NEET PG 2021

200 Question Recall and Explanation

- All 200 Questions with options
- In Depth explanation of each question
- Reference from Standard Textbooks
- Easy to read concepts
- Visual representations
- Predictive Questions

NEET PG

Recall 2021

We hope you enjoy using these notes

They have been hand-crafted with an obsessive attention to details, with aim of capturing the course's content in a way that's right for you.

Thank You

For the Students
from the Students

notespaedia

HOW TO USE THIS NOTE

High-Yield Series

- High-Yield content specifically designed for rapid revision
- Clinical based questions included
- Clinical formulas included
- Topics based on Previous Question papers included

CONGRATULATIONS

You now posses the book that will guide Medical PG Aspirants to score the best rank in PGMEE. With appropriate care, the binding should last the useful life of the book. Keep in mind that putting excessive flattening pressure on any binding will accelerate its failure. If you purchased a book that you believe is defective, please **immediately** return it to the place of purchase.

FOR BETTER RESULTS

Use as revision book :

Use this notes for revision as a supplement to your primary resource. These notes are specifically designed for active recall and visual memory for reinforcing the revision strategy. Best results are gained on quick recap a week before exam.

Consider *HIGH -YIELD NOTES* as your annotation hub :

Annotate material from your primary resource, such as lecture class or videos, into your notes. We have already referred and incorporated almost all information from the available sources. This will keep all the high-yield information you need in one place.

BOX ANNOTATION :

We have introduced concept of Box annotation

1. **RED BOX :** Includes the most repeated topic along with the next potential MCQ that we believe will be tested in future exam
2. **BLUE BOX :** Includes Previous AIIMS/ NEET PG /INICET recalls

COLOR CODE:

The notes are specifically color coded as follows

1. **BLACK :** Definition
2. **RED :** Clinical features
3. **BLUE:** Pathogenesis / Mechanism of Action
4. **VIOLET:** Investigation, Radiography and Lab Diagnosis
5. **GREEN:** Treatment

Prime Your Memory :

The visual representation of concepts incorporated into the notes serve as a useful way of retaining key associations and keeping high-yield facts fresh in your memory just prior to exams.

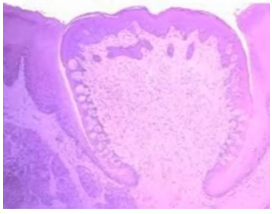
CONTRIBUTE TO ELITE NOTES :

No resource is error free and we do not guarantee the notes are free of errors. If you feel you want to add more contents or wish to be a part of Content Dev please contact us at **support@notespaedia.com**

Index

Anatomy

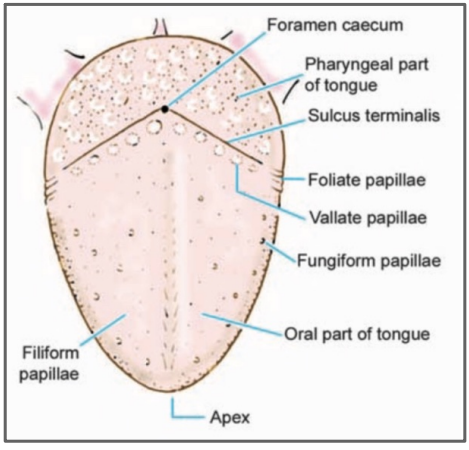
1. Given is a microscopic image of tongue. Identify the papillae.



A. Circumvalate
B. Fungiform
C. Filliform
D. Foliate

LINGUAL PAPILLAE

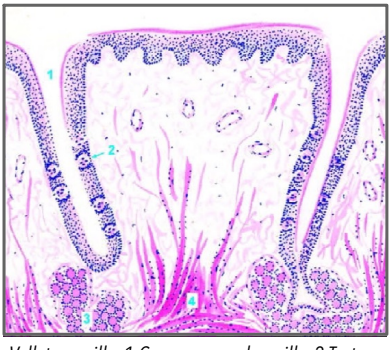
- Lingual papillae (singular papilla) are the small, nipple-like structures on the upper surface of the tongue that give it its characteristic rough texture.
- The four types of papillae on the human tongue have different structures and are accordingly classified as:
  - CIRCUMVALLATE (OR VALLATE),
  - FUNGIFORM,
  - FILIFORM,
  - FOLIATE.



(a) The most numerous papillae are small and conical in shape. They are called filiform papillae. The epithelium at the tips of these papillae is keratinized. It may project in the form of threads.

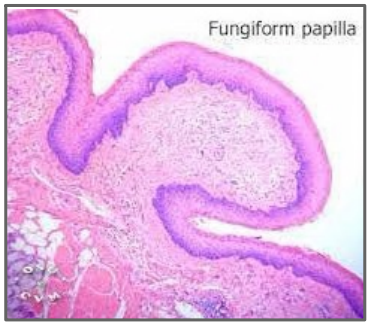
(b) At the apex of the tongue, and along its lateral margins there are larger papillae with rounded summits and narrower bases. These are called fungiform papillae. Fungiform papillae bear taste buds. In contrast to the filiform papillae the epithelium on fungiform papillae is (as a rule) not keratinized.

(c) The largest papillae of the tongue are called circumvallate papillae. They are arranged in a row just anterior to the sulcus terminalis. When viewed from the surface each papilla is seen to have a circular top demarcated from the rest of the mucosa by a groove. In sections through the papilla it is seen that the papilla has a circumferential 'lateral wall' that lies in the depth of the groove.Taste buds are present on this wall, and also on the 'outer' wall of the groove.

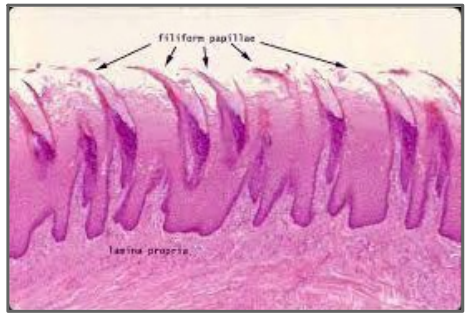


Vallate papilla. 1-Groove around papilla. 2-Taste bud. 3- Serous glands of Von Ebner. 4-Muscle extending into papilla.

Reference: HUMAN HISTOLOGY INDERBIR SINGH pg 232
Reference: Notespaedia Anatomy image bank, Pg 59



Fungiform papillae



Filiform papillae

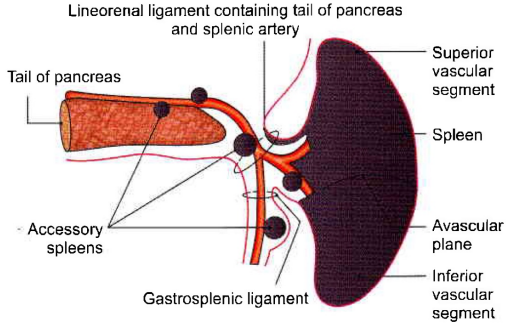
2. A patient of splenomegaly reaching 15 cm costal margin. Which structure prevents spleen from vertically going down?

A. Tail of pancreas
B. Phrenicocolic ligament
C. Hilum of spleen
D. Linorenal ligament

PERITONEAL RELATIONS OF SPLEEN

The spleen is surrounded by peritoneum, and is. suspended by following ligaments.

- The gastrosplenic ligament extends from the hilum of the spleen to the greater curvature of the stomach. It contains the short gastric vessels and associated lymphatics and sympathetic nerves
- The lienorenal ligament extends from the hilum of the spleen to the anterior surface of the left kidney. It contains the tail of the pancreas, the splenic vessels, and associated pancreaticosplenic lymph nodes, lymphatics and sympathetic nerves.
- The phrenicocolic ligament is not attached to the spleen, but supports its anterior end. It is a horizontal fold of peritoneum extending from the splenic flexure of colon to the diaphragm opposite the 11th rib in the midaxillary line. It limits the upper end of the left paracolic gutter.

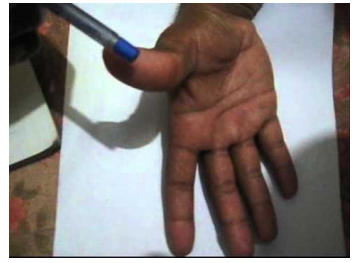


Peritoneal ligaments attached to the spleen, and common sites of accessory spleen

Reference:Human Anatomy By B D Chaurasia, lower limb and abdomen, pg 295

HEPATIC FLEXURE	SPLENIC FLEXURE
• Placed anteriorly on right side • Right angle • Lies on right kidney • Supplied by right colic aftery • No ligament is attached • Lies at level of L2 vertebra	• Placed posteriorly on left side • Acute angle • Lies on spleen • Supplied by left colic artery • Phrenicocolic ligament is attached • Lies at level of T12 vertebra

3. Image of pen test is given. Identify the nerve being tested.



A. Median nerve
B. Ulnar nerve
C. Posterior Interosseous nerve
D. Musculocutaneous nerve

MEDIAN NERVE INJURY

MAJOR MOTOR BRANCHES

- In the arm ——— Nil
- In the forearm ——
  - Proximal 1/3 — All the flexor muscles of the forearm, except the flexor carpi ulnaris and medial-half of the flexor digitorum profundus
  - Distal 1/3 ——— Nil
- In the hand ——— Thenar muscles* (three), First two lumbricals

The three muscles are flexor pollicis brevis, opponens pollicis and abductor pollicis.

TESTS FOR MEDIAN NERVE

BENEDICTION TEST

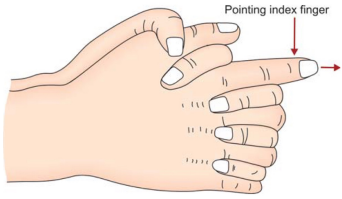
The patient is unable to flex the index and middle finger on lifting the hand

PEN TEST

The patient is unable to touch the pen due to the loss of action of abductor pollicis brevis

Reference:Human Anatomy By B D Chaurasia, upper limb pg 110
Reference: Notespaedia Orthopaedics image bank, Pg 51

POINTING INDEX OR OSCHNER'S CLASP TEST



When both the hands are clasped together, index and middle fingers, fail to flex due to the loss of action of long finger flexors of the index and middle fingers, which are supplied by the median nerve

4. A small boy with multiple humerus fractures following which there is loss of sensation over lateral side of forearm, difficulty in Flexion of elbow and supination of forearm.
Which nerve is involved?

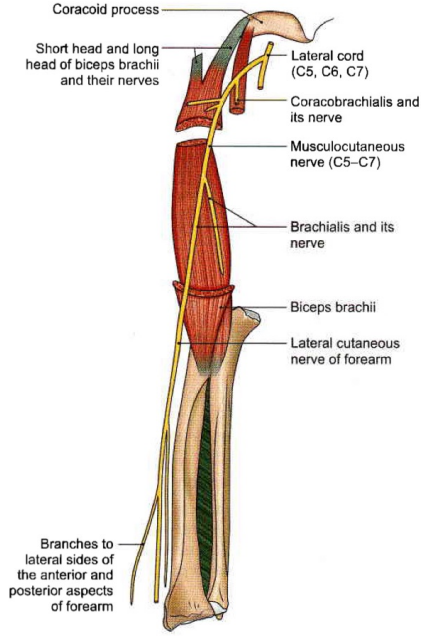
A. Median nerve
B. Musculocutaneous nerve
C. Axillary nerve
D. Radial nerve

MUSCULOCUTANEOUS NERVE

The musculocutaneous nerve is the main nerve of the front of the arm, and continues below the elbow as the lateral cutaneous nerve of the forearm

It is a branch of the lateral cord of the brachial plexus, arising at the lower border of the pectoralis minor in the axilla

ROOT VALUE : ventral rami of C5-C7 segments of spinal cord.



ORIGIN, COURSE OND TERMINATION

- Musculocutaneous nerve arises from the lateral cord of brachial plexus in the lower part of the axilla.
- It accompanies the third part of the axillary artery.
- It then enters the front of arm, where it pierces coracobrachialis muscle.
- Then it runs downwards and laterally between biceps brachii and brachialis muscles to reach the lateral side of the tendon of biceps brachii.
- It terminates by continuing as the lateral cutaneous nerve of forearm 2 cm above the bend of the elbow

Reference:Human Anatomy By B D Chaurasia, upper limb, pg 174

BRANCHES AND DISTRIBUTION

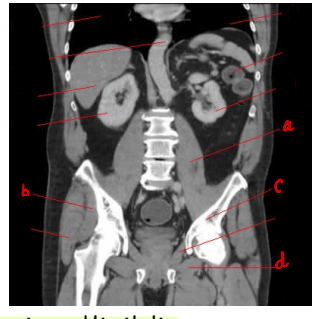
- Muscular: It supplies the following muscles of the front of the arm.
  - i. Coracobrachialis
  - ii. Biceps brachii,long and short heads
  - iii. Brachialis
- Cutaneous: Through the lateral cutaneous nerve of the forearm it supplies the skin of the lateral side of the forearm from the elbow to the wrist including the ball of the thumb
- Articular branches:
  - i. The elbow joint through its branch to the brachialis.
  - ii. The shoulder joint through a separate branch which enters the humerus along with its nutrient artery.
- Communicating branches : The musculocutaneous nerve through lateral cutaneous nerve of forearm communicates with the neighbouring nerve, namely the superficial branch of the radial nerve, the posterior cutaneous nerve of the forearm, and the palmar cutaneous branch of the median nerve.

CLINICAL ANATOMY

Injury causes:

- Motor functions – coracobrachialis, biceps brachii and brachialis muscles are affected:
  - Flexion at the shoulder and elbow are weakened but can still be performed by the pectoralis major and brachioradialis respectively.
  - Supination of the forearm is weak, but can still be performed by the brachioradialis.
- Sensory functions – loss of sensation over the lateral side of the forearm.

5. A child was brought to the hospital by his father with complaint of fever, low backache and persistent Flexion of the hip joint. He had history of spine TB in the past. On examination child has inguinal swelling. Identify the marked muscle responsible to be involved.



A. a
B. b
C. c
D. d

The marked structure 'a' is Psoas major. Fever, abdominal or back pain, and limitation of hip joint movement comprise the classic triad of psoas abscess. Mycobacterial psoas abscess is typically associated with spinal tuberculosis due to the extension of infection from lumbar vertebrae.

Reference:Human Anatomy By B D Chaurasia,abdomen pg 343

Psoas abscess formed due to tubercular infection of lumbar vertebrae can track down between psoas major muscles and its fascia to reachbehind the inguinal ligament into the femoral triangle. It may be mistaken for enlarged lymph nodes

PSOAS MAJOR

This is a fusiform muscle placed on the side of the lumbar spine and along the brim of the pelvis. The psoas and the iliacus are together known as the iliopsoas, due to their common inseftion and actions

ORIGIN

a. From anterior surfaces and lower borders of transverse process of all lumbar vertebrae
b. By 5 slips, one each from the bodies of two adjacent vertebrae and their intervertebral discs, from vertebrae, T12 to L5
c. From 4 tendinous arches extending across the constricted parts of the bodies of lumbar vertebrae, between the preceding slips. The origin is a continuous one from the lower border of T12 to upper border of L5

INSERTION

- The muscle passes behind the inguinal ligament and in front of the hip joint to enter the thigh.
- It ends in a tendon which receives the fibres of the iliacus on its lateral side.
- It is then inserted into the tip and medial part of the anterior surface of the lesser trochanter of the femur

NERVE SUPPLY

- Branches from the roots of spinal nerve L2, L3 and sometimes L4.

ACTIONS

1. With the iliacus, it acts as a powerful flexor of the hip, joint as in raising the trunk from recumbent to sitting posture
2. Helps in maintaining stability at the hip. Balances the trunk while sitting
3. When the muscle of one side acts alone, it brings about lateral flexion of the trunk on that side
4. It is a weak medial rotator of the hip. After fracture of the neck of the femur, the limb rotates laterally

CLINICAL ANATOMY

- The psoas is enclosed in the psoas sheath, a parl of the lumbar fascia. Pus from tubercular infection of the thoracic and lumbar vertebrae may track down through the sheath into the thigh, producing a soft swelling in the femoral triangle.
- The typical posture of a laterally rotated lower limb following fracture of the neck of the femur is produced by contraction of the psoas muscle.

6. Deoxygenated blood is returned to the placenta from the fetus through ?

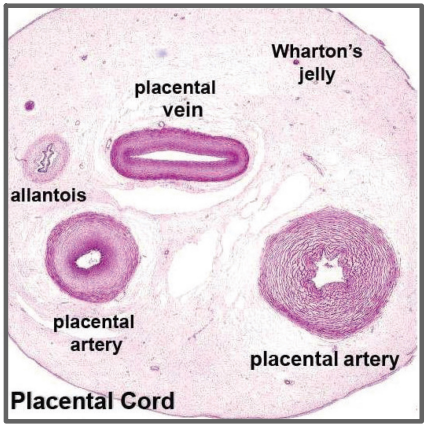
A. Umbilical artery

B. Umbilical vein

C. Ductus arteriosus

D. Ductus venosus

UMBILICAL CORD



- Develops from extraembryonic mesoderm
  1. Two umbilical arteries
  2. One umbilical vein (left)
  3. Wharton's jelly
  4. Remains of allantoic diverticulum
  5. Remains of vitellointestinal duct (remnant ofyolk sac)
- Umbilical arteries carry deoxygenated blood from fetus to the placenta
- Umbilical vein carries oxygenated blood from the placenta to the fetus.

CORD PROLAPSE → COMPRESSION → FETAL HYPOXIA

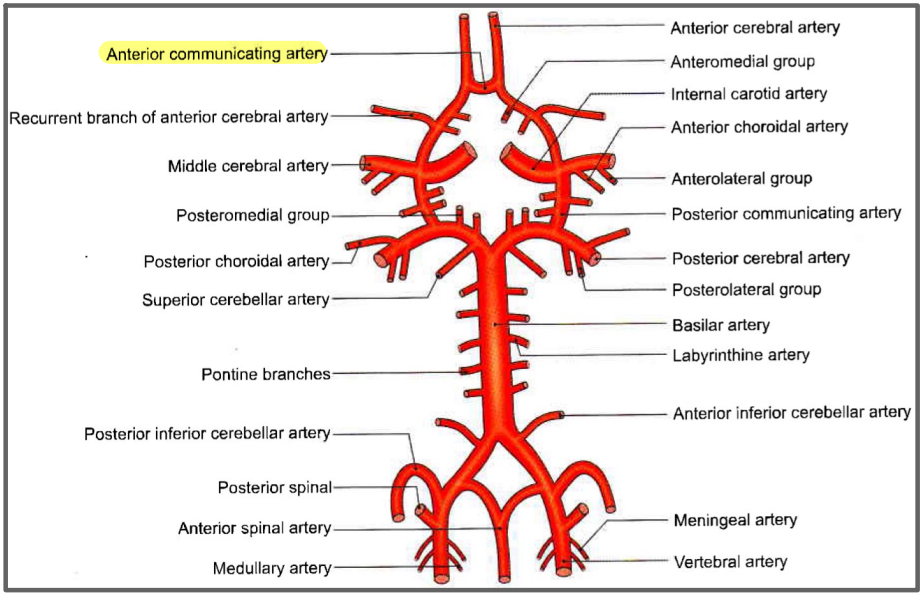
Fetal structure	Remnants after birth
1. Umbilical vein	Ligamentum teres hepatis
2. Ductus venosus	Ligamentum venosum
3. Foramen ovale	Fossa ovalis
4. Ductus arteriosus	Ligamentum arteriosum
5. Right and left umbilical arteries	• Superior vesicle arteries • Medial umbilical ligaments

Shunt	Functional closure	Anatomical closure
Ductus arteriosus	10 – 96 hrs after birth	2 – 3 wks after birth
Formamen ovale	Within several mins after birth	One year after birth
Ductus venosus	Within several mins after birth	3 – 7 days after birth

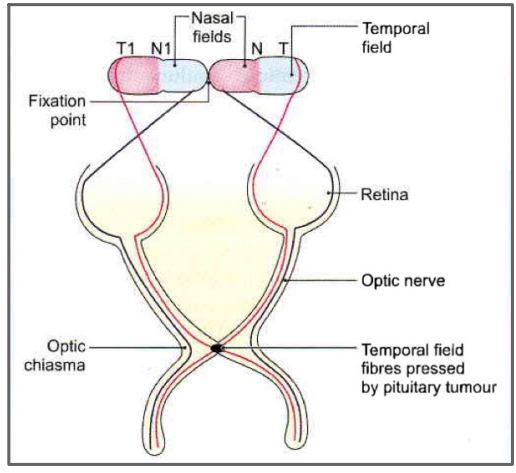
7. A patient is having visual disturbances. On investigating a vessel developed aneurysm on base of brain which was compressing optic chiasma. Involved vessel is?

