follicular through blood and anaplastic through both lymphatics and blood.
- Thyroid neoplasms typically present as distinct solitary nodules.
- Thyroid nodules are more likely to be benign than malignant.
- Iodine radioactive (I^{123}) uptake studies are of importance:
  - Increased uptake: Graves' disease or nodular goiter
  - Decreased uptake: adenoma and carcinoma (this warrants a biopsy by FNAC)

BENIGN TUMORS

- Follicular adenomas present as clinically solitary nodules and the distinction between a follicular carcinoma and an adenoma can only be made by histological examination.
- Follicular adenoma is characterized by benign proliferation of follicles surrounded by a fibrous capsule.
  - Follicles are usually non-functional however can secrete thyroid hormone.
- In adenoma there is no invasion of the capsule or of pericapsular blood vessels i.e. no capsular or vascular invasion.
- They can be functional (releasing hormones-'Hot nodules') or nonfunctional ('Cold nodule')
- Treatment is therefore by wide excision I.e. lobectomy. The remaining thyroid tissue is normal so that prolonged follow-up is unnecessary.
- It is doubtful if there is such an entity as a papillary adenoma and all papillary tumors should be considered as malignant even if encapsulated.

MALIGNANT TUMORS

- The vast majority of primary malignancies are carcinomas derived from follicular cells.
- Dunhill classified then into:
  - Histologically differentiated
    - Papillary
    - Follicular
  - Histologically undifferentiated

- Anaplastic
- Medullary
- Lymphoma (develop sometimes in autoimmune thyroiditis)
- Secondaries to the thyroid: secondary disease should be considered when there is a history of malignancy, particularly kidney and breast cancer and when the cytology of a thyroid swelling is atypical.

CLINICAL FEATURES OF THYROID CANCER

- Clinical features/ effects may be due to
  - Primary tumor:
    - Thyroid swelling:
      - A differentiated carcinoma may be suspiciously firm and irregular but often indistinguishable from a benign swelling.
      - Small papillary tumors may be impalpable even in the presence of lymphatic metastases.
      - Anaplastic growths are usually hard, irregular and infiltrating.
    - Enlarged cervical lymph nodes.
    - Dilated veins over goiter
    - Pain referred to the ear in infiltrative growths.
    - Recurrent laryngeal nerve paralysis (suggestive of locally advanced disease)
    - Pressure/infiltrative symptoms:
      - Dysphagia from compression or infiltration of esophagus
      - Dyspnea from compression of trachea or mets to the lungs
      - Dysphonia and hoarseness from external laryngeal involvement.
    - Symptoms due to functional tumors (those that produce hormones)
  - Secondary metastasis of tumor: mets to bones (flat bones of the skull, long bones, vertebrae)- Pathological fractures
  - Constitutional symptoms: malaise, weight loss, anorexia, fatigue, malaise, anemia

Tumors with mets to the bone
- Thyroid
- Breast
- Lung
- Kidney
- Prostate

HISTOLOGICAL ENTITIES

PAPILLARY CARCINOMA OF THYROID

- This is the most common type of thyroid carcinoma (60%).
- It is a differentiated tumor that contains a mixture of papillary (finger-like structures) and colloid-

filled follicles and in some follicular structure predominates.

- Note: Presence of any papillary structure or characteristic cytology means that the tumor will behave in a predictable fashion as a papillary carcinoma.
- Common in females and younger age group
- Etiology:
  - Radiation either external or radioactive iodine therapy.
  - TSH levels in the blood of these patients are high and so it is called as hormone dependent tumor.
  - The incidence of follicular carcinoma is high in endemic goitrous areas, possibly as a result of TSH stimulation.
- Clinically it presents as:
  - Soft or hard or firm, solid or cystic, solitary or multinodular thyroid swelling.
  - Multiple foci may occur in the same lobe as the primary or less commonly in both lobes.
  - Compression features are uncommon in papillary carcinoma of thyroid.
  - Often discrete lymph nodes in the neck (40%) are palpable.
  - May present with secondaries in neck lymph nodes with occult primary. It metastasizes to the regional lymph nodes.

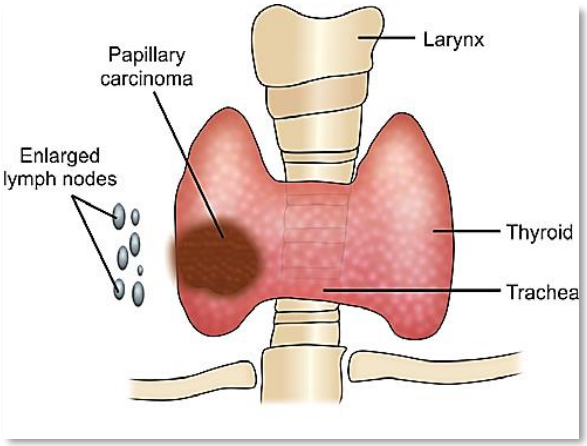


Figure 205: Papillary carcinoma

- Gross morphology: it can be soft, firm, hard, cystic. It can be solitary or multinodular. It contains brownish black fluid.
- Microscopy:
  - Shows cystic spaces, papillary projections with psammoma bodies (concentric layers of calcification), malignant cells with 'Orphan Annie eye' nuclei (intranuclear cytoplasmic inclusions)
  - Orphan Annie eye nuclei are identified in histology (Paraffin section of formalin tissue). It is not seen in FNAC. Orphan Annie is strip cartoon character with empty circle eyes.
  - Tall cell type of papillary carcinoma (10% of papillary carcinomas) is very aggressive type seen in elderly, 30% show capsular and vascular invasion with 25-30% 5-year survival rate.

- Columnar type is seen only in males with near 100% mortality in 5 years.
- Papillary carcinomas are rarely encapsulated.

- Papillary tumors are slow progressive and less aggressive.
- They generally have an excellent prognosis (90% 20-year survival rate)
- They are commonly multicentric and spread within the gland through intrathyroid lymphatics to other lobes, cones out of the capsule and spreads to cervical lymph nodes.
- Usually there is no blood spread.
- Extrthyroidal disease- invasion into thyroid capsule can cause blood born secondaries occasionally.
- Diagnosis:
  - TSH level in the blood is higher
  - Plain X-ray neck shows fine calcification whereas nodular goiter shows coarse- ring/rim calcification
  - U/S neck or CT scan neck (better) to identify non-palpable nodes in neck.
  - Radioisotope scan shows cold nodule
  - FNAC of thyroid nodule and lymph node

FOLLICULAR CARCINOMA

- Follicular carcinoma of the thyroid occurs in an older age group (40-50 years).
- It is slow-growing and eventually spreads by the hematogenous route.
- Most present as a solitary thyroid nodule. Multiple foci are rarely seen.
- Histology: follicular hyperplasia.
- The prognosis of the tumor depends on the degree of capsular and vascular invasion.
- Patients with minimal capsular invasion can be treated by thyroid lobectomy and TSH suppression with thyroxine (differentiated thyroid carcinomas are TSH responsive and it is therefore important to keep TSH suppressed by administering thyroxine).
- If there is significant capsular or vascular invasion the patient is best served by total thyroidectomy and subsequent radioiodine and thyroxine therapy.

Grading system for differentiated thyroid carcinoma.

Good prognosis
Female < 45 years
Male < 40 years
Tumour < 5 cm
Minimally invasive follicular carcinoma
Poor prognosis
Female > 45 years
Male > 40 years
Tumour > 5 cm
Any patient with distant metastasis
Extrathyroidal invasion

- Hurthle cell tumors are a variant of follicular neoplasm in which oxyphil cells predominate

histology. It carries poorer prognosis.

MEDULLARY CARCINOMA

- It is uncommon (5%) type of thyroid malignancy.
- It arises from the parafollicular 'C' cells (that produce calcitonin) which is derived from the neural crest. C cells are more in upper pole of the thyroid gland.
- FNAC shows 'amyloid stroma' (due to deposition of calcitonin). Amyloid can be visualized using Congo Red pigment.
- In these patients blood levels of calcitonin both basal as well as that following calcium or pentagastrin stimulation is high, a very useful tumor marker.
- Tumor also secretes serotonin (5-HT), prostaglandin, ACTH and vasoactive intestinal polypeptide (VIP).
- Diarrhea is a feature seen in 30% of cases and may be due to 5-hydroxytryptamine or prostaglandins produced by the tumor cells.
- It spreads mainly to lymph nodes (60%).
- It usually presents as a thyroid nodule in a sporadic form (70% of cases) or can be familial or in association with multiple endocrine neoplasia (MEN).
- Medullary carcinoma may be associated with MEN 2A and MEN 2B
  - MEN 2A: medullary carcinoma of thyroid + Pheochromocytoma + parathyroid adenoma
  - MEN 2B: medullary carcinoma of thyroid + pheochromocytoma + ganglioneuroma of oral mucosa
- Medullary carcinoma is associated with mutations in RET oncogene.
- Detection of RET mutation warrants prophylactic thyroidectomy.
- Clinical features:
  - Thyroid swelling often with enlargement of neck lymph nodes
  - Diarrhea, flushing (30%)
  - Hypertension, pheochromocytomas and mucosal neuromas with MEN 2B syndrome.
  - Paraneoplastic syndrome like Cushing's and carcinoids
  - Hypocalcemia (Chovstek sign- tapping over facial nerve in front of tragus causes twitching of facial muscles and Trousseau sign- carpal spasms induced by inflating a sphygmomanometer cuff above systolic pressure)
    - Paresthesia, muscle cramps, carpopedal spasms
- Patients with medullary thyroid carcinoma are best treated by total

thyroidectomy with central lymph node clearance.

- Post-operatively patients are given thyroxine replacement (but not TSH suppression), postoperatively calcitonin measurement is a useful tumor cell marker and can be used as a method of follow-up.
- The prognosis is approximately 60% 5-year survival.

ANAPLASTIC CARCINOMA

- This is an undifferentiated very aggressive carcinoma (1%) occurs in elderly females.
- It is a very aggressive tumor of short duration, presents with a swelling in thyroid region which is rapidly progressive causing:
  - Stridor and hoarseness of voice due to tracheal obstruction
  - Dysphagia
  - Fixity to the skin
- It spreads by blood and lymphatics.
- It is difficult to differentiate from Riedel thyroiditis.
  - Positive Berry's sign- involvement of carotid sheath leads to absence of carotid pulsation.
- Swelling is hard, with involvement of isthmus and lateral lobes.
- FNAC is diagnostic.
- Tracheostomy and isthmectomy has got a role to relieve respiratory obstruction temporarily.
- Treatment is external beam radiotherapy and/or chemotherapy, as usually thyroidectomy is not possible.
- Adriamycin as chemotherapy
- Prognosis is poor (patients die in 12 months). Life span is counted in few weeks to months only.

THYROID LYMPHOMA

- This is a rare tumor usually present in the elderly female with a background history of autoimmune (Hashimoto's thyroiditis) as a rapidly growing thyroid nodule.
- Presents with a large rubbery goiter.
- It may be diagnosed by FNA or Trucut biopsy.
- As for all lymphomas it should be staged with a bone marrow aspirate and CT scan of the chest and abdomen.
- If confined to the thyroid alone it may be treated by thyroid lobectomy with subsequent adjuvant radiotherapy and chemotherapy otherwise it is treated by chemoradiation alone.

STAGING: TMN STAGING

Tumour–node–metastasis staging (5th edition) of thyroid cancer

Tumour

TX	Primary cannot be assessed
T0	No evidence of primary
T1	Limited to thyroid, 1 cm or less
T2	Limited to thyroid, > 1 cm but < 4 cm
T3	Limited to thyroid, > 4 cm
T4	Extending beyond capsule, any size

Nodes

NX	Cannot be assessed
N0	No regional node metastases
N1	Regional node metastases

Metastases

MX	Cannot be assessed
M0	No metastases
M1	Metastases present

Stage	Under 45 years	Over 45 years
I	Any T, any N, M0	T1, N0, M0
II	Any T, any N, M1	T2, N0, M0 or T3, N0, M0
III		T4, N0, M0 or any T, N1, M0
IV		Any T, any N, M1

Note the effect of age on stage; only patients older than 45 years can have stage III or IV disease.

MANAGEMENT

- Contemporary guidelines increasingly recommend total thyroidectomy for virtually all cancer followed by thyroid hormone replacement.
- Conservative approach reserves total thyroidectomy for specific indication namely those in which there is a preoperative diagnosis of high-risk cancer, bilateral disease or when there is a clear indication for postoperative radiotherapy.
- "Small cancers" confined to one lobe can be managed by lobectomy and TSH suppression. Unfortunately, there is no general consensus on the definition of small cancer with arbitrary limits of between 1 and 4cm in use.
- Indications for thyroidectomy:
  - Malignant goiter

- Enlarged thyroid with pressure symptoms
- Thyrotoxic goiter unresponsive to medical treatment
- Cosmesis

- Complications of thyroidectomy:
  - Early
    - Recurrent laryngeal nerve damage (may be unilateral or bilateral)- leads to vocal cord collapse and upper airway obstruction
    - Laryngeal edema
    - Hemorrhage and Hematoma as a result of poor hemostasis- leads to upper airway obstruction
    - Tracheomalacia as seen with a large goiter that erodes the trachea that collapses after thyroidectomy resulting in upper airway obstruction
    - Upper airway obstruction
    - Hypocalcemia (removed parathyroid glands)- low calcium, Chvostek's sign and Trousseau sign. Treat with calcium gluconate
    - Tracheal perforation
    - Thyroid storm
      - Increased T3 and T4 levels with symptoms (Ill, Toxic looking, tachycardia, arrythmias, heart failure, pyrexia, tachypnea, sweating profusely, convulsions, confusion, coma, diarrhea, vomiting and acute abdomen)
      - Due to operation performed on a patient that is not rendered euthyroid.
      - Management
        - Admit patient to ICU (quiet room)
        - Temperature control (Fan, Cold compress/sponging, antipyretics)
        - Suppress T3, T4 and TSH production:
          - Beta blocker and Thioamide (Propylthiouracil 1st line) plus: Iodine compounds (e.g. Lugol's solution) to block T4 and T3 release (Give this 1hour after thioamide as otherwise it may increase thyroid hormone synthesis), Glucocorticoids (hydrocortisone or dexamethasone- to reduce conversion of T4 to T3) and Bile acid sequestrants (e.g. Cholestyramine) to prevent T4 and T3 reabsorption in the gut
        - Intravenous fluid
      - Prevention: Render the patient euthyroid prior to surgery
  - Intermediate
    - Infections

- DVT
- Hypostatic pneumonia

➢ Late

- Hypothyroidism
- Keloid and hypertrophic scars

SUMMARY OF THYROID CANCER

	Papillary	Follicular	Medullary	Anaplastic
Frequency	Most common	Not common in populations with adequate iodine	Not common	Rarest but worst prognosis
Risk factors	Radiation	Dyshormonogenesis	MEN II in 30-40%	• Prior diagnosis of well differentiated thyroid tumor • Iodine defieciny
Age group	30-40	40-50	50-60	60-70
Clinical features	• Painless mass • Dysphagia • Dyspnea • Hoarseness • Euthyroid	• Painless mass • Rarely: hyperfunctional	• Painless mass • Palpable lymph node (15-20%) • Dysphonia • Dyspnea • Hoarseness	• Rapidly enlarging mass (large mass at presentation) • Neck pain • Dysphagia • Hard, fixed lymph node (50%)
Diagnosis	• FNAC • CT or MRI (to assess local invasion)	• FNAC • CT or MRI (to assess local invasion)	• FNAC • Presence of amyloid is diagnostic (Congo red) • Check for calcitonin	• FNAC
Metastasis	Lymphatics	Blood	• Lymphatic (local neck and mediastinal nodes)	• Blood and lymphatics • Aggressive local disease • 30-50% have synchronous

			• Local (into trachea and esophagus)	pulmonary mets at time of diagnosis
Treatment	• Minimal cancer (<1.5cm): limited lobectomy and isthmectomy • Other: Total or near-total thyroidectomy for cancer >1.5cm • For +LN, modified radical neck dissection • I-131 ablation or thyroid suppression (with thyroid hormone) for patients with residual thyroid tissue or LN mets	• Minimal cancer (<4cm): lobectomy and isthmectomy • Other: Total or near-total thyroidectomy for cancer >4cm • For + LN, modified radical neck dissection • I-131 ablation for patients with residual thyroid tissue or LN mets	• Sporadic (80%): total thyroidectomy • Familial (20%): total thyroidectomy and central neck node dissection • No value for I-131 ablation • Follow patients with calcitonin levels	• Debulking resection of thyroid gland and adjacent structures • External radiation therapy • Doxorubicin-based chemotherapy
Prognosis	Worse for older patients with distant mets. Presence of + LN not strongly correlated with overall survival	Worse for older patients with distant mets, tumor size >4cm, high tumor grade. Presence of + LN not strongly correlated with overall survival		Poor
Survival	10-year survival: 74-93%	10-year survival: 60-80%	10-year survival: 70-80%	Median survival: 4-5 months

HYPERTHYROIDISM

- Thyrotoxicosis is a symptom complex due to raised levels of thyroid hormones.
- It refers to biochemical and physiological manifestations (e.g. hyperthermia) of excessive thyroid hormone.
  - Thyrotoxicosis= Excessive thyroid hormone release in blood + Clinical manifestations
- Hyperthyroidism is the term used to refer to overproduction of the thyroid hormones.
- Hyperthyroidism is divided into:
  - Primary thyrotoxicosis (pathology is in the thyroid gland)
    - Puberty, pregnancy, emotion and infection are the precipitating factors for primary thyrotoxicosis.
  - Secondary thyrotoxicosis (pathology not in thyroid gland)

Primary thyrotoxicosis	*Secondary thyrotoxicosis*
1. Symptoms appear first, then thyroid swelling	1. Thyroid swelling appears first
2. Goitre is diffuse, smooth, firm or soft, both lobes are involved with thrill and bruit	2. Swelling is large nodular, obvious
3. Features are much more severe compared to that of secondary toxicosis of short duration	3. Symptoms appear after long time, which is less severe and slowly progressive compared to primary toxicosis
4. Eye signs and exophthalmos are common	4. Eye signs are not common
5. As it is an autoimmune disease, there may be hepato-splenomegaly	5. Cardiac features are more common
6. It occurs in younger age group	6. It occurs in adult and elderly individual
7. Entire gland is overactive	7. Internodular tissues are overactive
8. There is no preexisting goitre	8. Occurs in a long standing preexisting multinodular goiter
Histologically, there is hyperplasia of acini, lined by columnar epithelium, often containing vacuolated colloid scalloping with pseudopapillary formation	

Figure 206: Primary thyrotoxicosis vs Secondary thyrotoxicosis

TYPES

- Diffuse toxic goiter (Primary thyrotoxicosis)
  - Graves' disease
  - Basedow's disease
- Toxic multinodular goiter (Secondary thyrotoxicosis): Plummer disease
- Toxic nodule (Goetsch's disease)
- Other causes of thyrotoxicosis without hyperthyroidism include:
  - Ectopic function thyroid

- Struma ovarii
- Functioning metastatic follicular carcinoma
- Trophoblastic tumors
- Thyrotoxicosis factitia- drug induced due to intake of L-thyroxine more than normal

GRAVES DISEASE

- This is an autoimmune thyrotoxicosis that is caused by an antibody against the TSH receptor (TSH receptor antibody).
- Graves' disease has genetic predisposition and it affects most females (8:1).
- Due to the stimulating TSH receptor antibodies, the acinar of the thyroid undergo hypertrophy and hyperplasia with absence of normal colloid in the tall columnar epithelium (normal is flat epithelium with colloid). As the cells are empty they look vacuolated. Tissues are highly vascular.



Figure 207: Patient with exophthalmos

- Clinical features are those of thyrotoxicosis. It is also associated with vitiligo. Exophthalmos producing substance causes Graves ophthalmopathy (manifested by lower eyelid retraction, lid lag, periorbital edema, chemosis or if severe proptosis and exophthalmos).
- Diffuse goiter, thyrotoxicosis and autoimmune manifestations (infiltrative ophthalmopathy, dermopathy, myopathy) are essential components of Graves' disease. Thyroid stimulating antibodies produced against thyroid antigen in Graves' disease which is directed to TSH receptor acting as TSH receptor antibody. TSH receptor antibody is observed only in Graves' disease.
- Examination reveals a moderate firm goiter which in severe thyrotoxicosis may have a vascular thrill and bruit.
- Investigations:
  - Suppressed TSH with raised free T4 and/or T3
  - 90% of patients have raised TSH antibodies
  - 70% of patients will have raised thyroid peroxidase antibodies.
- Treatment:
  - Medical

- Thyroid uptake blocking drugs to render the patient euthyroid
  - Carbimazole
    - Important side-effects: neutropenia (which presents as sore throat), profuse diarrhea or hepatocellular failure (which initially presents as jaundice), skin rash, nausea and joint pain
  - Propylthiouracil
- Beta-blockers (Propranolol) can be used if the patient is symptomatic with sweating, tremor or tachycardia
- Control of thyrotoxicosis usually takes about 6 weeks but maintenance is required for at least 18 months before cessation of carbimazole. Alternatively, higher doses of carbimazole can be taken in conjunction with thyroxine for about 12 months (block and replace).
- If the patient relapses after this then definitive long term treatment is required either radioactive iodine or if inappropriate (e.g. young children at home) surgery can be performed.

➢ Surgical

- Patient has to be rendered euthyroid before surgery.
- If surgery is the preferred option, then previously a subtotal thyroidectomy was performed to render the patient 'euthyroid' however 10% of these patients developed recurrent thyrotoxicosis and 70% develop hypothyroidism in the long run.
- Total thyroidectomy and long term thyroxine postoperative can be done.

TOXIC MULTINODULAR GOITER

- While the majority of multinodular goiters are non-toxic, over a period the presence of large functioning nodules may render the patient hyperthyroid leading to toxic multinodular goiter (Plummer's disease).
- The large goiter may extend retrosternally and cause tracheal deviation and compression occasionally leading to stridor (a low-pitched crowding noise on inspiration caused by narrowing of the trachea).
- Examination will reveal multiple nodules either bilaterally or unilaterally.
- If there is a dominant nodule within the gland then this should be investigated as for solitary thyroid nodule as the risk of malignancy in this nodule is about 10%.
- Elevation of the patient's arms may lead to venous congestion and stridor if there is retrosternal extension of the goiter (Pemberton's sign). The position of the trachea should be checked for possible tracheal deviation.
- Thyroid ophthalmopathy is not associated with multinodular goiter.

- Investigations:
  - TSH: reduced if toxic multinodular goiter.
  - Fine-needle aspiration of dominant nodule, if present
  - Ultrasound may confirm multiple nodules
  - X-ray of thoracic inlet and CT will delineate the extent of retrosternal extension and the degree of tracheal deviation and compression
- Treatment:
  - Total thyroidectomy should be performed for non-toxic multinodular goiter if there is retrosternal extension of the goiter, tracheal compression or if the patient finds the goiter cosmetically unacceptable.
  - Toxic multinodular goiter: treat with carbimazole initially and then either total thyroidectomy or radioiodine depending on the suitability of the patient.

SOLITARY TOXIC ADENOMA

- Occasionally, a single thyroid adenoma may be active enough to render the patient hyperthyroid.
- The patient normally presents with a solitary thyroid nodule and should be investigated as such in order to exclude malignancy.
- In patients with solitary toxic adenoma, TSH is suppressed and a $^{99m}TcO_4$ thyroid isotope scan will show a solitary 'hot' nodule.
- Treatment is initially with carbimazole followed by either thyroid lobectomy or radioactive iodine depending on preference.

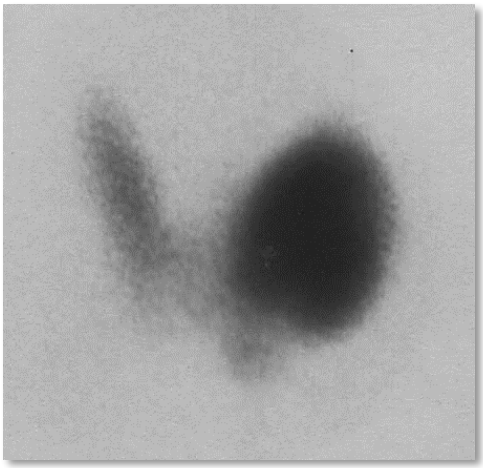


Figure 208: 'Hot' nodule on thyroid isotope scan

CLINICAL FEATURES OF THYROTOXICOSIS

- It is 8 times more common in females.
- Occurs in any age group.
  - Primary type is seen in younger group.
  - Secondary type is common in older age group.
- Note: Graves' disease often presents without any obvious thyroid swelling in the neck. Whenever there are unexplained behavioral problems, insomnia, myopathy, unexplained diarrhea or loss of weight, tachycardia, Graves' disease should be suspected and evaluated

SYMPTOMS

SYSTEM	SYMPTOMS
CENTRAL NERVOUS SYSTEM	• Irritability • Nervousness • Insomnia • Fine tremor (Due to diffuse irritability of grey matter) • Heightened emotions
CARDIOVASCULAR SYSTEM	• Palpitations (thyroid hormones increase expression of Beta 1 adrenergic receptors) • Shortness of breath at rest or on minimal exertion • Angina • Irregularity of heart rate (Arrhythmias) • Cardiac failure in the elderly • Undue fatigue and muscle weakness
GASTROINTESTINAL SYSTEM	• Weight loss in spite of increased appetite • Diarrhea (due to increased activity at ganglionic level) and malabsorption
GENITOURINARY SYSTEM	• Oligo or amenorrhea • Occasional urinary frequency
SKELETAL SYSTEM	• Increase in linear growth in children • Bone resorption and hypercalcemia • Decreased muscle mass with weakness
INTEGUMENTARY SYSTEM	• Hair loss, gynecomastia • Pruritus • Palmar erythema
METABOLIC SYSTEM	• Increase in basal metabolic rate (increase in the synthesis of sodium-potassium ATPase) • Hypocholesterolemia

	• Hyperglycemia (due gluconeogenesis and glycogenolysis)

- Sympathetic over activity causes dyspnea, palpation, heat intolerance, sweating, nervousness, increased appetite and decrease in weight.
- Because of the increased catabolism they have increased appetite, decreased weight and so also increased creatinine level which signifies myopathy (Due to more muscle catabolism).
- Fine tremor is due to diffuse irritability of grey matter.

SIGNS OF HYPERTHYROIDISM/ TOXICOSIS

EYE SIGNS IN TOXIC GOITRE

- Common in primary thyrotoxicosis.
- Lid lag, lid spasm can occur in secondary thyrotoxicosis also.
- Eye signs include:
  - Lid retraction: the upper eyelid is higher than normal and lower eyelid is in normal position with visible sclera adjacent to upper eyelid. It is due to sympathetic over activity causing spasm of involuntary smooth muscle of the levator palpebrae superioris. It is a sign of thyrotoxicosis not a sign of exophthalmos.
  - Lid Lag sign: it is inability of the upper eyelid to keep pace with the eyeball when it looks downwards to follow the examiners finger.
  - Staring gaze (absence of normal blinking)
  - Exophthalmos: this is proptosis of the eye, it is caused by infiltration of retrobulbar tissue with fluids and round cells with lid spasm of upper eyelid.
    - It is seen with Grave's disease and is believed that fibroblast have TSH receptors and in response to TSH like antibodies e.g. Grave's disease fibroblast produce mxyoid substances e.g. glycosaminoglycans (GAGs) which deposit in the retro-orbital tissues.

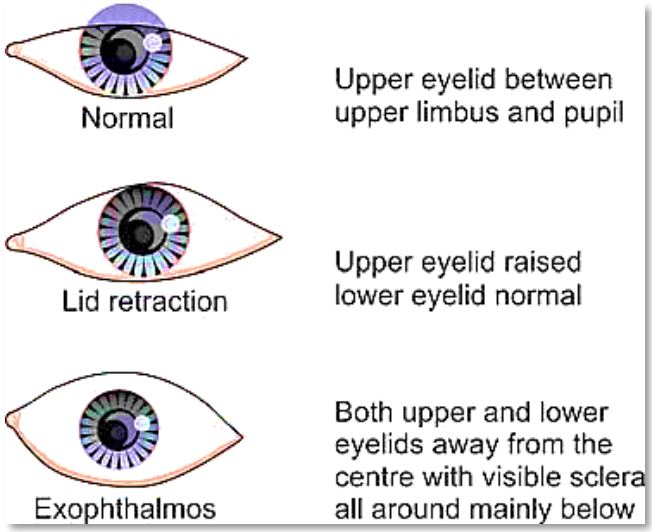


Figure 209: Eye signs in hyperthyroidism/Thyrotoxicosis

- Remember: antithyroid drugs may worsen exophthalmos and the patient should be observed once antithyroid drugs are started as steroid supplementation may be required.

CARDIAC MANIFESTATIONS

- Tachycardia (common), take sleeping pulse rate for 3 consecutive nights and average is taken as the value
  - Grade 1 <90b/m
  - Grade 2: 90-110b/min
  - Grade 3: >110 b/min
- Ectopic heart beats
- Multiple extrasystoles
- Paroxysmal atrial tachycardia & fibrillation
- Persistent atrial fibrillation (not responsive to digoxin)
- Pulsus paradoxus
- Wide pulse pressure

MYOPATHY

- Weakness of proximal muscles occurs i.e. front thigh muscle, arm muscles.
- Weakness is more when muscle contracts isometrically either while getting down steps or lifting a full bucket.
- Often when it is severe it resembles myasthenia gravis. Once hyperthyroidism is controlled recovery occurs.

PRETIBIAL MYXEDEMA

- This is a misnomer.
- It is often a feature of primary thyrotoxicosis.
- It is due to deposition of myxomatous tissue (mucin like deposits) in skin and subcutaneous plane. Glycosaminoglycans (hyaluronic acid) deposition occurs- same as exophthalmos.
- It is usually bilateral, symmetrical, shiny, red thickened dry skin with coarse hair in the feet and ankles.
- In severe cases skin of entire leg below the knee with involvement of foot and ankle can occur.
- It might or might not regress completely after treatment for toxicity.

- Skin becomes cyanotic when cold. Skin changes in toxicosis are called as thyroid dermopathy. They include- pretibial myxoedema, pruritus, palmar erythema, hair thinning, Dupuytren's contracture.

THYROID ACROPACHY

- Thyroid acropachy is clubbing of fingers and toes in primary thyrotoxicosis.
- Hypertrophic pulmonary osteoathropathy can develop.

OTHERS

- Thrill is felt in the upper pole of thyroid and also bruit on auscultation. It is because in upper pole, superior thyroid artery enters the gland superficially and so thrill and bruit can easily be felt. In lower pole inferior thyroid artery enters the gland from deeper plane and so thrill cannot be felt.
- Hepatosplenomegaly.

INVESTIGATIONS

- Thyroid function tests: Increased T3, T4 and low TSH. Sometimes T3 is the only thing elevated (T3 toxicosis)
  - Normal Free T3= 3-9 pmol/L
  - Normal Free T4= 8-26pmol/L
- Thyroglobulin antibodies
  - Raised in pregnancy, cirrhosis, hyperestrogenism
  - Decreased in high androgen levels, hypoproteinemia, acromegaly
- Thyroid antibody estimation- antithyroglobulin and TSH receptor antibody
- Radioisotope study (I-123 or ^{99m}Tc-technetium 99m) for hot nodules
  - I-131 is used for radioactive iodine therapy (beta rays are used)
  - I-123 is used for diagnostic studies (gamma rays are used)
- ECG- to look for cardiac involvement
- FBC- total count and neutrophil count are very essential base line investigations before starting antithyroid drugs (as it may cause agranulocytosis)

MANAGEMENT

- Medical (anti-thyroid drugs)
  - Indications:
    - Toxicity in pregnant women (propylthiouracil is preferred)
    - Toxicity in children and young adults
    - Before subtotal thyroidectomy to make the patient euthyroid. Note: antithyroid drugs should be continued during and after surgery for 7-10 days.
    - Soon after starting radioactive I-131 therapy for 6 to 12 weeks (effects of radiotherapy start only in 6 to 12 weeks)
  - Carbimazole:
    - Blocks thyroid hormone synthesis. It also suppresses the autoimmune process in Graves' disease
    - Dose: 5-10mg exactly 8 hourly/TID (as half-life of carbimazole is 8 hours). Each table is 5mg. It is usually given for 12-18 months.
    - Often Tri-iodothyronine 20micrograms QID or thyroxine 0.1 mg daily are given in combination with antithyroid drugs to prevent iatrogenic thyroid insufficiency or to prevent increase in size of goiter.
    - Side effects: fever, rashes, arthralgia, myalgia, neuritis, lymph node enlargement, liver cell dysfunction, psychosis, agranulocytosis
  - Methimazole: like carbimazole. Dose is 5 to 20 mg daily long acting, once a day dose.
  - Propylthiouracil:
    - Blocks thyroid hormone synthesis and peripheral conversion of T4 to T3
    - It also decreases the thyroid autoanibody levels.
    - Can be given for hyperthyroidism in children, pregnancy and lactation.
    - Dose: 200mg 8 hourly (TID)

- Side effects: dose unrelated hepatotoxicity, agranulocytosis, antineutrophilic cytoplasmic antibody (20% of patients on long term use)

➢ Propranolol
- Dose: 40mg TID
- It reduces the cardiac problems as well as blocks peripheral conversion of T4 to T3.
- Contraindications: bronchial asthma, heart block, cardiac failure.
- Long acting nadolol

➢ Lugol's iodine (5% iodine + 10% potassium iodide): decreases the vascularity of the gland and makes it more firm and easier to handle during surgery. Dose is 10-30 drops/day (minims) for 10 days prior to surgery. Potassium iodide tablets 60mg TID also can be given instead of Lugol's iodine. But its use at present is disqualified. (1 minim =1 drop. 1ml= 16 drops)
- Note: Lugo's iodine prevents the release of hormone from the gland (thyroid constipation). After 2 weeks, effects of Lugol's iodine is lost causing thyroid escape from iodine control.

• Surgical

➢ Indications:
- Failed medical treatment in primary thyrotoxicosis in young patients
- Autonomous toxic nodule
- Nodular toxic goiter
- When malignancy cannot be ruled out
- Graves' disease in children, Graves with nodules
- Need for antithyroid drugs for more than 2 years
- Large goiter, substernal/intrathoracic goiter
- Pressure symptoms, Graves ophthalmopathy
- Amiodarone induced thyrotoxicity

➢ Procedures done:
- Subtotal thyroidectomy: both lobes with isthmus are remove and a tissue equivalent to pulp of finger is retained at the lower pole of the gland on both side (5-8g)
- Hemithyroidectomy: this is done in an autonomous nodule. The entire lateral lobe with the whole of isthmus is removed.
- Total thyroidectomy: this is the better option in Graves disease to achieve the lowest relapse rate and successful stabilization of thyroid ophthalmopathy as it clears the antigenic focus in thyroid completely.

➢ Complications of thyroid surgery:

- Damage to the recurrent laryngeal nerve leading to palsy (2% of thyroidectomies) and causes hoarseness.
- Damage to the external branch of the superior laryngeal nerve, leading to palsy and causing hoarseness.
- Hypocalcemia due to damaged parathyroid (5% of thyroidectomies)
- Hemorrhage, potentially causing laryngeal edema and respiratory compromise.

- Radioiodine therapy
  - Indications:
    - Primary thyrotoxicosis after 45 years of age
    - Autonomous toxic nodule
    - Recurrent thyrotoxicosis
  - Radioiodine destroys the cells and causes the complete ablation of thyroid gland. It is given only after the age of 45 years as the chances of genetic mutation (Damage), leukemia, carcinomas are high in younger individuals.
  - Usual dose is 5 to 10 millicurie or 160 microcurie/g of thyroid.
  - The patient is made euthyroid using antihyroid drugs, drug is discontinued for 5 days, I-131 300-600 MBq is given orally, antithyroid drugs are started after 7 days and are continued for 8 weeks. In 30% of patients additional 2 or 3 doses may be required.
  - It takes 3 months to get full responses and so until then, the patient has to take antithyroid drugs. Often additional 1 or 2 doses of radioiodine are required to have complete ablation. Eventually they go for hypothyroidism and so require maintenance dose of L-thyroxine 0.1 mg daily.
  - To give therapeutic dose, patient should be admitted and isolated for 7 days (half-life) to prevent irradiation. It is given orally soon after getting from the manufacturer without much delay to have optimal efficacy.

Choice of therapies			
	Condition	***Age***	***Treatment***
1.	*Diffuse toxic goitre*:		
	a. Small goitre	Over 45 years	Antithyroid drugs for 18 months
	b. Large goitre	Under 45 years	Radioiodine therapy Surgery (*Subtotal thyroidectomy*)
2.	*Toxic nodular goitre*		Surgery (Subtotal thyroidectomy)
			Initially antithyroid drugs are given to make the patient euthyroid before surgery
3.	*Toxic solitary nodule*	Over 45 years	Radioiodine
		Under 45 years	Surgery (Hemithyroidectomy)
4.	*Recurrent thyrotoxicosis after surgery*	Under 45 years	Antithyroid drugs
		Over 45 years	Radioiodine therapy
5.	*Failure of antithyroid drugs or radioiodine therapy*		Surgery

Figure 210: Choice of therapies

HYPOTHYROIDISM

- This is characterized by low thyroid hormones.
- It is common among females (10:1).

CLASSIFICATION

- It may be classified as:
  - Primary- due to thyroid disease or removal of the thyroid.
  - Secondary- due to hypopituitarism
  - Tertiary- due to hypothalamic disease

ETIOLOGY

- Classified as:
  - Congenital:
    - Agenesis or dysgenesis of the thyroid
    - Enzyme deficiency (Dyshormonogenetic goiter: peroxidase enzyme deficiency)
  - Acquired:
    - Iodine deficiency (commonest cause in developing countries)

- Hashimoto's thyroiditis (commonest cause in developed countries): antibodies raised against the enzyme thyroid peroxidase and thyroglobulin (anti-thyroglobulin antibodies)
- Radioiodine
- Drugs: lithium, amiodarone
- After thyroidectomy (common cause)

CRETINISM	MYXEDEMA
• This is hypothyroidism in younger children. • It is due to inadequate thyroid hormone production during fetal and neonatal period. • Cause: ➢ Thyroid agenesis ➢ Inborn error of thyroid metabolism ➢ Dyshormonogenetic goiter: peroxidase enzyme deficiency ➢ Maternal hypothyroidism during early pregnancy ➢ Iodine deficiency (endemic) • Clinical features: ➢ Mental disability (thyroid hormones required for normal	• This is hypothyroidism in adults • Causes: ➢ Iodine deficiency ➢ Hashimoto thyroiditis (commonest cause of hypothyroidism where iodine is sufficient) ➢ Drug e.g. lithium ➢ Surgical removal or radioablation of thyroid • Clinical features: ➢ General: Tiredness, weight gain, cold intolerance, goiter, hyperlipidemia, hypercholesterolemia ➢ Cardiovascular: bradycardia with decreased cardiac output

brain development- formation of neuronal synapses) ➢ Short stature with skeletal abnormalities ➢ Delayed puberty ➢ Coarse facial features ➢ Enlarged tongue ➢ Typical hoarse cry ➢ Thickened skin ➢ Umbilical hernia • It is treated with L-thyroxine OD in the morning	(Shortness of breath and fatigue), angina, cardiac failure, pericardial effusion ➢ Hematological: anemia ➢ Gastrointestinal: constipation, ileus ➢ Reproductive: infertility, menorrhagia, galactorrhea ➢ Dermatological: dry skin, vitiligo, alopecia, erythema ➢ Others: Carpal tunnel syndrome, myalgia, hoarseness, deafness, ataxia, depression, psychosis (myxedema madness) • Treatment is with L-thyroxine/Levothyronine, Glucocorticoids, and antibiotics

- Examination will usually reveal a small firm goiter. If a nodule is present, then this should be investigated as for a solitary thyroid nodule in order to exclude malignancy.
- Thyroid lymphoma usually develops on a background of autoimmune hypothyroidism.
- Extreme hypothyroidism can present as myxedema madness with confusion leading to coma.
- Note: myxedema is severe thyroid failure

INVESTIGATIONS

- TSH: elevated in hypothyroidism with decreased free T4 and/or T3
- Antibodies: Thyroid peroxidase and antithyroglobulin

TREATMENT

- Patients are rendered euthyroid by treatment with Levothyroxine (L-thyroxine).
- In the elderly patients with ischemic heart disease, L-thyroxine should be introduced cautiously starting at 25-50 μg and increasing the dose every fortnight in order to avoid precipitating tachyarrhythmias and cardiac failure.
- Normal thyroxine dose= 75-150 μg.
- Patients should have their TSH checked every 12-18 months thereafter to ensure correct thyroxine dosage.

- For some patients, liothyronine (T3) is an alternative. Initial rapid response can be achieved by giving L- iodothyronine 20 μg TID.
- Myxedema coma is characterized by:
  - Hypothermia (precipitated by cold)- Drug of choice- T3 (levothyroinin) 10 μg IV QID
  - Hypotension (precipitated by infection or trauma)- Levothyroxine 300 μg IV/ 1000 μg orally (large dose)
  - Hyponatremia- give glucocorticoids
  - Hypoventilation- give antibiotics
  - Hypoglycemia- slow warming
  - Bradycardia- electrolyte management
  - Skin that is cold like a todd
  - Coma

PARATHYROID GLAND

HYPERPARATHYROIDISM

HYPOPARATHYROIDISM

ADRENAL GLANDS

CUSHING'S SYNDROME

HYPERALDOSTERONISM

ADRENAL TUMORS

PHAEOCHROMOCYTOMA

ADRENAL INCIDENTALOMA

MULTIPLE ENDOCRINE NEOPLASIA

PITUITARY GLAND

TUMORS OF ANTERIOR PITUITARY

BREAST

- All patients presenting with a breast lump should have a triple assessment including:
  - Clinical assessment:
    - History
    - Physical examination
  - Radiological assessment:
    - Ultrasound (patients below the age of 35)
    - Mammogram (patients above 35 years) or
    - Ultrasound is particularly useful in assessing whether a lump is solid or cystic.
    - Mammography is good at determining whether benign lumps are very well defined or have a surrounding halo. Breast cancers are commonly associated with speculation, architectural distortion or malignant microcalcification
  - Pathological assessment:
    - Fine needle aspiration: a fine needle attached to a syringe is inserted into the lump and cells are withdrawn by making several passes through the lump with negative pressure (6ml). Once cells are withdrawn from the breast lump, they can either be fixed or air-dried on a slide immediately or drawn into a transport medium and sent to the lab for them to be centrifuged and plated on a glass slide, fixed and stained with either hematoxylin and eosin or Papanicolaou stain.
      - Advantage: it allows drainage of a cyst (if fluid is present, then diagnosis is invariably benign). If the fluid withdrawn is bloody then further investigation is needed.

Classification of cell from FNAC

C1- inadequate

C2- normal/benign

C3- atypia (probably benign)

C4- atypia probably malignant

C5- malignant

C5 diagnosis on cytology that is congruous with the clinical and radiological features allows the surgeon to carry out a definitive surgical procedure for breast cancer

- Disadvantage: a breast cyst can be associated with presence of an intracystic papilloma (rarely carcinoma) and in these cases excision of the cyst is necessary. Clinically these cysts recur and an intracystic abnormality is visible on ultrasound.

- Trucut biopsy: when cytology is inadequate or unhelpful the next step in diagnosis is core biopsy.
  - This is a minimally invasive procedure where under local anesthesia a section of breast tissue is obtained from the lump.
  - This carries some advantages over fine-needle aspiration cytology in that a histological assessment of tumor grade (when a lump is malignant) can be made and estrogen receptor status can also be assessed.

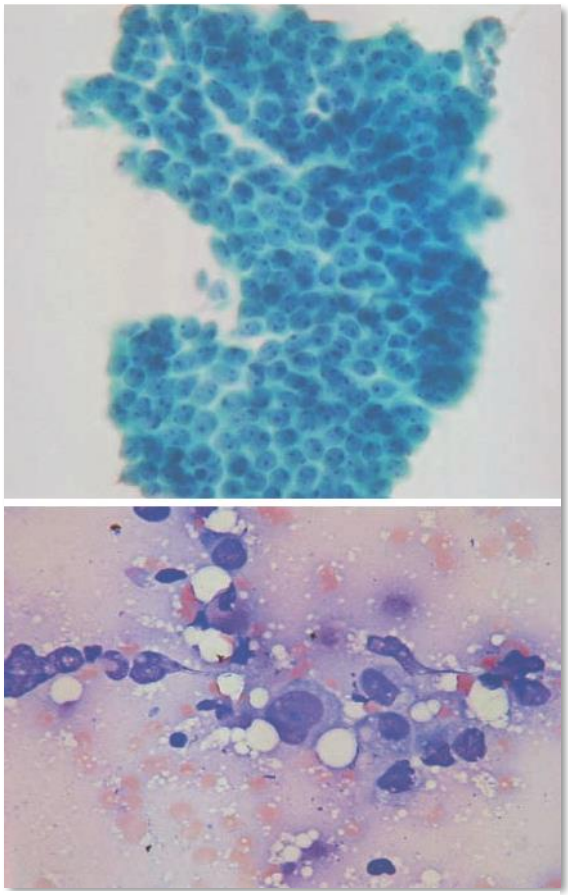


Figure 211: Breast cytology- Benign cytology (Up) and Malignant cytology (below)

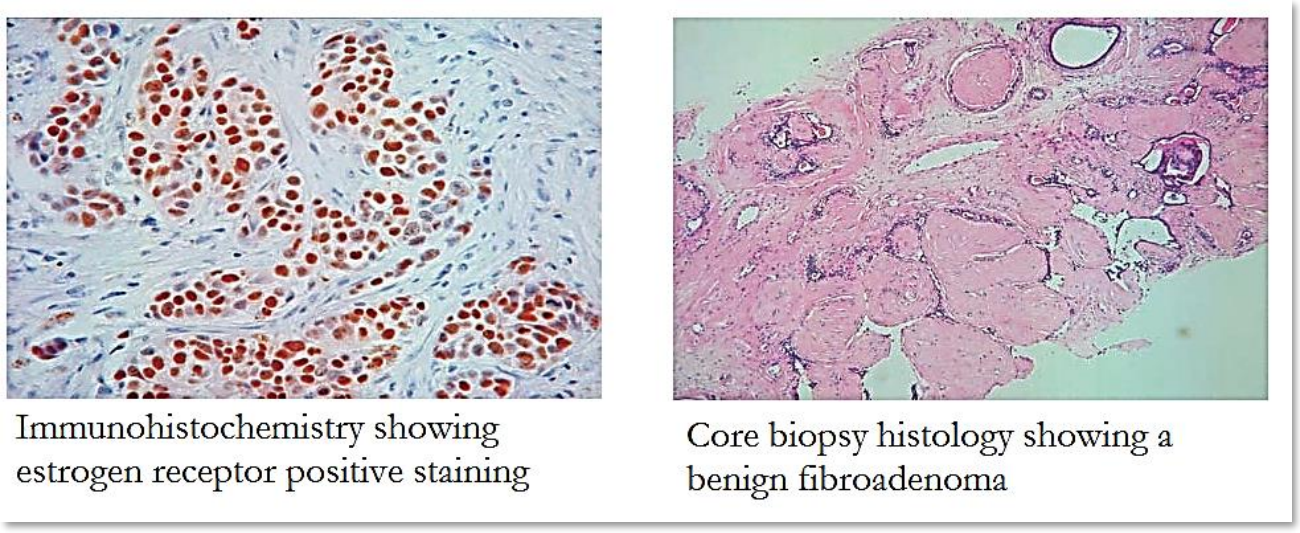


Figure 212: Breast histology from biopsy

APPROACH TO A BREAST LUMP

HISTORY

- Demographic history: Age (young- benign conditions, old- malignant)
- Presenting complaint: breast lump
- History of presenting complaint:
  - When was the lump first noticed? (duration)
  - What brought it to the patients' notice?
  - What symptoms does it cause?
    - Skin changes (ulceration, dimpling)
    - Nipple changes (retraction)
    - Nipple discharge
  - Has it changed? How much has it changed since it was first noticed? Has it increased or decreased in size?
  - Has it ever disappeared or healed?
  - Does the patient have any other lumps?
  - What does the patient think caused it?
- Review of systems: Associated symptoms:
  - Dyspnea, chest pain, cough, hemoptysis (lung mets)
  - Jaundice or abdominal distension (liver metastasis)
  - Back pain (bone metastasis especially to lumbar spine)
  - Hard nodules on skin (skin metastasis)
  - Swelling of arms (Axillary lymphadenopathy)
- Past Medical and surgical history
  - History of any operation (lumpectomy, excisions, biopsy).
  - History of breast cancer, ovarian cancer
  - History of radiation exposure
  - Use of Estrogen based oral contraceptives/ hormonal replacement.
- Gynae history:
  - Last menstrual period
  - Menstrual regularity and volume
  - Age of menarche/menopause
  - Age of 1st pregnancy or 1st child
  - Number of children (if any)
  - Breast feeding or not + last time of breast feed
- Family history: History and number of breast cancer in 1st degree relative (mother, sister, daughter)
- Social history
  - Diet and obesity
  - History of alcohol/smoking

EXAMINATION OF THE BREAST

- Before the examination always:
  - Wash hands.
  - Greet the patient and introduce yourself.
  - Explain the examination to the patient and gain consent.
- Explain the need for a chaperone: "One of the female ward staff members will be present throughout the examination, acting as a chaperone, is it ok?"
- When explaining the examination this may be a good time to ask if she has noticed any lumps or other problems (discharge, tenderness) and if she performs a monthly breast self-examination.
  - If she does not, teach her good technique and watch as she repeats the steps of examination after you, giving helpful correction as needed.
- Patients sometimes say that their lump can only be felt when they adopt a certain posture e.g. standing or lying on one side, and they should therefore be examined in this position as well.
- An adequate inspection requires full exposure of the chest but later as you examine one breast you may find it helpful to cover the other. Expose all of the top half of the trunk.

Self-breast examination

Adult women of all ages are encouraged to perform a monthly self-breast examination as follows:

- In the shower:
  - With the pads of the middle 3 fingers, check the entire breast and armpit area pressing down with light, medium and firm pressure
  - Check both breasts each month feeling for any lump, thickening, hardened knot, or any other breast changes
- In front of a mirror:
  - Visually inspect the breast with the arms on the side, next raise them high overhead. Look for any changes in the contour, any swelling, or dimpling of the skin or changes in the nipples
  - Next rest the palms on the hips and press firmly to flex the chest muscle. Left and right breasts will not exactly match (this is normal for some women), look for dimpling, puckering or changes particularly on one side.
- Lying down:
  - When lying down the breast tissue evenly spreads on the chest wall. By placing a pillow under the right shoulder and the right arm behind the head use the pads of the left fingers to palpate the entire right breast covering the entire areas and armpits. Use light, medium and firm pressure noting any masses or lumps
  - Repeat the process on the other breast
  - Squeeze the nipples and check for discharge

- Because breasts tend to swell and become more nodular before menses as a result of increasing estrogen stimulation, the best time for examination is 5 to 7 days after the onset of menstruation.
- Nodules appearing during the premenstrual phase should be reevaluated at this later time.

INSPECTION

GENERAL INSPECTION

- Inspection is done with the patient seated. Ask the patient to sit upright, ideally on the side of the bed.
- Note any obvious masses, swelling, skin changes, scars, asymmetry or nipple discharge.

CLOSE INSPECTION

- Inspect the breast and nipples with the patient in the following positions:
  - With the arms on the side laying on the thighs relaxed
  - Arms pressed against the hips and push inwards (to tense pectoralis major): this creates tension on the suspensory ligaments.





- Check for:
  - Scars- small scars (lumpectomy)/ Large diagonal scars (mastectomy)
  - Size: varies enormously in different individuals.
  - Symmetry: it is quite normal for there to be a difference between the side. However, any marked size difference of recent onset is likely to be caused by significant pathology.
  - Masses: note the size and position- look for overlying skin changes.
  - Skin changes: color, thickening of the skin and unusually prominent pores which may accompany lymphatic obstruction. The skin may be pulled in or puckered by an underlying cancer.
    - Redness (erythema) indicates local infection or superficial malignancy
    - Thickening and prominent pores suggest a breast cancer.
    - Puckering- underlying malignant mass

- There may be edema caused by obstruction of skin lymphatics by cancer cells, which is commonly referred to as peau d'orange.
  - The skin has ridges or appears pitted like the skin of an orange (peau d'orange) caused by a buildup of fluid and edema in the breast.
  - Indicates inflammatory breast cancer

➢ Characteristics of the nipple
- Contours: look for changes such as masses, dimpling or flattening
  - Flattening of the normally convex breast suggests cancer.
- Nipple retraction- congenital/ underlying tumor/ ductal ectasia
- Direction in which the nipples point
  - Asymmetry of directions in which nipples point suggests an underlying cancer
- Any rash, ulceration or any discharge
  - Rash or ulceration (Scale) in Paget's disease of the breast.



➢ Nipple inversion
- Fixed or Recent flattening or depression of the nipple suggests nipple retraction. A retracted nipple may also be broadened and thickened suggesting an underlying cancer.

➢ Arms over the head

➢ Arms over the head while leaning forward



- Placing the arms over the hips, head and leaning forward helps to bring out dimpling or retraction that may otherwise be invisible.
  - Ask the patient to press her hands against her hips to contract the pectoral muscles as well as to place the hands over the head and lean forward.
  - Inspect the breast contours carefully in each position. If the breasts are large or pendulous, it may be useful to have the patient stand and lean forward, supported by the back of the chair or the examiner's hands.
  - Dimpling or retraction of the breast in these positions suggests an underlying cancer. When a cancer or its associated fibrous strands are



attached to both the skin and the fascia overlying the pectoral muscles, pectoral contraction can draw the skin inwards, causing dimpling.

- Occasionally, these signs may be associated with benign lesions such as posttraumatic fat necrosis or mammy duct ectasia but they must always be evaluated with great care.

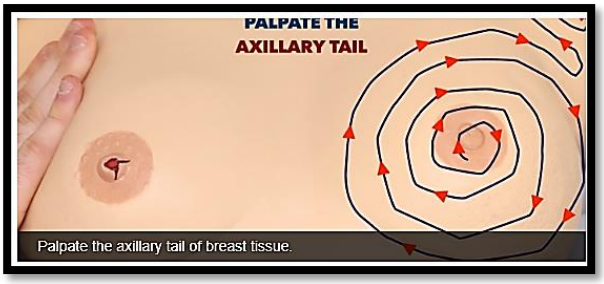
PALPATION

- Palpation is best performed when the breast tissue is flattened, the patient should thus be supine.
- Before examining the breast ask the patient if they have any nodes they can feel or pain in any breast, if present this breast should be examined last.
  - Always Examine the asymptomatic side first.
- A thorough examination will take 3 minutes for each breast.



- Palpate all the four quadrants of the breast including the axillary tail.
  - Use the finger-pads of the second, third and fourth fingers, keeping the fingers slightly flexed.
  - It is important to be systematic:
    - Clock face method- examine each "hour" of the breast
    - Spiral method- start at the nipple and work outwards in a concentric circular motion
  - Palpate in small, concentric circles at each examining point, if possible applying light, medium and deep pressure. You need to press more firmly to reach the deeper tissues of a large breast. When pressing deeply on the breast do not mistake a normal rib for a hard breast mass.
- Examine the breast tissue carefully for:
  - Consistency of the tissue: normal consistency varies widely, depending in part on the relative proportions of firmer glandular tissue and soft fat. Physiologic nodularity may be present, increasing before menses. There may be a firm transverse ridge of compressed tissue along the lower margin of the breast, especially in large breasts. This is the normal inframammary ridge, not a tumor.
  - Tenderness, as in premenstrual fullness

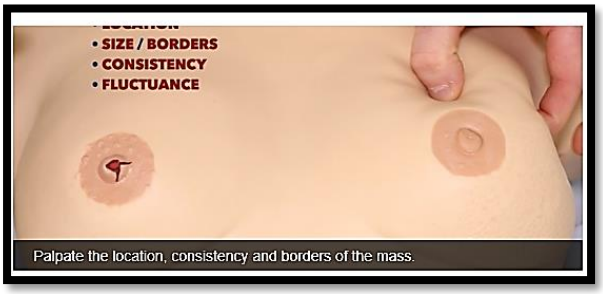
- If a mass is detected, ensure you complete a thorough examination of the remaining breast tissue before examining the mass further.
- Palpate the axillary tail of the breast:
  - Breast tissue often extends into the axilla and is known as the axillary tail.
  - The majority of breast cancers develop in the upper outer quadrant so it's essential this area is examined thoroughly



- If the patient reports having nipple discharge, ask them to squeeze the nipple to demonstrate this:
  - Yellow/ green discharge- infection
  - Greenish discharge-duct ectasia
  - Bloody discharge- more suspicious of malignancy e.g. papilloma.

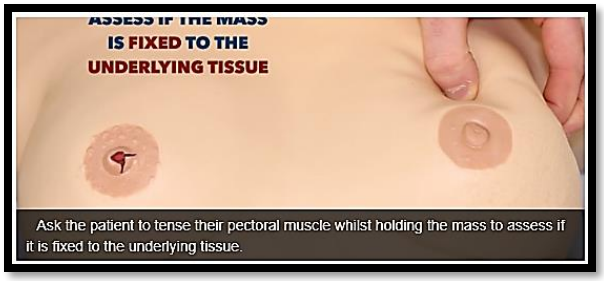


- Repeat palpation on the other breast, asking the patient to place their alternate hand behind their head.
- For every lump the following should be mentioned
  - Position/Location:
    - which quadrant of the breast? (e.g. upper outer quadrant)
    - Using the clock face metaphor, what "hour" on the clock is the mass located at? E.g. 2 o'clock
    - How far away from the nipple is the mass located? E.g. 4cm.
  - Size and shape:
    - What are the approximate dimensions of the mass? E.g. 2cm X 3cm
    - What shape is the mass? E.g. spherical/elongated/ irregular
  - Consistency: smooth, firm, stony, rubbery
  - Overlying skin changes: erythema/puckering





- Mobility:
  - Does if move freely
  - Does it move with overlying skin
  - Does it move with pectoral contraction
- Fluctuance: hold the mass by its side then apply pressure with another finger to the center of the mass. If the mass is fluid filled e.g. cyst then you should feel the sides bulging outwards



- Palpate each nipple, noting its elasticity.

AXILLA

- The axillary lymph nodes can now be palpated for lymphadenopathy (malignancy/ infection):
  - Have the patient sit on the edge of the bed facing you.
  - Support the patient's arm on the side being examined with your forearm.
  - If you're examining the right axilla, use your right arm to support the patient's (vice versa for left).
  - Palpate the axilla with your free hand covering the lateral, anterior, posterior walls, central and apex of the axilla.



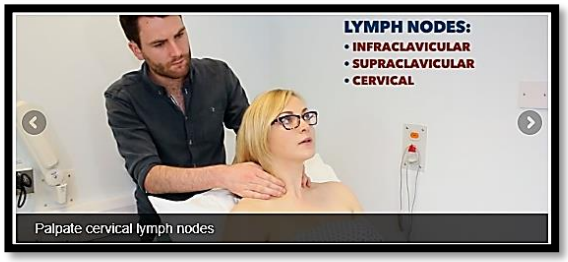


- Other lymph nodes to palpate include: submental, submandibular, pre-auricular, subauricular, post-auricular, sub-occipital, cervical, supraclavicular, infraclavicular and parasternal.



- To complete the examination
  - Thank the patient and cover them up.
  - Summarize finding.
  - Suggest further assessments and investigations:
    - Biopsy (FNA/ core biopsy)- to determine histology.
    - Ultrasound or Mammography depending on the age
    - Chest X-ray (if necessary)





BREAST ANATOMY

- The breast is a modified apocrine sweat gland derived from ectoderm as branching epithelial cords which form lactiferous ducts.
- About 15-20 lobes develop during puberty, each of which drains into a single lactiferous duct.
- True secretory alveoli develop during pregnancy and lactation under the influence of estrogen, progesterone and prolactin.
- The mammary gland develops embryologically as invaginations of surface ectoderm along two ventral lines (milk line/ intrauterine milk ridge/anterior mammary ridge)- which run from the axillae to the vulva.
- Breast tissue (and pathology) can develop anywhere along this milk line- e.g. Supernumerary nipples.



Figure 213: Accessory nipple (Image adapted from SRB's Manual of Surgery)

- The breast located vertically between the 2nd and 6th rib in the mid-clavicular line and lies over the pectoralis major, serratus anterior and external oblique muscles.
- Horizontally it lies from side of sternum to the mid-axillary line two-thirds of the breast rests upon pectoralis major and a third on the serratus anterior.
- At its lower medial quadrant, it rests on the external oblique aponeurosis, which separates it from the rectus abdominis
- The breast lies in superficial fascia. The superficial fascia is divided into a superficial layer and a deep layer.
- Below the superficial fascia lies the retromammary space (within the loose areolar tissue), deep pectoral fascia and muscles (pectoralis major, serratus anterior, external oblique) and the chest wall.
- Retromammary bursa/space: located between deep layer of superficial fascia and pectoral (Deep) fascia allowing free mobility of breast.
- The breast varies in size and shape among different individuals but it is characterized by fullness in the lower area and ptosis or drooping, which becomes pronounced with increasing age.

GROSS ANATOMY

- Grossly the breast consists of:
  - Nipple: located at 4th intercostal space just below the center/summit of the breast.
    - It Contains circular and longitudinal muscles to make nipple stiff or flat.
    - It is pierced by 15-20 lactiferous ducts.
    - Each duct independently opens into the nipple.
    - It has rich sensory nerve endings and contains modified sweat and sebaceous glands.
  - Areola: Circular pigmented area around the nipple.
    - Rich in modified sebaceous glands which enlarge during pregnancy and lactation as Montgomery tubercles. They secrete oily lubricant to nipple and areola.
    - Areola and nipple do not contain hair and fat beneath.
  - Axillary tail of spence:
    - This is a prolongation from the outer part of the gland which passes up to the level of the third rib in the axilla through a defect in the deep fascia (Foramen of Langer) where it is in direct contact with the main lymph nodes of the breast (Anterior axillary nodes).
    - When the axillary tail enlarges it is often mistaken for a lipoma

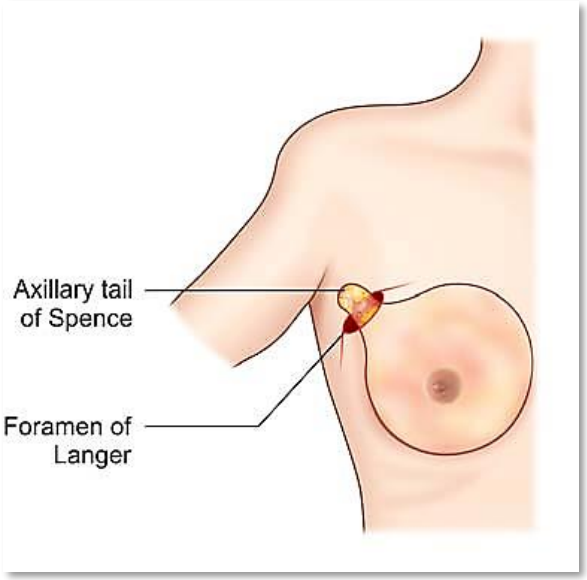


Figure 214: Axillary tail of Spence (Image adapted from SRB's Manual of Surgery)



Figure 215: Hypertrophy of axillary tail (Image adapted from SRB's Manual of Surgery)

- The breast is anchored to the overlying skin and to the underlying pectoral fascia by bands of connective tissue called ligament of cooper.
  - Contraction of these strands in cancer due to malignant cell invasion may cause dimpling of the skin or attachment of the growth to skin, which in turn cannot be pinched off from the lump.
  - If cancer grows along the ligament of cooper binding the breast to the pectoral fascia, the breast gets fixed to the pectoralis major. It then cannot be moved along the long axis of the muscles.

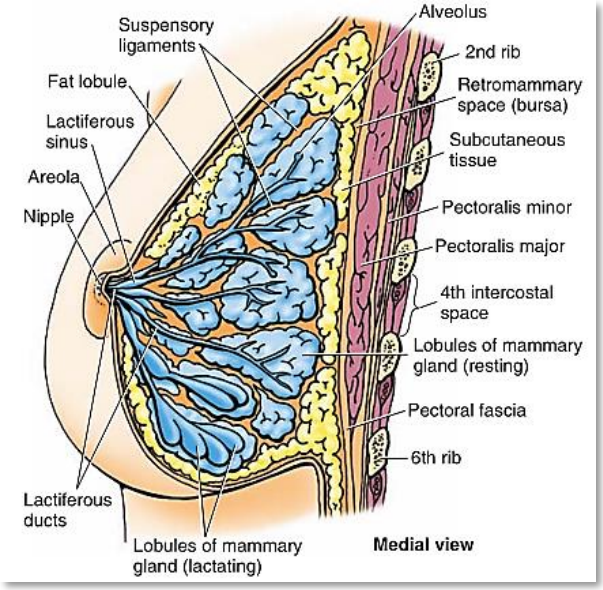
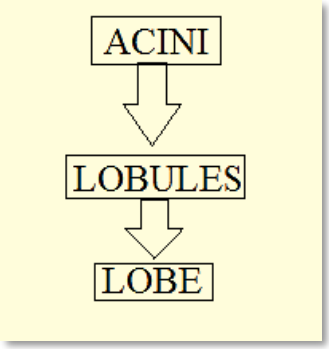


Figure 216: Structure of the breast

HISTOLOGY

- Breast tissue consists of stromal tissue and parenchyma.
- The breast is composed of acini which make up lobules, aggregation of which form the lobes of the gland.



- Approximately 10-100 alveoli (Acini) form lobules.
- Lobules make milk that drains via ducts to the nipple.
- The lobes are arranged in a radiating fashion like the spokes of a wheel and converge on the nipple, each lobe is drained by a lactiferous duct.
- The functional unit of the breast parenchyma is the terminal duct-lobular unit.
- About 15-20 lobes develop during puberty, each which drains into a single lactiferous duct.
- True secretory alveoli develop during pregnancy and lactation under the influenced of:
  - Estrogen-ductal proliferation
  - Progesterone- glandular proliferation
  - Prolactin- milk production
  - Oxytocin- milk secretion

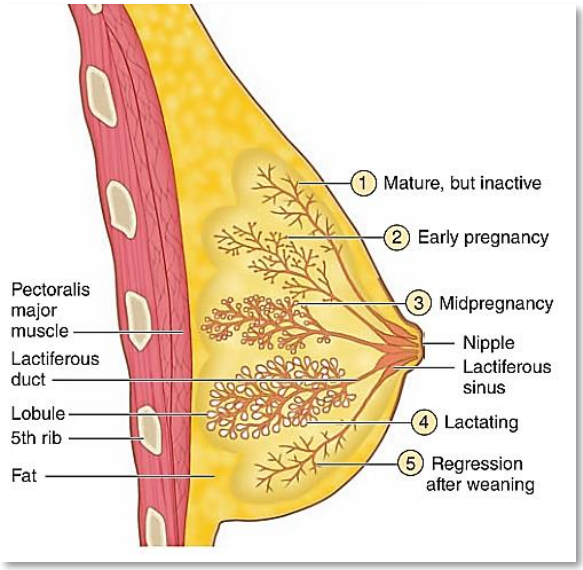


Figure 217: Histology of the breast at different stages

- All the ducts and lobules are lined by 2 layers of epithelium:
  - Luminal cell layer:
    - Alveolus- cuboidal (in rest) and columnar (in lactation)
    - Smaller duct- single layer of columnar epithelium
    - Larger ducts- many layered columnar
    - Lactiferous duct- stratified squamous
  - Myoepithelial cell layer

BLOOD SUPPLY, NERVE INNERVATION AND LYMPHATICS

- Arterial supply:
  - Perforating cutaneous branches of internal mammary/thoracic artery (from the subclavian) to the 2nd, 3rd and 4th intercostal space-60%
  - Lateral thoracic (from 2nd part of axillary artery)-30%
  - Lateral branch of the 2nd, 3rd and 4th intercostal arteries.
  - Pectoral branches of thoracoacromion artery
  - Superior thoracic artery
- Venous drainage
  - Superficial veins from the breast characterized by their proximity to the skin drain to axillary, internal mammary and intercostal vessels.
- Nerve innervation: anterior and lateral cutaneous branches of 4th to 6th intercostal nerves. (note: milk secretion under control of oxytocin not by nerves). The nipple is innervated by T4.
- Lymphatics:
  - Lymphatics drain predominantly into the axillary, internal mammary and central lymph nodes
  - Axillary lymph nodes drain about 80% of the breast whilst supraclavicular lymph nodes drain 20%
  - Axillary lymph nodes include:
    - Anterior group (Main drainage node)- along lateral thoracic
    - Central/medial group (next common)
    - Lateral group

- Posterior group
- Apical group

➢ These can be divided into surgical (Berg's) levels by the pectoralis minor muscles
  - Level 1: below and lateral to the pectoralis minor (anterior, lateral and posterior)
  - Level 2: behind/beneath the pectoralis minor muscle (central)
  - Level 3: above and medial to the pectoralis minor (Apical)

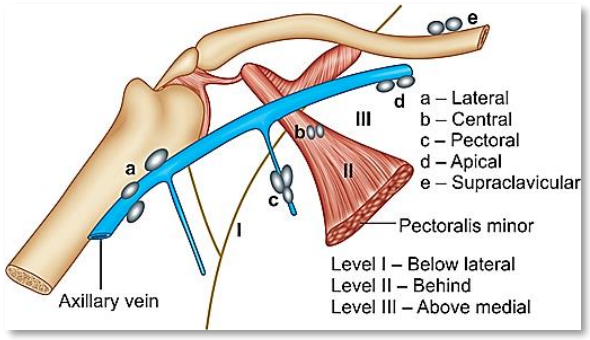


Figure 218: Levels of lymphatic drainage of the breast (Image adapted from SRB's Manual of Surgery)

➢ Spread restricted to level 1 nodes carries better prognosis.
➢ Spread to level 2 has poor prognosis whilst spread to level 3 carries the worst prognosis.

BREAST IMAGING: MAMMOGRAPHY

- This is a plain X-ray of soft tissue of the breast using low voltage and high ampearage X-rays.
- Two films are taken:
  ➢ Craniocaudal: from above downward
  ➢ Mediolateral: from side to side
- Mammography is done in patients above 35 years old. In those below 35 years an ultrasound is done
- Findings:
  ➢ Microcalcifications: signify malignancy. They are <0.5mm pleomorphic/heterogenous, grouped, fine, linear and branching
  ➢ Soft tissue shadow may be smooth, round/oval and regular in benign conditions or irregular carcinomas. Lobulated mass is also suggestive of carcinoma
  ➢ Size and location of mass lesion
  ➢ Spiculations, duct distortion (features of malignancy)
  ➢ Margin may be circumscribed (benign lesion), microlobulated, obscure, ill-defined, spiculated or stellate.