A. Anterior cerebral artery
B. Anterior choroidal artery
C. Anterior communicating artery
D. Posterior communicating artery

CIRCLE OF WILLIS (CIRCULUS ARTERIOSUS)



- It is an arterial circle, situated at the base of brain in the interpeduncular fossa. It is formed by the anterior and middle cerebral branches of internal carotid and the posterior cerebral branches of basilar artery.
- The two anterior cerebral arteries are connected by anterior communicating artety; the middle and posterior cerebral arteries of same side are united by the posterior communicating artery
- Pressure over the central part of optic chiasma causes bitemporal hemianopia



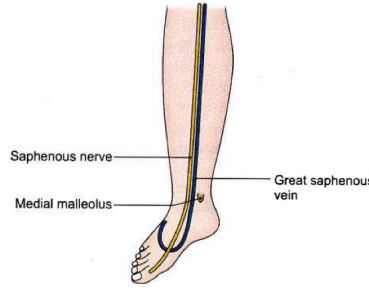
Reference:Human Anatomy By B D Chaurasia, pg 459
Reference: Notespaedia Anatomy image bank , pg 123

8. A patient underwent CABG for which saphenous vein graft taken and during surgery GSV removed. The complication that can occur is ?

A. Saphenous nerve injury
B. Sural nerve injury
C. Common peroneal nerve injury
D. Deep peroneal nerve injury

SAPHENOUS NERVE

- The saphenous nerve (L3, L4) is a branch of the posterior division of the femoral nerve.
- It arises in the femoral triangle.
- It pierces the deep fascia on the medial side of the knee between the sartorius and the gracilis, and descends close to the great saphenous vein.
- It supplies the skin of most of the medial area of the leg, and the medial border of the foot up to the ball of the big toe.
- During venesection this nerve should not be injured.
- "Cut open procedure"/venesection is done on the great saphenous vein as it lies in front of medial malleolus. This vein is used for transfusion of blood/ fluids in case of non- availability or collapse of other veins. Saphenous nerve is identified and not injured as it lies anterior to the great saphenous vein

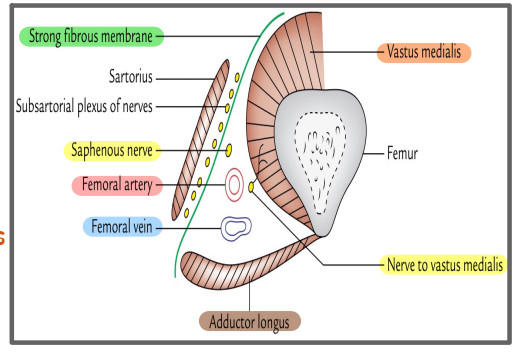


ADDUCTOR CANAL

SUBSARTORIAL CANAL/ HUNTER'S CANAL

CONTENTS

1. Femoral artery.
2. Femoral vein.
3. Saphenous nerve.
4. Nerve to vastus medialis.



BOUNDARIES

- Anterolateral wall: vastus medialis
- Posterior (floor): adductor longus
- Medial (roof): strong fibrous membrane overlapped by the sartorius muscle.

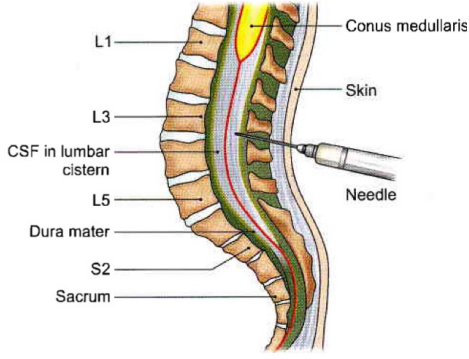
Reference:Human Anatomy By B D Chaurasia, lower limb, pg 56
Reference: Notespaedia Anatomy image bank , pg 66

9. Last structure encountered during lumbar puncture?

A. Ligamentum flavum
B. Dura
C. Arachnoid
D. Pia

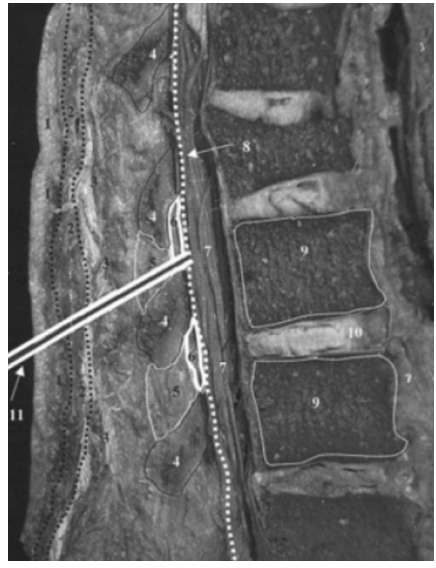
LUMBAR PUNCTURE

- **Lumbar puncture in adult:**
  Patient is lying on side with maximally flexed spine.
  A line is taken between highest points of iliac spine at L4 level.
  Skin locally anaesthetized, and lumbar puncture needle with trocar inserted carefully between L3 and L4 spines.
- **Lumbar puncture in infant, children:**
  During 2nd month of life, spinal cord usually reaches L3 level. Lumbar puncture needle is introduced in flexed spine between L4 and L5.



- The Lumbar Puncture needle pierces in order:

Skin,
↓
Subcutaneous tissue,
↓
Supraspinous ligament,
↓
Interspinous ligament,
↓
Ligamentum flavum,
↓
Epidural space containing the internal vertebral venous plexus,
↓
Dura,
↓
Arachnoid
↓
Subarachnoid space.
↓
CSF



Sagittal section of lumbar vertebrae illustrating the course of the lumbar puncture needle through skin (1), subcutaneous tissue (2), supraspinous ligament (3), interspinous ligament (5) between the spinous processes (4), ligamentum flavum (6), dura mater (8), into the subarachnoid space and between the nerve roots of the cauda equina (7). Lumbar vertebral bodies (9), intervertebral disc (10), and lumbar puncture needle (11).

Reference:Human Anatomy By B D Chaurasia, head and neck, pg 185

1. What is the acid base mechanism in chronic kidney disease?

A.Metabolic alkolosis

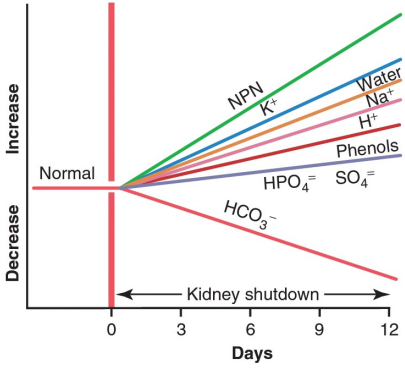
B.Metabolic acidosis

C.Respiratory acidosis

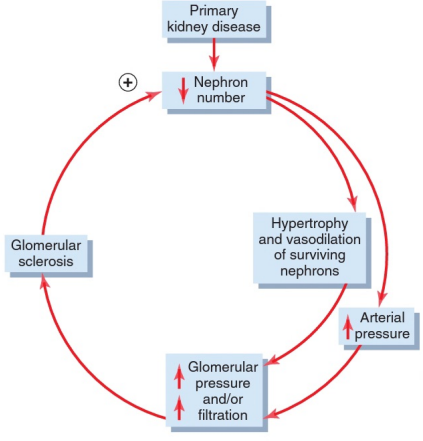
D.Respiratory alkalosis

CHRONIC KIDNEY DISEASE

- CKD is usually defined as the presence of kidney damage or decreased kidney function that persists for at least 3 months.
- CKD is often associated with progressive and irreversible loss of large numbers of functioning nephrons.



Effect of kidney failure on extracellular fluid constituents. NPN, nonprotein nitrogens.



The vicious circle that can occur with primary kidney disease. Loss of nephrons because of disease may increase pressure and flow in the surviving glomerular capillaries, which in turn may eventually injure these "normal"ncapillaries as well, thus causing progressive sclerosis and eventual loss of these glomeruli.

ACIDOSIS IN CHRONIC KIDNEY DISEASE.

- When kidney function declines markedly, there is a buildup of the anions of weak acids in the body fluids that are not being excreted by the kidneys.
- In addition, the decreased glomerular filtration rate reduces excretion of phosphates and NH4 +, which reduces the amount of HCO3 – added back to the body fluids.
- Thus, chronic renal failure can be associated with severe metabolic acidosis.

2. Receptor for absorption of glucose in GIT when person is given ORS?

A. SGLT1
B. SGLT2
C. GLUT4
D. GLUT2

SECONDARY ACTIVE REABSORPTION THROUGH THE TUBULAR MEMBRANE.

- Sodium glucose co-transporters (SGLT2 and SGLT1) are located on the brush border of proximal tubular cells and carry glucose into the cell cytoplasm against a concentration gradient.
- Approximately 90 percent of the filtered glucose is reabsorbed by SGLT2 in the early part of the proximal tubule (S1 segment), and the residual 10 percent is transported by SGLT1 in the latter segments of the proximal tubule.
- On the basolateral side of the membrane, glucose diffuses out of the cell into the interstitial spaces with the help of glucose transporters—GLUT2 in the S1 segment and GLUT1 in the latter part (S3 segment) of the proximal tubule.

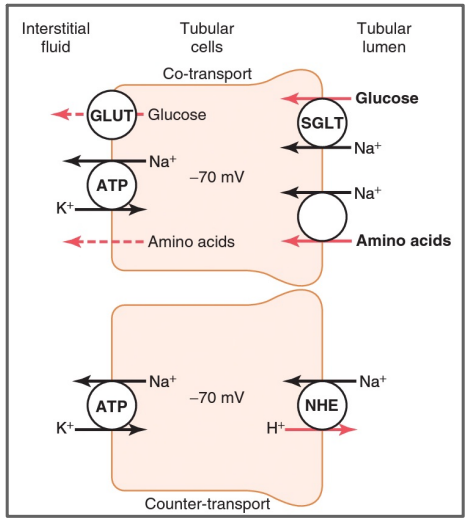


Figure shows secondary active transport of glucose and amino acids in the proximal tubule.
In both instances, specific carrier proteins in the brush border combine with a sodium ion and an amino acid or a glucose molecule at the same time.
These transport mechanisms are so efficient that they remove virtually all the glucose and amino acids from the tubular lumen.
After entry into the cell, glucose and amino acids exit across the basolateral membranes by diffusion, driven by the high glucose and amino acid concentrations in the cell facilitated by specific transport proteins.

Reference :Guyton and Hall Textbook of Medical Physiology pg 349

SODIUM GLUCOSE CO-TRANSPORTERS

- The transport of glucose and galactose is dependent on Na + in the intestinal lumen; a high concentration of Na + on the mucosal surface of the cells facilitates and a low concentration inhibits sugar infl ux into the epithelial cells.
- This is because these sugars and Na + share the same cotransporter, or symport, the sodium-dependent glucose transporter (SGLT, Na + glucose cotransporter).
- The members of this family of transporters, SGLT 1 and SGLT 2, resemble the glucose transporters (GLUTs) responsible for facilitated diff usion.
- SGLT-1 is responsible for uptake of dietary glucose from the gut.
- The related transporter, SGLT-2, is responsible for glucose transport out of the renal tubules .
- Because the intracellular Na + concentration is low in intestinal cells (as it is in other cells), Na + moves into the cell along its concentration gradient. Glucose moves with the Na + and is released in the cell .
- The Na + is transported into the lateral intercellular spaces, and the glucose is transported by GLUT 2 into the interstitium and thence to the capillaries.
- Thus, glucose transport is an example of secondary active transport.
- When the Na + / glucose cotransporter is congenitally defective, the resulting glucose/ galactose malabsorption causes severe diarrhea that is oft en fatal if glucose and galactose are not promptly removed from the diet.

GLUCOSE TRANSPORTERS

	Function	K_m (mM)[a]	Major Sites of Expression
Secondary active transport (Na^+-glucose cotransport)			
SGLT 1	Absorption of glucose	0.1–1.0	Small intestine, renal tubules
SGLT 2	Absorption of glucose	1.6	Renal tubules
Facilitated diffusion			
GLUT 1	Basal glucose uptake	1–2	Placenta, blood-brain barrier, brain, red cells, kidneys, colon, many other organs
GLUT 2	B-cell glucose sensor; transport out of intestinal and renal epithelial cells	12–20	B cells of islets, liver, epithelial cells of small intestine, kidneys
GLUT 3	Basal glucose uptake	<1	Brain, placenta, kidneys, many other organs
GLUT 4	Insulin-stimulated glucose uptake	5	Skeletal and cardiac muscle, adipose tissue, other tissues
GLUT 5	Fructose transport	1–2	Jejunum, sperm
GLUT 6	Unknown	—	Brain, spleen and leukocytes
GLUT 7	Glucose 6-phosphate transporter in endoplasmic reticulum	—	Liver

3. A 30 yr old male weighing 70kg had serum sodium of 120 meq /L. What is the total sodium deficit?

A)280meq
B)480meq
C)840meq
D)1400meq

TARGET NA: 135 - 145 mEq/L (average: 140 meq/L)

NA DEFICIT = 0.6 X WT. IN KG X (DESIRED NA – ACTUAL NA)

- Na deficit = 0.6 x (70 kg) x (140 - 120)

  = 840 mEq/L

- Correction rate: <0.5 meq/L/hr
- First 8 hrs - 50% of calculated Na
- Next 16 hrs - other 50%

Risk of development of Osmotic demyelination syndrome in rapid correction of hyponatremia

0.5 times the weight in kilos for women, 0.6 for men

4. A research fellow was studying of volumes and electrolyte the differences in different compartments. While experiment he took a sample and checked the electrolyte levels

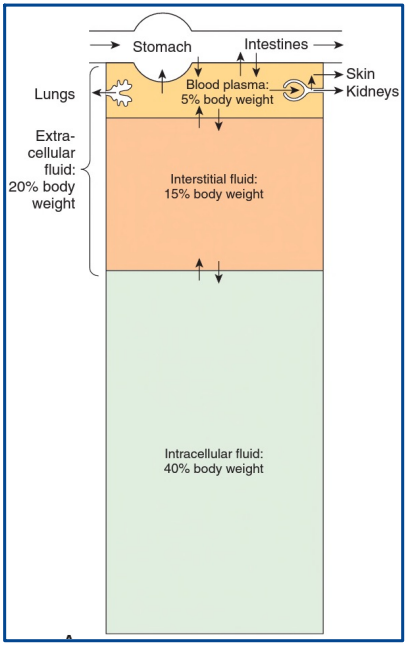
Na^+ 10
Cl^- 15
K^+ 140

indicates which compartment?

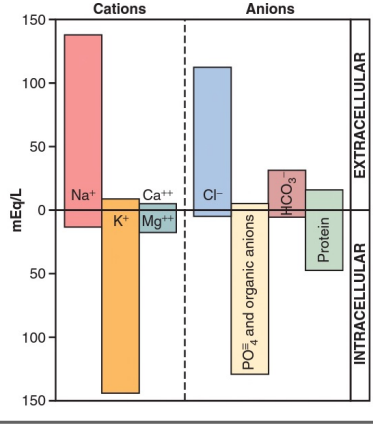
A. ICF
B. ECF
C. Interstitial fluid
D. Plasma

From the electrolyte levels, the compartment is Intracellular fluid.

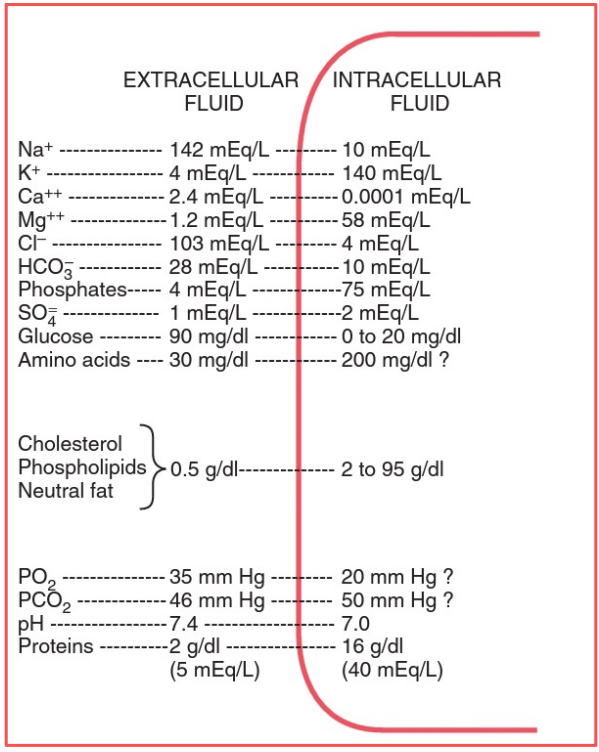
- The intracellular fluid constitutes about 40 percent of the total body weight in an "average" person.
- The two largest compartments of the extracellular fluid are the interstitial fluid, which makes up more than three fourths (11 liters) of the extracellular fluid, and the plasma, which makes up almost one fourth of the extracellular fluid, or about 3 liters.



	Plasma (mOsm/L H_2O)	Interstitial (mOsm/L H_2O)	Intracellular (mOsm/L H_2O)
Na^+	142	139	14
K^+	4.2	4.0	140
Ca^{++}	1.3	1.2	0
Mg^{++}	0.8	0.7	20
Cl^-	106	108	4
HCO_3^-	24	28.3	10
HPO_4^-, $H_2PO_4^-$	2	2	11
SO_4^-	0.5	0.5	1
Phosphocreatine			45
Carnosine			14
Amino acids	2	2	8
Creatine	0.2	0.2	9
Lactate	1.2	1.2	1.5
Adenosine triphosphate			5
Hexose monophosphate			3.7
Glucose	5.6	5.6	
Protein	1.2	0.2	4
Urea	4	4	4
Others	4.8	3.9	10
Total mOsm/L	299.8	300.8	301.2
Corrected osmolar activity (mOsm/L)	282.0	281.0	281.0
Total osmotic pressure at 37°C (mm Hg)	5441	5423	5423



Major cations and anions of the intracellular and extracellular fluids.The concentrations of Ca++ and Mg++ represent the sum of these two ions. The concentrations shown represent the total of free ions and complexed ions.