Figure 219: Mammography showing calcified lactiferous duct with a benign lesion (Image from SRB's Manual of Surgery 2nd Ed)

BREAST PATHOLOGY

- These include:
  - Congenital anomalies
  - Inflammatory conditions
  - Benign tumors (fibroadenomas, papillomas) and fibrocystic changes (nodular lesions, cysts and epithelial hyperplasia)
  - Breast cancer
- Note:
  - Breast lump diagnosis varies with age
  - Fibroadenomas are common in young women
  - Cysts are common in women aged 30-50 years
  - All new lumps in women over 50 years old are cancer until proven otherwise

CONGENITAL ANOMALIES

- Complete absence of breast development (amazia) can occur and is usually found in association with lack of development of the pectoralis major muscle (Poland's syndrome).
  - Poland syndrome: Amazia, Absence of sternal portion of pectoralis major, male predominance
- Other causes for absence of a breast include:
  - Inadvertent excision of the breast bud (breast lumps should not be excised in children)
  - Radiotherapy to the chest wall in children
- Accessory nipples (as discussed earlier). Besides being curiosities, these are also subject to the same diseases that affect the normal breast.

INFLMMATORY CONDITIONS

MASTALGIA

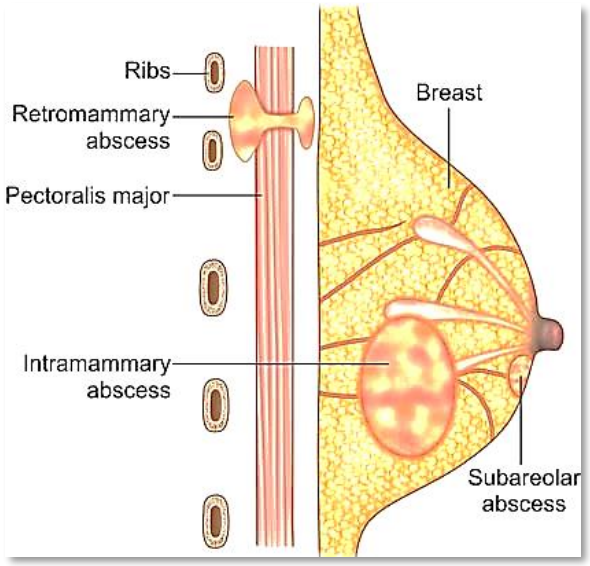
- This is pain in the breast.
- Incidence: 45% (21% is severe)
- Mostly etiology is unknown and poorly understood.
- It is not usually associated with breast cancer.
- Mastalgia is seen in patients on antiretroviral therapy, caffeine,

tobacco, large pendulous breasts etc.

- Breast pain can be divided into:
  - Cyclical- 65%
  - Noncyclical- 30%
  - Chest pain-5%
- Cyclical
  - It is extremely common
  - Pain related to menstrual cycles. When this discomfort is severe or prolonged beyond the usual 3-5 days it requires intervention.
  - It is due to an increase in fluid retention however diuretics have no role in its management.
  - Cyclical mastalgia is usually seen in ANDI (Aberration in normal development and involution) pathologies like fibrocystadenosis.
  - It Present in women of menstruating age group.
  - It is bilateral diffuse with "heavy feeling".
  - Treatment:
    - Reassurance that it is a physiological condition and not associated with increased risk of breast cancer.
    - Evening primrose oil 1000mg for 3 months (containing free fatty acids particularly gamma-linoleic acid) have been shown to improve cyclic mastalgia. It is thought to influence lipid metabolism and improve transport across intracellular membranes.
    - In refractory cases drugs can be used:
      - Bromocriptine (2.5 mg BD), Vitamin B6 & B12 and analgesics
      - Tamoxifen usually 20mg TID for 3 months (not supported by trials yet)
- Non-cyclical
  - Other causes of breast pain are peri-ductal mastitis, malignancy, cervical root pain, musculoskeletal pain, previous surgery, Tietze's syndrome (costochondritis)
  - It is unilateral, chronic, burning or dragging in nature, occurs bother in pre- and postmenopausal age group.
  - 5% of breast cancers present as pain during first presentation.
  - Treatment:
    - Identify cause
    - Malignancy should be ruled out
    - Avoid coffee and stress
    - Proper support to breast
    - NSAIDs can be given.
- Tietze's syndrome: costochondritis of 2nd costal cartilage, commonly seen in females, mimics mastalgia.

MASTITIS

- Inflammation of the breast tissue.
- Consists of
  - Subareolar mastitis
  - Intramammary mastitis
  - Retromammary mastitis (submammary)



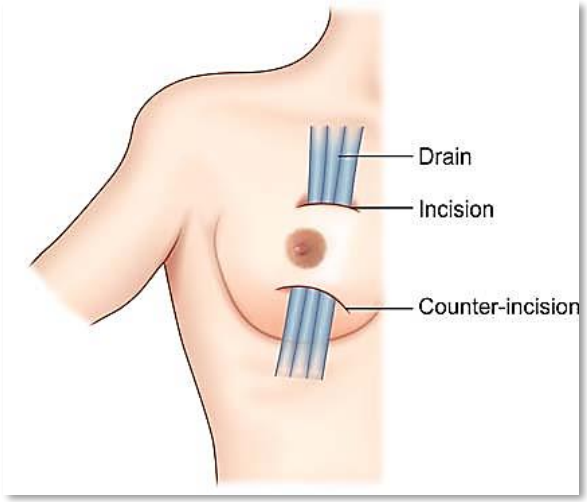
- Subareolar mastitis
  - Due to infection under areola caused by cracks in the nipple or areola. Results from an infection.
  - Associated with duct ectasia- causing formation of abscess, sinus and fistula
  - Common in non-lactating women
  - Clinical features:
    - Red, inflamed, edematous areola with a tender swelling underneath.
    - Nipple retraction may develop
  - Differential: paget's disease of the nipple
  - Treatment: under cover of antibiotics (Flucloxacillin) pus is drained by making a subareolar incision.
- Intramammary mastitis
  - Types:
    - Lactational abscess of the breast
    - Non-lactational abscess of the breast
  - Lactational abscess
    - Seen in lactating women.
    - Occurs in 3% of breastfeeding mother usually up to 6 months of lactation.
    - Mode of infection:
      - Bacteria (*Staph aureus*- most common) enters during sucking through cracked nipples occasionally it can spread by hematogenous spread.

Precipitating factors

- Cracked nipple
- Retracted nipple
- Improper cleaning of the nipple
- Inadequate milk sucking by baby or milk expression causing stasis
- Infection from the mouth of the baby
- Haematoma getting infected

    - *Staph aureus* causes clotting of milk in the blocked duct and multiply.

- Duct initially gets blocked by epithelial debris or by retracted nipple. Initially it begins in one quadrant but later involves entire breast.
- Clinical features
  - Continuous throbbing breast pain and high grade fever
  - Diffuse redness, tenderness, warmness and brawny induration of breast.
  - Purulent nipple discharge
  - Entire breast may get involved eventually
  - Occasionally tender fluctuant swelling (10%) may be felt, ulceration and discharge can occur at a later period.
  - Difficult to differential initial stage of mastitis (stage of cellulitis) from stage of breast abscess formation.
  - When it is treated by antibiotics without incision and drainage eventually it may get organized to form a non-tender, hard breast lump with sterile pus inside- stage of antibioma formation.
- Differential: inflammatory carcinoma of breast.
- Treatment:
  - Antibiotics- cephalosporins, flucloxacillin, and amoxicillin,
  - Repeated US guided aspirations (avoids surgery and scars)
  - Drainage under general anesthesia, a counter incision may be needed
  - It is not advisable to wait till the formation of abscess. Often takes very long time to heal after surgery causing distress to patient and surgeon.



Indications for drainage in mastitis/breast abscess

- Mastitis not resolving with antibiotics in 48 hours
- Persistent fever and progression of mastitis
- Brawny induration

Do not wait for abscess to form (fluctuation to develop)

- Complications:
  - Antibioma formation
  - Sinus formation, skin necrosis, fistula formation
  - Recurrent infection, bacteremia, septicemia.
- Suppression of lactation if often required by giving Bromocyptine 2.5 mg BD for 2 weeks

➢ Non-lactational abscess
- Common in duct ectasia and periareolar infection
- Common organisms: bacteroides, anaerobic streptococci, enterococci and gram negative organisms.
- Commonly recurrent with tender swelling under the areola
- Treatment:
  - Antibiotics
  - Repeated aspirations
  - Drainage and later cone excision of the duct is done

- Retromammary mastitis

➢ Due to tuberculosis of the intercostal lymph nodes.
- Other causes: TB of ribs, suppuration of intercostal lymph nodes, empyema necessitans, infected hematoma.

➢ Breast is normal.

➢ Investigations:
- CXR, FNAC, ESR
- Peripheral smear
- U/S of breast and chest wall
- Often CT scan chest may be needed

➢ Treatment:
- Treat cause
- Drainage through submammary/retromammary incision.

ANTIBIOMA

- If intramammary mastitis is not drained but only treated by antibiotics, pus localizes and becomes sterile (flaques) with a thick fibrous tissue cover and it is called as antibioma.
- Clinical features:

➢ Previous history of mastitis treated with antibiotics

➢ Swelling which is painless, smooth, nontender, hard, fixed to breast tissue without involving the pectorals and chest wall.

- Investigations

➢ FNAC

➢ Mammography

➢ U/S breast

- Treatment: excision (submammary incision). Later antibiotics are given. It should be sent for histology.

TUBERCULOSIS OF THE BREAST

- Rare, infection reaches through blood or retrograde lymphatic spread from lymph nodes of axilla.
- Associated with active pulmonary tuberculosis
- Common in lactation
- Nipple and areola are not commonly involved.
- Presents as a swelling in the breast with cold abscess, sinuses and a typical bluish appearance of surrounding skin with matted lymph nodes in the ipsilateral axilla.
- Lump- irregular ill-defined, peau d'orange, discharge, sinus, matted axillary nodes often with sinus are the features.



Figure 220: Tuberculosis of breast showing undermined lesion

- Differential: Carcinoma breast
- Investigations:
  - FNAC
  - Frozen section biopsy is useful to differentiate from carcinoma
  - Excision biopsy
- Treatment:
  - Antituberculous drugs (INH, rifampicin, ethambutol, pyrazinamide)
  - Drainage of cold abscess
- Note: mastectomy is not done

MAMMARY DUCT ECTASIA (PLASMA CELL MASTITIS)

- This is dilatation of lactiferous ducts due to myoepithelial relaxation of duct wall with periductal mastitis.
- Also known as 'plasma cell mastitis' as periductal inflammation contains plasma cells.
- Commonly many ducts are involved.
- Hormonally induced myoepithelial relaxation with poor ductal absorption of secretions and desquamated cells causing obstruction are the probable other causes and features.
- It is an uncommon condition usually encountered in parous women between 40 and 60 years of age.
- Clinical features
  - Greenish discharge or creamy paste like nipple discharge

- Indurate mass under the areolar which is often tender

- Differential: carcinoma of breast
- Investigations:
  - Discharge study: FNAC
  - Mammography

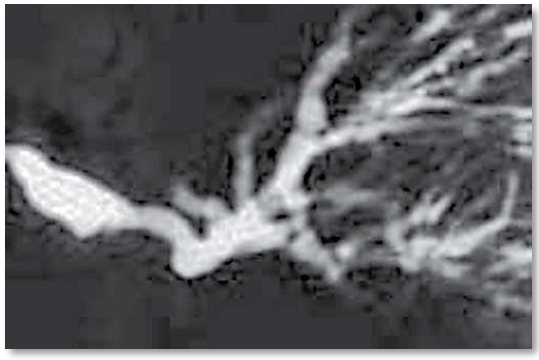


Figure 221: Duct ectasia

- Treatment:
  - Stop smoking
  - Cone excision of involved major duct (Adair-Hadfield operation)
  - Antibiotics
  - Melhem Novel modified breast ductal system excision

TRAUMATIC FAT NECROSIS

- This is a fibrous scar in the breast tissue caused by injury, hematoma and necrosis of breast fat with subsequent scarring.
- There is history of breast trauma in 50% of cases.
- True is due to either direct or indirect.
- There may be associated superficial ecchymoses.
- Pathogenesis:
  - Capillary ooze causes triglyceride in the fat to dissociate into fatty acids.
  - It combines with calcium from the blood resulting in saponification which causes inflammatory reaction and later presents as a non-progressive swelling in the breast.
- Clinical features:
  - Painless swelling in the breast which is smooth, hard, nontender and adherent to breast tissue
  - Bilateral and multifocal
  - It is nonprogressive, nonregressive.
  - Nipple retraction at a later stage. Slit like retraction of nipple due to fibrosis occurs
  - Abscess and fistula formation
  - Common in smokers- in relation of arterial pathology
  - Common also in multiple pregnancies, perimenopausal age, hyperprolactin status
  - May present with mastalgia
  - Axillary nodes may be palpable as nonspecific
  - Secondary bacterial infection (anaerobic) is common.
- Investigations
  - FNAC: chalky fluid with fat globules
  - Histology: periductal cellular infiltrates and fibrosis

- Mammography to rule out malignancy
- Differential diagnosis: carcinoma breast
- Treatment: surgical excision to exclude malignancy.

BENIGN TUMORS AND FIBROCYSTIC CHANGES

- These are due to Aberrations/abnormalities of normal development and involution of the breast (ANDI) pathologies.
- The term ANDI is broad and covers benign conditions of the breast, many of which have overlapping features.
- ANDI are common in younger premenopausal women and often cause considerable anxiety.
- Any breast lump should be evaluated by triple assessment for risk of malignancy whatever the likely diagnosis.

GYNECOMASTIA

- This is the presence of breasts in the male resembling those of the sexually mature female.
- It is usually physiological but often needs to be investigated for hormonal causes:
  - Estrogen or steroid therapy
  - Liver disease
  - Pituitary gland tumor
  - Testicular tumor
  - Use of spironolactone
- Treatment involves addressing the underlying cause however in severe cases is by excision of breast tissue.

BREAST CYST

- These are fluid filled cavities lined by epithelium.
- Incidence: 1 in 14 females, above the age of 35 up to menopause (30-40s).
  - Uncommon after menopause.
  - Hormone replacement can cause cyst formation in old women.
- They arise from destruction and dilatation of breast lobules and terminal ductules. It is due to nonintegrated stromal and epithelial involution.
- Cysts may be microscopic or macroscopic.
- They contain straw colored, green colored or opaque fluid.
- Cyst size varies with menstruation due to influence of ovarian hormones.
- They can present as:
  - Multiple (50%) and often bilateral.
  - Tender
- Cysts can be recurrent (50%).
- On examination they are smooth, soft, fluctuant often transilluminating well-localised swellings.
- Investigations:
  - U/S of breast

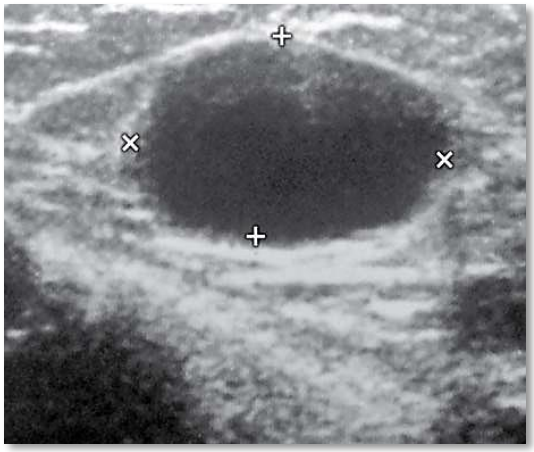


Figure 222: Ultrasound showing a breast cyst

- FNAC
- Mammography to rule out associated carcinoma

- Differential diagnosis:
  - Bloodgood cyst
  - Hematoma cystic necrosis in a carcinoma
  - Brodie's disease (phyllodes)
  - Galactocele
  - Lymph cyst
  - Hydatid cyst
- Treatment:
  - Reassurance
  - Aspiration for 2 times
  - Surgical excision is done if cyst recurs after two aspirations or if there is bloody discharge or residual mass if felt after aspiration.

Cystic swellings of the breast

- Bloodgood cyst
- Breast abscess
- Hydatid cyst
- Galactocele
- Serocystic disease of Brodie
- Cystic necrosis in carcinoma breast
- Lymph cyst
- Haematoma in breast

FIBROCYSTIC CHANGE

- Due to Aberration of Normal Development and Involution (ANDI) of normal breast tissue.
- Presently called cyclical mastalgia with nodularity.
- The term fibroadneosis refers to a spectrum of conditions that commonly present as lumpiness or discomfort on a background of generally lumpy breasts.
- It is an estrogen dependent condition however, Estrogenic therapy and oral contraceptives do not seem to increase the incidence of these alterations and oral contraceptives may in, fact decrease the risk.
- It is due to development of fibrosis and cysts in the breast.
- It is the most common breast disease (pre-menopausal) and occurs in the upper outer quadrant. The changes tend to arise during reproductive age and are most likely a consequence of the cyclic breast changes that occur normally in the menstrual cycle.

- It is an exaggerated response of breast stroma and epithelium to hormones and growth factors.
- It is rare in nulliparous/ovulating/oral contraceptive pill taking women.
- Breast involution causes changes at the microscopic level including fibrosis (increase in fibrous tissue), adenosis (increase in glandular elements) and apocrine changes (the normal epithelium lining the ducts becomes sweat gland-type epithelium).
- In addition, minimal hyperplasia of the epithelial cells lining the ducts occurs and small breast cysts may develop
- Stages:
  - Stage I-Stromal proliferation or hyperplasia
  - Stage II- Adenosis (increased glands)
  - Stage III- cyst formation. Cysts have a blue-dome appearance on gross exam.
- Microscopic changes include:
  - Non-proliferative changes (Common)
    - Stromal fibrosis with dilation of ducts
    - Microcyst formation
  - Proliferative changes
    - Epitheliosis (in ducts and acini): normal ducts and lobules are lined by 2 layers of cells (luminal cells overlying myoepithelial cells). Epithelial hyperplasia is recognized by the presence of more than 2 cell layers. It ranges from mild and orderly to atypical hyperplasia (resembling carcinoma in situ)
    - Sclerosing Adenosis: mimics carcinoma. Lesions contain marked intralobular fibrosis and proliferation of small ductules and acini
    - Papillomatosis-within the duct, often with apocrine metaplasia
- Clinical presentation:
  - Presents during menstruating age group (35-50 years) as a bilateral, painful, diffuse, granular, tender, swelling which is better felt with palpating fingers (lumpy breast).
  - Common in upper outer quadrant.
  - Pain and tenderness are more just prior to menstruation (cyclical mastalgia).
  - It subsides during pregnancy, lactation and after menopause.
  - Discharge from the nipple when present will be serous or occasionally greenish.
- Investigation
  - FNAC (Epitheliosis, when florid is undoubtedly premalignant)

- Ultrasound
- Mammography

- Treatment:
  - Conservative (preferred)
    - Reassurance, avoid caffeine, chocolate, salt
    - Medical drugs: indicated when fibroadenosis is not increasing in size, no nipple discharge especially blood, no psychological effect
  - Surgery
    - Subcutaneous mastectomy (preserves nipple, areola and skin) with prosthesis placement – only in severe, persistent disease
    - Excision of the cyst or localized excision of the diseased tissue
    - Indications:
      - Intractable pain
      - Florid epitheliosis on FNAC
      - Blood good cyst
      - Persistent bloody discharge
      - Psychological reason
- Note: this condition is benign but some fibrocystic-related changes are associated with an increased risk for invasive carcinoma (increased risk applies to both breast)
  - Minimal or no increased risk: Fibrosis, cystic changes, mild hyperplasia and apocrine metaplasia
  - Slightly increased risk (1.5-2 times)- moderate to florid hyperplasia (without atypia), Ductal papillomatosis and Sclerosing adenosis
  - Significant risk (5 times risk): Atypical hyperplasia whether ductular or lobular
- Fibrocystic change can only be distinguished from cancer through biopsy and histologic examination.
- Proliferative fibrocystic changes usually are bilateral and multifocal and are associated with increased risk of subsequent carcinoma in both breasts.

FIBROADENOMA

- This is a benign encapsulated tumor commonly occurring in young females aged 20-30.
- Presently it is considered as hyperplasia of a single lobule of the beast (classified under ANDI)
- It is the most common benign tumor of the breast below the age of 30 years in females.
  - Incidence: 15% of palpable breast lumps
  - It is common in blacks.
  - It is bilateral in 20% of cases and multiple in 20% of cases.
- It is due to an aberration in normal development (AND) of a lobule, although it shows similar hormonal

activities of normal breast like lactation, perimenopausal involution.

- Fibroadenoma does not turn into malignancy.
  - 30% of fibroadenomas may disappear or reduce in size in 2-4 years.
  - 10-15% will increase in size progressively. They do not occur after menopause unless women are on hormones.
- Presentation:
  - Painless swelling in one of the quadrants
  - Smooth, firm, nontender
  - Well-localized and moves freely within the breast tissue (mouse in the breast)
  - Axillary lymph nodes are not enlarged
- Types:
  - Gross
    - Soft-common after 30 years, more cellular, often bilateral
    - Hard- common below 30 years, more fibrous
    - Giant (>5cm in size) common in Africa
  - Microscopic:
    - Intracanalicular- large and soft- mainly cellular. Stroma with distorted duct. It carries the potential to undergo malignant transformation.
    - Pericanalicular- small and hard- mainly fibrous. Stroma with normal duct.
- Juvenile fibroadenoma:
  - Occurs in adolescent girls.
  - Rapid growth with stromal and epithelial hyperplasia
  - Does not show any alteration in stromal epithelial balance or cellular atypia or periductal cellular concentration.
  - Clinically mimics phyllodes tumor but does not turn into a phyllodes tumor or a carcinoma.



Figure 223: Large fibroadenoma left breast in a 14-year-old female

- Complex fibroadenoma:
  - Occurs in older age group
  - Typical fibroadenoma with fibrocystic changes (apocrine metaplasia, cyst formation, sclerosing adenosis)
  - 15% of proven fibroadenomas are complex
  - Occasionally it may turn into malignancy unlike usual fibroadenomas.

- Core biopsy is needed to confirm the condition.

- Investigations:
  - Mammography (well localized smooth regular shadow)
  - FNAC
  - Ultrasound (to confirm solid nature)

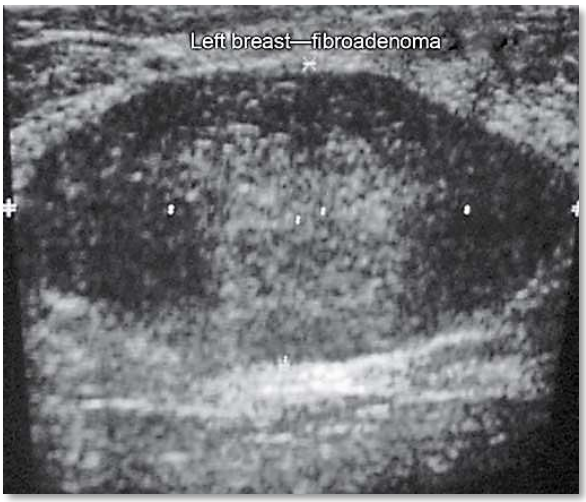


Figure 224: Fibroadenoma on ultrasound

- Treatment:
  - Excision through a circumareolar incision (Webster's) or Submammary incision (Galliard Thomas incision) under general anesthesia.

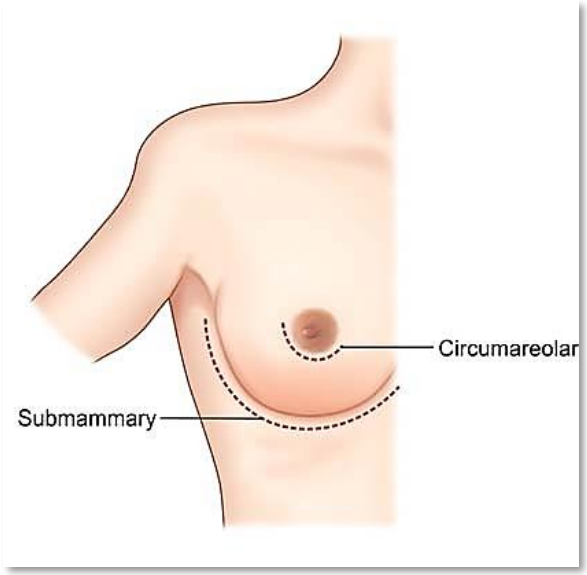


Figure 225: Incisions for excision of complex fibroadenoma

  - Fibroadenoma which is small (<3cm)/ single/ age<30 years can be left alone with regular follow-up with U/S at 6 monthly intervals. But anxiousness of patient and parents find difficult for this conservative approach.
  - Indications for surgery
    - Size> 3cm
    - Multiple
    - Giant type (usually >5cm)
    - Recurrence
    - Cosmesis
    - Complex type

HYPERPLASIA OF THE BREAST

- Asymptomatic and is usually discovered by chance.
- There is an increased in the number of epithelial cells of the lobules and ducts.
- Types:
  a. Ductal hyperplasia
  b. Lobular hyperplasia
- Grading based on microscopy is as:
  (1) Mild
  (2) Moderate
  (3) Florid (more extensive)
- This is associated with natural changes especially in women >35 years.

ATYPICAL HYPERPLASIA

- Abnormal-appearing cells (pattern or shape)
- Types:
  a. Atypical ductal hyperplasia
  b. Atypical ductal lobular hyperplasia
- Sometimes discovered by a breast biopsy.
- There is increased risk of developing cancer in atypical ductal hyperplasia.

DUCT PAPILLOMA

- Duct papilloma are epithelium-lined-true-polyps of breast lactiferous ducts.
  - ➢ They are usually <1cm in size often with a small lump under areola. But can attain large size.
  - ➢ Vascular stalk is present usually.
- They often project out like a pedunculated mass.
- Rarely a cystic soft swelling may be present underneath which is probably due to obstruction of the duct by papilloma.
- Duct papilloma is the most common cause of bloody discharge from nipple.
- It is usually single, from a single lactiferous duct.
- It blocks the duct causing ductal dilatation.
- Presentation:
  - ➢ Papilliferous swelling (projection) usually seen near the nipple orifice.



Figure 226: Ductal papilloma

  - ➢ Blood stained discharge from the nipple. Serosanguinous discharge can also occur.
  - ➢ Single papilloma is not premalignant but multiple

papillomas in many ducts can be premalignant.

- Peripheral papilloma should be differentiated from invasive papillary carcinoma

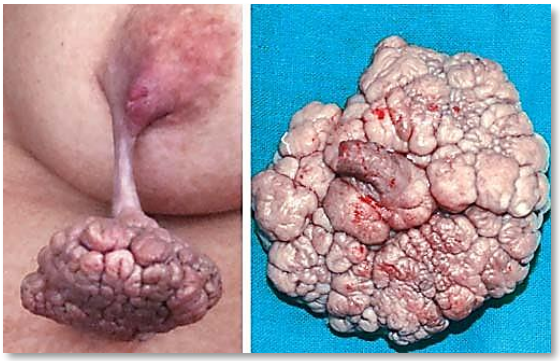


Figure 227: Papilloma breast which is large. Usually it is small intraductal

- Types:
  - Subareolar- common
  - Peripheral- occasional
  - Solitary- common
  - Multiple
  - Unilateral- common
  - Bilateral
- Investigation:
  - Discharge study (FNAC)
  - Injection of contrast into the duct (Ductogram)
  - Mammography may show dense lesion under the areola
- Treatment:
  - Microdochectomy: probed lactiferous duct is opened and the papilloma is excised using tennis racquet incision.
    - Probing of lactiferous duct is done up to 5cm deep and excised with part of the areolar skin.
- Note:
  - Intraductal papillary carcinoma arises usually not from a preexisting duct papilloma but as de novo
  - Papillomatosis is hyperplasia of the duct lining, filling the duct like a polyp without a pedicle/stalk (not a true polyp), it is not duct papilloma but part of fibrocystadenosis.
  - Axillary nodes are not involved.

ADENOSIS OF THE BREAST

- Noncancerous enlargement of the breast lobules.
- Adenosis can look like breast cancer on mammograms, so biopsy may be needed to rule out breast cancer.

PHYLLODES TUMOR

- Also known as cystosarcoma phyllodes/ serocystic disease of Brodie.
- They are not simply giant fibroadenoma.
- Show wide spectrum of activity ranging from almost benign (85%) to locally aggressive and sometimes metastatic tumor (15%).
- They occur in premenopausal women (30-50 years).
- Presentation:
  - Unilateral

- Grow rapidly to attain a large size with bosselated surface.
- Swelling is smooth, non-tender, soft, fluctuant with necrosis of skin over the summit due to pressure.
- Differs from carcinoma because:
  - Skin over breast is stretched, red and with dilated veins.
  - Tumor is warmer, not fixed to skin or deeper muscles or chest wall.
  - Nipple retraction is absent.
  - Lymph nodes are usually not involved



Figure 228: Phyllodes tumor

- Recurrence is common.
- Investigations:
  - U/S
  - FNAC, core biopsy
  - Mammography
  - Chest X-ray CT chest in malignancy to seen secondaries
- Treatment:
  - Excision or subcutaneous mastectomy
  - If malignant (Sarcoma) total mastectomy is indicated.

GALACTORRHOEA

- It is a secretion of milk not related to pregnancy or lactation.
- It is not a symptom of breast cancer.
- It is always bilateral.
  - Primary galactorrhoea: Stress and other factor. It is physiological in puberty or menopause. Reassurance is the treatment
  - Secondary galactorrhoea:
    - Dopamine receptor antagonists: haloperidol, methyldopa, chlorpromazine, metoclopramide or by hyperprolactinaemia due to pituitary tumors. It enhances the prolactin activity.
    - Hypothyroidism
    - Drugs like oral contraceptives, atenolol, clonidine, ranitidine
    - Ectopic prolactin secreting tumors usually from lungs (bronchogenic carcinoma)
    - Chronic renal failure
- Management
  - Estimation of serum prolactin, T3, T4, TSH, CT/MRI head.

- Bromocriptine therapy
- Treatment of cause. Causative drug should be stopped and different drugs should be used for needed condition

- Note: Witch's milk is secretion of milk in both male and infants due to maternal hormonal effects in fetus. It lasts up to 3 weeks after child birth.

GALACTOCELE

- Arises during lactation from cystic dilation of an obstructed duct.

BREAST CANCER

- Breast cancer is the most common carcinoma in women by incidence (excluding skin).
- Breast cancer is the second most common cause of cancer mortality. (Most common being lung cancer).

RISK FACTORS

- These can be classified as
  - Modifiable
  - Non-modifiable

NON-MODIFIABLE

- Gender: it is more common in females (99%) than in males (1%)
- Age: Breast cancer presents most commonly in a postmenopausal female but it can also present in younger females (this is suggestive of hereditary causes).
- Early menarche/ late menopause: increased duration of estrogen exposure.
- Genetics: Presence of a 1st degree relative (mother, sister, daughter)- incidence is 2-5%.
  - BRCA1, BRCA2, P53, HNPCC and AT genes are associated
- Atypical hyperplasia (5 times increased risk).

MODIFIABLE

- Obesity: adipose tissue can convert Androgen to estrone (a type of estrogen) so this increases the risk of breast cancer.
- Diet low with phytoestrogens and high alcohol intake have high risk of breast cancer. Vitamin C reduces the risk.
- Null-parity and multiparity both increase risk
- Use of estrogen-based contraceptive increases the risk

Risk factors classification		
Slight to moderate risk	***Moderate to high-risk***	***Very high-risk***
• Florid hyperplasia	• Age > 60 years	• Therapeutic radiation
• Solid duct papilloma	• ATD / ALS/ LCIS	• Family history of breast cancer in two 1st degree relatives
• Obesity, alcohol, HRT	• History of DCIS	• Family history of breast and ovarian cancer
• Nulliparity	• Cancer on one side breast	• BRCA1 and BRCA2 mutation carrier or 1st degree relative with mutation
• Early menarche, late menopause		

Figure 229: Risk factor classification for breast cancer

- Early child-bearing and breastfeeding reduces the chances of malignancy.
  - ➢ Early first child birth reduces the risk.
  - ➢ Late first child birth after 35 years increases the risk.
- Carcinoma in one breast increases the risk of developing carcinoma on opposite breast by 3-4 times. Incidence of bilateral carcinoma is 2%.
- Mutation of tumor suppressor genes BRCA1/ BRCA 2 is thought to be involved with high risk of breast carcinoma.
  - ➢ BRCA 1 mutation is having more risk (35-45%) than BRCA 2 mutation.
  - ➢ BRCA 1 is found on the long arm of chromosome 17. It is seen in breast (medullary carcinoma) and ovarian carcinoma (serous carcinoma)
    - ○ p53 gene is also found on Chromosome 17
  - ➢ BRCA 2 is found on the long arm of chromosome 13. Associated with breast cancer in males.
    - ○ Retinoblastoma gene is also found on chromosome 13.
- Occasionally mutation of BRCA 3 and p53 suppressor gene is also involve/d.

CLASSIFICATION OF BREAST CARCINOMA

- Recall the functional unit of the breast is the terminal duct-lobular unit.
- Breast carcinoma arising from lactiferous ducts is called as ductal carcinoma.
- Breast carcinoma arising from lobules is called as lobular carcinoma.
- In-situ-carcinoma is pre-invasive carcinoma which has not breached

the epithelial basement membrane. It may either be:

- Ductal Carcinoma In Situ (DCIS)- 90%
- Lobular carcinoma in Situ (LCIS)-10%

- Invasive carcinoma can occur eventually; this can either be:
  - Invasive ductal carcinoma (most common type-80%)- has many subtypes
  - Invasive lobular carcinoma (multifocal and often bilateral)
- The carcinoma can also arise from mixed connective tissue and epithelial tissue:
  - Phyllodes, angiosarcoma, carcinosarcoma
- The degree of differentiation of the tumor is usually described using 3 grades:
  - Well-differentiated (grade 1)
  - Moderately-differentiated (grade 2)
  - Poorly-differentiated (grade 3)

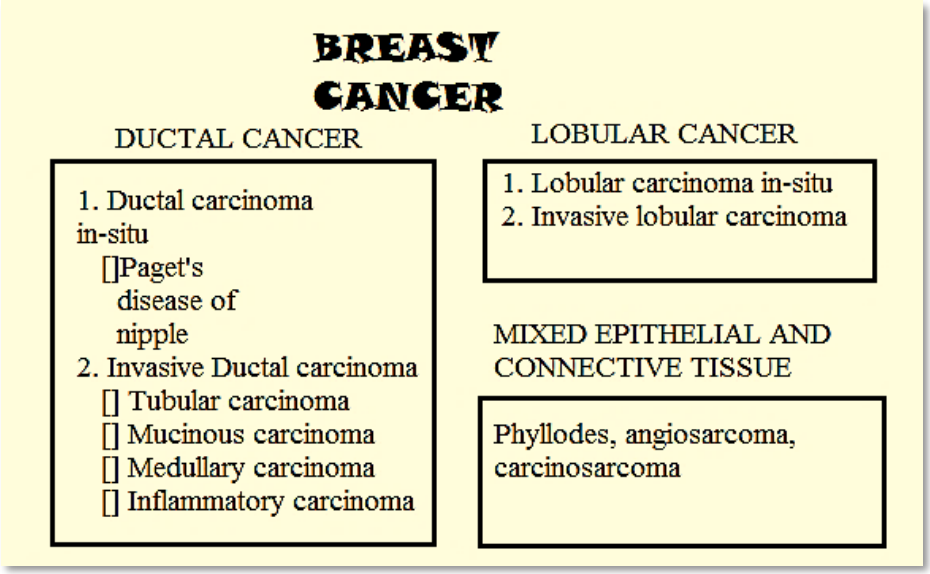


Figure 230: Classification of breast cancer

DUCTAL CARCINOMA IN-SITU (DCIS)

- Malignant proliferation of cells in ducts.
- No invasion of basement membrane.
- Detected as calcification on mammography (not detected as a mass- does not produce a mass because it is limited to the ducts).
- Comedo type has high grade cells with necrosis and dystrophic calcification in center of ducts.
  - Note: calcification is not always indicative of breast cancer. Calcification can also be seen in:

- Sclerosing adenosis (fibrocystic change)
- Traumatic fat necrosis- due to saponification of fats

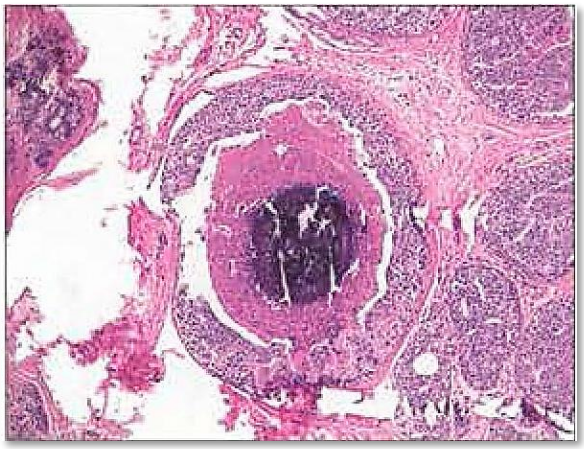


Figure 231: Ductal carcinoma in-situ, comedo type

- Biopsy of calcification is often necessary to distinguish between benign and malignant conditions.

PAGET DISEASE OF NIPPLE

- This is DCIS that has extended up the ducts to the skin of the nipple.
- It presents as nipple ulceration and erythema.
- It presents as an eczema-like condition of the nipple and areola which persists despite local treatment. The nipple is eroded slowly and eventually disappears.
- If left, the underlying carcinoma will sooner or later become more clinically evident.
- Nipple eczema should be biopsied if there is any doubt about its causes.



Figure 232: Paget's disease of Nipple

- It is Almost always associated with an underlying carcinoma.
- Recall: In extra-mammary Paget's disease (malignant cells within epidermis of Vulva) there is no underlying carcinoma.
- Microscopic appearance:
  - Large, ovoid cells with abundant clear pale staining cytoplasm in the malphighian layer of the epidermis.

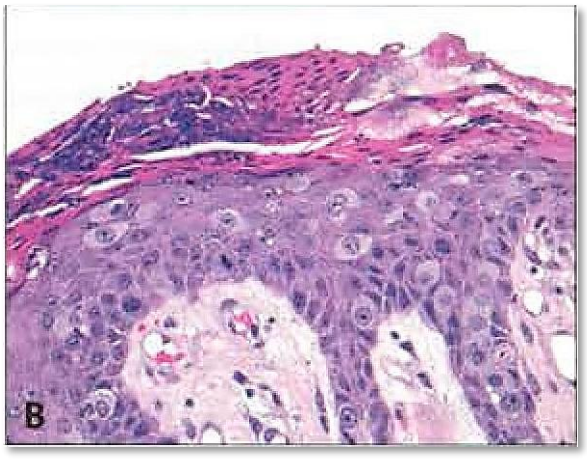


Figure 233: Paget's disease of the nipple, Microscopic appearance

INVASIVE DUCTAL CARCINOMA

- Classically forms duct-like structures.
- Most common type of invasive carcinoma in the breast, accounting for more than 80% of cases.
- Presents as a mass detected by physical examination or by mammography
  - Clinically detected masses are usually 2cm or greater.
  - Mammographically detected masses are usually 1cm or greater.
  - Advanced tumors may result in dimpling of skin or retraction of nipple.
    - Recall that retraction of nipple can also be due to inflammatory conditions
- Biopsy usually shows duct like structures in a desmoplastic stroma.
  - Desmoplastic stroma: connective tissue growing with the tumor. Connective tissue is there to support the tumor
- Important subtypes of invasive ductal carcinoma include:
  - Tubular carcinoma
  - Mucinous carcinoma
  - Medullary carcinoma
  - Inflammatory carcinoma

TUBULAR CARCINOMA

- Characterized by well differentiated tubules that lack myoepithelial cells (which distinguishes them from normal breast tissue which has 2 types of epithelia)
- Good Prognosis.

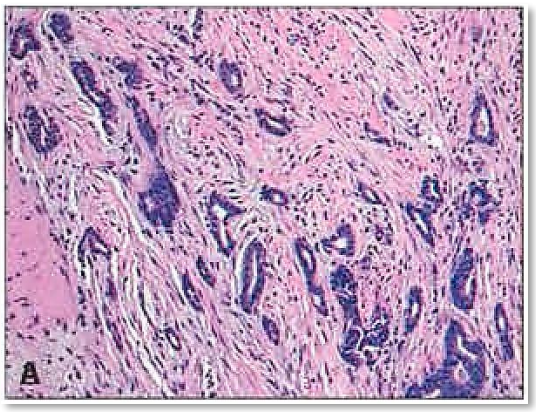


Figure 234: Tumor cells in a desmoplastic stroma (invasive ductal carcinoma-tubular subtype)

- Features that help to determine malignancy:
  - Desmoplastic stroma
  - Absence of 2 lining cell types: epithelial cells and myoepithelial cells

MUCINOUS CARCINOMA

- Characterized by carcinoma with abundant extracellular mucin ('tumor cells floating in a mucus pool')

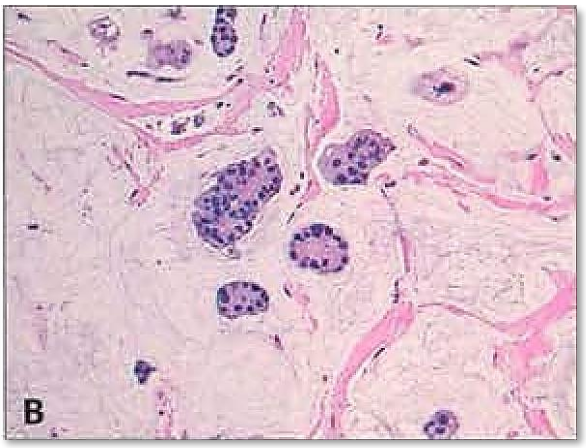


Figure 235: Mucinous breast cancer (invasive ductal carcinoma)

- Tends to occur in older women (average age is 70 years)
- Relatively good prognosis

MEDULLARY CARCINOMA

- Characterized by large, high grade cells growing in sheets with associated lymphocytes and plasma cells.
- Grows as a well-circumscribed mass that can mimic fibroadenoma on mammography.
- Relatively good prognosis.
- Increased incidence in BRCA 1 carriers.

INFLAMMATORY CARCINOMA

- Characterized by carcinoma in dermal lymphatics.

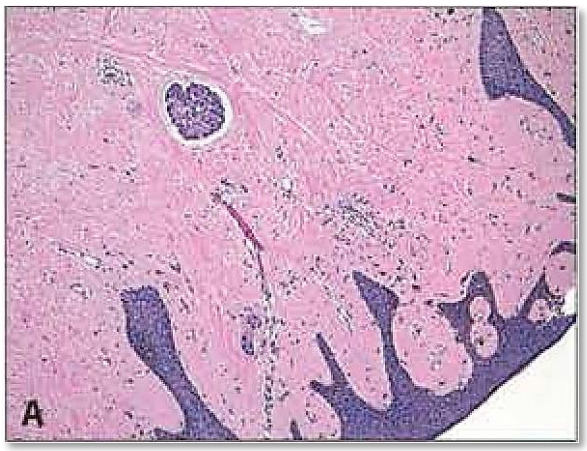


Figure 236: Inflammatory carcinoma

- Presents classically as:
  - Inflamed
  - Swollen breast (tumor cells block drainage of lymphatics) with no discrete mass, can be mistake for acute mastitis (rule out mastitis- give antibiotics and review in 5-10 days)



Figure 237: Peau d'orange, characteristic of inflammatory carcinoma

- Poor prognosis

LOBULAR CARCINOMA INSITU

- Malignant proliferation of cells in lobules with no invasion of the basement membrane.
- LCIS does not produce a mass or calcification and is usually discovered incidentally on biopsy.
- Characterized by dyscohesive cells lacking E-cadherin protein.
- Often multifocal and bilateral
- Treatment is tamoxifen (to reduce the risk of subsequent carcinoma) and close follow up, low risk of progression to invasive carcinoma.

INVASIVE LOBULAR CARCINOMA

- Characterized by cells growing in a single-file.
- Cells may exhibit signet-ring morphology.

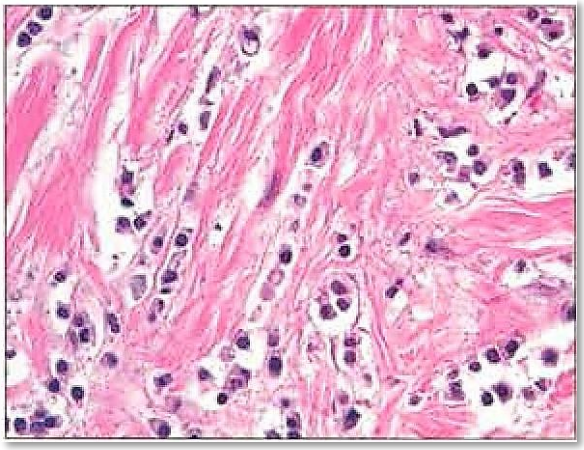


Figure 238: Invasive lobular carcinoma

CLINICAL PRESENTATION

- Although any portion of the breast including the axillary tail may be involved, breast cancer is found most frequently in the upper outer quadrant because this is where most breast tissue is found.
- Cutaneous manifestations:
  - Peau d'orange: due to obstruction of dermal lymphatics, openings of the sebaceous glands and hair follicles get buried in edema giving rise to orange peel appearance.
  - Dimpling of skin: due to infiltration of ligament of Cooper
  - Retraction of nipple: due to infiltration of lactiferous duct
  - Ulceration, discharge from the nipple and areola
  - Skin ulceration and fungation
  - Cancer-en-cuirasse: skin over the chest wall and breast is studded with cancer nodules. appearing like an armour coat.
  - Tethering to skin
- Most breast cancers will present as a hard lump which may be associated with in-drawing of the nipple.
- Spread to deeper plane
  - Into pectoralis major muscle (is confirmed by observing the restricted mobility of the

swelling while contracting the pectoralis muscle)

- Into latissimus dorsi muscle (extending the shoulder against resistance)
- Into serratus anterior (by pushing the wall with hands without flexing the elbow)
- Into chest wall (breast will not fall forward when leaning forward and while raising the arms above the shoulder, breast will not move upwards as it is fixed to the chest wall)

DIAGNOSIS

- This is done by triple assessment:
  - History and physical examination
  - Diagnostic imaging
    - Mammogram (for females above 35 years): shows irregular, speculated, radiopaque mass with micro-calcification
    - Ultrasound (for females below 35)- solid mass or cyst
    - Reasons for using US in patients below 35:
      - Breast density difference
      - Technical problems (small breast)
    - X-ray: metastasis
  - Histology/ Cytology:
    - FNAC (Fine Needle Aspiration and Cytology)
      - Advantages: least painful, can be done on outpatient basis, reliable and cheaper

Reliability of FNAC and mammography		
	FNAC	*Mammography*
Sensitivity (true positivity)	90-98%	90%
Specificity (without false-positive)	98-100%	90%
False-negative	2-10%	10%
False-positive	Near 1-5%	10%

FNAC scoring

C_0 : No epithelial cells
C_1 : Scanty epithelial cells, benign
C_2 : Benign cells
C_3 : Atypical cells
C_4 : Suspicious cells
C_5 : Malignant cells

Figure 239: FNAC vs Mammography

    - Core needle biopsy for histology

SENTINEL LYMPH NODE BIOSPY

- This procedure consists of injecting a vital blue dye or radioactive sulfur colloid of both around the tumor.
- The surgeon often waits 5 minutes in the case of the dye and a few hours in the case of colloid and dissects the axilla removing only a few nodes that either stained blue or radioactive.

- The principle behind this is that the first nodes that drain the dye are the most likely to first drain the tumor.
- Morbidity is significantly decreased as compared to a full axillary node dissection and in experienced hands, the procedure is close to 100% accurate in correctly describing the presence or absence of tumor in the axilla.

PROGNOSIS OF BREAST CANCER

- Based on the TMN staging
  - T-tumor size
    - T0- no palpable tumor
    - Tis- tumor in situ
    - T1- tumor 2cm or less in its greatest dimensions, not fixed/tethering
    - T2- tumor 2-5cm in its greatest dimensions, nipple retraction or tethering
    - T3- tumor more than 5cm (5-10cm) in its greatest dimensions, infiltration or ulceration
    - T4- any size invading skin or chest wall- not including pectoral muscles (10cm), ulceration greater than the distance of the lump, skin edema or skin ulceration or satellite skin nodules confined to the same breast or inflammatory carcinoma
  - M- metastasis
    - M0- no distant metastasis
    - M1- Metastasis present (including ipsilateral supraclavicular lymph nodes)
  - N- metastasis to local lymph nodes
    - N0- no palpable axillary lymphadenopathy
    - N1- mobile palpable axillary lymphadenopathy
    - N2- fixed palpable axillary lymphadenopathy
    - N3- palpable supraclavicular lymphadenopathy
- Metastasis is most important factor (mets out of axilla= poor prognosis) but because most patients do not present with this, spread to axillary lymph nodes becomes the most useful factor.
- Sentinel node biopsy is used to assess axillary lymph nodes (based on the principle that axillary lymph nodes drain in levels- 1, 2 and 3)
  - If level 1 lymph nodes are affected there is a chance that the tumor has spread to other lymph nodes so these other nodes can also be removed
  - If level 1 lymph nodes are not affected then the tumor has likely not spread and no need to remove the axillary lymph nodes.

STAGING OF BREAST CACNER

STAGE	TUMOR	NODES	METS
0	Tis	N0	M0
I	T1	N0	M0
IIA	T0, 1 T2	N1 N0	MO MO
IIB	T2 T3	N1 N0	MO MO
IIIA	T0,1, 2 T3	N2 N1, N2	MO MO
IIIB	T4 Any T	N1, 2 N3	MO M1
IV	Any T	Any N	M1

PROGNOSTIC AND PREDICTIVE FACTORS

- Most important factors are:
  - ER: estrogen receptor
  - PR: progesterone receptor
  - HER_2/neu gene amplification
- ER and PR associated with response to anti-estrogenic agents (tamoxifen)
- HER_2/neu gene amplification is associated with response to trastuzumab
- Recall HER_2/neu is a cell surface growth factor receptor, if a cell has excess HER_2/neu receptor through gene amplification there is growth and proliferation cells predisposing an individual to malignancy.
- An individual can thus be positive or negative for ER, PR and/or HER_2/neu gene amplification.
- When cell is negative for all it is termed 'Triple negative' and carries a poor prognosis.
- African American women have increased propensity to develop triple negative carcinoma.

HEREDITARY BREAST CANCER

- 10% of all breast cancer.
- Features suggestive of hereditary breast cancer:
  - Multiple 1st degree relative with cancer
  - Tumor at premenopausal age
  - Multiple tumors.

NOTTINGHAM PROGNOSTIC INDEX

- This is used to determine prognosis following surgery for breast cancer.
- It is calculated using 3 pathological criteria:
  - Size of the lesion (S)
  - Number of involved lymph nodes (N)
    - 0 Nodes= 1
    - 1-3 Nodes= 2
    - >3 Nodes=3
  - Grade of the tumor (G)
    - Grade 1= 1
    - Grade 2= 2
    - Grade 3=3
- The index is calculated using this formula:
  - NPI= [0.2 x S] + N + G
- Prognosis:

- 93% 5-year survival
  - >/=2.0 to </=2.4
- 85% 5-year survival
  - >2.4 to </=3.4
- 70% 5-year survival
  - >3.4 to </=5.4
- 50% 5-year survival
  - >5.4

MALE BREAST CANCER

- Rare (1%)
- Presents as a subareolar mass under the nipple in older males (majority of breast tissue in males is in the subareolar region), may produce nipple discharge (tumor developing underneath nipple).
- Usually invasive ductal carcinoma because in the male breast there is development of terminal duct but there is no proper development of lobules.
- Association with BRCA 2 mutation and Klinefelter syndrome.

SPREAD OF BREAST CANCER

- Local spread:
  - Tumor increases in size and invades other portions of the breast.
  - It tends to involve the skin and to penetrate the pectoral muscles and even the chest wall if diagnosed late.
- Lymphatic metastasis
  - Occurs primarily to axillary and internal mammary lymph nodes.
  - Tumors in the posterior 1/3 of the beast are more likely to drain to the internal mammary nodes.
  - Involvement of supraclavicular nodes and any contralateral lymph nodes represents advanced disease.
- Spread by bloodstream (hematogenous)
  - Often metastasizes to bone.
  - Frequent sites include lumbar vertebrae, femur, thoracic vertebrae, ribs and skull.
  - Deposits are generally osteolytic.
  - Metastasis may also commonly occur to the liver, lungs and brain and occasionally the adrenals and ovaries.

TREATMENT OF BREAST CANCER

- Treatment is stage dependent.
- Primary aim is to remove tumor.
- Management is usually a combined approach:
  - Medical therapy:
    - Radiotherapy
    - Hormone therapy
    - Chemotherapy
  - Surgical therapy

SURGICAL THERAPY

- Indications for mastectomy:

- Large tumor/breast size
- Multifocal tumor (tumor tissues within the same quadrant at multiple foci)
- Extensive premalignant change
- Tumors in pregnancy (particularly first trimester)
- Recurrence of breast cancer following previous breast conservation

- Surgical procedures:
  - Lumpectomy: resection of the mass
  - Quadrantectomy
  - Semi-mastectomy
  - Total (Simple) mastectomy
    - Structures removed: tumor, entire breast + axillary tail, areola, nipple, skin over the breast.
    - Often the patient is subjected to radiotherapy later (external) to axilla
  - Total mastectomy with axillary clearance (Commonly used procedure)
    - Structures removed: tumor, entire breast + axillary tail, areola, nipple, skin over the breast, axillary fat, fascia and lymph nodes (Level I and II)
  - Radical mastectomy
  - Modified radical mastectomy: removal of all breast tissue and axillary node dissection, sparing all motor nerves and muscles of the chest.
    - Patey's operation
    - Scanlon's operation
    - Auchincloss modified radical mastectomy
    - Halsted radical mastectomy

MEDICAL THERAPY

RADIOTHERAPY

- This is done by external radiotherapy over the breast area, axilla (in some patients if axillary dissection is not done or more than 4 positive axillary nodes), internal mammary and supraclavicular area.
  - Total dosage: 5000 cGY units
  - 200-cGY units daily- 5 days a week for 6 weeks
- Indications for radiotherapy:
  - Patients who undergo conservative breast surgery, breast is irradiated after surgery
  - After total mastectomy, external irradiation is given to the axilla
  - Patients with high risk of local relapse after surgery:
    - Invasive carcinoma
    - Extensive in situ carcinoma
    - Patient under 35 years
    - Multifocal disease
  - Bone secondaries to palliate pain and swelling. If there is a pathological fracture in the bone, internal fixation along with external irradiation is done

- Inflammatory breast cancer
- Preoperative to shrink tumor size and downstage the tumor for operation
- Atrophic scirrhous carcinoma of the breast (curative radiotherapy)
- >4 positive lymph nodes in the axilla, pectoral fascia involvement, positive surgical margins, extranodal spread or in patients with axillary status not known/not assessed.

HORMONE THERAPY

- Hormone therapy is used in ER/PR positive patients in all age groups (it was previously only used in perimenopausal aged women).
- Hormone therapy is easy to administer and safe. It additional provides prophylaxis against carcinoma of the opposite breast.
- Hormone therapy is also used in metastatic breast cancer.
- Hormone therapy with tamoxifen is useful in breast cancer in elderly (positive ER) after wide local excision or occasionally as tamoxifen alone.
- Hormone therapy is not used in ER negative patients
- The drugs used are shown in the table below.
- Modalities used for hormone therapy:
  - Estrogen receptor antagonist e.g. tamoxifen
  - Ovarian ablation by surgery (bilateral oophorectomy) or by radiation
  - LHRH agonist (medical oophorectomy)
  - Oral aromatase inhibitors for postmenopausal women
  - Adrenalectomy or pituitary ablation
  - Progesterone receptor antagonist
  - Androgens- injection of testosterone propionate 100mg IM 3 times a week
  - Aminoglutethimide- blocks the synthesis of steroids by inhibiting conversion of cholesterol to pregnenolone- medical adrenalectomy
  - Progestogens e.g. medroxyprogesterone acetate

- Conservative breast surgery consists of wide local excision of the tumor.
- Early breast cancer

DRUG	MECHANISM OF ACTION	DOSE	Advantages	ADVERSE EFFECTS
Tamoxifen	Estrogen receptor antagonist It blocks cytosolic estrogen receptor	20mg OD for 5 years or 10mg BID (half life for tamoxifen is 7 days, it takes 4 weeks to show benefits, it reduces the cholesterol and also CVS morbidity)	1. Reduces recurrence by 25% 2. Improves prognosis 3. Used in all ages with ER positive tumors 4. Cheap, easy, less toxic, very effective 5. Equally effective in carcinoma male breast 6. Under trial for benign diseases e.g. ANDI, cyclical mastalgia	Tamoxifen flare- flushing, tachycardia, sweating, genital itching, vaginal atrophy and dryness (premenopausal), vaginal discharge (postmenopausal), fluid retention, weight gain. Others: Nausea, bone pain (loss of bone density in premenopausal women), amenorrhea, endometrial hyperplasia/carcinoma
Medroxyprogesterone	Progestogen	400 mg		Nausea, flushing, vaginal bleeding
Aminoglutethimide	Aromatase inhibitor	250 mg QID	More effective than tamoxifen	Rash, dizziness, lethargy, Cushing facies
Arimidex	Aromatase inhibitor	1mg		Lethargy, GI upset
Letrozole	Non-steroidal competitive inhibitor of the enzyme Aromatase that converts androgens to estrogen (Aromatization)	2.5mg OD It reduces estrogen level by 98%. It is half life is 45hours. It decreases bone density	1. More effective than tamoxifen 2. Used as adjuvant endocrine therapy in post-menopausal women with hormone sensitive breast cancer, metastatic and recurrent cases. It slows down and stops growth of estrogen sensitive breast tumors	Vaginal dryness, night sweats, hot flushes, vaginal bleeding, CVS problems and osteoporosis
Zoladex (Goserelin)	LHRH agonist	3.6 mg monthly		Amenorrhea, nausea, it is expensive
Diethylstilbestrol	Estrogen	15mg daily		Fluid retention, vomiting, thrombosis, hypercalcemia
Fluoxymestrone	Androgen	30mg daily		Masculinization, nausea
It is more beneficial in ER positive patients, perimenopausal patients with positive node, postmenopausal patients. Presently tamoxifen and oophorectomy are commonly used hormonal therapies				

Presentation	Pathology
Painless lump	• Fibroadenoma • Fibrocystic change • Carcinoma • Cyst • Fat necrosis • Area of fibroadenosis • Lipoma of breast • Phyllodes tumor (Brodie tumor)
Painful lump	• Menstrual change • Area of Fibroadenosis • Cyst • Periductal mastitis

	• Abscess (postpartum or lactational) • Carcinoma (sometimes)
Pain and tenderness but no lump	• Cyclic breast pain • Non-cyclical breast pain • Carcinoma (very rare)
Nipple discharge	• Duct ectasia- Green-brownish discharge • Intraductal papilloma- Bloody discharge • Associated cysts • Galactocele
Changes in nipple and/or areolar	• Duct ectasia • Carcinoma • Paget's disease • Eczema
Changes in breast size and shape	• Pregnancy • Carcinoma • Benign hypertrophy • Rare large tumors

CHAPTER 17: UROLOGY

UROLOGY

HEMATURIA

HEMATURIA

- Hematuria is the presence of blood in urine.
- In a healthy individual <1µL of blood per day in urine i.e. 1 million RBCs (approximately 1 RBC passed per glomerulus).
  - ➢ Normal urine should contain <3 red blood cells per high power field of spun urine.

- Hematuria can usually be detected 1-5 RBC/ µL on dipstick or >3-5 RBC/high power field (HPF) on microscopy.
- Types of hematuria include:
  - Gross (macroscopic, frank): blood is visible to unaided eye
  - Microscopic: >5RBCs per high power field
- Hematuria may be:
  - Early (initial): more likely to be urethral in origin, distal to external sphincter.
  - Terminal hematuria: more likely to be bladder neck or prostate in origin
  - Diffuse (total) hematuria: source is in the bladder or upper urinary tract (ureteric or kidney)
- Discoloration of urine from pigments such as food coloring (e.g. beetroots), Drugs (rifampicin, sulfasalazine) and myoglobin is known as false hematuria. Brown urine, blood + on dipstick but no RBCs on microscopy.
- Silent hematuria is due to tumors of kidney or bladder unless proven otherwise.

ETIOLOGY

- Kidney causes:
  - Benign causes
    - Pyelonephritis
    - Glomerulonephritis
    - Infarct
    - Renal stones
    - Tuberculosis
    - Trauma
    - Polycystic kidney disease
    - Simple renal cyst
  - Malignant causes
    - Renal tumor
    - Papillary carcinoma
- Ureteric causes
  - Benign causes
    - Ureteric stone
    - Bleeding disorders
  - Malignant causes: ureteric tumor
- Bladder causes
  - Benign causes:
    - Cystitis
    - Tuberculosis
    - Bladder stones
    - Endemic hematuria (Schistosomiasis)
    - Benign prostatic hyperplasia
    - Prostatitis
  - Malignant causes
    - Bladder tumors jo
    - Carcinoma prostate
- Urethral causes
  - Benign causes:
    - Urethritis
    - Urethral injury
  - Malignant causes: uretheral tumors

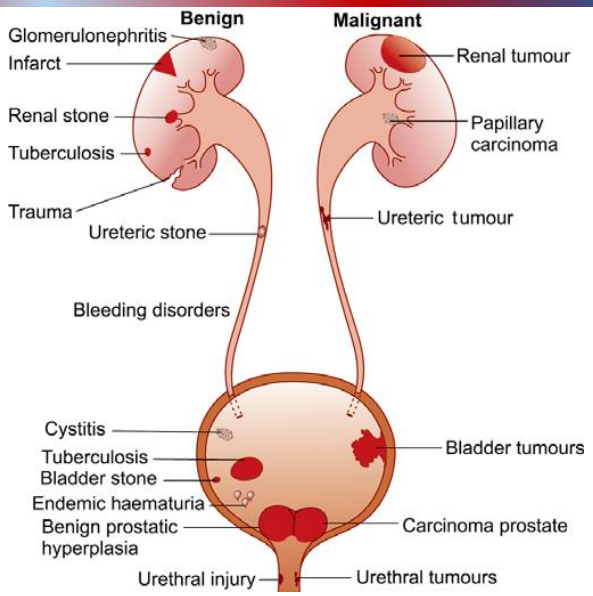


Figure 240: Causes of hematuria (Image adapted from SRB's manual of Surgery)

- Note: mild hematuria is a normal finding in a patient with recent urologic instrumentation (including catheterization)

CLINICAL ASSESSMENT

- On history focus on whether hematuria is:
  - Painful or painless
  - Gross (visible to the naked eye) or microscopic
  - Initial (only at the start of the stream)
  - Terminal (only at the end of stream)
  - Clots present
  - Hemospermia (blood also in semen)

INVESTIGATIONS

- Urine:
  - Urine microscopy, culture and sensitivity (urine test for hematuria Benzidine test)
    - In working up microhematuria, assess for the presence of red cell casts, which are present in glomerular bleeding
  - Urinary cytology for diagnosing urothelial malignancy
  - Urinalysis for urinary tract infections. False positive on dipstick occurs when myoglobin is present.
- Blood:
  - Bleeding time, clotting time, prothrombin time, platelet count for blood dyscrasias
  - Renal function tests- blood urea, serum creatinine
- Imaging
  - Kidney, urinary bladder and ureter (KUB) X-ray
  - Ultrasound to look for the stone, tumor in the urinary tract.
  - Cystourethroscopy to look for bladder or uretheral pathology
  - IVU look for function of the kidneys
  - CT abdomen

MANAGEMENT

- Identify and treat cause
- Blood transfusion
- Antibiotics

- Nephroureterectomy for renal cell carcinoma, removal of stone for kidney, ureter, urinary bladder.
- Treatment of bladder tumor by cystoscopic resection, intravesical chemotherapy using BCG, radiotherapy, systemic chemotherapy
- Treatment of medical cause like glomerulonephritis
- Correction of BPH
- Correction of bleeding diathesis

INFECTIONS OF THE UROGENITAL SYSTEM

-

INCONTINENCE AND NEUROPATHIC BLADDER DISORDERS

-

CYSTIC DISEASES OF KIDNEY

TYPES

1. Genetic
   - Adult polycystic kidney disease (Autosomal dominant)
   - Infantile polycystic kidney disease (autosomal recessive)- fatal
     - Presents in infants
     - Worsening renal failure and HTN
     - Newborns may present with Potter sequence
     - Associated with congenital hepatic fibrosis and hepatic cysts
2. Non-genetic:
   - Simple cyst,
   - Multicystic kidney,
   - Medullary sponge kidney
3. Acquired renal cystic disease may develop in patient on long-term dialysis.

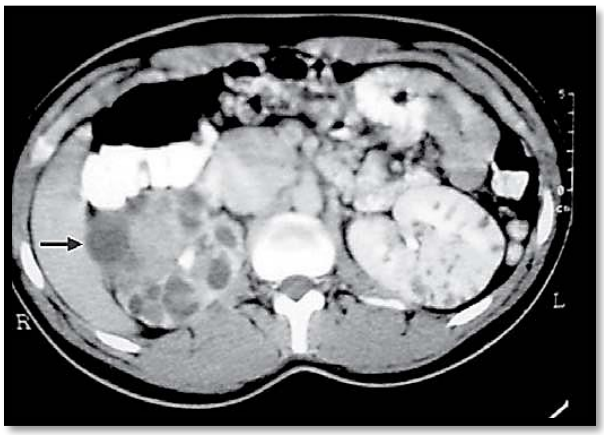
POLYCYSTIC KIDNEY DISEASE (PCKD)

- Adult PCKD is inherited as autosomal dominant disease.
  - Mutation is in Adult polycystic kidney disease 1 (APKD 1) or APKD 2.
  - The mutation is present at birth but cysts develop over time.

- It is common in females.
- It is bilateral and presents in third decade.
  - One side presents little earlier than other side
- Cyst formation occurs at the junction of the distal tubule and the collecting duct. (both in renal cortex and medulla)
- PCKD is usually associated with:
  - Polycystic disease of liver (18%), pancreas and lungs
  - Berry aneurysm in the circle of Willis.
  - Mitral valve prolapse
- Gross: multiple cysts with a clear or brownish fluid (due to hemorrhage)
- Clinical features:
  - Renal mass which is lobular, firm, mobile, moves with respiration, ballotable with dull renal angle and resonant band in front.
  - Pain, which is due to stretch of renal capsule or haemorrhage into a cyst.
  - Hematuria (25%)- due to overdistended cyst rupturing into the renal pelvis.
  - Infection is due to statsis
  - Hypertension (75%)
  - Worsening renal failure
  - Uremia occurs in later stage due to renal failure
- Differential diagnosis:
  - Renal cell carcinoma
  - Hydronephrosis
  - Solitary renal cyst

INVESTIGATIONS

- U/S confirms the presence of cysts
- Intravenous urogram- spider leg pattern with an elongated compressed renal pelvis, narrowed and stretched calyces.
- Blood urea and serum creatinine
- Urine shows low specific gravity



(CT showing bilateral, multiple kidney cysts)

TREATMENT

- Wait and watch policy
- If one of the cyst overdistends causing pain, hemorrhage, infection then surgical intervention is required.
- Rovsing operation: The kidney is exposed. The cyst is opening. The fluid is evacuated. The cut edge is marsupialised.
- Presently U/S guided aspiration is done as a simpler approach.

- Laparoscopic/retropertoneoscopic aspiration/ de-roofing of renal cyst.
- Once renal failure sets in then initial hemodialysis followed by bilateral nephrectomy is done and later renal transplantation should be planned for.

SOLITARY RENAL CYST

- Solitary renal cyst is never congenital.
- It is due to an earlier trauma or infection resulting in blockage of tubule, leading to cyst formation.
- It usually:
  - Unilateral
  - Presents as a renal mass which is smooth, often tender if infected or haemorrhagic.
- Differential:
  - Renal neoplasm
  - Hydronephrosis
  - Polycystic disease
  - Hydatid cyst

INVESTIGATIONS

- U/S and Intravenous urogram confirms diagnosis
- CT scan

TREATMENT

- Kidney is exposed. The cyst is aspirated and a portion of the cyst wall us removed (Kirwin's operation) and cavity is filled with perinephric fat.
- Occasionally, if the cyst is in one of the pole, partial nephrectomy is done.
- Laparoscopic approach.

URINARY STONES

•

URINARY RETENTION

- This is incomplete voiding of the bladder.
  - As the bladder fills with urine, the bladder muscle (detrusor) is stretched. There is an awareness of filling at a volume of 150-250ml and a desire to void when normal bladder capacity (400-500ml) is reached.
  - Micturition is achieved by relaxation of the pelvic floor musculature including the voluntary sphincter. The trigone contracts, opening the bladder neck and closing the ureteric orifices thus preventing urine reflux during voiding.
  - The detrusor then contracts, expelling the stored urine.
  - The pelvic nerves supplying the bladder and urethra with motor and sensory fibers synapse in the micturition center located in the S2-4 segments of the spinal cord which corresponds with the T2-L1 vertebral level.

- This center is connected to centers in the pons and cerebrum (especially frontal lobes) which exert an inhibitory effect that is central to voluntary control.

- Urinary retention can be classified as:
  - Acute urinary retention: complete inability to urinate
  - Chronic urinary retention

ACUTE URINARY RETENTION

- Patients want to void but cannot resulting in a huge distended palpable bladder.

ETIOLOGY

- Male:
  - Obstruction: Bladder outlet obstruction (commonest cause), Prostate enlargement (can be precipitated by cold, drugs, abundant fluid intake) and Urethral stricture
  - Inflammation: Acute urethritis or prostatitis, Balanitis
  - Anatomical: Phimosis
- Female:
  - Obstruction: bladder neck obstruction (rare)
  - Inflammation: Vulvovaginitis and Vaginal lichen planus
  - Anatomical: retroverted gravid uterus
- Both:
  - Urinary obstruction: Blood clot, Urinary tract stones, fecal impaction and Genitourinary prolapse.
  - Neurogenic (injury or disease of the spinal cord)
  - Drugs: anticholinergics, opioids, epidural, tricyclic antidepressants, sympathomimetic, spinal anesthesia
  - Others: Rupture of the urethra, Smooth muscle cell dysfunction associated with ageing, anal pain (hemorrhoidectomy), intensive postoperative analgesic treatment
  - Postoperative urinary retention: it is very common, and sometimes masquerades as incontinence. The patient may not feel the need to void because of post-op pain medication etc. but will report that every few minutes there is involuntary release of small amounts of urine. A huge distended bladder will be palpable confirming overflow incontinence from retention. Indwelling bladder catheter is needed.