5. A 58 years old man was brought to the emergency with confusion and lethargy. His temperature is 36.7 C, blood pressure is 86/58 mmHg, pulse is 120/min, and respirations are 10/min. pH is 7.59, pCO2 49 mmHg and p02 85mm Hg. Which of the following condition best describe his acid base status?

A. Respiratory alkalosis
B. Respiratory acidosis
C. Metabolic alkalosis
D. Metabolic acidosis

ACID-BASE DISORDERS

Step 1. Check the pH
pH< 7.36, acidosis
pH> 7.44, alkalosis

Step 2. Is primary process metabolic or respiratory? For this, check pC02 and HC03

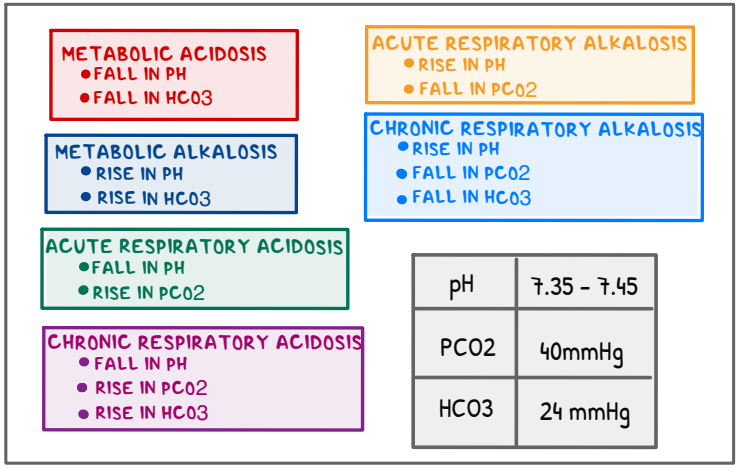
Step 3. Choose appropriate compensation for acid-base disorder

Step 4. Determine if degree of compensation is appropriate or not

Step 5. If metabolic acidosis present, calculate the anion gap

Step 6. If high anion gap acidosis, calculate delta-delta gap

Reference : Guyton and Hall Textbook of Physiology, 13th edition, pg 425



7. A 56 year old man had come to the hospital for dialysis. Before sending him for dialysis an arterial blood sample was taken for analysis. Which of the following laboratory results below indicates compensated metabolic acidosis?

A. Low PCO2, normal bicarbonate and, high pH
B. Low PCO2, low bicarbonate, low pH
C. High PCO2, high bicarbonate and, low pH
D. High PCO2, low bicarbonate and High Ph

METABOLIC ACIDOSIS

ANION GAP

Measure of acid-base balance

$AG = (NA+) - [(CL-) + (HCO3-)]$

NA+		
CL-	HCO3-	ANION GAP

MNEMONIC MUD PILES

MNEMONIC USED CARP

HIGH ANION GAP METABOLIC ACIDOSIS

1. Methanol
2. Uraemia
3. Diabetic ketoacidosis
4. Phenformin, Paraldehyde
5. INH, Iron
6. Lactic acid
7. Ethanol, Ethylene glycol
8. Salicylates

NORMAL ANION GAP METABOLIC ACIDOSIS

1. Ureteroenterostomy
2. Small bowel fistula
3. Extra chloride
4. Diarrhoea
5. Carbonic anhydride inhibitor
6. Addison's disease
7. Renal tubular acidosis
8. Pancreatic fistula

8. A young woman is found comatose, having taken an unknown number of sleeping pills an unknown time before. An arterial blood sample yields the following values: pH —7.10, HCO3- 32 meq/liter, PaCO2 68 mmHg. This patient's acid-base status is most accurately described as

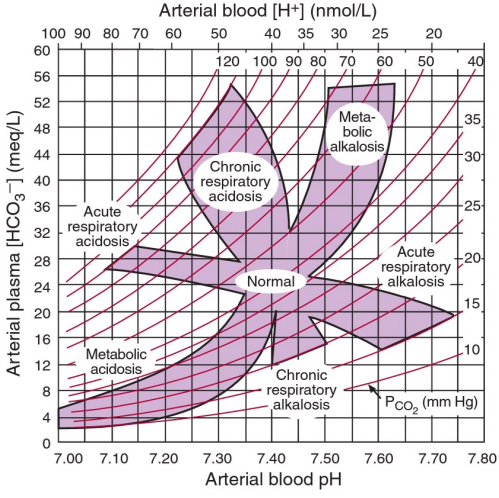
A. Uncompensated metabolic acidosis

B. Uncompensated respiratory acidosis

C. Respiratory alkalosis with partial renal compensation

D. Respiratory acidosis with partial renal compensation

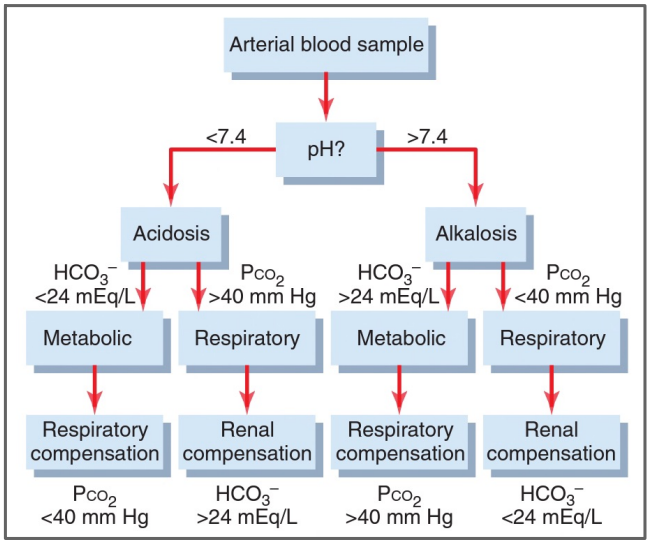
ACID–BASE NOMOGRAM



Acid-base nomogram. Changes in the PCO2(curved lines), plasma HCO 3- , and pH (or [H +]) of arterial blood in respiratory and metabolic acidosis are shown. Note the shifts in HCO 3- and pH as acute respiratory acidosis and alkalosis are compensated, producing their chronic counterparts

Condition	Arterial Plasma pH	HCO_3^- (mEq/L)	P_{CO_2} (mm Hg)	Cause
Normal	7.40	24.1	40	
Metabolic acidosis	7.28	18.1	40	NH_4Cl ingestion
	6.96	5.0	23	Diabetic acidosis
Metabolic alkalosis	7.50	30.1	40	$NaHCO_3$ ingestion
	7.56	49.8	58	Prolonged vomiting
Respiratory acidosis	7.34	25.0	48	Breathing 7% CO_2
	7.34	33.5	64	Emphysema
Respiratory alkalosis	7.53	22.0	27	Voluntary hyper-ventilation
	7.48	18.7	26	Three-week residence at 4000-m altitude

	pH	H^+	P_{CO_2}	HCO_3^-
Normal	7.4	40 mEq/L	40 mm Hg	24 mEq/L
Respiratory acidosis	↓	↑	↑↑	↑
Respiratory alkalosis	↑	↓	↓↓	↓
Metabolic acidosis	↓	↑	↓	↓↓
Metabolic alkalosis	↑	↓	↑	↑↑



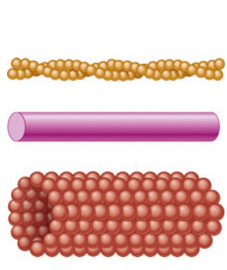
Analysis of simple acidbase disorders. If the com pensatory responses are markedly different from those shown at the bottom of the figure, one should suspect a mixed acidbase disorder.

8. A research scholar while studying cytoskeleton, came across several strands of fibrous proteins that are wound together in stable structure and distribute tensile forces across cells in the tissue. Which one of the following structure is best suited?

A. Intermediate filaments
B. Microtubules
C. Microfilaments
D. All of the above

CYTOSKELETON

- A system of fibers that not only maintains the structure of the cell but also permits it to change shape and move.
- The cytoskeleton is made up primarily of microtubules, intermediate filaments, and microfilaments, along with proteins that anchor them and tie them together.
- In addition, proteins and organelles move along microtubules and microfi laments from one part of the cell to another, propelled by molecular motors.



Cytoskeletal filaments	Diameter (nm)	Protein subunit
Microfilament	7	Actin
Intermediate filament	10	Several proteins
Microtubule	25	Tubulin

Cytoskeletal elements of the cell.

The cytoskeleton is a network of three types of filaments that provide structural integrity to the cell as well as a means for trafficking of organelles and other structures around the cell.

- Actin filaments are important in cellular contraction, migration, and signaling.They also provide the backbone for muscle contraction.
- Intermediate filaments are primarily structural.
- Microtubules provide a dynamic structure in cells that allows for the movement of cellular components around the cell.

MICROTUBULES

- Dynamic portion of the cytoskeleton.
- They provide the tracks along which several diff erent molecular motors move transport vesicles, organelles such as secretory granules, and mitochondria from one part of the cell to another.
- Long, hollow structures with 5-nm walls surrounding a cavity 15 nm in diameter.
- They are made up of two globular protein subunits: α- and β-tubulin.
- A third subunit, γ-tubulin, is associated with the production of microtubules by the centrosomes.
- The α and β subunits form heterodimers, which aggregate to form long tubes made up of stacked rings, with each ring usually containing 13 subunits.
- The tubules interact with GTP to facilitate their formation.

INTERMEDIATE FILAMENTS

- 8–14 nm in diameter and are made up of various subunits.
- Some of these filaments connect the nuclear membrane to the cell membrane.
- They form a flexible scaffolding for the cell and help it resist external pressure.
- In their absence, cells rupture more easily, and when they are abnormal in humans, blistering of the skin is common.
- The proteins that make up intermediate filaments are cell-type specifi c, and are thus frequently used as cellular markers.

MICROFILAMENTS

- Long solid fi bers with a 4–6 nm diameter that are made up of actin.
- Although actin is most often associated with muscle contraction, it is present in all types of cells.
- It is the most abundant protein in mammalian cells, sometimes accounting for as much as 15% of the total protein in the cell.

Reference:Ganong's Review of Medical Physiology pg 40

9. Patient presents with breathing with generalized weakness. On auscultation a mid-diastolic murmur with prominent 'a' waves are seen in?

A. TS

B. MS

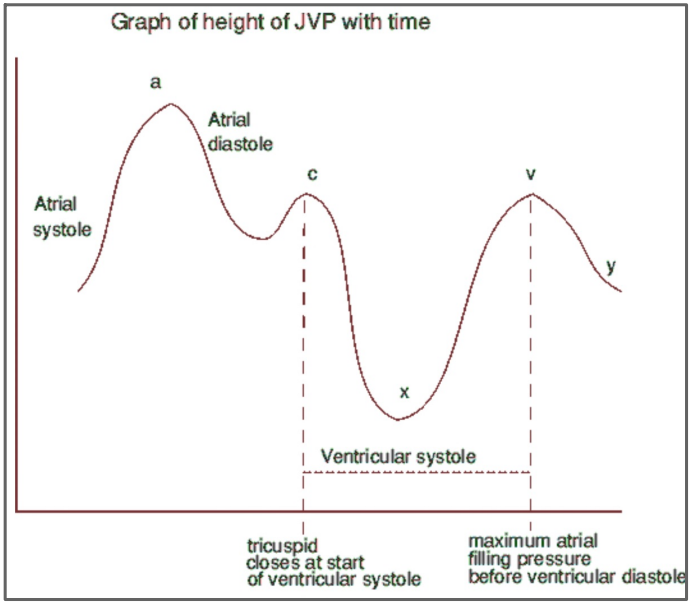
C. MR

D. TR

JVP

Jugular Venous Pulse: defined as the oscillating top of vertical column of blood in right IJV that reflects pressure changes in Right Atrium in cardiac cycle.

Jugular Venous Pressure: Vertical height of oscillating column of blood.



- a' wave:
  - Prominent:
    1. RV hypertrophy (↑ resist of filling)
    2. Pulmonary stenosis.
    3. Pulmonary hypertension.
    4. Tricuspid stenosis.
  - Absence: Atrial fibrillation, TR.
  - Cannon wave: Complete AV block, atrial flutter, ventricular extrasystole.
- 'c' wave: Prominent in TR; absent in constrictive pericarditis.
- 'v' wave: Prominent in constrictive pericarditis.

ABNORMALITIES OF JVP

1) Raised JVP with normal waveform
   - right heart failure
   - fluid overload
   - bradycardia
2) Raised JVP with absent pulsation
   - SVC obstruction - full dilated jugular veins, no pulsation, oedematous face and neck
3) Large a wave
   - tricuspid stenosis - atria contracts against stiff tricuspid and so pressure in atria rises higher than normal
   - pulmonary hypertension - there are generally higher pressures on the right side of the heart
   - pulmonary stenosis
4) Extra-large a wave = Cannon wave

Occurs when atrium contracts against closed tricuspid eg

   - complete heart block
   - atrial flutter
   - single chamber pacing
   - nodal rhythm (AV node is in charge)
   - ventricular extra-systole
   - ventricular tachycardia
5) Absent a wave
   - atrial fibrillation
6) Systolic waves = combined c-v waves = big v waves
   - tricuspid regurgitation
7) The slow y descent occurs in tricuspid stenosis
8) Paradoxical JVP = Kussmaul's sign

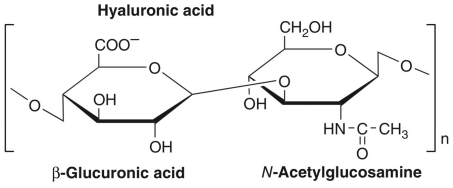
Biochemistry

1. Corneal transparency is decided by
A.CHONDROITIN SULPHATE
B.HYALURONIC ACID
C.KERATIN SULPHATE
D.HEPARIN SULPHATE

MUCOPOLYSACCHARIDES

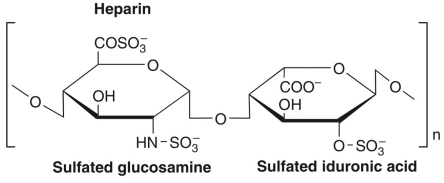
- a.k.a Glycosaminoglycans (GAG).
- are Heteropolysaccharides containing uronic acid and amino sugars (Acetylayed amino groups ,sulfate and carboxyl groups are also present.)
- Presence of charged groups attract water molecules and produce viscous solution.
- Excreted in abnormal amounts in Lysosomal Storage disorders.
- Detected by 2D Gel electrophoresis techniques, CPC test,Cetavlon test and Alicante Blue staining.

HYALURONIC ACID



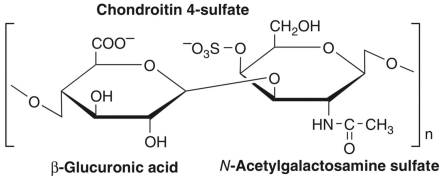
Present in connective tissues,tendons, synovial fluid,vitreous humor.
Lubricant in joint cavities.

HEPARIN



Repeated units of Sulfated glucosamine-alpha-1,4-Iduronic acid.
Anticoagulant - activates Anti-Thrombin III
Present in Liver,Lungs,Spleen and Monocytes.

CHONDROITIN SULFATE



Repeated units of D-Glucuronic acid-beta-1,3 N - Acetyl galactosamine-4 sulfate.
Present in cartilage,bone,tendons,cornea and skin.

KERATIN SULFATE

Only GAG which doesnot contain any uronic acid.
Repeating units - Galctose and N-Acetyl glucosamine in Beta linkage.
Present in Cornea and Tendons

DERMATAN SULFATE

Contains L- Iduronic acid and N-Acetyl Galactosamine in Beta-1,3 linkages.
Present in Skin,Blood vessels and Heart valves

2. A patient complains of knee pain. Routine investigations are unremarkable and still, the patient is unsatisfied. Urine turns black on standing, what is the enzyme involved?

A. Homogentisate oxidase
B. Xanthine oxidase
C. Tyrosine transaminase
D. 4- fumarylacetoacetase

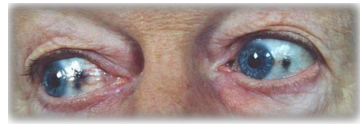
The diagnosis is ALKAPTONURIA caused by deficiency of Homogentisate oxidase

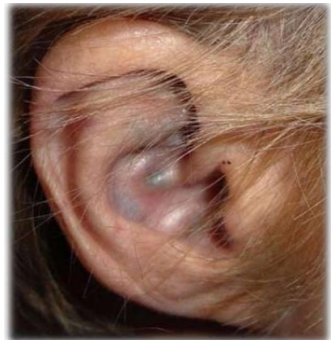
ALKAPTONURIA

Autosomal Recessive.
Caused by Homogentisic acid oxidase deficiency.

CLINICAL MANIFESTATION

- Blackening of urine on standing
- Ochronosis - accumulation of black polymer of homogentisic acid.
- Arthritis - Acute exacerbations present. Radiologic features similar to Osteoarthritis.





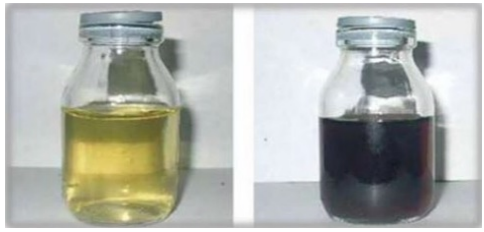
Dark spots on sclera and cartilage

DIAGNOSIS

- Massive excretion of homogentisic acid on urine testing.
- Tyrosine levels - Normal.
- Benedict's test - Strongly Positive.
- Ferric Chloride test positive.

TREATMENT

- Symptomatic
- Nitisinone combined with Phenylalanine and Tyrosine
- restricted diet.

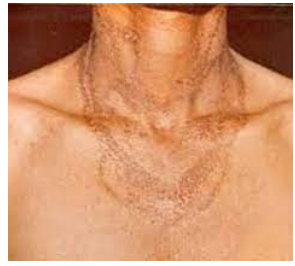


Blackening of Urine

Reference : Textbook of Biochemistry for Medical Students by DM Vasudevan 7th Ed. Pg 237
Nelson Testbook of Pediatrics 20th Ed. Pg 642
Reference: Notespaedia Biochemistry Image bank pg 15

3. A middle aged farmer grew mainly maize as staple food. He presents to the opd as shown in the image. Deficiency of which vitamin is shown?

A. Niacin
B. Zinc
C. Thiamine
D. Pyridoxine



VITAMIN B 3

a.k.a Niacin, Nicotinic acid , Pellagra preventing Factor.
Niacinamide - active form
Co-enzyme forms :
- i. NAD +
- ii. NADPH +

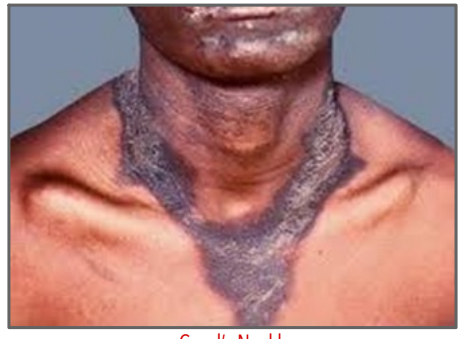
60mg of Tryptophan is equivalent to 1mg of Niacin

Quinolinate phosphoribosyl transferase is Rate limiting enzyme in conversion of Niacin to NAD +

CAUSES OF NIACIN DEFICIENCY

i. Dietary deficiency of Tryptophan
   - Maize as staple diet
   - Sorghum/Johar/Guinea corn as staple diet

ii. Deficient synthesis
iii. Isoniazid
iv. Hartnup disease
v. Carcinoid sydnrome



Casal's Necklace

PELLAGRA

Caused by deficiency of Tryptophan and Niacin.
Symptoms :
- i. Dermatitis - Casal's Necklace
- ii. Diarrhoea
- iii. Dementia- Delirium common in A/C Pellagra

ADVERSE EFFECTS

i. Flushing - transient vasodilation due to histamine release.
ii. Liver damage - modest elevations in serum transaminase and unconjugated bilirubin is seen.
iii. Diabetes - can cause hyperglycemia by insulin resistance in Diabetic patient.
iv. Niacin Maculopathy - Thickening of Macula and Retina leading to blurred vision and blindness.