CLINICAL FEATURES

- No urine is passed for several hours.
- Pain is present
- Bladder is visible, palpable, tender and dull to percussion
- Potential neurological causes should be excluded by checking reflexes in the lower limbs and perianal sensation.
- It can lead to hydronephrosis

INVESTIGATION

- Urinalysis
- Bloods: FBC, Urea and electrolytes, Glucose
- Ultrasound of bladder to check for post void residual, and kidney scan for hydronephrosis
- Other imaging: KUB X-ray/ CT (mass, stone), MRI (neurological)

MANAGEMENT

- Treatment is to pass a fine urethral catheter (14F-Frech gauge is defined as the circumference in millimeters) and arrange urological management to treat underling cause.
  - Wash hands thoroughly and don sterile gloves.
  - Clean genitalia using soapy antiseptic
  - Lignocaine gel is inserted into the urethra, warning the patient that it may sting. The jelly should be massaged posteriorly in an attempt to anesthetize the sphincter region and it is of advantage to place a penile clamp for several minutes.
  - A small Foley catheter should be passed while the penis is held taut.
  - In female patients the labia should be parted using the middle and index fingers of the left hand which should not be moved once cleaning has been performed.
  - Once urine begins to drain it is wise to pass a few more centimeters of catheter into the bladder before the balloon is inflated to avoid inflation in the prostate. FORCE MUST NOT BE USED.
- In postoperative retention a warm bath can help.
- After catheterization:
  - Record the volume of urine drained
  - Examine the abdomen to exclude other pathology (rupture of an aortic aneurysm, ureteric colic or diverticulitis can cause confusion)

- If the catheter will not pass, it is usually due to poor technique, lack of anesthesia, traumatization of the urethra or a urethral stricture.
- Catheter should be placed for at least 3 days.
- Suprapubic puncture:
  - Suprapubic puncture with commercially available catheters such as Cystofix or Lawrence Add-a-Cath catheter is straightforward provided that the bladder is palpable.
  - The skin, fascia and retropubic space are anesthetized with 0.5% lignocaine. Correct placement is confirmed by aspiration.
  - A large-bore needle is placed into the bladder, down which a fine catheter is passed (Cystofix) and then secured in position. The other option is to place a plastic suprapubic trocar and cannula which has a removable plastic strip on the side.
  - A standard 12F Foley catheter can be passed down the cannula, the balloon is inflated, the cannula is extracted and the strip is pulled away from the catheter (Add-a-Cath)
  - If urine cannot be aspirated through the fine-bore needle, passing a suprapubic trocar should not be attempted.
  - If these devices are not available a catheter can be placed in the bladder under direct vision through a small incision under local anesthetic.
- Urethral instrumentation in a patient with a known stricture should be done by an experienced urologist that may elect to dilate the stricture or take the patient to theatre to carry out an optical urethrotomy.

CHRONIC URINARY RETENTION

- In chronic retention there is no pain.
- These patients are at risk of upper tract dilation because of high intravesical tension- they require urgent urological referral.
- Men with impaired renal function may develop post-obstructive diuresis following catheterization. Such men need careful monitoring with replacement of inappropriate urinary losses by intravenous saline, they are also at risk of hematuria as the distended urinary tract empties.
- Often it is several days before full renal recovery occurs.

URINARY TRACT OBSTRUCTION

- Obstruction of the urinary tract may occur at any level from the external urinary meatus to the ureteropelvic junction (junction between the renal pelvis and the ureter).
- Obstruction may be classified according to site as:
  - Upper urinary tract obstruction: bladder and uppers
  - Lower urinary tract obstruction: distal to the bladder

- Within each type of obstruction further sub-classification can be done including:
  - Etiology: congenital vs acquired
  - Duration: Acute vs Chronic
  - Degree of obstruction: Partial vs complete

UPPER URINARY TRACT OBSTRUCTION

- Upper urinary tract obstruction may result in renal impairment especially if the obstruction is prolonged.
- The kidney responds to distal obstruction by a decrease in ipsilateral renal blood flow with a concomitant increase in contralateral renal blood flow.
- Other protective mechanisms:
  - Ureteric dilation: this dissipates the increased pressure to some extent
  - Urine reabsorption: through renal lymphatic and collecting ducts (pyelointerstitial back-flow)
  - Papillary shutdown
- Continued unrelieved obstruction produces renal impairment due to a combination of pressure and ischemic atrophy.
- Upper urinary tract obstruction can be:
  - Acute: symptomatic with loin pain
  - Chronic: asymptomatic or produce vague symptoms
- Diagnosis depends mainly on IVU with judicious application of specialized radiographic techniques such as isotopic renography, ascending ureterography and perfusion studies (Whitaker test).
- On basis of clinical and radiological findings obstruction may be found to be
  - Unilateral or bilateral
  - Complete or partial
  - Acute or chronic

UNILATERAL UPPER TRACT OBSTRUCTION

- Lesions can be
  - Intraluminal: within the ureteric lumen
  - Intramural: within the ureteric wall
  - Extramural: outside the wall of the ureter compressing it

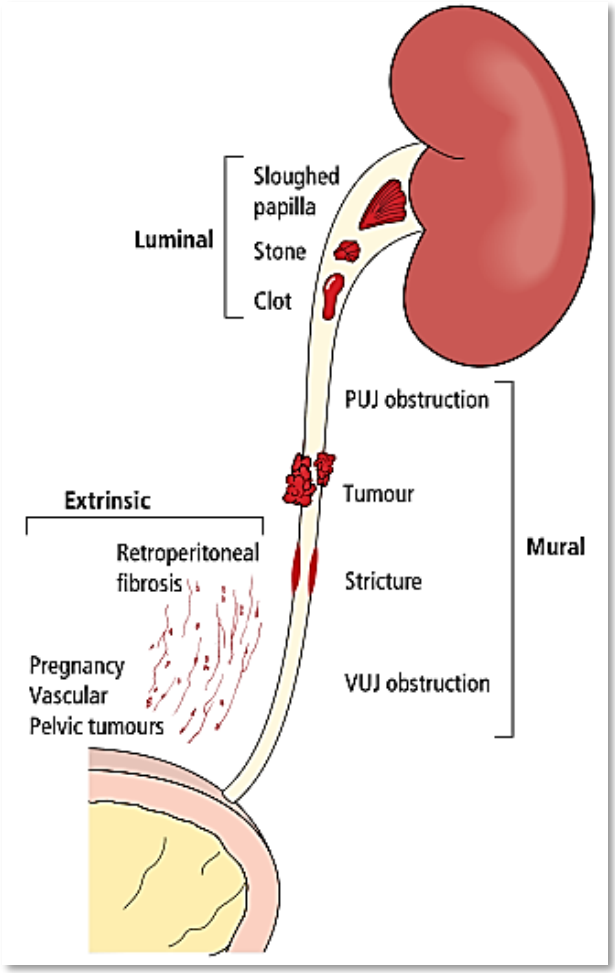


Figure 241: Causes of unilateral ureteric obstruction

INTRALUMINAL

- Causes:
  - Calculi (most common): presents with acute ureteric colic associated with hematuria. A plain abdominal film demonstrates the calculus in 90% of patients and IVU will confirm the diagnosis and degree of obstruction. (Management covered earlier)
  - Clots from bleeding in the renal pelvis or kidney (transitional cell carcinoma, renal cell carcinoma): may mimic ureteric colic but findings of X-ray of a filling defect in the renal pelvis or distorted calyces gives a clue to the correct diagnosis.
  - Renal papillary necrosis: is uncommon but the sloughed papilla may obstruct the ureter mimicking the clinical picture of ureteric stone. Causes include sickle cell disease, diabetes mellitus, and chronic use of NSAIDs. Symptoms are pain associated with hematuria and sterile pyuria. A typical IVU shows defects in the medulla where the papillae were and a non-calcified obstructing ureteric lesion. Sloughed papillae are managed like ureteric calculi.

INTRAMURAL OBSTRUCTION

- Congenital obstruction of the pelviureteric junction:
  - This is a common ureteric abnormality caused by a failure of transmission of peristalsis from the pelvis to the ureter.
  - This results in a rise in intrapelvic pressure as the renal pelvis and calyces dilate.
  - Gradual deterioration of renal function on the affected side is the rule.
  - The condition may be asymptomatic or it may cause loin pain especially after an increased fluid load (Dietl's crises).
  - IVU will show dilated calyces and pelvis with obstruction at the level of the pelvic-ureteric junction (PUJ). The ureter is not visible, if it can be seen on IVU then the diagnosis of PUJ obstruction is incorrect.
  - A renogram with diuretic will confirm the obstructive nature of the lesion and give rough estimate of renal function on the obstructed side.
  - Treatment is surgical. If renal function is poor, nephrectomy is the most appropriate treatment provided the condition is not bilateral. If renal function is good then a reconstructive operation (pyeloplasty) is done.
- Obstruction of the ureterovesical junction: Obstruction may also occur at the ureterovesical junction (obstructed megaureter) and failure of transmission of peristalsis at the junction is believed to be the mechanism. Pain, hematuria and infection are usual presenting symptoms. IVU shows a dilated ureter with less dilated proximally and blunting of the calyces. A diuretic renogram to confirm obstruction is indicated. Treatment consists of reimplantation of the ureter after excision of the diseased distal portion.
- Ureteric strictures: may occur after ureteroscopy and stone manipulation, pelvic surgery, irradiation (for carcinoma of the prostate, cervix or bladder) or chronic inflammatory conditions (tuberculosis or schistosomiasis)
- Ureteric tumors: account for 1% of all urothelial tumors and may present as ureteric pain with hematuria mimicking a stone. Diagnosis depends on urine for cytology and IVU. Treatment consists of nephrouretectomy and regular follow-up cystoscopy as recurrences in the bladder are common.

EXTRAMURAL OBSTRUCTION

- Extramural obstruction of the ureter is rare.
- Can be caused by:
  - Retroperitoneal fibrosis
  - Pregnancy

- Pelvic tumors

BILATERAL UPPER TRACT OBSTRUCTION

- Causes include:
  - Retroperitoneal and pelvic tumors: may obstruct both ureters, most tumors in the retroperitoneum are malignant, invasion arising from the cervix, prostate, bladder, breast, colon, ovary or uterus. The remainder are primary retroperitoneal tumors such as lymphoma or sarcoma. When treatment of the underlying tumor is feasible the ureters may be reimplanted or urinary diversion may be considered.

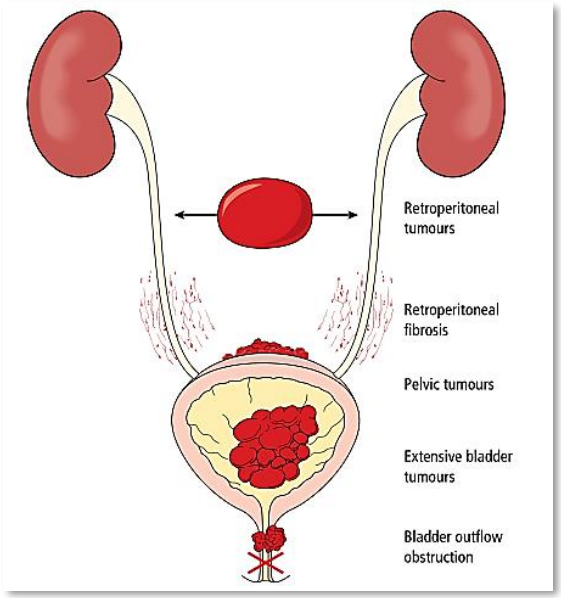


Figure 242: Bilateral causes of upper tract obstruction

  - Retroperitoneal fibrosis:
    - This is usually idiopathic though in some cases is secondary to malignant disease of the retroperitoneum, irradiation-induced fibrosis, or drugs such as methysergide or beta blockers.
    - As the fibrotic plaque develops the ureters are pulled towards the midline and progressively obstructed. This may present as backache and gradual renal impairment. Hypertension and signs of distal venous occlusion are frequently associated.
    - Renal failure occurs in some cases.
    - Men are affected more than women and the disease usually presents in middle age.
    - Lab investigations may reveal renal function impairment, anemia and a raised ESR.
    - An IVU will show deviation of the ureters towards the midline and hydronephrosis.
    - CT guided biopsy of the plaque is necessary to rule out underlying malignancy.
    - Initial treatment consists of relieving the ureteric obstruction, although freeing the ureters from the fibrous plaque (ureterolysis) and wrapping the

ureters in omental tubes to prevent recurrence may occasionally be undertaken.

- Extensive bladder tumors
- Bladder outflow obstruction

LOWER URINARY TRACT OBSTRUCTION

- Outflow obstruction of the lower urinary tract remains one of the most prevalent and clinically significant diseases in urology.
- Symptoms referable to the lower urinary tract are categorized into:
  - Obstructive:
    - Weak stream
    - Hesitancy
    - Intermittency
    - Dribbling
    - Straining to void
  - Irritative:
    - Urinary Frequency
    - Urgency
    - Nocturia
    - Dysuria
    - Urge incontinence
- The term 'prostatism' refers to a combination of these symptoms including frequency, urgency, hesitancy and weak stream.
- While the name implies that prostatic obstruction is the cause, this is not always the case as bladder instability in patients with no evidence of obstruction can present in an identical fashion.
- The differential diagnosis of lower urinary tract obstruction can be classified as:
  - Structural causes
    - Congenital
      - Meatal stricture
      - Posterior urethral valves
    - Acquired
      - Urethral stricture
      - Phimosis
      - BPH
      - CA prostate
      - Bladder neck contracture
      - CA bladder

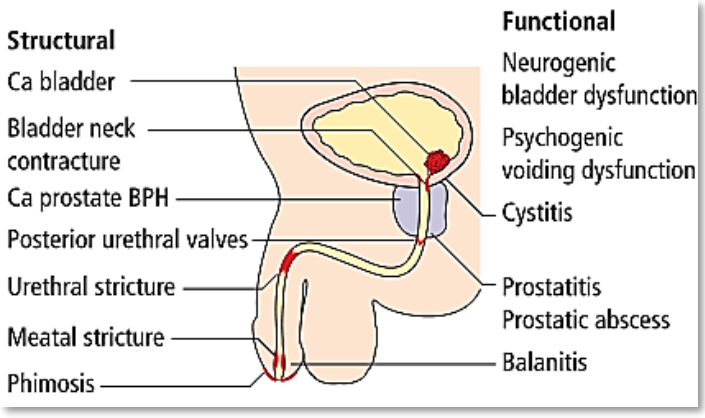


Figure 243: Differential diagnosis of lower urinary tract obstruction

- Functional causes
  - Neurogenic bladder dysfunction
  - Psychogenic voiding dysfunction
  - Cystitis
  - Prostatitis
  - Prostatic abscess
  - Balanitis

CONDITION	FEATURE
STRUCTURAL CAUSES	
Meatal stricture	• Obstruction of the distal urethra • Typically seen in newborn or infant males • There is usually a congenital web of epithelial tissue permitting only a pinpoint opening the meatus.
Phimosis	• In uncircumcised males, chronic inflammation and scarring of the prepuce (Phimosis) can cause obstruction of urine
Urethral stenosis and strictures	• Urethral stenosis in females is rare and may be related to trauma or inflammation • Urethral stricture is a frequent problem seen in adult males. Although a common sequel to gonococcal urethritis in the past, the majority of strictures now seen are the result of trauma to the urethra from instrumentation or catheterization
Posterior urethral valves	• These are congenital mucosal folds in the area of the membranous urethra • They are the most common cause of proximal urethra obstruction in male infants. • In adults, obstruction in this region can be both functional and anatomical. Failure of the external urethral sphincter to relax secondary to spasm during micturition will impede flow. • Several neurological diseases including spinal cord injury and multiple sclerosis as well as psychogenic causes will result in this type of obstruction.
Ca prostate	• This is a less common cause of obstruction as most cancers occur in the peripheral regions of the prostate
BPH	• This is the most common cause of obstruction in adult males • It tends to occur in the periurethral transition zone.

Bladder neck contracture	• Contracture of the bladder neck is a common cause of obstruction and typically seen secondary to surgery or trauma • Neurological and idiopathic dysfunctions are less common and are characterized by failure of the vesical neck to open completely during micturition without anatomical cause. This may mimic BPH but usually occurs in a younger age group. • In female patients who have had vaginal deliveries or pelvic surgery, cystocele may develop causing a range of symptoms from obstruction and retention to urinary incontinence
CA bladder	• Physical obstruction
	FUNCTIONAL CAUSES
Neurogenic bladder dysfunction	• Peripheral neuropathies may adversely affect the detrusor muscle contraction by involving the autonomic nerve supply. • Causes: diabetes mellitus, chronic alcoholism, uremia, Guillain-Barre syndrome and trauma • Certain drugs may exacerbate obstructive symptoms and precipitate retention. These include anticholinergics (phenothiazine and some antianxiety medication) inhibit detrusor contractility, alpha adrenergic agonist (pseudoephedrine, ephedrine and phenylpropanolamine) increase sympathetic tone of the bladder neck and prostatic urethra. • Both temporary and permanent detrusor dysfunction can be seen from prolonged over-distension or ischemia of the bladder (myogenic failure)
Psychogenic voiding dysfunction	• Inability of the bladder to void due to psychological issues
Cystitis	• Infection and inflammation of the bladder
Prostatitis Prostatic abscess	• Infectious processes of the prostate can also lead to obstructive symptoms owing to surrounding inflammation and edema and include acute prostatitis and prostatic abscesses
Balanitis	• Obstruction of the distal urethra due to inflammation of the glans penis

BENIGN PROSTATIC HYPERPLASIA

- Benign prostatic hyperplasia (BPH) is a common benign conditions of the prostate seen in older men.
- It is clinically important because it is the most common cause of bladder outlet obstruction in men over 50 year of age.
- Incidence in men between 60 and 90 years is slightly over 50%. Early changes probably occur between the ages of 30 and 40 years.
- If left untreated, bladder outlet obstruction can lead to urinary tract infection and bladder stones secondary to stasis from incomplete bladder emptying, bladder decompensation resulting in chronic urinary retention with overflow and most serious of all renal failure secondary to high-pressure urinary retention.
- The initial change in BPH is the development of microscopic stromal nodules around the periurethral glands. Around these nodules glandular hyperplasia originates.

SURGICAL ANATOMY

- The prostate is an accessory gland of male reproductive system. It is composed of glandular tissue embedded in fibromuscular stroma. It surrounds the first 3cm of urethra.
- The prostate consist of 3 structures:
  - 5 anatomical lobes
    - 1 Medial/middle lobe
    - 2 lateral lobes
    - 1 anterior and 1 posterior lobe
  - 3 types Secreting glands: secrete into the urethra
    - Urethral glands
    - Submucous glands
    - Prostatic glands (Carcinoma prostate normally arises from these glands)
  - Capsule

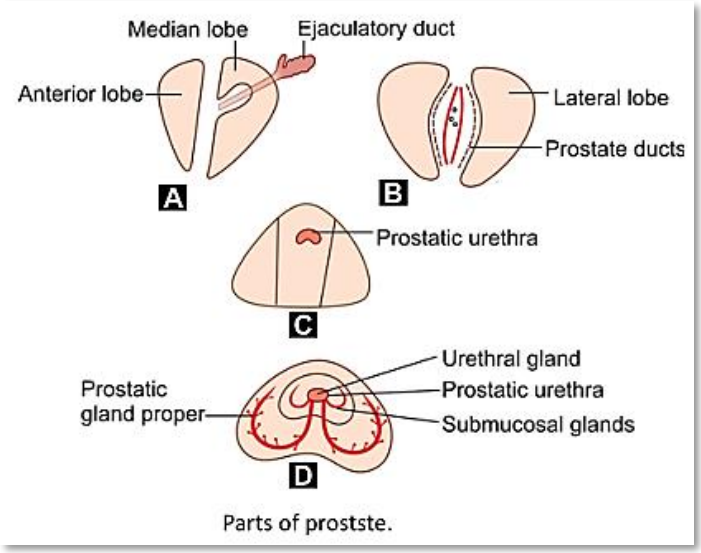


Figure 244: The prostate

- True capsule (encloses all lobes): formed by the condensation of the peripheral parts of the gland, continues with the stroma of the gland
- Prostatic sheath /False capsule (blends with pelvic fascia)
- False capsule only present and formed by compression of the posterior lobe e.g. in BPH

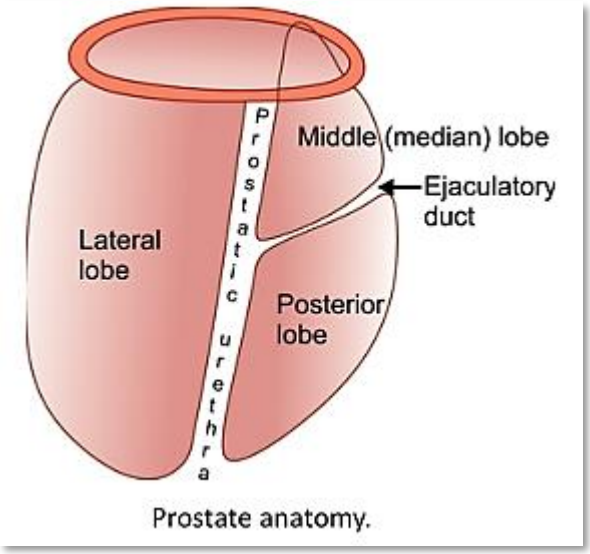


Figure 245: Prostate anatomy

- The prostate according to McNeal divides the prostate into 3 zones:
  - Peripheral zone- prone for carcinoma
  - Periurethral transition zone where BPH arises
  - Central zone
- Note:
  - Prostate carcinoma develops in prostatic gland proper
  - BPH develops in submucosal glands
- Acid phosphatase:
  - This is an enzyme that splits organic phosphates.
  - It is found in many human tissue, but more concentrated in prostate.
  - It is active at pH5
  - Acid phosphatase secreted by prostate drains into the urethra through prostatic ducts and so blood levels of this enzyme remain low.
  - Serum acid phosphatase estimation should be done on empty stomach because heavy meals alter the level of the acid phosphatase
  - Normal value= 0-5 King Armstrong units per 100ml of serum
  - It is raised significantly in carcinoma prostate with metastases
  - It does not increase in BPH.
  - Slight increase in acid phosphatase level occurs in acute phosphatase level occurs in acute prostatitis, Paget's disease of bone and hepatic cirrhosis.
  - Prostatic fraction of acid phosphatase is more relevant in carcinoma prostate.
- Prostate specific antigen
  - It is a protease produced from the prostatic epithelium secreted in the semen to cleave and liquefy the seminal coagulum formed after ejaculation
  - PSA is organ specific.
  - Normal value=4ng/ml of plasma (more than 10ng/ml is significant)

- PSA elevation is seen in carcinoma, prostatic hyperplasia and prostatitis. Increase is much more in carcinoma than in benign conditions.
- PSA is in 2 forms:
  - Major (bound form): increases in carcinoma
  - Minor (free form): increases in benign conditions
- PSA density i.e. PSA level per gram of prostate tissue is more relevant
- Serial estimation of PSA is very useful to suspect spread and recurrence after treatment
- 25% of men with PSA 4-10 ng/ml show prostate carcinoma
- 20% of men with normal PSA (1-4ng/ml) will show prostate carcinoma

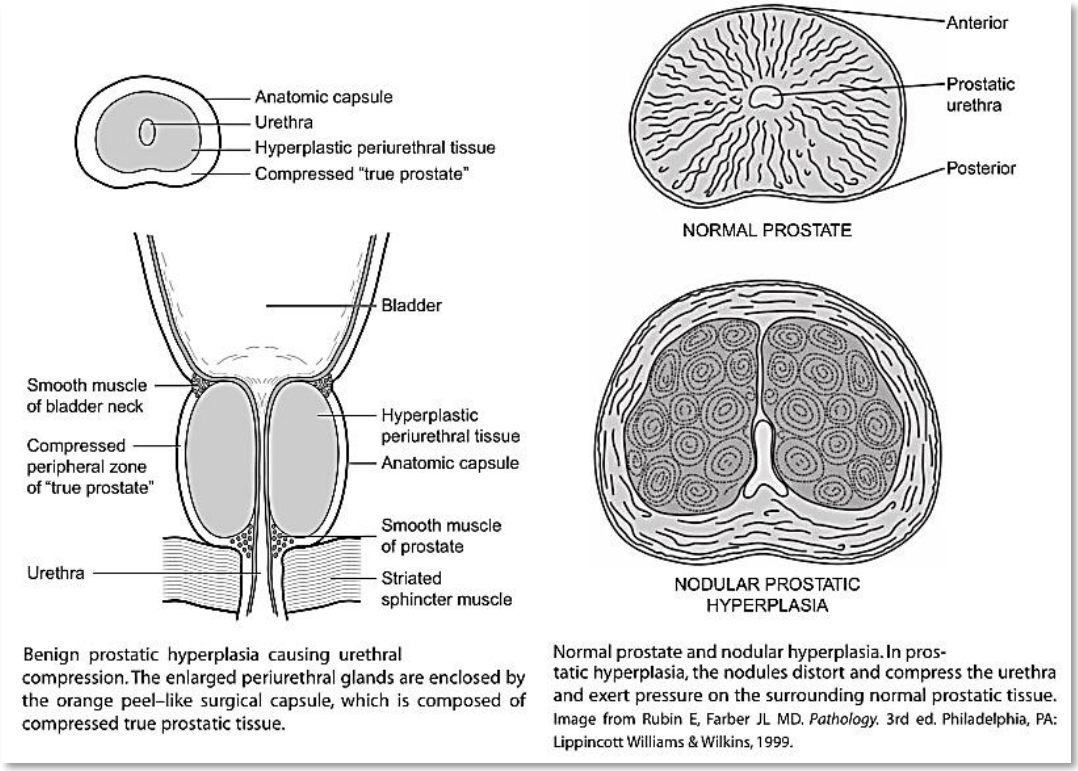


Figure 246: BPH

PATHOGENESIS

- The etiology of BPH is uncertain, although it is probably multifactorial with a hormonal element present.
- The gland is influenced by steroid hormones.

- Recall that the hypothalamus produces luteinizing hormone releasing hormone which stimulates the anterior pituitary lobe to produce luteinizing hormone.
- Luteinizing hormone in turn causes the testes (Leydig cells) to produce testosterone.
- Testosterone is convert to dihydrotestosterone (DHT) and this stimulates prostatic hyperplasia.
- Additionally, estrogen has a negative feedback on the hypothalamic-pituitary axis.
- The exact mechanism of BPH remains unclear.
- Interestingly, BPH does not occur in castrated men or pseudohermaphrodites both whom lack the active metabolite of testosterone, dihydrotestosterone (DHT).
  - The enzyme that catalyze the conversion of testosterone to dihydrotestosterone is 5-alpha reductase.
- Estrogens have also been implicated in prostatic hyperplasia, because in aging men, the levels of estrogen rise and those of androgens fall.
- The specific area of cellular hyperplasia is the transitional zone or periurethral area of the prostate.
- The periurethral glandular elements undergo hyperplasia causing an increase in glandular mass that results in compression of the prostatic urethra and the onset of obstructive symptoms.

RISK FACTORS

- Age >50 years
- Hormonal: increase in estrogen and decrease in androgens.
- Neoplastic: fibrosis, adenosis, myosis (fibromyoadenoma)

PATHOLOGY

- Prostate grows into the bladder
- Prostate grows outside bladder
- Effects: BPH induces gradual changes in the urinary tract which result from interactions between prostatic urethral resistance and intravesical pressure.
  - Urethral:
    - Compressed laterally
    - Enlongated (from 1.5” to 4”)
    - Increased curvature
  - Bladder
    - Hypertrophy of bladder (trabeculations)
    - Sacculation (diverticulum)
  - Ureter and Kidney
    - Hydroureter
    - Hydronephrosis
    - Renal failure

CLINICAL MANIFESTATIONS

HISTORY

- Variable symptoms. Symptoms are both obstructive and Irritative.
- Any older man presenting with obstructive urinary symptoms must be suspected of having BPH.
- Symptoms include:
  - Obstructive symptoms:
    - Urinary hesitancy: due to prolonged time required for the detrusor muscles to overcome the increased urethral resistance
    - Decreased force of urinary stream- poor urinary stream due to compression of the urethra.
    - Intermittency, terminal dribbling and sensation of incomplete bladder voiding: when detrusor muscle can no longer maintain the increased pressure required to empty the bladder.
  - Irritative symptoms:
    - Frequency- earliest and commonest due to irritation of the bladder trigone, residual urine in post prostatic pouch and cystitis.
    - Nocturia: when patients are asleep, cortical inhibition is decreased as is urethral tone, leading to nocturia.
    - Urgency- due to stretching of internal sphincter by enlarged gland, urine trickle and irritate the prostatic urethra.
    - Dysuria- pain on urination
    - Incontinence is a late finding in BPH. When large volumes of residual urine accumulate in the bladder and overcome urethral resistance this is called overflow incontinence.
- Acute urinary retention may develop as a result of a precipitation factor aggravating existing BPH e.g. drugs (alpha adrenergic drugs, anticholinergics, and psychotropic agents), alcohol, cold and infection
- Secondary symptoms:
  - High postvoid residual volumes promote bacterial growth, leading to urinary tract infection.
  - Stasis can also promote the formation of bladder calculi.
  - Most seriously, high pressure chronic retention can cause bilateral hydroureteronephrosis and subsequent renal failure.

- Other symptoms include hematuria from vesical piles, increased libido which is followed by impotence.

PHYSICAL EXAMINATION

- Physical signs attributable to BPH tend to occur late in the disease.
- Rectal examination reveals and enlarged symmetric rubbery gland (smooth surface).
  - Right and left lobes are often not discernible although asymmetry is common while consistency of the gland may either be soft or firm depending on whether there are more glandular or fibromuscular elements.
  - Seminal vesicles are usually not palpable with enlarged glands.
  - If irregularities of the prostate are felt on digital rectal examination including firm nodules, induration or generally hard, prostate carcinoma is more likely and it requires further investigations
  - The size of the gland has no relationship to symptomatology. A small gland may produce a high degree of outflow obstruction whereas a large gland may produce no symptoms at all.
- The suprapubic region should be palpated to rule out a grossly distended bladder.
- Examination of the abdomen may reveal a distended bladder or occasionally a palpable kidney and flank tenderness from hydronephrosis or pyelonephritis.
- In advanced cases signs of uremia secondary to renal failure may be present: Hypertension, tachycardia and tachypnea from metabolic acidosis and anemia as well as neurological changes and uremic fetor.
- Urinalysis should be done to look out for evidence of infection and hematuria. If hematuria exists other causes referable to the urinary tract should be excluded.

DIAGNOSIS

- Urine microscopy, culture and sensitivity- infections
- Serum urea, electrolytes and creatinine levels should be checked for evidence of renal insufficiency.
- Full blood count- anemia
- Prostate specific antigen (PSA) to rule out CA prostate.
- Kidney ureter and bladder (KUB) X ray- stones (stasis)
- Intravenous urogram and KUB ultrasound- hydronephrosis, hydroureter, residual urine, diverticulae.
- Residual volume measurement- the patient empties bladder then catheterize them (normal residual ~ 30mls), residual volume greater than 100ml would signify failure of the bladder to empty completely.
- Cystoscopy- it can be done to evaluate BPH prior to transurethral resection of the prostate. It includes evaluation of the ureteric orifices, presence of bladder tumors or stones, size and length of the prostate and condition of the urethra.
- Urodynamic studies: Urinary flow rate
  - A flow rate of less than 10ml/sec is evidence of bladder outlet obstruction, BPH, detrusor instability, neuropathic bladder (diabetes, parkinsonism, Alzheimer, CVA or neurosyphilis)

TREATMENT

MEDICAL TREATMENT

- Goal of drug therapy is to relax smooth muscle in the prostate and bladder neck or to induce regression of cellular hyperplasia thereby enhancing urinary outflow from the bladder to the urethra.
- Alpha blockade of adrenergic receptors produces smooth muscle relaxation of both prostate and bladder neck.
  - An infrequent side effect to alpha-antagonist (e.g. terazosin, tamsulosin) is postural hypotension (2-8%).
  - Tamsulosin 400 micrograms PO OD
  - Useful in small prostates causing obstructive uropathy due to BPH
- Prostatic hyperplasia can also be treated with 5-alpha reductase inhibitor (e.g. finasteride) that block the conversion of testosterone to DHT without lowering the serum levels of circulation testosterone.

- Effectiveness of 5-alpha reductase inhibitors is less than half that seen with alpha blockers.
- Useful in big prostates causing obstructive uropathy

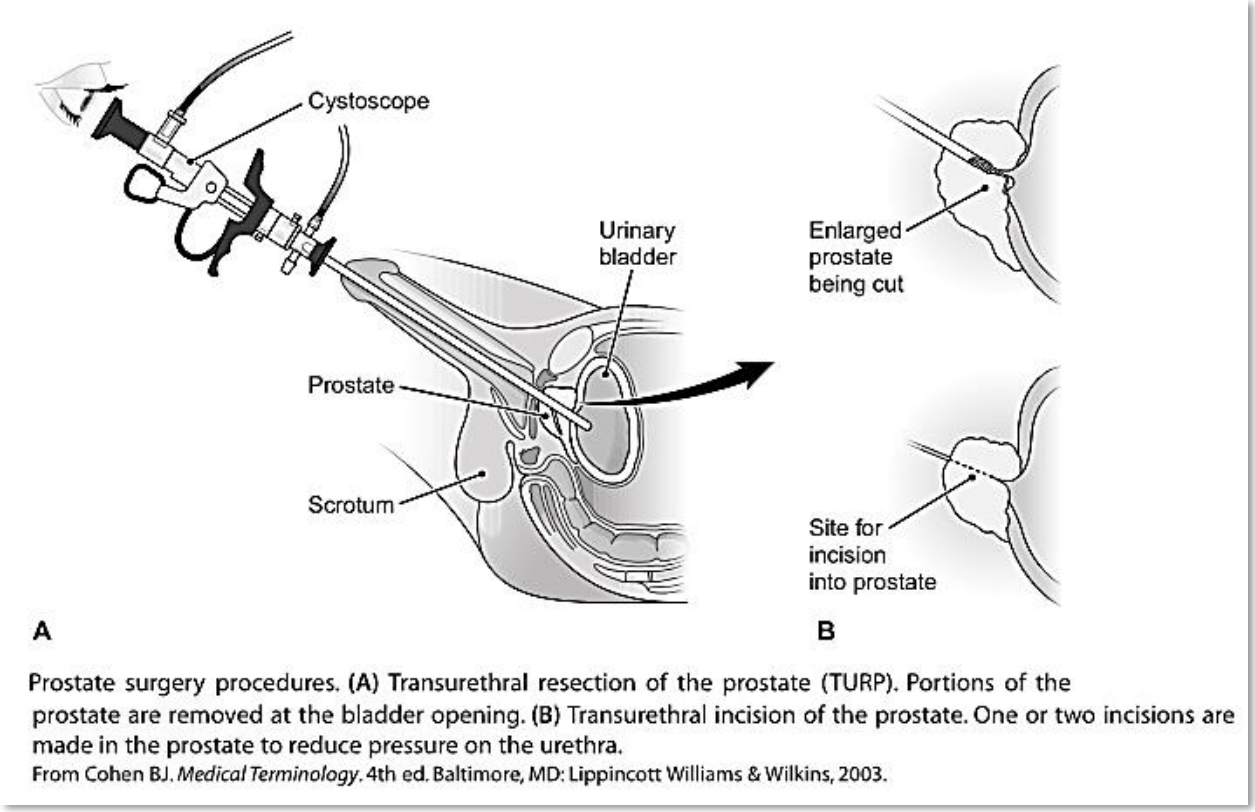
- Both drugs are usually taken for 6 moths- 1 year to see effects
- There is need to follow up the patient.
- Finasteride 5mg daily, review treatment at 3-6 months and then every 6-12 months (may require several months treatment before benefit is obtained)
- Other medical interventions:
  - Intermittent self-catheterization
  - Transurethral balloon dilatation of the prostate
  - Microwave hyperthermic prostatic therapy
  - Prostatic urethral stents
  - Transurethral needle ablation
  - High-intensity focused ultrasound.

SURGICAL

- Surgical relief of obstruction is necessary when medical therapy fails.
- Indications:
  - Failed medical treatment
  - Complications (bleeding hydronephrosis, uremia, bladder calculi, urinary sepsis, acute or chronic urinary retention)
  - Post-void residual volume >100mL
  - Acute urinary retention
  - Chronic urinary retention with overflow dribbling
  - Gross hematuria on more than one occasion
  - Recurrent urinary tract infection
  - Patient request for restoration of normal voiding pattern because of excessive nocturia or dribbling.
- Procedures include:
  - Open method- prostatectomy
    - Hospital stay around 10 days
    - Limited for patients with extremely large glands that are not suitable for TURP because of gland size and time required for resection.
    - Greater blood loss and higher morbidity and mortality risk
    - Majority have a good quality of life after prostatectomy.
  - Closed method: Transurethral resection of prostate (TURP)- preferred
    - Hospital stay around 5 days

- The adenomatous portion of the prostate is removed via a resectoscope using electrocautery from within the prostatic urethra.

- Open method can be done through
  - Transversical prostatectomy (Wilson Hey procedure)
  - Retropubic prostalectomy (Millin procedure)
  - Perineal prostalectomy (Young procedure)- not practiced.
- The procedure of choice is transurethral resection of the prostate.
  - With the patient in the lithotomy position, the resectoscope is introduced via the urethra into the bladder.
  - The occlusive prostate tissue is identified and under direct vision, the tissue is shaved away using an electrified wire loop.
  - As the bladder is constantly irrigated with non-electrolytic isotonic solution containing sorbitol or glycine, extravasated blood and tissue fragments are evacuated.
  - An indwelling catheter is left in place for 1-7 days.



Prostate surgery procedures. (A) Transurethral resection of the prostate (TURP). Portions of the prostate are removed at the bladder opening. (B) Transurethral incision of the prostate. One or two incisions are made in the prostate to reduce pressure on the urethra.
From Cohen BJ. *Medical Terminology*. 4th ed. Baltimore, MD: Lippincott Williams & Wilkins, 2003.

Figure 247: Transurethral resection of the prostate

POST OPERATIVE CARE

- Establish continuous drainage with normal saline.
- High urine output (essential)- oral fluids and IV fluids.
- Early ambulation to avoid DVT and hypostatic pneumonia.

COMPLICATIONS OF PROSTATECTOMY

- Early
  - Hemorrhage (lavage, clot retention)
  - Bladder tamponade-this is an emergence insert 22-24G catheter to decompress
  - Hyponatremic hypochloremic metabolis acidosis (TURP syndrome) due to absorption of irrigation fluid containing sorbitol or glycine via venous sinuses causing hyponatremia and metabolic acidosis in 2% of patients
- Intermediate:
  - Infection (UTI)- broad spectrum antibiotics
  - Incontinence of urine- physio, investigate for damage to internal urethral sphincter
- Late:
  - Urethral stricture
  - Renal failure
  - Sterility- retrograde ejaculation
  - Impotence
  - Bladder neck contractures

CARCINOMA PROSTATE

- This is the most common malignant tumor in men over 65 years.
- It is the second most frequent cause of cancer deaths.
- It occurs in the peripheral zone in prostatic gland proper i.e. commonly in posterior lobe. So prostatectomy from BPH does not provide protection against the development of carcinoma prostate.
- Incidence is 70% in men over 80.

ETIOLOGY

- The following have been suggested as possible etiological factors in the development of prostatic carcinoma:
  - Age
  - Race
  - Environmental factors
  - Diet
  - Nationality
  - Endocrine environment
  - Viral infection

PATHOLOGY

- The most common type of prostate tumor is the adenocarcinoma which arises from the glandular epithelium. It comprises 95% of tumors.
  - It arises in the outer part of the prostate gland or peripheral zone. As the tumor enlarges it spreads medially into the remainder of the gland and outwardly to the surrounding tissue particularly the seminal vesicles.
  - Invasion of the rectum is uncommon as it is protected by the fascia of Denonvilliers. The tethering of the rectal mucosa that may occur does not indicate invasion of the rectum but rather of the fascia beneath it.
- Grading of carcinoma is based on de-differentiation as proposed by Gleason (Gleason grading system):
  - A tumor is assigned a single grade on a scale of 2-10
  - 8-10 indicates a poorly differentiated tumor
  - 2-4 indicates a well-differentiated tumor
- A Gleason score more than 7 carries poor prognosis.
- Types of carcinoma prostate:

- Microscopically latent
- Tumors incidentally found either by TURP or by PSA estimation
- Early localized carcinoma
- Advanced local prostatic carcinoma
- Metastatic carcinoma either into the bone commonly or other organs

CLINICAL FEATURES

- Commonly asymptomatic
- Most patients will present with symptoms of bladder outflow obstruction:
  - Poor stream
  - Hesitancy
  - Nocturia
  - Incomplete bladder emptying
- Sometimes the tumor may present as acute urinary retention
- 40% of patients may present with symptoms of advanced prostatic carcinoma caused by either
  - Ureteric obstruction
  - Bony metastasis: the pain occurs classically at night, waking the patient from sleep. The pain may be relieved by getting out of bed and walking for a while
- On examination
  - Prostate may feel normal
  - On per rectal examination, prostate feels hard, nodular, irregular often with loss of median groove.
  - In localized prostatic carcinoma there may be a hard nodule in one of the lobes or a hard area which has spread into the opposite lobe but with no evidence of spread outside the gland
  - If digital rectal examination of prostate reveals the presence of locally invasive prostate cancer, there may be as hard mass spreading outside the boundaries of the prostate and involving the seminal vesicles or the side wall of the pelvis. In this instance, the examining finger may feel a hard prostate that may be irregular and associated with a fixed gland and sometimes tethering of the rectal mucosa.

DIFFERENTIAL DIAGNOSIS

- Other causes of retention urine
- Other cause of back pain

SPREAD

- It spreads via 2 ways:
  - Local spread:
    - Upward into seminal vesicles, bladder neck, trigone, later into both ureters causing anuria.
    - Downward into distal sphincter.
  - Blood spread:
    - Into bones commonly the pelvic bone, lumbar vertebrae, femoral head, ribs, skull- in that order.
    - Pathological fractures can occur in long bones and vertebrae. Paraplegia may occur if spine is involved.
    - Rarely spread to the liver and lungs can occur.
  - Lymphatic spread:
    - Into the obturator lymph nodes, then to internal iliac lymph nodes.
    - Through seminal vesicles into external iliac and retroperitoneal lymph nodes.
    - Eventually mediastinal, left supraclavicular lymph nodes get involved.

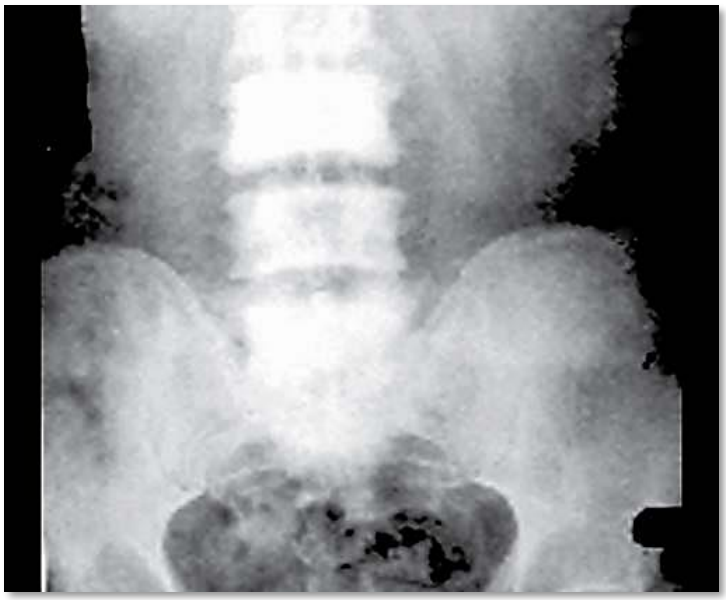


Figure 248: A plain X-ray AP view of lumbar spine in a patient with prostate carcinoma. Note the typical osteoslcerotic secondaries

INVESTIGATIONS

- These include:
  - Full blood count and peripheral smear: in metastatic disease there may be leucoerythroblastic reaction with bone marrow invasion causing anemia, thrombocytopenia, DIC and increased fibrinogen degradation products. Anemia may also be due to renal failure
  - Kidney function tests (Blood urea, serum creatinine and electrolytes)
  - Liver function test
  - Prostate specific antigen (PSA): more than 10nmol/ml is suggestive
  - Prostatic fraction of acid phosphatase is increased
  - Chest X-ray
  - Imaging:
    - Plain X-ray, KUB: may show dense coarse sclerotic secondaries. Osteolytic or combination of lytic and sclerotic lesions are also often seen.
    - Transrectal ultrasound-TRUS (including guided needle biopsy of the prostate)
    - Isotope bone scan for the presence of bony metastases
    - MRI to evaluate local invasion and regional lymphadenopathy
    - U/S abdomen to see tumor extension into the bladder and to see kidneys for hydronephrosis
    - Technetium radioisotope bone scan to see secondaries
  - Transrectal prostatic biopsy. 10 biopsy cores are taken.

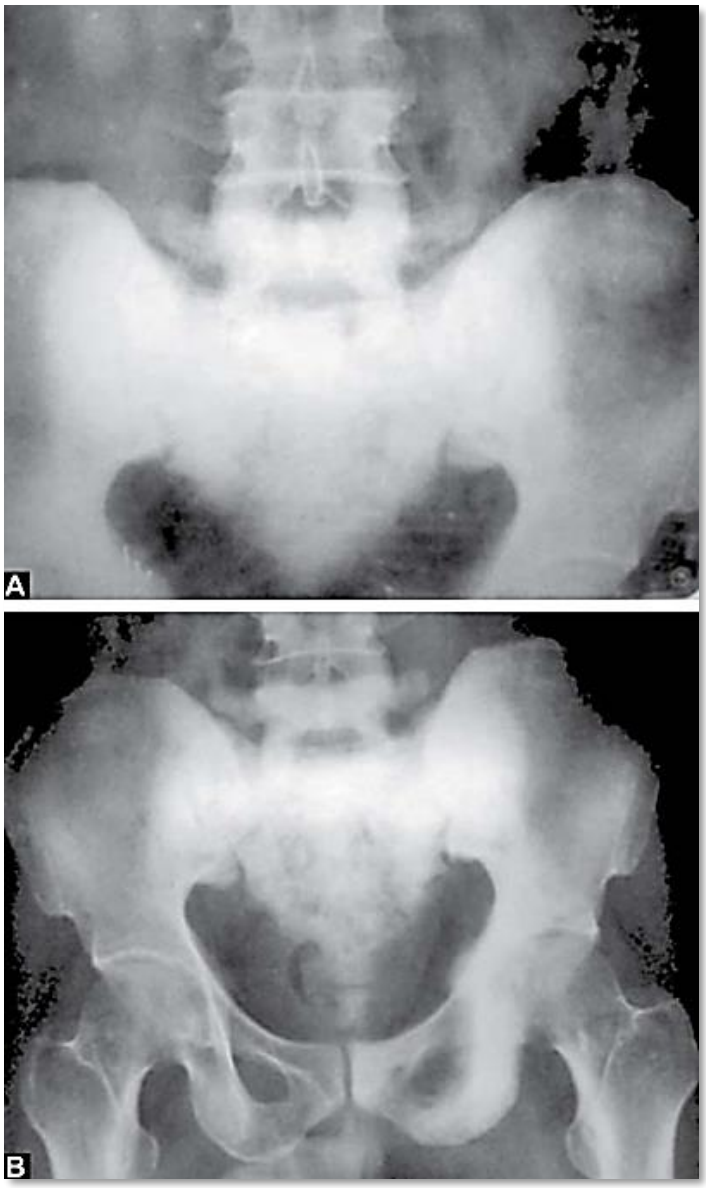


Figure 249: Osteoblastic secondaries in pelvic boens and lumbar spine

MANAGEMENT

- Management is dependent on staging (TMN staging)
  - T1- incidentally confirmed carcinoma after prostatectomy
    - T1a- tumor occupying less than 5% of specimen
    - T1b- tumor occupying more than 5% of specimen
    - T1c- tumor impalpable but suspected by high PSA
  - T2- tumor nodule palpable on rectal examination
    - T2a- single nodule palpable on digital examination within prostate capsule
    - T2b- nodule involving both lobes
  - T3- Tumor extends through the capsule
    - T3a- extension through capsule one or both
    - T3b- extension to seminal vesicles
    - T4- tumor which is fixed or spread to adjacent structures like pelvic wall and rectum other than seminal vesicles

Note:
For T1a progress rate is slow—15-20% in 8 years
For T1b and T2 it is 35%
For T3/T4 M0 it is 50% in 5 years with bone spread
For M1 average survival is 3 years

- Staging of carcinoma prostate
  - Occult- diagnosed after investigation due to suspicion
  - Stage I (T1)- tumor confined to prostate/local nodule
  - Stage II (T2)- tumor involving capsule or diffuse type
  - Stage III (T3)- Tumor involving seminal vesicle
  - Stage IV (T4)- extension into adjacent tissue

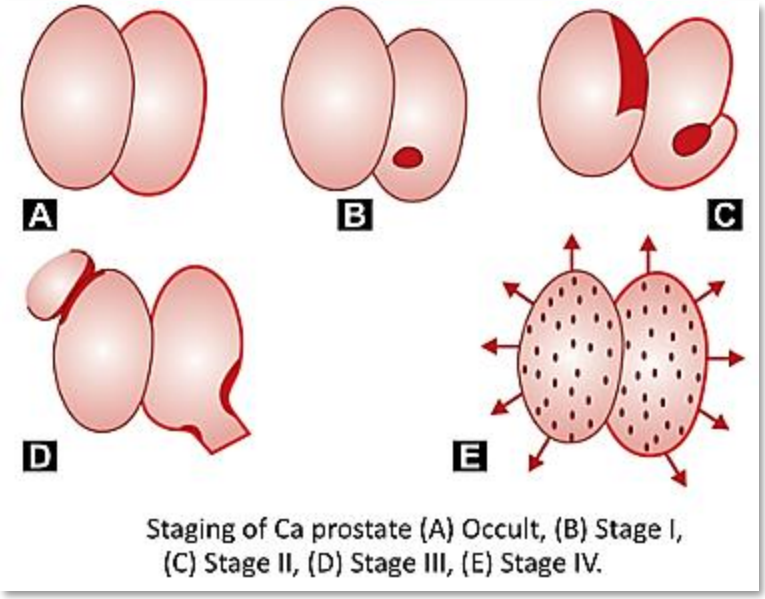


Figure 250: Staging of prostate carcinoma

- If a carcinoma of the prostate is suspected, tissue must be removed for histological examination and confirmation of diagnosis by:
  - TURP (especially with outflow tract obstruction present)
  - TRUS-guided needle biopsy

UNSUSPECTED CARCINOMA OF PROSTATE (STAGE T0)

- If the prostate feels normal on examination but the specimen from TURP has revealed well-differentiated tumor in some of the removed tissue, the recommended method is:
  - Observe patient
  - Regular digital rectal examination (or ultrasound)
  - Regular assessment of serum PSA

LOCALIZED PROSTATIC CARCINOMA (STAGES T1 AND T2)

- There are 3 treatment options:
  - Radical prostatectomy: the prostate, seminal vesicle, distal sphincter are removed along with reconstruction of the urethra. It associated with very low incidence of urinary incontinence and impotence
  - Radical radiotherapy: may be associated with cystitis and proctitis
  - Interstitial irradiation with ^{125}I and ^{198}Au
- The long term survival of patients treated by irradiation or radical surgery is comparable.

LOCAL SPREAD (STAGES T3 AND T4)

- External-beam radiotherapy is the treatment of choice.
- In some cases, particularly those with incipient ureteric obstruction, endocrine therapy may be required in addition to irradiation. Methods of therapy include:
  - Luteinizing hormone- releasing hormone agonists (leuprolide, Goserelin)
  - Antiandrogens (cyproterone or flutamide)
  - Diethylstibestrol
  - Progestational agents

MESTASTIC DISEASE (STAGES T1-T4, M1)

- The treatment centers on androgen deprivation because the prostate gland is very androgen sensitive.
- Methods of therapy:
  - Luteinizing hormone- releasing hormone agonists (leuprolide, Goserelin)
  - Antiandrogens (cyproterone or flutamide)
  - Bilateral orchidectomy
  - Diethylstibestrol-
    - Initially given intravenously and later orally. It is very effective.

- Initially IV 100mg/ daily later orally 100mg/daily
- Response starts appearing in 48 hours
- Complications: cardiac congestion, DVT, gynecomastia.

➢ Progestational agents

RENAL CARCINOMA

NEPHROBLASTOMA (WILMS TUMOR)

TUMORS OF THE RENAL PELVIS AND URETER

CARCINOMA OF THE BLADDER

TESTICULAR CANCER

CANCER OF THE PENIS

TESTICULAR TORSION

- This is twist of the spermatic cord causing testicular ischemia.
- Peak incidence during the first year of life and at puberty around the age of 13 (When growth rates are the fastest)
- Undescended testis carries a higher risk of ipsilateral torsion.
- Salvage rate is 80-100% if surgical intervention is <6 hours from onset of pain.
- Testicular torsion is frequently preceded by strenuous activity or athletic event.
- Testicular torsion can be intermittent in which pain passes. However, surgery is still advisable especially if testicle(s) have a transverse lie.

PATHOPHYSIOLOGY

- The testicle twists on its spermatic cord, causing obstruction of venous return leading to swelling.
- If obstruction persists venous thrombosis occurs followed by arterial occlusion.
- Infarction ensues quickly in the absence of blood supply.
- "Bell-clapper deformity" seen: insufficient attachment of the testicle to the tunica vaginalis allowing for a pathologically large degree of rotational freedom. This increases risk of torsion.

CLINICAL FEATURES

- Acute onset of severe pain in testicle (making walking uncomfortable), lower abdomen or the inguinal canal.
- Scrotum is elevated, swollen and tender
- May be accompanied by nausea or vomiting.
- Classic signs: high-riding testis with a horizontal lie. However, this is often difficult to distinguish because of degree of swelling in the scrotum
- Presence of a reactive hydrocele.
- Loss of cremasteric reflex (The cremasteric reflex is elicited by stroking the medial aspect of the patient's thigh, resulting in a contraction of the cremasteric muscle elevating the scrotum and ipsilateral testis).
- Prehn's sign: decrease in scrotal pain with elevation of the testicle. This action relieves pain in epididymitis but exacerbates it in testicular torsion.

DIAGNOSIS

- Since epididymitis is most commonly confused with testicular torsion, several tests may aid in distinguishing the two:
  - ➢ Urinalysis and FBC are usually unremarkable in testicular torsion. Most patients will be afebrile.
- Since the ability to salvage the affected testis is dependent on how quickly the testis becomes destorsed, time is of the testis! If the diagnosis is thought to be torsion, surgical exploration is indicated.
- Color Doppler ultrasound (US) or nuclear scintigraphy have become the test of choice, showing the presence or absence of blood flow to the suspected testis.
- ALWAYS suspect torsion in a patient with inguinal/abdominal pain and an empty scrotum.

MANAGEMENT

- Surgical repair is the definitive treatment.
- If the testis is viable, bilateral orchiopexy/orchidopexy (detorsion

and fixation with suture) is performed.

- If the tissue is necrotic, the testicle is removed to prevent gangrene. The contralateral testis undergoes orchiopexy as a prophylactic measure.
- Note: the higher the undescended testicle, the higher the lifelong risk of cancer (irrespective of whether the testicle is brought into the scrotum surgically).

TESTICULAR/SCROTAL MASSES AND SWELLINGS

NEUROSURGERY

Cranial Foramina

- **Optic canal:** Optic nerve and ophthalmic artery.
- **Superior orbital fissure:** Cranial nerves (CN) III, IV, VI, and V_1.
- **Foramen rotundum:** CN V_2.
- **Foramen ovale:** CN V_3.
- **Carotid canal:** Internal carotid artery.
- **Internal acoustic meatus:** CN VII and VIII.
- **Stylomastoid foramen:** CN VII and stylomastoid artery.
- **Jugular foramen:** Internal jugular vein and CN IX–XI.
- **Hypoglossal canal:** CN XII.
- **Foramen spinosum:** Middle meningeal artery and vein.
- **Foramen magnum:** Spinal cord, CN XI (spinal accessory) and vertebral, posterior, and anterior spinal arteries.

Internal carotid artery (ICA) does not produce branches until it enters the petrous bone, where it gives off geographically occult feeders to the middle and inner ear.

Major ICA Branches Visible with Angiography

Meningohypophyseal, inferolateral trunk, ophthalmic, posterior communicating, anterior choroidal, middle cerebral, anterior cerebral.

Anterior Cerebral Artery (ACA) Branches

Medial lenticulostriates, anterior communication, recurrent artery of Heubner, orbitofrontal, frontopolar, pericallosal, callosomarginal.

Middle Cerebral Artery (MCA) Branches

Lateral lenticulostriates, anterior temporal, posterior cerebral, ascending frontal, lateral orbitofrontal, precentral, central, anterior parietal, posterior parietal, angular.

Vertebral Artery Branches

Posterior meningeal, anterior spinal, posterior inferior cerebellar, vertebrals fuse to form the basilar artery.

Basilar Artery Branches

Anterior inferior cerebellar, pontine perforators, superior cerebellar, posterior cerebral.

Posterior Cerebral Artery Branches

Posterior thalamoperforators, medial posterior choroidal, lateral posterior choroidal, thalamogeniculates, inferior temporals, parieto-occipital, calcarine, posterior pericallosal.

HEAD INJURY

- Head injury defined as traumatic damage that affects either the skull or brain.

- Every effort must be made to discover its underlying cause, paying particular attention to:
  - The likely speed of impact
  - Any events that may have led to the injury (e.g. epilepsy, subarachnoid hemorrhage, alcohol consumption)
  - Any events after its occurrence (e.g. vomiting, epilepsy, talking)
- Recall that the components of the cranial cavity include:
  - Brain tissue: makes up 80% of intracranial volume. It is partially compartmentalized by the reflections of dura (falx cerebri and tentorium cerebelli). Cranial nerve III rungs along the edge of the tentorium cerebelli. The brain weighs about 1400g.
  - Blood: the brain receives about 15% of the cardiac output (75ml of blood). The brain is responsible for 20% of total body oxygen consumption.
  - CSF: this is formed primarily by the choroid plexus at a rate of about 500ml/day with 150ml of CSF circulating at a given moment. It cushions the brain. It is drained by the arachnoid villi.
- The Monro-kellie hypothesis entails the pressure-volume relationship between intracranial pressure, volume of CSF, blood and cerebral perfusion pressure (CPP).
  - The sum of the volume of the brain, blood and CSF within the skull must remain constant, therefore, an increase in one of the above must be offset by decreased volume of others.
  - It states that the cranial compartment is incompressible and that the volume inside the cranium is fixed such that any increase in one of the components (blood, CSF or brain tissue) is at the expense of the others.
- Recall also the 5 layers of SCALP:
  - S-skin

Cerebral perfusion pressure (CPP)

- CPP= MAP-ICP
- MAP= mean arterial blood pressure (avoid systolic blood pressure <90mmHg)
- MAP=$\frac{2}{3}(DP)+\frac{1}{3}(SP)$
- Normal MAP= 70-100 mmHg (65-110 mmHg)
- ICP= intracranial pressure, Normal ICP= 5-10 mmHg
- ICP>20 mmHg should be treated
- Normal CPP= 60-80 mmHg
- Maintaining CPP between 50 and 70mmHg in non-operative brain injury is the fundamental treatment
- Concept of CPP is important in hypertensive patients. Lowering the BP too fast will also decrease the CPP, creating a new problem

$$MAP \simeq DP + \frac{1}{3}(SP - DP)$$

or equivalently

$$MAP \simeq \frac{2}{3}(DP) + \frac{1}{3}(SP)$$

or equivalently

$$MAP \simeq \frac{(2 \times DP) + SP}{3}$$

or equivalently

$$MAP \simeq DP + \frac{1}{3}PP$$

where PP is the pulse pressure, $SP - DP$

- C-dense connective tissue (bleeds the most)
- A-aponeurotica Galia
- L-loose connective tissue
- P-periosteum

- The scalp is highly vascular and may be the source of major blood loss.
- Most head injuries are caused by blows to the head from numerous causes including motor vehicle accidents, assaults and falls from heights.
- The brain within the skull is liable to injury when deceleration occurs i.e. when the neck flexes, extends or rotates. As the brain moves within the cranial cavity, it may strike sharp objects such as the sphenoid wing and the frontal and occipital poles.
- Points where the brain is tethered such as the foramen magnum and the cranial nerves are also potential sites of injury. Shaking of the brain when the skull moves at high speed therefore results in hemorrhage in the subarachnoid space and at the frontal, temporal and occipital poles and in tearing of nerves and vessels. The damage can occur with the head without being struck, for example in a high-speed car crash or in a fall from a height in which the body decelerates rapidly. It may also be associated with direct damage from a blow or a penetrating wound.
- Head injury is one of the most common cause of death and disability.

CLASSIFICATION

- Head injury is classified in the following ways:
  1. General classification
  2. According to Mode of injury (Etiological classification)
  3. Pathological classification
  4. Clinical classification (according to severity-GCS)

GENERAL CLASSIFICATION

- Injury is classified as:
  - Open/ Penetrating
  - Closed/ Blunt

MODE OF INJURY (ETIOLOGICAL CLASSIFICATION)

- These include
  - Missile injury

- High velocity: Gunshot wound
  - Produce focal and diffuse widespread distraction.
- Low velocity: Catapult, golf ball, skull fractures and epidural hematoma (injury to middle meningeal artery)
  - May cause focal injury

➢ Non-missile injuries

- High velocity
  - Typically motor accidents
  - Acceleration/deceleration, rotational & direct impact injuries associated with extensive brain contusion, subdural or intracerebral hematoma with significant morbidity and mortality.
- Low velocity
  - Striking the head against an object (Coup-Contra Coup injury)
  - During a fall or in assault
  - Injury localized at the site of impact (Coup)
  - Injury at opposite site of impact (Contra-coup)
  - Consciousness may not be altered.

DIFFUSE INTRACRANIAL LESIONS

CONCUSSION

- Altered mental state that may or may not include loss of consciousness (momentous loss of consciousness). It is caused by impairment of the reticular activating system.
- Also known as mild traumatic brain injury.
- Diagnosis is clinical with no CT signs.
- Recovery is often complete, however, residual effects such as headache may last for some time.

DIFFUSE AXONAL INJURY

- This is caused by microscopic shearing of nerve fibers, scattered microscopic abnormalities.
- It frequently requires intubation, hyperventilation, CPP monitoring and admission to a neurosurgical intensive care unit (ICU).
- No mass lesion is seen on CT in Diffuse axonal injury.
- Patients are often comatose for prolonged periods of time (mortality= 33%).

FOCAL INTRACRANIAL LESIONS

CONTUSSION

- Injury results from focal trauma to the parenchymal blood vessels leading to petechial hemorrhages and edema (hemorrhagic brain contusion with edema)
- It may occur directly under the site of impact (coup) or on the contralateral side (contrecoup).
- May result in an expanding mass effect
- Insult is to the gray matter which gradually infiltrates the white matter.
- Most common locations are sub-frontal cortex and frontal lobe.
- Presents with focal neurologic deficits, prolonged confusion or coma.
- They require close observation.
- They progress to herniation, rapidly deteriorate and die.

INTRECEREBRAL HEMORRHAGE

- Caused by traumatic tearing of intracranial blood vessels. It is difficult to differentiate from a contusion.
- Include: Epidural and subdural hematoma (discussed later)

PATHOLOGICAL

- Includes:
  - Primary injury
  - Secondary injury

PRIMARY INJURY

- This arise from the initial injury at the time of impact.
- Results of mechanical forces producing tissue deformation.
- Injury thresholds for different tissues differ. Survival depends on the energy reaching the brain which in turn depends on the velocity of impact for example hitting a brick wall at 110km/h will be fatal but a kick to the head playing rugby will probably not. Similarly, a shot from a high velocity rifle will prove fatal but a shot from an air rifle will probably not.
- The mechanism is similar however the degree is different.
- Severe primary damage is characterized by coma from impact. The ability to talk, even a few simple words, after injury indicates that whatever else may happen, the primary injury was not severe and the injury is theoretically survivable.
- Range:
  - Scalp laceration

- Skull fractures
- Vascular- epidural, subdural, intracerebral/intraventricular and subarachnoid hemorrhage
- Meninges
- Cerebral lacerations
- Neuronal + axonal injury (without sign on head or face)

SECONDARY INJURY

- These are further insults to the damaged brain. They may further exacerbate primary injury.
- They are important to understand as they can often be prevented and occasionally reversed, whereas nothing can be done about primary damage (except avoiding the accident in the first place).
- The main secondary effects are respiratory complications, perfusion failure, intracranial hematoma, cerebral swelling, epilepsy, infection and hydrocephalus.
- Secondary injury is often due to secondary insults that include:
  - Poor oxygenation (hypo-ventilation)
  - Poor perfusion (hypo-perfusion)
  - Hypoglycemia of traumatized brain tissue.
- Secondary brain injury:
  - Hypoxic hypoxia, hypercapnia and obstruction to breathing: head injury patients are especially prone to respiratory problems because of lack of central drive, airway obstruction, hemothorax or pneumothorax and/or aspiration pneumonia. The most important aspect of care in the head-injured patient is care of the chest.
  - Ischemic hypoxia and infarction: perfusion failure will lead to cerebral ischemia and worsening of the clinical state. Head injury itself is rarely a cause of hypotension and other causes need to be investigated (e.g. rupture spleen). Resuscitation must be rapid. Patients with head injury accompanied by a systolic blood pressure of less than 60mmHg for more than a few minutes rarely survive.
  - Hypoglycemia
  - Cerebral edema: there is increased ICP which leads to cerebral perfusion failure which in turn causes ischemia leading to more brain swelling and a further increase in ICP. This vicious cycle tends to be worse in those with severe primary damage.
  - Cerebral herniation

- Infection: development of meningitis or an abscess after injury can reverse a good recovery and must be watched for and treated vigorously.
- Hypotension
- Hypo and hyperthermia
- Hematoma (>150ml the brain cannot compensate): intracerebral, subdural and epidural hematomata occur and can lead to deterioration following head injury.
  - Intracerebral and acute subdural hematomata are usually associated with severe primary injury and carry a bad prognosis.
  - In contrast subacute subdural and epidural hematomata are often associated with little or no primary injury and bleed slowly over a few hours or days, removal before intracranial pressure is excessive can lead to complete recovery.
- Seizures: fits are common in head injury and cause ischemia while they are occurring. They must be stopped rapidly using IV diazepam in small doses followed by phenytoin or valproate to prevent recurrence.

SEVERITY OF BRAIN INJURY (CLINICAL CLASSIFICATION)

- Most used in assessment and management.
- Can be easily done by most health workers and is easily interpreted.
- It is very useful in assessing progress of the injury
- It is assessed using the Glasgow Coma Scale (GCS)

Value	Eye (Out of 4)	Verbal response (out of 5)	Motor response (out of 6)
6			Obeys commands
5		Oriented & converses (gives name, place and date)	Localizes to pain (brings hand above clavicle to stimulus on head/neck)
4	Opens eyes spontaneously	Disoriented but Converses (communicates coherently)	Withdraws from pain (bends arm at elbow rapidly but features not predominantly abnormal)
3	Opens to verbal command (sound)	Inappropriate response (intelligible single words)	Decorticate/ Abnormal flexion (Bends arms at elbow rapidly but features clearly predominantly abnormal)-"Mummy pose"

2	Opens to pain	Inappropriate sounds (only moans/groans)	Decerebrate/ Extension (Extends arms at elbow)
1	No response	No response	No response

- Note:
  - Decorticate= flexion (upper limbs) and extension of lower limbs
  - Decerebrate= mid brain injury, all limbs are in extension
- Note:
  - GCS of 13-15 indicates mild head injury
  - GCS 9-12 indicates moderate head injury
  - GCS of 3-8 indicates severe brain injury
    - GCS< 8: comatose

PEDIATRIC MODIFICATION

Age	Eye	Verbal	Motor	Total
0-6M	4	Cries-2	Flexion-3	9
6 -12M	4	Vocalises-3	Localizes-4	11
1-2Y	4	Words-4	Localizes-4	12
2-5Y	4	Words-4	Obeys-5	13
5 Y+	4	Orienteted-5	Obeys-5	14

CLINICAL ASSESSMENT

- Primary survey- ABCs (with cervical collar until fracture ruled out by imaging)
- Secondary survey
  - Full history and possible also collateral history
  - Complete Examination
  - Investigations

HISTORY

- Mechanism of injury: direct trauma to the head or deceleration
- Time of injury
- History of loss of consciousness & did the patient regain it (Lucid interval).
- History of convulsions
- History of bleeding from ears/nose or any discharge
- Headache, nausea, vomiting, a falling pulse rate and rising blood pressure indicate cerebral edema
- Last meal

- Co-morbidities
- Drugs- aspirin

EXAMINATION

- Complete neurological examination including:
  - GCS
  - Pupil light responses and reflexes: pupillary inequalities or abnormal light reflex indicate intracranial hemorrhage
  - Tone
  - Power
  - Reflexes
  - Sensation
  - Co-ordination
- Examine other systems also fully
  - Pulse
  - Temperature
  - Blood pressure
  - Respiratory rate

INVESTIGATIONS

DIAGNOSTIC

1. Imaging
   - CT scan (because this is an emergency CT is used for diagnosis, it is cheaper and faster than MRI): it shows contusions, hematomata, midline shift, fractures, ventricle & brainstem injury, hydrocephalus and cerebral edema
   - MRI
   - X-ray- skull fractures, relative position of the calcified pineal gland, presence of intracranial air.
     - Skull X-ray: AP, lateral and Towne's views-Cervical fractures/displacement
     - Serial X-rays (Cervical spine, chest, abdomen and pelvic)
   - Carotid arteriography

SUPPORTIVE

1. Full blood count and Cross match/ Group & save
2. Abdominal ultrasound: for any abdominal injuries
3. Urea and Electrolytes
4. Creatinine

5. Liver function tests
6. Blood sugar
7. Arterial blood gases

INDICATIONS

INDICATIONS FOR ADMISSION TO A GENERAL HOSPITAL

- Oriented patients with:
  - Skull fracture or suture diastasis
  - Persisting neurological symptoms or signs
  - Confusion or any other depression of the level of consciousness at the time of examination
  - Difficulty in assessing the patient (e.g. due to alcohol, young age, epilepsy, attempted suicide)
  - Inadequate social conditions or lack of responsible adult or relative
  - Co-existence of other medical conditions (e.g. coagulation disorders)
- All patients with impaired consciousness

INDICATATION FOR SKULL RADIOGRAPHY AFTER RECENT HEAD INJURY

- Oriented patient with:
  - History of loss of consciousness or amnesia
  - Suspected penetrating injury
  - CSF or blood loss from the nose or ear
  - Scalp laceration (to bone or >5cm long), bruise or swelling
  - Violent mechanism of injury
  - Persistent headache or vomiting
  - Fall from a significant height in a child (depends in part on the age of a child) and/or on to a hard surface, tense fontanelle, suspected non-accidental injury
- Patient with impaired consciousness or neurological signs all patients, unless urgent CT is performed.

INDICATIONS FOR URGENT CT SCANNING AND/OR NEUROSURGICAL CONSULTATION

- Coma, confusion or neurological signs persisting after resuscitation
- Deteriorating consciousness or progressive neurological signs
- Fractured skull in combination with
  - Confusion or other impairment of consciousness
  - Epileptic seizures
  - Neurological symptoms or signs

- Open injury with
  - Depressed compound fracture of skull vault
  - Fractured base of skull
  - Penetrating injury
- Unstable systemic state precluding transfer to neurosurgery
- Diagnosis uncertain
- Tense fontanelle or suture diastasis in a child

INDICATIONS FOR NEUROSURGICAL CONSULTATION AFTER CT SCANNING

- Abnormal CT scan
- High or mixed density intracranial lesion
- Midline shift
- Obliteration of third ventricles
- Relative dilatation of lateral ventricle(s)
- Obliteration of basal cisterns
- Intracranial air
- Subarachnoid or intraventricular hemorrhage

MANAGEMENT

GENERAL MANGEMENT

- Primary survey- ABCs
  - Head must be positioned at 30 degrees in neutral position.
  - Fluids- Crystalloid (NS), DO NOT GIVE DEXTROSE!
    - If they are hypoglycemic give a bolus of 50% dextrose
  - Oxygen saturation should be maintained at 90%
  - BP systolic >90mmHg
  - Mean arterial pressure should not go below 70 (keep at 80)
- Protection of airway using mouth gag, endotracheal intubation or tracheostomy whenever required.

- Throat suction, bladder and bowel care and good nursing (hourly turning to prevent decubitus ulcers).
- Nasal oxygen or often ventilator support
- IV fluids initially later NG tube feeding
  - Feeding within first 24 hours and assess if they can swallow (assess if NG tube is required). Aim to attain normal diet by day 7
- Electrolyte maintenance
- Prevent hyper- or hypothermia
- Patients susceptible to seizure should start on prophylactic medication (Carbamazepine, phenytoin and phenobarbital) If patient starts to seize treat for seizures.
- SEDATION SHOULD BE AVOIDED!
- Antibiotics especially in patients with fractures: traditionally prophylactic antibiotics have been given to head injury patients but are less commonly used nowadays. Persistent rhinorrhea or otorrhea requires surgical treatment to repair the skull base defect
  - Antibiotics like penicillins, ampicillins are given.
- Give analgesia for pain (non-sedating) and Anti-acids (to prevent Cushing's ulcers).
- Corticosteroids either dexamethasone or betamethasone are used commonly but their beneficial effect is not confirmed.
- Dealing with any scalp wound:
  - Shave hair for at least 2cm around the wound
  - Remove foreign bodies
  - Debride the skin edges
  - Suture the scalp in a single layer, leaving stitches in for at least 7 days
  - Use lidocaine with adrenaline to reduce bleeding.
- Indications for surgery (craniotomy):
  - Acute extradural/epidural hematoma
  - Acute subdural hematoma
  - Depressed skull fracture
- Procedure: Craniotomy is done and cranial flap is raised. Clot is evacuated followed by applying hitch stitches between dural layer and scalp. Post-Op antibiotics, analgesic and anticonvulsants are given.

MANAGEMENT OF RAISED INTRACRANIAL PRESSURE

- Elevate head at 30 degrees
- Diuretics
  - Mannitol 20% (0.5-1g/kg- slow infusion in 30 minutes)- 200ml IV TDS or
  - Furosemide 40mg IV TDS. It should not be given in case of intracranial hematoma
- Hypertonic saline (3% saline)
- Hyperventilation (decrease carbon dioxide partial pressure and increase oxygenation- causes vasoconstriction)
  - Only done in 30 minutes when other measures have failed
- Decompressive craniotomy (ventriculostomy with burr holes)

Measures to lower ICP: "HIVED"

- Hyperventilation
- Intubation with pretreatment and sedation
- Ventriculostomy (burr hole)
- Elevate the head of the bed
- Diuretics (mannitol)

COMPLI CATIONS OF HEAD INJURIES

Cushing's triad for ICP

- Bradycardia
- Hypertension and a widening pulse pressure
- Decreased respiration/apnea

EARLY

- Brainstem injury- due to coning
- Compression over cerebellum and medulla
- CSF rhinorrhea: indicate a tear in the dura following fracture involving the sinuses-frontal, ethmoid, sphenoid sinuses. Meningitis is the common complication of CSF rhinorrhea. Treatment is initially conservative (10-day IV antibiotics and observations). Surgeries include: reduction of fracture of middle third face and exploration of anterior cranial fossa.
- CSF otorrhea
- Meningitis
- Pituitary damage and endocrine failure- requires high dose of hydrocortisone 200mg QID.
- Aerocele
- Depressed fractures: often causes injury to dural venous sinuses and may lead to torrential hemorrhage which may be life threatening. So such depressed fractures should never be elevated.

LATE

- Chronic subdural hematoma
- Early post-traumatic epilepsy- they need anticonvulsants for 3 years

- Late post-traumatic epilepsy due to scarring and gliosis of cerebrum
- Post-traumatic amnesia
- Post traumatic hydrocephalus
- Post-traumatic headache

INTRACRANIAL HEMORRHAGE

EPIDURAL/EXTRADURAL HEMATOMA

- This is collection of blood in the extradural space between the dura and the skull.
- The most common site is temporoparietal region. They are usually associated with fractures of the temporoparietal region.
- It can be unilateral or bilateral.
- Commonly affected vessels:
  - Middle meningeal veins
  - Anterior branch of middle meningeal artery
  - Posterior branch of middle meningeal artery
- It may also result from a torn dural venous sinus

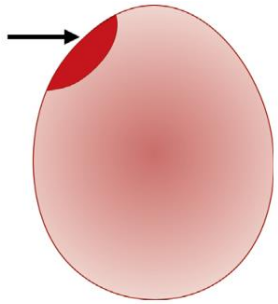
PATHOLOGY

- Direct blow, like from cricket ball or road traffic accidents or fall and impact or coup and contre-coup injuries lead to fracture of thin temporal bone and tear of vessels.
- Bleeding initially outward towards the scalp and under temporalis leading to formation of a hematoma.
- Gradual stripping of dura from skull and collection of blood occurs.
- In 6-12 hours epidural hematoma occurs which raises the intracranial pressure leading to coning of supratentorial content (uncus of temporal lobe) through tentorial hiatus.
- Shift of midbrain towards opposite side which gets injured by sharp edge of the tentorial cerebelli.
- Corticospinal tract before decussation on opposite side gets injured so hemiparesis and pupillary changes occur on the same side of hematoma. This effect is called as Kernohan's notch effect.

CLINICAL FEATURES

- History of transient loss of consciousness following a blow or a fall.
- Patient soon regains consciousness and again after 6-12 hours starts deteriorating (Lucid interval).
- Later the patient presents with: confusion, irritability, drowsiness, hemiparesis on same side of the injury.
- Initially pupillary constriction and later pupillary dilation occurs on the same side, finally becomes totally unconscious- Hutchinsonian pupils.
- Death can occur if immediate surgical intervention is not done.

- Features of raised intracranial pressure may be seen:
  - Hypertension
  - Bradycardia
  - Vomiting
  - Seizures (occasionally)
- Wound and hematoma in the temporal region of the scalp may be seen



INVESTIGATIONS

- X-ray skull may show fracture of temporal bone
- Electrolyte estimation
- CT scan head is diagnostic. Epidural hematoma shows biconvex lesion.

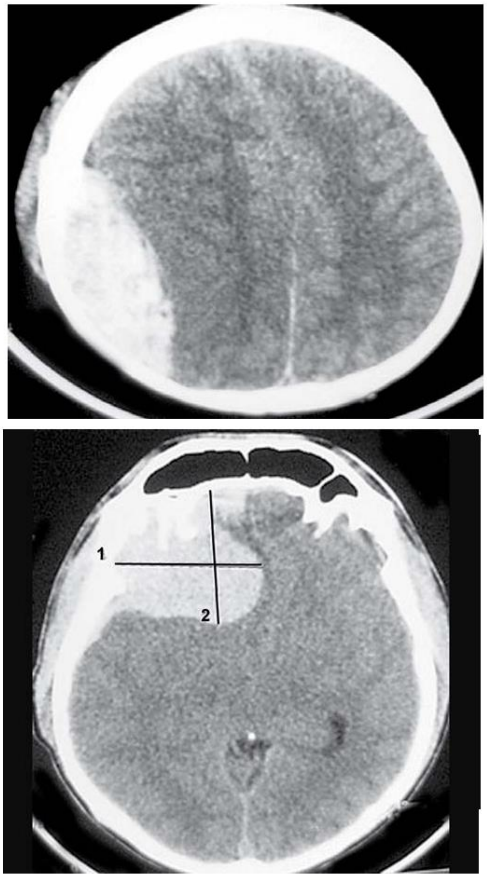


Figure 251: Epidural hematoma

TREATMENT

- Immediate surgical intervention is a must to save the life of the patient.
- Craniotomy is done and cranial flaps are raised. The dura is opened and the clot is evacuated. The dura is fixed to galea using interrupted sutures- Hitch stitches.
  - 5cm vertical incision in parietal region above the zygoma.
  - Galea is incised. Skull is opened using perforator and burr
  - Meninges are kept aside
  - Black currant jelly clot is evacuated
  - Bleeding vessels are cauterized-bipolar cautery
  - Dural hitch stitches are placed
  - Opposite side if needed should be evacuated
- Antibiotics, analgesia and anticonvulsants are given postoperatively.
- Recovery is good after surgery.

COMPLICATIONS

- Meningitis
- Post-traumatic epilepsy
- Post-traumatic amnesia

SUBDURAL HEMATOMA

- These are divided into:
  - Acute subdural hematoma (<24 hours)
  - Subacute (24hrs-2 weeks)
  - Chronic subdural hematoma (>2 weeks)

ACUTE SUBDURAL HEMATOMA

- This is a collection of blood between the dura and the brain. It is due to injury to the cortical veins and often due to laceration of cortex of brain which bleeds and blood gets collected in the subdural space forming a hematoma.
- The hematoma is extensive and diffuse.
- There is no lucid interval. There is severe primary brain damage.
- Hematoma may be of coup and contra-coupe type.
- Loss of consciousness occurs immediately after trauma and is progressive
- Convulsion is common
- Features of raised ICP are obvious: high blood pressure, bradycardia and vomiting
- Focal neurological deficits or hemiparesis can occur
- CT scan shows concavo-convex lesion.
- Treatment:
  - Antibiotics
  - Anticonvulsants
  - Surgical decompression is done by craniotomy.

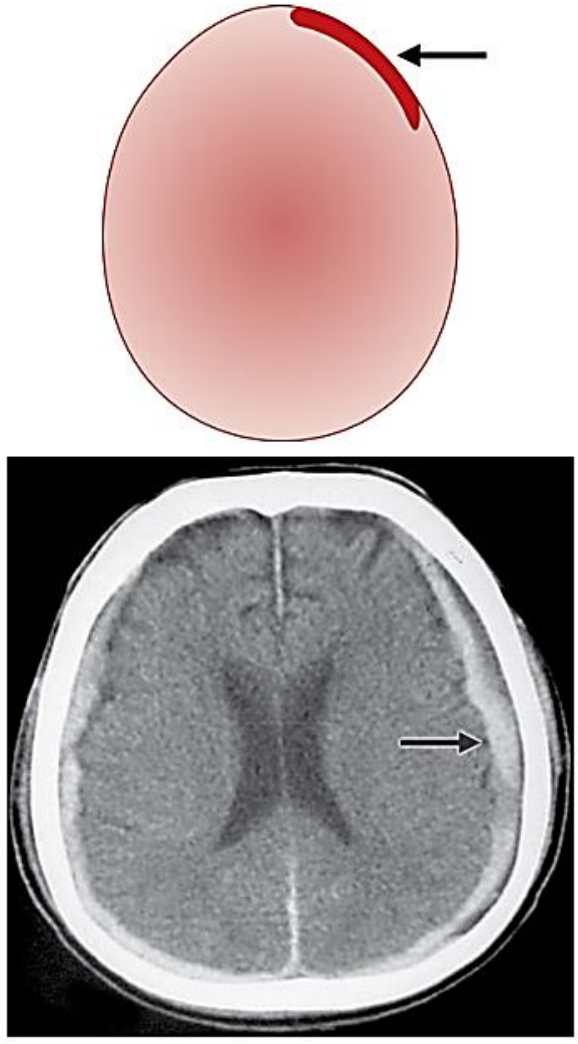


Figure 252: Subdural hematoma

CHRONIC SUBDURAL HEMATOMA

- This is due to rupture of veins between dura and brain (cerebral hemispheres), causing gradual collection of blood in subdural space.
- It is commonly seen in elderly people following minor trauma e.g. fall, slipping (which might have gone unnoticed)
- In elderly people, the brain atrophies and even minor injuries can cause shearing and bleeding from these veins,

- Blood gradually collects over 2-6 weeks. Plasma and cellular components get separated. Eventually cellular part gets absorbed leaving only fluid component called chronic subdural hygroma.
- Usual hematoma collection is 60-120ml and often in 50% of cases is bilateral.
- Patients present with confusion, disorientation, gradually with altered level of consciousness and drowsiness.
- Later convulsions, features of intracranial hypertension and features of coning develop.
- Extensor plantar response and pupillary changes develop eventually.
- CT shows concavo-convex lesion.
- Treatment:
  - Craniotomy and evacuation of clot is done when required on both sides.
  - Antibiotics
  - Anticonvulsants for 3 years.
- Complications:
  - Epilepsy
  - Meningitis
  - Coning
  - Neurological deficits

CHRONIC SUBDURAL EMPYEMA

- This may be a primary infection of the subdural space from sinusitis focus causing suppuration and pus formation.
- It can be a complication of chronic subdural hematoma
- It is due to secondary bacterial infection of collected clot/fluid.
- Infection is from sinusitis scalp (common)/through earlier trauma wound/hematogenous
- Commonly gram positive organisms like *Streptococci (viridans/milleri)* cause empyema however other virulent organisms like gram negative bacteria occasionally can cause it.
- There is cortical venous thrombophlebitis and cortical infarction
- Headache, fever, meningism and convulsions are the features.
- MRI is ideal than CT to diagnose
- Treatment: antibiotics, craniotomy and drainage, anticonvulsants, ICU care, proper monitoring, regular follow up.
- It carries 10% mortality.

SUBARACHNOID HEMORRHAGE

- This is a type of intracranial hemorrhage into the subarachnoid space usually from basal cisterns.
- It may be spontaneous or following trauma.
- Causes:
  - Intracranial aneurysm (50%)
  - Hypertension
  - A-V malformations
  - Blood dyscrasias
  - Anticoagulant drugs
  - Brain tumors (malignant)
- Clinical features:
  - Sudden onset of severe headache (thunderclap headache) with vomiting.
  - Features of raised intracranial pressure: hypertension, bradycardia and vomiting
  - Photophobia
  - Neck stiffness
  - Focal neurological deficits: hemiplegia, dysphasia
  - Eye changes: ptosis, dilated pupil, changes in the eyeball movement
  - Sudden loss of consciousness
  - Features of brain edema and cerebral ischemia
  - In 40% of recovered patients, rebleeding occurs in 6-8 weeks which is commonly fatal.
- Hunt and Hess grading for SAH:
  - Grade 1: asymptomatic
  - Grade 2: severe headache and neck stiffness
  - Grade 3: drowsy, confused or mild focal deficit
  - Grade 4: stupor, hemiparesis
  - Grade 5: decerebrate rigidity, coma
- Fischer grading of SAH:
  - I- minimal <8mm size
  - II- moderate 8-15mm size
  - III-severe >15mm size
- Differential diagnosis:
  - Meningitis
  - Coning due to any cause

- Investigations:
  - Lumbar puncture should be done to differentiate from meningitis. It has to be done carefully as it may precipitate coning. In subarachnoid hemorrhage, blood strained CSF is collected (Xanthochromic)
  - CT scan
  - Carotid and vertebral angiogram
- Treatment:
  - Clipping or wrapping the aneurysm
  - Craniotomy and proceed.
  - Ligation of common carotid artery-there is risk of hemiplegia
  - Therapeutic embolization
  - Excision of vascular malformations
  - Coiling of aneurysms with instruments

DEPRESSED SKULL FRACTURE



Figure 253: Depressed skull fracture

- This means fracture depression is more than the depth of inner table of the skull.
- It may result in:
  - Tear in the dura beneath
  - Hematoma in the deeper plane
  - Injury to the cerebrum
  - Injury to the venous sinuses-may cause life-threatening hemorrhage. Fracture should not be elevated in such occasion as it itself can precipitate bleeding.
  - Convulsions
  - Meningitis
- Investigations:
  - CT scan
- Treatment:
  - Antibiotics, anticonvulsants
  - Elevation of the depressed fracture: burr holes are made in the adjacent normal skull. Fracture is elevated. Bony fragments and necrotic material are removed. Dural tear is closed with interrupted sutures.

BASILAR FRACTURE

- Signs include periorbital ecchymoses (raccoon's eyes), retroauricular ecchymoses (Battle's sign), otorrhea, Rhinorrhea, hemotympanum and cranial nerve palsies.
- A ring test for CSF rhinorrhea (in the presence of epistaxis): Sample of blood from nose placed on filter paper to test for the presence of CSF. If present, a large transparent ring will be seen encircling a clot of blood.

SPINAL TRAUMA

SPINAL TRAUMA

- Spinal trauma may involve injury to the spinal column, spinal cord or both.
- Recall: the vertebral column is composed of a series of motion segments. A motion segment consists of two adjacent vertebrae, intervertebral disc and ligamentous restraints. The neural elements are contained within the spinal canal.
- Spinal injury is 1/10 as common as head injury but the basic pathology is the same.
  - Primary injury occurs when the skeletal structures fail to dissipate the energy of the primary mechanical insult, resulting in direct energy transfer to the neural elements. The injury may occur directly by flexion, extension, axial loading, rotation or traction or by compression of the cord by a fragment of bone and/or disc.
  - Secondary injury: hemorrhage, edema and ischemia result in a biochemical cascade that causes the secondary injury. This may result in further loss of neurological function. Prolonged hypotension and poor oxygenation compound the situation so it is important that the patient is adequately resuscitated. The spinal column should be realigned as soon as possible in cases of cord injury in which dislocation or subluxation is present. Ongoing spinal cord compression from disc material and/or bony fragments may require surgical intervention. Secondary effects including hypoxia and perfusion failure can also occur.
- Compression of the cord is less common than compression of the brain following head injury.

- The causes of cord compression are bone fragments, hematomata and acute disc prolapses.
- Over 50% of spinal injuries occur in the cervical spine with the remainder being divided between the thoracic spine, the thoracolumbar junction and the lumbosacral region.
  - Upper cervical spinal injury is uncommon as the canal is capacious. The cervicothoracic junction is a transitional zone going from mobile to fixed and is thus prone to injury. It may be difficult to visualize this area on radiography.
  - The thoracic spine is relatively rigid because of the stabilizing influence of the thorax. Significant disruption of the thoracic spine requires major energy transfer and associated visceral and vascular injuries are common.
  - The thoracolumbar junction is another transitional zone that is prone to injury, this is the second most common area of injury.
- Recall:
  - There are 7 cervical, 12 thoracic, 5 lumbar, 5 sacral and 4 coccygeal vertebrae.
  - The spinal cord extends from the foramen magnum to the T12/L1 junction where it ends as the conus Medullaris below of which the cauda equine lies.
  - The spinal cord consists of central grey matter (neuronal cell bodies) surrounded by white matter (axons).
  - The lateral spinothalamic tracts transmit pain and temperature sensation, the lateral corticospinal tracts are responsible for motor function and the posterior columns transmit position, vibration and deep pressure sensation.
  - A spinal nerve root exits on both sides of the spinal cord at each level. There are 8 cervical nerve roots. The C1 root exits above the C1 body, the C2 root between C1 and C2 and the C8 root exits below C7. All thoracic (12) and lumbar nerve roots (5) exit below the pedicle of the same number.

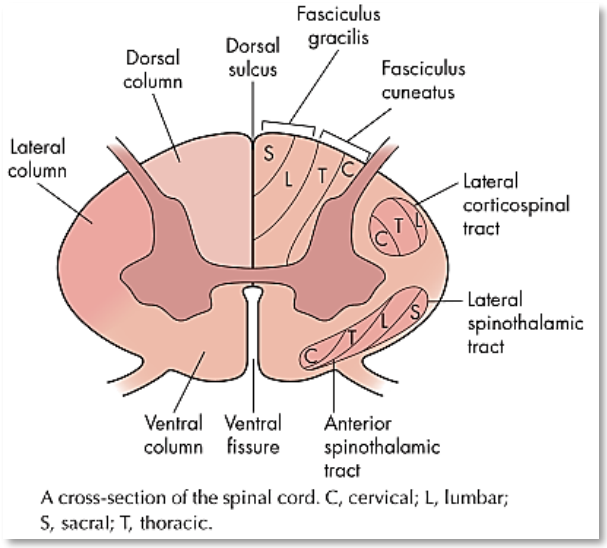


Figure 254: Cross-section of the spinal cord

- The cervical spine is the region more vulnerable to injury.
- The thoracic spine is relatively protected due to limited mobility from support of the rib cage (T1-T10) however the spinal canal through which the spinal cord

traverses is relatively narrow in this region. Therefore, when injuries to this region do occur they usually have devastating results.

- The thoracolumbar junction (T11-L1) is a fairly vulnerable region as it is the area between the relatively inflexible thoracic region and the flexible lumbar region.
- The lumbosacral region (L2 and below) contains the region of the spinal canal below which the spinal cord proper ends and the cauda equine begins.
- Recall the spinal column can be divided into 3 columns: anterior, middle and posterior. When all 3 columns are injured the spine is unstable. Instability may also exist in some two-column injuries.

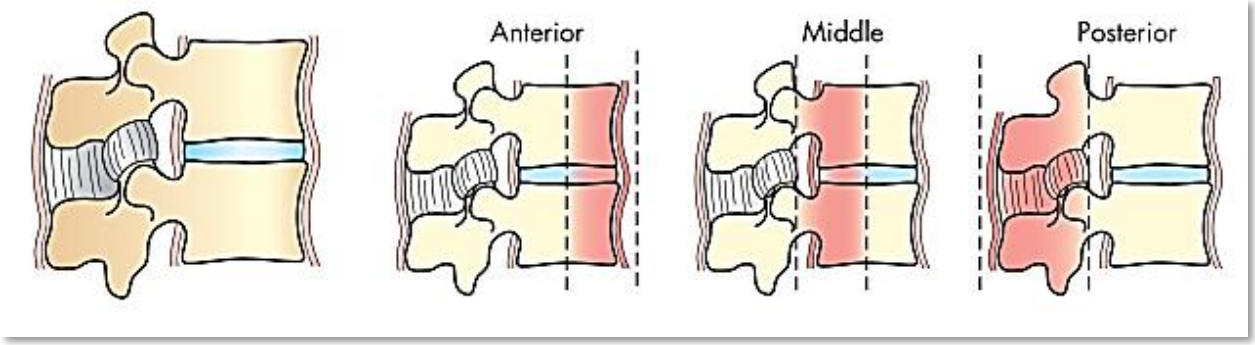


Figure 255: The three-column model of spinal stability

- There should be a high index of suspicion of spinal injury if any of the following are evident:
  - Neurological deficit
  - Multiple injuries
  - Head injuries
  - Facial injury
  - High energy injury e.g. fall from a height
  - Abdominal bruising from a seatbelt, suggestive of a possible lumbar spine injury
- In a conscious patient spinal injury can be excluded if:
  - There is no pain
  - Palpation of the spine is non-tender
  - Neurological examination is normal
  - There is a pain-free range of movement

CLASSIFICATION OF SPINAL TRAUMA

- Spinal injuries are classified based on:
  - Severity:
    - Complete: rendering the patient functionless below the level of the lesion
    - Incomplete: some sensorimotor function is preserved
  - Level of neurological (sensory and motor) and bony involvement
  - Fracture/dislocation type (mechanism, stable vs unstable)

PATHOPHYSIOLOGY

- Spinal cord injury leads to either temporary or permanent dysfunction.
- Spinal injury occurs due to
  - Direct injury due to bullet firing or accidents (on road, in working place, during communal violence etc.)
  - Compression by bone fragments, hematoma or disk material
  - Ischemia due to rupture of spinal arteries.
- Complete transection of the spinal cord causes immediate loss of sensation and voluntary movement below the level of the lesion. In quick transection of the spinal cord the patient feels themselves cut into 2.
- For a while, the patient's mind remains clear but they feel as if the lower part of the body below the injury does not exist. This is because the higher centers remain unaffected but the spinal centers below the level of the injury loose the function.
- The effects of complete transection of the spinal cord occur in 3 stages:
  - Stage of spinal shock
  - Stage of reflex activity
  - Stage of reflex failure
- If the spinal cord is gravely injured but does not suffer complete division, the condition is called incomplete transection.
- The stages above are seen also in incomplete transection of the spinal cord though the features in the second stage differ.

STAGE OF SPINAL SHOCK

- This occurs immediately after the injury. It also known as the stage of flaccidity.
- It may last from seconds to weeks (3 weeks) and does not signify permanent spinal cord damage therefore long-term prognosis cannot be postulated until spinal shock has resolved.

- Flaccid Paralysis (loss of muscle tone) occurs in 2 times or in all 4 limbs. Paralysis depends upon the level of the injury.
  - Injury in the cervical region leads to paralysis of all 4 limbs (quadriplegia/tetraplegia)
  - Injury to the thoracic, lumbar or sacral segments including cauda equine and conus Medullaris causes paralysis of the lower limbs (paraplegia)
- All reflexes are lost due to injury to the anterior and posterior nerve roots.
- All sensations are lost due to injury to the posterior nerve roots and sensory neurons in the posterior gray horn.
- Some visceral organs are also affected especially the urinary bladder and rectum which become paralyzed. The heart rate is decreased and the pulse becomes weak and thready.
- Venous return is also reduced. In complete transection of the spinal cord muscle tone is lost and there is flaccid paralysis so skeletal muscles cannot contract to push blood back to the heart (muscle pump) in addition, the limbs are immobile and smooth muscles of blood vessels also loss their tone. Blood accumulates in the blood vessels of the limbs so they become cold and blue.
- The effect on blood pressure depends upon the level of the injury:
  - Lesions anywhere below L2 will not affect blood pressure as much because the sympathetic vasoconstrictor fibers leave the spinal cord between T1 and L2 segments.
  - Lesions at or above T2 segment will cause blood pressure to fall drastically. Mean arterial pressure falls below 40mmHg.
- Complete transection at the cervical region above C3 can be very fatal because the diaphragm and other respiratory muscles are cut off from respiratory centers. It can cause paralysis of respiratory muscles leading to sudden arrest of breathing.
- Crush injuries at sacral segments of the spinal cord results in atonic bladder and loss of micturition reflex.

Neurogenic shock: is a state of vasomotor instability resulting from impairment of the descending sympathetic pathways in the spinal cord or simply a loss of sympathetic tone.

Clinical features:

- Flaccid paralysis
- Hypotension
- Bradycardia
- Cutaneous vasodilation
- Normal to wide pulse pressure

STAGE OF REFLEX ACTIVITY

- This is also caused the stage of recovery. After 3 weeks period, depending largely upon the general health of the patient, the reflex activity begins to return to the isolated segments of spinal cord below the lesion.
- In complete spinal injury:
  - Functional activities return to smooth muscle first.
  - Sympathetic tone to blood vessels returns next as the neurons in gray horn act independently of vasomotor center. When tone in blood vessels is restored the blood pressure returns back to normal.
  - After 3 months tone in skeletal muscle returns. First to the flexors. Though the tonicity is returned, it is not complete even in flexor muscles. The muscles remain hypotonic. Limbs in this condition tend to adopt a position of slight flexion and the paralysis is therefor called paraplegia in flexion. Limbs cannot support the weight of the body.
  - After a few weeks reflex movements occur. First flexor reflexes (which can be elicited by painful stimulus). The first reflex to appear is the Babinski reflex.
  - After 1 to 5 weeks extensor reflexes return, initially the knee jerk and then the ankle jerk.
  - In some cases, a widespread reaction can be elicited by scratching the skin over the lower limbs or the anterior abdominal wall, depending on the level of the lesion. This reaction constitutes the spasm in flexor muscles of both the lower limbs, evacuation of urinary bladder and profuse sweating. It is called the mass reflex.
- In incomplete spinal injury
  - Tone returns to extensors first and not flexor. This is because in incomplete transection, some of the descending fibers in the lateral column of the cord especially vestibulospinal and reticulospinal tract may escape injury so some connections persist between the brainstem and the spinal cord. Fibers of these tracts mainly reinforce the activity of extensor motor neurons.
  - Because of the extensor hypertonia, the lower limbs are extended at the hip and knee with toes pointing slightly downwards, this condition is known as paraplegia in extension.
  - The stretch reflex reappears first. Flexor reflexes return later. Philipson reflex (clasp knife reflex can be elicited)
    - Philipson reflex (Clasp knife reflex): In the upper limb, some resistance is offered when the arm is flexed at the elbow joint passively i.e. the arm

cannot be flexed, this resistance is offered because of the stretch reflex developed in the triceps muscles however if the forearm is flexed forcefully the resistance to flexion is abolished suddenly leading to quick flexion of arm (just like how a clasp-knife works)

- Mass reflex as seen in complete transection is not seen here.

STAGE OF REFLEX FAILURE

- Though the reflex movements return, muscles below the level of injury have less power and less resistance.
- Usually general condition of the patient starts to deteriorate.
- General infections become common.
- Due to this, the failure of reflex function develops. The reflexes become more difficult to elicit. The threshold for stimulus increases.
- Mass reflex is abolished and the muscles become extremely flaccid and undergo wasting.

CLINICAL FEATURES

- Flaccid paraplegia with no preservation of sensation usually indicates a complete lesion.
- If any sensorimotor function below the level of the injury is seen this constitutes incomplete injury.
  - Sacral sparing is a sign of incomplete spinal injury.
  - The sacral dermatomes are the most resistant to injury and sacral sparing is a cause for slight optimism (better prognosis)
  - In sacral sparing there is perianal sensation, voluntary anal sphincter contraction or voluntary toe flexion.

PHYSICAL EXAMINATION

- Classification of spinal injuries as complete or incomplete requires a proper neurologic examination
  - The entire spine must be palpated and the overlying skin inspected. A formal spinal log roll must be performed to achieve this. Significant swelling, tenderness or palpable steps or gaps suggest a spinal injury. Note the presence of any wounds that might suggest penetrating trauma and



Figure 256: Spinal log roll

document the condition of the skin, particularly over the pressure areas.

- The exam should include testing the 3 readily assessable long spinal tracts:
  - Corticospinal tract: located in the posterolateral of the spinal cord and responsible for ipsilateral motor function. It is tested by voluntary muscle contraction (Grade the power).
  - Spinothalamic tract: located in the anterolateral aspect of the spinal cord. It is responsible for contralateral pain (pin prick) and temperature sensation and tested as such.
  - Posterior columns: located in the posterior aspect of the spinal cord. It is responsible for ipsilateral position and vibratory sense and some light touch sensation. Tested using a tuning fork and position sense of the fingers and toes.
- Deep tendon reflexes and sacral reflexes may be preserved in complete injuries.

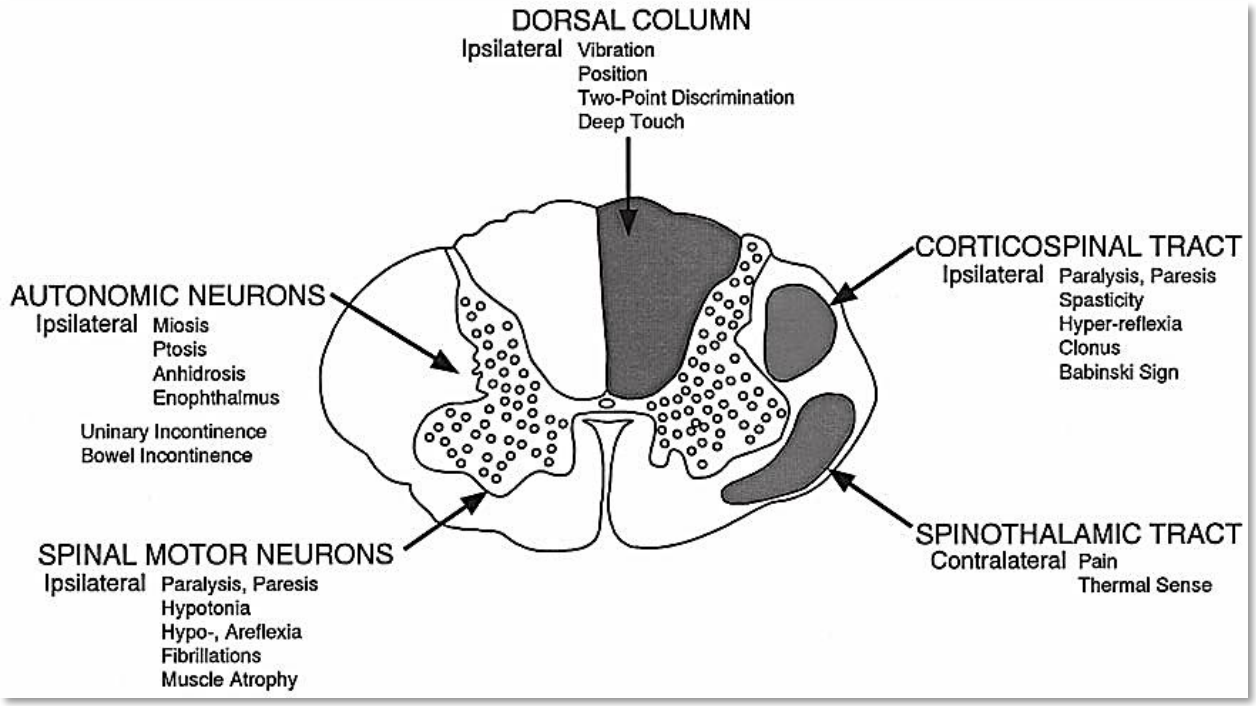


Figure 257: Cross-section of the spinal cord and tracts

SPINAL CORD SYNDROME

ANTERIOR CORD SYNDROME

- Pattern seen with injury to the anterior potion of the spinal cord or with compression of the anterior spinal arteries (artery of Adamkiewicz).
- It involves full or partial loss of bilateral pain and temperature sensation (spinothalamic tract) and paraplegia (corticospinal tract) with preservation of posterior column function
- It is often seen with flexion injuries and carries a poor prognosis.

BROWN-SEQUARD SYNDROME

- This is seen with hemi-section of the spinal cord usually secondary to a penetrating injury, but may also be seen with disk protrusion, herniation or tumor.
- It consists of ipsilateral loss of motor function (Corticospinal tract) and posterior column function with contralateral loss of pain and temperature sensation.

CENTRAL CORD SYNDROME

- Pattern is seen with injury to the central area of the spinal cord often in patients with a preexisting narrowing of the spinal canal.
- Usually seen with hyperextension injuries, its cause is usually attributed to buckling of the ligamentum flavum into the cord and/or an ischemic etiology in the distribution of branches of the anterior spinal artery.
- It is characterized by weakness greater in the upper extremities than the lower extremities and distal worse than proximal.
- It has a better prognosis than the other partial cord syndromes with a characteristic pattern of recovery (lower extremity recovery progressing upward to upper extremity recovery, then the hands recover strength).

ASSESSMENT

- Assessment should answer the following questions:
  - Is there any neurological injury?
  - What level is the injury? (the lowest neurological level with normal neurological function)
  - Is the cord lesion complete or incomplete?
  - Is the bony architecture stable or unstable?
- Mechanisms suspicious for spinal injury:
  - Diving
  - Fall from >10feet

- Injury above level of shoulders (cervical spine)
- Electrocution
- High speed motor vehicle crash
- Rugby or football injury (tackling)

- If the lesion is incomplete, every effort must be made to prevent deterioration. Thus the spine must be held in a stable position, hypoxia prevented and hypotension corrected.
- Early investigation with MRI will exclude cord compression.
  - Cervical spine films are indicated for: tenderness along cervical spine, neurologic deficit, good mechanism of injury, presence of distracting injury and patients with altered sensorium.
- If the bony architecture is displaced or thought to be unstable, it should be reduced and stabilized.
- When the cervical spine is injured this can be done with traction only but if the thoracic and lumbar spines are involved open reduction may be required.
- It is now common to perform an early operative fixation with bone grafts and metal for both incomplete and complete lesions. This allows early mobilization which in turn reduces the risk of chest complications and deep vein thrombosis.
- At the earliest opportunity the patient should be transferred to a spinal injuries unit.

MANAGEMENT

- Always start with the ABCs of trauma resuscitation.
- Maintain spinal immobilization throughout the resuscitation.
- Estimate level of neurologic dysfunction during the secondary survey
- Obtain appropriate diagnostic studies
- Establish early neurosurgical consultation
- If blunt spinal cord injury is diagnosed, begin high-dose methylprednisolone (must be given within 8 hours of injury and not for penetrating injury).
  - Loading dose of 30mg/kg over 15 minutes during hour 1 followed by a continuous infusion of 5.4mg/kg/hr, the infusion is continued for 23 hours if the bolus is given within 3 hours of injury or for 47 hours if the bolus is given within 8 hours of injury.

CERVICAL SPINAL INJURY



Figure 258: Radiography of cervical spine showing fracture dislocation of the spine at C2/C3 level, so called hangman's fracture

- Injury to the cervical spine may accompany a head injury and fractures and dislocations in this area are common after road traffic accidents (most common cause) and falls.
- These injuries are usually classified based on:
  - Mechanism: Flexion, extension, compression, rotation or a combination of these
  - Location
  - Stability
- If a patient complains of neck pain or weakness and/or numbness in the limbs or if there is loss of sphincter control, an injury to the cervical spine should be suspected.
- Precautions should be taken to prevent further damage.
- Extension and rotation of the neck should be prevented preferably by halter tractions.
- Skull calipers may be applied later.
- Sandbands may help movement in the emergency situation.
- Collars should be maintained in position until radiography has been completed.
- 4 vies of the cervical spine should be obtained (lateral, anteroposterior, oblique and odontoid)
- Lateral radiographs of the cervical spine, including all 7 cervical vertebrae are done soon as possible. A lateral view alone will miss 10% of cervical spine injuries.
- Movement of the patient is especially hazardous. 5 individuals are needed to move the patient, keep the head and neck immobile.
- Patients unconscious following a head injury should be assumed to have a cervical injury until proved otherwise by normal radiographs of the cervical spine.

CERVICAL SPINE FRACTURES AND DISLOCATION

JEFFERSON FRACTURE

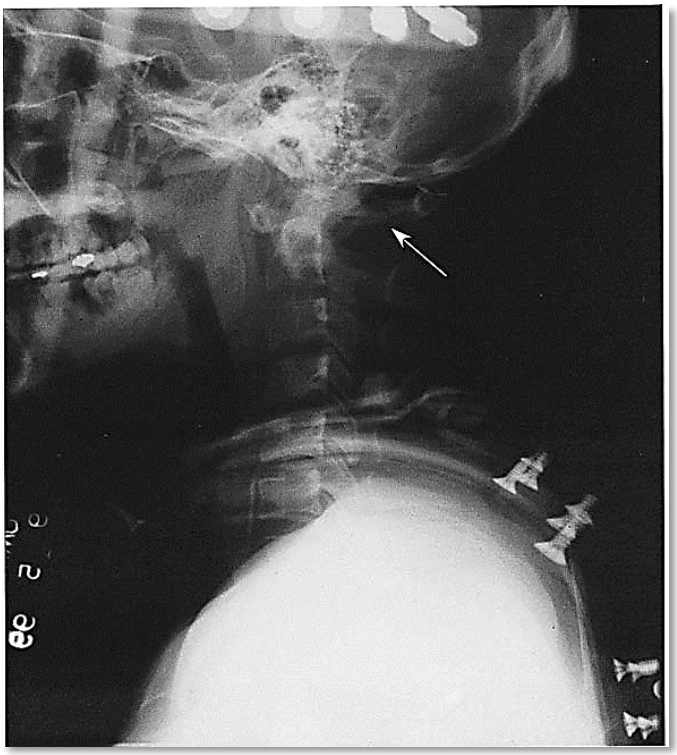


Figure 259: Jefferson fracture

- A Jefferson fracture is a bone fracture of the anterior and posterior arches of the C1 (Atlas) vertebra, though it may also appear as a 3 or 2-part fracture.
- It is a burst fracture.
- It is the most common C1 fracture.
- Results from axial loading on the back of the head or hyperextension of the neck such as when the patient falls directly on his or her head or something falls on the patient's head
- Consider all C1 fractures unstable even though most are not associated with spinal cord injury
- Clinical features:
  - Pain in the upper neck but no neurological signs.
  - There may be damage to arteries in the neck resulting in lateral medullary syndrome, Horner's syndrome, ataxia and inability to sense pain or temperature.
- The use of surgery to treat a Jefferson fracture is somewhat controversial.
- Non-surgical treatment varies depending if the fracture is stable or unstable, defined by an intact or broken transverse ligament and degree of fracture of the anterior arch.
- An intact ligament requires use of soft or hard collar.
- A rupture ligament may require traction, a halo or surgery.
  - Note: the use of rigid halos can lead to intracranial infections and are often uncomfortable for individuals wearing them, and may be replaced with a more flexible alternative depending on the stability of the injured bones but treatment of a stable injury with a halo collar can result in a full recovery.
  - Surgical treatment involves fusion or fixation of the first 3 cervical vertebrae

- Fusion may occur immediately or later during treatment in cases where non-surgical interventions are unsuccessful.
- A primary factor in deciding between surgical and non-surgical intervention is the degree of stability as the presence of damage to other cervical vertebrae.

- Though a serious injury, the long term consequences of this fracture are uncertain and may not impact longevity or abilities even if untreated. Conservative management with an immobilization device can produce excellent long-term recovery.

ODONTOID FRACTURES

- Type 1: involves only the tip of the dens (stable)
- Type 2: involves only the base of the dense (unstable)
- Type 3: fracture through the base and body of C2 (generally unstable
- Note: some type 1 fractures may be stable when the transverse ligament remains intact.

Steels Rule of thirds in regarding to cervical injury

Recall: the anteroposterior diameter of the ring of the atlas is approximately 3cm.

The odontoid process and the spinal cord are each approximately 1cm in diameter

According to steels rule of thirds, the remaining centimeter of free space available for cord allows for some degree of pathologic displacement

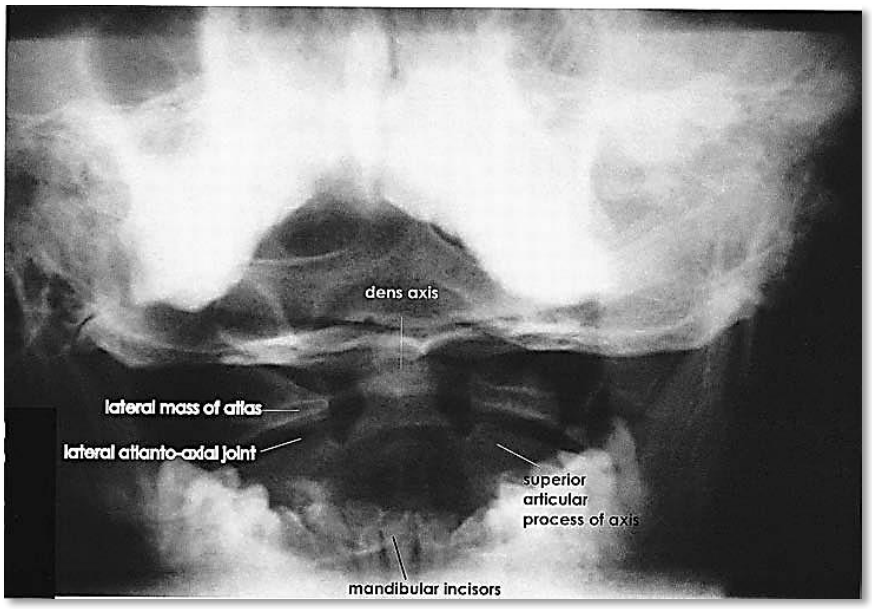


Figure 260: Normal odontoid (open mouth) view of cervical spine

HANGMAN'S FRACTURE

- This involves fracture of both C2 (Axis) pedicles ("posterior elements")
- Usually due to a hyperextension mechanism such as falls (especially in adults), and motor accidents due to impacts of high force causing extension of the neck and great axial load onto the C2 vertebra.
- This type of fracture is often also seen in judiciary hanging.
- Unstable fracture, however, often not associated with spinal cord injury because the spinal canal is at its widest through C2.
- Management is both non-surgical and surgical.

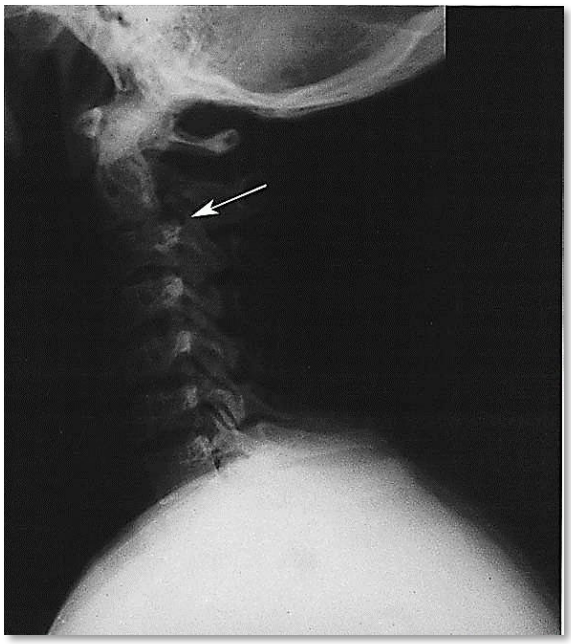


Figure 261: Hangman fracture

CLAY SHOVELER'S FRACTURE

- Usually a flexion injury resulting in an avulsion of the tip of the spinous process (C7>C6>T1)
- It may also result from a direct blow.
- In Australia in the 1930s men digging deep ditches tossed clay 10 to 15 feet above their heads using long handled shovels. Instead of separating, the sticky clay

would sometimes stick to the shovel, the worker would hear a pop and feel a sudden pain between the shoulder blades unable to continue working.

- The mechanism of injury is believed to be secondary to muscle pull and reflex with force transmission through the supraspinous ligaments. The tremendous forces pulls on the spinous process producing an avulsion fracture.
- The fracture is diagnosed by plain film examination.



Figure 262: Clay Shoveler's fracture

THORACIC SPINE FRACTURES

- Most injuries occur at the junction between the relatively fixed upper thoracic spine and the mobile thoracolumbar region (T10-L5). Frequency L1> L2>T1
- When thoracic fractures do take place the results are particularly more devastating.
- This occurs because the spinal canal through this region is relatively narrow and the blood supply to this region is in a watershed area (the greater radicular artery of Adamkiewicz enters the spinal canal at L1 but provides blood flow as high as T4).
- Most thoracic spine fractures are caused by hyperflexion leading to a wedge or compression fracture of the vertebral body.
- Most fractures/dislocations in this area are considered stable because of the surrounding normal bony thorax however neurological impairment resulting from injuries in this area is often complete.

THORACOLUMBAR JUNCTION AND LUMBAR SPINE FRACTURES AND DISLOCATIONS

- Results from axial loading and flexion
- Potentially unstable
- Neurologic injury is uncommon but may result in spinal cord compression.

DISTRACTION OR SEAT-BELT INJURY

- Frequently referred to as a chance fracture.
- Horizontal fracture through the vertebral body, spinous processes, laminae, pedicles and tearing of the posterior spinous ligament.
- It is caused by an acceleration-deceleration injury of a mobile person moving forward into a fixed seat belt.
- Abdominal injuries frequently coexist with this fracture/dislocation

THORACIC OUTLET SYNDROME

- Subclavian artery/vein and brachial plexus pass through a space defined by the clavicle and first rib (thoracic outlet).
- Vascular compromise is more common than neurologic:
  - Unilateral Raynaud's phenomenon
  - Adson's sign- loss of radial pulse on abduction and external rotation of the arm
- It can present with T1 sensory loss, wasting of thenar muscles.
- It is caused by:
  - Fibrous band compressing C8/T1 roots (inferior trunk)
  - Elongated C7 transverse process- "Cervical rib"
- Treatment: surgical lysis of fibrosis band or removal of C7 transverse process by either transaxillary or supraclavicular approach to thoracic outlet.

CNS TUMORS

HYDROCEPHALUS

MISCELLANEOUS

CELLULITIS AND ERYSIPELAS

- Cellulitis is inflammation of the dermis and subcutaneous tissue. It most commonly Group A *Streptococci* and *Staphylococcus aureus*.
- Erysipelas is a superficial cellulitis affecting the dermis and upper subcutaneous tissue. It is commonly due to Group A Streptococci.
- Cellulitis results from pathogen entry through broken skin. Precipitating lesions: trauma, ulcer, bite, skin damage from chronic condition.

RISK FACTORS

- Previous cellulitis
- Chronic disease: Diabetes, chronic kidney or liver disease, cancer
- Immunodeficiency
- Venous insufficiency
- Age
- Skin disease e.g. tinea pedis

CLINICAL FEATURES

- Skin:
  - Inflammation: painful, hot, red, swollen
  - Cellulitis is typically poorly demarcated, while erysipelas is raised and well-defined
  - May be associated with a skin abscess
- Commonest sites:
  - Lower legs. Bilateral lower leg cellulitis rare and is often misdiagnosis of venous eczema
  - Erysipelas may also occur on the face where it can have an (Asymmetrical) butterfly distribution on the cheeks and bridge of nose.
  - Cannula site, possibly with phlebitis
- Non-dermal features:
  - Lymphadenopathy
  - Systemic symptoms: fever. Commoner with erysipelas, where it may precede skin signs by hours.

INVESTIGATIONS AND DIAGNOSIS

- Diagnosis is usually clinical
- Affected area:
  - Look for portal of entry and ask about trauma
  - Draw around edge of area to monitor progress
  - Swab for culture only needed for severe/resistant infection or unusual exposures (penetrating injury, water-borne, acquired abroad)
- Blood investigations:
  - Full blood count: increased WBC
  - C-reactive protein: increased
  - Blood cultures
  - D-dimers (to rule out DVT)
- Imaging:
  - Foot X-ray for osteomyelitis
  - Ultrasound and Well's score to rule out DVT

MANAGEMENT

- Antibiotics
  - Flucloxacillin PO for 5-7 days 1st line for most (for patient allergic to penicillin use doxycycline or clarithromycin PO)
  - If near eyes or nose or for human/animal bite wounds use Co-amoxiclav PO
  - If severe, cefuroxime, ceftriaxone, flucloxacillin, co-amoxiclav, or clindamycin IV
  - If MRSA, vancomycin, teicoplanin or linezolid IV
- Analgesia:
  - Simple analgesia PO
  - Leg elevation can ease pain
- If at cannula site: remove cannula, re-site, and culture needle tip.

COMPLICATIONS

- Thrombophlebitis
- Sepsis
- Toxic shock syndrome
- Lymphangitis and secondary lymphedema
- Cavernous sinus thrombosis if facial

NECROTIZING FASCIITIS

- This is a rare but life-threatening infection of soft tissue with high mortality rate

PATHOPHYSIOLOGY

- Bacteria enters through break in the skin. May follow surgery, trauma, IV injection or insect bite.
- Infection spreads rapidly across fascial layer, leading to tissue death of fascia and subcutaneous tissue.
- Can be:
  - Polymicrobial (type 1)
  - Monomicrobial (type 2): Group A streptococcus is a the most common cause with tissue destruction driven by exotoxins A, B and C
- Fournier's gangrene is a polymicrobial necrotizing fasciitis of the male perineum usually following breach in GI or urethral mucosal. It is commonest in older men.

RISK FACTORS

- IV drug use
- Obesity
- Diabetes

CLINICAL FEATURES

- Rapidly expanding, inflamed area of skin. May progress to bullae and dusky purplish discoloration
- Sever pain out of proportion to skin signs
- Skin crepitus: crackly on palpation
- Sepsis and systemic symptoms

INVESTIGATIONS

- Bloods:
  - FBC: increased WBC
  - Increased CK
  - Increased lactate
- X-ray, CT or MRI may aid diagnosis, showing gas in soft tissue but should not delay referral and surgery if clinical suspicion is high

MANAGEMENT

- Urgent surgical debridement
- IV antibiotics: carbapenem + Clindamycin +/- MRSA cover (e.g. vancomycin)
- Consider IVIG though evidence is limited

GANGRENE

GAS GANGRENE

PATHOPHYSIOLOGY

- Myonecrosis due to alpha-toxin from Clostridium perfrigens. Occasionally caused by other clostridia species.
- Clostridia usually inhabits soil or normal gut flora. They enter muscle in major trauma or GI surgery. Rarely it is non-traumatic (spontaneous) due to colorectal carcinoma or immunosuppression.

CLINICAL FEATURES

- Pain which may have sudden onset and be out of proportion to signs
- Skin crepitus: crackly on palpation
- Skin becomes swollen, dark purple and bullae form
- Septic shock

MANAGEMENT

- Surgical debridement
- IV antibiotics

HAND INFECTIONS

ISCHEMIA AND SWELLING OF THE LOWER LIMBS

SKIN

- The skin is the largest organ in the body (by mass i.e. 4kg).
- Functions of the skin include:
  - Protection:
    - It acts as a physical barrier against thermal and mechanism insults e.g. friction, shearing force and other material
    - It provides protection against infection (innate immunity), chemicals, ultraviolet irradiation (this is a function of melanin) and desiccation from trans-epidermal water loss. The skin participates in antigen presentation/immunological reactions/wound healing.
  - Sensation: pain, touch and temperature
  - Synthetic function:
    - Ultraviolet induced synthesis of vitamin D: cells of the skin synthesize vitamin D3 needed for calcium metabolism and proper bone formation.
    - Hormonal e.g. testosterone
  - Homeostatic function: Temperature and water regulation
    - Constant body temperature is maintained by the skin's insulating components e.g. fatty layer and hair on the head and its mechanism for accelerating heat loss (Sweat production and a dense superficial microvasculature.
  - Sexual signaling: production of sex pheromones by apocrine sweat glands that are important in attraction.
- The skin consists of 3 distinct layers:
  - Epidermis (ectodermal origin): consists of stratified squamous epithelium mainly of keratinocytes which rest on a basement membrane separated from the underlying layers. The epidermis adheres to the dermis by interlocking of its downward projections (epidermal ridges or rete pegs) with upward projections of the dermis (dermal papillae). The epidermis is divided into the following layers depending on whether it is thick skin or thin skin:
    - Stratum basale (basal layer): contains stem cells that divide to produce keratinocytes which constitute 90% of the epidermis and melanocytes which produce melanin that gives the skin its pigmentation (Recall: different color skins are due to the amount of melanin produced and not the necessarily the number)
    - Stratum spinosum: contains cells in layers held tightly by desmosomes which are visualized as bridges/spikes /spines crossing the cells

- Stratum granulosum: contains keratohyalin granules that produce profilaggrin (pro-filament aggregation protein)
- Stratum lucidum: Only seen in thick skin
- Stratum corneum: Upper cells lose their nuclei and become surrounded by a tough impermeable envelope of various proteins
- Remember the mnemonic: "Black Shoes Go Looking Clean" for histological layers of the epidermis

➢ Dermis (mesodermal origin): less cellular and contains blood vessels, nerves, glands (Sweat and sebaceous) and epidermal derived appendages (hair follicles and sweat glands)
- Papillary dermis: The upper layer of the dermis. It is a thin layer and contains dermal papillae, loose connective tissue, type I and II collagen, fibroblast, mast cells, macrophages and other leukocytes.
- Reticular dermis: The deeper layer of the dermis. It is thick and consists of dense irregular connective tissues (type I collagen) and a network of elastic fibers to provide elasticity.

➢ Subcutaneous layer/hypodermis: contains adipose tissue

- The cells of the epidermis include:
  - ➢ Keratinocytes: account for 90% of the epidermal cells. They are derived from the basal cells. It takes 14 days for keratinocytes to reach the stratum corneum and another 14 days for the cells to desquamate (a total of 28 days).
  - ➢ Melanocytes: found in the basal layer and secrete the pigment melanin which is synthesized from tyrosine and packaged into melanosomes transferring it to the keratinocytes via dendritic processes. These are derived from neural crest cells
  - ➢ Merkel cells: play a role in sensation (fine touch). They are found in the basal layer and originate either from neural crest or epidermal keratinocytes
  - ➢ Langerhan's cells: these are dendritic bone marrow derived antigen presenting cells found in the basal layer.

SKIN TUMORS

- Skin tumors of clinical importance include:
  - ➢ Malignant melanoma
  - ➢ Basal cell carcinoma
  - ➢ Squamous cell carcinoma

- Basal cell and squamous cell carcinoma are less aggressive forms and common types of skin cancer whilst malignant melanoma is uncommon but the most aggressive skin cancer of the three.
- It is generally accepted that these cancers are due to sun exposure.
- Skin cancer seems to be more prevalent in people with light skin or numerus moles.

MALIGNANT MELANOMA

- Melanoma is the most frequent cause of death of all skin cancer types.
- It results from malignant transformation of the normal melanocytes usually located in the basal layer of the epidermis.
- Many melanomas are curable by surgical excision.

RISK FACTORS

- Mole showing persistent changes in shape, size, or color. A mole that shows rapid growth or heterogenous pigmentation should be evaluated and possibly biopsied to rule out melanoma.
- Giant naevi or persons having more than 100 nevi, atypical/dysplastic naevi
- Personal history or family history of melanoma
- Excess sun exposure (especially in childhood) and sunburn: UV light is suspected to play a role in the development of all types of skin cancer including melanoma. Although the precise etiologic role of UV light in the malignant transformation of skin cells remain unresolved, both UV-A and UV-B are thought to have carcinogenic potential. UV-A penetrate deep into the dermis, damaging connective tissue and intrinsic skin elasticity. Excessive UVB exposure results in sunburn. (A simple mnemonic is that UVA Age and tans hence its use in sunbeds while UVB Burns and causes cancer)
- Fair complexion
- Tendency to freckle

CLINICAL FEATURES

- Remember the ABCDE's of melanoma (5 signs of melanoma):
  - A- asymmetric shape
  - B- boarders are irregular
  - C- color is mottled or hyperpigmented
  - D-diameter is large (>6mm)
  - E- Enlargement or Evolving lesion
- Common sites:

- Back (males)
- Legs (females)

TYPES OF MELANOMA

- They include:
  - Superficial spreading melanoma
    - Commonest type worldwide
    - Can occur anywhere on both sun-exposed and non-exposed areas.
    - Average age of diagnosis: 40 to 50 years
    - Lesions are commonly on the upper back and lower legs
    - Lesions show heterogenous pigmentation with irregular margins.
    - Growth phase is radial with horizontal spread
  - Lentigo maligna melanoma
    - Usually seen in older individuals (70 years)
    - Lesions appear on sun-exposed surfaces, particularly the malar region of the cheek and temple
    - Lesion exhibit horizontal spread
    - Carries the best prognosis
  - Acral lentiginous menaloma
    - Unusual distribution. Lesions appear on palms, soles, nail beds r mucous membranes (most commonly the vulva, other sites include anus, nasopharynx, sinuses and oral cavity).
    - It is common in Africa
    - Average age of diagnosis=60 years.
    - Spread is in a horizontal pattern
  - Nodular melanoma
    - Can occur at any site and has an a very early malignant potential secondary to a predominantly vertical growth phase. (in contrast to the other 3 variants that show radial growth phases with horizontal spread).
    - Nodular lesions have well-circumscribed borders and uniform black or brown coloring.
    - It is the most aggressive and carries the worst prognosis.
  - Amelanotic

STAGING

- Stage 1: local disease <1.5mm
- Stage II: local disease >1.5mm
- Stage III: regional disease

- Stage IV: metastatic disease

CLARK LEVELS

- This provides an anatomic description of tumor invasion.
  - Level 1: Tumor confined the epidermis
  - Level 2: Tumor extends beyond epidermis into the papillary dermis
  - Level 3: Tumor extends to papillary dermis
  - Level 4: Tumor extends to reticular dermis
  - Level 5: Tumor extends subcutaneous layer (Full thickness)

BRESLOW SCALE

- The Breslow scale defines primary melanomas that are <0.76mm thick as local tumors

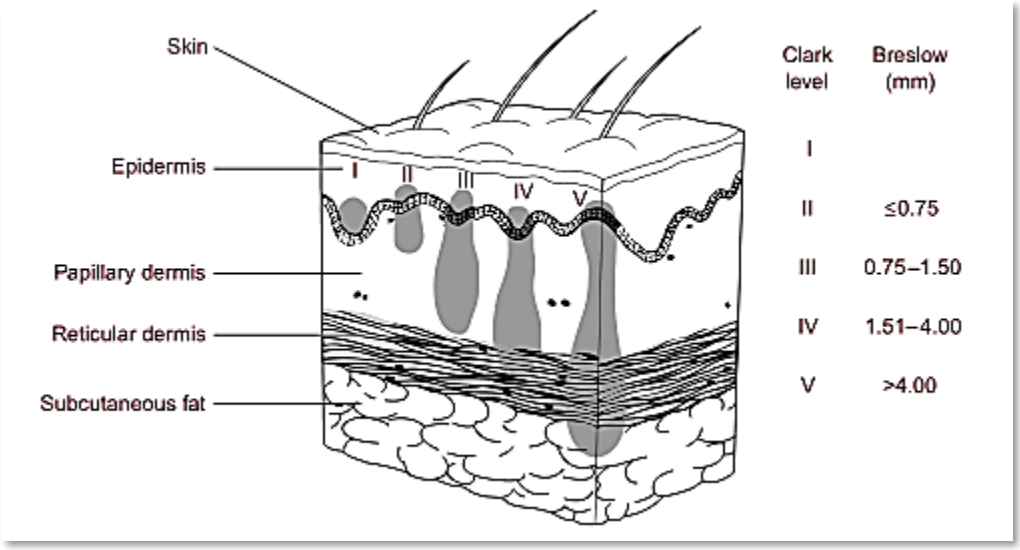


Figure 263: The Clark and Breslow classification for melanoma (Image from Blueprints Surgery)

TNM STAGING

- Stage 1: T1 lesion (≤0.76 mm thick) or T2 lesion (0.76 to 1.50mm) with negative nodes and no mets.
- Stage 2: T3 (1.51-4.00 mm thick) or T4 lesions (>4. 00 mm thick) with negative nodes and no mets
- Stage 3: fewer than 3 regional mets (N1) and no distant mets
- Stage 4: Mets in the skin or subcutaneous, distant lymph node mets or visceral mets

DIAGNOSIS

- Biopsy of the lesion for histology.

MANAGEMENT

- Management depends on the thickness of the primary lesion, location and stage of the cancer.
- Primary choice of management: Wide local excision
  - Most tissue defects are primarily closed without skin graft.
  - If primary biopsy specimen are found to have tumor-negative margins, no further surgical treatment is required.
  - Primary mucosal melanomas have poor outcomes because disease is usually extensive
  - Nail bed lesions require amputation at the distal interphalangeal joint for finger primaries and the interphalangeal joint for thumb primaries
  - Margins for surgical excision:
    - In situ: 5mm margin
    - <1 mm thick: 1 cm margin
    - >1mm thick: 2 cm margin
- Regarding regional disease, the performance of elective regional lymph node dissection for nonpalpable nodes is not routine. The thickness of the primary melanoma is used to predict the chance of regional lymph node metastases.
  - Thin lesions limited to the epidermis have a low likelihood of lymph node metastasis whereas thick lesions invading the subcutaneous fat have higher chance of spread
  - Regional lymphadenectomy is usually only performed for thick lesions with a likelihood of nodal spread
  - Sentinel lymph node biopsy can be done. Using lymphoscintigraphym the sentinel node of the first order lymph node basin into which tumor initially drains is identified. Technetium and a sulfur colloid are circumferentially injected around the primary lesion. Technetium drain via lymphatics to the sentinel node which is identified using a handheld Geiger counter. The sentinel node is excised and evaluated microscopically for evidence of metastasis. Sentinel lymph node biopsy is usually considered for lesions >1mm in thickness.
- In metastatic disease, individuals with a single in identifiable metastatic lesion may benefit from surgical resection.

- Resection is not generally recommended for patients with multiple metastatic lesions.
  - Sites of metastasis: Skin, lung, brain, bone, liver, and the gastrointestinal tract
  - Treatment options: surgery, radiation, chemotherapy and isolated limb perfusion

BASAL CELL CARCINOMA

- This is the most common form of skin cancer in Caucasians. It is rare in Asians and exceedingly rare in darkly pigmented individuals.
- The predominant cause is long-term excess exposure to UV-B radiation.
- BCC is a disease of adults and tumors arise from sun-exposed skin, namely the head and neck.
- The cell of origin of BCC has been thought to be the basal cell of the epidermis however an alternative theory suggests that the originating cell type is a pluripotent epithelial cell.
- BCC is the commonest but least dangerous skin cancer. It is also known as 'Rodent ulcer' as they burrow through tissue causing local damage but almost never metastasize.
- BCC is categorized into 3 types:
  - Noduloulcerative (most common)
  - Superficial
  - Sclerosing

CLINICAL FEATURES

- Shiny nodule 'pearly' with rolled, red telangiectatic edge and eroded center.
- Slowly increases in size
- Usually on sun-exposed sites especially upper 2/3 of face.

TYPES OF BASAL CELL CARCINOMA

NODULOULCERATIVE

- This is the most common type of BCC.
- Lesions have a pearly, dome-shaped, nodular appearance with associated telangiectasia and an ulcerated center.
- Telangiectasia is secondary to tumor-induced angiogenesis and ulceration results from outgrowth of the local blood supply.
- Tumors <1cm in diameter are rarely invasive and can be treated with cautery and curettage or cryosurgery.

- Tumors >1cm are treated with surgical excision. High risk sites of tumor growth are areas with underlying bone and cartilage (i.e. nose, ear), because the growing tumor tends to track along these structures. High risk tumors and recurrent tumors should be treated with Moh micrographic surgery to ensure complete excision.

SUPERFICIAL

- This is the second most common BCC.
- Lesions usually appear on the trunk and proximal extremities and clinically resemble thin, scaly, pink plaques with irregular margins.
- These horizontally expanding tumors are often dismissed as dermatitis, subsequently tumors may reach diameters of several centimeters by the time of diagnosis. By this late stage, ulceration and deep dermal invasion are present.
- Standard treatment has been wide-margin excision with skin graft if necessary. However, this approach may be unacceptably morbid leaving a large skin defect.
- Topical chemotherapy with fluorouracil, cryosurgery and cautery/curettage has shown cure rates similar to those of traditional wide excision

SCLEROSING

- This is the least common type. The anatomic distribution is similar to that of noduloulcerative but histologically the lesions appear as narrow cords of tumor cells encased in a proliferation of connective tissue.
- Macroscopically lesions are smooth atrophic and indurated and easily mimic scar tissue. This deceptive appearance is unfortunate because sclerosing tumors are more aggressive than other basal cell tumors.
- The growth pattern follows tissue planes and neurovascular bundles, resulting in deep soft tissue invasion. Moh micrographic surgery is preferred management technique.

MANAGEMENT

- Wide local excision biopsy with 4mm margin is the gold standard.
- Alternatives:
  - Mohs' micrographic surgery: segments are excised in stages and examined histologically. Consider for difficult sites where tissue preservation is important e.g. eyelid.
  - Curettage: Removal of tumor soft tissue and cautery of the base. Consider for small BCCs (<1 cm) but downside is that margins are difficult to assess.

- Topical cryotherapy or medication (5-fluorouracil, imiquimod). Incisional biopsy is still needed to confirm the diagnosis. Higher recurrence rates than surgery.
- Radiotherapy: an alternative or adjunct to surgery. Good cure rate but may have poor cosmetic outcome.

SQUAMOUS CELL CARCINOMA

- This is second most common form of skin cancer after BCC.
- Tumors arise from the skin and the oral and anogenital mucosa. Multiple predisposing factors for development of SCC include:
  - Sunlight exposure
  - Human papilloma virus infection
  - Immunosuppression: Transplant recipients, HIV infection
  - Chronic ulcers
  - Ionizing radiation (X-rays, gamma rays)
  - Tobacco use
  - Scars (burn injury)
  - Xeroderma pigmentosum
- Squamous cell carcinoma is a malignant proliferation of keratinocytes (squamous cells).
- Most remain localized, but 5% metastasize usually to lymph nodes.
- Marjolin's ulcer is a rare SCC sub-type which arises within existing ulcers or chronically damage skin.

CLINICAL FEATURES

- SCC appears as an indurated nodule or plaque often with ulceration which has slowly evolved over time. Most lesions are on sun-exposed areas, such as the face, ears and upper extremities.
  - Ulcerated, irregular lesion with hard raised edge. May be painful. Increases in size over months.
- Caucasians exhibit pinkish lesions, whereas darker-skinned individuals have hypo- or hyperpigmented lesions.
- Regional lymphadenopathy occurs in 35% of SCCs arising in the lip and mouth.
- Aberrant keratinization is often seen in SCC occasionally causing the growth of cutaneous horns. The base of a cutaneous horn should always be examined for the presence of squamous cell cancer.

MANAGEMENT

- Treatment is tumor removal by wide local surgical excision with 4mm margin.
- Consider topical imiquimod or 5FU if superficial
- The remaining defect is closed either primarily for smaller lesion or by skin grafting or flap reconstruction for larger lesions. Cryosurgery or cautery/curettage can also be used for small tumors.
- Prognosis is excellent.

PROCEDURES

SCRUBBING UP AND GOWNING

DONNING GLOVES

SUTURE AND SUTURING TECHNIQUES
LOCAL ANESTHETICS

TYPES OF SUTURES

SUTURING TECHNIQUES

AIRWAY DEVICE INSERTION
LARYNGEAL MASK

ENDOTRACHEAL INTUBATION

CRICOTHYROIDOTOMY

TUBE THORACOSTOMY

NEEDLE THORACOCENTESIS

URINARY CATHETERIZATION

SUPRAPUBIC CATHETERIZATION

DIGITAL RECTAL EXAMINATION

DRAWING BLOOD AND INTRAVENOUS CANNULATION

DRAWING BLOOD

- Before drawing blood ensure you:
  - Know the patient
  - Know the tests to be ordered for i.e. you must know the correct bottle with the correct additive to use
  - Gain consent from the patient
  - Complete the blood forms (lab request forms) for the tests required.
  - Label bottles fully with name, hospital number, date of birth and date of sample
- Equipment:
  - Blood taking device (vacutainers, syringe, butterfly)
  - Tourniquet
  - Alcohol swab
  - Cottonwool ball
  - Plaster
  - Blood forms
- Procedure:
  - Gather your equipment as listed above
  - Ensure the prerequisites are met as stated above
  - Introduce yourself to the patient and explain what you are going to do and why

- Attach the tourniquet to the upper arm of the patient and ask them to open and close their hand (making a fist)
- Select a vein, usually the antecubital fossa is the best. Also make sure you are not taking blood from an arm that has an intravenous drip running into it.
- Swab the selected vein with the alcohol
- Warn the patient you are about to start and insert the needle at 30^{o} to the skin
- Insert a bottle into the vacutainer barrel, if the needle in the vein, the bottle will fill with blood, if not adjust the needle
- Fill all the bottle required
- Remove tourniquet
- Place cottonwool ball over needle and withdraw it from the skin, the patient should apply pressure for 5min.
- Dispose of the sharps.
- Placed labeled bottle with the completed forms in a bag for transfer to the laboratory.
- Apply a small plaster to the patient's arm.