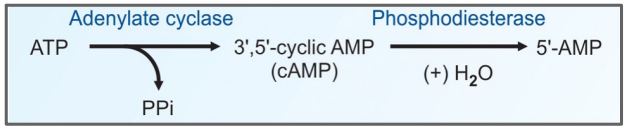
4. A 52-year-old male with Asthma presents to the emergency room in respiratory distress. The attending physician uses epinephrine to produce bronchodilation. Because epinephrine activates B-adrenergic receptors, it will relieve the symptoms. It acts through which enzyme?

A. Adenylate cyclase
B. Guanylyl cyclase
C. IP3/ DAG
D. Tyrosine kinase

C-AMP

- c-AMP is formed from ATP by the action of adenylate cyclase.
- c-AMP acts as a second messenger for many hormones, e.g. epinephrine, glucagon, etc.

c-AMP affects a wide range of cellular processes by acting as a second messenger.



Synthesis, degradation of cyclic AMP

- Cyclic AMP is a second messenger produced in the cell in response to activation of adenylate cyclase by active G-protein.
- During hormonal stimulation, cyclic AMP level in the cell increases several times.
- The level of cyclic AMP in the cell is regulated by its rate of production by adenylate cyclase (AC) and hydrolyzis by phosphodiesterase (PDE).
- The action of PDE is also regulated by hormones and drugs.
- Therefore, cellular level of cyclic AMP can be increased by inhibition of PDE.
- For example insulin activates PDE, decreasing the cellular level of cAMP while caffeine and theophylline inhibit PDEs increasing cAMP levels.

Hormones stimulate adenyl cyclase:
ACTH, ADH, Calcitonin, CRH, FSH, Glucagon, epinephrine, hCG, LH, LPH, MSH, PTH and TSH.
Hormones inhibit adenyl cyclase:
Acetylcholine, angiotensin II and somatostatin.

5. What type of defect is seen in HNPCC?

A. Base-excision repair
B. Nucleotide-excision repair
C. Mismatch repair
D. Non-homologus end joining repair

DNA REPAIR MECHANISMS

Mechanism	*Defect*	*Repair*
Mismatch repair	Copying error 1–5 bases unpaired	Strand cutting, exonuclease digestion
Nucleotide exicision repair (NER)	Chemical damage to a segment	30 bases removed; then correct bases added
Base excision repair	Chemical damage to single base	Base removed by N-glycosylase; new base added
Double strand break	Free radicals and radiation	Unwinding, alignment, ligation

DISEASES ASSOCIATED WITH DNA REPAIR MECHANISMS

DEFECTIVE NONHOMOLOGOUS END-JOINING REPAIR (NHEJ)

1. SEVERE COMBINED IMMUNODEFICIENCY DISEASE (SCID)
2. RADIATION-SENSITIVE SEVERE COMBINED IMMUNODEFICIENCY DISEASE (RS-SCID)

DEFECTIVE HOMOLOGOUS REPAIR (HR)

1. AT-LIKE DISORDER (ATLD)
2. NIJMEGEN BREAKAGE SYNDROME (NBS)
3. BLOOM SYNDROME (BS)
4. WERNER SYNDROME (WS)
5. ROTHMUND-THOMSON SYNDROME (RTS)
6. BREAST CANCER SUSCEPTIBILITY 1 AND 2 (BRCA1, BRCA2)

DEFECTIVE DNA NUCLEOTIDE EXCISION REPAIR (NER)

1. XERODERMA PIGMENTOSUM (XP)
2. COCKAYNE SYNDROME (CS)
3. TRICHOTHIODYSTROPHY (TTD)

DEFECTIVE DNA BASE EXCISION REPAIR (BER)

1. MUTYH-ASSOCIATED POLYPOSIS (MAP)

DEFECTIVE DNA MISMATCH REPAIR (MMR)

1. HEREDITARY NONPOLYPOSIS COLORECTAL CANCER (HNPCC)

Reference : Harper's Illustrated Biochemistry 31st Ed. Pg 370
Reference: Notespaedia Biochemistry Image bank pg 90

DISEASES ASSOCIATED WITH DNA REPAIR MECHANISMS

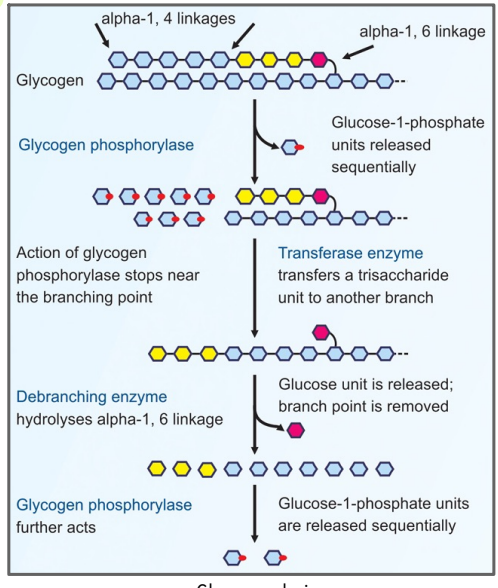
- **Xeroderma Pigmentosum** (XP): Defective NER mechanism; sensitivity to UV light; skin cancers
- **Ataxia Telangiectasia** (AT): Defective ATM gene; sensitivity to UV light; lymphoreticular neoplasms
- **Fanconi's Anemia** (1927): Defective genes are in chromosomes 20q and 9q. Defect in DNA cross link repair; increased occurrence of cancer
- **Bloom's Syndrome** (1954): Gene is in 15q. Defect in DNA ligase or helicase; lymphoreticular malignancies
- **Cockayne Syndrome** (1946): Defect in NER mechanism; transcrition factor coupled repair, transcription factor II H is defective; stunted growth and mental retardation.
- **Hereditary Polyposis Colon Cancer** (Lynch syndrome): Defective gene in chromosome 2. Defect in hMSH1 and 2 genes; mismatch repair is defective.

6. A person has a meal at 8 pm at night and records blood glucose at 7 am on the next day which showed a value of 180mg/dL. What's the source of this glucose ?

A. Dietary Glucose
B. Hepatic Gluconeogenesis
C. Hepatic Glycogenolysis
D. Muscle glycogenolysis

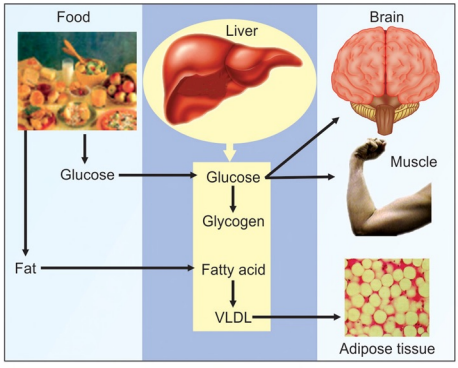
HEPATIC GLYCOGENOLYSIS

- Glycogen is the storage form of carbohydrates in the human body.
- The major sites of storage are liver and muscle.
- The major function of liver glycogen is to provide glucose during fasting.
- When blood glucose level lowers, liver glycogen is broken down and helps to maintain blood glucose level.
- After taking food, blood sugar tends to rise, which causes glycogen deposition in liver.
- About 5 hours after taking food, the blood sugar tends to fall. But, glycogen is lyzed to glucose so that the energy needs are met.
- After about 18 hours fasting, most of the liver glycogen is depleted, when depot fats are hydrolyzed and energy requirement is met by fatty acid oxidation



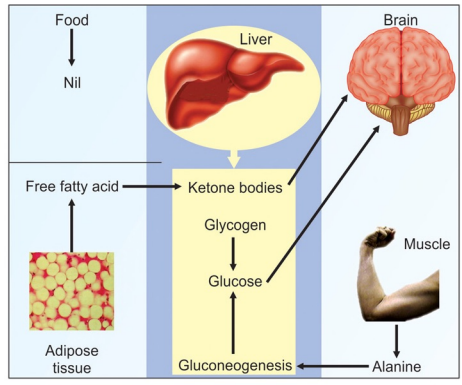
Glycogenolysis

LIVER METABOLISM IN FED STATE:



Under well-fed conditions, the liver takes up glucose from circulation and stores it as glycogen. Similarly the fatty acids synthesised by the liver are incorporated into VLDL and secreted into bloodstream. Liver is the major site of degradation of amino acids and detoxification of ammonia into urea

DURING STARVATION:



Liver provides glucose by glycogenolysis and later by gluconeogenesis so that the obligatory requirements of the brain are met. Moreover, liver also produces the ketone bodies, an alternate source of fuel. But the liver cannot use ketone bodies as its own fuel.

7. A patient presented with tendon xanthoma. Serum cholesterol and LDL were elevated and was treated with statins. The probable disorder is?

A. Familial Chylomicronemia syndrome
B. Familial hypercholesterolemia
C. Familial dysbetalipoproteinemia
D. Familial combined hyperlipidemia

The disorder is familial hypercholesterolemia (Type II A)

Table 13.2 Fredrickson (WHO) classification of hyper lipoproteinemia

Type	*Elevated plasma lipoprotein*	*Elevated plasma lipid*	*Metabolic defect*	*Risk*
Type I	Chylomicron	Triacylglycerol	Deficiency of lipoprotein lipase or apo C-II	Pancreatitis
Type IIa	LDL	Cholesterol	Deficiency of functional LDL-receptors	Coronary heart disease
Type IIb	LDL and VLDL	Triacylglycerol Cholesterol	Overproduction of apo B	Coronary heart disease
Type III	Chylomicron remnants, VLDL remnants	Triacylglycerol Cholesterol	Abnormal apo E	Vascular disease
Type IV	VLDL	Triacylglycerol Cholesterol	Overproduction of triacylglycerol	Coronary heart disease
Type V	VLDL, Chylomicron	Triacylglycerol	Seondary to other disease	Coronary heart disease

Reference: ESSENTIALS OF BIOCHEMISTRY BIOCHEMISTRY, Pankaja Naik pg 212

1. Type I (Hyperchylomicronemia):

- This is due to either deficiency of the enzyme, lipoprotein lipase or lack of apo C which is required as an activator of lipoprotein lipase.
- The enzyme defect leads to impaired hydrolysis of chylomicrons resulting in increased level of plasma chylomicron and triacylglycerol.

2. Type IIa (Familial hypercholesterolemia):

- Due to deficiency of functional LDL receptors in liver and extrahepatic tissues, cellular uptake of LDL is impaired resulting in high LDL and hypercholesterolemia.
- The LDL receptor defect may be due to the following reasons.
    1. LDL receptor deficiency.
    2. Defective binding of B-100 to the receptor. A substitution of glutamine for arginine at 3500th amino acid results in poor binding to LDL receptors. This defect is known as B-3500 or familial defective apo B.
    3. Receptor-LDL complex is not internalized.

3. Type IIb (Hypercholesterolemia):

- There is elevation of both LDL and VLDL resulting in elevated level of cholesterol and triacylglycerol.
- This is believed to be due to excessive production of apo B.
- Secondary type II hyperlipoproteinemia is seen in obesity, diabetes mellitus, hypothyroidism and nephrotic syndrome.

4. Type III (Dysbetalipoproteinemia):

- There is accumulation of chylomicron remnants and VLDL remnants in plasma resulting in hypercholesterolemia and hypertriglyceridemia.
- This is believed to be due to a genetic defect in apo E, which cannot bind to the hepatic apo E receptors.

5. Type IV (Familial endogenous hypertriglyceridemia):

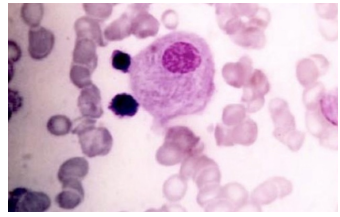
- This is due to overproduction of triacylglycerol by liver with a concomitant elevation in plasma VLDL.
- It may be associated with diabetes mellitus, obesity, alcoholism and impaired glucose tolerance.

6. TypeV:

- There is an increase in both chylomicrons and VLDL, so triacylglycerol levels are increased.
- This is usually secondary to other disorders like obesity, diabetes, alcoholism and renal disease.

8. A five-year-old male patient presented with Anemia, bone pain, hepatosplenomegaly. Bone marrow aspirate smear is as shown. What is the lipid accumulated in this disease.

A. Glucocereberoside
B. Hexosamindase A
C. u-Galactosidase
D. B-Galactosidase



The smear shows Gaucher's cells. Glucocerebroside is the lipid accumulating in Gaucher's disease.

SPHINGOLIPIDOSES

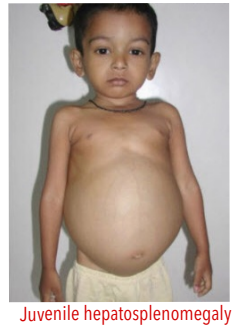
No	Disease	Enzyme defect	Lipid accumulating	Salient features
1.	Gaucher's disease	β-glucosidase	Glucocerebroside	3 types—adult, infantile, juvenile hepatosplenomegaly, erosion of bone, moderate anemia
2.	Niemann-Pick disease	Sphingomyelinase	Sphingomyelin	Severe CNS damage, mental retardation, hepatosplenomegaly Cherry red spot in macula. Death occurs by 2 years of age
3.	Krabbe's leukodystrophy	β-galactosidase	Galactocerebroside	Severe mental retardation. Total absence of myelin in CNS Globoid bodies in white matter
4.	Metachromatic leukodystrophy	Sulfatidesulfatase	Sulfogalactocerebroside	Accumulates in most tissues. Neurological deficit, difficulty in speech and optic atrophy. Demyelination is also seen
5.	Fabry's disease	α-galactosidase	Ceramide trihexoside	Kidney is the site of accumulation. Progressive renal failure Death by 5 years of age. Purplish papules appear. 'X' linked inheritence
6.	Tay Sach's disease	Hexosaminidase A	Ganglioside (GM2)	Incidence 1 in 6000 births. Mental retardation. Cherry red spot in the macula. Progressive deterioration. Death by 3–4 years
7.	Generalized gangliosidoses	β-galactosidase	Ganglioside (GM1)	Mental retardation, hepatomegaly, skeletal deformities. Foam cells in bone marrow. Cherry red spot in the retina
8.	Lactosyl ceramidoses	β-galactosidase	Lactosyl ceramide	Mainly CNS and reticuloendothelial system affected
9.	Sandhoff's disease	Hexosaminidase A and B	Globoside	Neurological deficit, mental retardation

GAUCHER'S DISEASE:

- The inherited deficiency β-glucosidase impairs the hydrolysis of glucocerebrosides, which results in accumulation of glucocerebrosides in brain, liver, spleen, and bone marrow.
- This disorder is associated with mental retardation and enlargement of liver and spleen.
- Enzyme defect : Beta - glucosidase
- Lipid accumulating - Glucocerebroside

CLINICAL FEATURES

- Bruising from thrombocytopenia
  Chronic fatigue secondary to anemia
- hepatomegaly with or without elevated liver function test results
- Splenomegaly
- Bone pain



Juvenile hepatosplenomegaly

Reference: Textbook of Biochemistry DM Vasudevan, pg 193

Reference: Notespaedia Biochemistry Image bank pg 81

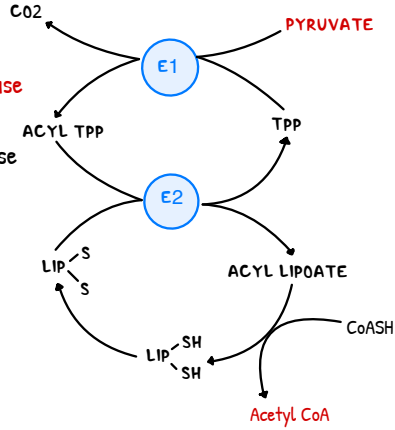
9. What is E1 and E2 ?

a. Pyruvate carboxylase, Dihydrolipoyl Acetyl transferase

b. Pyruvate dehydrogenase, Dihydrolipoyl Acetyl transferase

c. Pyruvate decarboxylase, Dihydrolipoyl Acetylase

d. Pyruvate decarboxylase, Dihydrolipoyl Acetyl transferase



The image shows Pyruvate Dehydrogenase Complex

The enzymes are:

E1 : Pyruvate dehydrogenase

E2: Dihydrolipoyl Acetyl transferase

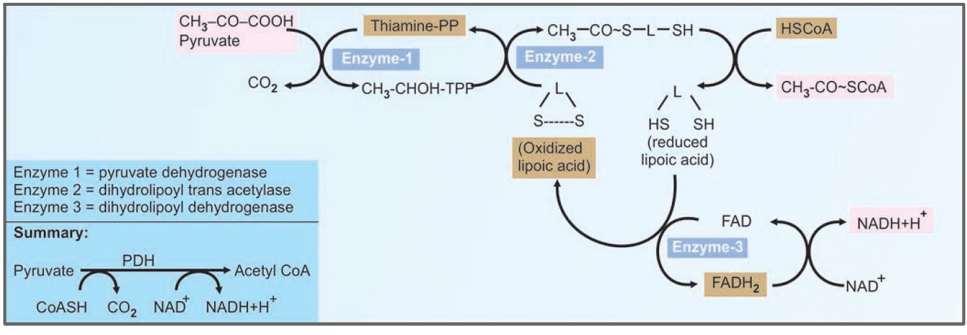
PYRUVATE DEHYDROGENASE COMPLEX

- Inside the mitochondria, pyruvate is oxidatively decarboxylated to acetyl CoA by pyruvate dehydrogenase (PDH).
- It is a multi-enzyme complex with 5 co-enzymes and 3 apo-enzymes.
- The co-enzymes needed are:
  1. Thiamine pyrophosphate (TPP)
  2. Co-enzyme A (CoA)
  3. FAD
  4. NAD+
  5. Lipoamide.
- The enzyme part of the PDH complex is made up of three component enzymes

A. Pyruvate dehydrogenase (Enzyme 1): Catalyzes oxidative decarboxylation. TPP is required for this step. So, Thiamine, a B complex group vitamin is essential for utilization of pyruvate. An enzyme bound hydroxyl ethyl TPP is formed.

B. Dihydrolipoyl transacetylase (Enzyme 2): Then, hydroxyethyl group is oxidized to form an acetyl group and then transferred from TPP to lipoamide to form acetyl lipoamide.

C. Dihydrolipoyl dehydrogenase (Enzyme 3): The last step is the oxidation of lipoamide. At the end of the reaction the cofactors, namely TPP, lipoamide and FAD are regenerated



10. In an experiment conducted by a student on ETC , when pyruvate and one inhibitor is added, it resulted in inhibition of oxidative phosphorylation. Which inhibitor most likely used by the student.