Blood collection tubes (Vacutainer system)

EDTA liquid	Forms calcium salts to remove calcium	Haematology (FBC) and blood bank (crossmatch): gently invert bottle after filling to prevent clotting and platelet clumping
Sodium citrate	Forms calcium salts to remove calcium	Coagulation tests (INR), full draw required
Plasma separating tube (PST) with lithium heparin	Anticoagulates with lithium heparin; plasma is separated with PST gel at the bottom of the tube	Chemistries
None	Serum separator tube (SST) contains a gel at the bottom to separate blood from serum on centrifugation	Chemistries, Immunology and Serology
None	Blood clots, and the serum is separated by centrifugation	Chemistries, Immunology and Serology, some blood bank profiles
Sodium fluoride and potassium oxalate	Antiglycolytic agent preserves glucose for up to five days	For glucose levels
Sodium citrate (buffered)	Forms calcium salts to remove calcium	Erythrocyte Sedimentation Rate (ESR)

Correct order of draw
(as recommended by the National Committee for Clinical Laboratory Standards, 1991 Guidelines Reference H3A3)
- Blood culture (Bacteriology)
- Plain or serum (no additives)
- Coagulation profiles
- Other additive tubes, e.g. EDTA, lithium heparin, glucose

Figure 264: Blood collection bottles

PERFORMING A FEMORAL TAP

- Equipment:
  - Alcohol swab
  - Syringe

- Cottonwool ball
- Plaster
- Blood forms

- Procedure:
  - Introduce yourself to the patient and explain what you are going to do and why
  - The patient should be supine with the groin and leg extended and slightly abducted
  - Locate the femoral artery, halfway between the anterior superior iliac spine and pubic symphysis, 2cm below the inguinal ligament. You should be able to feel the pulsating of the femoral artery. The femoral vein lies medial to the femoral artery, mark out the femoral vein.
  - Clean the skin directly over the vein with an alcohol swab
  - Fix the vein between two fingers while inserting the syringe and needle at 90^{o}
  - Slowly advance the needle until there is free flow into the synrine
  - Withdraw the needle and apply the cottonwool ball pressure for 5 minutes
  - Place the blood in the appropriate blood bottle and send to the lab.

INSERTION OF AN INTRAVENOUS CANNULA

- Equipment:
  - Intravenous cannula:
    - Blue 22G (very small)
    - Pink 20G (small)
    - Green 18G (average)
    - Grey 16G (large)
    - Brown 14G (very large)
  - Tourniquet
  - Saline flush or intravenous fluid with giving set
  - Securing dressing
  - Sharps bin

- Procedure:
  - Gather your equipment, introduce yourself to the patient and explain what you are about to do.

- Apply a tourniquet to upper arm and ask the patient to open and close their hand
- Select a vein that is palpable and straight, if the cannula is for long-term use, it is best situated away from joints e.g. lower forearm cephalic vein. In an emergency, select a large-bore cannula in an antecubital fossa vein.
- Take the cannula, remove the sheath, open the wings and introduce the needle into the vein at 30^{o} to the skin. Once the needle is in the vein there will be a visible flash-back in the barrel of the needle.
- Slide the cannula into the vein over the needle while removing the needle
- Remove the tourniquet, remove the needle and put white cap on the end of the cannula
- Dispose of sharps
- Apply a dressing to the cannula
- Flush the cannula with saline
- Eliminate any air bubbles from the giving set of the intravenous fluid and then attach it to the cannula
- Set infusion rate and note time infusion started

VENOUS CUT-DOWN

- If emergency intravenous access is required and no other sites are available, then a venous cut-down can be performed. The most consistent and easily accessible large vein is the long saphenous vein, alternative sites include the antecubital fossa and the wrist. Aseptic technique should be used.
- Procedure:
  - Infiltrate the skin with local anesthetic
  - A transverse incision is made 1cm above and anterior to the medial malleolus
  - The vein is gently dissected out, to control the vein two ligatures are passed under it
  - The digital ligature is tied, the other is left untied until a cannula is inserted
  - A large bore cannula is inserted through the skin and into the vein, the proximal ligature is then tied to keep the cannula in place
  - The skin is closed and the cannula secured with a further suture and a dressing.

CENTRAL VENOUS CANNULATION

- Access to the central veins is a common requirement in modern practice and is important for:
  - Measurement of central venous pressure

- Infusion of irritant drugs e.g. amiodarone, dopamine, streptokinase
- Infusion of parenteral nutrition
- Measurement of pulmonary artery wedge pressure
- Infusion of chemotherapeutic agents
- Placement of pacing wires

- It is important to note that although fluids can obviously be given through a central line, it is not the method of choice if fluid needs to be infused quickly as central lines tend to be long and thin.
- 2 different approaches can be used:
  - Supraclavicular approach: preferred vein to cannulate is the right internal jugular vein because it is easily accessible, has a straight path to the right atrium and possible injury to the thoracic duct is eliminated. There is also less risk of damaging the pleural cap and causing a pneumothorax hence it should be the method of choice for the less experienced practitioner.
  - Infraclavicular approach: used by more experienced practitioner, there is an increased risk of pneumothorax and should not be used in patients with clotting abnormalities as it is difficult to stem bleeding from the subclavian vein if hemorrhage occurs. This approach makes nursing care of the central line easier and is more comfortable for the patient.
- Equipment:
  - Central line set:
    - Central venous catheter
    - Syringes
    - Introducing wire
    - Cannulation needles
    - Scalpel blade
    - Track dilator
  - Saline
  - Local anesthetic
  - Suture material
  - Antiseptic
  - Transparent dressing
  - Giving set
  - Sterile pack
  - Sterile glove
- Supraclavicular Procedure (internal jugular approach):

- You need an assistant to pass the items while you are sterile. Maintain the uttermost sterility during the procedure
- Explain the procedure to the patient.
- Remove the pillows from under the head and tilt the patient 30^{o} head down. Some patients may not tolerate this especially those in heart failure.
- Place a bag of fluids under the right shoulder to bring the vessels forward
- Expose the area and turn the patient's head to the left

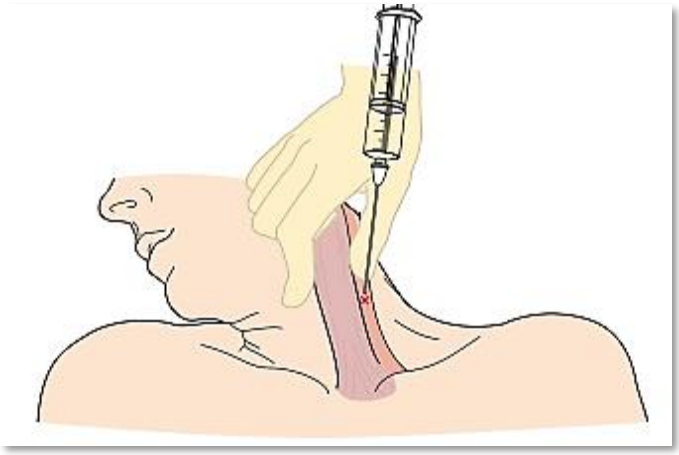


Figure 265: Insertion of a central venous cannula: internal jugular approach

- Scrub and don sterile gloves. With patient in position paint the area where you are going to inset the central line with antiseptic
- Inject local anesthetic into the skin and deeper tissues at the site of cannulation
- The right internal jugular vein runs from the base of the skull anterolaterally in the carotid sheath below the sternocleidomastoid muscle to meet the innominate vein and thence into the superior vena cava.
- The vein can be approached either high or low.
- In the high approach the landmark for cannulation is half distance along a line between the sternal head of the clavicle and the mastoid and lateral to the sternocleidomastoid muscle.
- Enter the skin with a needle connected to a syringe with saline. Advance the needle deep to the sternocleidomastoid and point towards the suprasternal notch until you freely aspirate blood.
- In the low approach to the vein, the landmark is the triangle formed by the sternal and clavicular heads of the sternocleidomastoid, the vein lies between them.

- Infrasternal procedure (subclavian approach)
  - In this approach, the entry point of the needle should be 3cm below the midpoint of the clavicle, with the needle angled towards the jugular notch.
  - Occasionally, the needle may abut against the clavicular periosteum, this will be painful, so it is important to anesthetize down the clavicle. If the clavicle is encountered, then gentle downward pressure should be applied to the needle to help it enter the vein.

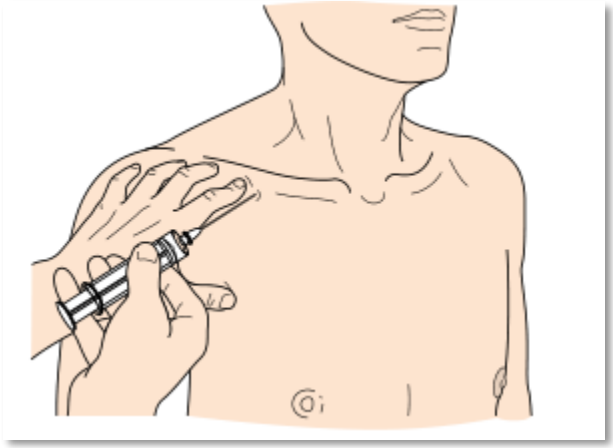


Figure 266: Insertion of a central venous cannula: Subclavian approach

- Seldinger technique:
  - This is common to whichever method of insertion is used.
  - After the vein has been cannulated, a flexible wire with a leading J end is inserted into the needle and fed through the vein towards the right atrium.
  - ECG monitoring helps gauge whether the wire has been inserted too far and is impinging on the myocardium.
  - When the wire has been placed, the needle is removed over the top of it. The track formed by the wire and placing a dilating device over it. The central venous catheter is then fed into the vein over the wire, the wire is removed through the cannula.
  - The cannula is aspirated and flushed with saline to check it is working, is sutured in place and a transparent dressing applied and a giving set attached to the line. Once the patient has received a CXR to check position and exclude a pneumothorax, the line can then be used.
- Complications of central line insertion
  - Early
    - Hemorrhage
    - Misplacement
    - Pneumothorax
  - Intermediate
    - Infection
    - Blockage
  - Late
    - Scarring
    - Myocardial damage

NASOGASTRIC TUBE INSERTION

- This is a tube inserted into the stomach via the nose and is used for either drainage or feeding.
- The most commonly used nasogastric tubes (NGT) are of the drainage type, are made of plastic and are called Ryle's tubes (16F).
- Tubes used for feeding are likely to be placed for a longer period of time, are thinner and made of silicon (Fine bore nasogastric tubes) which is softer and hence more comfortable for the patient and blocks less readily.
- Passage of an NGT is contraindicated in patient with a head injury because of the risk of passing the tube through a fractured cribriform plate into the brain. If drainage of the stomach is required, an orogastric tube can be passed.
- This is a skill that is important to know. Most medical schools now have mannequins to practice this on, although it's not frequently examined upon. However, it is very useful to know how to do- you don't want your first time to be the middle of the night as a junior doctor when the nurses who normally do it, call you as they cannot!
- Equipment:
  - NGT made stiffer by storing in refrigerator
  - Lubricating jelly
  - Bladder syringe
  - Drainage bag
  - Securing tape
  - Cup of water
  - Litmus paper
- Procedure
  - Wash your hands, don glove, introduce yourself and explain to the patient that the procedure is not particularly pleasant and insertion of the tube may cause them to retch.
  - Position the patient upright and facing forward
  - Inspect the nose for any obvious deformities

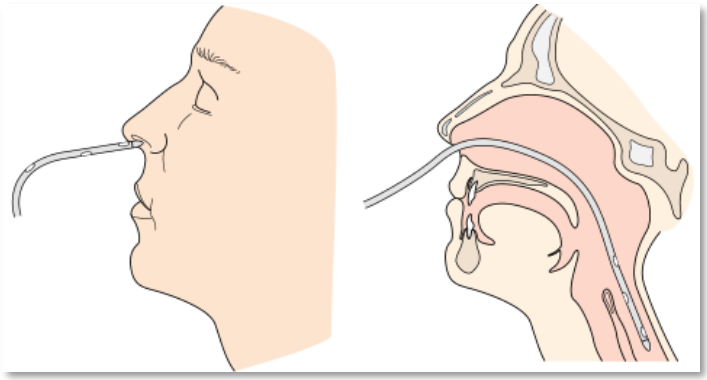


Figure 267: Insertion of a nasogastric tube

- Estimate the length of the tube to be inserted. Do this by measuring the NG tube from the tip of the nose, to the earlobe and then the xiphisternum (5cm below it).
- Lubricate the tube
- Insert the tube into nostril pointing towards the occiput. If you encounter any resistance change to the other nostril.
- Ask the patient to swallow a mouthful of water, and as they do advance the tube
- To check the tube is in stomach aspirate contents and test for acidity (pH 1-5.5) with litmus. Alternatively instill 30ml of air with the bladder syringe and auscultate the epigastrium for bubbling
- Secure the tube to the nose and attach a drainage bag
- Document the result in the patient's notes.

- Insertion of a feeding tube is similar to that described above, except that feeding tubes have a wire within them as they have insufficient rigidity for insertion into the stomach. An X-ray is taken of the epigastrium to check the position of the wire. When in the correct position the wire is removed leaving the feeding tube in place.

ANSWERS TO CLINICAL BOX

A CASE OF EPIGASTRIC PAIN AND VOMITING

1. Likely diagnosis: Acute pancreatitis because:
   - History: age, positive alcohol drinking, vomiting and epigastric pain radiating to the back
   - Raise serum amylase (although serum lipase would have been more specific, serum amylase can be elevated in a patient with gastric perforation due to systemic absorption of pancreatic enzymes from the abdominal cavity, an amylase rise of over 1000IU/dl is usually diagnostic of acute pancreatitis)
   - Macrocytosis which suggests the alcoholic etiology
   - Note: an ultrasound may be needed to rule out gallstones
2. Differential diagnosis to exclude:
   - Gallstones (ultrasound)
   - Perforated peptic ulcer (Erect chest X-ray which would show free subphrenic air in 90% of cases)
3. Ranson's criteria is used to grade the severity of alcoholic pancreatitis but it takes 48 hours before the score can be used. Each fulfilled criterion scores a point and the total indicates the severity.
   - On admission
     - Age>55 years
     - WBC >16 x 10^9L
     - LDH >600 IU/L
     - AST> 120IU/L
     - Glucose >10 mmol/L
     - Fluid sequestration >6L
   - Within 48 hours
     - Hematocrit fall >10%
     - Urea rise >0.9 mmol/L
     - Calcium <2 mmol/L
     - Partial pressure of oxygen <60mmHg
     - Base deficit >4
   - Points
     - 0-2= 2% mortality
     - 3-4= 15% mortality
     - 5-6= 40% mortality
     - >7= 100% mortality

4. Causes of acute pancreatitis
   - Alcohol and gallstones (common=80%)
   - Trauma (RTA)
   - Hypercalcemia, hyperlipidemia, scorpion stings, rupture of posterior duodenal ulcer
   - Rare= infections (mumps, coxsackie B virus), drugs (thiazide, corticosteroids, salicylates, cimetidine), pancreatic structural anomalies (pancreatic divisum), ampullary or pancreatic tumors
5. Causes of elevated serum amylase
   - Perforated peptic ulcer
   - Mesenteric infarction
   - Cholecystitis
   - Generalized peritonitis
   - Intestinal obstruction
   - Rupture ectopic pregnancy
   - Diabetic ketoacidosis
   - Liver failure
   - Bowel perforation
   - Renal failure
   - Rupture abdominal aortic aneurysm
6. Management
   - Medical:
     - Admit to ICU
     - ABCs- prompt fluid resuscitation, a urinary catheter and central venous pressure monitoring, fresh frozen plasma and blood transfusion
     - Total parenteral feeding
     - Antibiotics: 3rd Gen cephalosporins (cefotaxime), imipenem
     - Anticholinergics
     - Calcium gluconate 10ml 10% 8 hourly
     - IV omeprazole 40mg BD
     - Analgesics- pethidine (Avoid morphine)
   - Surgical: open surgery is the gold standard
7. Complications
   - Local: pancreatic pseudocyst, abscess formation, biliary obstruction, fistula formation, thrombosis
   - Systemic: renal failure, septic shock, hypovolemic shock, electrolyte imbalance, ARDS, respiratory failure, multi-organ failure

END OF BOOK REVISION

TRUE/FALSE

1. Long term sequelae of head injury:
   A. Brain atrophy is a feature
   B. Focal neurological deficits are not a feature
   C. Memory deficits are not a feature
   D. Epilepsy is a feature

2. Clinical classification of head injury is based on:
   A. Mode of injury
   B. Whether it is open or closed
   C. Glasgow coma scale
   D. Pathological lesion

3. Subdural hematoma follows:
   A. Tearing of superior sagittal sinus
   B. Tearing of cerebral veins
   C. Middle meningeal artery laceration
   D. Vertebral artery laceration

4. Head injury patient with subdural hematoma
   A. Is likely to deteriorate faster if blood pressure is reducing
   B. Is likely to deteriorate if blood pressure is increasing
   C. Is likely to deteriorate faster if pupils are starting to become unequal
   D. Is likely to deteriorate faster if hemiparesis is starting to present

5. The main source of bleeding in a deep scalp laceration is
   A. Periosteum
   B. Loose connective tissue
   C. Aponeurotica galea
   D. Dense connective tissue

6. Epidural hematoma in head injury patient follows:
   A. Tearing of cerebral veins
   B. Brain laceration
   C. Middle meningeal artery laceration
   D. Superior sagittal sinus tear

7. Head injury patient who presents with skull fracture crossing the midline
   A. Is likely to have epidural hematoma from superior sagittal sinus laceration
   B. Is likely to have epidural hematoma from middle meningeal artery laceration
   C. Is likely to have cerebral spinal fluid leaking from both ears and nose
   D. Is likely to have fracture dislocation of C5/C6 cervical spine

8. Burns are classified in terms of depth as
   A. Superficial partial thickness
   B. Deep partial thickness
   C. Full thickness
   D. Full thickness is also known as second degree burns

9. The rule of 9 for estimating extent of burns is as follows:
   A. Head and neck 9%
   B. Front of trunk 18%
   C. Perineum 11%
   D. Upper limbs 18%

10. Cerebrospinal otorrhea:
   A. Fracture of the patients' temporal bone is always present
   B. Fracture of the base of the skull is the cause
   C. Immediate surgical repair is indicated
   D. Infection is common
   E. Indicates injury to the 6^{th} cranial nerve

11. The comatose patient with a head injury:
   A. Is nursed on his side to prevent aspiration of stomach content
   B. Must have endotracheal intubation if pharyngeal reflexes are absent
   C. Must have a nasogastric tube inserted
   D. A urinary catheter is useful for monitoring fluid balance
   E. A suction machine is essential

12. Urine output in a burnt patient:
    A. In children an output of 1ml/kg/hr must be maintained
    B. In adult ensure an output of 70ml/hour
    C. Catheterize all patients with extensive burns
    D. In a 3-year-old female child size 24 Foley is used
    E. Antibiotics are routinely given to catheterized patient

13. Cerebrospinal otorrhea complicating head injury requires:
    A. Antibiotic ear drops
    B. Plugging the external auditory meatus
    C. Syringing with saline
    D. Early dural repair
    E. Parenteral antibiotics

14. Systemic effects of burns
    A. Hyper-metabolism
    B. Immunosuppression
    C. SIRS
    D. Keloids
    E. Contractures

15. Complications of burns
    A. Marjolin's ulcer
    B. Keloids
    C. Systemic inflammatory response syndrome
    D. Renal failure
    E. Airway obstruction

16. Triage refers to:
    A. Surgical wound debridement
    B. Categorizing and sorting patients according to priority
    C. Casualty evacuation
    D. Only B and C are correct

17. In mass casualty injuries, the following injuries require immediate treatment
    A. Sucking wounds of chest
    B. Flail chest
    C. Tension pneumothorax
    D. Penetrating wound of the abdomen

18. Complications of intercostal drain insertion:
    A. Pneumothorax
    B. Injury to intercostal neurovascular bundle
    C. Lung and liver trauma
    D. Surgical emphysema

19. The edge of a tuberculous ulcer is
    A. Shelving
    B. Rolled up
    C. Undermined
    D. Punched out

20. The edge of a malignant ulcer is
    A. Shelving
    B. Rolled up
    C. Undermined
    D. Punched out

21. Debridement is
    A. The process of making an incision
    B. The removal of foreign material or dead tissue
    C. The process of obtaining hemostasis
    D. None of the above

22. About ulcers:
    A. Diabetic ulcer is an example of a specific ulcer
    B. Malignant melanoma ulcer is always pigmented
    C. Arterial ulcers are located on the medial aspect of the leg
    D. Trophic ulcers are painful

23. Acute raised intracranial pressure is treated by:
    A. Mannitol
    B. Furosemide
    C. Hyperventilation
    D. Steroids
    E. Raising the foot end of the bed

24. Decubitus ulcers
    A. Only occur in comatose patients
    B. Are a form of ischemic necrosis
    C. Cause septicemia
    D. Antibiotics cure the condition
    E. Surgery is of value in selected cases

25. History taking from a patient
    A. Accounts for 10% of the diagnostic process
    B. Open ended questions direct the interview
    C. Referral letter may reveal important information
    D. Occupation of the patient is not important
    E. Smoking is associated with pancreatitis

26. Skin grafting
    A. Auto graft refers to the use of pig skin
    B. Xenograft usually takes and becomes part of the body skin
    C. Allograft fails to provide any normal function
    D. Split thickness skin grafting needs pedicles to survive
    E. Split thickness graft heals with excellent appearance

27. Early signs of a rising intracranial pressure include:
    A. Bradycardia
    B. Deteriorating conscious level
    C. Hypothermia
    D. Hypertension
    E. Tachypnea

28. The Glasgow coma chart includes details on the following
    A. Pupillary size
    B. Motor response
    C. Urine output
    D. Hemoglobin level
    E. Injury severity score

29. Pneumothorax
    A. Occurs spontaneously sometimes
    B. Bullae are a cause
    C. Is a complication of flail chest
    D. Thoracotomy is frequently indicated
    E. A chest tube is always necessary

30. Spontaneous pneumothorax is caused by
    A. Tuberculosis
    B. Emphysematous bullae
    C. Solitary lung cyst
    D. Chronic bronchitis
    E. Idiopathic

31. Decerebrate rigidity
    A. Is commonly seen in patients with Glasgow coma scale of 8 or less
    B. Is commonly seen in patients with Glasgow coma scale of 12 or more
    C. Is commonly seen in a patient with stiff extension of limbs
    D. Is commonly seen in a patient who is well oriented in person, space and time.

32. Brainstem injury is mostly likely to have occurred in patient who presents with:
    A. Glasgow coma scale 15/15
    B. Glasgow coma scale 15/15 and unequal pupils
    C. Cerebral spinal fluid leak from both ears
    D. Occipital scalp laceration

33. All end colostomies are:
    A. Permanent colostomies
    B. Defunctioning colostomies
    C. Not suitable for ano-rectal malformation
    D. Hartmann's colostomies

34. Complications of colostomy include
    A. Bleeding
    B. Diarrhea
    C. Prolapse
    D. Retraction
    E. Parastomal hernia

35. Types of colostomy
    A. Spectacle colostomy
    B. Double barrel colostomy
    C. Ileostomy
    D. Gastrostomy
    E. Pharyngostomy

36. Indications for permanent end-colostomy
    A. Stage 1 carcinoma of the sigmoid
    B. Carcinoma of the caecum
    C. High fistula-in-ano
    D. Inoperable vesicle-vaginal fistula
    E. Duke-B squamous cell carcinoma of the anus

37. A patient with flail chest
    A. Does not require a chest drain
    B. Rarely requires ventilation
    C. Should be nursed in a dark room
    D. The flail segment moves in on inspiration
    E. Should be treated in the intensive care unit

38. Complications of defunctioning loop colostomy of the sigmoid colon include
    A. Electrolyte imbalance
    B. Excoriation of skin
    C. Retraction
    D. Prolapse
    E. Internal hernia

39. Snake bite results in the following:
    A. Visible puncture wounds
    B. Swelling of the bitten limb
    C. Occasionally gangrene
    D. Disseminated intravascular coagulation frequently
    E. Fat embolism

40. In a jaundiced patient with palpable gallbladder, the cause of jaundice is most likely to be:
    A. Gall stones in the gall bladder
    B. Gall stone in the common bile duct
    C. Cancer of head of pancreas
    D. Cancer left kidney

41. Generalized peritonitis is likely to follow
    A. Delayed diagnosis of perforated duodenal ulcer
    B. Acute pancreatitis
    C. Ruptured appendicitis
    D. Torsion of the testis

42. Bilateral lower abdominal pain in a female patient is most likely caused by
    A. Ectopic pregnancy
    B. Appendicitis
    C. Salpingitis
    D. Ureteric stone

43. Small intestinal obstruction is likely to present with
    A. Radiating pain to the left shoulder
    B. Spreading pain to the right iliac fossa
    C. Colic pain in the umbilical region
    D. Early vomiting

44. During wound healing fibroblasts migrates across the following protein:
    A. Fibrillin
    B. Fibronectin
    C. Elastin
    D. Laminin

45. Regarding collagen
    A. It is composed to tropocollagen
    B. It is synthesized mostly by mast cells
    C. Type II collagen is most common in connective tissue proper
    D. None above are correct

46. Basement membrane consists of:
    A. Type I collagen
    B. Type II collagen
    C. Type III collagen
    D. Type IV collagen

47. The following factors do not contribute to wound disruption
    A. Massive blood transfusion
    B. Anemia
    C. Abdominal distention
    D. Vitamin C deficiency

48. 7 days after suturing, tensile strength of a wound will be:
    A. 5% of the uninjured tissue
    B. 20% of the uninjured tissue
    C. 30% of the uninjured tissue
    D. 50% of the uninjured tissue

49. Acute appendicitis
    A. Pain can be referred to the right subcostal region
    B. Pain can shift to right iliac fossa
    C. Can be associated with vomiting
    D. Can be associated with left hip joint flexion

50. Large bowel intestinal obstruction
    A. Early vomiting is a feature
    B. Gross abdominal distension is a feature
    C. Sigmoid volvulus is a possible cause
    D. Rectal carcinoma cannot be the cause

51. Wound healing
    A. Is impaired in anemic patients
    B. Impaired in hematoma formation
    C. Impaired in hypoproteinemia
    D. Stimulated by steroids

52. Intestinal obstruction
    A. Can be classified depending on the age of the patient
    B. Can be made on erect clinical examination
    C. Can be due to electrolyte imbalance
    D. Pyloric stenosis is caused by lower intestinal obstruction

53. Obstructive jaundice
    A. Is associated with biliary atresia
    B. Is associated with itching of the skin
    C. A patient with obstructive jaundice passes pale stool
    D. Obstructive jaundice can be diagnosed by supine X-ray

54. Complications of intercostal drain insertion
    A. Pneumothorax
    B. Injury to intercostal neurovascular bundle
    C. Lung and liver trauma
    D. Surgical emphysema

55. In acute appendicitis
    A. Central or epigastric pain radiates to right iliac fossa
    B. Central or epigastric pain shifts to right iliac fossa
    C. Pain precedes nausea and vomiting
    D. Nausea and vomiting precedes pain

56. In acute appendicitis
    A. Mantrel (Alvarado) scores of 7 out of 10 is highly suggestive of appendicitis
    B. Right iliac fossa (McBurney's point) tenderness and leukocytosis are allotted 2 points each on mantrel scale
    C. Rovsing's sign and obturator test are contributory to diagnosis
    D. Digital rectal examination has no value

57. In wound healing
    A. Cytokines are solely produced by hemopoeitic cells
    B. Cytokines optimize cellular response in inflammation
    C. Interleukins are not classified as cytokines
    D. Macrophages actively synthesize cytokine growth factors for angiogenesis

58. In a completely healed wound, the tensile strength will be
    A. 20% of uninjured tissue
    B. 40% of uninjured tissue
    C. 60% of uninjured tissue
    D. 80% of uninjured tissue

59. Wound closure using suture is termed
    A. Closure by primary intention
    B. Closure by secondary intention
    C. Hemostasis
    D. Only (C) is correct

60. Factors which affect wound healing include:
    A. Chronic illness
    B. Radiation therapy
    C. Nutrition
    D. All above are correct

61. The following conditions are associated with intestinal obstruction
    A. Imperforate anus
    B. Hirschprung's disease
    C. Peritonitis
    D. Massive potassium loss

62. Common causes of intestinal obstruction in the pediatric age group include:
    A. Diaphragm of the duodenum
    B. Ileal duplication
    C. Intussusception
    D. Imperforate anus
    E. Peritonitis following a ruptured appendix

63. In the University Teaching Hospital colostomies are commonly performed for the following conditions
    A. Volvulus of the sigmoid colon
    B. Vesicovaginal fistula
    C. An obstructed umbilical hernia
    D. Imperforate anus
    E. Meckel's diverticulum

64. Paralytic intestinal obstruction occurs in
    A. Hypokalemia
    B. Hyponatremia
    C. Peritonitis
    D. Hemoperitoneum
    E. Retroperitoneal hematoma

65. Mechanical intestinal obstruction occurs in
    A. Internal hernia
    B. Intussusception
    C. Gastric polyps
    D. Gall stone ileus
    E. Volvulus of the cecum

66. Common causes of peritonitis in Lusaka are
    A. Tuberculosis
    B. Perforated appendix
    C. Typhoid perforation
    D. Biliary leak
    E. Perforated diverticulum of the sigmoid colon

67. Shift of the falx cerebri occurs in
    A. Hydrocephalus
    B. Astrocytoma
    C. Meningitis
    D. Barry aneurysm of the Circle of Willis
    E. Extradural hematoma

68. Gall stones cause
    A. Acute cholecystitis
    B. Acalculous cholecystitis
    C. Obstructive jaundice
    D. Carcinoma of the gall bladder
    E. Torsion of the gall bladder

69. Factors which are known to cause perforation of the ileum include
    A. Trauma
    B. Typhoid
    C. Gall stones
    D. Foreign body
    E. Diverticulum of the ileum

70. Mechanical ileus is a complication of
    A. Hypokalemia
    B. Pelvic peritonitis
    C. Adhesions
    D. Internal hernia
    E. Paraplegia of acute onset

71. Paralytic ileus is a complication of
    A. Retroperitoneal hematoma
    B. Paraplegia of slow onset
    C. Hyperkalemia
    D. Hypercalcemia
    E. Peritonitis

72. Medical conditions which are known to present as acute abdomen include:
    A. Sickle cell disease
    B. Pneumonia
    C. Hypocalcemia
    D. Addison's disease
    E. Hemophilia

73. Early signs and symptoms of compartment syndrome include:
    A. Absent pulse
    B. Paresthesia
    C. Inability to move toes or fingers
    D. A compartment pressure of less than 10mmHg
    E. Decrease in limb pain

74. Pre-operative care before laparotomy for intestinal obstruction
    A. Nasogastric tube is mandatory
    B. Urethral catheterization may be avoided to prevent infection
    C. Antibiotic use should only be instituted after knowing the cause of obstruction
    D. X-match should be done for all patients
    E. Raised serum urea could be treated by furosemide to flash it through the kidneys

75. Factors that increase wound infection postoperatively include
    A. Wound irrigation
    B. Tissue damage
    C. Hematoma formation
    D. Bacteria load <100 000/g tissue
    E. Keeping clean incised wounds dressed for 48 hours

76. Regarding incisions
    A. Keloids form more readily on sternal area than abdomen
    B. Langers lines are not used for placing incisions
    C. Avoid old scars when re-operating
    D. Incised wounds heal with minimal scarring
    E. Traumatised skin edges can be sites of wound breakdown later

77. The signs of uncomplicated volvulus are:
    A. Gross abdominal distention
    B. Ballooning of the rectum
    C. Dyspnea
    D. Severe tenderness on abdominal palpation

78. The usual symptoms of appendicitis are
    A. Central abdominal pain with severe vomiting
    B. Fever over 40^{o}C
    C. Central abdominal pain moving to right iliac fossa
    D. Nausea and anorexia
    E. Painful micturition

79. The symptoms of acute uncomplicated small gut obstruction are:
    A. Continuous severe abdominal pain
    B. Absolute constipation
    C. Abdominal distension
    D. Bilious vomiting
    E. Colicky suprapubic pain

80. The signs of acute uncomplicated small gut obstruction are:
    A. Oliguria
    B. Abdominal distension
    C. Tinkling bowel sounds
    D. Absence of guarding and rebound tenderness
    E. Multiple fluid levels on erect film of abdomen

81. Wound healing by secondary intention occurs:
    A. When the wound edges are not approximated
    B. When there is excessive skin loss
    C. When the wound is infected
    D. In anemia patients
    E. In jaundiced patients

82. Sigmoid volvulus
    A. Is initially treated with a rectal tube
    B. Is common in African female
    C. Rectal bleeding is common
    D. Is an emergency best treated by barium enema
    E. Resection and primary anastomosis is an acceptable form of treatment

83. Paralytic ileus (adynamic obstruction) occurs in
    A. Patients following laparotomy
    B. Peritonitis
    C. Uremia
    D. Hypokalemia
    E. Hypocalcemia

84. Clinical features of acute intestinal obstruction include:
    A. Abdominal pains
    B. Melena
    C. Vomiting
    D. Distention of abdomen
    E. Constipation

85. Acute intussusception in adults occurs due to
    A. Gall stones
    B. Change of diet
    C. Maximum aggregation of Peyer's patches
    D. Intestinal polyps
    E. Head injury

86. The following are predisposing causes of sigmoid volvulus
    A. Chronic constipation
    B. Redundant pelvic mesocolon
    C. Refined dietary intake
    D. Hypokalemia
    E. Uremia

87. The differential diagnosis of acute appendicitis includes:
    A. Tonsillitis
    B. Acute cholecystitis
    C. Carcinoma of the cecum
    D. Ectopic gestation
    E. Infection in urinary tract

88. Burst abdomen is caused by:
    A. Wound infection
    B. Leak of pancreatic enzymes
    C. Anemia
    D. Jaundice
    E. Persistent cough

89. In fracture of the base of the skull the following nerves are commonly injured
    A. Olfactory
    B. Facial
    C. Vestibulocochlear
    D. Accessory
    E. Hypoglossal

90. Keloid characteristics include
    A. Onset is often delayed for months
    B. Is cured by cytotoxics
    C. Regresses spontaneously
    D. Pressure garments are useful following surgery
    E. Radiotherapy is contraindicated

91. The epidermis of the skin consists of the following:
    A. Stratum corneum
    B. Stratum Lucidum
    C. Stratum granulosum
    D. Stratum Malpighi
    E. Stratum basalis

92. Complications of blood transfusion include:
    A. Congestive cardiac failure
    B. Antibody reaction
    C. Infection
    D. Disseminated intravascular coagulation
    E. Fat embolism

93. Inguinal hernia
    A. Indirect hernia is more common than direct hernia
    B. Strangulation is more common with direct hernia than with indirect hernia
    C. Obstruction is more common with indirect inguinal hernia than direct hernia
    D. Direct hernia is likely to protrude through Hesselbach's triangle

94. Differential diagnosis of inguino-scrotal mass includes:
    A. Hernia
    B. Hydrocele
    C. Meningocele
    D. Lipoma

95. The following conditions are associated with abdominal hernias:
    A. Chronic cough
    B. Multiple pregnancies in women
    C. Poor surgical abdominal wound management
    D. Ascites

96. An indirect inguinal hernia
    A. Is the commonest hernia in young adults
    B. Exits through the femoral ring
    C. Has its neck medial to the inferior epigastric vessels
    D. Does not occur in female patients
    E. Is treated with a truss

97. Umbilical hernias:
    A. Are common in Zambian children
    B. All need herniorrhaphy before 2 years of age
    C. Does not give rise to complications
    D. All are Paraumbilical
    E. Adults do not have umbilical hernias

98. Features associated with incompatible blood transfusion include:
    A. Loin pain
    B. Tachycardia
    C. Bradycardia
    D. Cyanosis
    E. Jaundice

99. Congenital diaphragmatic hernia
    A. Are common
    B. Always occur in anterior mediastinum
    C. Is invariably fatal
    D. Diagnosis is often missed initially in small hernias
    E. Are best operated upon at 10 years

100. Concerning hernias
    A. Inguinal hernias are more common on the right
    B. Femoral hernias are more common in females than in males
    C. Inguinal hernias are more common than femoral hernias in females
    D. Paraumbilical hernias are acquired
    E. Umbilical hernias are always congenital

101. Risk factors for developing inguinal hernias include
   A. Male factor
   B. Enlarged prostate
   C. Chronic cough
   D. Ascites
   E. Chronic constipation

102. Differential diagnosis of inguinal hernia in a female are:
   A. A hydrocele of the canal of Nuck
   B. Femoral hernia
   C. An encysted hydrocele of the cord
   D. Lipoma of the cord
   E. Spermatocele

103. An inguinal hernia in a 3-month old baby boy should be treated
   A. Because small gut strangulation is a risk
   B. By a truss
   C. By early operation
   D. Left alone because it resolves spontaneously
   E. By surgery only if the hernia becomes irreducible

104. Quadriplegia
   A. Can be caused by fracture dislocation at C5/C6 cervical spine
   B. Can be caused by fracture dislocation at T5/T6 thoracic spine
   C. Can be caused by Hangman fracture
   D. Hypoventilation is not a feature

105. Second cervical vertebra and odontoid fracture
   A. Basal odontoid fracture extending into body of C2 is type III fracture and is unstable
   B. Basal odontoid fracture is type I and is stable
   C. Apical fracture of odontoid process is type II
   D. Jefferson’s fracture is C3 fracture

106. Patient who presents with goiter, asphyxia and hoarseness of voice:
   A. Is likely to have malignant goiter
   B. Is likely to have retrosternal goiter
   C. Is likely to have unequal pupils
   D. Is likely to have dilated pupils

107. Preoperative treatment of toxic goiter
   A. Iodine can be given in combination with carbimazole or propranolol
   B. Iodine can be given and reduces vascularity of goiter
   C. Nadolol is not a beta-blocker to be used in treating toxic goiter
   D. It is not necessary to reduce toxic goiter to euthyroid status before operations

108. Secondary thyrotoxicosis
   A. Occurs in older patients than those with primary thyrotoxicosis
   B. Eye signs including lid lag and exophthalmos are absent
   C. Early signs of cardiac decompensation like ankle edema and dyspnea are a feature
   D. Orthopnea is not a feature

109. Malignant thyroid
   A. Papillary carcinoma is most uncommon
   B. Anaplastic carcinoma is seen in old patients
   C. Follicular carcinoma is seen in 20% of patients of middle age
   D. Medullary carcinoma is the commonest malignancy

110. Hashimoto’s disease
   A. Is common in post-menopausal women
   B. On inspection one lobe of the thyroid is larger than the other
   C. Symptoms of hypothyroidism are absent
   D. Blood test may show antibodies to the thyroid gland

111. Thyroglossal cyst
   A. It is anterior cervical midline mass
   B. Subhyoid is rare site for thyroglossal cyst
   C. Thyroglossal fistula is a rare post-operative complication
   D. Moves upwards when the tongue is protruded

112. The following vessels supply thyroid gland
   A. Superior thyroid from external carotid artery
   B. Inferior thyroid from thyrocervical trunk of subclavian artery
   C. Thyroidea ima artery from arch of aorta
   D. All above except “B” and “C”

113. Malignant goiter
   A. Medullary carcinoma is the commonest
   B. Papillary carcinoma is the commonest
   C. Bone metastasis may occur
   D. Is slow growing

114. Toxic goiter
   A. Patient has good appetite
   B. Patient gains weight rapidly
   C. Lid lag is not a feature
   D. Proximal myopathy is a feature

115. Goiter
   A. Is more common in males
   B. Compression signs include hoarseness of voice
   C. Moves with protrusion of the tongue
   D. Moves with swallowing

116. Toxic goiter
   A. Lid lag is a feature
   B. Lid retraction is a feature
   C. Exophthalmos is a feature in secondary thyrotoxicosis
   D. Anxiety state is a feature

117. In thyroid function
   A. Coupling stage in thyroid hormone synthesis is catalyzed by deiodinase enzyme
   B. T4 is metabolically more active than T3
   C. Thyroid hormone lack in children affects physical growth and mental development
   D. TSH, T3, T4 and radioisotope thyroid scan are useful indices for thyroid gland activity

118. Examination of non-toxic goiter:
   A. Includes asking the patient to swallow on inspection
   B. Palpation is best done from the front
   C. Palpation is best done behind
   D. Determining lower edges helps to check for retrosternal goiter

119. Patient who presents with goiter causing tracheal obstruction
   A. Stridor is a presenting feature
   B. Dyspnea is a presenting feature
   C. Kocher's test is usually negative
   D. Kocher's test is usually positive

120. Toxic goiter
   A. Exophthalmos is not a feature
   B. Lid lag is a feature
   C. Hot and moist skin and palms is a feature
   D. Rapid pulse rate is not present during sleep

121. Secondary thyrotoxicosis
   A. Patients are younger than those with primary thyrotoxicosis
   B. Eye signs (exophthalmos, lid lag) are absent
   C. Atrial fibrillation is a feature
   D. Dyspnea is not a clinical feature

122. Paraplegia can be caused by
   A. Stab cervical injury
   B. Fracture dislocation of thoracolumbar spine
   C. Spinal tuberculosis
   D. Schistosomiasis

123. Odontoid fracture
   A. Is likely to involve C1
   B. Is likely to involve C2
   C. Type I fracture is stable
   D. Type III fracture is stable

124. Cervical spinal fracture
   A. Fracture at C4/C5 level spares phrenic nerve
   B. Hangman's fracture refers to the fractures at C5/C6
   C. During hanging there is spinal hyperextension and distribution by body weight
   D. Open mouth X-ray is called town view

125. Regarding iodine metabolism
   A. Uptake is solely by thyroid gland
   B. Daily iodine requirement is 100micrograms
   C. Iodine is trapped as iodide
   D. Iodine is organified by carboxylase enzyme

126. Regarding thyroid hormone synthesis
   A. Coupling precedes organification
   B. T3 and T4 are stored in the follicle bound to thyroglobulin molecule
   C. T4 is physiologically more active than T3
   D. Serum T3 is 100-160ng/100ml and T4 60-140m/L

127. Carcinoma of the thyroid
   A. Vascular and capsular invasion is diagnostic of follicular carcinoma
   B. Psammoma bodies and orphan Annie eyed nuclei are diagnostic of medullary carcinoma
   C. Follicular carcinoma metastases is mainly by blood stream
   D. Anaplastic carcinoma has the worst prognosis

128. Medullary carcinoma
   A. Arises from parafollicular "P" cells
   B. Tumor may be sporadic or familial
   C. Pituitary adenoma, parathyroid adenoma and pancreatic adenoma associated with medullary carcinoma constitutes MEN type I syndrome
   D. Tumors in (C) above constitutes MEN type II syndrome

129. Parathyroid adenoma, pheochromocytoma, medullary carcinoma and mucocutaneous vacuromas constitutes:
   A. Sipple syndrome
   B. Marfan syndrome
   C. Hashimoto thyroiditis
   D. None of the above

130. Tumors of the thyroid gland include
   A. Lymphoma
   B. Follicular carcinoma
   C. Anaplastic carcinoma
   D. Warthin's tumor
   E. Giant cell tumor

131. Cervical injuries
   A. Are commonly associated with head injuries
   B. X-ray of the cervical spine down to C6 is adequate
   C. An anteroposterior X-ray is more informative than a lateral view
   D. Injuries at cord level C5 will cause respiratory arrest
   E. During radiological evaluation the shoulders are pulled down caudally

132. Cervical spinal injury
   A. Is common in young children
   B. Odontoid peg is the commonest site of injury
   C. CT scan is required in all cases for diagnosis
   D. The phrenic nerve is involved if the injury is below C5
   E. Is common in the head injured comatose patient

133. Carcinoma of the thyroid
   A. A common type is papillary carcinoma
   B. Medullary carcinoma is associated with pheochromocytoma
   C. Follicular carcinoma secretes calcitonin
   D. Cold nodules on thyroid scan are associated with malignancy
   E. Is seen following irradiation in childhood

134. Patients with spinal cord injury develop
   A. Bronchopneumonia
   B. Pressure sores
   C. Pyelonephritis
   D. Amyloidosis
   E. Priapism

135. Post thyroidectomy complications include
   A. Airway obstruction due to tracheomalacia
   B. Recurrent laryngeal nerve palsy
   C. External laryngeal nerve palsy
   D. Hypercalcemia
   E. Voice change

136. The differential diagnosis of mid-line tumors in the neck includes:
   A. Dermoid cysts
   B. Thyroglossal cyst
   C. Goiter
   D. Bronchial cyst
   E. Pharyngeal pouch

137. Pathological fractures may be associated with
   A. Scurvy
   B. Hyperparathyroidism
   C. Bone cysts
   D. All of the above

138. The most frequent offending agent in osteomyelitis of the spine is:
   A. Streptococcus
   B. Salmonella
   C. Tubercle bacillus
   D. Staphylococcus

139. The following sites are associated with non-union
   A. Colle's fracture
   B. Carpal navicular fracture
   C. Patella fracture
   D. Clavicle fracture

140. A 3-year-old child with a femoral fracture should be immobilized by:
   A. Skeletal traction
   B. POP splint
   C. Intramedullary nailing
   D. Bryant's traction

141. The following factors favor non-unions after fracture:
   A. Muscle interposition
   B. Hematoma at fracture site
   C. Poor blood supply
   D. Infection

142. The following statements are true of Perkins traction
   A. It is a form of fixed skeletal traction
   B. It can be used both in children and adults
   C. It was invented by Prof Sir George Perkins
   D. All above correct except "C"

143. Perkins's exercises:
   A. Are commenced 2 weeks after application of traction kit
   B. Are commenced within a week after application of traction kit
   C. Are commenced after 3 weeks when pain at fracture site has subsided
   D. All above correct except "B"

144. Perkins exercises:
A. Ensure accelerated femoral fracture union
B. Delay femoral fracture union due to mobility while performing exercise
C. Are designed to accelerate femoral fracture union and forestall knee stiffness
D. All above correct except “A”

145. Regarding fracture classification
A. Garden classification refers to fracture neck of femur
B. Salter-Harris classification refers to epiphyseal growth plate fractures
C. Le forte classification refers to tarsal fractures
D. All above correct

146. Ankle joint mortice
A. Mortice formed by medial and lateral malleolus only
B. Mortice formed by medial and lateral malleolus and tibio-fibular syndesmosis
C. Mortice formed by (b) above and articular surface of tibia
D. Only (b) above correct

147. Weber “C” ankle fracture:
A. Is fracture below tibio-fibular syndesmosis
B. Is fracture at level of tibio-fibular syndesmosis
C. Is fracture above tibio-fibular syndesmosis
D. Any fracture at ankle joint

148. Torn anterior cruciate ligament diagnosis, the following test are positive:
A. Anterior drawer test
B. Mc-Murray test
C. Lachman test
D. Pivot shift

149. Torn posterior cruciate ligament diagnosis, the following tests are positive:
A. Lachman test
B. Apley’s grinding test
C. Posterior drawer test
D. All of the above correct

150. In classical degenerative osteoarthritis, the following are noted:
   A. Osteophytes (lipping)
   B. Narrowing joint space
   C. Subchondral bone sclerosis
   D. Subchondral bone cysts

151. Total hip replacement involves
   A. Replacement of femoral head and acetabular cup
   B. Replacement of femoral shaft with artificial implant
   C. Both hips replacement with custom made implants
   D. Replacement of only acetabular cup

152. Anterior shoulder dislocation may be reduced by
   A. Kocher's maneuver
   B. Hippocratic method
   C. Stimson's technique
   D. Socrate's method

153. The following factors favor non-union of a fracture
   A. Soft tissue inter-position
   B. Hematoma at fracture site
   C. Poor blood supply
   D. When there are two bones if the other bone is intact

154. Salter-Harris type II epiphyseal fractures involves
   A. It is a "T" shaped fracture extending into the proximal joint and metaphysis
   B. It extends into metaphysis with a triangular component
   C. It extends into proximal joint with partially crushed epiphysis
   D. None of the above

155. In posterior hip dislocation
   A. Dislocation puts sciatic nerve at risk of injury
   B. Reduction is achieved under general anesthesia with patient on the floor
   C. Skin or skeletal traction should be applied after successful reduction
   D. All above correct except "C"

156. In shoulder dislocation
   A. Posterior dislocation is commoner than anterior dislocation
   B. Axillary nerve is at risk of injury
   C. Kocher's manoeuver may be employed to reduce dislocation
   D. All above are correct except "C"

157. In elbow dislocation
   A. Anterior dislocation describes humerus lying anterior to radius.
   B. Posterior dislocation describes humerus lying posterior to radius
   C. The forearm bones provide key to description of the type of dislocation
   D. None of above correct

158. In lower limb deformities
   A. Genu valgus refers to bow legs
   B. Genu varus refers to knock knees
   C. Foot drop refers to injury of common fibular nerve
   D. All above are correct

159. Secondary metastases to the spine can originate from cancer of
   A. Thyroid
   B. Breast
   C. Lung
   D. Prostate

160. Collar and Cuff sling
   A. Is an example of external splint
   B. Is suitable for displaced humeral fracture
   C. Can be used to immobilize femoral fractures in children
   D. The flexion angle at the elbow is less than 90 degrees

161. Intramedullary nail is
   A. External fixation
   B. Internal fixation
   C. Suitable for fracture of the femoral shaft
   D. Also known as Kuntscher nail

162. The following tumors readily metastasize to bone
   A. Rectal adenocarcinoma
   B. Basal cell carcinoma of skin
   C. Follicular carcinoma of the thyroid
   D. Prostatic adenocarcinoma
   E. Renal adenocarcinoma

163. Dislocations
   A. Do not require urgent management
   B. Post-reduction immobilization is required for 3 months
   C. The medial nerve is damaged in shoulder dislocations
   D. The ulnar nerve is damaged in lunate dislocations
   E. All open dislocations require operative reduction

164. Osteoarthritis of the knee joint is a complication of
   A. Hemophilia
   B. A malunited femoral fracture
   C. Total menisectomy
   D. Valgus deformity of the knee
   E. Gross obesity

165. Supracondylar fractures of the humerus
   A. Always check the radial artery pulse
   B. Always check nail bed capillary filling in absence of the radial pulse
   C. Extension improves limb circulation
   D. Nerve damage is uncommon
   E. Non-union is uncommon

166. The following fractures commonly lead to ischemic necrosis of a part of the bone
   A. Femoral neck
   B. Humeral neck
   C. Scaphoid
   D. Talus neck
   E. Femoral condyle

167. The following fractures lead to nerve damage in some patients:
   A. Femoral neck
   B. Neck of fibula
   C. Medial epicondyle of humerus
   D. Radial shaft
   E. Humeral shaft

168. Fracture of the pelvis:
   A. A stable fracture requires 3 months bed rest
   B. A displaced sacroiliac joint requires skeletal traction
   C. Blood transfusion is essential in extensive pelvic fractures
   D. Isolated undisplaced iliac bone fractures do not require immobilization
   E. Fractures of the pubic rami are relatively common

169. The following are flexors of the wrist joint:
   A. Brachioradialis
   B. Flexor carpi ulnaris
   C. Pronator teres
   D. Flexor digitorum profundus
   E. Flexor pollicis brevis

170. Osteosarcoma is treated by
   A. Radiotherapy
   B. Chemotherapy and surgery
   C. Immunotherapy
   D. Antibiotics
   E. Vitamin D

171. The following laboratory tests are useful in evaluating diseases related to bones
   A. Serum parathyroid hormone
   B. C-reactive protein
   C. Urinalysis for specific proteins
   D. Fasting blood glucose
   E. Serum uric acid levels

172. Osteoporosis
   A. Is common in premenopausal women
   B. Is treated with estrogens
   C. Causes pathological fractures
   D. Erythrocyte sedimentation rate is elevated
   E. Once established cannot be reversed

173. Primary osteoarthritis
   A. Starts as a disorder of articular cartilage
   B. Results in periarticular osteoporosis
   C. Is associated with peripheral vascular disease
   D. Occurs more commonly in females
   E. Is common in Zambia

174. Dupuytren's contractures:
   A. Causes extension deformity of the wrist
   B. Is familial
   C. The foot can be involved occasionally
   D. Is treated by steroids
   E. Trauma is an etiologic factor

175. Carpal Tunnel syndrome
   A. Occurs in middle aged men only
   B. Symptoms are related to thumb only
   C. Does not cause muscle wasting
   D. Surgery is indicated
   E. Symptoms are worse at night

176. Septic arthritis is best managed by
   A. Aspiration of the joint
   B. Antibiotics
   C. Rest and elevation
   D. Gypsona immobilization
   E. Arthrotomy, antibiotics and rest for the joint

177. Acute osteomyelitis
   A. Brodie's abscess is a variety of acute osteomyelitis
   B. A common causative organism is Staphylococcus aureus
   C. Bone drilling must be done early to prevent further damage
   D. X-rays do not show changes in the first week
   E. Is common in Zambian children

178. Tuberculosis of the spine
   A. Is common in Zambia
   B. Usually affects the vertebral bodies
   C. Can be adequately treated by anti-tuberculous drugs alone
   D. Never produces sequestra
   E. Is not diagnosed unless a paraplegia is present

179. A 3-year-old child develops pain in the hip joint, is pyrexial and has a high erythrocyte sedimentation rate:
   A. Treat by traction alone
   B. Treat by antibiotics alone
   C. Blood culture are to be done
   D. Explore the hip joint surgically
   E. Take a careful history from the mother and do a full physical examination at admission

180. Poliomyelitis
   A. Anterior horn cells are affected
   B. Flaccid paralysis develops
   C. Joint contracture may follow muscle imbalance
   D. Tendon division may improve function
   E. Spread is by nasopharyngeal droplets

181. The following statements are true or false
   A. The radial nerve is injured in humeral fractures
   B. The ulnar nerve is injured in dislocated shoulder
   C. A Colles fractures commonly injures the median nerve
   D. Wrist drop occurs with median nerve palsy
   E. Circumflex nerve injury causes inability to adduct the shoulder

182. Acute osteomyelitis of the tibia
   A. Not to be drained unless bone changes are present on X-ray
   B. Staphylococcus aureus is the causative organism in all cases
   C. Requires immediate drainage
   D. X-Ray changes are visible within a few days of onset of infection
   E. Antibiotics are not indicated

183. Ulnar nerve palsy
   A. Occurs in fracture of the lateral epicondyle of the humerus
   B. Results in wasting of thenar muscles
   C. The little finger cannot be abducted against resistance
   D. The ulnar side of the hand becomes dry (no sweating)
   E. Dermatome involved is C5

184. Osteoporosis
   A. Is a hereditary disease
   B. Is same as Osteomalacia
   C. Is curable
   D. Is common in young premenopausal women
   E. Responds to vitamin A

185. The following nerves are injured in a dislocation shoulder
   A. Medial
   B. Circumflex
   C. Axillary
   D. Suprascapular
   E. The nerve which supplies the deltoid muscle

186. Concerning the menisci of the knee:
   A. Injuries are common in elderly people
   B. A tear is associated with osteoarthritis of the knee
   C. Twists at the knee is the usual cause of meniscal tears
   D. Injuries to the lateral one is commoner
   E. One function is to improve lubrication

187. A 39-year-old male with a suspected intervertebral disc prolapse suddenly develops urinary retention. The following measures should be taken
A. Urgent radiculogram
B. Catheterisation
C. Urgent exploration of spine
D. Bed rest and skeletal traction
E. Send to the next urology clinic

188. A 4-year-old child develops pain in the hip joint with restriction of all movements, pyrexia, high erythrocyte sedimentation rate and a high white blood cell count
A. Treat with skin traction alone
B. Give aspirin and bed rest
C. Take blood cultures
D. Explore the hip surgically
E. Is commonly associated with trauma

189. Osteoarthritis has the following features:
A. Occurs in the hand bones
B. Is age related commonly
C. Often follows joint injury
D. Is commoner in males
E. Results in periarticular osteoporosis

190. Sickle cell disease
A. Is hereditary
B. Is due to an abnormal hemoglobin
C. Femoral epiphyseal infarction is a complication
D. Steroids are indicated in all cases
E. Is associated with osteomyelitis

191. Following a nerve injury
A. Neuropraxia has the best prognosis for recovery
B. Neurotmesis has the worst prognosis for recovery
C. Axonotemesis describes complete transection of the nerve
D. Axons regenerate at the rate of 1 mm per day
E. Axons regenerate at the rate of 1cm per day

192. The following fractures occur at the wrist
   A. Colles
   B. Barton's
   C. Smiths
   D. Pott's
   E. De Quervain's

193. The following fractures usually require open reduction and internal fixation
   A. Oblique shaft fractures of the humerus
   B. Colles fracture
   C. Greenstick forearm fractures in children
   D. A displaced fracture of the lateral condyle of the elbow in a 10-year-old
   E. A subcapital femoral fracture in an elderly female

194. A fracture of the carpal scaphoid
   A. Is frequently caused by a fall on the outstretched hand
   B. Usually occurs through the waist of the scaphoid
   C. Avascular necrosis of the distal fragment is a possibility
   D. Is commoner in the younger patient than the old
   E. Non-union is possible

195. In spinal Tuberculosis
   A. Infection starts in bone adjacent to a disc
   B. New bone formation is minimal in active infection
   C. Radiographs usually show destruction of the body of a vertebra without involvement of the disc
   D. Psoas abscess is a complication
   E. Paraplegia is a complication

196. Poliomyelitis
   A. Is caused by damage to anterior horn cells
   B. Leg length inequality is a possibility
   C. Short rigid scoliosis is usually associated with it
   D. Foot neuropathic ulcers are common
   E. Osteoarthritis is a common feature of the affected limb joints

197. The following muscles in the hand are supplied by the median nerve
A. Abductor pollicis brevis
B. The first dorsal interosseous muscle
C. Adductor pollicis
D. Abductor digiti minimi
E. Opponens pollicis

198. In chronic osteomyelitis of the femur
A. New bone outside the cortex is the involucrum
B. Sequestrum is more radiopaque than normal bone
C. A loose dead bone is called sequela
D. Cloacae drain pus through the cortex
E. Long standing sinuses heal with antibiotics

199. The early clinical signs and symptoms of ischemia in the forearm and hand following closed reduction of a supracondylar fracture are
A. Paralysis of median nerve
B. Gangrene of tips of fingers
C. Fixed contracture of the flexor muscles
D. Pain, especially on extension of the fingers
E. Absent radial pulse and poor capillary return

200. The following are complications of a Colles fracture at the wrist
A. Rupute of the extensor pollicis longus
B. Frequent non-union
C. Median nerve compression
D. Malunion
E. Inferior radioulnar subluxation

201. Classification of fractures
A. Radiologically as open or closed fractures
B. Clinically as compound fracture or closed fracture
C. Comminuted fracture when more than 2 fragments are present
D. Pathological fractures when fracture is due to tumor deposit
E. Supracondylar fracture refers to anatomical classification

202. Concerning fractures of the neck of femur
A. They are more common in elderly men
B. They are usually caused by high energy trauma in the elderly
C. Avascular necrosis of the head of the femur is common complication
D. If treated conservatively they have a higher incidence on non-union
E. They are best managed by skeletal traction

203. Femoral shaft fractures
A. Are associated with an average blood loss of 1000-1500ml
B. In the young patient are usually associated with other life-threatening injuries
C. Are usually adequately treated with traction
D. Are commonly complicated by compartment syndrome
E. Are commonly associated with shortening of the affected limb

204. Regarding Perkin's traction
A. The usual weight applied is 1/10 of the patient's body weight
B. Leg lengths are measured once weekly for the first 2 weeks after application
C. Exercises are best commenced within the first 4 days of applying traction
D. The foot end of the bed is normally elevated by 4cm for every Kg of weight
E. A Denham pin may be used for applying the traction

205. Concerning open fractures
A. They are more common in the tibia than in any other long bone
B. They cannot be complicated by compartment syndrome
C. They are commonly associated with delayed union and non-union in the tibia
D. X-rays are not necessary
E. Are best treated with an external fixator

206. The following displacements are characteristic of Colles' fracture
A. Volar (anterior) displacement of the distal fragment
B. Apex volar angulation
C. Ulnar deviation of the distal fragment
D. Impaction
E. 'Dinner fork' deformity

207. Supracondylar fractures of the humerus in children
   A. Are usually of the flexion type
   B. Commonly occur after a fall on an outstretched hand
   C. Are partly caused by the olecranon being forced into the olecranon fossa
   D. Can be complicated by myositis ossificans
   E. If presenting with extensive swelling should be manipulated and immobilized in a collar and cuff

208. Concerning fracture healing
   A. Hematoma formation at the fracture site does not influence the rate of fracture healing
   B. It is affected by the nutritional status
   C. Upper limb fractures in children take about 3 weeks to heal
   D. It is influenced by the type of bone involved
   E. It is affected by the hemoglobin level of the patient

209. Concerning acute hematogenous osteomyelitis
   A. It is commonly caused by Salmonella Spp
   B. Organism normally settle in the metaphysis of long bones
   C. In infants it may lead to septic arthritis
   D. Radiological signs are present within 24 hours of the infection
   E. It is best managed conservatively

210. Differential diagnoses of acute osteomyelitis include:
   A. Fracture
   B. Sickle cell crisis
   C. Rheumatic fever
   D. Cellulitis
   E. Septic arthritis

211. The criteria for diagnosis of septic arthritis in patients with negative cultures (according to Morrey and Associates) include:
   A. Pain in suspected joint worsened by movement
   B. Systemic symptoms
   C. Poor response to antibiotic therapy
   D. Swelling of suspected joint
   E. Fever of more than 38.3 degrees Celsius

212. Fracture of clavicle
A. Is usually compound
B. Is best treated by open internal fixation
C. Is usually associated with blood vessel injury
D. Is treated by POP immobilization
E. Can be treated by long arm sling alone

213. In Tuberculosis of bone
A. The local reaction is characterized by extensive new bone formation
B. The metaphysis of a bone is the commonest site of involvement
C. The infection is usually secondary to primary focus elsewhere in the body
D. Extension of the bone abscess into a joint is common
E. Associated abscesses show few signs of inflammation

214. Features occurring in children differ from those in adults in the following respects
A. Fractures unite readily
B. Malunion can be corrected by growth
C. Joint stiffness is rare after immobilization
D. Fractures involve the epiphyseal plate on occasion
E. Fractures usually occur with minimal trauma

215. In fracture of the shaft of the adult femur
A. The estimated blood loss is at least 1 liter
B. Skeletal traction and Perkin's exercises are effective treatment
C. Healing is faster in Zambians than in Europeans
D. Internal fixation allows earlier mobilization
E. Internal fixation is the treatment of choice in Zambia

216. Carpal tunnel syndrome
A. Is most frequent in middle aged men
B. Symptoms are related to all fingers and the thumb
C. Does not cause muscle wasting
D. Surgical treatment is usually curative
E. The symptoms are worse at night

217. Dislocation of the hip
   A. Common variety is a posterior dislocation
   B. Delay in reduction does not increase the risk of late complications
   C. Is overlooked if there is an associated fracture of the shaft of femur
   D. Leads to dorsiflexion of the foot and great toes
   E. Sciatic nerve injury occurs in 50% of patients

218. The following fractures are rarely adequately treated by closed manipulation:
   A. Bennet's fracture
   B. Colles fracture
   C. Phalages of digits
   D. Monteggia
   E. Tibial condyle

219. Internal hemorrhoids are arranged in the following manner with the patient in lithotomy:
   A. 3, 7 and 11 O'clock
   B. 3, 7 and 9 O'clock
   C. 7, 9 and 11 O'clock
   D. 3, 9 and 11 O'clock

220. GoodSall's rule is related to
   A. Anal fissure
   B. Anal fistula
   C. Hemorrhoids
   D. Anal abscesses

221. Common causes for rectal bleeding
   A. Internal hemorrhoids
   B. Anal fissure
   C. Carcinoma rectum
   D. All correct except (B)

222. Concerning piles
   A. First degree piles bleed but do not prolapse
   B. Second degree piles prolapse with defecation and reduce spontaneously
   C. Third degree piles are thrombosed piles whether internal or external
   D. None above are correct

223. Treatment for first degree piles
   A. Hemorrhoidectomy
   B. High fiber diet
   C. Suppositories
   D. All above correct

224. Hemorrhoids
   A. Always cause pain and bleeding
   B. Sometimes cause pain and bleeding
   C. Are always treated by operation
   D. Can be due to increased intra-abdominal pressure
   E. Are noted in pregnancy

225. The adult anus
   A. Is 1cm long
   B. Is covered by serosal lining
   C. Blood supply is from 3 rectal arteries
   D. Has 2 main parts meeting at perineal line
   E. Inferior half lymphatics drainage is to the superficial inguinal nodes

226. Thrombosed hemorrhoids
   A. Are treated by hemorrhoidectomy
   B. Can be commonly seen at three sites: 4, 7 and 11 O'clock
   C. Are due to arterial anastomosis
   D. Usually have a sentinel pile
   E. Common in diabetic patients

227. Digital rectal examination is diagnostic in
   A. First degree hemorrhoids
   B. Low carcinoma of the rectum
   C. Fissure in ano
   D. Acute cystitis
   E. Hirschprung's disease

228. Axillary tail of the breast lies in
   A. Upper outer quadrant
   B. Upper inner quadrant
   C. Lower outer quadrant
   D. Lower inner quadrant

229. The breast tissue is located
   A. Within subcutaneous layer
   B. Within superficial fascia between upper and lower layers
   C. Rests directly on pectoral fascia
   D. Only "A" above is correct

230. Fibroadenoma of breast
   A. Commonest benign tumor
   B. It may be peri or intra canalicular
   C. It lacks a definite capsule
   D. Recurrence after excision is rare

231. Blood stained discharged from nipple may represent
   A. Duct papilloma
   B. Pagets disease of nipple
   C. Galactocele
   D. Only "B" and "C" correct

232. The following breast tumors may undergo malignant transformation
   A. Phylloides tumor
   B. Multiple duct papillomatosis
   C. Intracanalicular fibroadenoma
   D. All above

233. Change of breast may also be named as
   A. ANDI
   B. Benign mammary dysplasia
   C. Fibroadenosis
   D. Chronic mastitis

234. Reference to ANDI of breast, histological features include:
   A. Adenosis
   B. Epitheliosis
   C. Duct ectasia
   D. Fibrosis

235. In Lactational mastitis
   A. Cracked nipple permits entry of bacteria
   B. Staphylococcus aureus is the main pathogen
   C. There is good response to broad spectrum antibiotics
   D. May be complicated by breast abscess

236. The following cannot affect breast tissue
   A. TB
   B. Syphilis
   C. Fungal infection
   D. All above

237. Triple assessment of breast pathology
   A. Is only relevant to malignant conditions
   B. Involves taking history and performing clinical examinations only
   C. Involves as well U/S scan for females above 35 years old and mammogram for females below 35 years of age
   D. Fine needle aspiration cytology (FNAC) is preferred to incisional or excisional biopsy snice it is specific and easy to interpret

238. Pagets disease of the nipple
   A. Usually presents as bilateral nipple eczema
   B. Unilateral nipple eczema
   C. Related to underlying duct carcinoma
   D. All above true except (B)

239. Fibroadenoma of breast
   A. It may present as mobile, painless mass
   B. Has ill-defined edges
   C. It may be intra or peri-canalicular
   D. Recurrence common after excision

240. Fibrocystic breast disease presents as:
   A. Fibroadenosis
   B. Cystic formation
   C. Epitheliosis
   D. Fibrosis and lymphocytosis

241. The following drugs are used in breast cancer
   A. Cyclophosphamide
   B. 5-fluoro-uracil
   C. Methotrexate
   D. Adriamycin

242. The following features present advanved breast cancer
   A. Peau d'orange
   B. Nipple retraction/ulceration/bleeding
   C. Fixity to underlying pectoral musclce
   D. Hepatomegaly

243. Phyllodes tumor on histology
   A. Might be benign
   B. Might be borderline
   C. Might be frankly malignant
   D. Cellular stroma is characteristic

244. Breast tissue lies
   A. Within retromandibular space
   B. Deep to pectoral fascia
   C. Within superficial fascia
   D. May extend to the axilla

245. Breast cancer
   A. Carcinoma in-situ tumor has not yet breached basement membrane
   B. Lobular carcinoma tends to be multicentric in origin more than ductal carcinoma
   C. Ductal carcinoma tends to be multicentric in origin more than lobular carcinoma
   D. None of the above

246. Breast cancer markers/receptors include:
   A. ER/PR
   B. HER-2/NEU
   C. Tamoxifen is useful in ER positive receptors
   D. Trastuzumab (Herceptin) is useful in advanced breast cancer that are HER-2/NEU receptor negative

247. Supraradical mastectomy involves
   A. Mastectomy, axillary node clearance and excision of pectoral muscles
   B. The clavicle and sternum were spared
   C. Procedure abandoned due to post op arm edema and weak upper limb
   D. Operation is now obsolete

248. Radical mastectomy
   A. Improves on supraradical mastectomy
   B. It spared clavicle and sternum
   C. Pectoralis major was spared
   D. Also referred to as complete Halsted

249. Patey's mastectomy
   A. Improved on radical mastectomy
   B. It spares pectoral major muscles
   C. Axillary and pectoral nodes clearance done
   D. Power limb was an advantage

250. Lymph node involvement in breast cancer includes the following
   A. Axillary lymph nodes
   B. Supraclavicular lymph nodes
   C. Cervical lymph nodes
   D. Inguinal lymph nodes

251. Triple assessment is used in making a diagnosis of breast disease and consists of the following:
   A. Clinical examination, imaging, and fine needle aspiration cytology
   B. Triple imaging-ultrasound, chest X-ray, mammography
   C. Triple diagnosis- mammography, radioisotope studies and CAT-scan
   D. Nipple cytology, thermography and MRI

252. A breast lump in an adolescent can be one of the following
   A. Fibroadenoma
   B. Giant fibroadenoma
   C. Galactocele
   D. Dysgerminoma
   E. Hurtle cell tumor