A. Oligomycin

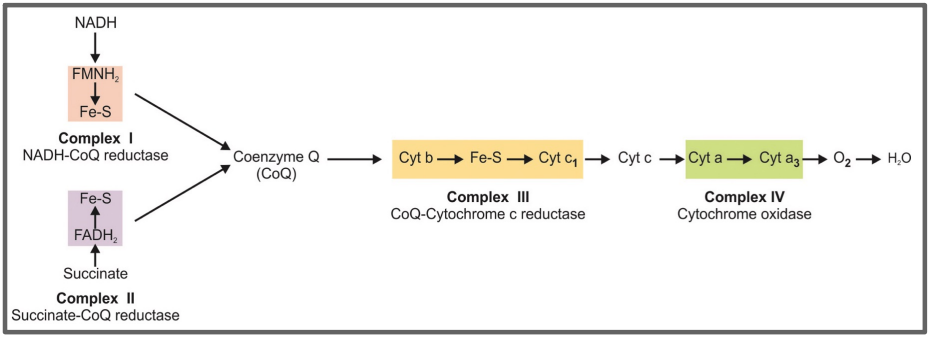
B. 2,4-DNP

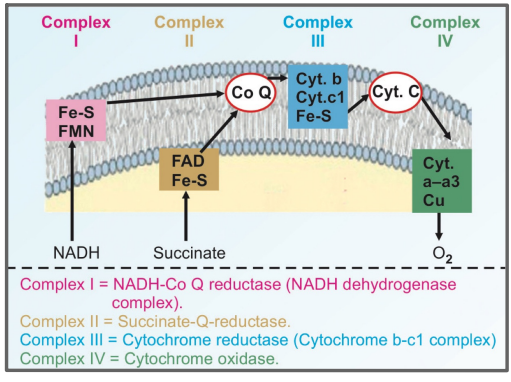
C. Antimycin

D. Rotinonc

ELECTRON TRANSPORT CHAIN (ETC)

- The electron transport chain (ETC) oxidizes NADH and FADH2 by transferring electrons by a series of oxidation reduction reactions to 02, the terminal electron acceptor.
- In the presence of 02, the ETC converts reducing equivalents into energy, (ATP) by oxidative phosphorylation.
- Present in the inner mitochondrial membrane
- The electrons flow from the more electronegative components to the more electropositive components.
- There are four distinct multi-protein complexes; Complex-I, II, III and IV. These are connected by two mobile carriers, co-enzyme Q and cytochrome c.

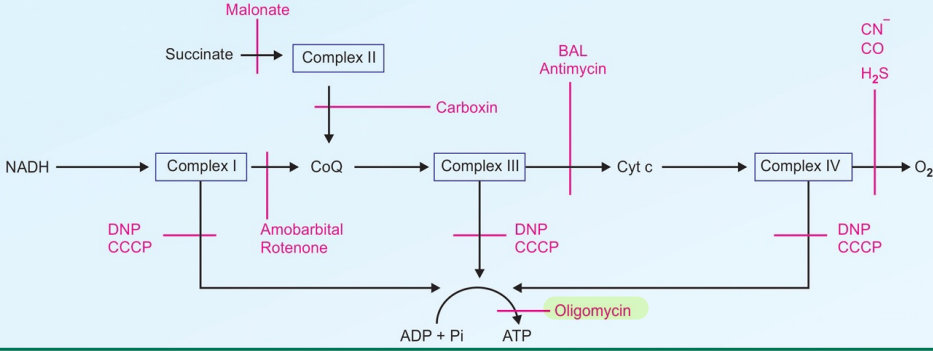




COMPOUNDS WHICH AFFECT ELECTRON TRANSPORT CHAIN AND OXIDATIVE PHOSPHORYLATION

1. Complex I to Co-Q specific inhibitors
   - i. Alkylguanides (guanethide), hypotensive drug
   - ii. Rotenone, insecticide and fish poison
   - iii. Barbiturates (amobarbital), sedative
   - iv. Chlorpromazine, tranquilizer
   - v. Piericidin, antibiotic
2. Complex II to Co-Q
   - i. Carboxin
3. Complex III to cytochrome c inhibitors
   - i. BAL (British anti-lewisite), antidote of war gas
   - ii. Naphthoquinone
   - iii. Antimycin
4. Complex IV inhibitors
   - i. Carbon monoxide, inhibits cellular respiration
   - ii. Cyanide (CN^-)
   - iii. Azide ($N3^-$)
   - iv. Hydrogen sulfide (H_2S)
5. Site between succinate dehydrogenase and Co-Q
   - i. Carboxin, inhibits transfer of ions from $FADH_2$
   - ii. Malonate, competitive inhibitor of succinate DH
6. Inhibitors of oxidative phosphorylation
   - i. Atractyloside, inhibits translocase
   - ii. Oligomycin, inhibits flow of protons through Fo
   - iii. Ionophores, e.g. Valinomycin
7. Uncouplers
   - i. 2,4-dinitrophenol (2,4-DNP)
   - ii. 2,4-dinitrocresol (2,4-DNC)
   - iii. CCCP (chlorocarbonylcyanidephenyl hydrazone)
8. Physiological uncouplers
   - i. Thyroxine, in high doses
   - ii. Thermogenin in brown adipose tissue

INHIBITORS OF ELECTRON TRANSPORT CHAIN AND OXIDATIVE PHOSPHORYLATION.



Reference: Textbook of Biochemistry DM Vasudevan, pg 266

11. In a patient with cystathionine B synthase deficiency, which aminoacid supplementation should be given?

A. Methionine

B. Serine

C. Cysteine

D. Tyrosine

HOMOCYSTINURIAS

Homocystinurias are a group of disorder of methionine metabolism.

It is characterized by high blood and urinary levels of homocysteine and methionine.

Four metabolic defects cause four types of homocystinuria.

HOMOCYSTINURIA TYPE-I

• It is due to defect in the enzyme cystathionine synthase, which converts homocysteine to cysthathionine.

As a result, homocysteine accumulates in blood and appears in urine.

• The accumulation of homocysteine causes skeletal abnormalities, ectopia lentis (dislocation of the lenses in the eyes), osteoporosis, mental retardation and thrombosis.

• Thrombosis may result in myocardial infarction, pulmonary embolism or stroke.

• A deficiency of cystathionine synthase is the most common cause of homocystinuria.

Other types of homocystinuria are due to defects in the remethylation of homocysteine to form methionine.

HOMOCYSTINURIA TYPE-II AND III

• In type-II and III, there is deficiency in synthesis of N5-methyltetrahydrofolate and methyl B12 respectively. Both N5-methyl THF and methyl B12 are required for the remethylation of homocysteine to form methionine.

HOMOCYSTINURIA TYPE-IV

It is due to defective intestinal absorption of vitamin B12.

Type	*Defect*
Homocystinuria-I	Cystathionine-β-synthase
Homocystinuria-II	Synthesis of N^5 methyl THF
Homocystinuria-III	Deficiency of methyl B_{12}
Homocystinuria-IV	Defective intestinal absorption of vitamin B_{12}

TREATMENT

• The biochemical defect in cystathionine synthase can be corrected in some cases by providing pyridoxine (vitamin B6). Pyridoxine is needed to activate the enzyme cystathionine synthase.

• Those with complete enzyme deficiency should be treated with a diet low in methionine and supplemented with cysteine.

• Vitamin B12 may be given in instances of vitamin B12 deficiency.

Pathology

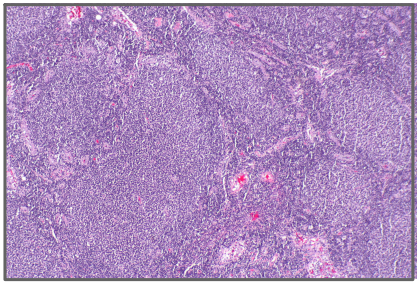
1. Patient with generalised fatigue,tiredness.Primary clinical examination is uneventful.Normal TLC and DLC.No immature cells seen.Superficial discrete lymph nodes enlarged.On biopsy showed effaced architecture,indented nucleus,prominent nucleolus containing atypical cells.CD 10 and BCL 2 Positive.

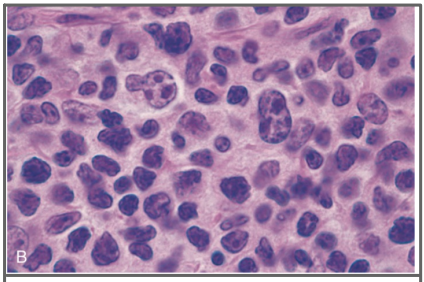
A. Follicular lymphoma
B. Burkitts lymphoma
C. Non Hodgkin's lymphoma
D. Mycoses fungoides

- Based on the given clinical features and biopsy findings is suggestive of Follicular lymphoma

FOLLICULAR LYMPHOMA

- Most common form of indolent NHL.
- Follicular lymphoma is strongly associated with chromosomal translocations involving BCL2.
- Its hallmark is a (14;18) translocation that juxtaposes the IGH locus on chromosome 14 and the BCL2 locus on chromosome 18.





At high magnification, small lymphoid cells with condensed chromatin and irregular or cleaved nuclear outlines (centrocytes) are mixed with a population of larger cells with nucleoli (centroblasts).

MORPHOLOGY

- In most cases, a nodular or nodular and diffuse growth pattern is observed in involved lymph nodes.
- Two principal cell types are present in varying proportions :
  (1) small cells with irregular or cleaved nuclear contours and scant cytoplasm, referred to as centrocytes (small cleaved cells), and
  (2) larger cells with open nuclear chromatin, several nucleoli, and modest amounts of cytoplasm, referred to as centroblasts

CLINICAL FEATURES

- Painless, generalized lymphadenopathy.
- Involvement of extranodal sites, such as the gastrointestinal tract, central nervous system, or testis, is relatively uncommon.

BURKITT LYMPHOMA

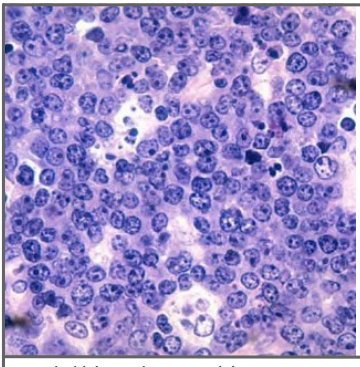
- All forms of Burkitt lymphoma are associated with translocations of the MYC gene on chromosome 8 that lead to increased MYC protein levels.

Reference :
Robbins & Cotran Pathologic Basis of Disease, 10th Ed. Pg : 599,601
Notespaedia Pathology IB Pg : 116

- MYC is a master transcriptional regulator that increases the expression of genes that are required for aerobic glycolysis, the so-called Warburg effect .
- The MYC translocation usually associated with the IGH locus [t(8;14)], but may also be the Igκ [t(2;8)] or λ [t(8;22)] light chain loci.
- Sequencing of the genomes of Burkitt lymphoma cells has revealed that most tumors have mutations that increase the activity of the transcription factor TCF3 (also known as E2A).

MORPHOLOGY

- Involved tissues are effaced by a diffuse infiltrate of intermediate- sized lymphoid cells 10 to 25 μm in diameter with round or oval nuclei, coarse chromatin, several nucleoli, and a moderate amount of cytoplasm



Burkitt lymphoma. At low power, numerous pale tingible body macrophages are evident, producing a "starry sky" appearance.

IMMUNOPHENOTYPE

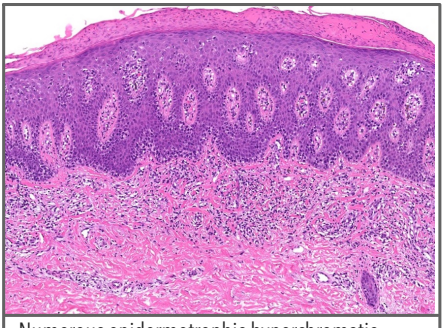
- These are tumors of mature B cells that express surface IgM, CD19, CD20, CD10, and BCL6.
- Almost always fails to express the antiapoptotic protein BCL2.

CLINICAL FEATURES

- Most tumors manifest at extranodal sites.
- Endemic :
  - presents as a mass involving the mandible
  - abdominal viscera such as kidney, ovaries and adrenal glands are involved.
- Sporadic :
  - appears as a mass involving the ileocecum and peritoneum.

MYCOSIS FUNGOIDES (CUTANEOUS T-CELL LYMPHOMA)

- May occur at any age, but most commonly afflicts persons older than age 40.
- Lesions usually involve truncal areas and include scaly, red-brown patches; raised, scaling plaques that may be confused with psoriasis; and fungating nodules.
- Seeding of the blood by malignant T cells is accompanied by diffuse erythema and scaling of the entire body surface (erythroderma), a condition known as Sézary syndrome



Numerous epidermotrophic,hyperchromatic lymphocytes and a band like dermal lymphoid infiltrate are present amid background psoriasiform acanthosis and dermal fibroplasia

Reference :
Robbins & Cotran Pathologic Basis of Disease, 10th Ed. Pg : 1152

2. A patient with substernal mass was diagnosed with red cell aplasia,not responding to blood transfusion.Decreased Hb, TLC normal.What is the most probable cause of aplasia ?

A. Bronchogenic Carcinoma
B. Non Hodgkins Lymphoma
C. Thymic neoplasia
D. Retrosternal goitre

- Substernal mass with red cell aplasia not responding to blood transfusion is suggestive of Thymic Neoplasia

THYMOMA

- A diversity of neoplasms may arise in the thymus—germ cell tumors, lymphomas, carcinoids, and others—but the designation "thymoma" is restricted to tumors of thymic epithelial cells.
- Classification three histologic subtypes :
  - Tumors that are cytologically benign and noninvasive
  - Tumors that are cytologically benign but invasive or metastatic
  - Tumors that are cytologically malignant (thymic carcinoma)
- Usually occur in adults older than 40 years of age.
- Males and females are affected equally.
- Account for 20% to 30% of tumors in the anterosuperior mediastinum
- Macroscopically, thymomas are lobulated, firm, gray-white masses of up to 15 to 20 cm in size.
- They sometimes have areas of cystic necrosis and calcification.
- Noninvasive thymomas :
  - Are most often composed of medullary-type epithelial cells or a mixture of medullary- and cortical-type epithelial cells.
- Invasive thymoma :
  - Refers to a tumor that is cytologically benign but locally invasive.
  - These tumors are much more likely to metastasize.
  - The epithelial cells are most commonly of the cortical variety, with abundant cytoplasm and rounded vesicular nuclei

CLINICAL FEATURES

- Symptoms stemming from impingement on mediastinal structures.

- Pure Red Cell Aplasia Association :
  - Acute : Parvovirus B19 infection
  - Chronic : Associated with thymoma, large granular lymphocytic leukemia, presence of neutralizing antibodies against erythropoietin, and other autoimmune phenomenon

Reference :
Robbins & Cotran Pathologic Basis of Disease, 10th Ed.

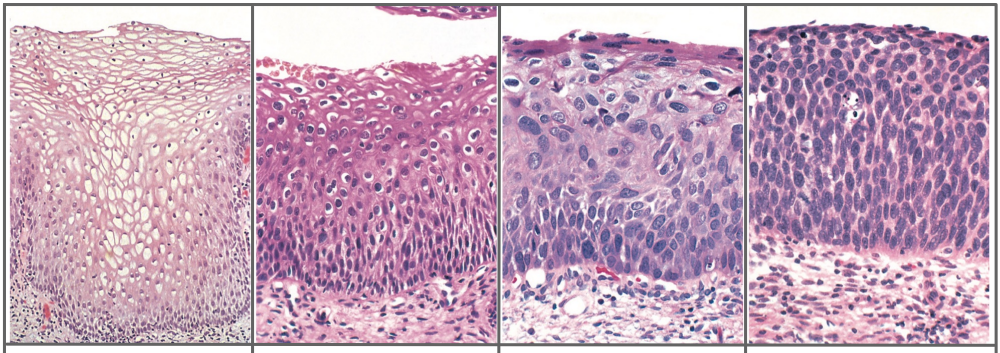
3. 21yr old female with PAP Smear with hyperchromatic nuclei,pleomorphism with low maturation index involving almost all thickness.What is this called ?

A. Metaplasia
B. Dysplasia
C. Hyperplasia
D. Carcinoma

- PAP Smear with hyperchromatic nuclei,pleomorphism with low maturation index involving almost all thickness is suggestive of CIN III.

CERVICAL INTRAEPITHELIAL NEOPLASIA (SQUAMOUS INTRAEPITHELIAL LESIONS)

- Cervical intraepithelial neoplasia (CIN) classification with :
  - Mild dysplasia termed CIN I,
  - Moderate dysplasia termed CIN II, and
  - Severe dysplasia termed CIN III.
- The three-tier classification syste has been recently simplified to a two-tiered system, with
  - CIN I renamed low-grade squamous intraepithelial lesion (LSIL) and
  - CIN II and CIN III combined into one category referred to as high-grade squamous intraepithelial lesion (HSIL)
- LSIL does not progress directly to invasive carcinoma, and, in fact, most cases regress spontaneously; only a small percentage progress to HSIL.
- By contrast to LSIL, HSIL is considered to be at high risk for progression to carcinoma.
- More than 80% of LSILs and 100% of HSILs are associated with high-risk HPVs, with HPV-16 being the most common HPV type in both lesions.



Normal	CIN I	CIN II	CN III

Spectrum of cervical intraepithelial neoplasia: normal squamous epithelium for comparison; low-grade squamous intraepithelial lesion (cervical intraepithelial neoplasia [CIN] I) with koilocytic atypia; high-grade squamous intraepithelial lesion (HSIL) (CIN II) with progressive atypia and expansion of the immature basal cells above the lower third of the epithelial thickness; HSIL (CIN III) with diffuse atypia, loss of maturation, and expansion of the immature basal cells to the epithelial surface.

Reference :
Robbins & Cotran Pathologic Basis of Disease, 10th Ed.
Notespaedia Pathology IB Pg 23.

METAPLASIA	HYPERPLASIA	DYSPLASIA
● Replacement of one type of cell with another type. ● Nearly always found in association with tissue damage, repair, and regeneration. ● Eg : - Squamous metaplasia of the bronchial epithelium in chronic smokers - Barrett esophagus : squamous epithelium of the esophagus, replaced by glandular (gastric or intestinal) epithelium	● Is an increase in the number of cells in an organ or tissue in response to a stimulus. ● Physiologic hyperplasia : Due to the action of hormones or growth factors occurs when there is a need to increase functional capacity of hormone sensitive organs, or when there is need for compensatory increase after damage or resection. ● Pathologic hyperplasia : Caused by excessive or inappropriate actions of hormones or growth factors acting on target cells.	● Means "disordered growth." ● Seen in epithelial cells. ● Cells may exhibit considerable pleomorphism and often contain large hyperchromatic nuclei with a high nuclear-to-cytoplasmic ratio. ● Typically show architectural disarray and a loss of orderly differentiation. Eg : - CIN

4. A patient presented with h/o fatigue,weight loss,infection has a history of benzene exposure for past 20 yrs.Systemic examination is normal.He is most likely to have which carcinoma ?

A. Lung
B. Bladder
C. Blood
D. Skin

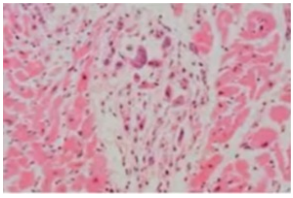
● Likely carcinoma to occur after chronic benzene exposure is AML (Acute Myeloid Leukemia)

Occupational Cancers	
Agents or Groups of Agents	Human Cancers for Which Reasonable Evidence Is Available
Arsenic and arsenic compounds	Lung carcinoma, skin carcinoma
Asbestos	Lung, esophageal, gastric, and colon carcinoma; mesothelioma
Benzene	Acute myeloid leukemia
Beryllium and beryllium compounds	Lung carcinoma
Cadmium and cadmium compounds	Prostate carcinoma
Chromium compounds	Lung carcinoma

Occupational Cancers	
Agents or Groups of Agents	Human Cancers for Which Reasonable Evidence Is Available
Nickel compounds	Lung and oropharyngeal carcinoma
Radon and its decay products	Lung carcinoma
Vinyl chloride	Hepatic angiosarcoma

6. A 34yr old woman presented with fever,migratory arthritis of lower large joints,tachycardia.Murmur is Pancystolic.On Echo Mitral regurgitation is present.The biopsy shows the following ?

A. Aschoff body
B. Granuloma anulare
C. Granulomatous vasculitis
D. Epitheloid granuloma



RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

- Rheumatic fever (RF) is an acute, immunologically mediated, multisystem inflammatory disease classically occurring a few weeks after an episode of group A streptococcal pharyngitis.

PATHOGENESIS

- Acute rheumatic fever results from host immune responses to group A streptococcal antigens that cross-react with host proteins.
- Antibody binding can activate complement, as well as recruit Fc-receptor bearing cells (neutrophils and macrophages); cytokine production by the stimulated T cells leads to macrophage activation (e.g., within Aschoff bodies).

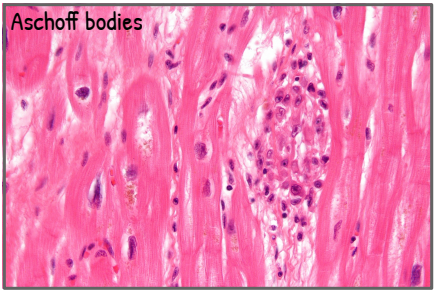
MORPHOLOGY

- During Acute RF,focal inflammatory lesions are found in various tissues.
- Distinctive lesions occur in the heart, called Aschoff bodies, consisting of foci of T lymphocytes, occasional plasma cells,and plump activated macrophages called Anitschkow cells (pathognomonic for RF).
- During Acute RF,diffuse inflammation and Aschoff bodies may be found in any of the 3 layers of the heart.
- Subendocardial lesions,perhaps exacerbated by regurgitant jets, can induce irregular thickenings called MacCallum plaques,usually in the left atrium.
- The cardinal anatomic changes of the mitral valve in Chronic RHD are leaflet thickening, commissural fusion and short ening, and thickening and fusion of the tendinous cords

Reference :
Robbins & Cotran Pathologic Basis of Disease, 10th Ed.
Notespaedia Pathology IB Pg 124.

CLINICAL FEATURES

- Constellation of findings :
  (1) Migratory polyarthritis of the large joints,
  (2) Pancarditis,
  (3) Subcutaneous nodules,
  (4) Erythema marginatum of the skin, and
  (5) Sydenham chorea, a neurologic disorder with involuntary rapid, purposeless movements.



7. A 30 year old female IT professional came to OPD with c/o of easy fatigue. She gives a history of sitting in front of computer for 12 - 14 hrs per day.She usually orders non vegetarian food from outside with less fruits and veggies.Upon blood examination,it showed Hb - 9g%,MCV - 120.What is the most likely cause of her condition ?

A. Folic Acid deficiency
B. Cyanocobalamin deficiency
C. Massive Blood loss
D. Sideroblastic Anemia

- Based on history given and blood examination showing Hb -9%,MCV - 120 mostly suggestive of Megaloblastic anemia.
- Megaloblastic anemia is due to : Cyanocobalamin deficiency > Folic acid deficiency

MEGALOBLASTIC ANEMIAS

- The common theme among the various causes of megaloblastic anemia is an impairment of DNA synthesis that leads to ineffective hematopoiesis and distinctive morphologic changes, including abnormally large erythroid precursors and red cells.

CAUSES

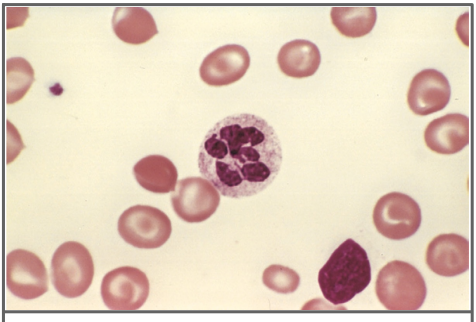
- Decreased Intake : Inadequate diet, vegetarianism
- Impaired Absorption :
  i. Intrinsic factor deficiency :
    - Pernicious anemia
    - Gastrectomy
  ii.Malabsorption states Diffuse intestinal disease (e.g., lymphoma, systemic sclerosis)
  iii.Ileal resection,ileitis
    - Competitive parasitic uptake
    - Fish tapeworm infestation
  iv.Bacterial overgrowth in blind loops and diverticula of bowel

Reference :
Robbins & Cotran Pathologic Basis of Disease, 10th Ed.

- **Folic Acid Deficiency**
  - i. Decreased Intake :
    - Inadequate diet, alcoholism, infancy
    - Impaired Absorption
    - Malabsorption states
    - Intrinsic intestinal disease
    - Anticonvulsants, oral contraceptives
    - Increased Loss
    - Hemodialysis
  - ii. Increased Requirement :
    - Pregnancy, infancy, disseminated cancer, markedly increased hematopoiesis
  - iii. Impaired Utilization : Folic acid antagonists
- **Unresponsive to Vitamin B12 or Folic Acid Therapy :**
  - Metabolic Inhibitors of DNA Synthesis and/or Folate Metabolism (e.g., Methotrexate)

MORPHOLOGY

- Peripheral blood findings :
  - Presence of red cells that are macrocytic and oval (macro-ovalocytes) is highly characteristic.
  - Because they are larger than normal and contain ample hemoglobin, most macrocytes lack the central pallor of normal red cells and even appear "hyperchromic," but the MCHC is not elevated.
  - There is marked variation in the size (anisocytosis) and shape (poikilocytosis) of red cells.
  - The reticulocyte count is low.
  - While nuclear maturation is delayed, cytoplasmic maturation and hemoglobin accumulation proceed at a normal pace, leading to nuclear-to-cytoplasmic asynchrony.



Megaloblastic anemia : A peripheral blood smear shows a hypersegmented neutrophil with a six-lobed nucleus.

SIDEROBLASTIC ANEMIA

- Sideroblastic anemia is a type of anemia that results from abnormal utilization of iron during erythropoiesis.
- Sideroblastic anemia is known to cause microcytic and macrocytic anemia depending on what type of mutation led to it.
- Ring sideroblasts are erythroid precursors containing deposits of non-heme iron in mitochondria forming a ring-like distribution around the nucleus.
- Diagnosed by the presence of ring sideroblasts in the bone marrow.
- The red blood cells that contain these iron inclusions are called siderocytes.

- On complete blood count :
  - Mean corpuscular volume is low showing microcytosis;
  - Low mean corpuscular hemoglobin and
  - Increased red blood cell distribution width.
- Treatment :
  - Oral pyridoxine 50-100mg/day has been proven to partially or completely correct anemia.
  - It is recommended to start deferoxamine or oral chelators when serum ferritin is more than 1000 ng/L.
  - Iron overload, if left untreated could result in unresponsiveness of pyridoxine.

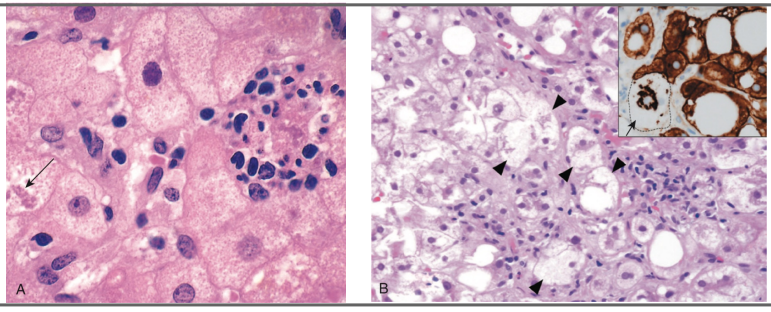
8. A patient presented with cirrhosis.On liver biopsy showed Eosinophilic material cytokeratin positive. (CK 8+,CK 18+).This is most likely composed of ?

A. Actin C. Intermediate
B. Microtubules D. Fibronectin

- Based on the biopsy findings eosinophilic material cytokeratin and CK 8+ ,CK1 8+ is suggestive of Mallory Denk Bodies

ALCOHOLIC (STEATO-) HEPATITIS

1. Hepatocyte swelling and necrosis
2. Mallory-Denk bodies :
   - These are usually present as clumped, amorphous, eosinophilic material in ballooned hepatocytes.
   - They are made up of tangled skeins of intermediate filaments such as keratins 8 and 18 in complex with other proteins such as ubiquitin.
   - These inclusions are a characteristic but not specific feature of alcoholic liver disease.
3. Neutrophilic reaction :
   - Neutrophils permeate the hepatic lobule and accumulate around degenerating hepatocytes, particularly those having Mallory-Denk bodies.
   - They may be more or less admixed with mononuclear cells



A, Alcoholic hepatitis with clustered inflammatory cells marking the site of a necrotic hepatocyte. A Mallory Denk body is present in another hepatocyte (arrow).
B, Alcoholic steatohepatitis with many ballooned hepatocytes (arrowheads). Clusters of inflammatory cells are also present; inset shows immunostaining for keratins 8 and 18 (brown), with most hepatocytes, including those with fat vacuoles, showing normal cytoplasmic staining.

9.Mismatch repair defect is seen in :

A. Xeroderma Pigmentosa

B. HNPCC

C. Familial adenomatous polyposis

D. Ataxia Telengectasia

HEREDITARY NONPOLYPOSIS COLON CANCER SYNDROME

- Is an autosomal dominant disorder characterized by familial carcinomas of the colon affecting predominantly the cecum and proximal colon.
- Results from defects in a family of genes encoding a group of proteins that work together to carry out DNA mismatch repair.
- One of the hallmarks of patients with mismatch-repair defects is microsatellite instability.
- Germline mutations in the MSH2 and MLH1 genes each account for approximately 30% of cases.

DNA REPAIR DEFECT	CONDITION
Nucleotide excision repair	Xeroderma Pigmentosum Cockayne Syndrome (CS); UV-Sensitive Syndrome Cerebro-Oculo-Facio-Skeletal syndrome (COFS)
Non-homologous end joining (NHEJ)	Severe Combined Immunodeficiency Disease (SCID) Radiation Sensitive Severe Combined Immunodeficiency Disease(RS-SCID)
DNA Base Excision Repair (BER)	MUTYH-Associated Polyposis
Homologous Repair	AT- Like disorder Bloom Syndrome Werner syndrome Rothmund-Thomson Syndrome (RTS) Breast Cancer susceptibility (BRCA 1 and 2)

10. 20yr old boy presents with gum bleeding and easy bruisibility.Fever for one month Hb 3,TLC 15,000,Platelet count 15000.On examination Pallor present,Petechial rash present all over the body.Peripheral smear shows :macrocytes,hypocellular bone marrow,absent megakaryocyte,no immature cells.What is most probable diagnosis ?

A. Disseminated TB involving bone ematirx

B. Idiopathic Acquired Aplastic anemia

C. Paroxysmal Nocturnal Hemoglobinuria

D. Myelodysplastic syndrome

- Based on the given history and examination findings the most probable diagnosis is Idiopathic Acquired Aplastic anemia

Reference :
Robbins & Cotran Pathologic Basis of Disease, 10th Ed.
Notespaedia Pathology IB Pg 74,92

APLASTIC ANEMIA

- Aplastic anemia refers to a syndrome of chronic primary hematopoietic failure and attendant pancytopenia (anemia, neutropenia, and thrombocytopenia).

ETIOLOGY

- **Acquired :**
  - Idiopathic
  - Acquired stem cell defects
  - Immune mediated
- **Physical Agents :**
  - Whole-body irradiation
  - Viral Infections Hepatitis (unknown virus)
  - Cytomegalovirus infections
  - Epstein-Barr virus infections
  - Herpes zoster (varicella zoster)
- **Chemical Agents :**
  - i. Dose related
    - Alkylating agents
    - Antimetabolites
    - Benzene
    - Chloramphenicol
    - Inorganic arsenicals
  - ii. Idiosyncratic
    - Chloramphenicol
    - Phenylbutazone
    - Organic arsenicals
    - Methylphenylethylhydantoin
    - Carbamazepine
    - Penicillamine
    - Gold salts
- **Inherited :**
  - Fanconi anemia
  - Telomerase defects

PATHOGENESIS

- Two major etiologies have been invoked :
  - i. An extrinsic, immune-mediated suppression of marrow progenitors, and
  - ii. An intrinsic abnormality of stem cells

CLINICAL FEATURES

- Progressive weakness, pallor, and dyspnea;
- Petechiae and ecchymoses;
- Frequent and persistent minor infections or the sudden onset of chills, fever, and prostration.
- Splenomegaly is characteristically absent; if it is present, the diagnosis of aplastic anemia should be seriously questioned.

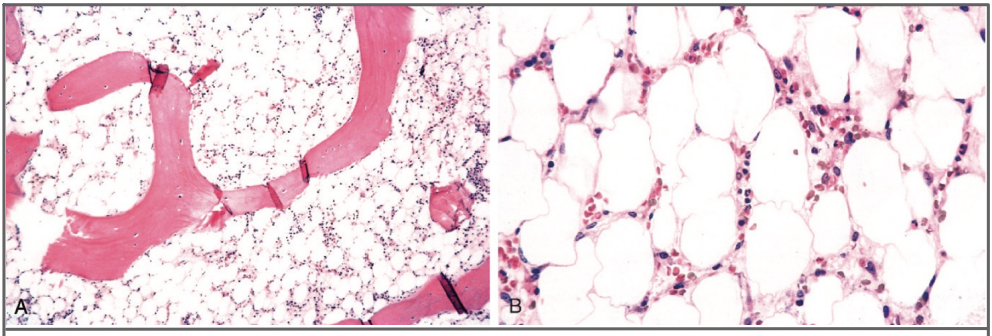
DIAGNOSIS

- Anemia : Hb decreased
- Platelet count : decreased
- Bone marrow biopsy
- Thrombocytopenia
- Neutropenia

TREATMENT

- Bone marrow transplantation is the treatment of choice in those with a suitable donor and provides a 5-year survival of more than 75%.
- Older patients or those without suitable donors often respond well to immunosuppressive therapy.

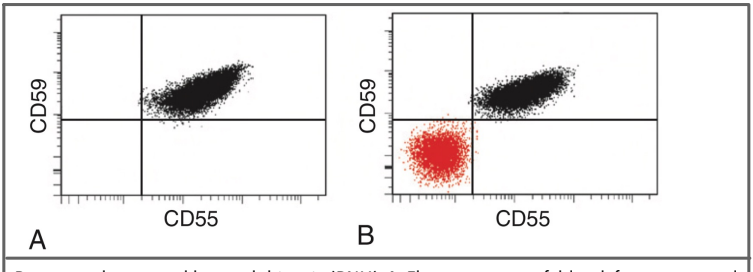
Reference :
Robbins and Cotran Pathologic Basis of Disease 9th Ed. Pg : 653
Notespaedia Pathology IB Pg : 93



Aplastic anemia (bone marrow biopsy).Markedly hypocellular marrow contains mainly fat cells. A,Low power. B, High power.

PAROXYSMAL NOCTURNAL HEMOGLOBINURIA

- Disease that results from acquired mutations in the phosphatidylinositol glycan complementation group A gene (PIGA), an enzyme that is essential for the synthesis of certain membrane-associated complement regulatory proteins.
- PNH blood cells are deficient in three GPI-linked proteins that regulate complement activity :
  (1) decay- accelerating factor, or CD55;
  (2) membrane inhibitor of reactive lysis, or CD59 (Most important); and
  (3) C8 binding protein
- Red cells deficient in these GPI-linked factors are abnormally susceptible to lysis or injury by complement
- Manifests as intravascular hemolysis (caused by the C5b-C9 membrane attack complex).
- The anemia is variable but usually mild to moderate in severity.
- The loss of heme iron in the urine (hemosiderinuria)
  Diagnosed by flow cytometry, which provides a sensitive means for detecting red cells that are deficient in GPI-linked proteins such as CD59



Paroxysmal nocturnal hemoglobinuria (PNH). A, Flow cytogram of blood from a normal individual shows that the red cells express two phosphatidylinositol glycan (PIG)-linked membrane proteins, CD55 and CD59, on their surfaces. B, Flow cytogram of blood from a patient with PNH shows a population of red cells that is deficient in both CD55 and CD59. As is typical of PNH, a second population of CD55+/CD59+ red cells that is derived from residual normal hematopoietic stem cells is also present.

Reference :
Robbins and Cotran Pathologic Basis of Disease 9th Ed.

MYELODYSPLASTIC SYNDROMES

- Refers to a group of clonal stem cell disorders characterized by maturation defects that are associated with ineffective hematopoiesis and a high risk of transformation to AML.
- Bone marrow is cellular
  - Partly or wholly replaced by the clonal progeny of a neoplastic multipotent stem cell that retains the capacity to differentiate but does so in an ineffective and disordered fashion.
  - Patients have peripheral blood cytopenias.

CLINICAL FEATURES

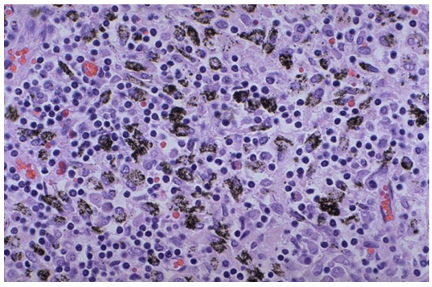
- It presents with weakness, infections, and hemorrhages, all due to pancytopenia.
- Patients often succumb to the complications of thrombocytopenia (bleeding) and neutropenia (infection).

12. Patient with Squamous cell carcinoma of lung. During pulmonectomy, there was blackish hilar lymph node of 1 cm.Identify the cause of pigmentation:

A. Asbestos
B. Anthracotic pigment.
C. Lipofuscin
D. Melanin

SMALL CELL CARCINOMA

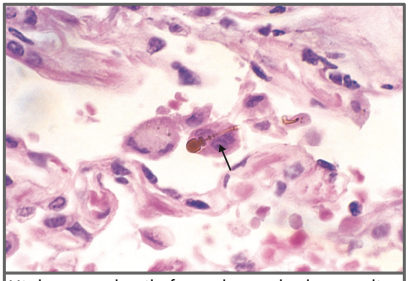
- Is a highly malignant tumor with a strong relationship to cigarette smoking; only about 1% occurs in nonsmokers.
- May arise in major bronchi or in the periphery of the lung.
- No known pre-invasive phase.
- Most aggressive of lung tumors, metastasizing widely and fatal.
- Comprised of relatively small cells with scant cytoplasm, ill-defined cell borders, finely granular nuclear chromatin (salt and pepper pattern), and absent or inconspicuous nucleoli.
- The cells are round, oval, or spindle- shaped, and nuclear molding is prominent.
- There is no absolute size for the tumor cells, but in general they are smaller than three times the diameter of a small resting lymphocyte (a size of about 25 μm).
- The mitotic count is high.
- Necrosis is common and often extensive.
- Basophilic staining of vascular walls due to encrustation by DNA from necrotic tumor cells (Azzopardi effect) is frequently present.
- On Electron microscopy :
  - Shows dense-core neurosecretory granules
  - Expression of neuroendocrine markers such as chromogranin, synaptophysin, and CD56; and the ability of some of these tumors to secrete hormones



- Here is anthracotic pigment in macrophages in a hilar lymph node.
- Anthracosis is nothing more than accumulation of carbon pigment from breathing dirty air.
- Smokers have the most pronounced anthracosis.
- The anthracotic pigment looks bad, but it causes no major organ dysfunction.