253. An enlarged prostate
   A. Can be determined by digital rectal examination
   B. Can be identified by ultrasound evaluation
   C. Doses not cause acute urinary retention
   D. Prostatic specific antigen is an indicator of malignant disease in the prostate
   E. Acid phosphatase is a specific test for prostatic cancer

254. Prostatectomy is performed using one of the following approaches
   A. Suprapubic transvesical
   B. Retropubic (Millin's)
   C. Transurethral
   D. Perineal
   E. Pfannenstial incision

255. Complication of prostatectomy include
   A. Excessive bleeding
   B. Inflammation of the vas deferens
   C. Bladder neck stricture
   D. Gastric atony
   E. Subphrenic abscess

256. Cancer of the prostate
   A. Is common in the elderly
   B. Is often asymptomatic
   C. Treatment of choice is prostatectomy
   D. Prostate specific antigen is a reliable guide to prognosis
   E. Metastases are always osteolytic

257. Fibroadenoma of the breast
   A. Occurs in early adult life
   B. May be multiple
   C. Adheres to the pectoralis major
   D. Is painful
   E. Disappears spontaneously

258. In hypernatremia
   A. Fever and edema are absent
   B. Restlessness and raised BP are present
   C. There is increased urine output
   D. Only C is correct

259. In hyperkalemia patient will present with:
   A. Increased reflexes and ECG changes
   B. ECG shows inverted “T” waves
   C. ECG shows peaked “T” waves
   D. Oliguria, muscle weakness and respiratory distress are a feature

260. Treatment of hyperkalemia include:
   A. Kayexalate
   B. IV insulin and glucose
   C. Lasix and stop potassium containing fluid
   D. Dialysis if severe

261. In respiratory alkalosis
   A. PCO_2 rises
   B. PCO_2 falls
   C. PCO_2 remains the same
   D. None above are correct

262. In metabolic acidosis
   A. CO_2 combining power rises
   B. CO_2 combining power falls
   C. CO_2 combining power remains the same
   D. Only C is correct

263. Under normal conditions the colloid osmotic pressure of blood is about
   A. 75mmHg
   B. 50mmHg
   C. 25mmHg
   D. 10mmHg

264. Signs and symptoms of hyperkalemia include
   A. Peaked “T” waves on ECG
   B. Increased cardiac contractility
   C. Muscle weakness
   D. Respiratory distress

265. Treatment of hypernatremia include:
A. Stop IV normal saline
B. Glucose and insulin intravenously
C. Lasix
D. Dialysis if severe

266. Administration of potassium to correct hypokalemia
A. Give potassium as intravenous bolus
B. Not more than 20mEq with 40mEq as maximum
C. Ensure good urine output
D. Cardiac monitor not essential

267. The following intravenous solutions are commonly used to treat hemorrhagic shock in the University Teaching Hospital
A. 0.9% saline
B. 5% dextrose
C. Ringer's lactate
D. Hypertonic saline
E. Darrow's solution

268. Septic shock
A. Is particularly associated with Celestin tube insertion
B. Is often due to gram negative organisms
C. Is particularly seen in patients with indwelling intravenous catheters
D. Surgery for acute cholecystitis is a cause
E. Does not occur in gram positive septicemia

269. The following intravenous fluids contain potassium
A. Darrow's
B. Ringer's
C. Blood
D. Dextrose 5%
E. Dextran solution

270. Patients needing supplementary feeds include those with
   A. Recent weight gain
   B. Serum albumin >30g/dl
   C. Serum albumin <30g/dl
   D. Negative nitrogen balance
   E. Positive nitrogen balance

271. Parenteral feeds
   A. Best feeding method
   B. Has least side effects
   C. Requires feeding nasogastric tube
   D. Very expensive
   E. May be the only alternative in conditions like acute pancreatitis

272. Body fluids
   A. A 70kg man has 42L as total body water
   B. Interstitial fluids in a 70kg man is 11 Liters
   C. 3 L of plasma in a 60kg man
   D. 15L in a 60kg man is extracellular fluid
   E. Potassium is an extracellular ion

273. Pancreatitis
   A. Can be the cause of chemical peritonitis
   B. Absence of bowel sounds is a feature
   C. Serum amylase is not raised
   D. Laparotomy is indicated in all cases

274. Complications of acute pancreatitis are
   A. Pseudocyst formation
   B. Disseminated intravascular coagulation
   C. Left basal effusion (pleural)
   D. Renal failure
   E. Ascites

275. Gall stones cause
   A. Acute cholecystitis
   B. Acalculous cholecystitis
   C. Obstructive jaundice
   D. Carcinoma of the gall bladder
   E. Torsion of the gall bladder

276. Acute cholecystitis
   A. Does not occur in sicklers
   B. Is usually treated by cholecystostomy
   C. Is usually associated with urinary bladder stones
   D. Can cause pain in the right shoulder
   E. Causes pain and tenderness in the left hypochondrium

277. Acute pancreatitis
   A. Often results in hypoxemia
   B. Usually gives rise to Cullen's sign
   C. Causes hypercalcemia
   D. Can be caused by mumps
   E. Occurs after biliary surgery sometimes

278. Esophagus
   A. Below the pulmonary roots the vagus nerves are in contact with esophagus
   B. The left trunk of the vagus nerve is on the anterior side of the esophagus
   C. The right trunk of the vagus nerve is on the posterior side of the esophagus
   D. The longest part of esophagus is intra-abdominal

279. Cancer of the esophagus
   A. Dysphagia is initially for solids
   B. Dysphagia is initially for liquids
   C. Barium swallow X-ray shows a smooth constriction giving a rat-tail appearance
   D. Barium swallow X-ray shows irregular and sharp edge constriction giving shouldering appearance

280. Signs of advanced cancer of the esophagus include:
A. Hoarseness of voice as a sign of involvement of recurrent laryngeal nerve
B. Weight loss of more than 20%
C. Absence of palpable supraclavicular lymph nodes
D. Presence of palpable lymph nodes

281. Esophageal stricture
A. Can be caused by achalasia of the cardia
B. Inflammation of esophagus does not cause strictures
C. Can be caused by cancer of esophagus
D. Can be caused by ingestion of corrosive chemicals

282. Cancer esophagus presents with
A. Weight gain
B. Dysphagia of solids
C. Odynophagia
D. Regurgitation is not a feature

283. Carcinoma of the esophagus is commonly treated by
A. Surgery
B. Radiotherapy
C. Immunotherapy
D. Chemotherapy
E. Gastrostomy

284. Carcinoma of the esophagus
A. Lower third is a common site
B. Is adenocarcicnoma in 90% of cases
C. Is radiosensitive
D. Metastases the scalene node
E. Presents early in Zambia

285. Hepatocellular carcinoma
   A. Is the commonest tumor of men in Zambia
   B. Is not associated with post-necrotic cirrhosis
   C. Is related to ingestion of aflatoxins
   D. Presents with intermittent abdominal pain
   E. Rarely causes early weight loss

286. Incisional hernias are related to
   A. Wound infections
   B. Anemia and malnutrition
   C. Obesity
   D. The use of absorbable suture materials

287. The differential diagnosis of an inguinal hernia in a female include:
   A. Vaginal hydrocele
   B. Encysted hydrocele of the cord
   C. A femoral hernia
   D. None of the above

288. A strangulated hernia
   A. Is tense
   B. Is tender
   C. Has a transmitted cough impulse
   D. Requires elective surgery

289. A partial thickness burn
   A. May heal without grafting
   B. May deteriorate into full thickness skin loss
   C. Rarely causes severe physiological derangement of the patient
   D. Heals within 7 days in the absence of infection

290. Burns
   A. History taking should always include- Time of burn, cause, smoke inhalation, trauma
   B. On examination the severity is determined by burn depth, burn size, areas of circumferential full thickness
   C. Full thickness burns will produce very painful charred skin
   D. Escharotomy may help in reducing respiratory embarrassment

291. In burns
   A. A man with 30% superficial burns does not require fluid therapy
   B. A female with 20% superficial burns does not require ATT on admission
   C. Parkland's formula considers fluids therapy in the first 36 hours
   D. Browder and Lund charts are used to estimate percentage surface area burnt

292. The skin
   A. Is a multilayered organ
   B. Good approximation of layers is not vital
   C. Crush injury heals better than an incision
   D. Langer's lines run perpendicular to muscle axis
   E. Langer lines run mainly longitudinal to muscle axis

293. Upper gastrointestinal hemorrhage can be caused by:
   A. Mallory-Weis tear
   B. Sigmoid volvulus
   C. Periportal fibrosis due to Bilharzia
   D. Perforated anterior duodenal ulcer

294. Zollinger-Ellison syndrome
   A. Multiple ulcerations are a feature
   B. Increased vagal activity is the main cause
   C. Fresh red blood rectal bleeding is a common feature
   D. Gastrin producing endocrine tumor is a common feature

295. Pyloric stenosis in adult patient:
A. Annulus pancreas can be the cause
B. Chronic duodenal ulcer can be the cause
C. Meckel's diverticulum can be the cause
D. Cancer of pancreas can be the cause

296. In a patient with peptic ulcer disease
A. Histamine production is increased by stimuli from both vagus nerves and gastrin
B. The process of exporting hydrogen ions from parietal cells via protein pump is not a feature
C. Histamine acts on parietal cells via H2-receptors
D. Melena stools is not a feature

297. Investigating a patient who presents with hematemesis includes:
A. Esophagoscopy
B. Gastroduodenoscopy
C. Lower midline laparotomy is advisable
D. Right subcostal mini-laparatomy is advisable

298. Brain abscess in the frontal lobe
A. Is most likely a complication of chronic otitis media
B. Is not likely a complication of paranasal sinusitis
C. Is most likely a complication of infected occipital laceration
D. Is most likely a complication of infected wound on the temporal area of the head

299. Large supratentotrial space occupying lesions is likely to present with
A. Full consciousness
B. Unequal pupils
C. Contralateral hemiplegia
D. Paraplegia

300. Common causes of congenital hydrocephalus include:
   A. Brain abscess
   B. Dandy-walker cyst
   C. Arterial venous malformation
   D. Anold-Chiari malformation

301. Submandibular gland tumors
   A. 50% are malignant
   B. Facial nerve weakness is a feature
   C. Ulceration of overlying skin is not a feature
   D. Cervical lymph nodes enlargement is a feature

302. Parotid gland tumors
   A. Most common are benign pleomorphic adenomas
   B. 10% are malignant
   C. Rapid growth with infiltrating of overlying skin is a sign of high-grade malignancy
   D. Enlarged palpable cervical lymph nodes are not a feature

303. Bilateral paralysis of genioglossus leads to
   A. Atrophy of the tongue
   B. Tongue falls back
   C. Obstruction of respiratory passage
   D. All above true except “B”

304. Structures embedded in the parotid gland include
   A. Facial nerve
   B. Retromandibular vein
   C. External carotid artery
   D. Parotid lymph nodes

305. The following are examples of non-absorbable suture materials
   A. Silk
   B. Vicryl
   C. Nylon
   D. Stainless steel

306. The following suture are
   A. Silk is natural material
   B. Nylon is synthetic material
   C. Vicryl is natural material
   D. Stainless steel is synthetic material

307. Knot security after wound suturing
   A. Best with vicryl
   B. Good with nylon
   C. Worst with silk
   D. All above correct

308. Clostridium Welchii is
   A. Causative agent of wet gangrene
   B. Causative agent of gas gangrene
   C. Aerobe, sporulating, gram negative bacillus
   D. Proliferates in dirty wounds

309. In acute limb ischemia
   A. Thrombo-embolism may be a causative factor
   B. In-situ blockage of artery by thrombus is factor
   C. Buerger's test is positive
   D. Ankle brachial pressure index is useful indicator of severity of ischemia

310. Parotid benign tumors
   A. Make up 80 to 90% of parotid tumors
   B. Are pleomorphic adenomas
   C. Are soft on palpation
   D. Mostly arise in the superficial lobe

311. HIV-associated sialadenitis of parotid gland
   A. Chronic parotitis in children is pathognomonic of HIV infection
   B. Facial disfigurement is a feature
   C. CT scan shows characteristics swiss cheese appearance of multiple cystic lesions
   D. Cosmetic surgery is not advisable

312. Causes of leucopenia
   A. Lymphoma
   B. Sever infections
   C. Disease of the spleen
   D. Autoimmune disorders

313. Causes of leukocytosis
   A. Inflammatory conditions like arthritis
   B. Stress
   C. Allergic conditions like asthma
   D. Pregnancy

314. A rise in monocytes may indicate the following:
   A. Chronic inflammatory disease
   B. Parasitic infestation
   C. Tuberculosis
   D. Oral infection

315. Post-op fever after abdominal surgery
   A. Is caused by release of pyrogens
   B. Intra-abdominal collections
   C. Blood and platelet infusions
   D. Pulmonary complications like pneumonia or empyema

316. Unilateral ureteric obstruction may be caused by
   A. Ureteropelvic stenosis
   B. Ureteric stone
   C. Posterior urethral valves
   D. Neurogenic bladder

317. Unilateral renal artery obstruction may be associated with
   A. Increased renin release by the obstructed kidney
   B. Angiotensin release and increased aldosterone production by adrenal cortex
   C. Increased sodium and water retention
   D. Hypotension

318. Chronic bilharzia can cause
   A. Bilateral ureteric obstruction
   B. Transitional cell carcinoma
   C. Bilateral hydronephrosis
   D. Squamous cell carcinoma of the bladder

319. Management of obstructive uropathy may include
   A. Catheterization
   B. Bilateral orchidectomy
   C. Endoscopic ureterotomy
   D. Urethroplasty

320. Parotid inflammatory swelling
   A. Can be caused by mumps
   B. Is rare in children
   C. Can be HIV associated
   D. Does not cause facial disfigurement

321. Superficial parotidectomy
   A. Is indicated for benign and low grade malignant tumors
   B. Incision is lazy-“S”
   C. Facial nerve injury is rare
   D. Skin flap is developed to reach anterior border of the gland

322. Parotidectomy complications
   A. Frey’s syndrome
   B. Facial nerve palsy
   C. Hemorrhage
   D. Raised T3 and T4

323. Lung cancer
   A. Small cell lung cancer makes up 20%
   B. Metastasis by lymphatics is rare
   C. Metastasis by blood borne spread is common
   D. Surgery greatly improves survival

324. When considering fluid spacing
   A. First spacing is normal exchanging between intravascular and extravascular space
   B. Second spacing is accumulation fluid into tissues as edema
   C. Third spacing is accumulation of fluid in body cavities e.g. Ascites, pleural effusion
   D. All above correct except “C”

325. If vagus nerves are severed the respiratory rate
   A. Is increased
   B. Is decreased
   C. Remains constant
   D. Ceases

326. Destruction of pneumotaxic center located in the pons causes
   A. Apneustic respiration
   B. Forceful expiration
   C. Accelerated respiration
   D. Apnea

327. Median nerve
   A. Is formed from medial and lateral cods
   B. Innervates all flexor compartment muscles except flexor carpi ulnaris
   C. Is also known as the labourer’s nerve
   D. Only (a) is correct

328. Radial nerve
   A. Is formed from posterior cord
   B. Innervates all extensor compartment muscles except anconeus
   C. Is prone to injury in fractures of the shaft of humerus
   D. Injury results in wrist drop

329. Ulnar nerve
   A. Is a branch of the posterior cord
   B. Is known as the musician’s nerve
   C. Is the major innervation of muscles of the hand
   D. Lesion of the nerve results in ape thumb

330. A 3/0 suture is
A. Larger in diameter than 4/0
B. Smaller in diameter than 4/0
C. Is too small for skin closure
D. Can be used in an adult patient to close the abdominal sheath

331. Boil (furuncle)
A. Can occur on any part of the body except the face
B. Is infection of hair follicle
C. Can cause signs of inflammation
D. Surgical drainage is not indicated

332. Carbuncle
A. Can occur on any part of the body except the neck
B. Is necrotic infection of contiguous hair follicles
C. Patients present with fever and malaise
D. Both surgical drainage and antibiotics are not required for treatment

333. Lymphangitis
A. Common causative organism include streptococci
B. Blood cultures are usually positive
C. Lymph nodes draining the infected area are usually swollen and painful
D. Presents with systemic inflammatory response syndrome (SIRS)

334. Regulations of fluids and electrolytes movement across all membranes is by
A. Diffusion/filtration
B. Active transport
C. Osmosis
D. Characteristics of the membrane itself

335. Atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) are hormones involved in regulation of
A. pH of the body fluids
B. Water and electrolyte balance
C. ANP is produced in atrium
D. BNP is produced in ventricles

336. A white leg is due to
   A. Femoral deep vein thrombosis
   B. Lymphatic obstruction
   C. Femoral vein thrombosis and lymphatic obstruction
   D. None of the above

337. The following are irritative symptoms of bladder outlet obstruction secondary to BPH:
   A. Weak stream
   B. Nocturia
   C. Frequency
   D. Intermittence

338. Ilio-Psoas muscle for hip flexion is innervated by
   A. L1, 2 segments
   B. L4, 5 segments
   C. L5, S1 segments
   D. S1, S2 segments

339. Secondary metastasis to the spine can originate from
   A. Thyroid cancer
   B. Breast cancer
   C. Lung cancer
   D. Prostate cancer

340. A seroma is also known as
   A. A hematoma
   B. A chyloma
   C. A collection of serum in the tissue
   D. All above correct

341. Choice of suture material
   A. Is irrelevant
   B. Depends upon the tissue and area of the body
   C. Is usually left up to the scrub nurse
   D. Is whatever the hospital gives you

342. About swellings
   A. A punctum is diagnostic of a lipoma
   B. A cyst is lined by some form of epithelium and is fluid-filled
   C. A sebaceous cyst exhibits pseudo-fluctuation
   D. Carotid body tumor moves only in one plane vertically

343. The following are the reasons why investigations are done
   A. Medico-legal purpose
   B. To make a diagnosis
   C. To establish fitness before surgery
   D. For monitoring of a disease process

344. Hypertrophic pyloric stenosis
   A. Is never seen in adults
   B. Projectile vomiting occurs
   C. A mass is never palpable
   D. Diarrhea is common
   E. Is associated with other congenital abnormalities

345. Indications for surgery in peptic ulcer disease include:
   A. Intractable pain
   B. Pyloric stenosis
   C. Absence of healing after 3 weeks of medical treatment
   D. Acute gastric erosion due to analgesic abuse
   E. Single episode of hematesis

346. Treatment of peptic ulcer disease includes:
   A. Antacids
   B. Antibiotics
   C. Prostaglandin analogues e.g. misoprostol
   D. Omeprazole
   E. Cimetidine

347. Operations performed for peptic ulcer disease includes
   A. Gastrostomy
   B. Gastropexy
   C. Vagotomy and gastrojejunostomy
   D. Billroth gastrectomy
   E. Polya gastrectomy

348. Which of the following conditions give rise to hemoperitoneum?
   A. A ruptured ectopic pregnancy
   B. Rupture of the bladder neck
   C. An injured spleen
   D. Spinal injuries
   E. An extraperitoneal isolated injury to the kidney

349. Post-operative pulmonary complications commonly occur:
   A. In patients with chronic bronchitis
   B. In smokers
   C. In patients with severe pain
   D. Following herniorrhaphy
   E. Following splenectomy

350. Open cardiac operations require one or more of the following
   A. Extracorporeal circulation
   B. Hypothermia
   C. Cardioplegia
   D. Right second intercostal posterolateral thoracotomy
   E. Sternotomy

351. Cardiac arrest is caused by
   A. Anoxia
   B. Excessive intravenous potassium
   C. Exsanguinating bleeding
   D. Frusemide
   E. Intravenous glucose

352. Cardiac tamponade following trauma:
A. Heart sounds are usually muffled
B. There is venous hypertension
C. There is arterial hypotension
D. Urgent surgical treatment is indicated
E. Urgent artificial ventilation is required preoperatively in all cases

353. Spinal (Lumbar) puncture:
A. Is usually performed at L1-L2 level
B. The cerebrospinal fluid is located in the subdural space
C. The cord ends at a higher level in an infant
D. Must not be performed in the presence of a raised intracranial pressure
E. A complication is venous bleeding

354. The following tumors readily metastasize to bone
A. Rectal adenocarcinoma
B. Basal cell carcinoma of skin
C. Follicular carcinoma of the thyroid
D. Prostatic adenocarcinoma
E. Renal adenocarcinoma

355. A lipoma has the following features
A. A slipping edge
B. Transillumination
C. Is tender to touch
D. Has a central punctum
E. Is only found in subcutaneous tissue

356. Tumors of the parotid include
A. Pleomorphic adenoma
B. Adenolymphoma
C. Papillary carcinoma
D. An enlarged intraparotid lymph node

357. Which investigations demonstrate occlusion of one common iliac artery
A. Inferior venacavogram
B. Translumbar aortogram
C. Doppler ultrasound
D. Intravenous pyelogram
E. Retrograde femoral catheterization of the other side

358. Varicose veins
A. Are tortuous elongated veins
B. Pain is a late feature
C. It is also known as venous hypertensive disease
D. Elastic support stocking are useful
E. Injection sclerotherapy is indicated in some patients

359. Hemorrhage
A. Primary hemorrhage occurs within 24 hours
B. Secondary hemorrhage occurs after a week to 10 days
C. Bleeding time is a measure of fibrinogen activity
D. The normal clotting time is up to 20 minutes
E. Bleeding in cirrhotics is immediately controlled by administering oral vitamin K

360. Common cause of pyrexia within 2 days of surgery include:
A. Chest infection
B. Blood transfusion
C. Wound infection
D. Venous thrombosis
E. Allergy to the anesthetic agent

361. Bacteroides species
A. Are common in the mouth
B. Are the commonest large bowel commensal
C. Are sensitive to metronidazole
D. Causes necrotizing fasciitis
E. Cause Crohn’s disease

362. An anteroposterior chest X-ray (done to demonstrate a left subphrenic abscess) will show:
A. Obliteration of the costophrenic angle on the same side
B. Collapse of the left upper lobe
C. Lack of movement on the right cupula of the hemidiaphragm on screening
D. A fluid level below the left hemidiaphragm
E. Shift of the gastric fundus

363. Fournier's gangrene of the scrotum
A. Is secondary to tuberculosis
B. Is very uncommon in Zambians
C. Minor trauma to the scrotal skin is an etiological factor
D. Metronidazole is indicated
E. Surgery is contraindication

364. The following antibiotics are effective against anaerobic organisms
A. Chloramphenicol
B. Erythromycin
C. Cloxacillin
D. Clindamycin
E. Actinomycin

365. Hand infections
A. The hand is rested with extension of all joints
B. Osteomyelitis is common in untreated pulp space infection
C. Paronychia refers to infection in the palm
D. Commonly follow minor neglected injuries
E. A common infecting organism is *E. coli*

366. Complications of nasogastric tubes include
A. Gastric perforation
B. Otitis media
C. Chest infection
D. Ulceration of nares
E. Esophageal stricture following prolonged intubation

367. Tetanus
A. Is caused by an anerobic bacillus
B. Is associated with a hypercatabolic state
C. Occurs only after major trauma
D. Tetanus toxoid gives artificial immunity
E. Common cause of death is respiratory failure

368. Streptomycin
A. Is an aminoglycoside
B. It is bactericidal in high dose
C. Causes pseudomembranous colitis
D. Causes jaundice
E. Causes ototoxicity

369. A venous cut down can be done on the following veins
A. Cubital
B. Cephalic
C. Subclavian
D. Umbilical
E. Posterior tibial

370. Torsion of the testis
A. If in doubt of the diagnosis explore
B. In unilateral torsion bilateral fixation is necessary
C. Does not occur in children
D. Is confused with a hydrocele
E. A horizontally placed low testis is diagnostic

371. Congenital hydrocele
A. Can be repaired by Lord's procedure
B. Is due to closure of the processus vaginalis
C. Phenol may be used to treat the hydrocele
D. May be due to epididymorchitis
E. Presents as inguinal hernia

372. Anesthetic drugs
   A. Diazepam may be used for premedication
   B. Mist magnesium trisilicate for premedication for cesarian section
   C. Thiopentone for induction
   D. Etomidate for reversal of muscle relaxation
   E. Morphine for post-operative analgesia

373. Post-operative wound breakdown may be due to
   A. Use of tight suturing technique
   B. Prolonged steroid use before surgery
   C. Reduced serum protein
   D. Protein energy malnutrition
   E. Diabetes mellitus

374. Metabolic response to trauma
   A. May be caused by fracture of a distal phalanx
   B. Good recovery follows steroid treatment
   C. Head injury may be the cause
   D. Patient may gain weight in the flow phase
   E. May lead to systemic inflammatory response syndrome

375. Peptic ulcer disease
   A. With hematemsis may be due to malignant transformation
   B. Can be duodenal if pain wakes patient after midnight
   C. Can be duodenal if pain is experienced immediately after meals
   D. Can be gastric if experienced immediately after food
   E. Can be a cause of acute abdomen

376. Cranial nerve that supplies superior oblique which elevates and adducts the eye is:
   A. 7^{th} cranial nerve
   B. 6^{th} cranial nerve
   C. 5^{th} cranial nerve
   D. 4^{th} cranial nerve

377. Sensory component of 5th cranial nerve is tested by
   A. Wrinkle formation on the forehead
   B. Touch sensation on the face
   C. Lifting the shoulder
   D. Corneal reflex

378. When a patient has unilateral vagus nerve lesion and is asked to open the mouth and say AAAG.. The uvula moves and points
   A. Away from the lesion
   B. Towards the lesion
   C. Stays in the midline
   D. Elongates within the midline

379. When a patient has unilateral hypoglossal lesion and is asked to protrude the tongue
   A. The tongue points away from the lesion
   B. The tongue points to the side of the lesion
   C. The tongue will show wasting
   D. The tongue will show fasciculations

380. When a patient sustains cranial nerve XI lesion
   A. Patient is unable to elevate (shrug) the affected shoulder
   B. Patient is able to elevate the shoulder against examiners resistance
   C. Sternomastoid muscle power is spared
   D. When patient is asked to rotate the chin against examiners resistance he/she fails to do so

381. When a patient has unilateral cranial nerve V motor component lesion
   A. Masseter muscle will feel weak on palpation as patient clenches the teeth
   B. Temporal muscle will feel well contracted as patient clenches the teeth
   C. The jaw will deviate to the paralyzed pterygoid muscle when patient opens the mouth
   D. The jaw will deviate away from the paralyzed pterygoid muscle when patient is asked to open the mouth

382. Bilateral 6th cranial nerve palsy is a sign of
   A. Cingulate gyrus herniation
   B. Uncal herniation
   C. Raised intracranial pressure
   D. Tonsillar herniation

383. Hyper-resonant chest is likely to be caused by
   A. Hemothorax
   B. Pleural effusion
   C. Pneumothorax
   D. Tension pneumothorax

384. Secondary brain injury
   A. Can result from hypoxia
   B. Can result from hypoperfusion
   C. Can result from hypoglycemia
   D. Can result from any of the above

385. Breast cancer is common in women with
   A. Positive family history
   B. History of having breast fed their children
   C. History of previous radiation exposure
   D. Having the first child at early age

386. Causes of surgical jaundice include:
   A. Gall stones
   B. Massive hemolysis
   C. Ascaris lumbricoides
   D. Malignancy of the head of pancreas

387. The most frequent anatomic site for squamous cell carcinoma of the esophagus is
   A. Upper third
   B. Middle third
   C. Lower third
   D. Gastro-esophageal junction

388. Pathological fractures may be associated with
   A. Scurvy
   B. Hyperparathyroidism
   C. Bone cyst
   D. All of the above

389. Perkin's exercises:
   A. Are commenced 2 weeks after application of traction kit
   B. Are commenced within a week after application of traction kit
   C. Are commenced after 3 weeks when pain at fracture site has subsided
   D. All above correct except "B"

390. Tachypnea
   A. Is respiratory rate of less than 10 per minute in adult patients
   B. Can be caused by tension pneumothorax
   C. Can be caused by hemothorax
   D. Is never associated with tachycardia

391. About investigations
   A. Gastroscopy is an example of an imaging technique
   B. For biliary pathology, ultrasound is superior to computerized tomography
   C. Plain X-rays are sufficient in order to make a diagnosis of fractures
   D. There are other methods apart from histology and cytology that are used to confirm cancer

392. Patients with central nervous system lesion on observation may have
   A. Epigastric peristalsis
   B. Ptosis
   C. Tremors
   D. Diaphragmatic respirations

393. These are major symptoms associated with intestinal obstruction
   A. Diarrhea
   B. Change in skin coloration
   C. Hematuria
   D. Melena stool

394. Oculomotor nerve lesion presents with
   A. Pupil dilation
   B. Ophthalmoplegia
   C. Lateral rectus palsy
   D. Loss of corneal reflex

395. Origin of oculomotor nerve (CN III) is
   A. Medulla-oblongata
   B. Pons
   C. Mid-brain
   D. Cerebellum

396. Open skull fracture has occurred when there is
   A. Cerebral spinal fluid leak from the ear
   B. Depressed skull fracture under the scalp wound
   C. Subdural hematoma with superficial bruise on the forehead
   D. Subarachnoid hemorrhage regardless of the cause

397. Patients who present with acute abdominal pain starting in the umbilical region and shifts to the right iliac fossa is most likely caused by
   A. Perforated duodenal ulcer
   B. Diverticulitis
   C. Appendicitis
   D. Salpingitis

398. Murphy's sign
   A. Helps to diagnose ureteric stones
   B. Helps to diagnose ectopic pregnancy
   C. Helps to diagnose appendicitis
   D. Helps to diagnose cholecystitis

399. Small intestinal obstruction is likely to present with
   A. Radiating pain to the left shoulder
   B. Spreading pain to the right iliac fossa
   C. Colic pain in the umbilical region
   D. Shifting pain to the right iliac fossa

400. Imperforate anus
   A. Maybe related to urogenital fistulae
   B. X-rays (invertogram) is not helpful
   C. Colostomy may be useful initially
   D. Cannot be a cause of intestinal obstruction
   E. May present with constipation

401. Drugs used for maintenance in anesthesia
   A. Nitrous oxide
   B. Halothane
   C. Isoflurane
   D. Oxygen
   E. Enflurane

402. Hirschprung's disease
   A. Lacks nerve tissue in the sub-mucosa layer
   B. A concentration of nerve tissue in the submucosa layer
   C. History of constipation is characteristic
   D. History of anal bleeding is characteristic
   E. Definitive diagnosis is made on X-rays

403. The gastrointestinal system has
   A. 4 basilica layers
   B. Mucosa lined by epithelium
   C. Muscularis mucosa to produce local movement of mucosa
   D. 5 basic layers
   E. Serosal lining covering mucosa

404. Normal blood values
   A. Total white cell count- 14 x 10^9/L
   B. Serum creatinine- 200umol/L
   C. Urea- 10mmol/L
   D. Sodium 155mmol/L
   E. Potassium 6.5 mmol/L

405. During surgical procedures
A. Doors to operating theaters must always be left open
B. Ventilators can reduce the atmospheric bacterial load
C. The patient is not the most important source of wound infection
D. Shaving must be done ideally the day before operation
E. Commonest organism to cause infection is streptococcus

406. Concerning antisepsis
A. Antisepsis has to do with reducing the bacterial load
B. Sterilization completely destroys all viable organisms
C. Disinfection reduces the bacterial load
D. Disinfection has to do with reducing the tissue bacterial load
E. Ultraviolet light is one way of sterilization

407. Fatty acids
A. Are 75% stored as glycogen in the liver
B. Glycogen stores used up in the stressful situations within 48 hours
C. Digestion is mainly by pancreatitic lipase
D. Liver stores maintain blood levels
E. Insulin is not important in cell uptake of carbohydrate

408. Most of protein digestion and absorption occurs
A. Terminal ileum
B. Stomach and duodenum
C. Stomach and colon
D. In the liver and gall bladder
E. Stomach

409. Concerning suturing of incision
A. Subcuticular sutures are not good for cosmesis
B. Mattress suture is good for hemostasis
C. Continuous sutures are ideal if infection is anticipated
D. Continuous sutures are ideal for long incisions
E. Head and neck sutures must be removed only after the 5^{th} days

410. Monofilament sutures
   A. Cause least tissue reaction
   B. Causes least wound infection
   C. Causes most tissue reaction and scarring
   D. Can be braised
   E. Can be absorbable

411. Nylon suture
   A. Absorbable suture and gives little tissue reaction
   B. Non-absorbable and gives a lot of tissue reaction
   C. Non-absorbable and gives little tissue reaction
   D. Synthetic
   E. Naturally occurring

412. On examination of the abdomen
   A. Spleen is examined starting from the right iliac fossa
   B. There are 9 surface division
   C. Inspection is done after palpation
   D. Percussion is done after palpation
   E. Normal bowel sounds heard every 2 seconds

413. Peptic ulcer disease
   A. With hematesis may be due to malignant transformation
   B. Can be duodenal if pain wakes patient after midnight
   C. Can be duodenal if pain is experienced immediately after meals
   D. Can be gastric if experienced immediately after food
   E. Can be cause of acute abdomen

414. Congenital hydrocele
   A. Can be repaired by Lord’s procedure
   B. Is due to closure of the processus vaginalis
   C. Phenol may be used to treat the hydrocele
   D. May be due to epididymorchitis
   E. Presents as inguinal hernia

415. Patients burns major burns
A. Are in negative nitrogen balance
B. Have normal calorie requirement
C. Do not generally become anemic
D. Are resistant to septicemia

416. Patients with raised intracranial pressure
A. Cerebral perfusion pressure is maintained by reduction of arterial pressure
B. Cerebral spinal fluid shifting
C. Uncal herniation is not a feature
D. Coma scale remains unaltered

417. The parafollicular cells of thyroid secrete
A. Calcitonin
B. Amyloid
C. Parathormone
D. Follicle stimulating hormone

418. An indirect inguinal hernia would lie in relation to the inferior epigastric artery:
A. Medial
B. Lateral
C. Anterior
D. Posterior

419. Fissure-in-Ano is located characteristically:
A. Midline anteriorly
B. Midline posteriorly
C. Right lateral quadrant
D. Left lateral quadrant

420. Post-traumatic intracranial space occupying lesion is likely to present with:
A. Decreased pulse rate
B. Increased blood pressure
C. Irregular pulse
D. Glasgow coma scale of 2/15

421. Paradoxical (Diaphragmatic) respiration can be caused by
   A. Odontoid fracture dislocation
   B. C2/3 hangman's fracture
   C. C5/6 fracture dislocation
   D. T112/L1 fracture dislocation

422. Concerning blood groups
   A. Blood group A is associated with increased risk of gastric cancer
   B. The risk of gastric cancer is associated with blood group B
   C. The risk of gastric cancer is associated with blood group O
   D. Only (b) and (c) correct

423. Acute abdomen can be caused by:
   A. Perforated duodenal ulcer
   B. Sigmoid volvulus
   C. Pancreatitis
   D. Subcutaneous lipoma

424. The following are true of breast cancer
   A. Lobular carcinoma is characterized by 'Indian File' pattern of tumors cells
   B. Lobular carcinoma tends to be familial, multifocal and bilateral
   C. Carcinoma-in-situ implies non-invasive cancer that has not breached basement membrane
   D. Macroscopically schirrhous carcinoma is hard, grey and grates on cutting

425. Symptoms of obstructive uropathy include the following:
   A. Weak stream, hesitancy
   B. Dribbling, straining to void
   C. Polyuria, oliguria
   D. Frequency, urgency, urge incontinence

426. Which are the causes of dysphagia
   A. Carcinoma, goiter, thymoma, mediastinal lymphadenopathy, aortic aneurysm
   B. Motor neuron disease, rabies, tetanus, pseudobulbar paralysis
   C. Achalasia
   D. Pneumonia, TB of spine

427. Concerning tumors, the term ‘well differentiated’ means
   A. The cells are so aggressive, wild and uncontrolled and keep reproducing to no good end
   B. Cells and tissue that have mature (specialized) structures and functions
   C. Cancer cells look more like normal cells under a microscope
   D. The cells tend to grow and spread more quickly than poorly differentiated cancer cells

428. About 85-90% radio-opaque urinary stones include the following
   A. Calcium oxalate stones
   B. Pure uric acid stones
   C. Sodium urate and ammonium stones
   D. Sulphur containing cysteine

429. Diabetic foot
   A. Presence of pathologies such as infection, ulcer and neuropathic osteoarthropathy
   B. Is always present in chronic diabetes mellitus
   C. Any chronic foot ulcer that eventually causes diabetes mellitus
   D. Patients have associated reduced ability to feel pain

430. Chest injuries
   A. Tension pneumothorax causes so-called ‘paradoxical breathing’
   B. May cause a rupture of the diaphragm
   C. A flail chest result from a wound in the chest wall which acts as a valve that permits air to enter the pleural cavity but prevents its escape
   D. May cause pulmonary contusion

431. Glasgow coma sale
   A. Is a neurological scale which aims to give an objective way of recording the conscious state of a person
   B. Is composed of eye, verbal and motor response
   C. Glasgow coma scale as high as ≥13 signifies minor head injury
   D. The light response and size of the pupil are part of the Glasgow coma scale assessment

432. Fournier's gangrene of the scrotum is associated with
   A. Infection
   B. Minor injuries
   C. No obvious cause
   D. Filariasis

433. Fibroadenoma
   A. Benign tumor with a mixture of stroma and epithelial tissue of breast
   B. Painless, firm, solitary, mobile, slowly growing lump in the breast of women of child-bearing years
   C. Leiomyoma in the breast tissue
   D. Benign tumor of the fibrous tissue in the sweat glands of the breast tissue

434. Cervical sympathectomy is procedure which is indicated for the treatment of
   A. Causalgia
   B. Raynaud's disease
   C. PUD
   D. Cervical rib

435. The following are causes of secondary varicose veins
   A. Klippel Trenuaney syndrome
   B. Oral contraceptive pills
   C. Pelvic tumors
   D. Exercises

436. Varicose veins are one of the causes of thrombophlebitis other causes include
   A. Blood transfusion
   B. Enteral feeding
   C. Chemotherapy
   D. Trauma

437. High risk group in the development of DVT would include the following features:
   A. Operation <30 minutes
   B. Obesity
   C. >40 years age
   D. Stroke patient

438. The development of skin malignancy pre-malignant lesions would include:
A. Solar keratosis
B. Keloids
C. Radio-dermatitis
D. Rodent ulcer

439. In hyperthyroidism
A. Serum creatinine is increased
B. Serum cholesterol is increased
C. Serum calcitonin is increased
D. Serum iodine is reduced

440. Causes of pulsatile thyroid swelling
A. Follicular carcinoma
B. Primary thyrotoxicosis
C. Secondary thyrotoxicosis
D. Papillary carcinoma

441. Medullary carcinoma of the thyroid (MCT):
A. Arises from parafollicular ‘c’ cells
B. Associated with MEN I syndrome
C. Associated with MEN IIa syndrome
D. Sporadic is the commonest

442. Tetany is a condition where there is hyper-excitability of peripheral nerves. The causes include
A. Hypoparathyroidism
B. Respiratory alkalosis
C. Pyloric stenosis
D. Chronic renal failure

443. In etiopathogenesis of carcinoma of the esophagus, precancerous conditions include:
A. Plummer vinson syndrome
B. Achalasia cardia
C. Corrosive stricture
D. Keratosis palmaris

444. In gastric outlet obstruction electrolyte changes
A. Hypernatremia
B. Hyperkalemia
C. Hypochloremic alkalosis
D. Hypocalcemia

445. In hepatocellular carcinoma alpha-fetoprotein levels are increased. What other conditions would increase the alpha-fetoprotein levels:
A. Carcinoma stomach
B. Carcinoma pancreas
C. Hepatoma
D. Embryonal cell carcinoma of testis

446. An increase in the level of alkaline phosphatase would be caused by
A. Biliary cirrhosis
B. Metastasis in the liver
C. Hepatocellular carcinoma
D. Hepatitis

447. Acute pancreatitis can be caused by drugs which include
A. Estrogen
B. Diuretics
C. Antifungals
D. None of the above

448. Complications of acute pancreatitis include the following except:
A. Shock
B. Hypocalcemia
C. Pleural effusion
D. Respiratory insufficiency

449. The following are components of wound healing except:
A. Wound contraction
B. Healing by primary intention
C. Epithelization
D. Scar formation

450. Factors affecting wound healing include:
A. Jaundice
B. Debilitation
C. Gender
D. Parity

451. Ludwig's angina precipitating factors:
A. Cachexia
B. Chemotherapy
C. Dental caries
D. Immobility

452. In a patient with established Tetanus, the possible causes of death would include
A. Aspiration
B. Cardiac arrhythmias
C. DVT
D. Laryngeal spasm

453. Tropical ulcers occur in Tropical countries. The precipitating factors are:
A. Malnutrition
B. Trauma
C. Insect bite
D. HIV infection

454. The following are causative factors in acute empyema thoracis:
A. Lung infection
B. Subdiaphragmatic abscess
C. Ruptured esophagus
D. Gunshot bullet lodging in pleural cavity

455. The following may complicate chronic empyema thoracis
A. Amyloidosis
B. Brain abscess
C. Bronchopleural fistula
D. Chronic anemia

456.

ESSAYS

1. Write short notes on:
   a. Salter-Harris classification of epiphyseal fractures
   b. Gatland classification of supracondylar fractures
   c. Draw and label patterns of fracture of neck of femur

2. Questions:
   a. Define excised and incised wound
   b. Describe briefly physiology of healing of excised wound
   c. List complications of wound healing

3. Questions
   a. Briefly trace iodine in food to iodine in thyroid hormone
   b. Draw homeostatic loop for T3 and T4
   c. List signs/ symptoms of thyrotoxicosis

4. Write short notes on
   a. Colles's fracture
   b. Colostomy
   c. Shoulder dislocation

5. Question
   a. List complications of thyroidectomy
   b. Discuss management of thyroid storm (Crisis)
   c. Discuss briefly clinical presentation of hypoparathyroidism

6. Write short notes on metabolic body response to acute blood loss.

7. Write short notes on value of sentinel node in investigating breast cancer

8. Discuss briefly common patterns and presentation of hand infection.

9. An 8-year-old child weighing 25kg has 20% burns which occurred at 04:00 hours and arrives in hospital at 08:00hrs. Calculate fluid requirement using Parkland formula:
   a. Resuscitation fluid
   b. Maintenance fluid
10. Write short notes on the complications of blood transfusion

11. What are signs and symptoms of acute appendicitis. Explain the anatomical basis for these features, where applicable.

12. You are called to a surgical ward, 2 hours after thyroidectomy of a patient with thyrotoxicosis. Write short notes on what complications your patient might have now and in the near future.

13. How would you investigate the cause of fever in a patient who develops a fever 3 or 4 days after abdominal surgery?

14. A 70-year-old man presents with acute retention in a male surgical ward. Take a brief relevant and focused history in order to arrive at a diagnosis

15. Persistent generalized lymphadenopathy is a common presentation of patients to a surgical clinical in Zambia. How would you evaluate such patients to come up with a diagnosis? Include in your answer the possible histological diagnostic description of the common causes.

16. Graves disease is fairly common condition in young women in Zambia. Write short notes on eye signs and give reasons why these signs do occur.

17. List the possible causes of PUD. Clearly explain how helicobacter pylori causes this disease

18. What is gangrene? What are the etiological factors of the different types of gangrene? Your short notes should include pathophysiology of each type of gangrene.

19. A 26-year-old man has been hit by a moving vehicle. He is brought to casualty in a coma. How would you evaluate this patient?

20. Write shore notes on presentation and diagnosis of cervical injury

21. Write the hemodynamic changes caused by space occupying lesions in head injury patients.
22. How much fluid are you going to give a 3-year old child weighing 15Kg with 20% burns in the first 48 hours?

23. There has been a plane crash in Ngwerere area with a few survivors. How would you handle the 13 survivors who have been brought to casualty?

24. A 65-year-old male patient presents to the casualty with a very painful ulcer on the right foot. He is a known hypertension and has been on treatment for several years. On examination has a moderate sized ulcer on the dorsum of the foot which is very painful. The ulcer has punched out edge and the base is pale. The SHO makes a diagnosis of a venous ulcer.
    A. What other history would you want to get from this patient?
    B. What further clinical examination would you do?
    C. Was the diagnosis correct? Give your reasons?
    D. What investigation is indicated
    E. What is the diagnosis and state your reasons?

25. Discuss the immediate management of a 5-year-old child with 15% burns.

26. Write short notes on flail chest.

27. The signs of intracranial bleeding after a head injury are
    A. Conscious level .....
    B. Pupil on side of lesion .....
    C. Pulse rate .....
    D. Blood pressure .....
    E. Respiration .....

28. A 65-year-old patient presents with a painful ulcer on the dorsum of his foot. He complains that he is unable to sleep at night and the pain is not relieved by moderate pain killer

A. List 2 differential diagnosis for this ulcer
B. What is the most likely diagnosis?
C. What 4 important things would you ask in the history to determine the etiologic factors?
D. What 2 blood investigations are indicated?
E. What radiological investigation is indicated?
F. Describe the following terms: a. Ankle Brachial pulse index and b. critical ischemia?

29. A 35-year-old patient is seen in casualty following a road traffic accident (RTA). The patient smells of alcohol and has a laceration on the occipital area. Answer the questions below briefly.
A. What two things would you ask in the history?
B. Explain how you would examine the occipital laceration
C. List 4 localizing signs you would look for
D. List 4 reasons you might ask for a CT scan in this patient

30. List the various indications for doing a colostomy

31. Discuss in brief the causes of surgical jaundice

32. Write short notes about Steel's rule application in a patient with cervical spine injury

33. A 34 year old woman presents with mild yellowness of eyes, pain in the right hypochondrium and fever. She gives history of previous episodes which resolve on antibiotic treatment. Answer the questions below:
A. What is the most likely diagnosis?
B. What is the most likely cause?
C. What other 2 things would you ask for in the history?
D. What 2 blood investigations would you ask for?
E. What 2 signs would be present on abdominal examination?
F. What radiological investigation is indicated?

34. Answer the following questions:
A. Outline briefly the operative blood transfusion check list
B. Discuss measures that you would take to treat an adverse blood reaction

35. Answer the following questions
    A. Define goiter
    B. Discuss briefly pre-operative investigations for goiter
    C. List possible post-operative complications of thyroidectomy
    D. How would you recognize and abort a thyroid storm

36. Give an account of thyroid swellings

37. A 46-year-old Zambian female presents to clinic 4 with a thyroid swelling which is painless and slow growing.
    A. What 4 things would you ask in the history to determine the cause?
    B. What 4 examinations would you do to determine whether the patient is euthyroid or toxic?
    C. What specific blood tests are indicated?
    D. What 3 radiological investigations would you do?
    E. List 4 indications for surgery in this patient

38. Answer the following questions
    A. Define osteoarthritis
    B. Discuss briefly etiological factors in general and clinical presentation of osteoarthritis of lumbar spine
    C. List the four classical radiological findings in osteoarthritis
    D. List the treatment modalities available for osteoarthritis.

39. Define the following
    A. Fistula
    B. Fissure
    C. Ulcer
    D. Granulation tissue
    E. Paralytic ileus

40. Write short notes on the following
    A. Define sentinel lymph node
    B. Discuss briefly value of sentinel axillary lymph node biopsy in management of breast cancer

41. A 37-year-old female patient is seen in clinic 4 with a lump in the breast and an axillary swelling. She gives history of weight loss and back ache. Answer the questions below.
    A. List 2 differential diagnosis
    B. What is the most likely diagnosis?
    C. What 4 important questions would you ask to determine the possible etiological factors?
    D. List 2 investigations you would do to confirm your diagnosis
    E. What 2 radiological investigations would you do to determine whether metastasis is present?
    F. What is the value of the sentinel node in this condition?

42. A 56-year-old patient is seen in casualty with history of failing to pass urine. He has severe pain and abdominal distention.
    A. What is the diagnosis?
    B. List 2 possible conditions that can lead to this complication
    C. List 4 questions you would ask to differentiate the 2 conditions
    D. What single physical examination is necessary in this patient?
    E. What one endoscopy and one radiological investigation should be done?

43. A 40-year-old man comes to the emergency ward with acute urinary retention. What are the possible etiologies, briefly outline how you would investigate.

44. A 34-year-old woman presents with mild yellowness of eyes, pain in the right hypochondrium and fever. She gives history of previous episodes which resolve on antibiotic treatment. Answer the questions below
    A. What is the most likely diagnosis?
    B. What is the most likely causes?
    C. What other 2 things would you ask for in the history?
    D. What 2 blood investigations would you ask for?
    E. What 2 signs would be present on abdominal examination?
    F. What radiological investigation is indicated?

45. A beautiful 40-year-old female patient is seen in casualty with right upper quadrant pain which radiates to the scapula. She gives history of intermittent attacks associated with fatty foods. On examination Murphy's sign and

Moynihan's sign are both positive. The SHO on call makes a diagnosis of Acute pancreatitis and requests some blood tests. The result shows serum amylase 100 u/L, Alkaline Phosphates 300 u/L, SGPT 30u/L, SGOT 31u/L and bilirubin 25 micronmols/L. The SHO is uncertain of the interpretation of the results and calls the medical Registrar on call. The Registrar takes the history on the phone. The medical Registrar is extremely upset with the young SHO, but nonetheless recommends IV antibiotics, analgesia and an urgent U/S. By the morning round the SHO has altered his diagnosis and takes the credit for an astute diagnosis from the newly appointed Prof of Surgery.

A. What other important history should be taken?
B. What diagnosis do both the history and examination point to?
C. What do the investigations show?
D. Is a plain X-ray indicated in this patient?
E. Why was the medical registrar so upset?
F. What were the surgical principles on which the medical registrar gave his advise?

46. Answer the following questions:
    A. Define compartment syndrome and list major etiological factors
    B. Discuss briefly the pathophysiology of compartment syndrome
    C. With aid of labelled diagram, explain Whitesides saline/needle technique for measuring intracompartment pressure in a limb

47. Write short notes on assessment of a patient with burns

48. Write short notes on usefulness of intravenous urogram (IVU) in a patient with bilateral hydronephrosis

49. Describe the steps involved in the synthesis of Tri-iodothyronine (T3) and thyroxine (T3). Comment on homeostatic (regulatory) feed-back loop

50. Answer the following
    A. Define osteoarthritis
    B. Discuss briefly pathogenesis of degenerative osteoarthritis
    C. List the four cardinal radiological features of osteoarthritis

51. List baseline investigations and their significance in preparation of a patient due for thyroidectomy

52. Answer the following questions
    A. Describe phases of fracture healing in a long tubular bone
    B. List in tabular form complications associated with fractures
    C. List the methods available for reduction of fractures

53. Write short notes on:
    A. Autologous blood transfusion, types, uses and limitations
    B. Pre-operative blood transfusion check list
    C. Steps to be taken in aborting hemolytic blood transfusion reaction

54. Write short notes on how you would manage a patient you found to have a ruptured liver during an operation in a district hospital.

55. Discuss the various hormones that are involved following the ruptured spleen in a 25-year-old man.

56. A 47-year-old female patient is admitted to casualty with swelling of her left leg of spontaneous onset. She has a fever and the leg feels warm
    A. List 2 differentials diagnosis
    B. What bacteria is likely to be the cause?
    C. What 2 important questions would you ask for in the history to help determine the etiological factors?
    D. What 4 signs would you look for in the examination?
    E. What 2 blood investigations would you do?
    F. What antibiotic is indicated and what route of administration is recommended?

57. A 15-year-old boy presents to the casualty department with scrotal pain. He gives a history of sudden onset with no history of trauma. Answer the questions below.
    A. What are the 2 main differentials?
    B. What 4 things would you ask for in the history to differentiate the two?
    C. What is the most likely diagnosis?
    D. What is Prehn's test? Describe how it is done.
    E. What radiological investigation is indicated?

58. A 12 year old boy presents to clinic 3 with a swelling on the upper leg which is fast growing. The child is ill and give history of weight loss
    A. List 2 differential diagnosis
    B. What radiological investigation might distinguish the two?
    C. What other radiological investigation can be done?
    D. List 2 X-ray findings in osteosarcoma

59. A game ranger is brought to the casualty with a gunshot wound to the right leg. The wound is dirty and 5cm in length. The patient has palpable pulses and nerve function is normal.
    A. What type of fracture has the patient had?
    B. What is the classification of open tibia fractures?
    C. How would you classify this patient's fracture?
    D. What blood investigations would you do?
    E. What 2 organisms would be present on pus swab of this wound?

ANSWERS TO TRUE/FALSE

1. Long term sequelae of head injury:
   A. Brain atrophy is a Feature-True
   B. Focal neurological deficits are not a Feature-False
   C. Memory deficits are not a Feature-False
   D. Epilepsy is a Feature-True

2. Clinical classification of head injury is based on:
   A. Mode of Injury-False
   B. Whether it is open or Closed-False
   C. Glasgow coma Scale-True
   D. Pathological Lesion-False

3. Subdural hematoma follows:
   A. Tearing of superior sagittal Sinus-False
   B. Tearing of cerebral Veins-True
   C. Middle meningeal artery Laceration-False
   D. Vertebral artery Laceration-False

4. Head injury patient with subdural hematoma
   A. Is likely to deteriorate faster if blood pressure is Reducing-False
   B. Is likely to deteriorate if blood pressure is Increasing-True
   C. Is likely to deteriorate faster if pupils are starting to become Unequal-True
   D. Is likely to deteriorate faster if hemiparesis is starting to Present-True

5. The main source of bleeding in a deep scalp laceration is
   A. Periosteum-False
   B. Loose connective Tissue-False
   C. Aponeurotica galea-False
   D. Dense connective Tissue-True

6. Epidural hematoma in head injury patient follows:
   A. Tearing of cerebral Veins-False
   B. Brain Laceration-False
   C. Middle meningeal artery Laceration-True
   D. Superior sagittal sinus Tear-True

7. Head injury patient who presents with skull fracture crossing the midline
   A. Is likely to have epidural hematoma from superior sagittal sinus Laceration-True
   B. Is likely to have epidural hematoma from middle meningeal artery Laceration-True
   C. Is likely to have cerebral spinal fluid leaking from both ears and Nose-False
   D. Is likely to have fracture dislocation of C5/C6 cervical Spine-False

8. Burns are classified in terms of depth as
   A. Superficial partial Thickness-True
   B. Deep partial Thickness-True
   C. Full Thickness-True
   D. Full thickness is also known as second degree Burns-False

9. The rule of 9 for estimating extent of burns is as follows:
   A. Head and neck 9%-True
   B. Front of trunk 18%-True
   C. Perineum 11%-False
   D. Upper limbs 18%-True

10. Cerebrospinal otorrhea:
   A. Fracture of the patients' temporal bone is always Present-False
   B. Fracture of the base of the skull is the Cause-True
   C. Immediate surgical repair is Indicated-False
   D. Infection is Common-True
   E. Indicates injury to the 6th cranial Nerve-True

11. The comatose patient with a head injury:
   A. Is nursed on his side to prevent aspiration of stomach Content-True
   B. Must have endotracheal intubation if pharyngeal reflexes are Absent-True
   C. Must have a nasogastric tube Inserted-True

D. A urinary catheter is useful for monitoring fluid Balance-True
E. A suction machine is Essential-True

12. Urine output in a burnt patient:
    A. In children an output of 1ml/kg/hr must be Maintained-True
    B. In adult ensure an output of 70ml/Hour-True
    C. Catheterize all patients with extensive Burns-True
    D. In a 3-year-old female child size 24 Foley is Used-False
    E. Antibiotics are routinely given to catheterized Patient-False

13. Cerebrospinal otorrhea complicating head injury requires:
    A. Antibiotic ear Drops-True
    B. Plugging the external auditory Meatus-False
    C. Syringing with Saline-False
    D. Early dural Repair-True
    E. Parenteral Antibiotics-True

14. Systemic effects of burns
    A. Hyper-Metabolism-True
    B. Immunosuppression-True
    C. SIRS-True
    D. Keloids-False
    E. Contractures-False

15. Complications of burns
    A. Marjolin's Ulcer-True
    B. Keloids-True
    C. Systemic inflammatory response Syndrome-True
    D. Renal Failure-True
    E. Airway Obstruction-True

16. Triage refers to:
    A. Surgical wound Debridement-False
    B. Categorizing and sorting patients according to Priority-True
    C. Casualty Evacuation-False

D. Only B and C Are Correct-False

17. In mass casualty injuries, the following injuries require immediate treatment
    A. Sucking wounds of Chest-True
    B. Flail Chest-True
    C. Tension Pneumothorax-True
    D. Penetrating wound of the Abdomen-True

18. Complications of intercostal drain insertion:
    A. Pneumothorax-True
    B. Injury to intercostal neurovascular Bundle-True
    C. Lung and liver Trauma-True
    D. Surgical Emphysema-True

19. The edge of a tuberculous ulcer is
    A. Shelving-False
    B. Rolled up-False
    C. Undermined-True
    D. Punched out-False

20. The edge of a malignant ulcer is
    A. Shelving-False
    B. Rolled up-True
    C. Undermined-False
    D. Punched out-False

21. Debridement is
    A. The process of making an Incision-False
    B. The removal of foreign material or dead Tissue-True
    C. The process of obtaining Hemostasis-False
    D. None of the Above-False

22. About ulcers:
    A. Diabetic ulcer is an example of a specific Ulcer-False

B. Malignant melanoma ulcer is always Pigmented-True
C. Arterial ulcers are located on the medial aspect of the Leg-False
D. Trophic ulcers are Painful-True

23. Acute raised intracranial pressure is treated by:
A. Mannitol-True
B. Furosemide-True
C. Hyperventilation-True
D. Steroids-True
E. Raising the foot end of the Bed-False

24. Decubitus ulcers
A. Only occur in comatose Patients-False
B. Are a form of ischemic Necrosis- True
C. Cause Septicemia-True
D. Antibiotics cure the Condition-False
E. Surgery is of value in selected Cases-True

25. History taking from a patient
A. Accounts for 10% of the diagnostic Process-False
B. Open ended questions direct the Interview-False
C. Referral letter may reveal important Information-True
D. Occupation of the patient is not Important-False
E. Smoking is associated with Pancreatitis-True

26. Skin grafting
A. Auto graft refers to the use of pig Skin-False
B. Xenograft usually takes and becomes part of the body Skin-False
C. Allograft fails to provide any normal Function-False
D. Split thickness skin grafting needs pedicles to Survive-True
E. Split thickness graft heals with excellent Appearance-True

27. Early signs of a rising intracranial pressure include:
A. Bradycardia-True
B. Deteriorating conscious Level-False

C. Hypothermia-False
D. Hypertension-True
E. Tachypnea-False

28. The Glasgow coma chart includes details on the following
A. Pupillary Size-True
B. Motor Response-True
C. Urine Output-False
D. Hemoglobin Level-False
E. Injury severity Score-True

29. Pneumothorax
A. Occurs spontaneously Sometimes-True
B. Bullae are a Cause-True
C. Is a complication of flail Chest-True
D. Thoracotomy is frequently Indicated-True
E. A chest tube is always Necessary-False

30. Spontaneous pneumothorax is caused by
A. Tuberculosis-True
B. Emphysematous Bullae-True
C. Solitary lung Cyst-True
D. Chronic Bronchitis-True
E. Idiopathic-True

31. Decerebrate rigidity
A. Is commonly seen in patients with Glasgow coma scale of 8 or Less-True
B. Is commonly seen in patients with Glasgow coma scale of 12 or more-False
C. Is commonly seen in a patient with stiff extension of Limbs-True
D. Is commonly seen in a patient who is well oriented in person, space and Time-False

32. Brainstem injury is mostly likely to have occurred in patient who presents with:
A. Glasgow coma scale 15/15-False
B. Glasgow coma scale 15/15 and unequal Pupils-False
C. Cerebral spinal fluid leak from both Ears-True

D. Occipital scalp Laceration-True

33. All end colostomies are:
    A. Permanent Colostomies-True
    B. Defunctioning colostomies- True
    C. Not suitable for ano-rectal Malformation-False
    D. Hartmann's Colostomies-False

34. Complications of colostomy include
    A. Bleeding-True
    B. Diarrhea-True
    C. Prolapse-True
    D. Retraction-True
    E. Parastomal Hernia-True

35. Types of colostomy
    A. Spectacle Colostomy-True
    B. Double barrel Colostomy-True
    C. Ileostomy-False
    D. Gastrostomy-False
    E. Pharyngostomy-False

36. Indications for permanent end-colostomy
    A. Stage 1 carcinoma of the Sigmoid-False
    B. Carcinoma of the Caecum-True
    C. High fistula-in-ano-True
    D. Inoperable vesicle-vaginal Fistula-False
    E. Duke-B squamous cell carcinoma of the Anus-False

37. A patient with flail chest
    A. Does not require a chest Drain-True
    B. Rarely requires Ventilation-True
    C. Should be nursed in a dark Room-False
    D. The flail segment moves in on Inspiration-True

E. Should be treated in the intensive care Unit-True

38. Complications of defunctioning loop colostomy of the sigmoid colon include
    A. Electrolyte Imbalance-True
    B. Excoriation of Skin-False
    C. Retraction-True
    D. Prolapse-True
    E. Internal Hernia-True

39. Snake bite results in the following:
    A. Visible puncture Wounds-True
    B. Swelling of the bitten Limb-True
    C. Occasionally Gangrene-True
    D. Disseminated intravascular coagulation Frequently-True
    E. Fat Embolism-False

40. In a jaundiced patient with palpable gallbladder, the cause of jaundice is most likely to be:
    A. Gall stones in the gall Bladder-False
    B. Gall stone in the common bile Duct-False
    C. Cancer of head of Pancreas-True
    D. Cancer left Kidney-False

41. Generalized peritonitis is likely to follow
    A. Delayed diagnosis of perforated duodenal Ulcer-True
    B. Acute Pancreatitis-True
    C. Ruptured Appendicitis-True
    D. Torsion of the Testis-False

42. Bilateral lower abdominal pain in a female patient is most likely caused by
    A. Ectopic Pregnancy-True
    B. Appendicitis-True
    C. Salpingitis-True
    D. Ureteric Stone-True

43. Small intestinal obstruction is likely to present with
    A. Radiating pain to the left Shoulder-False
    B. Spreading pain to the right iliac Fossa-False
    C. Colic pain in the umbilical region -True
    D. Early Vomiting-True

44. During wound healing fibroblasts migrates across the following protein:
    A. Fibrillin-True
    B. Fibronectin-True
    C. Elastin-True
    D. Laminin-False

45. Regarding collagen
    A. It is composed to tropocollagen-True
    B. It is synthesized mostly by mast Cells-False
    C. Type II collagen is most common in connective tissue Proper-False
    D. None above are Correct-False

46. Basement membrane consists of:
    A. Type I Collagen-False
    B. Type II Collagen-False
    C. Type III Collagen-False
    D. Type IV Collagen-True

47. The following factors do not contribute to wound disruption
    A. Massive blood Transfusion-False
    B. Anemia-False
    C. Abdominal Distention-False
    D. Vitamin C Deficiency-False

48. 7 days after suturing, tensile strength of a wound will be:
    A. 5% of the uninjured Tissue-True
    B. 20% of the uninjured Tissue-False

C. 30% of the uninjured Tissue-False
D. 50% of the uninjured Tissue-False

49. Acute appendicitis
A. Pain can be referred to the right subcostal Region-False
B. Pain can shift to right iliac Fossa-True
C. Can be associated with Vomiting-True
D. Can be associated with left hip joint Flexion-False

50. Large bowel intestinal obstruction
A. Early vomiting is a Feature-False
B. Gross abdominal distension is a Feature-True
C. Sigmoid volvulus is a possible Cause-True
D. Rectal carcinoma cannot be the Cause-False

51. Wound healing
A. Is impaired in anemic Patients-True
B. Impaired in hematoma Formation-True
C. Impaired in hypoproteinemia-True
D. Stimulated by Steroids-False

52. Intestinal obstruction
A. Can be classified depending on the age of the Patient-True
B. Can be made on erect clinical Examination-True
C. Can be due to electrolyte Imbalance-True
D. Pyloric stenosis is caused by lower intestinal Obstruction-False

53. Obstructive jaundice
A. Is associated with biliary Atresia-True
B. Is associated with itching of the Skin-True
C. A patient with obstructive jaundice passes pale Stool-True
D. Obstructive jaundice can be diagnosed by supine X-Ray-False

54. Complications of intercostal drain insertion
A. Pneumothorax-True
B. Injury to intercostal neurovascular Bundle-True

C. Lung and liver Trauma-True
D. Surgical Emphysema-True

55. In acute appendicitis
A. Central or epigastric pain radiates to right iliac Fossa-False
B. Central or epigastric pain shifts to right iliac Fossa-True
C. Pain precedes nausea and Vomiting-True
D. Nausea and vomiting precedes Pain-False

56. In acute appendicitis
A. Mantrel (Alvarado) scores of 7 out of 10 is highly suggestive of Appendicitis-True
B. Right iliac fossa (McBurney's point) tenderness and leukocytosis are allotted 2 points each on Mantrel Scale-True
C. Rovsing's sign and obturator test are contributory to Diagnosis-True
D. Digital rectal examination has no Value-False

57. In wound healing
A. Cytokines are solely produced by hemopoeitic Cells-False
B. Cytokines optimize cellular response in Inflammation-True
C. Interleukins are not classified as Cytokines-False
D. Macrophages actively synthesize cytokine growth factors for Angiogenesis-True

58. In a completely healed wound, the tensile strength will be
A. 20% of uninjured Tissue-False
B. 40% of uninjured Tissue-False
C. 60% of uninjured Tissue-False
D. 80% of uninjured Tissue-True

59. Wound closure using suture is termed
A. Closure by primary Intention-True
B. Closure by secondary Intention-False
C. Hemostasis-False
D. Only (C) is Correct-False

60. Factors which affect wound healing include:
    A. Chronic Illness-True
    B. Radiation Therapy-True
    C. Nutrition-True
    D. All above are Correct-True

61. The following conditions are associated with intestinal obstruction
    A. Imperforate Anus-True
    B. Hirschprung's Disease-True
    C. Peritonitis-False
    D. Massive potassium Loss-True

62. Common causes of intestinal obstruction in the pediatric age group include:
    A. Diaphragm of the Duodenum-True
    B. Ileal Duplication-False
    C. Intussusception-True
    D. Imperforate Anus-True
    E. Peritonitis following a ruptured Appendix-False

63. In the University Teaching Hospital colostomies are commonly performed for the following conditions
    A. Volvulus of the sigmoid Colon-True
    B. Vesicovaginal Fistula-False
    C. An obstructed umbilical Hernia-False
    D. Imperforate Anus-False
    E. Meckel's Diverticulum-False

64. Paralytic intestinal obstruction occurs in
    A. Hypokalemia-True
    B. Hyponatremia-True
    C. Peritonitis-True
    D. Hemoperitoneum-True
    E. Retroperitoneal Hematoma-True

65. Mechanical intestinal obstruction occurs in
    A. Internal Hernia-True
    B. Intussusception-True
    C. Gastric Polyps-True
    D. Gall stone Ileus-True
    E. Volvulus of the Cecum-True

66. Common causes of peritonitis in Lusaka are
    A. Tuberculosis-True
    B. Perforated Appendix-True
    C. Typhoid Perforation-True
    D. Biliary Leak-False
    E. Perforated diverticulum of the sigmoid Colon-False

67. Shift of the falx cerebri occurs in
    A. Hydrocephalus-False
    B. Astrocytoma-True
    C. Meningitis-False
    D. Barry aneurysm of the Circle of Willis-True
    E. Extradural Hematoma-True

68. Gall stones cause
    A. Acute cholecystitis-True
    B. Acalculous cholecystitis-False
    C. Obstructive Jaundice-True
    D. Carcinoma of the gall Bladder-True
    E. Torsion of the gall Bladder-True

69. Factors which are known to cause perforation of the ileum include
    A. Trauma-True
    B. Typhoid-True
    C. Gall Stones-False
    D. Foreign Body-True

E. Diverticulum of the Ileum-False

70. Mechanical ileus is a complication of
A. Hypokalemia-False
B. Pelvic Peritonitis-False
C. Adhesions-True
D. Internal Hernia-True
E. Paraplegia of acute Onset-False

71. Paralytic ileus is a complication of
A. Retroperitoneal Hematoma-True
B. Paraplegia of slow Onset-True
C. Hyperkalemia-False
D. Hypercalcemia-True
E. Peritonitis-True

72. Medical conditions which are known to present as acute abdomen include:
A. Sickle cell Disease-True
B. Pneumonia-True
C. Hypocalcemia-True
D. Addison's disease-False
E. Hemophilia-True

73. Early signs and symptoms of compartment syndrome include:
A. Absent Pulse-True
B. Paresthesia-True
C. Inability to move toes or Fingers-False
D. A compartment pressure of less than 10mmHg-False
E. Decrease in limb Pain-False

74. Pre-operative care before laparotomy for intestinal obstruction
A. Nasogastric tube is Mandatory-True
B. Urethral catheterization may be avoided to prevent Infection-False

C. Antibiotic use should only be instituted after knowing the cause of Obstruction-False
D. X-match should be done for all Patients-True
E. Raised serum urea could be treated by furosemide to flash it through the Kidneys-True

75. Factors that increase wound infection postoperatively include
A. Wound Irrigation-False
B. Tissue Damage-True
C. Hematoma formation- True
D. Bacteria load <100 000/g Tissue-False
E. Keeping clean incised wounds dressed for 48 Hours-False

76. Regarding incisions
A. Keloids form more readily on sternal area than Abdomen-True
B. Langers lines are not used for placing Incisions-False
C. Avoid old scars when re-Operating-True
D. Incised wounds heal with minimal Scarring-True
E. Traumatized skin edges can be sites of wound breakdown Later-True

77. The signs of uncomplicated volvulus are:
A. Gross abdominal distention- True
B. Ballooning of the Rectum-False
C. Dyspnea-False
D. Severe tenderness on abdominal Palpation-True

78. The usual symptoms of appendicitis are
A. Central abdominal pain with severe Vomiting-True
B. Fever over 40^{o}C-True
C. Central abdominal pain moving to right iliac Fossa-True
D. Nausea and Anorexia-True
E. Painful Micturition-False

79. The symptoms of acute uncomplicated small gut obstruction are:
A. Continuous severe abdominal Pain-False
B. Absolute Constipation-False
C. Abdominal Distension-False

D. Bilious Vomiting-True
E. Colicky suprapubic Pain-False

80. The signs of acute uncomplicated small gut obstruction are:
A. Oliguria-False
B. Abdominal Distension-False
C. Tinkling bowel Sounds-False
D. Absence of guarding and rebound Tenderness-True
E. Multiple fluid levels on erect film of Abdomen-True

81. Wound healing by secondary intention occurs:
A. When the wound edges are not Approximated-True
B. When there is excessive skin Loss-True
C. When the wound is Infected-True
D. In anemia Patients-False
E. In jaundiced patients-False

82. Sigmoid volvulus
A. Is initially treated with a rectal Tube-True
B. Is common in African Female-False
C. Rectal bleeding is Common-False
D. Is an emergency best treated by barium Enema-False
E. Resection and primary anastomosis is an acceptable form of Treatment-True