ASBESTOSIS

- Marked by diffuse pulmonary interstitial fibrosis, which is distinguished from diffuse interstitial fibrosis resulting from other causes only by the presence of asbestos bodies.
- Asbestos bodies :
  - Are golden brown, fusiform or beaded rods with a translucent center that consist of asbestos fibers coated with an iron-containing proteinaceous material
- Other inorganic particulates may become coated with similar iron-protein complexes and are called ferruginous bodies.
- Pleural plaques :
  - Most common manifestation of asbestos exposure, are well-circumscribed plaques of dense collagen that are often calcified



High-power detail of an asbestos body, revealing the typical beading and knobbed ends (arrow).

13. A patient is diagnosed of multiple sclerosis. The disease mainly affects?

A. Astrocytes

B. Oligodendrocytes

C. Glial cells

D. ependymal cells

MULTIPLE SCLEROSIS

• An autoimmune inflammatory disease characterized by demyelination and axonal injury

• Acute plaques (representing perivascular inflammation) progress to chronic plaques (representing demyelination)

- Multiple or disseminated sclerosis is the most common of the CNS demyelinating diseases.
- The usual age at onset is 20 to 40 years. The disease presents as recurrent attacks of focal neurologic disorder with predilection for involvement of the spinal cord, optic nerve and brain.

- Clinical Features
  - The first attack usually begins with a single sign or symptom, most commonly optic neuritis, followed by recovery. As the disease becomes more progressive, remissions become infrequent and incomplete.

MORPHOLOGIC FEATURES

The pathologic hallmark is the presence of many scattered discrete areas of demyelination termed plaques.

- **GROSSLY**, plaques appear as grey-pink, swollen, sharply defined, usually bilaterally symmetric areas in the white matter.
- **MICROSCOPICALLY**, the features vary according to the age of the plaque:
- In active enlarging plaques,

  the histologic features are accumulation of lymphocytes and macrophages around venules and at the plaque margin where demye lination is occurring. In addition,

  there is loss of oligodendrocytes and presence of reactive astro cytosis with numerous lipid-laden macrophages (micro glia) in the plaque.

  The axons in the plaque are generally intact.
- In old inactive plaques,

  there is no perivascular inflammatory cell infiltrate and nearly total absence of oligodendrocytes.

  Demyelination in the plaque area is complete as there is only limited regeneration of myelin.

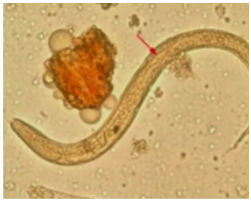
  Gliosis is well-developed but astrocytes are less prominent.

  Some axonal loss may be present.

Reference: Harsh Mohan textbook of Pathology, pg 876

1. A patient is on long term steroids for chronic urticaria/recurrent eczema is now presenting to the clinic with itching and nocturnal cough.Bronchospasm Alevolar Lavage was done which showed multiple small larvae.What is your diagnosis ?

A.Capillaria Philippinensis
B.Ankylostoma canium
C.Strongyloides stercolis
D.Enterobius vermicularis

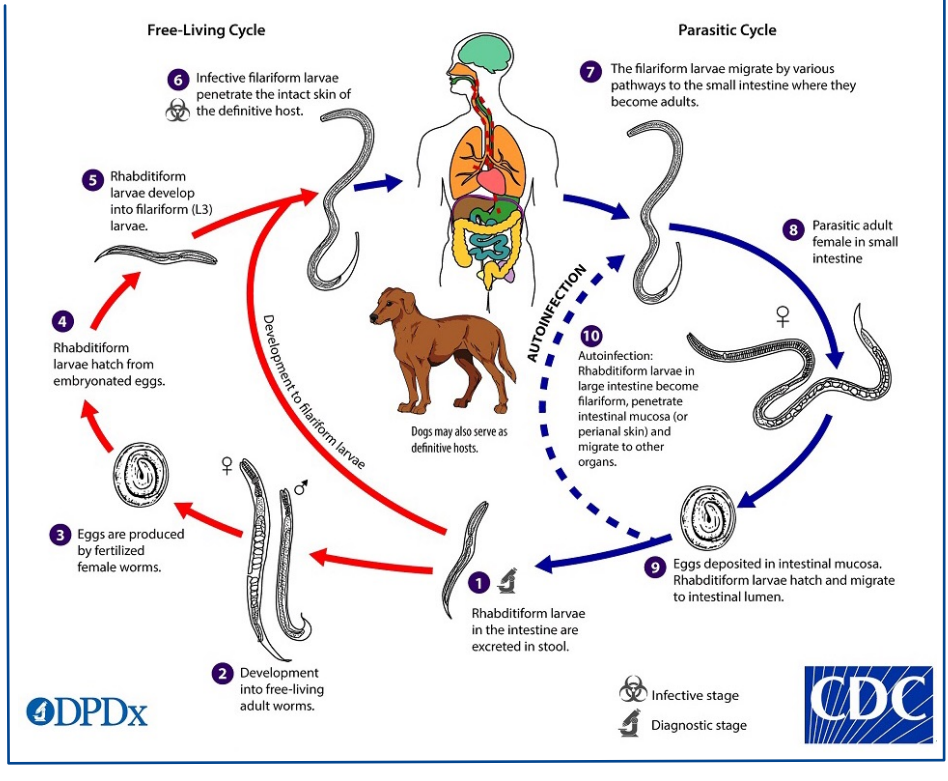


- Based on given history and multiple larvae in BAL is suggestive of Strongyloides

STRONGYLOIDES STERCORALIS

- Pulmonary capillaries into the alveoli, small hemorrhages may occur in the alveoli and bronchioles.
- Bronchopneumonia may be present,which may progress to chronic bronchitis and asthmatic symptoms in some patients.
- Larva of Strongyloides may be found in the sputum of these patients.

LIFE CYCLE OF STRONGLOIDES STERCORALIS



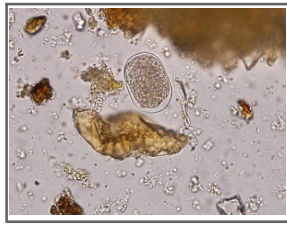
Reference :
Paniker's Textbook of Medical Parasitology 8th Ed

CLINICAL MANIFESTATIONS

- **Cutaneous Manifestations :**
  - Dermatitis, with erythema and itching at the site of penetration of the filariform larva, particularly when large numbers oflarvae enter the skin.
  - Pruritus and urticaria, particularly around the perianal skin and buttocks, are symptoms of chronic strongyloidiasis.
  - Term larva currens (meaning racing larvae) has been applied to the rapidly progressing linear or serpiginous urticarial tracks caused by migrating filariform larvae.
  - These often follow autoinfection and start perianally.
- **Pulmonary Manifestations :**
  - When the larva escape from the pulmonary capillaries into the alveoli, small hemorrhages may occur in the alveoli and bronchioles.
  - Bronchopneumonia may be present, which may progress to chronic bronchitis and asthmatic symptoms in some patients.
  - Larva of Strongyloides may be found in the sputum of these patients.
- **Intestinal Manifestations :**
  - The symptoms may resemble those of peptic ulcer or of malabsorption syndrome.
  - Mucus diarrhea is often present
  - In heavy infection, the mucosa may be honeycombed with the worm and there may be extensive sloughing, causing dysenteric stools.
  - Other manifestations are protein-losing enteropathy and paralytic ileus.

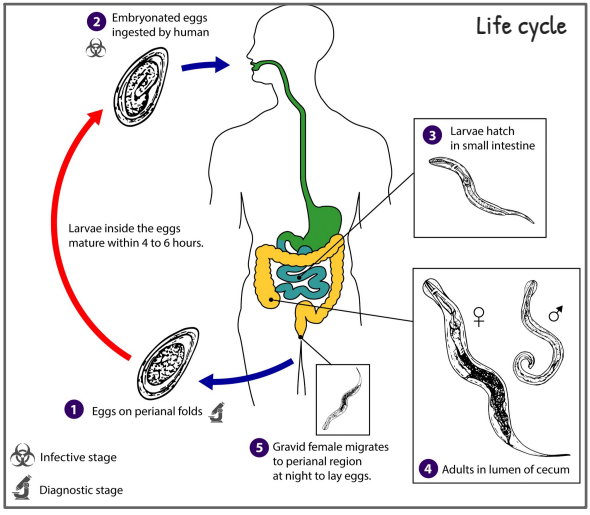
LABORATORY DIAGNOSIS

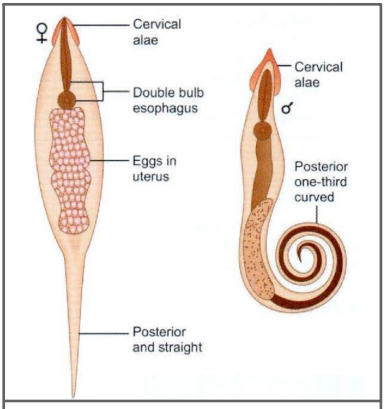
- **Microscopy :**
  - Direct wet mount of stool : Demonstrates rhabditiform larva (definitive diagnosis}
  - Stool concentrations methods:
    - i.Formol ether concentration
    - ii.Baermann's funnel gauze
  - Demonstration of larva in sputum or duodenal aspirates or jejunal biopsies
- **Stool culture :**
  - Done when larvae are scanty in stools
  - Methods used :
    - i. Agar plate culture
    - ii.Charcoal culture method
- **Blood examination :**
  - Peripheral eosinophilia
  - Raised serum IgE levels



Eggs are conspicuous within the uterus ofgravid female. Each uterus contains 8-10 eggs arranged anteroposteriorly in a single row.

ENTEROBIUS VERMICULARIS





Adult worm of Enterobius vermicularis (male and female)

- Enterobius uermicularis is considered to be world's most common parasite, which specially affects the children.

CLINICAL FEATURES

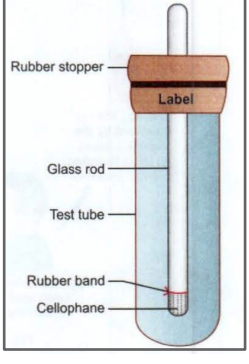
- Occurs mostly in children.
- It is more common in females than in males.
- About one-third of infections are asymptomatic.
- Pruritus ani
- Nocturnal enuresis

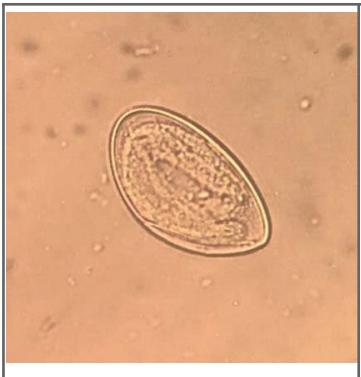
LABORATORY DIAGNOSIS

i. Detection of egg :
- Under finger nails
- NIH Swab Method
- Scotch Tape Method

ii. Detection of adult worm :
- Stool sample

NIH Swab

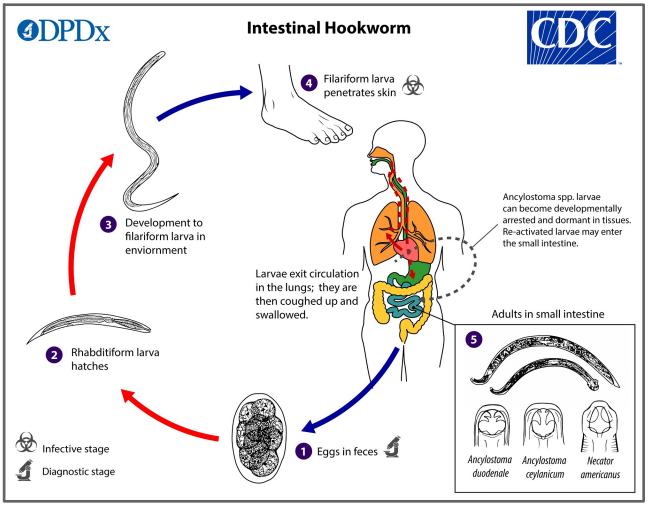


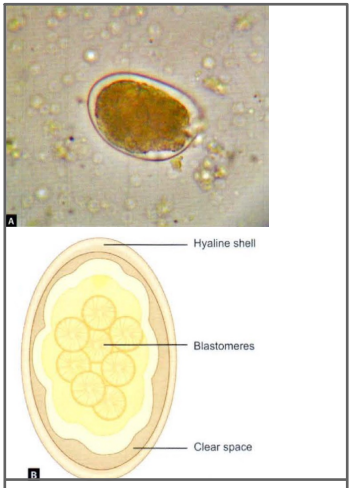


- Egg is colorless and not bile-stained.
- It floats in saturated salt solution.
- Has a characteristic shape, being elongated ovoid, flattened on one side and convex on the other (plano-convex), measuring 50-60µm by 20-30 µm.
- The egg contains a tadpole-shaped coiled embryo, which is fully formed, but becomes infectious only 6 hours after beingdeposited on the skin.

Reference :
Paniker's Textbook of Medical Parasitology 8th Ed

ANCYLOSTOMA DUODENALE





Egg of Ancylostoma duodenale.
(A) As seen under microscope; and
(B) Schematic diagram

CLINICAL FEATURES

i. Ground itch
ii. Creeping eruption: ft is formed due to subcutaneous migration of filariform larvae.
iii. Mild transient pneumonitis, or bronchitis

LABORATORYDIAGNOSIS

i. Direct methods :
- Demonstration of eggs in feces : Direct wet microscopy or by concentration method or in duodenal aspirate.
- Demonstration of adult Worm in feces or duodenal aspirate (specific diagnosis)
- Stool culture by Harada-Mori method

ii. Indirect methods :
- Blood examination Microcytic hypochromic anemia and eosinophilia
- Stool examination : To demonstrate presence of occult blood and Charcot-Leyden crystals
- Chest X-ray

TREATMENT

- Effective drug is Albendazole (400 mg single dose) or Mebendazole (500 mg once).
- Pyrantel pamoate (11 mg/kg x 3 days) is also effective and can be used in pregnancy.
- Treatment of hookworm disease also includes relief of anemia.

Reference :
Paniker's Textbook of Medical Parasitology 8th Ed

CAPILLARIA PHILIPPINENSIS

- C.phillippinensisis a small nematode, about 3-4 mm long.
- It belongs to the superfamilyTrichuroidea.

CLINICAL FEATURES

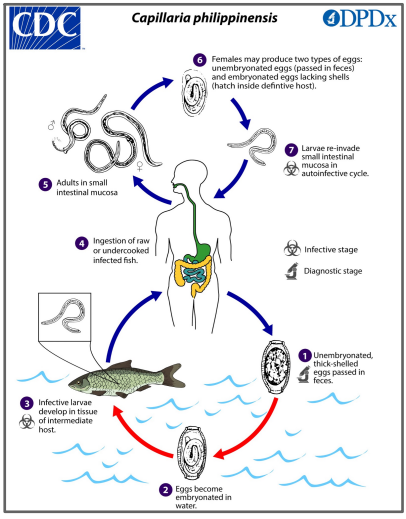
- Malabsorption syndrome with severe diarrhea,
- Borborygmi and
- Abdominal pain.

DIAGNOSIS

- Made by detection of the eggs, larvae and adults in stools.
- The eggs resemble those of Trichuris trichiura, but are smaller.

TREATMENT

- Mebendazole is useful in treatment.



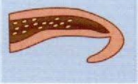
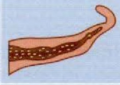
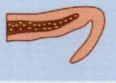
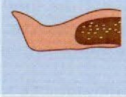
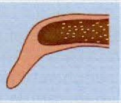
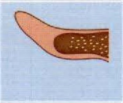
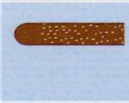
2. Name the parasite whose microfilariae have a sheath with no nuclei at the tail end :

A. W.bancrofti
B. Bruges malayi
C. Loa loa
D. B.timori

- Microfilariae parasites with don't have nuclei to extend to the tip of tail are :

i. W.bancrofti
ii. Mansonella ozzardi
iii. Onchocerca volvulus

Head and tail ends of microfilariae found in humans

Species	*Wuchereria bancrofti*	*Brugia malayi*	*Loa loa*	*Mansonella perstans*	*Mansonella ozzardi*	*Onchocerca volvulus*
Shape						
Posterior end						
Tail nuclei	Nuclei do not extend to the tip of tail	2 nuclei at the tip of the tail	Nuclei form continuous row in the tip of the tail	Nuclei extend to the tip of the tail	Nuclei do not extend to the tip of the tail	Nuclei do not extend to the tip of the tail
Anterior end						
Size	300 × 8 µm	220 × 6 µm	270 × 8 µm	180 × 4 µm	220 × 4 µm	200 × 360 µm
Sheathed/unsheathed	Sheathed	Sheathed	Sheathed	Unsheathed	Unsheathed	Unsheathed
Habitat	Blood	Blood	Blood	Blood	Blood	Skin, eye

Reference :
Paniker's Textbook of Medical Parasitology 8th Ed

3. A patient from Uttar Pradesh are presented to OPD with fever.On examination hepatomegaly was present.He was subjected to bone marrow biopsy which revealed macrophages under microscopy containing organism showing its kinetoplast stage.What is the vector causing the condition ?

A. Tsetse fly
B. Sandfly
C. Anopheles
D. Ixodes

- Based on history given by patient he is suffering from Leishmaniasis.

LEISHMANIA

- All members of the genus Leishmania are obligate intracellularparasites that pass their life cycle in two hosts :
  (1) The mammalian host, and
  (2) the insect vector, female sandfly.

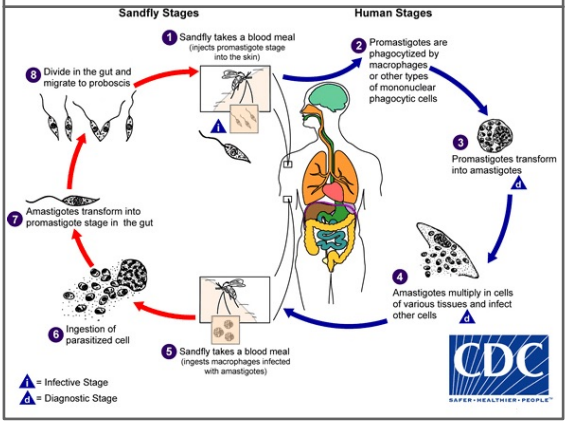
- Across the tropics, three different diseases are caused by various species ofgenus Leishmania. These are :
  i. Visceral leishmaniasis : The species L. donouani complex infecting internal organs (liver, spleen and bone marrow) of human is the causative parasite.
  ii. Cutaneous leishmaniasis: The species L. tropica complex, L. aethiopica, L. major and L. mexicana complex are the causative parasite.
  iii. Mucocutaneous leishmaniasis: It is caused by the L. braziliensis complex.