83. Paralytic ileus (adynamic obstruction) occurs in
A. Patients following Laparotomy-True
B. Peritonitis- True
C. Uremia- True
D. Hypokalemia-True
E. Hypocalcemia-False

84. Clinical features of acute intestinal obstruction include:
A. Abdominal Pains-True
B. Melena-False
C. Vomiting-True

D. Distention of Abdomen-True
E. Constipation-True

85. Acute intussusception in adults occurs due to
A. Gall Stones-False
B. Change of Diet-True
C. Maximum aggregation of Peyer's Patches-True
D. Intestinal Polyps-True
E. Head Injury-False

86. The following are predisposing causes of sigmoid volvulus
A. Chronic Constipation-False
B. Redundant pelvic mesocolon-True
C. Refined dietary Intake-False
D. Hypokalemia-False
E. Uremia-False

87. The differential diagnosis of acute appendicitis includes:
A. Tonsillitis-False
B. Acute cholecystitis-False
C. Carcinoma of the Cecum-True
D. Ectopic Gestation-True
E. Infection in urinary Tract-True

88. Burst abdomen is caused by:
A. Wound infection- True
B. Leak of pancreatic Enzymes-True
C. Anemia-True
D. Jaundice- True
E. Persistent Cough-True

89. In fracture of the base of the skull the following nerves are commonly injured
A. Olfactory-True
A. Facial-True
B. Vestibulocochlear-True

C. Accessory- True
D. Hypoglossal-True

90. Keloid characteristics include
A. Onset is often delayed for months- True
B. Is cured by cytotoxics- False
C. Regresses Spontaneously-False
D. Pressure garments are useful following surgery- True
E. Radiotherapy is contraindicated- True

91. The epidermis of the skin consists of the following:
A. Stratum corneum-True
B. Stratum Lucidum-True
C. Stratum Granulosum-True
D. Stratum Malpighi-True
E. Stratum Basalis-True

92. Complications of blood transfusion include:
A. Congestive cardiac Failure-True
B. Antibody Reaction- True
C. Infection- True
D. Disseminated intravascular Coagulation-True
E. Fat embolism- False

93. Inguinal hernia
A. Indirect hernia is more common than direct Hernia-True
B. Strangulation is more common with direct hernia than with indirect Hernia- False
C. Obstruction is more common with indirect inguinal hernia than direct hernia- True
D. Direct hernia is likely to protrude through Hesselbach's triangle- True

94. Differential diagnosis of inguino-scrotal mass includes:
A. Hernia-True
B. Hydrocele-True

C. Meningocele-False
D. Lipoma-True

95. The following conditions are associated with abdominal hernias:
A. Chronic Cough-True
B. Multiple pregnancies in Women-True
C. Poor surgical abdominal wound Management-True
D. Ascites-True

96. An indirect inguinal hernia
A. Is the commonest hernia in young Adults-True
B. Exits through the femoral Ring-False
C. Has its neck medial to the inferior epigastric Vessels-False
D. Does not occur in female Patients-False
E. Is treated with a Truss-True

97. Umbilical hernias:
A. Are common in Zambian Children-True
B. All need herniorrhaphy before 2 years of Age-False
C. Does not give rise to Complications-False
D. All are Paraumbilical-False
E. Adults do not have umbilical Hernias-False

98. Features associated with incompatible blood transfusion include:
A. Loin pain- True
B. Tachycardia- False
C. Bradycardia- True
D. Cyanosis- True
E. Jaundice- True

99. Congenital diaphragmatic hernia
A. Are Common-True
B. Always occur in anterior mediastinum- False
C. Is invariably Fatal-False
D. Diagnosis is often missed initially in small hernias- True

E. Are best operated upon at 10 Years-False

100. Concerning hernias
A. Inguinal hernias are more common on the Right-True
B. Femoral hernias are more common in females than in Males-True
C. Inguinal hernias are more common than femoral hernias in Females-False
D. Paraumbilical hernias are Acquired-True
E. Umbilical hernias are always Congenital-True

101. Risk factors for developing inguinal hernias include
A. Male factor-True
B. Enlarged prostate-True
C. Chronic cough-True
D. Ascites-True
E. Chronic constipation-True

102. Differential diagnosis of inguinal hernia in a female are:
A. A hydrocele of the canal of Nuck-True
B. Femoral hernia-True
C. An encysted hydrocele of the cord-False
D. Lipoma of the cord-False
E. Spermatocele-False

103. An inguinal hernia in a 3-month old baby boy should be treated
A. Because small gut strangulation is a risk-True
B. By a truss-False
C. By early operation-True
D. Left alone because it resolves spontaneously-False
E. By surgery only if the hernia becomes irreducible-False

104. Quadriplegia
A. Can be caused by fracture dislocation at C5/C6 cervical spine-True
B. Can be caused by fracture dislocation at T5/T6 thoracic spine-False
C. Can be caused by Hangman fracture-True

D. Hypoventilation is not a feature-False

105. Second cervical vertebra and odontoid fracture
   A. Basal odontoid fracture extending into body of C2 is type III fracture and is unstable-True
   B. Basal odontoid fracture is type I and is stable-False
   C. Apical fracture of odontoid process is type II-False
   D. Jefferson's fracture is C3 fracture-False

106. Patient who presents with goiter, asphyxia and hoarseness of voice:
   A. Is likely to have malignant goiter-True
   B. Is likely to have retrosternal goiter-True
   C. Is likely to have unequal pupils-False
   D. Is likely to have dilated pupils-False

107. Preoperative treatment of toxic goiter
   A. Iodine can be given in combination with carbimazole or propranolol-True
   B. Iodine can be given and reduces vascularity of goiter-True
   C. Nadolol is not a beta-blocker to be used in treating toxic goiter-False
   D. It is not necessary to reduce toxic goiter to euthyroid status before operations-False

108. Secondary thyrotoxicosis
   A. Occurs in older patients than those with primary thyrotoxicosis-True
   B. Eye signs including lid lag and exophthalmos are absent-True
   C. Early signs of cardiac decompensation like ankle edema and dyspnea are a feature-True
   D. Orthopnea is not a feature-False

109. Malignant thyroid
   A. Papillary carcinoma is most uncommon-False
   B. Anaplastic carcinoma is seen in old patients-True
   C. Follicular carcinoma is seen in 20% of patients of middle age-True
   D. Medullary carcinoma is the commonest malignancy-False

110. Hashimoto's disease
   A. Is common in post-menopausal women-False
   B. On inspection one lobe of the thyroid is larger than the other-False
   C. Symptoms of hypothyroidism are absent-False
   D. Blood test may show antibodies to the thyroid gland-True

111. Thyroglossal cyst
   A. It is anterior cervical midline mass-True
   B. Subhyoid is rare site for thyroglossal cyst-False
   C. Thyroglossal fistula is a rare post-operative complication-False
   D. Moves upwards when the tongue is protruded-True

112. The following vessels supply thyroid gland
   A. Superior thyroid from external carotid artery-True
   B. Inferior thyroid from thyrocervical trunk of subclavian artery-True
   C. Thyroidea ima artery from arch of aorta-True
   D. All above except "B" and "C"-False

113. Malignant goiter
   A. Medullary carcinoma is the commonest-False
   B. Papillary carcinoma is the commonest-True
   C. Bone metastasis may occur-True
   D. Is slow growing-False

114. Toxic goiter
   A. Patient has good appetite-True
   B. Patient gains weight rapidly-False
   C. Lid lag is not a feature-False
   D. Proximal myopathy is a feature-True

115. Goiter
   A. Is more common in males-False

B. Compression signs include hoarseness of voice-True
C. Moves with protrusion of the tongue-False
D. Moves with swallowing-True

116. Toxic goiter
A. Lid lag is a feature-True
B. Lid retraction is a feature- True
C. Exophthalmos is a feature in secondary thyrotoxicosis-False
D. Anxiety state is a feature-True

117. In thyroid function
A. Coupling stage in thyroid hormone synthesis is catalyzed by deiodinase enzyme-False
B. T4 is metabolically more active than T3-False
C. Thyroid hormone lack in children affects physical growth and mental development-True
D. TSH, T3, T4 and radioisotope thyroid scan are useful indices for thyroid gland activity-True

118. Examination of non-toxic goiter:
A. Includes asking the patient to swallow on inspection-True
B. Palpation is best done from the front-False
C. Palpation is best done behind-True
D. Determining lower edges helps to check for retrosternal goiter-True

119. Patient who presents with goiter causing tracheal obstruction
A. Stridor is a presenting feature-True
B. Dyspnea is a presenting feature-True
C. Kocher's test is usually negative- False
D. Kocher's test is usually positive-True

120. Toxic goiter
A. Exophthalmos is not a feature-False
B. Lid lag is a feature-True
C. Hot and moist skin and palms is a feature-True
D. Rapid pulse rate is not present during sleep- False

121. Secondary thyrotoxicosis
   A. Patients are younger than those with primary thyrotoxicosis-False
   B. Eye signs (exophthalmos, lid lag) are absent-True
   C. Atrial fibrillation is a feature-True
   D. Dyspnea is not a clinical feature-False

122. Paraplegia can be caused by
   A. Stab cervical injury-True
   B. Fracture dislocation of thoracolumbar spine-True
   C. Spinal tuberculosis-True
   D. Schistosomiasis-True

123. Odontoid fracture
   A. Is likely to involve C1-False
   B. Is likely to involve C2-True
   C. Type I fracture is stable-True
   D. Type III fracture is stable-False

124. Cervical spinal fracture
   A. Fracture at C4/C5 level spares phrenic nerve-False
   B. Hangman's fracture refers to the fractures at C5/C6-False
   C. During hanging there is spinal hyperextension and distribution by body weight-True
   D. Open mouth X-ray is called town view-False

125. Regarding iodine metabolism
   A. Uptake is solely by thyroid gland-True
   B. Daily iodine requirement is 100micrograms-True
   C. Iodine is trapped as iodide-True
   D. Iodine is organified by carboxylase enzyme-False

126. Regarding thyroid hormone synthesis

A. Coupling precedes organification-False
B. T3 and T4 are stored in the follicle bound to thyroglobulin molecule-True
C. T4 is physiologically more active than T3-False
D. Serum T3 is 100-160ng/100ml and T4 60-140m/L-False

127. Carcinoma of the thyroid
A. Vascular and capsular invasion is diagnostic of follicular carcinoma-True
B. Psammoma bodies and orphan Annie eyed nuclei are diagnostic of medullary carcinoma-False
C. Follicular carcinoma metastases is mainly by blood stream-True
D. Anaplastic carcinoma has the worst prognosis-True

128. Medullary carcinoma
A. Arises from parafollicular "P" cells-False
B. Tumor may be sporadic or familial-True
C. Pituitary adenoma, parathyroid adenoma and pancreatic adenoma associated with medullary carcinoma constitutes MEN type I syndrome-False
D. Tumors in (C) above constitutes MEN type II syndrome-False

129. Parathyroid adenoma, pheochromocytoma, medullary carcinoma and mucocutaneous vacuromas constitutes:
A. Sipple syndrome-True
B. Marfan syndrome-False
C. Hashimoto thyroiditis-False
D. None of the above-False

130. Tumors of the thyroid gland include
A. Lymphoma-True
B. Follicular carcinoma-True
C. Anaplastic carcinoma-True
D. Warthin's tumor-False
E. Giant cell tumor-False

131. Cervical injuries
A. Are commonly associated with head injuries-True
B. X-ray of the cervical spine down to C6 is adequate-False
C. An anteroposterior X-ray is more informative than a lateral view-True

D. Injuries at cord level C5 will cause respiratory arrest-False
E. During radiological evaluation the shoulders are pulled down caudally-False

132. Cervical spinal injury
A. Is common in young children-False
B. Odontoid peg is the commonest site of injury-False
C. CT scan is required in all cases for diagnosis-False
D. The phrenic nerve is involved if the injury is below C5-False
E. Is common in the head injured comatose patient-True

133. Carcinoma of the thyroid
A. A common type is papillary carcinoma-True
B. Medullary carcinoma is associated with pheochromocytoma-True
C. Follicular carcinoma secretes calcitonin-False
D. Cold nodules on thyroid scan are associated with malignancy-True
E. Is seen following irradiation in childhood-True

134. Patients with spinal cord injury develop
A. Bronchopneumonia-True
B. Pressure sores-True
C. Pyelonephritis-True
D. Amyloidosis-False
E. Priapism-True

135. Post thyroidectomy complications include
A. Airway obstruction due to tracheomalacia-True
B. Recurrent laryngeal nerve palsy-True
C. External laryngeal nerve palsy-False
D. Hypercalcemia-False
E. Voice change-True

136. The differential diagnosis of mid-line tumors in the neck includes:
A. Dermoid cysts-True
B. Thyroglossal cyst-True
C. Goiter-True

D. Bronchial cyst-True
E. Pharyngeal pouch-True

137. Pathological fractures may be associated with
A. Scurvy-True
B. Hyperparathyroidism-True
C. Bone cysts-True
D. All of the above-True

138. The most frequent offending agent in osteomyelitis of the spine is:
A. Streptococcus
B. Salmonella
C. Tubercle bacillus
D. Staphylococcus-True

139. The following sites are associated with non-union
E. Colle's fracture
F. Carpal navicular fracture
G. Patella fracture
H. Clavicle fracture

140. A 3-year-old child with a femoral fracture should be immobilized by:
E. Skeletal traction
F. POP splint
G. Intramedullary nailing
H. Bryant's traction

141. The following factors favor non-unions after fracture:
E. Muscle interposition
F. Hematoma at fracture site
G. Poor blood supply
H. Infection

142. The following statements are true of Perkins traction
E. It is a form of fixed skeletal traction

F. It can be used both in children and adults
G. It was invented by Prof Sir George Perkins
H. All above correct except “C”

143. Perkins’s exercises:
E. Are commenced 2 weeks after application of traction kit
F. Are commenced within a week after application of traction kit
G. Are commenced after 3 weeks when pain at fracture site has subsided
H. All above correct except “B”

144. Perkins exercises:
E. Ensure accelerated femoral fracture union
F. Delay femoral fracture union due to mobility while performing exercise
G. Are designed to accelerate femoral fracture union and forestall knee stiffness
H. All above correct except “A”

145. Regarding fracture classification
E. Garden classification refers to fracture neck of femur
F. Salter-Harris classification refers to epiphyseal growth plate fractures
G. Le forte classification refers to tarsal fractures
H. All above correct

146. Ankle joint mortice
E. Mortice formed by medial and lateral malleolus only
F. Mortice formed by medial and lateral malleolus and tibio-fibular syndesmosis
G. Mortice formed by (b) above and articular surface of tibia
H. Only (b) above correct

147. Weber “C” ankle fracture:
E. Is fracture below tibio-fibular syndesmosis
F. Is fracture at level of tibio-fibular syndesmosis
G. Is fracture above tibio-fibular syndesmosis
H. Any fracture at ankle joint

148. Torn anterior cruciate ligament diagnosis, the following test are positive:
E. Anterior drawer test

F. Mc-Murray test
G. Lachman test
H. Pivot shift

149. Torn posterior cruciate ligament diagnosis, the following tests are positive:
E. Lachman test
F. Apley's grinding test
G. Posterior drawer test
H. All of the above correct

150. In classical degenerative osteoarthritis, the following are noted:
E. Osteophytes (lipping)
F. Narrowing joint space
G. Subchondral bone sclerosis
H. Subchondral bone cysts

151. Total hip replacement involves
E. Replacement of femoral head and acetabular cup
F. Replacement of femoral shaft with artificial implant
G. Both hips replacement with custom made implants
H. Replacement of only acetabular cup

152. Anterior shoulder dislocation may be reduced by
E. Kocher's maneuver
F. Hippocratic method
G. Stimson's technique
H. Socrate's method

153. The following factors favor non-union of a fracture
E. Soft tissue inter-position
F. Hematoma at fracture site
G. Poor blood supply
H. When there are two bones if the other bone is intact

154. Salter-Harris type II epiphyseal fractures involves
E. It is a "T" shaped fracture extending into the proximal joint and metaphysis

F. It extends into metaphysis with a triangular component
G. It extends into proximal joint with partially crushed epiphysis
H. None of the above

155. In posterior hip dislocation
E. Dislocation puts sciatic nerve at risk of injury
F. Reduction is achieved under general anesthesia with patient on the floor
G. Skin or skeletal traction should be applied after successful reduction
H. All above correct except “C”

156. In shoulder dislocation
E. Posterior dislocation is commoner than anterior dislocation
F. Axillary nerve is at risk of injury
G. Kocher’s manoeuver may be employed to reduce dislocation
H. All above are correct except “C”

157. In elbow dislocation
E. Anterior dislocation describes humerus lying anterior to radius.
F. Posterior dislocation describes humerus lying posterior to radius
G. The forearm bones provide key to description of the type of dislocation
H. None of above correct

158. In lower limb deformities
E. Genu valgus refers to bow legs
F. Genu varus refers to knock knees
G. Foot drop refers to injury of common fibular nerve
H. All above are correct

159. Secondary metastases to the spine can originate from cancer of
E. Thyroid
F. Breast
G. Lung
H. Prostate

160. Collar and Cuff sling
E. Is an example of external splint

F. Is suitable for displaced humeral fracture
G. Can be used to immobilize femoral fractures in children
H. The flexion angle at the elbow is less than 90 degrees

161. Intramedullary nail is
E. External fixation
F. Internal fixation
G. Suitable for fracture of the femoral shaft
H. Also known as Kuntscher nail

162. The following tumors readily metastasize to bone
F. Rectal adenocarcinoma
G. Basal cell carcinoma of skin
H. Follicular carcinoma of the thyroid
I. Prostatic adenocarcinoma
J. Renal adenocarcinoma

163. Dislocations
F. Do not require urgent management
G. Post-reduction immobilization is required for 3 months
H. The medial nerve is damaged in shoulder dislocations
I. The ulnar nerve is damaged in lunate dislocations
J. All open dislocations require operative reduction

164. Osteoarthritis of the knee joint is a complication of
F. Hemophilia
G. A malunited femoral fracture
H. Total menisectomy
I. Valgus deformity of the knee
J. Gross obesity

165. Supracondylar fractures of the humerus
F. Always check the radial artery pulse
G. Always check nail bed capillary filling in absence of the radial pulse
H. Extension improves limb circulation
I. Nerve damage is uncommon

J. Non-union is uncommon

166. The following fractures commonly lead to ischemic necrosis of a part of the bone
F. Femoral neck
G. Humeral neck
H. Scaphoid
I. Talus neck
J. Femoral condyle

167. The following fractures lead to nerve damage in some patients:
F. Femoral neck
G. Neck of fibula
H. Medial epicondyle of humerus
I. Radial shaft
J. Humeral shaft

168. Fracture of the pelvis:
F. A stable fracture requires 3 months bed rest
G. A displaced sacroiliac joint requires skeletal traction
H. Blood transfusion is essential in extensive pelvic fractures
I. Isolated undisplaced iliac bone fractures do not require immobilization
J. Fractures of the pubic rami are relatively common

169. The following are flexors of the wrist joint:
F. Brachioradialis
G. Flexor carpi ulnaris
H. Pronator teres
I. Flexor digitorum profundus
J. Flexor pollicis brevis

170. Osteosarcoma is treated by
F. Radiotherapy
G. Chemotherapy and surgery
H. Immunotherapy

I. Antibiotics
J. Vitamin D

171. The following laboratory tests are useful in evaluating diseases related to bones
F. Serum parathyroid hormone
G. C-reactive protein
H. Urinalysis for specific proteins
I. Fasting blood glucose
J. Serum uric acid levels

172. Osteoporosis
F. Is common in premenopausal women
G. Is treated with estrogens
H. Causes pathological fractures
I. Erythrocyte sedimentation rate is elevated
J. Once established cannot be reversed

173. Primary osteoarthritis
F. Starts as a disorder of articular cartilage
G. Results in periarticular osteoporosis
H. Is associated with peripheral vascular disease
I. Occurs more commonly in females
J. Is common in Zambia

174. Dupuytren's contractures:
F. Causes extension deformity of the wrist
G. Is familial
H. The foot can be involved occasionally
I. Is treated by steroids
J. Trauma is an etiologic factor

175. Carpal Tunnel syndrome
F. Occurs in middle aged men only
G. Symptoms are related to thumb only
H. Does not cause muscle wasting
I. Surgery is indicated

J. Symptoms are worse at night

176. Septic arthritis is best managed by
F. Aspiration of the joint
G. Antibiotics
H. Rest and elevation
I. Gypsona immobilization
J. Arthrotomy, antibiotics and rest for the joint

177. Acute osteomyelitis
F. Brodie's abscess is a variety of acute osteomyelitis
G. A common causative organism is Staphylococcus aureus
H. Bone drilling must be done early to prevent further damage
I. X-rays do not show changes in the first week
J. Is common in Zambian children

178. Tuberculosis of the spine
F. Is common in Zambia
G. Usually affects the vertebral bodies
H. Can be adequately treated by anti-tuberculous drugs alone
I. Never produces sequestra
J. Is not diagnosed unless a paraplegia is present

179. A 3-year-old child develops pain in the hip joint, is pyrexial and has a high erythrocyte sedimentation rate:
F. Treat by traction alone
G. Treat by antibiotics alone
H. Blood culture are to be done
I. Explore the hip joint surgically
J. Take a careful history from the mother and do a full physical examination at admission

180. Poliomyelitis
F. Anterior horn cells are affected
G. Flaccid paralysis develops
H. Joint contracture may follow muscle imbalance

I. Tendon division may improve function
J. Spread is by nasopharyngeal droplets

181. The following statements are true or false
F. The radial nerve is injured in humeral fractures
G. The ulnar nerve is injured in dislocated shoulder
H. A Colles fractures commonly injures the median nerve
I. Wrist drop occurs with median nerve palsy
J. Circumflex nerve injury causes inability to adduct the shoulder

182. Acute osteomyelitis of the tibia
F. Not to be drained unless bone changes are present on X-ray
G. Staphylococcus aureus is the causative organism in all cases
H. Requires immediate drainage
I. X-Ray changes are visible within a few days of onset of infection
J. Antibiotics are not indicated

183. Ulnar nerve palsy
F. Occurs in fracture of the lateral epicondyle of the humerus
G. Results in wasting of thenar muscles
H. The little finger cannot be abducted against resistance
I. The ulnar side of the hand becomes dry (no sweating)
J. Dermatome involved is C5

184. Osteoporosis
F. Is a hereditary disease
G. Is same as Osteomalacia
H. Is curable
I. Is common in young premenopausal women
J. Responds to vitamin A

185. The following nerves are injured in a dislocation shoulder
F. Medial
G. Circumflex
H. Axillary
I. Suprascapular
J. The nerve which supplies the deltoid muscle

186. Concerning the menisci of the knee:
F. Injuries are common in elderly people
G. A tear is associated with osteoarthritis of the knee
H. Twists at the knee is the usual cause of meniscal tears
I. Injuries to the lateral one is commoner
J. One function is to improve lubrication

187. A 39-year-old male with a suspected intervertebral disc prolapse suddenly develops urinary retention. The following measures should be taken
F. Urgent radiculogram
G. Catheterisation
H. Urgent exploration of spine
I. Bed rest and skeletal traction
J. Send to the next urology clinic

188. A 4-year-old child develops pain in the hip joint with restriction of all movements, pyrexia, high erythrocyte sedimentation rate and a high white blood cell count
F. Treat with skin traction alone
G. Give aspirin and bed rest
H. Take blood cultures
I. Explore the hip surgically
J. Is commonly associated with trauma

189. Osteoarthritis has the following features:
F. Occurs in the hand bones
G. Is age related commonly
H. Often follows joint injury
I. Is commoner in males
J. Results in periarticular osteoporosis

190. Sickle cell disease
F. Is hereditary
G. Is due to an abnormal hemoglobin

H. Femoral epiphyseal infarction is a complication
I. Steroids are indicated in all cases
J. Is associated with osteomyelitis

191. Following a nerve injury
F. Neuropraxia has the best prognosis for recovery
G. Neurotmesis has the worst prognosis for recovery
H. Axonotemesis describes complete transection of the nerve
I. Axons regenerate at the rate of 1 mm per day
J. Axons regenerate at the rate of 1cm per day

192. The following fractures occur at the wrist
F. Colles
G. Barton's
H. Smiths
I. Pott's
J. De Quervain's

193. The following fractures usually require open reduction and internal fixation
F. Oblique shaft fractures of the humerus
G. Colles fracture
H. Greenstick forearm fractures in children
I. A displaced fracture of the lateral condyle of the elbow in a 10-year-old
J. A subcapital femoral fracture in an elderly female

194. A fracture of the carpal scaphoid
F. Is frequently caused by a fall on the outstretched hand
G. Usually occurs through the waist of the scaphoid
H. Avascular necrosis of the distal fragment is a possibility
I. Is commoner in the younger patient than the old
J. Non-union is possible

195. In spinal Tuberculosis
F. Infection starts in bone adjacent to a disc
G. New bone formation is minimal in active infection
H. Radiographs usually show destruction of the body of a vertebra without involvement of the disc

I. Psoas abscess is a complication
J. Paraplegia is a complication

196. Poliomyelitis
F. Is caused by damage to anterior horn cells
G. Leg length inequality is a possibility
H. Short rigid scoliosis is usually associated with it
I. Foot neuropathic ulcers are common
J. Osteoarthritis is a common feature of the affected limb joints

197. The following muscles in the hand are supplied by the median nerve
F. Abductor pollicis brevis
G. The first dorsal interosseous muscle
H. Adductor pollicis
I. Abductor digiti minimi
J. Opponens pollicis

198. In chronic osteomyelitis of the femur
F. New bone outside the cortex is the involucrum
G. Sequestrum is more radiopaque than normal bone
H. A loose dead bone is called sequela
I. Cloacae drain pus through the cortex
J. Long standing sinuses heal with antibiotics

199. The early clinical signs and symptoms of ischemia in the forearm and hand following closed reduction of a supracondylar fracture are
F. Paralysis of median nerve
G. Gangrene of tips of fingers
H. Fixed contracture of the flexor muscles
I. Pain, especially on extension of the fingers
J. Absent radial pulse and poor capillary return

200. The following are complications of a Colles fracture at the wrist
F. Rupute of the extensor pollicis longus
G. Frequent non-union
H. Median nerve compression
I. Malunion

J. Inferior radioulnar subluxation

201. Classification of fractures
F. Radiologically as open or closed fractures
G. Clinically as compound fracture or closed fracture
H. Comminuted fracture when more than 2 fragments are present
I. Pathological fractures when fracture is due to tumor deposit
J. Supracondylar fracture refers to anatomical classification

202. Concerning fractures of the neck of femur
F. They are more common in elderly men
G. They are usually caused by high energy trauma in the elderly
H. Avascular necrosis of the head of the femur is common complication
I. If treated conservatively they have a higher incidence on non-union
J. They are best managed by skeletal traction

203. Femoral shaft fractures
F. Are associated with an average blood loss of 1000-1500ml
G. In the young patient are usually associated with other life-threatening injuries
H. Are usually adequately treated with traction
I. Are commonly complicated by compartment syndrome
J. Are commonly associated with shortening of the affected limb

204. Regarding Perkin's traction
F. The usual weight applied is 1/10 of the patient's body weight
G. Leg lengths are measured once weekly for the first 2 weeks after application
H. Exercises are best commenced within the first 4 days of applying traction
I. The foot end of the bed is normally elevated by 4cm for every Kg of weight
J. A Denham pin may be used for applying the traction

205. Concerning open fractures
F. They are more common in the tibia than in any other long bone
G. They cannot be complicated by compartment syndrome
H. They are commonly associated with delayed union and non-union in the tibia
I. X-rays are not necessary
J. Are best treated with an external fixator

206. The following displacements are characteristic of Colles' fracture
   F. Volar (anterior) displacement of the distal fragment
   G. Apex volar angulation
   H. Ulnar deviation of the distal fragment
   I. Impaction
   J. 'Dinner fork' deformity

207. Supracondylar fractures of the humerus in children
   F. Are usually of the flexion type
   G. Commonly occur after a fall on an outstretched hand
   H. Are partly caused by the olecranon being forced into the olecranon fossa
   I. Can be complicated by myositis ossificans
   J. If presenting with extensive swelling should be manipulated and immobilized in a collar and cuff

208. Concerning fracture healing
   F. Hematoma formation at the fracture site does not influence the rate of fracture healing
   G. It is affected by the nutritional status
   H. Upper limb fractures in children take about 3 weeks to heal
   I. It is influenced by the type of bone involved
   J. It is affected by the hemoglobin level of the patient

209. Concerning acute hematogenous osteomyelitis
   F. It is commonly caused by Salmonella Spp
   G. Organism normally settle in the metaphysis of long bones
   H. In infants it may lead to septic arthritis
   I. Radiological signs are present within 24 hours of the infection
   J. It is best managed conservatively

210. Differential diagnoses of acute osteomyelitis include:
   F. Fracture
   G. Sickle cell crisis
   H. Rheumatic fever

I. Cellulitis
J. Septic arthritis

211. The criteria for diagnosis of septic arthritis in patients with negative cultures (according to Morrey and Associates) include:
F. Pain in suspected joint worsened by movement
G. Systemic symptoms
H. Poor response to antibiotic therapy
I. Swelling of suspected joint
J. Fever of more than 38.3 degrees Celsius

212. Fracture of clavicle
F. Is usually compound
G. Is best treated by open internal fixation
H. Is usually associated with blood vessel injury
I. Is treated by POP immobilization
J. Can be treated by long arm sling alone

213. In Tuberculosis of bone
F. The local reaction is characterized by extensive new bone formation
G. The metaphysis of a bone is the commonest site of involvement
H. The infection is usually secondary to primary focus elsewhere in the body
I. Extension of the bone abscess into a joint is common
J. Associated abscesses show few signs of inflammation

214. Features occurring in children differ from those in adults in the following respects
F. Fractures unite readily
G. Malunion can be corrected by growth
H. Joint stiffness is rare after immobilization
I. Fractures involve the epiphyseal plate on occasion
J. Fractures usually occur with minimal trauma

215. In fracture of the shaft of the adult femur
F. The estimated blood loss is at least 1 liter
G. Skeletal traction and Perkin's exercises are effective treatment
H. Healing is faster in Zambians than in Europeans

I. Internal fixation allows earlier mobilization
J. Internal fixation is the treatment of choice in Zambia

216. Carpal tunnel syndrome
F. Is most frequent in middle aged men
G. Symptoms are related to all fingers and the thumb
H. Does not cause muscle wasting
I. Surgical treatment is usually curative
J. The symptoms are worse at night

217. Dislocation of the hip
F. Common variety is a posterior dislocation
G. Delay in reduction does not increase the risk of late complications
H. Is overlooked if there is an associated fracture of the shaft of femur
I. Leads to dorsiflexion of the foot and great toes
J. Sciatic nerve injury occurs in 50% of patients

218. The following fractures are rarely adequately treated by closed manipulation:
F. Bennet's fracture
G. Colles fracture
H. Phalages of digits
I. Monteggia
J. Tibial condyle

219. Internal hemorrhoids are arranged in the following manner with the patient in lithotomy:
A. 3, 7 and 11 O'clock-True
B. 3, 7 and 9 O'clock-False
C. 7, 9 and 11 O'clock- False
D. 3, 9 and 11 O'clock-False

220. GoodSall's rule is related to
A. Anal fissure-False
B. Anal fistula-True
C. Hemorrhoids-False
D. Anal abscesses-False

221. Common causes for rectal bleeding
   A. Internal hemorrhoids-True
   B. Anal fissure-True
   C. Carcinoma rectum-True
   D. All correct except (B)-False

222. Concerning piles
   A. First degree piles bleed but do not prolapse-True
   B. Second degree piles prolapse with defecation and reduce spontaneously-True
   C. Third degree piles are thrombosed piles whether internal or external-False
   D. None above are correct-False

223. Treatment for first degree piles
   A. Hemorrhoidectomy-False
   B. High fiber diet-True
   C. Suppositories-True
   D. All above correct-False

224. Hemorrhoids
   A. Always cause pain and bleeding-False
   B. Sometimes cause pain and bleeding-True
   C. Are always treated by operation-False
   D. Can be due to increased intra-abdominal pressure-True
   E. Are noted in pregnancy-True

225. The adult anus
   A. Is 1cm long-False
   B. Is covered by serosal lining-False
   C. Blood supply is from 3 rectal arteries-True
   D. Has 2 main parts meeting at perineal line-False
   E. Inferior half lymphatics drainage is to the superficial inguinal nodes-False

226. Thrombosed hemorrhoids
   A. Are treated by hemorrhoidectomy-True
   B. Can be commonly seen at three sites: 4, 7 and 11 O'clock-False
   C. Are due to arterial anastomosis-True
   D. Usually have a sentinel pile-True
   E. Common in diabetic patients-True

227. Digital rectal examination is diagnostic in
   A. First degree hemorrhoids-True
   B. Low carcinoma of the rectum-True
   C. Fissure in ano-True
   D. Acute cystitis-False
   E. Hirschprung's disease-True

228. Axillary tail of the breast lies in
   A. Upper outer quadrant-True
   B. Upper inner quadrant-False
   C. Lower outer quadrant-False
   D. Lower inner quadrant-False

229. The breast tissue is located
   A. Within subcutaneous layer-False
   B. Within superficial fascia between upper and lower layers-True
   C. Rests directly on pectoral fascia-False
   D. Only "A" above is correct-False

230. Fibroadenoma of breast
   A. Commonest benign tumor-True
   B. It may be peri or intra canalicular-True
   C. It lacks a definite capsule-True
   D. Recurrence after excision is rare-True

231. Blood stained discharged from nipple may represent
   A. Duct papilloma-True

B. Pagets disease of nipple-True
C. Galactocele-False
D. Only “B” and “C” correct-False

232. The following breast tumors may undergo malignant transformation
A. Phylloides tumor-True
B. Multiple duct papillomatosis-True
C. Intracanalicular fibroadenoma-True
D. All above-True

233. Change of breast may also be named as
A. ANDI-True
B. Benign mammary dysplasia-True
C. Fibroadenosis-True
D. Chronic mastitis-True

234. Reference to ANDI of breast, histological features include:
A. Adenosis-True
B. Epitheliosis-True
C. Duct ectasia-False
D. Fibrosis-True

235. In Lactational mastitis
A. Cracked nipple permits entry of bacteria-True
B. Staphylococcus aureus is the main pathogen-True
C. There is good response to broad spectrum antibiotics-True
D. May be complicated by breast abscess-True

236. The following cannot affect breast tissue
A. TB-False
B. Syphilis-False
C. Fungal infection-False
D. All above-False

237. Triple assessment of breast pathology
   A. Is only relevant to malignant conditions-False
   B. Involves taking history and performing clinical examinations only-False
   C. Involves as well U/S scan for females above 35 years old and mammogram for females below 35 years of age-False
   D. Fine needle aspiration cytology (FNAC) is preferred to incisional or excisional biopsy snice it is specific and easy to interpret- True

238. Pagets disease of the nipple
   A. Usually presents as bilateral nipple eczema-False
   B. Unilateral nipple eczema-True
   C. Related to underlying duct carcinoma-True
   D. All above true except (B)-False

239. Fibroadenoma of breast
   A. It may present as mobile, painless mass-True
   B. Has ill-defined edges-False
   C. It may be intra or peri-canalicular-True
   D. Recurrence common after excision-False

240. Fibrocystic breast disease presents as:
   A. Fibroadenosis-True
   B. Cystic formation-True
   C. Epitheliosis- True
   D. Fibrosis and lymphocytosis-True

241. The following drugs are used in breast cancer
   A. Cyclophosphamide-True
   B. 5-fluoro-uracil-True
   C. Methotrexate-True
   D. Adriamycin-True

242. The following features present advanced breast cancer
   A. Peau d'orange-True

B. Nipple retraction/ulceration/bleeding-True
C. Fixity to underlying pectoral muscle-True
D. Hepatomegaly-True

243. Phyllodes tumor on histology
A. Might be benign-True
B. Might be borderline-True
C. Might be frankly malignant-True
D. Cellular stroma is characteristic-True

244. Breast tissue lies
A. Within retromandibular space-False
B. Deep to pectoral fascia-False
C. Within superficial fascia-True
D. May extend to the axilla-True

245. Breast cancer
A. Carcinoma in-situ tumor has not yet breached basement membrane-True
B. Lobular carcinoma tends to be multicentric in origin more than ductal carcinoma-True
C. Ductal carcinoma tends to be multicentric in origin more than lobular carcinoma-False
D. None of the above-False

246. Breast cancer markers/receptors include:
A. ER/PR-True
B. HER-2/NEU-True
C. Tamoxifen is useful in ER positive receptors-True
D. Trastuzumab (Herceptin) is useful in advanced breast cancer that are HER-2/NEU receptor negative-False

247. Supraradical mastectomy involves
A. Mastectomy, axillary node clearance and excision of pectoral muscles-True
B. The clavicle and sternum were spared-True
C. Procedure abandoned due to post op arm edema and weak upper limb-True
D. Operation is now obsolete-True

248. Radical mastectomy
   A. Improves on supraradical mastectomy-False
   B. It spared clavicle and sternum-True
   C. Pectoralis major was spared-False
   D. Also referred to as complete Halsted-True

249. Patey's mastectomy
   A. Improved on radical mastectomy-True
   B. It spares pectoral major muscles-True
   C. Axillary and pectoral nodes clearance done-True
   D. Power limb was an advantage-True

250. Lymph node involvement in breast cancer includes the following
   A. Axillary lymph nodes-True
   B. Supraclavicular lymph nodes-True
   C. Cervical lymph nodes-True
   D. Inguinal lymph nodes-False

251. Triple assessment is used in making a diagnosis of breast disease and consists of the following:
   A. Clinical examination, imaging, and fine needle aspiration cytology-True
   B. Triple imaging-ultrasound, chest X-ray, mammography-False
   C. Triple diagnosis- mammography, radioisotope studies and CAT-scan-False
   D. Nipple cytology, thermography and MRI-False

252. A breast lump in an adolescent can be one of the following
   A. Fibroadenoma-True
   B. Giant fibroadenoma-True
   C. Galactocele-False
   D. Dysgerminoma-False
   E. Hurtle cell tumor-False

253. An enlarged prostate
   A. Can be determined by digital rectal examination-True
   B. Can be identified by ultrasound evaluation-True
   C. Doses not cause acute urinary retention-False
   D. Prostatic specific antigen is an indicator of malignant disease in the prostate-True
   E. Acid phosphatase is a specific test for prostatic cancer-True

254. Prostatectomy is performed using one of the following approaches
   A. Suprapubic transvesical-True
   B. Retropubic (Millin's)-True
   C. Transurethral-True
   D. Perineal-True
   E. Pfannenstial incision-False

255. Complication of prostatectomy include
   A. Excessive bleeding-True
   B. Inflammation of the vas deferens-True
   C. Bladder neck stricture-True
   D. Gastric atony-False
   E. Subphrenic abscess-False

256. Cancer of the prostate
   A. Is common in the elderly-True
   B. Is often asymptomatic-True
   C. Treatment of choice is prostatectomy-True
   D. Prostate specific antigen is a reliable guide to prognosis-True
   E. Metastases are always osteolytic-False

257. Fibroadenoma of the breast
   A. Occurs in early adult life-True
   B. May be multiple-True
   C. Adheres to the pectoralis major-False
   D. Is painful-False

E. Disappears spontaneously-False

258. In hypernatremia
A. Fever and edema are absent-True
B. Restlessness and raised BP are present-True
C. There is increased urine output-True
D. Only C is correct-False

259. In hyperkalemia patient will present with:
A. Increased reflexes and ECG changes-False
B. ECG shows inverted "T" waves-False
C. ECG shows peaked "T" waves-True
D. Oliguria, muscle weakness and respiratory distress are a feature-True

260. Treatment of hyperkalemia include:
A. Kayexalate-True
B. IV insulin and glucose-True
C. Lasix and stop potassium containing fluid-True
D. Dialysis if severe-True

261. In respiratory alkalosis
A. PCO_2 rises-False
B. PCO_2 falls-True
C. PCO_2 remains the same-False
D. None above are correct-False

262. In metabolic acidosis
A. CO_2 combining power rises-False
B. CO_2 combining power falls-True
C. CO_2 combining power remains the same-False
D. Only C is correct-False

263. Under normal conditions the colloid osmotic pressure of blood is about
A. 75mmHg-False
B. 50mmHg-False
C. 25mmHg-True

D. 10mmHg-False

264. Signs and symptoms of hyperkalemia include
A. Peaked “T” waves on ECG-True
B. Increased cardiac contractility-False
C. Muscle weakness-True
D. Respiratory distress-True

265. Treatment of hypernatremia include:
A. Stop IV normal saline-True
B. Glucose and insulin intravenously-False
C. Lasix-True
D. Dialysis if severe-True

266. Administration of potassium to correct hypokalemia
A. Give potassium as intravenous bolus-False
B. Not more than 20mEq with 40mEq as maximum-True
C. Ensure good urine output-True
D. Cardiac monitor not essential-False

267. The following intravenous solutions are commonly used to treat hemorrhagic shock in the University Teaching Hospital
A. 0.9% saline-True
B. 5% dextrose-False
C. Ringer’s lactate-True
D. Hypertonic saline-False
E. Darrow’s solution-False

268. Septic shock
A. Is particularly associated with Celestin tube insertion-False
B. Is often due to gram negative organisms-True
C. Is particularly seen in patients with indwelling intravenous catheters-True
D. Surgery for acute cholecystitis is a cause-False
E. Does not occur in gram positive septicemia-False

269. The following intravenous fluids contain potassium
   A. Darrow's-True
   B. Ringer's-True
   C. Blood-True
   D. Dextrose 5%-False
   E. Dextran solution-False

270. Patients needing supplementary feeds include those with
   A. Recent weight gain-False
   B. Serum albumin >30g/dl-False
   C. Serum albumin <30g/dl-True
   D. Negative nitrogen balance-True
   E. Positive nitrogen balance-False

271. Parenteral feeds
   A. Best feeding method-False
   B. Has least side effects-False
   C. Requires feeding nasogastric tube-False
   D. Very expensive-True
   E. May be the only alternative in conditions like acute pancreatitis-True

272. Body fluids
   A. A 70kg man has 42L as total body water-True
   B. Interstitial fluids in a 70kg man is 11 Liters-True
   C. 3 L of plasma in a 60kg man-False
   D. 15L in a 60kg man is extracellular fluid-False
   E. Potassium is an extracellular ion-False

273. Pancreatitis
   A. Can be the cause of chemical peritonitis-True
   B. Absence of bowel sounds is a feature-True
   C. Serum amylase is not raised-False
   D. Laparotomy is indicated in all cases-False

274. Complications of acute pancreatitis are
   A. Pseudocyst formation-True
   B. Disseminated intravascular coagulation-True
   C. Left basal effusion (pleural)-True
   D. Renal failure-True
   E. Ascites-True

275. Gall stones cause
   A. Acute cholecystitis-True
   B. Acalculous cholecystitis-False
   C. Obstructive jaundice-True
   D. Carcinoma of the gall bladder-True
   E. Torsion of the gall bladder-True

276. Acute cholecystitis
   A. Does not occur in sicklers-False
   B. Is usually treated by cholecystostomy-True
   C. Is usually associated with urinary bladder stones-False
   D. Can cause pain in the right shoulder-True
   E. Causes pain and tenderness in the left hypochondrium-False

277. Acute pancreatitis
   A. Often results in hypoxemia-True
   B. Usually gives rise to Cullen's sign-True
   C. Causes hypercalcemia-False
   D. Can be caused by mumps-True
   E. Occurs after biliary surgery sometimes-True

278. Esophagus
   A. Below the pulmonary roots the vagus nerves are in contact with esophagus-True
   B. The left trunk of the vagus nerve is on the anterior side of the esophagus -True
   C. The right trunk of the vagus nerve is on the posterior side of the esophagus-True

D. The longest part of esophagus is intra-abdominal-False

279. Cancer of the esophagus
A. Dysphagia is initially for solids-True
B. Dysphagia is initially for liquids-False
C. Barium swallow X-ray shows a smooth constriction giving a rat-tail appearance-False
D. Barium swallow X-ray shows irregular and sharp edge constriction giving shouldering appearance-True

280. Signs of advanced cancer of the esophagus include:
A. Hoarseness of voice as a sign of involvement of recurrent laryngeal nerve-True
B. Weight loss of more than 20%-True
C. Absence of palpable supraclavicular lymph nodes-False
D. Presence of palpable lymph nodes-True

281. Esophageal stricture
A. Can be caused by achalasia of the cardia-False
B. Inflammation of esophagus does not cause strictures-False
C. Can be caused by cancer of esophagus-True
D. Can be caused by ingestion of corrosive chemicals-True

282. Cancer esophagus presents with
A. Weight gain-False
B. Dysphagia of solids-True
C. Odynophagia- True
D. Regurgitation is not a feature-False

283. Carcinoma of the esophagus is commonly treated by
A. Surgery-True
B. Radiotherapy-True
C. Immunotherapy-False
D. Chemotherapy-True
E. Gastrostomy-False

284. Carcinoma of the esophagus
   A. Lower third is a common site-True
   B. Is adenocarcicnoma in 90% of cases-False
   C. Is radiosensitive-True
   D. Metastases the scalene node-True
   E. Presents early in Zambia-False-False

285. Hepatocellular carcinoma
   A. Is the commonest tumor of men in Zambia-False
   B. Is not associated with post-necrotic cirrhosis-False
   C. Is related to ingestion of aflatoxins-True
   D. Presents with intermittent abdominal pain-True
   E. Rarely causes early weight loss-False

286. Incisional hernias are related to
   A. Wound infections-True
   B. Anemia and malnutrition-True
   C. Obesity-True
   D. The use of absorbable suture materials-True

287. The differential diagnosis of an inguinal hernia in a female include:
   A. Vaginal hydrocele-True
   B. Encysted hydrocele of the cord-False
   C. A femoral hernia-True
   D. None of the above-False

288. A strangulated hernia
   A. Is tense-True
   B. Is tender-True
   C. Has a transmitted cough impulse-False
   D. Requires elective surgery-False

289. A partial thickness burn
   A. May heal without grafting-True

B. May deteriorate into full thickness skin loss-True
C. Rarely causes severe physiological derangement of the patient-True
D. Heals within 7 days in the absence of infection-False

290. Burns
A. History taking should always include- Time of burn, cause, smoke inhalation, trauma- True
B. On examination the severity is determined by burn depth, burn size, areas of circumferential full thickness-True
C. Full thickness burns will produce very painful charred skin-False
D. Escharotomy may help in reducing respiratory embarrassment-True

291. In burns
A. A man with 30% superficial burns does not require fluid therapy-False
B. A female with 20% superficial burns does not require ATT on admission-False
C. Parkland's formula considers fluids therapy in the first 36 hours-False
D. Browder and Lund charts are used to estimate percentage surface area burnt-True

292. The skin
A. Is a multilayered organ-True
B. Good approximation of layers is not vital-False
C. Crush injury heals better than an incision-False
D. Langer's lines run perpendicular to muscle axis-True
E. Langer lines run mainly longitudinal to muscle axis-False

293. Upper gastrointestinal hemorrhage can be caused by:
E. Mallory-Weis tear
F. Sigmoid volvulus
G. Periportal fibrosis due to Bilharzia
H. Perforated anterior duodenal ulcer

294. Zollinger-Ellison syndrome
E. Multiple ulcerations are a feature
F. Increased vagal activity is the main cause
G. Fresh red blood rectal bleeding is a common feature

H. Gastrin producing endocrine tumor is a common feature

295. Pyloric stenosis in adult patient:
E. Annulus pancreas can be the cause
F. Chronic duodenal ulcer can be the cause
G. Meckel's diverticulum can be the cause
H. Cancer of pancreas can be the cause

296. In a patient with peptic ulcer disease
E. Histamine production is increased by stimuli from both vagus nerves and gastrin
F. The process of exporting hydrogen ions from parietal cells via protein pump is not a feature
G. Histamine acts on parietal cells via H2-receptors
H. Melena stools is not a feature

297. Investigating a patient who presents with hematemesis includes:
E. Esophagoscopy
F. Gastroduodenoscopy
G. Lower midline laparotomy is advisable
H. Right subcostal mini-laparatomy is advisable

298. Brain abscess in the frontal lobe
E. Is most likely a complication of chronic otitis media
F. Is not likely a complication of paranasal sinusitis
G. Is most likely a complication of infected occipital laceration
H. Is most likely a complication of infected wound on the temporal area of the head

299. Large supratentotrial space occupying lesions is likely to present with
E. Full consciousness
F. Unequal pupils
G. Contralateral hemiplegia

H. Paraplegia

300. Common causes of congenital hydrocephalus include:
   E. Brain abscess
   F. Dandy-walker cyst
   G. Arterial venous malformation
   H. Anold-Chiari malformation

301. Submandibular gland tumors
   E. 50% are malignant
   F. Facial nerve weakness is a feature
   G. Ulceration of overlying skin is not a feature
   H. Cervical lymph nodes enlargement is a feature

302. Parotid gland tumors
   E. Most common are benign pleomorphic adenomas
   F. 10% are malignant
   G. Rapid growth with infiltrating of overlying skin is a sign of high-grade malignancy
   H. Enlarged palpable cervical lymph nodes are not a feature

303. Bilateral paralysis of genioglossus leads to
   E. Atrophy of the tongue
   F. Tongue falls back
   G. Obstruction of respiratory passage
   H. All above true except “B”

304. Structures embedded in the parotid gland include
   E. Facial nerve
   F. Retromandibular vein
   G. External carotid artery
   H. Parotid lymph nodes

305. The following are examples of non-absorbable suture materials
   E. Silk
   F. Vicryl
   G. Nylon
   H. Stainless steel
306. The following suture are
   E. Silk is natural material
   F. Nylon is synthetic material
   G. Vicryl is natural material
   H. Stainless steel is synthetic material

307. Knot security after wound suturing
   E. Best with vicryl
   F. Good with nylon
   G. Worst with silk
   H. All above correct

308. Clostridium Welchii is
   E. Causative agent of wet gangrene
   F. Causative agent of gas gangrene
   G. Aerobe, sporulating, gram negative bacillus
   H. Proliferates in dirty wounds

309. In acute limb ischemia
   E. Thrombo-embolism may be a causative factor
   F. In-situ blockage of artery by thrombus is factor
   G. Buerger's test is positive
   H. Ankle brachial pressure index is useful indicator of severity of ischemia

310. Parotid benign tumors
   E. Make up 80 to 90% of parotid tumors
   F. Are pleomorphic adenomas
   G. Are soft on palpation
   H. Mostly arise in the superficial lobe

311. HIV-associated sialadenitis of parotid gland

E. Chronic parotitis in children is pathognomonic of HIV infection
F. Facial disfigurement is a feature
G. CT scan shows characteristics swiss cheese appearance of multiple cystic lesions
H. Cosmetic surgery is not advisable

312. Causes of leucopenia
E. Lymphoma
F. Sever infections
G. Disease of the spleen
H. Autoimmune disorders

313. Causes of leukocytosis
E. Inflammatory conditions like arthritis
F. Stress
G. Allergic conditions like asthma
H. Pregnancy

314. A rise in monocytes may indicate the following:
E. Chronic inflammatory disease
F. Parasitic infestation
G. Tuberculosis
H. Oral infection

315. Post-op fever after abdominal surgery
E. Is caused by release of pyrogens
F. Intra-abdominal collections
G. Blood and platelet infusions
H. Pulmonary complications like pneumonia or empyema

316. Unilateral ureteric obstruction may be caused by
E. Ureteropelvic stenosis
F. Ureteric stone
G. Posterior urethral valves
H. Neurogenic bladder

317. Unilateral renal artery obstruction may be associated with

E. Increased renin release by the obstructed kidney
F. Angiotensin release and increased aldosterone production by adrenal cortex
G. Increased sodium and water retention
H. Hypotension

318. Chronic bilharzia can cause
E. Bilateral ureteric obstruction
F. Transitional cell carcinoma
G. Bilateral hydronephrosis
H. Squamous cell carcinoma of the bladder

319. Management of obstructive uropathy may include
E. Catheterization
F. Bilateral orchidectomy
G. Endoscopic ureterotomy
H. Urethroplasty

320. Parotid inflammatory swelling
E. Can be caused by mumps
F. Is rare in children
G. Can be HIV associated
H. Does not cause facial disfigurement

321. Superficial parotidectomy
E. Is indicated for benign and low grade malignant tumors
F. Incision is lazy-“S”
G. Facial nerve injury is rare
H. Skin flap is developed to reach anterior border of the gland

322. Parotidectomy complications
E. Frey’s syndrome
F. Facial nerve palsy
G. Hemorrhage
H. Raised T3 and T4

323. Lung cancer

E. Small cell lung cancer makes up 20%
F. Metastasis by lymphatics is rare
G. Metastasis by blood borne spread is common
H. Surgery greatly improves survival

324. When considering fluid spacing
E. First spacing is normal exchanging between intravascular and extravascular space
F. Second spacing is accumulation fluid into tissues as edema
G. Third spacing is accumulation of fluid in body cavities e.g. Ascites, pleural effusion
H. All above correct except “C”

325. If vagus nerves are severed the respiratory rate
E. Is increased
F. Is decreased
G. Remains constant
H. Ceases

326. Destruction of pneumotaxic center located in the pons causes
E. Apneustic respiration
F. Forceful expiration
G. Accelerated respiration
H. Apnea

327. Median nerve
E. Is formed from medial and lateral cods
F. Innervates all flexor compartment muscles except flexor carpi ulnaris
G. Is also known as the labourer’s nerve
H. Only (a) is correct

328. Radial nerve
E. Is formed from posterior cord
F. Innervates all extensor compartment muscles except anconeus
G. Is prone to injury in fractures of the shaft of humerus
H. Injury results in wrist drop

329. Ulnar nerve
   E. Is a branch of the posterior cord
   F. Is known as the musician's nerve
   G. Is the major innervation of muscles of the hand
   H. Lesion of the nerve results in ape thumb
330. A 3/0 suture is
   E. Larger in diameter than 4/0
   F. Smaller in diameter than 4/0
   G. Is too small for skin closure
   H. Can be used in an adult patient to close the abdominal sheath

331. Boil (furuncle)
   E. Can occur on any part of the body except the face
   F. Is infection of hair follicle
   G. Can cause signs of inflammation
   H. Surgical drainage is not indicated

332. Carbuncle
   E. Can occur on any part of the body except the neck
   F. Is necrotic infection of contiguous hair follicles
   G. Patients present with fever and malaise
   H. Both surgical drainage and antibiotics are not required for treatment

333. Lymphangitis
   E. Common causative organism include streptococci
   F. Blood cultures are usually positive
   G. Lymph nodes draining the infected area are usually swollen and painful
   H. Presents with systemic inflammatory response syndrome (SIRS)

334. Regulations of fluids and electrolytes movement across all membranes is by
   E. Diffusion/filtration
   F. Active transport
   G. Osmosis
   H. Characteristics of the membrane itself

335. Atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) are hormones involved in regulation of
E. pH of the body fluids
F. Water and electrolyte balance
G. ANP is produced in atrium
H. BNP is produced in ventricles

336. A white leg is due to
E. Femoral deep vein thrombosis
F. Lymphatic obstruction
G. Femoral vein thrombosis and lymphatic obstruction
H. None of the above

337. The following are irritative symptoms of bladder outlet obstruction secondary to BPH:
E. Weak stream
F. Nocturia
G. Frequency
H. Intermittence

338. Ilio-Psoas muscle for hip flexion is innervated by
E. L1, 2 segments
F. L4, 5 segments
G. L5, S1 segments
H. S1, S2 segments

339. Secondary metastasis to the spine can originate from
E. Thyroid cancer
F. Breast cancer
G. Lung cancer
H. Prostate cancer

340. A seroma is also known as
E. A hematoma
F. A chyloma
G. A collection of serum in the tissue
H. All above correct

341. Choice of suture material
   E. Is irrelevant
   F. Depends upon the tissue and area of the body
   G. Is usually left up to the scrub nurse
   H. Is whatever the hospital gives you

342. About swellings
   E. A punctum is diagnostic of a lipoma
   F. A cyst is lined by some form of epithelium and is fluid-filled
   G. A sebaceous cyst exhibits pseudo-fluctuation
   H. Carotid body tumor moves only in one plane vertically

343. The following are the reasons why investigations are done
   E. Medico-legal purpose
   F. To make a diagnosis
   G. To establish fitness before surgery
   H. For monitoring of a disease process

344. Hypertrophic pyloric stenosis
   F. Is never seen in adults
   G. Projectile vomiting occurs
   H. A mass is never palpable
   I. Diarrhea is common
   J. Is associated with other congenital abnormalities

345. Indications for surgery in peptic ulcer disease include:
   F. Intractable pain
   G. Pyloric stenosis
   H. Absence of healing after 3 weeks of medical treatment
   I. Acute gastric erosion due to analgesic abuse
   J. Single episode of hematesis

346. Treatment of peptic ulcer disease includes:
   F. Antacids
   G. Antibiotics
   H. Prostaglandin analogues e.g. misoprostol

I. Omeprazole
J. Cimetidine

347. Operations performed for peptic ulcer disease includes
F. Gastrostomy
G. Gastropexy
H. Vagotomy and gastrojejunostomy
I. Billroth gastrectomy
J. Polya gastrectomy

348. Which of the following conditions give rise to hemoperitoneum?
F. A ruptured ectopic pregnancy
G. Rupture of the bladder neck
H. An injured spleen
I. Spinal injuries
J. An extraperitoneal isolated injury to the kidney

349. Post-operative pulmonary complications commonly occur:
F. In patients with chronic bronchitis
G. In smokers
H. In patients with severe pain
I. Following herniorrhaphy
J. Following splenectomy

350. Open cardiac operations require one or more of the following
F. Extracorporeal circulation
G. Hypothermia
H. Cardioplegia
I. Right second intercostal posterolateral thoracotomy
J. Sternotomy

351. Cardiac arrest is caused by
F. Anoxia

G. Excessive intravenous potassium
H. Exsanguinating bleeding
I. Frusemide
J. Intravenous glucose

352. Cardiac tamponade following trauma:
F. Heart sounds are usually muffled
G. There is venous hypertension
H. There is arterial hypotension
I. Urgent surgical treatment is indicated
J. Urgent artificial ventilation is required preoperatively in all cases

353. Spinal (Lumbar) puncture:
F. Is usually performed at L1-L2 level
G. The cerebrospinal fluid is located in the subdural space
H. The cord ends at a higher level in an infant
I. Must not be performed in the presence of a raised intracranial pressure
J. A complication is venous bleeding

354. The following tumors readily metastasize to bone
F. Rectal adenocarcinoma
G. Basal cell carcinoma of skin
H. Follicular carcinoma of the thyroid
I. Prostatic adenocarcinoma
J. Renal adenocarcinoma

355. A lipoma has the following features
F. A slipping edge
G. Transillumination
H. Is tender to touch
I. Has a central punctum
J. Is only found in subcutaneous tissue

356. Tumors of the parotid include

E. Pleomorphic adenoma
F. Adenolymphoma
G. Papillary carcinoma
H. An enlarged intraparotid lymph node

357. Which investigations demonstrate occlusion of one common iliac artery
F. Inferior venacavogram
G. Translumbar aortogram
H. Doppler ultrasound
I. Intravenous pyelogram
J. Retrograde femoral catheterization of the other side

358. Varicose veins
F. Are tortuous elongated veins
G. Pain is a late feature
H. It is also known as venous hypertensive disease
I. Elastic support stocking are useful
J. Injection sclerotherapy is indicated in some patients

359. Hemorrhage
F. Primary hemorrhage occurs within 24 hours
G. Secondary hemorrhage occurs after a week to 10 days
H. Bleeding time is a measure of fibrinogen activity
I. The normal clotting time is up to 20 minutes
J. Bleeding in cirrhotics is immediately controlled by administering oral vitamin K

360. Common cause of pyrexia within 2 days of surgery include:
F. Chest infection
G. Blood transfusion
H. Wound infection
I. Venous thrombosis
J. Allergy to the anesthetic agent

361. Bacteroides species
   F. Are common in the mouth
   G. Are the commonest large bowel commensal
   H. Are sensitive to metronidazole
   I. Causes necrotizing fasciitis
   J. Cause Crohn's disease

362. An anteroposterior chest X-ray (done to demonstrate a left subphrenic abscess) will show:
   F. Obliteration of the costophrenic angle on the same side
   G. Collapse of the left upper lobe
   H. Lack of movement on the right cupula of the hemidiaphragm on screening
   I. A fluid level below the left hemidiaphragm
   J. Shift of the gastric fundus

363. Fournier's gangrene of the scrotum
   F. Is secondary to tuberculosis
   G. Is very uncommon in Zambians
   H. Minor trauma to the scrotal skin is an etiological factor
   I. Metronidazole is indicated
   J. Surgery is contraindication

364. The following antibiotics are effective against anaerobic organisms
   F. Chloramphenicol
   G. Erythromycin
   H. Cloxacillin
   I. Clindamycin
   J. Actinomycin

365. Hand infections
   F. The hand is rested with extension of all joints
   G. Osteomyelitis is common in untreated pulp space infection
   H. Paronychia refers to infection in the palm
   I. Commonly follow minor neglected injuries
   J. A common infecting organism is *E. coli*

366. Complications of nasogastric tubes include
   F. Gastric perforation
   G. Otitis media
   H. Chest infection
   I. Ulceration of nares
   J. Esophageal stricture following prolonged intubation

367. Tetanus
   F. Is caused by an anerobic bacillus
   G. Is associated with a hypercatabolic state
   H. Occurs only after major trauma
   I. Tetanus toxoid gives artificial immunity
   J. Common cause of death is respiratory failure

368. Streptomycin
   F. Is an aminoglycoside
   G. It is bactericidal in high dose
   H. Causes pseudomembranous colitis
   I. Causes jaundice
   J. Causes ototoxicity

369. A venous cut down can be done on the following veins
   F. Cubital
   G. Cephalic
   H. Subclavian
   I. Umbilical
   J. Posterior tibial

370. Torsion of the testis
   F. If in doubt of the diagnosis explore
   G. In unilateral torsion bilateral fixation is necessary
   H. Does not occur in children
   I. Is confused with a hydrocele
   J. A horizontally placed low testis is diagnostic

371. Congenital hydrocele

F. Can be repaired by Lord's procedure
G. Is due to closure of the processus vaginalis
H. Phenol may be used to treat the hydrocele
I. May be due to epididymorchitis
J. Presents as inguinal hernia

372. Anesthetic drugs
F. Diazepam may be used for premedication
G. Mist magnesium trisilicate for premedication for cesarian section
H. Thiopentone for induction
I. Etomidate for reversal of muscle relaxation
J. Morphine for post-operative analgesia

373. Post-operative wound breakdown may be due to
F. Use of tight suturing technique
G. Prolonged steroid use before surgery
H. Reduced serum protein
I. Protein energy malnutrition
J. Diabetes mellitus

374. Metabolic response to trauma
F. May be caused by fracture of a distal phalanx
G. Good recovery follows steroid treatment
H. Head injury may be the cause
I. Patient may gain weight in the flow phase
J. May lead to systemic inflammatory response syndrome

375. Peptic ulcer disease
F. With hematemsis may be due to malignant transformation
G. Can be duodenal if pain wakes patient after midnight
H. Can be duodenal if pain is experienced immediately after meals
I. Can be gastric if experienced immediately after food
J. Can be a cause of acute abdomen

376. Cranial nerve that supplies superior oblique which elevates and adducts the eye is:
E. 7th cranial nerve
F. 6th cranial nerve
G. 5th cranial nerve
H. 4th cranial nerve

377. Sensory component of 5th cranial nerve is tested by
E. Wrinkle formation on the forehead
F. Touch sensation on the face
G. Lifting the shoulder
H. Corneal reflex

378. When a patient has unilateral vagus nerve lesion and is asked to open the mouth and say AAAG.. The uvula moves and points
E. Away from the lesion
F. Towards the lesion
G. Stays in the midline
H. Elongates within the midline

379. When a patient has unilateral hypoglossal lesion and is asked to protrude the tongue
E. The tongue points away from the lesion
F. The tongue points to the side of the lesion
G. The tongue will show wasting
H. The tongue will show fasciculations

380. When a patient sustains cranial nerve XI lesion
E. Patient is unable to elevate (shrug) the affected shoulder
F. Patient is able to elevate the shoulder against examiners resistance
G. Sternomastoid muscle power is spared
H. When patient is asked to rotate the chin against examiners resistance he/she fails to do so

381. When a patient has unilateral cranial nerve V motor component lesion
   E. Masseter muscle will feel weak on palpation as patient clenches the teeth
   F. Temporal muscle will feel well contracted as patient clenches the teeth
   G. The jaw will deviate to the paralyzed pterygoid muscle when patient opens the mouth
   H. The jaw will deviate away from the paralyzed pterygoid muscle when patient is asked to open the mouth

382. Bilateral 6th cranial nerve palsy is a sign of
   E. Cingulate gyrus herniation
   F. Uncal herniation
   G. Raised intracranial pressure
   H. Tonsillar herniation

383. Hyper-resonant chest is likely to be caused by
   E. Hemothorax
   F. Pleural effusion
   G. Pneumothorax
   H. Tension pneumothorax

384. Secondary brain injury
   E. Can result from hypoxia
   F. Can result from hypoperfusion
   G. Can result from hypoglycemia
   H. Can result from any of the above

385. Breast cancer is common in women with
   E. Positive family history
   F. History of having breast fed their children
   G. History of previous radiation exposure
   H. Having the first child at early age

386. Causes of surgical jaundice include:
   E. Gall stones

F. Massive hemolysis
G. Ascaris lumbricoides
H. Malignancy of the head of pancreas

387. The most frequent anatomic site for squamous cell carcinoma of the esophagus is
E. Upper third
F. Middle third
G. Lower third
H. Gastro-esophageal junction

388. Pathological fractures may be associated with
E. Scurvy
F. Hyperparathyroidism
G. Bone cyst
H. All of the above

389. Perkin's exercises:
E. Are commenced 2 weeks after application of traction kit
F. Are commenced within a week after application of traction kit
G. Are commenced after 3 weeks when pain at fracture site has subsided
H. All above correct except "B"

390. Tachypnea
E. Is respiratory rate of less than 10 per minute in adult patients
F. Can be caused by tension pneumothorax
G. Can be caused by hemothorax
H. Is never associated with tachycardia

391. About investigations
E. Gastroscopy is an example of an imaging technique
F. For biliary pathology, ultrasound is superior to computerized tomography
G. Plain X-rays are sufficient in order to make a diagnosis of fractures
H. There are other methods apart from histology and cytology that are used to confirm cancer

392. Patients with central nervous system lesion on observation may have

E. Epigastric peristalsis
F. Ptosis
G. Tremors
H. Diaphragmatic respirations

393. These are major symptoms associated with intestinal obstruction
E. Diarrhea
F. Change in skin coloration
G. Hematuria
H. Melena stool

394. Oculomotor nerve lesion presents with
E. Pupil dilation
F. Ophthalmoplegia
G. Lateral rectus palsy
H. Loss of corneal reflex

395. Origin of oculomotor nerve (CN III) is
E. Medulla-oblongata
F. Pons
G. Mid-brain
H. Cerebellum

396. Open skull fracture has occurred when there is
E. Cerebral spinal fluid leak from the ear
F. Depressed skull fracture under the scalp wound
G. Subdural hematoma with superficial bruise on the forehead
H. Subarachnoid hemorrhage regardless of the cause

397. Patients who present with acute abdominal pain starting in the umbilical region and shifts to the right iliac fossa is most likely caused by
E. Perforated duodenal ulcer
F. Diverticulitis
G. Appendicitis
H. Salpingitis

398. Murphy's sign

E. Helps to diagnose ureteric stones
F. Helps to diagnose ectopic pregnancy
G. Helps to diagnose appendicitis
H. Helps to diagnose cholecystitis

399. Small intestinal obstruction is likely to present with
E. Radiating pain to the left shoulder
F. Spreading pain to the right iliac fossa
G. Colic pain in the umbilical region
H. Shifting pain to the right iliac fossa

400. Imperforate anus
F. Maybe related to urogenital fistulae
G. X-rays (invertogram) is not helpful
H. Colostomy may be useful initially
I. Cannot be a cause of intestinal obstruction
J. May present with constipation

401. Drugs used for maintenance in anesthesia
F. Nitrous oxide
G. Halothane
H. Isoflurane
I. Oxygen
J. Enflurane

402. Hirschprung's disease
F. Lacks nerve tissue in the sub-mucosa layer
G. A concentration of nerve tissue in the submucosa layer
H. History of constipation is characteristic
I. History of anal bleeding is characteristic
J. Definitive diagnosis is made on X-rays

403. The gastrointestinal system has
F. 4 basilica layers
G. Mucosa lined by epithelium
H. Muscularis mucosa to produce local movement of mucosa
I. 5 basic layers

J. Serosal lining covering mucosa

404. Normal blood values
F. Total white cell count- 14 x 10^9/L
G. Serum creatinine- 200umol/L
H. Urea- 10mmol/L
I. Sodium 155mmol/L
J. Potassium 6.5 mmol/L

405. During surgical procedures
F. Doors to operating theaters must always be left open
G. Ventilators can reduce the atmospheric bacterial load
H. The patient is not the most important source of wound infection
I. Shaving must be done ideally the day before operation
J. Commonest organism to cause infection is streptococcus

406. Concerning antisepsis
F. Antisepsis has to do with reducing the bacterial load
G. Sterilization completely destroys all viable organisms
H. Disinfection reduces the bacterial load
I. Disinfection has to do with reducing the tissue bacterial load
J. Ultraviolet light is one way of sterilization

407. Fatty acids
F. Are 75% stored as glycogen in the liver
G. Glycogen stores used up in the stressful situations within 48 hours
H. Digestion is mainly by pancreatitic lipase
I. Liver stores maintain blood levels
J. Insulin is not important in cell uptake of carbohydrate

408. Most of protein digestion and absorption occurs
F. Terminal ileum
G. Stomach and duodenum
H. Stomach and colon

I. In the liver and gall bladder
J. Stomach

409. Concerning suturing of incision
F. Subcuticular sutures are not good for cosmesis
G. Mattress suture is good for hemostasis
H. Continuous sutures are ideal if infection is anticipated
I. Continuous sutures are ideal for long incisions
J. Head and neck sutures must be removed only after the 5th days

410. Monofilament sutures
F. Cause least tissue reaction
G. Causes least wound infection
H. Causes most tissue reaction and scarring
I. Can be braised
J. Can be absorbable

411. Nylon suture
F. Absorbable suture and gives little tissue reaction
G. Non-absorbable and gives a lot of tissue reaction
H. Non-absorbable and gives little tissue reaction
I. Synthetic
J. Naturally occurring

412. On examination of the abdomen
F. Spleen is examined starting from the right iliac fossa
G. There are 9 surface division
H. Inspection is done after palpation
I. Percussion is done after palpation
J. Normal bowel sounds heard every 2 seconds

413. Peptic ulcer disease
F. With hematesis may be due to malignant transformation
G. Can be duodenal if pain wakes patient after midnight

H. Can be duodenal if pain is experienced immediately after meals
I. Can be gastric if experienced immediately after food
J. Can be cause of acute abdomen

414. Congenital hydrocele
F. Can be repaired by Lord's procedure
G. Is due to closure of the processus vaginalis
H. Phenol may be used to treat the hydrocele
I. May be due to epididymorchitis
J. Presents as inguinal hernia

415. Patients burns major burns
A. Are in negative nitrogen balance-True
B. Have normal calorie requirement-False
C. Do not generally become anemic-False
D. Are resistant to septicemia-False

416. Patients with raised intracranial pressure
A. Cerebral perfusion pressure is maintained by reduction of arterial pressure-False
B. Cerebral spinal fluid shifting-True
C. Uncal herniation is not a feature-False
D. Coma scale remains unaltered-False

417. The parafollicular cells of thyroid secrete
A. Calcitonin-True
B. Amyloid-False
C. Parathormone-False
D. Follicle stimulating hormone-False

418. An indirect inguinal hernia would lie in relation to the inferior epigastric artery:
A. Medial-False
B. Lateral-True
C. Anterior-False
D. Posterior-False

419. Fissure-in-Ano is located characteristically:
   A. Midline anteriorly-False
   B. Midline posteriorly-True
   C. Right lateral quadrant-False
   D. Left lateral quadrant-False

420. Post-traumatic intracranial space occupying lesion is likely to present with:
   A. Decreased pulse rate-True
   B. Increased blood pressure-True
   C. Irregular pulse-False
   D. Glasgow coma scale of 2/15-False

421. Paradoxical (Diaphragmatic) respiration can be caused by
   A. Odontoid fracture dislocation-False
   B. C2/3 hangman's fracture-False
   C. C5/6 fracture dislocation-True
   D. T112/L1 fracture dislocation-False

422. Concerning blood groups
   A. Blood group A is associated with increased risk of gastric cancer-True
   B. The risk of gastric cancer is associated with blood group B-False
   C. The risk of gastric cancer is associated with blood group O-False
   D. Only (b) and (c) are correct-False

423. Acute abdomen can be caused by:
   A. Perforated duodenal ulcer-True
   B. Sigmoid volvulus-True
   C. Pancreatitis-True
   D. Subcutaneous lipoma-False

424. The following are true of breast cancer
   A. Lobular carcinoma is characterized by 'Indian File' pattern of tumors cells-True
   B. Lobular carcinoma tends to be familial, multifocal and bilateral-True
   C. Carcinoma-in-situ implies non-invasive cancer that has not breached basement membrane-True

D. Macroscopically schirrhous carcinoma is hard, grey and grates on cutting-True

425. Symptoms of obstructive uropathy include the following:
A. Weak stream, hesitancy-True
B. Dribbling, straining to void-True
C. Polyuria, oliguria-False
D. Frequency, urgency, urge incontinence-True

426. Which are the causes of dysphagia
A. Carcinoma, goiter, thymoma, mediastinal lymphadenopathy, aortic aneurysm-True
B. Motor neuron disease, rabies, tetanus, pseudobulbar paralysis-True
C. Achalasia-True
D. Pneumonia, TB of spine-False

427. Concerning tumors, the term 'well differentiated' means
A. The cells are so aggressive, wild and uncontrolled and keep reproducing to no good end-False
B. Cells and tissue that have mature (specialized) structures and functions-False
C. Cancer cells look more like normal cells under a microscope-True
D. The cells tend to grow and spread more quickly than poorly differentiated cancer cells-False

428. About 85-90% radio-opaque urinary stones include the following
A. Calcium oxalate stones-True
B. Pure uric acid stones-False
C. Sodium urate and ammonium stones-True
D. Sulphur containing cysteine-True

429. Diabetic foot
A. Presence of pathologies such as infection, ulcer and neuropathic osteoarthropathy-True
B. Is always present in chronic diabetes mellitus-False
C. Any chronic foot ulcer that eventually causes diabetes mellitus-False
D. Patients have associated reduced ability to feel pain-True

430. Chest injuries
   A. Tension pneumothorax causes so-called ‘paradoxical breathing’-False
   B. May cause a rupture of the diaphragm-True
   C. A flail chest result from a wound in the chest wall which acts as a valve that permits air to enter the pleural cavity but prevents its escape-False
   D. May cause pulmonary contusion-True

431. Glasgow coma scale
   A. Is a neurological scale which aims to give an objective way of recording the conscious state of a person-True
   B. Is composed of eye, verbal and motor response-True
   C. Glasgow coma scale as high as ≥13 signifies minor head injury-True
   D. The light response and size of the pupil are part of the Glasgow coma scale assessment-True

432. Fournier’s gangrene of the scrotum is associated with
   A. Infection-True
   B. Minor injuries-False
   C. No obvious cause-False
   D. Filariasis-False

433. Fibroadenoma
   A. Benign tumor with a mixture of stroma and epithelial tissue of breast-True
   B. Painless, firm, solitary, mobile, slowly growing lump in the breast of women of child-bearing years-True
   C. Leiomyoma in the breast tissue-False
   D. Benign tumor of the fibrous tissue in the sweat glands of the breast tissue-False

434. Cervical sympathectomy is procedure which is indicated for the treatment of
   A. Causalgia-True
   B. Raynaud’s disease-True
   C. PUD-False
   D. Cervical rib-False

435. The following are causes of secondary varicose veins
   A. Klippel Trenuaney syndrome-True

B. Oral contraceptive pills-True
C. Pelvic tumors-True
D. Exercises-False

436. Varicose veins are one of the causes of thrombophlebitis other causes include
A. Blood transfusion-True
B. Enteral feeding-False
C. Chemotherapy-True
D. Trauma-True

437. High risk group in the development of DVT would include the following features:
A. Operation <30 minutes-False
B. Obesity-True
C. >40 years age -True
D. Stroke patient -True

438. The development of skin malignancy, pre-malignant lesions would include:
A. Solar keratosis-True
B. Keloids- False
C. Radio-dermatitis-True
D. Rodent ulcer-False

439. In hyperthyroidism
A. Serum creatinine is increased-True
B. Serum cholesterol is increased-False
C. Serum calcitonin is increased-False
D. Serum iodine is reduced-True

440. Causes of pulsatile thyroid swelling
A. Follicular carcinoma-False
B. Primary thyrotoxicosis-True
C. Secondary thyrotoxicosis-False
D. Papillary carcinoma-False

441. Medullary carcinoma of the thyroid (MCT):
A. Arises from parafollicular ‘c’ cells-True

B. Associated with MEN I syndrome-False
C. Associated with MEN IIa syndrome-True
D. Sporadic is the commonest-True

442. Tetany is a condition where there is hyper-excitability of peripheral nerves. The causes include
A. Hypoparathyroidism-True
B. Respiratory alkalosis-True
C. Pyloric stenosis-True
D. Chronic renal failure-True

443. In etiopathogenesis of carcinoma of the esophagus, precancerous conditions include:
A. Plummer vinson syndrome-True
B. Achalasia cardia-True
C. Corrosive stricture-True
D. Keratosis palmaris-False

444. In gastric outlet obstruction electrolyte changes
A. Hypernatremia- False
B. Hyperkalemia- False
C. Hypochloremic alkalosis-True
D. Hypocalcemia- False

445. In hepatocellular carcinoma alpha-fetoprotein levels are increased. What other conditions would increase the alpha-fetoprotein levels:
A. Carcinoma stomach- False
B. Carcinoma pancreas- False
C. Hepatoma-True
D. Embryonal cell carcinoma of testis-True

446. An increase in the level of alkaline phosphatase would be caused by
A. Biliary cirrhosis-True
B. Metastasis in the liver-True
C. Hepatocellular carcinoma- True
D. Hepatitis-True

447. Acute pancreatitis can be caused by drugs which include
   A. Estrogen-True
   B. Diuretics-True
   C. Antifungals-True
   D. None of the above-False

448. Complications of acute pancreatitis include the following except:
   A. Shock-False
   B. Hypocalcemia-False
   C. Pleural effusion-False
   D. Respiratory insufficiency-False

449. The following are components of wound healing except:
   A. Wound contraction-False
   B. Healing by primary intention-True
   C. Epithelization-False
   D. Scar formation-False

450. Factors affecting wound healing include:
   A. Jaundice-True
   B. Debilitation-True
   C. Gender-True
   D. Parity-False

451. Ludwig's angina precipitating factors:
   A. Cachexia-False
   B. Chemotherapy-False
   C. Dental caries-True
   D. Immobility-False

452. In a patient with established Tetanus, the possible causes of death would include
   A. Aspiration-True
   B. Cardiac arrhythmias-True
   C. DVT-True
   D. Laryngeal spasm-True

453. Tropical ulcers occur in Tropical countries. The precipitating factors are:

A. Malnutrition-True
B. Trauma-True
C. Insect bite-True
D. HIV infection-True

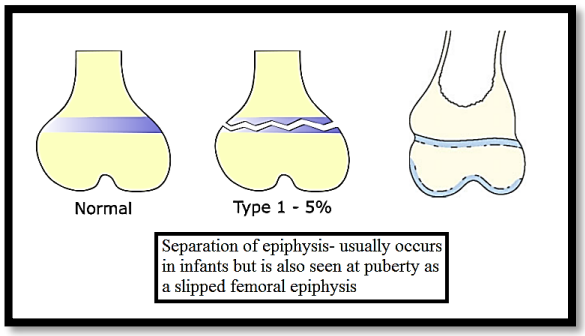
ANSWERS TO ESSAYS

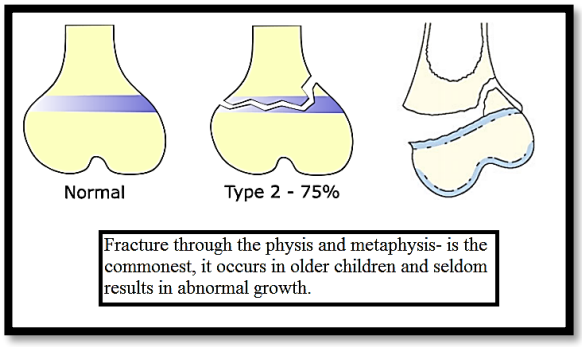
1. Write short notes on:
   a. Salter-Harris classification of epiphyseal fractures

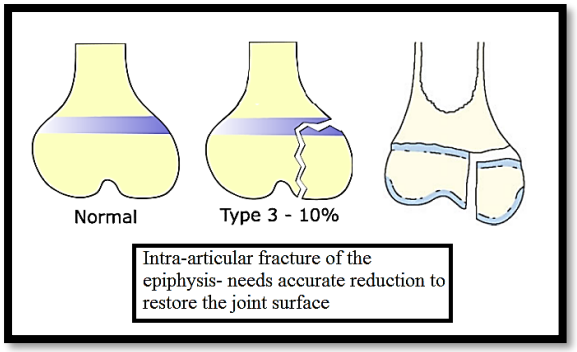
   Answer:

   Salter and Harris (1963) described 5 different types of epiphyseal fractures.

   - Type 1 ("Straight through")-5%
     - Fracture involves a transverse fracture through the hypertrophic or calcified zone of the epiphyseal plate
   - Type 2 ("Above")-75% (commonest)
     - Fracture involves growth plate; it extends into metaphysis above/ away from the joint but spares the epiphysis
     - Fracture often splits off a triangular metaphyseal fragment of bone (sometimes referred to as a Thurston-Holland fragment)
   - Type 3 ("Lower")-10%
     - The fracture involves the growth plate and epiphysis below the joint but spares the metaphysis
   - Type 4 ("Through everything")-10%

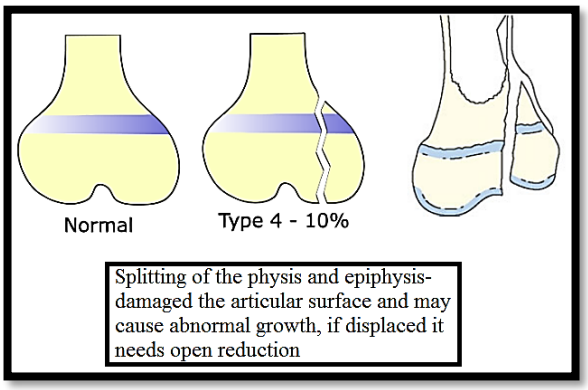


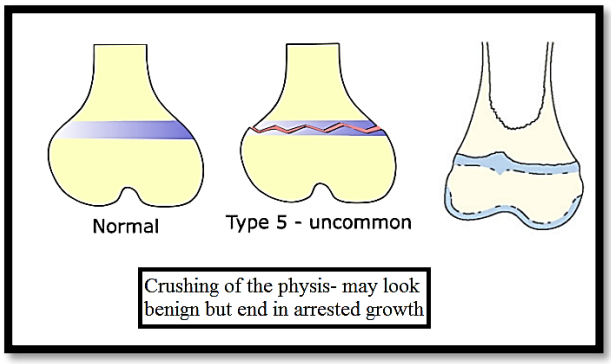




- Fracture involves the growth plate, metaphysis and epiphysis (Fracture forms a T)

➢ Type 5 ("Rammed/Crushed")-1%
- Due to longitudinal compression injury of the epiphyseal plate.
- There is no visible fracture but growth plate is crushed and this may arrest growth





b. Gatland classification of supracondylar fractures

Answer:

The Gatland classification is divided into 3 types (based on displacement)

➢ Type 1:
- This is an undisplaced supracondylar fracture
- Treatment: collar-cuff or backslab

➢ Type 2:
- Angulated fracture with posterior cortex still intact (periosteal hinge)
- Subdivided into:
  - Type IIA- less severe injury with distal fragment angulated
  - Type IIB- severe injury, distal fragments angulated and malrotated
- Treatment: manipulation under anesthesia and collar cuff or backslab (convert Type II to type I)

➢ Type 3:
- Completely displaced fracture (although the posterior periosteum is usually still preserved, which is usually still preserved, which will assist surgical reduction)

- Treatment: manipulation under anesthesia, K-wire (Kirschna wire) fixation, after 3 weeks take the patient to theater to remove the K-wires under general anesthesia and put a slab

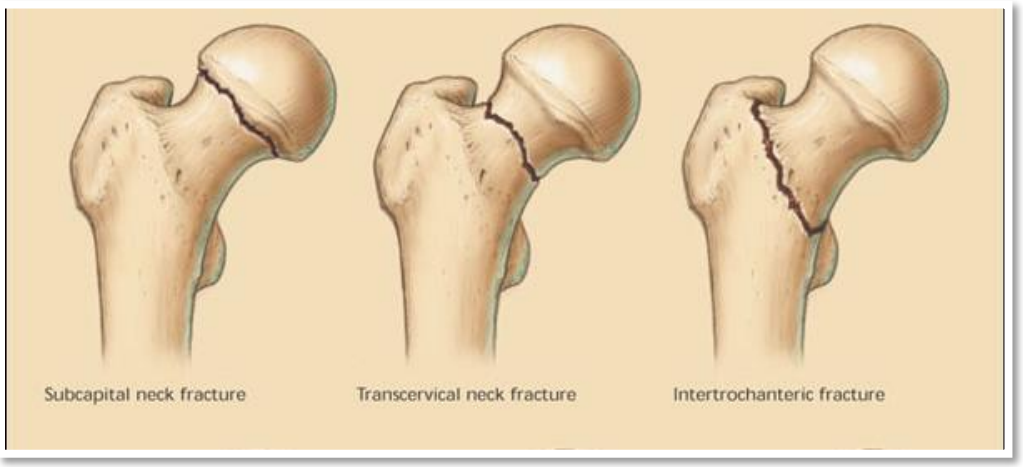
c. Draw and label patterns of fracture of neck of femur

Answer:

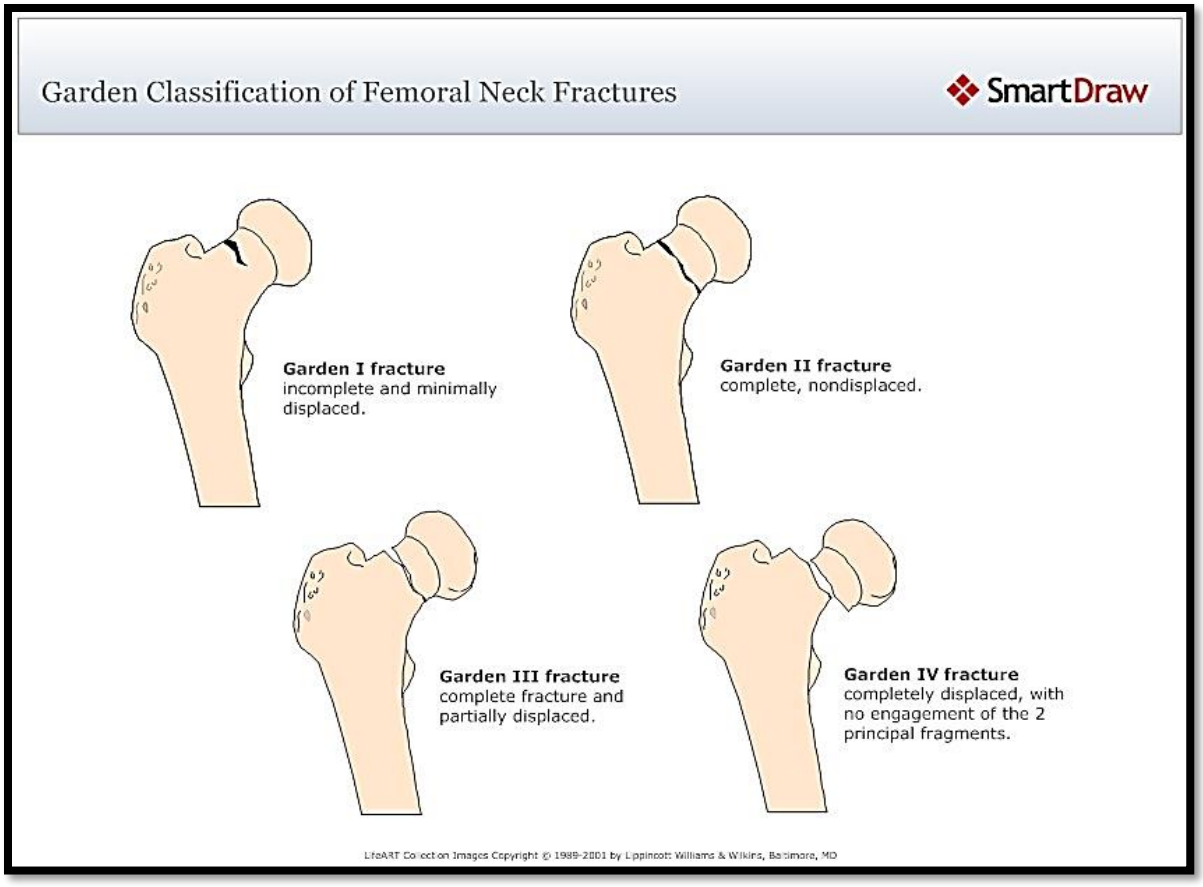
Fractures can be viewed based on

- Site of fracture (Anatomical classification)
- Displacement (Garden's classification)

Site of fracture, include: Subcapital, Transcervical and Basal



Garden's classification:



2. Questions:
   a. Define excised and incised wound

      Answer:

      - Excised wounds are wounds created by the act of cutting out/ severing/ removing undesired tissue from the body.
      - Incised wounds are wounds created by the act of cutting into tissue in order to enter or gain access to an internal part of the body. Edges are usually linear.

   b. Describe briefly physiology of healing of excised wound

      Answer:

      Excised wounds heal by fibrosis and results in a wide scar often hypertrophied and contracted. Re-epithelialisation occurs from remaining dermal elements or wound margins.

The stages of wound healing include:

- Stage of coagulation/hemostasis
  - Happens almost immediately following injury the clotting cascade is initiated
  - Local vasoconstriction occurs and a platelet clot forms
  - Platelet derived growth factor is released
- Stage of inflammation
  - Begins after vasodilation (0 to 4 days)
  - Inflammatory cells reach site of injury. Initially neutrophils and then macrophages which secrete fibroblast growth factor for angiogenesis and skeletal development as well as remove tissue debris.
  - Lymphocytes secrete inflammatory mediates and oxygen radicals
- Stage of proliferation/fibroplasia
  - There is formation of granulation tissue
  - Lasts 4 to 21 days
  - Fibroblast deposits type III collagen, capillaries provide nutrients.
- Stage of remodeling
  - Takes 3weeks to 18 months
  - There is maturation of collagen by cross-linking which is responsible for tensile strength of the scar.
  - There is differentiation of fibroblast into Myofibroblast which contracts wound.
  - Type III collagen is replaced with type I collagen and blood vessels atrophy.
  - Collagenase removes type III collagen and requires zinc as a co-factor

c. List complications of wound healing

Answer:

Early

- Wound infection
- Septicemia
- Septic arthritis

Late

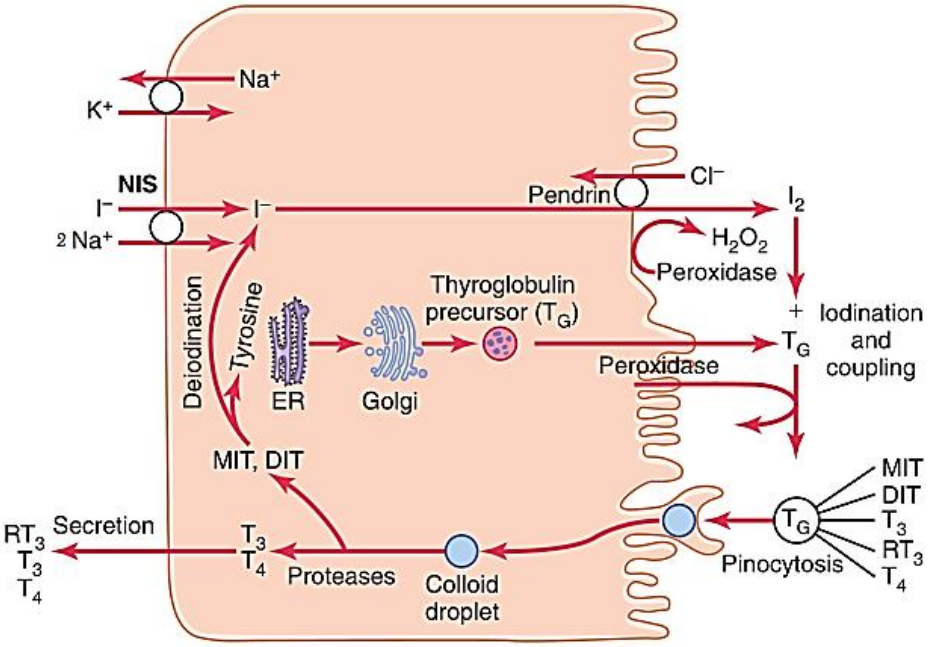
- Wound Dehiscence
- Contractures
- Hypertrophic scars

- Keloids
- Marjolins ulcer

3. Questions
   a. Briefly trace iodine in food to iodine in thyroid hormone

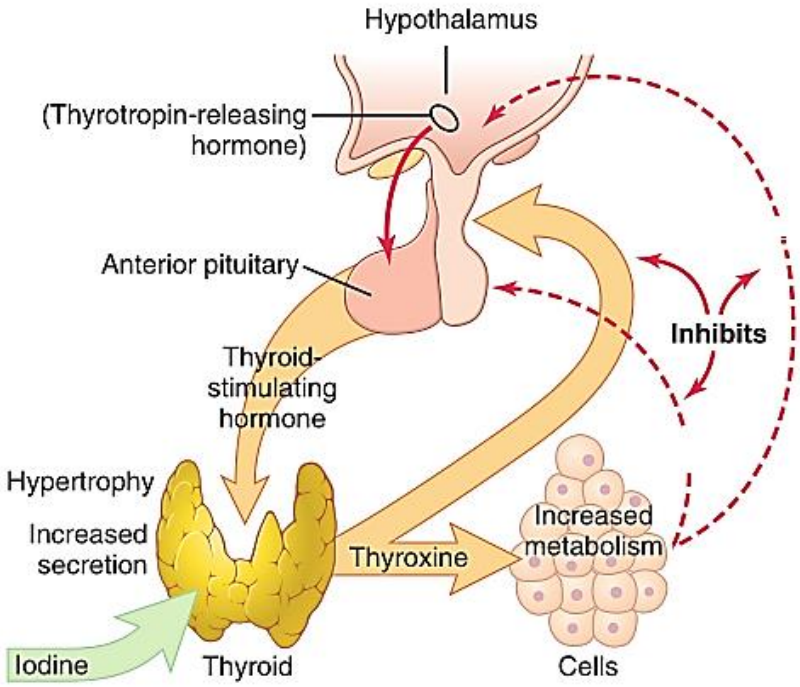
   Answer:

   - Around 50mg of iodine in the form of iodide is required per year (1mg per week). Source of the iodine is iodated salt. Iodine can also be found bound to protein as iodine this has to be converted to iodide (by glutathione) as this is the easiest form to absorbed
   - Iodides ingested orally are absorbed from the intestine into the blood in about the same manner as chlorides.
     - Absorption of sodium ions through the epithelium creates electronegativity in the chime and electropositivity in the paracellular spaces between the epithelial cells.
     - Iodide ions then move along this electrical gradient to "follow" the sodium ions.
   - Iodide in the blood stream is transported to the thyroid glandular cells and follicles.
   - On the basolateral side of the cells sodium-iodide symporter co-transports one iodide ion along with 2 sodium ions across the basolateral membrane into the cells. (energy to transport these ions against a concentration gradient comes from the sodium potassium ATPase pump).
   - In the follicular cells peroxidase converts iodide ions to an oxidized form of iodine. Iodine is transported to the colloid through pendrin (an antiporter for chloride and iodine), in the colloid iodine is then organified (also catalyzed by the enzyme peroxidase) to thyroglobulin where it binds tyrosine residues to form monoiodothyronine and diiodothyronine which can couple to form the hormones thyroxine (T4) and triiodothyronine (T3)- also in the presence of peroxidase enzyme
   - The thyroid hormones can then be released in blood via pinocytosis and secretion.



b. Draw homeostatic loop for T3 and T4

Answer:



c. List signs/ symptoms of thyrotoxicosis

Answer:

- Symptoms
  - Nervousness
  - Anxiety and hyperactivity
  - Increased sweating especially palms
  - Hyperthermia and Heat intolerance
  - Palpitations
  - Hyper-defecation
- Signs
  - Fine hand tremors
  - Warm moist hands
  - Tachycardia and atrial arrhythmias
  - Systolic hypertension with wide pulse pressure
  - Lig lag
  - Wide eyed stare gaze
  - Muscle weakness

4. Write short notes on
   a. Colles's fracture

Answer:

- This is a transverse fracture of the radius about 2.5cm above the wrist joint with the following displacement (Remember mnemonic "College Rotating Doors Shut I'm Late"):
  - Rotation
  - Dorsal tilt (proximal tilt)
  - Dorsal shift
  - Supination
  - Impaction
  - Lateral shift (radial shift)
  - Lateral tilt
- It is found in elderly due to falling on an outstretched hand.
- Clinical features: dinner fork deformity with prominence on the back of the wrist and depression in front, local tenderness and pain on wrist movement
- Treatment:
  - Closed manipulation under anesthesia

        - Correct the impaction first
        - Center the fragments (correct the radial displacement)
        - Correct the proximal tilt by dorsiflexing the wrist
        - Lock the arm towards the little finger using a slab ("corrected dinner fork appearance")
    - X-ray: after reduction (to check of fracture is reduced-post-reduction X-ray), after 24 hours (for neurovascular state), after 7 (to rule out any displacement, reduce in theatre if displaced), after 14 days and at 8 weeks

b. Colostomy

Answer:

- Definition: this is hollow iatrogenic mucocutaneous fistula between the colon and the skin.
- Types:
    - Temporary: done in conditions where diversion is required to facilitate healing distally in the rectum or distal colon. This type is closed once purpose is over. Sites of temporary colostomy include the right hypochondrium and left iliac fossa.
        - Examples: Loop colostomy, Devine's double barrel, spectacle, Hartmann
    - Permanent: this is always and end colostomy. It is placed in left iliac fossa, 6cm above and medial to the anterior superior iliac spine.
- Colostomy indications:
    - Congenital (congenital megacolon, anorectal malformations, imperforate anus, Hirschsprung disease, anal atresia, anorectal agenesis, anal stenosis) and
    - Acquired (Intestinal obstruction i.e. sigmoid volvulus, ischemic bowel disease, tumors i.e. anal and rectal carcinoma, perforation of left sided colon, high anal fistula, after Hartmann's operation)
- Complications:
    - Ischemic gangrene
    - Hemorrhage
    - Retraction
    - Prolapse
    - Parastomal hernia

c. Shoulder dislocation:

Answer:

- This is displacement of the head of the humerus from the glenoid cavity.
- It can be caused by
  - High speed trauma (traumatic shoulder dislocation)
  - Ligament overstrain (repetitive shoulder ligament overstrain e.g. with swimming, tennis, throwing sports e.g. baseball)
- Dislocation can either be partial (sublaxation) where the head of the humerus is partially dislodged from the glenoid cavity or complete (luxation) where the head of the humerus is completely dislodged from the glenoid cavity.
- Dislocation can either be in the anterior (common) or posterior direction.
- Signs and symptoms:
  - Deformity
  - Swelling
  - Numbness
  - Weakness
  - Bruising
  - Muscle spasms
  - History of trauma or sporting activity before injury
- Treatment: closed reduction
- Rehabilitation: immobilize in a sling for a few weeks, apply ice to shoulder 3 to 4 times.

5. Question
   a. List complications of thyroidectomy

Answer:

- Early
  - Bleeding
  - Hemorrhage and Hematoma
  - Injury to recurrent nerve
  - Tracheomalacia
  - Upper airway obstruction
  - Hypocalcemia
  - Tracheal perforation
  - Thyroid storm- atrial fibrillation
- Intermediate
  - Infections

- Late
  - Hypothyroidism
  - Hypocalcemia with pathological fractures
  - Keloids and hypertrophic scars

b. Discuss management of thyroid storm (Crisis)

Answer:

- Admit to ICU/High dependency unit
- Perform ABCs
- Rehydration: IV fluids and oral fluids
- Control hyperthermia (ice pack/Cold compress/sponging/fan, antipyretic-IV paracetamol)
- Administer beta blockers: Propranolol (avoid in asthmatic patients use calcium channel blockers instead such as verapamil and diltiazem)
- Administer antithyroid drugs e.g. propylthiouracil to block T4 and T3 synthesis and release
- Administer potassium iodide (Lugol's solution)
- Administer glucocorticoids e.g. hydrocortisone or dexamethasone (to reduce conversion of T4 to T3)
- Administer bile acid sequestrant e.g. cholestyramine to prevent T4 and T3 reabsorption from the gut
- When patient is stabilized (euthyroid) thyroidectomy can be performed with appropriate hormone replacement after

c. Discuss briefly clinical presentation of hypoparathyroidism

Answer:

Presents with hypocalcemia. Clinical features include:

- Neuromuscular excitability
  - Carpopedal spasm: tetany (involuntary contraction) of feet or hands
  - Chvostek's sign: facial twitch on parotid tap
  - Trousseau's sign: carpal tetany in response to a tightened Blood pressure cuff that is pumped above systolic blood pressure
  - Paresthesia of peripheries, tongue and mouth
  - Cramps and aches
  - Airway: laryngospasm and bronchospasms
- CNS

- Seizures
- Papilledema
- Dementia
- Parkinsonism
- Anxiety, Depression, fatigue

➢ Other signs (in chronic disease)

- Cataracts
- Dry, coarse, puffy skin
- Reduced tooth growth (if patient is young)

6. Write short notes on metabolic body response to acute blood loss.

Answer:

➢ There are 2 phases of the response

- Initial phase (EBB phase)
  - Occurs within the first 48hours to 2 days
  - Physiological role of the ERB phase is to conserve circulating volume and energy stores.
  - It is characterized by hypovolemia, decreased cardiac output, decreased metabolic rate, hypothermia and lactic acidosis.
  - Blood loss triggers the hypothalamus, pituitary glands, adrenals, thyroid gland, pancreas and renin angiotensin aldosterone axis. This results in release stress hormones (catecholamines, cortisol, glucagon, growth hormone) and other hormones such as aldosterone, angiotensin and thyroid hormones.
  - Catecholamines produce the following changes: increase in heart rate, respiratory rate, and temperature
    - Accentuated clinical signs e.g. tachycardia (more than 90b/m), tachypnea (more than 20b/min), hyperthermia (38 degrees or more), hypothermia (36 degrees or less) are indicative of systemic inflammatory response (SIRS)
    - Any 2 of the 4 parameters above= SIRS
  - Cortisol increases the blood sugar (through gluconeogenesis, glycogenolysis)
  - Aldosterone increases sodium and water reabsorption (under the control of ADH)

- WBC count also increases and there is also increase in the production of pro-inflammatory cytokine such as IL-1 and TNF-alpha

- Flow phase
  - Occurs from day 3 to about day 10.
  - Associated with chronic neuroendocrine response.
  - It is divided into:
    - Catabolic phase day 3 to 10
    - Anabolic phase: can last weeks depending on severity
  - It is associated with the mobilization of body energy stores for repair and replacement of damage tissue

7. Write short notes on value of sentinel node in investigating breast cancer

   Answer:

   The sentinel node is used during a sentinel lymph node biopsy. The procedure consists of injecting a vital blue dye or radioactive sulfur colloid or both around the tumor. The surgeon often waits 5 minutes in the case of the dyes and a few hours in the case of colloid and then dissects the axilla removing only a few nodes that either stained blue or radioactive. The principle behind this is that the first node that drains the dye are most likely to first drain the tumor as it metastasizes. In doing so the nodes are examined and if found to have signs of malignancy it indicates that the breast cancer may have metastasized from the local area however if the nodes show no signs of malignancy it is assumed that the tumor has not metastasize. This has helped reduce morbidity as compared to a full axillary node dissection and in experienced hands the procedure is close to 100% accurate in correctly describing the presence or absence of tumor in the axilla.

8. Discuss briefly common patterns and presentation of hand infection.

   Answer:

   - General features:
     - Infection spreads faster in all areas.
     - Infection causes edema over the dorsum of the hand due to lax skin and more lymphatic network even though infection per se is more over the volar aspect. Hand looks like a frog hand.
     - There is restricted movement of fingers and hands.
     - The hand functions like hook, pinch, grip, grasp are lost.
     - There is severe pain and tenderness with fever.

- Tender palpable axillary lymph nodes are often present
- They can be superficial or deep and can be localized or spreading

➢ Different types of hands infection include:

- Acute paronychia (most common hand infection, occurs in subcuticular area under the eponychium) presents as severe throbbing pain and tenderness with visible pus under the nail root. Nail on touch is very tender. Caused by minor injury, implicated organisms *Staphylococcus aureus* and *Streptococcus pyogenes*
- Chronic paronychia- commonly due to fungal infection (Candida), nail is diseased with ridges and pigmentation, itching in the nail bed, recurrent pain, and discharge. Secondary bacterial infection may supervene
- Terminal pulp space infection (felon)- second most common hand infection. Affect index and thumb mostly, usually caused by minor injury like finger prick. Presents with pain, tenderness, swelling in the terminal phalanx, fever and tender axillary lymph nodes. Implicated organisms include staphylococcus, streptococcus and gram-negative organisms
- Subungual infection
- Web space infection
- Mid-palmar space infection
- Thenar space infection
- Deep palmar abscess
- Acute suppurative tenosynovitis
- Chronic tenosynovitis of flexor tendon sheath of palm and forearm-compound palmar ganglion
- Lymphangitis of the hand
- Arthritis of hand joint
- Subcuticular abscess

9. An 8-year-old child weighing 25kg has 20% burns which occurred at 04:00 hours and arrives in hospital at 08:00hrs. Calculate fluid requirement using Parkland formula:
   a. Resuscitation fluid
   b. Maintenance fluid

   Answer:

a. Resuscitation fluid:
   - Volume of fluid in 24 hours= 4ml x Mass (kg) x % burns
   - Volume of fluid in 24 hours= 4 x 25 x 20
   - Volume of fluid in 24 hours= 2000ml
   - Give:
     - 1000ml of Ringer's Lactate Intravenously in 4hours (from 08:00 to 12:00hours) since child was burnt at 04:00 hours so 4hours from 8hours of first fluid has elapsed.
     - 1000ml of Ringer's Lactate intravenously in remaining 16hours (from 12:00hours to 04:00 next day).

b. Maintenance fluid:
   - 100ml/kg for first 10kg
   - 50ml/kg for next 10kg
   - 20ml/kg for the remaining 5kg
   - Total fluids in 24 hours= (100 x 10) + (50 x 10) + (20 x 5)
   - Total fluids in 24 hours= 1600ml
   - Give 1600ml in 24 hours dextrose-saline (5% dextrose)

10. Write short notes on the complications of blood transfusion

Answer:

- Acute hemolytic reactions due to ABO incompatibility (results from antibodies in recipient's plasma direct against antigens on donor's erythrocytes). It can be either
  - Intravascular hemolysis (RBCs are hemolyzed in the blood steam) it causes hemoglobinemia, hemoglobinuria, acute renal failure, DIC and complement-mediated cardiovascular collapse. Dyspnea, chest pain, sweating, fever with chills, tachycardia, hypotension and cardiac arrest occur in fatal type. Jaundice may also be seen.
  - Extravascular hemolysis: Due to Rh or non-ABO antigens. It is seen in patients usually exposed to antigen through prior pregnancies, transplantation or transfusion. Antibody titers are too low often to be detected during screening however production increases with re-exposure to the initial antigen. Antibodies do not activate complement (no intravascular hemolysis) instead they coat RBCs which are removed by splenic macrophages.

- Non-hemolytic Febrile reactions: due to formation of cytokines during storage of blood. Most common complication due to impurities like pyrogens in blood or in infusion set. Presents as headaches, fever, chills and rigors, tachycardia, nausea are other features. Transfusion is temporarily stopped or slowed down with administration of antipyretic.
- Allergic/anaphylactic reaction: urticaria and allergy to specific proteins in donor's plasma can occur.
- Congestive cardiac failure: occurs especially if large quantities of whole blood are transfused in chronic severe anemia, pregnancy, elderly patients, in patients who have cardiac problems.
- Infections: serum hepatitis, HIV infection, malaria infection, CMV infection, EBV infection, Syphilis infection, Trypanosoma cruzi infection
- Air embolism
- Thrombophlebitis
- Hemochormatosis/iron overload in patients with chronic renal failure receiving repeated blood transfusion
- Citrate intoxication causing bradycardia and hypocalcemia. (for every 4 units of blood 10ml of 10% calcium chloride or gluconate should be infused intravenously)
- Transfusion related acute lung injury
- Graft vs Host disease: occurs when donor lymphocytes mount immune response against the recipient lymphoid tissue.

11. What are signs and symptoms of acute appendicitis. Explain the anatomical basis for these features, where applicable.

Answer:

- Symptoms:
  - Pain: visceral pain starts around the umbilicus due to distension of the appendix, after a few hours somatic pain occurs in right iliac fossa (Blumberg sign) due to irritation of the parietal peritoneal because of inflamed appendix. The pain is severe and diffuse. Pain migrates to right iliac fossa with maximum tenderness at McBurney's point.
  - Vomiting and nausea due to reflex pylorospasm
  - Constipation (in postileal appendix)/diarrhea (pelvic appendix)
  - Anorexia and Low-grade fever
  - Urinary frequency: bladder irritation from inflamed appendix

- Signs
  - Tachycardia
  - Tenderness and rebound tenderness in right iliac fossa (Blumberg sign/release sign)
  - Pointing sign- Pain at Mc Burney's point
  - Signs indicative of peritoneal irritation
    - Rovsing sign- on pressing left iliac fossa, pain occurs in right iliac fossa due to shift of bowel loops which irritate the peritoneum
    - Psoas test- for retrocecal appendix, hyperextension of hip causes pain in the right iliac fossa.
    - Obturator test- For pelvic appendix internal rotation of right hip causes pain in right iliac fossa due to irritation of obturator internus muscle
    - Baldwing test- positive in retrocecal appendix- when legs are lifted off the bed with knees extended the patient complains of pain while pressing on abdomen (Ribs-ilium)

12. You are called to a surgical ward, 2 hours after thyroidectomy of a patient with thyrotoxicosis. Write short notes on what complications your patient might have now and in the near future.

Answer:

- Early
  - Bleeding and hematoma as a result of poor hemostasis (can lead to upper airway obstruction
  - Injury to recurrent laryngeal nerve (unilateral or bilateral)- leads to vocal cord collapse and upper airway obstruction
  - Laryngeal edema
  - Tracheomalacia with large goiter that erodes the trachea that collapses after thyroidectomy
  - Upper airways obstruction
  - Thyroid storm- atrial fibrillation
  - Tracheal perforation
  - Hypocalcemia with removal of all parathyroid glands
- Intermediate
  - Infections
  - DVT

- Hypostatic pneumonia

➢ Late
- Hypothyroidism
- Hypocalcemia with pathological fractures
- Keloids and hypertrophic scars

13. How would you investigate the cause of fever in a patient who develops a fever 3 or 4 days after abdominal surgery?

Answer:

➢ Blood investigations
  - Full blood count with differential- increase in WBC
  - ESR and C-reactive protein: increase
  - Blood culture: for sepsis
  - Urea and electrolytes, creatinine: to check renal function
  - Liver enzymes
➢ Pus swab- microscopy, culture and sensitivity (for any pus)
➢ Urine microscopy, culture and sensitivity
➢ Imaging
  - Chest X-ray (to rule out pulmonary sources of infection)
  - Abdominal ultrasound
  - CT scan

14. A 70-year-old man presents with acute retention in a male surgical ward. Take a brief relevant and focused history in order to arrive at a diagnosis.

Answer:

➢ Onset of the urinary retention
➢ Duration of urinary retention
➢ Number of times urine has been passed compared to normal
➢ Any associated symptoms:
  - Weight loss
  - Hematuria-initial, terminal or diffuse
  - Poor urinary stream
  - Terminal dribbling
  - Dysuria
  - Urgency
  - Intermittency
  - Back pain

- Any history of urinary tract infection?
- Any history of urinary stones?
- Any history of TB?
- Any history of acute renal failure?
- Last performed Digital rectal scan, any significant findings?
- History of prostate cancer screening?
- History of any urogenital surgeries or trauma

15. Persistent generalized lymphadenopathy is a common presentation of patients to a surgical clinical in Zambia. How would you evaluate such patients to come up with a diagnosis? Include in your answer the possible histological diagnostic description of the common causes.

Answer:

- History:
  - Cough? Productive or non-productive? If production, color and amount and is blood present?
  - Night sweats
  - Fatigue
  - Weight loss
  - Anorexia
  - Fever
  - Tested for HIV
- Physical examination
  - Generalized lymphadenopathy
  - High temperature, tonsils erythematous with a creamy grey exudate and appear almost confluent usually symmetrical, hepatosplenomegaly (infectious mononucleosis)
  - Oral thrush oral hairy leucoplakia on tongue, purple lesions on hard palate (HIV infection)
- Investigations
  - Lymph node biopsy
  - HIV Test, CD4 count and Viral load
  - Full blood count with differential count
  - Peripheral smear
- Differential diagnosis with histological findings
  - HIV infection: shows follicular hyperplasia
  - Tuberculosis adenitis: show caseaous necrosis

- Lymphoma: Reed-Sternberg cells seen (giant cells with large mirror image nuclei)
- Infectious mononucleosis: giant cells with 2 large mirror image nuclei (Reed-sternburg cells)
- Leukemia
- Hyperthyroidism
- Systemic lupus erythematosus

16. Graves disease is fairly common condition in young women in Zambia. Write short notes on eye signs and give reasons why these signs do occur.

Answer:

- Exophthalmos: this is proptosis of the eye, it is caused by infiltration of retrobulbar tissue with fluids and round cells with lid spasm of upper eyelid. In Grave's disease it is believed that the fibroblast have TSH receptors to TSH like antibodies, in Grave's disease fibroblast produces mxyoid substances such as glycosaminoglycans which deposit in the retro-orbital tissues.
- Lid retraction: the upper eyelid is higher than normal and lower eyelid is in normal position with visible sclera adjacent to upper eyelid. It is due to sympathetic over activity causing involuntary spasms of the smooth muscles of the levator palpebrae superioris.
- Staring gaze: this is due to the absence of normal blinking. First sign to appear. It is due to widening of palpebral fissure due to lid retraction and also due to contraction of voluntary part of the levator palpebrae superiors muscle.
- Lid lag (Von Graefe's sign): this is inability of the upper eyelid to keep pace with the eyeball when it looks downwards to follow the examiners finger

17. List the possible causes of PUD. Clearly explain how helicobacter pylori causes this disease

Answer:

- Causes of PUD
  - Infectious causes:
    - Helicobacter pylori
  - Non-infectious causes:

- Long term use of drugs e.g. Non-steroid anti-inflammatory drugs, steroids
- Alcohol and smoking
- Stress
- Burns (Curling's ulcer)
- Cerebral trauma (Cushing's ulcer)

➢ Helicobacter pylori and peptic ulcer disease

- The organism has the enzyme urease which has the ability to hydrolyze urea to form ammonia, a strong alkali. Ammonia through negative feedback causes the release of gastrin on the antral G cells which results in gastric acid hypersecretion.
- The bacterium produces enzymes e.g. dehydrogenase (converts alcohol to aldehyde which is toxic to mucosa), and endopeptidase which disrupts the gastric mucous barrier and the inflammation induce in the gastric epithelium is associated with the ultimate ulceration.
- Some strains produce cytotoxins such as cagA and vacA gene products which directly damage the cells of the gastrointestinal tract. The effect of the organism on the gastric epithelium is to incite a classic inflammatory response that involves the migration and degranulation of acute inflammatory cells such as neutrophils and also the accumulation of chronic inflammatory cells such as macrophages and lymphocytes.

18. What is gangrene? What are the etiological factors of the different types of gangrene? Your short notes should include pathophysiology of each type of gangrene.

Answer:

➢ Gangrene is macroscopic death of tissue in situ (in continuity with adjacent viable tissue) with or without putrefaction. The term necrosis may be used synonymously. It often affects the distal part of a limb because of arterial obstruction (from thrombosis, embolus or arteritis).

- A gangrenous part lacks arterial pulsation, venous return, capillary response to pressure, sensation, warmth and function. The color of the part changes through a variety of shades according to circumstances (pallor, dusky grey, mottled, purple) until finally taking on a the characteristic dark-brown, greenish-black or black appearance, which is caused by the disintegration of hemoglobin and the formation of iron sulphide.

➢ Causes

- Arterial occlusion e.g. atherosclerosis, emboli, diabetes, Raynaud's disease, ergots, arteritis, peripheral artery disease
- Infective: Boil, carbuncle, gas gangrene, Fournier's gangrene, Cancrum oris
- Traumatic: direct, indirect
- Physical: burns, scalds, frostbit, chemicals, irradiation, electrical
- Venous gangrene
- Smoking, obesity

➢ Types of gangrene

- Dry gangrene: occurs when the tissues are desiccated by gradual slowing of the bloodstream, it is typically the result of atheromatous occlusion of arteries. The affected part becomes dry and wrinkle, discolored from disintegration of hemoglobin and greasy to the touch.
- Wet gangrene: occurs when infection and putrefaction are present, the affected part becomes swollen and discolored and the epidermis may be raised in blebs.
- Gas gangrene: It is due to infection with Clostridium species. Myonecrsis is due to alpha-toxin from *Clostridium perfringens.*
- Internal gangrene: This affects one or more of the internal organs such as the intestines, gallbladder or appendix. It occurs when blood flow to an internal organ is blocked e.g. in appendicitis
- Fournier's gangrene (necrotizing fasciitis) this is a rare and fulminant form of necrotizing fasciitis of the male perineum and genital region frequently due to a synergistic polymicrobial infection Bacteria enters through break in the skin or it may follow surgery, trauma, IV injection or insect bite. Infection spreads rapidly across fascial layer, leading to tissue death of fascia and subcutaneous tissue.
- Progressive bacterial synergistic gangrene (Meleney's gangrene): this is a rare type of gangrene typically occurring after an operation with painful skin lesions developing one or two weeks after surgery. It is a spreading infectious type of gangrene and is often associated with Streptococcus (which produces tissue damage mediated by Exotoxins A, B and C) as well as other microbes.

19. A 26-year-old man has been hit by a moving vehicle. He is brought to casualty in a coma. How would you evaluate this patient?

Answer:

➢ Shout for help as this requires a team effort

➢ Perform primary survey (Perform ABCs)

- A- ensure airway is patent with cervical spine protection
  - Check for any signs of airway obstruction, foreign bodies, facial, mandibular or laryngeal fractures?
  - Suction any secretion
  - Maneuvers such as chin lift or jaw thrust can be done to establish a clear airway but protect the cervical spine at all times
  - If airway not secure assess for the need of intubation or performing a cricothyroidotomy or emergency tracheostomy
  - If patient can talk, the airway is likely to be safe.
- B- breathing
  - Evaluate breathing: perform a chest examination with adequate exposure. Watch for chest movements (note for any paradoxical movements in the case of flail chest that may need mechanical ventilation) check respiratory rate, palpate for any fractures or subcutaneous emphysema (which may require draining by needle thoracostomy with subsequent intercostal drainage until defect is repaired), percuss for any fluids in pleural cavity such as hemothorax that may require intercostal drainage. Auscultate to ensure there is air entry in the lungs.
  - Provide high flow oxygen through a rebreather mask if not intubated and ventilated.
- C- Circulation with hemorrhage control
  - Assess blood loss rapidly: observe level of consciousness, skin color, pulse, capillary refill time, bleeding
  - Gain venous access with 2 large bore cannulae, obtain blood for crossmatch and baseline investigations.
  - Start running fluids, Ringer's lactate/ normal saline rapidly as you await blood
  - Arrest any bleeding (Direct manual pressure should be used to stem visible bleeding), perform chest, abdominal and pelvic compression to rule out any fractures that may cause internal hemorrhage especially when not responding to fluid resuscitation.
  - Check for blood sugar
  - Catheterize the patient and monitor urine output
- D- disability (neurological status)
  - Assess level of consciousness, using Glasgow coma scale
  - Pupil size symmetry and reaction to light

- Assess level of any spinal cord injury (limb movements, spontaneous respiratory effort)
- Any lateralising signs

  - E-exposure/environmental control
    - Undress the patient but prevent hypothermia
    - Check for any other injuries, factures, lacerations
    - Monitor vitals for signs of deterioration

- Secondary survey
  - After patient is stabilized, Take a complete history and full physical examination
    - Ask about events that lead to injury,
    - AMPLE: any allergies, medication taken, past/present medical illnesses, last meal, Events surrounding injury
  - Document all measures done during primary survey
  - Perform X-rays (Head& neck, Chest and pelvic-traumatic series X-rays), ultrasounds (FAST scan) and other laboratory tests
  - If at any point patient deteriorate revert back to primary survey
- Definitive care: depends on underlying injuries present.

20. Write shore notes on presentation and diagnosis of cervical injury

21. Write the hemodynamic changes caused by space occupying lesions in head injury patients.

22. How much fluid are you going to give a 3-year old child weighing 15Kg with 20% burns in the first 48 hours?

    Answer:

    Volume of fluid in the first 24 hours= 4ml x Weight x % burns
    Volume of fluid in the first 24 hours= 4 x 20 x 15
    Volume of fluid in the first 24 hours= 1200ml

    In first 24 hours give 600ml of Ringer's lactate to run intravenously over 8 hours and 600ml of Ringer's lactate to run in the remaining 16 hours

Maintenance= (100x10) + (50 x 5)
Maintenance= 1250ml of dextrose-saline in 24 hours on day 2.

23. There has been a plane crash in Ngwerere area with a few survivors. How would you handle the 13 survivors who have been brought to casualty?

24. A 65-year-old male patient presents to the casualty with a very painful ulcer on the right foot. He is a known hypertension and has been on treatment for several years. On examination has a moderate sized ulcer on the dorsum of the foot which is very painful. The ulcer has punched out edge and the base is pale. The SHO makes a diagnosis of a venous ulcer.
    A. What other history would you want to get from this patient?
    B. What further clinical examination would you do?
    C. Was the diagnosis correct? Give your reasons?
    D. What investigation is indicated
    E. What is the diagnosis and state your reasons?

25. Discuss the immediate management of a 5-year-old child with 15% burns.
    Answer:
    Admit patient to burns ward
    ABCs
    - Secure airway- suction any secretions, assess for need for intubation or tracheostomy
    - Ensure child is breath and give child oxygen
    - Gain venous access (cannulate child)- send blood for group and save, Full blood count, Urea, Electrolytes and Creatinine, and Liver enzymes.

    Catheterize patient, monitor urine output (input/output chart) and Start fluids

    - Resuscitation fluid
        - Volume in first 24 hours= 4ml x 15 x (estimated weight of child approximately=20kg)
        - Volume in first 24 hours= 1200ml
        - Give 600ml in first 8 hours from time of burn injury and 600ml in next 16hours
        - Fluid of choice= Ringer's lactate

- Maintenance fluid
  - Volume= (100 x 10) + (50 x 10)
  - Volume in next 24 hours= 1500ml
  - Fluid of choice Dextrose 5%

Drugs given

- Analgesia
- Tetanus toxoid 0.5ml
- Broad spectrum antibiotics

Monitor vitals hourly

Daily wound cleaning with application of silver sulphadiazine

26. Write short notes on flail chest.

27. The signs of intracranial bleeding after a head injury are
    F. Conscious level .....
    G. Pupil on side of lesion .....
    H. Pulse rate .....
    I. Blood pressure .....
    J. Respiration .....

28. A 65-year-old patient presents with a painful ulcer on the dorsum of his foot. He complains that he is unable to sleep at night and the pain is not relieved by moderate pain killer

    A. List 2 differential diagnosis for this ulcer

    Answer:

    - Ischemic ulcers (arterial ulcer)
    - Venous ulcer

    B. What is the most likely diagnosis?

    Answer: Ischemic ulcer

    C. What 4 important things would you ask in the history to determine the etiologic factors?

Answer:

- Medical history of diabetes and hypertension
- History of stroke, myocardial infarction
- Intermittent claudication
- Diet/Cholesterol levels

D. What 2 blood investigations are indicated?

Answer:

- Random blood sugar
- Cholesterol levels

E. What radiological investigation is indicated?

Answer: Doppler ultrasound

F. Describe the following terms: a. Ankle Brachial pulse index and b. critical ischemia?

Answer

- Ankle brachial pulse index is the ratio of brachial pressure over that of the ankle pressure. It is usually 1
- Critical ischemia is present when this ratio is less than 0.6

29. A 35-year-old patient is seen in casualty following a road traffic accident (RTA). The patient smells of alcohol and has a laceration on the occipital area. Answer the questions below briefly.
    E. What two things would you ask in the history?
    F. Explain how you would examine the occipital laceration
    G. List 4 localizing signs you would look for
    H. List 4 reasons you might ask for a CT scan in this patient

31. List the various indications for doing a colostomy

32. Discuss in brief the causes of surgical jaundice

33. Write short notes about Steel's rule application in a patient with cervical spine injury

34. A 34 year old woman presents with mild yellowness of eyes, pain in the right hypochondrium and fever. She gives history of previous episodes which resolve on antibiotic treatment. Answer the questions below:
    G. What is the most likely diagnosis?
    H. What is the most likely cause?
    I. What other 2 things would you ask for in the history?
    J. What 2 blood investigations would you ask for?
    K. What 2 signs would be present on abdominal examination?
    L. What radiological investigation is indicated?

35. Answer the following questions:
    C. Outline briefly the operative blood transfusion check list
    D. Discuss measures that you would take to treat an adverse blood reaction

36. Answer the following questions
    E. Define goiter
    F. Discuss briefly pre-operative investigations for goiter
    G. List possible post-operative complications of thyroidectomy
    H. How would you recognize and abort a thyroid storm

37. Give an account of thyroid swellings

38. A 46-year-old Zambian female presents to clinic 4 with a thyroid swelling which is painless and slow growing.
    F. What 4 things would you ask in the history to determine the cause?
    G. What 4 examinations would you do to determine whether the patient is euthyroid or toxic?
    H. What specific blood tests are indicated?
    I. What 3 radiological investigations would you do?
    J. List 4 indications for surgery in this patient

39. Answer the following questions
    E. Define osteoarthritis
    F. Discuss briefly etiological factors in general and clinical presentation of osteoarthritis of lumbar spine
    G. List the four classical radiological findings in osteoarthritis

H. List the treatment modalities available for osteoarthritis.

40. Define the following
    F. Fistula
    G. Fissure
    H. Ulcer
    I. Granulation tissue
    J. Paralytic ileus

41. Write short notes on the following
    C. Define sentinel lymph node
    D. Discuss briefly value of sentinel axillary lymph node biopsy in management of breast cancer

42. A 37-year-old female patient is seen in clinic 4 with a lump in the breast and an axillary swelling. She gives history of weight loss and back ache. Answer the questions below.
    G. List 2 differential diagnosis
    H. What is the most likely diagnosis?
    I. What 4 important questions would you ask to determine the possible etiological factors?
    J. List 2 investigations you would do to confirm your diagnosis
    K. What 2 radiological investigations would you do to determine whether metastasis is present?
    L. What is the value of the sentinel node in this condition?

43. A 56-year-old patient is seen in casualty with history of failing to pass urine. He has severe pain and abdominal distention.
    F. What is the diagnosis?
    G. List 2 possible conditions that can lead to this complication
    H. List 4 questions you would ask to differentiate the 2 conditions
    I. What single physical examination is necessary in this patient?
    J. What one endoscopy and one radiological investigation should be done?

44. A 40-year-old man comes to the emergency ward with acute urinary retention. What are the possible etiologies, briefly outline how you would investigate.

45. A 34-year-old woman presents with mild yellowness of eyes, pain in the right hypochondrium and fever. She gives history of previous episodes which resolve on antibiotic treatment. Answer the questions below
    G. What is the most likely diagnosis?
    H. What is the most likely causes?
    I. What other 2 things would you ask for in the history?
    J. What 2 blood investigations would you ask for?
    K. What 2 signs would be present on abdominal examination?
    L. What radiological investigation is indicated?

46. A beautiful 40-year-old female patient is seen in casualty with right upper quadrant pain which radiates to the scapula. She gives history of intermittent attacks associated with fatty foods. On examination Murphy's sign and Moynihan's sign are both positive. The SHO on call makes a diagnosis of Acute pancreatitis and requests some blood tests. The result shows serum amylase 100 u/L, Alkaline Phosphates 300 u/L, SGPT 30u/L, SGOT 31u/L and bilirubin 25 micronmols/L. The SHO is uncertain of the interpretation of the results and calls the medical Registrar on call. The Registrar takes the history on the phone. The medical Registrar is extremely upset with the young SHO, but nonetheless recommends IV antibiotics, analgesia and an urgent U/S. By the morning round the SHO has altered his diagnosis and takes the credit for an astute diagnosis from the newly appointed Prof of Surgery.
    G. What other important history should be taken?
    H. What diagnosis do both the history and examination point to?
    I. What do the investigations show?
    J. Is a plain X-ray indicated in this patient?
    K. Why was the medical registrar so upset?
    L. What were the surgical principles on which the medical registrar gave his advise?

47. Answer the following questions:

A. Define compartment syndrome and list major etiological factors

Answer:

Compartment syndrome is an entity that results from increased intra-compartmental pressure within a limited space compromising the circulation and function of tissues within that closed space. It is characterized by severe, progressive and persistent pain that poorly responds to analgesics and is aggravated by passive muscle stretch as well as paresthesia, paralysis, pulselessness, pallor and poikilothermia.

Major etiological factors include:

- Trauma:
  Fractures of the lower limb or upper limbs,
  Soft tissue crush injuries
  Vascular injuries
  Circumferential burns affecting a compartment,
  High velocity injuries: gun injuries
  Snake bikes
- Iatrogenic: application of tight dressings/plasters
- Lying on one limb in comatous patients

B. Discuss briefly the pathophysiology of compartment syndrome

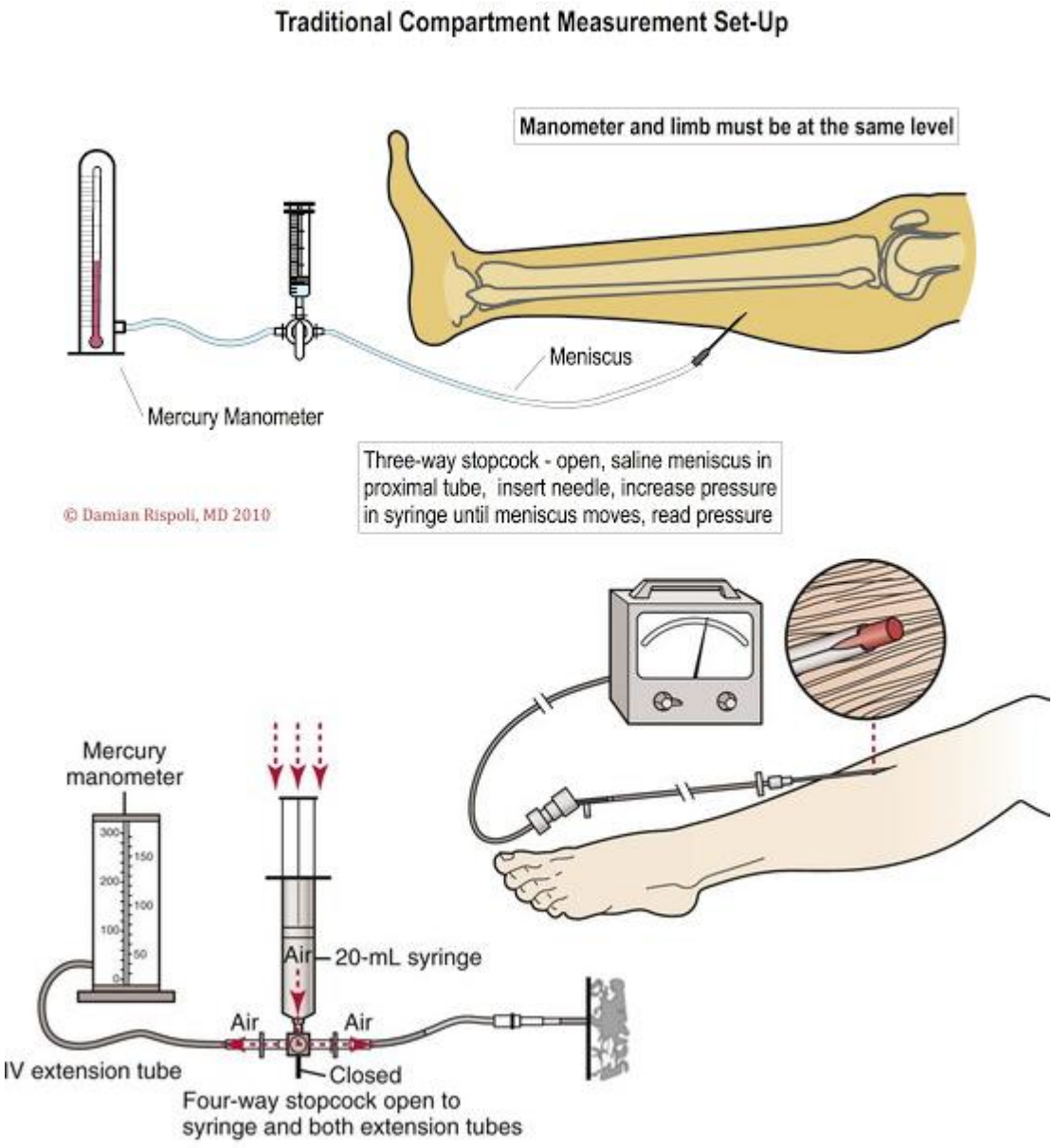
Answer:

Edema that is often a sequela of trauma occurring in a limited anatomical space raises the tissue intra-compartmental hydrostatic pressure. As tissue pressure rises, the pressure on and within the local venous system also rises. Arterial resistance subsequently increases and inflow is reduced. If untreated there is vascular compromise and the enclosed muscle becomes ischemic. Prolonged ischemia results in necrosis and ultimately, fibrosis of the muscle and surrounding soft-tissue structures. As pressure increases there is loss of sensation in the distribution of the involved nerves and ultimately the local pulses are lost (though sometimes the pulse may be present).

C. With aid of labelled diagram, explain Whitesides saline/needle technique for measuring intracompartment pressure in a limb

Answer:

Intracompartment pressure measured using the Whitesides saline/needle technique uses an 18G needle connected via a 3 way stop cock to an air filled 20ml syringe. Air filled tubing is connected to a mercury manometer and a small amount of saline sits in the tube connected to the needle. Compression of the syringe raises the pressure till saline flows into the compartment (this is indicated by meniscus moving). Below is a diagram illustrating this



48. Write short notes on assessment of a patient with burns

Answer:

- History:
  - Time and mechanism of the injury
    - Time the person was burnt
    - Type of burn i.e. scald (what was the liquid, if tea/coffee was there milk in it), flame, electrical (what voltage, was there a flash/arching, what was the contact time) or chemical (Acid or alkaline, how much exposure?)
  - Circumstances around the burn
    - How was the person put out?
    - How long were they alight for?
    - What first aid was carried out? If cooling was performed, what with and for how long?
    - Concomitant injuries (i.e. fall from height, road traffic accident, explosions)
    - Likelihood of inhalational injury (did the event occur in an enclosed space?)
    - Any comorbidities present
    - Any suspicion of non-accidental injury?
- Physical assessment:
  - Assess burn percentage and severity using:
    - Lund and Browder chart (most accurate)
    - Wallace Rule of nines (in adults): Head and neck (9%), Upper limbs (9% each), Lower limbs (18% each) Trunk-36% (18% in front and 18% at the back) and Perineum-1%
    - Rule of sevens (in children): Head and neck (28%), Upper limbs (7% each), Lower limbs (14% each) Trunk-28% (14% in front and 14% at the back) and Perineum-2%
    - Rule of palms (the palms represents 1% of total body surface area
  - Examine systems:
    - Neurological examination: look for alteration of mental status (confusion)
    - Respiratory examination: look out for signs of inhalational burns (soot in mouth or nostrils, burnt nasal hair, stridor, respiratory distress, circumferential burns on chest)

- Cardiovascular system: look for signs of shock (tachycardia, hypotension)
- GIT examination
- Musculoskeletal and skin: look for fractures and assess skin for burn percentage area

- Investigations:
  - Full blood count to pick up any cytopenias
  - Group and save/ Cross match
  - Urea, electrolytes and creatinine to assess renal function and electrolyte imbalance
  - Liver enzymes and function tests
  - Pus swab
  - For electrical burns: 12 lead ECG monitoring and cardiac enzymes
  - For inhalation burns: chest X-ray and Arterial blood gases

49. Write short notes on usefulness of intravenous urogram (IVU) in a patient with bilateral hydronephrosis

50. Describe the steps involved in the synthesis of Tri-iodothyronine (T3) and thyroxine (T3). Comment on homeostatic (regulatory) feed-back loop

51. Answer the following
    D. Define osteoarthritis
    E. Discuss briefly pathogenesis of degenerative osteoarthritis
    F. List the four cardinal radiological features of osteoarthritis

52. List baseline investigations and their significance in preparation of a patient due for thyroidectomy

53. Answer the following questions
    D. Describe phases of fracture healing in a long tubular bone
    E. List in tabular form complications associated with fractures
    F. List the methods available for reduction of fractures

54. Write short notes on:
    D. Autologous blood transfusion, types, uses and limitations
    E. Pre-operative blood transfusion check list

F. Steps to be taken in aborting hemolytic blood transfusion reaction

55. Write short notes on how you would manage a patient you found to have a ruptured liver during an operation in a district hospital.

56. Discuss the various hormones that are involved following the ruptured spleen in a 25-year-old man.

57. A 47-year-old female patient is admitted to casualty with swelling of her left leg of spontaneous onset. She has a fever and the leg feels warm

A. List 2 differentials diagnosis

Answer:

Cellulitis

Deep vein thrombosis

B. What bacteria is likely to be the cause?

Answer: Streptococcus pyogenes

C. What 2 important questions would you ask for in the history to help determine the etiological factors?

Answer:

- Past medical history of diabetes/immunosuppression
- History of trauma/injury

D. What 4 signs would you look for in the examination?

Answer:

- Swelling
- Hotness
- Redness
- Pain

E. What 2 blood investigations would you do?

Answer:

- Full blood count with differential count
- Blood culture/C-reactive protein

F. What antibiotic is indicated and what route of administration is recommended?

Answer: Penicillins intravenous

58. A 15-year-old boy presents to the casualty department with scrotal pain. He gives a history of sudden onset with no history of trauma. Answer the questions below.

A. What are the 2 main differentials?

Answer:
Testicular torsion
Epididymorchitis

B. What 4 things would you ask for in the history to differentiate the two?

Answer:

- History of nausea and vomiting
- Previous episode
- Precipitating factors e.g. sex, masturbation, pornographic materials
- History of urethritis

C. What is the most likely diagnosis?

Answer: Testicular torsion

D. What is Prehn's test? Describe how it is done.

Answer: Prehn's test is done by elevation of the testis. In testicular torsion the pain increase whilst in epididymitis it reliefs the pain

E. What radiological investigation is indicated?

Answer: Color doppler ultrasound

59. A 12-year-old boy presents to clinic 3 with a swelling on the upper leg which is fast growing. The child is ill and give history of weight loss

A. List 2 differential diagnosis

Answer:
Osteosarcoma
Osteomyelitis

B. What radiological investigation might distinguish the two?

Answer: Plain X-ray

C. What other radiological investigation can be done?

Answer: Technetium bone scan

D. List 2 X-ray findings in osteosarcoma
Answer:
Codman triangle
Sunray appearance/spicules

60. A game ranger is brought to the casualty with a gunshot wound to the right leg. The wound is dirty and 5cm in length. The patient has palpable pulses and nerve function is normal.

A. What type of fracture has the patient had?
Answer: Open tibia fracture

B. What is the classification of open tibia fractures?
Answer:
This is the Gustillo-Anderson classification. It consists of 3 types
Type 1: Wounds are <1cm and clean (mild contamination), simple fracture pattern and there is no skin crushing
Type 2: Wounds measuring 1-10 cm but without significant soft tissue crushing, flaps, degloving or contusion. Fracture pattern is more complex and there is moderate contamination
Type 3: Wounds >10cm, also includes injuries older than 8 hours. There is severe contamination. It has 3 subtypes
- Type 3A: no periosteal stripping
- Type 3B: periosteal stripping
- Type 3C: neurovascular damage

C. How would you classify this patient's fracture?
Answer: Type 3B

D. What blood investigations would you do?
Answer:
- Full blood count with differential white cell count
- Blood cultures

E. What 2 organisms would be present on pus swab of this wound?
Answer:

- Anaerobic *Clostridium tetani*
- Bacteroides

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