CLINICAL FEATURES

- High-grade fever which may be remittent with twice daily spikes or intermittent or less commonly continuous .
- Splenomegaly starts early and is progressive and massive. It is usually soft and nontender.
- Hepatomegaly is moderate.
- Lymphadenopathy.
- Skin becomes dry, rough and darkly pigmented.
- The hair becomes thin and brittle.
- Cachexia with marked anemia, emaciation and loss of weight is seen.
- Ascites and edema may occur due to hypoalbuminemia.

Reference :
Paniker's Textbook of Medical Parasitology 8th Ed

Distribution and disease caused by Leishmania spp.



MORPHOLOGY

- The parasite exists in two forms :
  1. Amastigote form: In humans and other mammals.
  2. Promastigote form: In the sandfly and in artificial culture.

LABORATORY DIAGNOSIS

i. Direct evidence :

- Demonstration of LD bodies : In stained smears of thick blood film, splenic, bone marrow, and lymph node aspirate
- Culture : In NNN medium or Schneider's liquid medium to demonstrate promastigote form
- Animal inoculation : In hamster or mice

ii. Indirect evidence :

(A) Serodiagnosis :

- Detection of antigen : ELISA
- Detection of antibody
  - (a) CFT using WKK antigen
  - (b) DAT
  - (c) IFAT
  - (d) CIEP
  - (e) DOT-ELISA
  - (f) ICT using rK39 antigen

(B) Molecular diagnosis :

- DNA probe
- PCR

(C) Non specific Serum Test :

- Aldehyde test or
- Chopra's antimony
- The tests are test positive in hypergammaglobulinemia

(D) Skin test : Leishmanin or Montenegro test

(E) Blood picture :

- Anemia
- Progressive leukopenia
- Reverse albumin : globulin ratio

TSETSE FLY

- Is a vector for Trypanosoma Brucei
- Clinical Features of Trypanosomiasis :
  - i. Painless chancre
  - ii. Winterbottom's sign : is a swelling of lymph nodes (lymphadenopathy) along the posterior cervical lymph node chain.
  - iii. Hematological manifestations include :
    - Anemia,
    - Moderate leukocytosis and
    - Thrombocytopenia.
- Abnormalities in cerebrospinal fluid (CSF) include raised intracranial pressure, pleocytosis and raised total protein concentrations.

ANOPHELES MOSQUITO

- Anopheles gambiae is one of the best known, because of its predominant role in the transmission of the most dangerous malaria parasite species (to humans) – Plasmodium falciparum.
- It is also a vector for Lymphatic filariasis

IXODES

- It includes important disease vectors of animals and humans (tick-borne disease), and some species (notably Ixodes holocyclus) inject toxins that can cause paralysis.
- Some ticks in this genus may transmit the pathogenic bacterium Borrelia burgdorferi responsible for causing Lyme disease.
- Additional organisms that may be transmitted by Ixodes are parasites from the genus Babesia, which cause babesiosis, and bacteria from the related genus Anaplasma, which cause anaplasmosis.

4.A Truck driver came with complains of pain in genital area.On examination there was ulcer in genital area.He gives h/o of unsafe intercourse.Which among the following can be used for visualising motility of organism ?

A.Dark field microscope
B.Electron microscope
C.Fluorescent microscope
D.Light microscope

- From the given history he had an unsafe intercourse following which an ulcer developed in genital area, most probable diagnosis is Syphilis caused by Treponema pallidum.
- Inorder to visualise motility of the organism Dark field microscope is used.

SYPHILIS

- Causative agent : Treponema pallidum
- It is a thin, delicate spirochete with tapering ends, about 10 µm long (range 4–14 µm) and 0.1– 0.2 µm wide.
- It has about ten regular spirals, which are sharp and angular, at regular intervals of about 1 µm.
- It is actively motile, exhibiting rotation around the long axis, backward and forward movements, and flexion of the whole body.
- Can be acquired by the venereal or non-venereal route or be congenital or acquired.
  1. Venereal syphilis is acquired by sexual contact :
     - The primary lesion in syphilis is the chancre at the site of entry of the spirochete
     - It is known as 'hard chancre' to distinguish it from the non-indurated lesions of 'soft chancre' caused by H.ducreyi.
     - Secondary syphilis sets in 1–3 months after the primary lesion heals.
     - After the secondary lesions disappear, there is a period of quiescence known as latent syphilis.
  2. Late tertiary or quanternary syphilis :
  3. Congenital syphilis, where infection is transmitted from mother to fetus transplacentally, the manifestations and course are different.
- It can be stained by silver impregnation methods.
- Fontana's method is useful for staining films and Levaditi's method for tissue sections.

LABORATORY DIAGNOSIS

- Consists of demonstration of the spirochetes under the microscope and of antibodies in serum or CSF.
- Microscopy :
  i. Dark ground examination
  ii. Direct fluorescent antibody test for T.pallidum (DFA-TP) is a better and safer method for microscopic diagnosis.
  iii. Silver impregnation smears can be stained by methods.

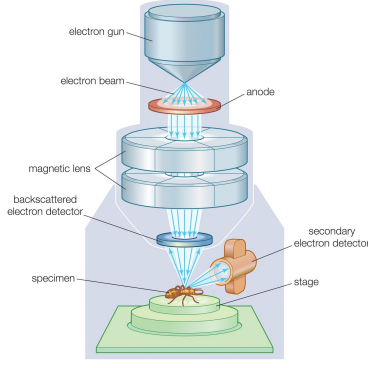
Reference :
Ananthanarayan and Paniker's Textbook of Microbiology 10th Ed.

- **Serological tests** : These tests form the mainstay of laboratory diagnosis.
  - i. Reagin antibody tests :
    - Use the lipoidal or cardiolipin antigens and are known as standard tests for syphilis (STS).
- **Specific T.pallidum tests** :
  - i. Treponema pallidum immobilisation (TPI) : **Gold standard in syphilis serology.**
  - ii. Fluorescent treponemal antibody (FTA) test
  - iii. T.pallidum hemagglutination assay (TPHA)
  - iv. Enzyme immunoassays (EIA) have been developed using T.pallidum antigens
- **Response to treatment** :
  - Quantitative tests are useful in monitoring the patient's response to treatment
  - VDRL or RPR is preferred because they usually become negative following treatment.
- **Diagnosis: TPHA and FTA-ABS** are helpful in excluding or confirming the diagnosis of syphilis and for identifying BFP reactions.

Motile spirochetes as seen under Dark field microscope

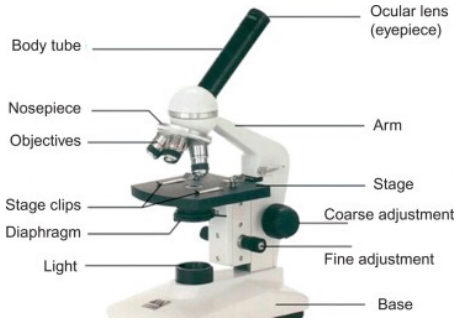
ELECTRON MICROSCOPE

- Is a microscope that uses a beam of accelerated electrons as a source of illumination.
- Applications :
  - Electron microscopes are used to investigate the ultrastructure of a wide range of biological and inorganic specimens including microorganisms, cells, large molecules, biopsy samples, metals, and crystals.
  - Industrially, electron microscopes are often used for quality control and failure analysis.



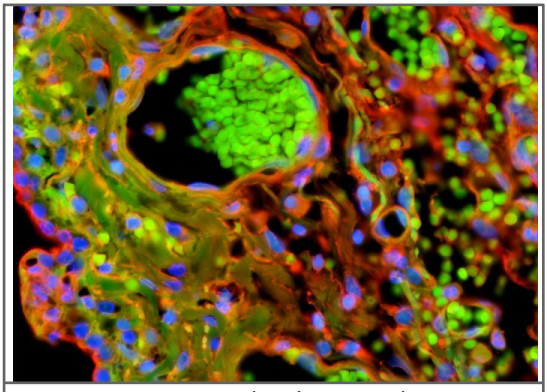
LIGHT MICROSCOPE

- Is a type of microscope that commonly uses visible light and a system of lenses to generate magnified images of small objects.
- They can be used to examine a wide variety of types of specimen, frequently with minimal preparation.



FLUORESCENCE MICROSCOPE

- Is an optical microscope that uses fluorescence instead of, or in addition to, scattering, reflection, and attenuation or absorption, to study the properties of organic or inorganic substances.



Human Lung Tissue under Fluorescent microscope

5.A patient was suspected to have brucellosis.Serum sample was sent for standard agglutination test.It came out to be negative initially but after dilution of sample,the test was positive.What could be reason for initial negative test ?

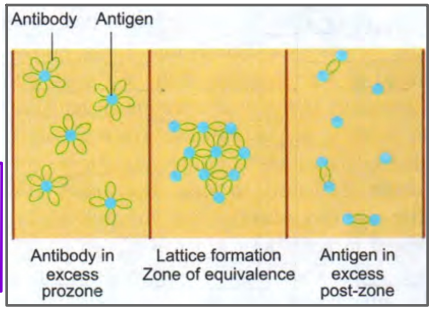
A. Postzone phenomenon
B. Prozone phenomenon
C. Complement activation
D. Inadequate antibodies

- STAT has been used extensively for many years in the diagnosis and surveillance of brucellosis.
- The serum (tube) agglutination test (SAT) detects antibodies to the S-LPS (smooth (S) lipopolysaccharide (LPS)

Reference :
Notespaedia Microbiology IB Pg : 31
Ananthanarayan and Paniker's Textbook of Microbiology 10th Ed.

POST-ZONE PHENOMENON

- This is caused by the presence of excess antigen in the test system.
- No visible reaction will occur.



PROZONE PHENOMENON

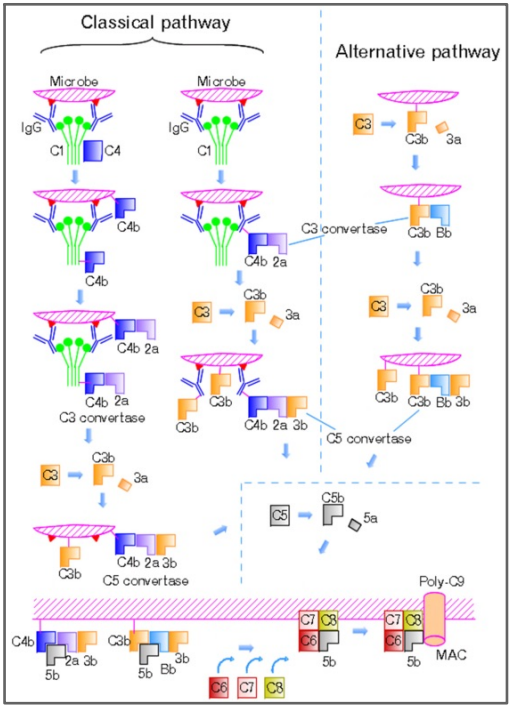
- This is caused by excess antibody in the test system.
- Failure of a visible reaction is due to inhibition of lattice formation by the excess antibody.

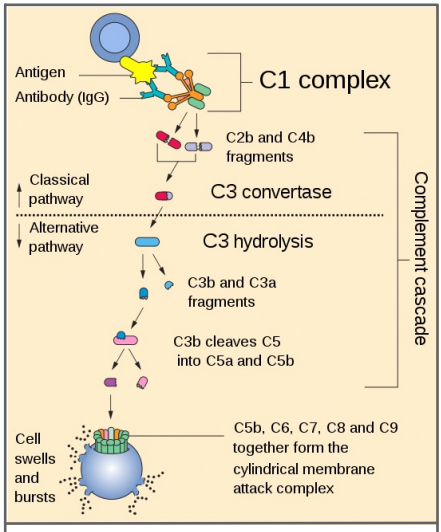
COMPLEMENT ACTIVATION

- Complement is normally present in the body in an inactive form but when its activity is induced by antigen- antibody combination or other stimuli, C components react in a specific sequence as a cascade.

CLASSICAL COMPLEMENT PATHWAY

- The chain of events in which complement components react in a specific sequence following activation C1qrs of and typically culminate in immune cytolysis is known as the classical pathway
- Step :





The classical complement pathway leading into a complement cascade that is shared with the alternate pathway.

7. A 11 yr old child presents with bluish white spots in the buccal mucosa followed by skin rashes. What is the genome of the causative organism ?

A. Single stranded naked RNA
B. Single stranded enveloped RNA
C. Double stranded naked RNA
D. Double stranded enveloped RNA

- From the given most probable diagnosis is Measles.

MEASLES

- Is a Morbillivirus, of the family Paramyxoviridae, and is an enveloped single-stranded negative-sense RNA virus

CLINICAL FEATURES

- Prodromal malaise, fever, conjunctival injection, cough and nasal discharge.
- Koplik's spot :
  - Develop on the buccal mucosa and occasionally on the conjunctiva and intestinal mucosa.
  - The prodromal illness subsides within a day or two of appearance of the rash.
  - The red maculopapular rash of measles typically appears on the forehead first and spreads downwards, to disappear in the same sequence 3-6 days later, leaving behind a brownish discolouration and finely granular desquamation
  - Cytopathic effects consist of multinucleate syncytium formation, with numerous acidophilic nuclear and cytoplasmic inclusions.
  - Multinucleate giant cells (Warthin- Finkeldey cells) are also found in the lymphoid tissues of patients.

LABORATORY DIAGNOSIS

- Specimen :
  - Nasal secretions, throat, conjunctiva and blood can be used.
  - CSF is collected in SSPE.
- Direct microscopy :
  - A simple diagnostic test, which can be used even before the rash appears, is the demonstration of multinucleated giant cells in Giemsa- stained smears of nasal secretions.
- IFA :
  - The measles virus antigen can be detected in cells of nasal secretions by immunofluorescence.
- Virus isolation :
  - Virus can be isolated from the nose, throat, conjunctiva and blood during the prodromal phase and up to about two days after the appearance of the rash.
  - The virus may be obtained from urine for a few more days.
  - Primary human or monkey kidney and amnion cells are most useful.
  - Cytopathic changes may take up to a week to develop, but earlier diagnosis of viral growth is possible by immunofluorescence.

- PCR :
  - Reverse transcriptase PCR is a sensitive and specific method of diagnosis.

8. A group of friends had a party and ate pastries from a shop late in the night.All developed vomiting early morning.Identify the most probable causative organism ?

A. Shigella dysenteriae
B. Emetic form of Bacillus cereus
C. Staph aureus
D. Salmonella enteritidis

Incubation period	Symptoms	Common Food Sources
1 -6 hrs		
Staph.aureus	Nausea,Vomiting,Diarrhoea	Ham,Poultry,potato or egg salad,Myaonnaise,Cream pastries.
Bacillus cereus	Nausea,Vomiting,Diarrhoea	Fried rice
8 - 16hrs		
Clostridium perfringens	Abdominal cramps, Diarrhoea(Vomiting rare)	Beef poultry,legumes,gravies
B.cereus	Abdominal cramps, Diarrhoea(Vomiting rare)	Meats,vegetables,dried beans,cereals
> 16hrs		
V.cholerae	Watery diarrhoea	Shellfish,water
Enterotoxigenic E.coli	Watery diarrhoea	Salads cheese,meats,water
Enterohemorrhagic E.coli	Bloody diarrhoea	Ground beef,Roast beef,Salami, Raw milk,Raw vegetables
Salmonella spp.	Inflammatory diarrhoea	Beef,poultry,eggs,dairy products
Shigella	Dysentry	Potato or egg salad,lettuce,Raw vegetables

9. A known case of AIDS with productive cough and fever was found to have consolidation in right infracapsualr area with X ray showing Right lower lobe consolidation with CD 4 counts of 55 per micro litre.What is the most common cause of this ?

A. Staph aureus
B. Streptococcus pneumoniae
C. Mycoplasma
D. Pneumocyctis jiroverci

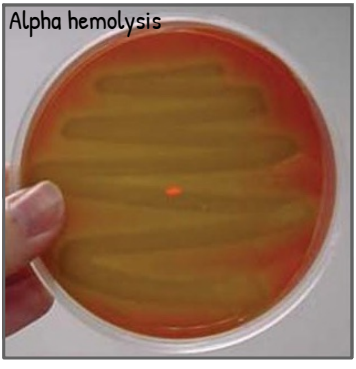
- Currently, the most frequent diagnosis in developed countries is bacterial pneumonia, especially Pneumococcal pneumonia, the second most frequent cause is Pneumocystis pneumonia and the third is Tuberculosis.

STREPTOCOCCUS PNEUMONIAE

- Gram-positive lanceolate-shaped diplococcus (commonly referred to as Pneumococci).
- They are the single most prevalent bacterial agents in pneumonia and in otitis media in children.
- They can also cause sinusitis, bronchitis, bacteremia, meningitis and other infections.

CULTURAL CHARACTERISTICS

- They are aerobes and facultative anaerobes, the optimum temperature being 37°C (range 25-42°C) and pH 7.8 (range 6.5-8.3).
- Growth is improved by 5-10% C02.
- On Blood agar :
  - After incubation for 18 hours, the colonies are small (0.5-1 mm), dome-shaped glistening, with an area of green, discolouration (alpha hemolysis) around them, resembling colonies of viridans streptococci.
  - Draughtsman or carrom coin appearance
- Under anaerobic conditions colonies on blood agar are surrounded by a zone of beta hemolysis due to oxygen-labile hemolysin O.
- Biochemical reactions :
  - Catalase and oxidase negative.
  - Fermentation is tested in Hiss's serum sugars.
  - Bile solubility test : S.pneumoniae are bile soluble.



LABORATORY DIAGNOSIS

1. Specimen : Sputum,CSF, blood for culture and urine are used for antigen detection.
2 . Microscopy: In the acute phase of lobar pneumonia, the rusty sputum contains S.pneumoniae in large numbers, with hardly any other kind of bacterium.

Reference :
https://erj.ersjournals.com/content/39/3/730

3. Biomarkers :
- CRP testing, by passive agglutination using latex particles coated with anti-CRP antibody, a routine diagnostic procedure.
- Procalcitonin is another biomarker which is elevated in invasive pneumococcal disease.

4. Antigen detection :
- Culture negative diagnosis by demonstrating the SSS in CSF by precipitation with antisera or the latex agglutination test.

TREATMENT

- Antibiotic of choice is parenteral penicillin in ,severe cases and amoxycillin in milder ones.
- Vancomycin is to be reserved for life-threatening illnesses with highly resistant strains.

PNEUMOCYSTIS JIROVECI

- Pneumocystis pneumonia is one ofthe common opportunistic infections in AIDS.

PATHOGENESIS AND CLINICAL FEATURES

Infection is transmitted by respiratory droplets and is asymptomatic in ipmunocompetent individuals.

LABORATORY DIAGNOSIS

- Microscopy :
  - Trophozoites can be demonstrated by Giemsa, tolcidine blue, methenamine silver and calcofluor white stains.
  - The cyst wall stains black with methenamine silver stain.
- Culture : The organism cannot be cultured.
- Serology :
  - Complement fixation test with titres of 1:4 or above indicate active disease.
  - Latex agglutination test can also be used.

TREATMENT

- Trimethoprim-sulfamethoxazole (TMP-SMZ) and Pentamidine isothionate are the drugs of choice for the treatment of acute cases.

10. A patient presented with 70% burns.A test was done on a sample collected from the burn site is as shown in the picture in suscpicion of probable causative organism which is an obligate aerobe.What is the microbe ?

A. Pseudomonas
B. E.coli
C. Klebsiella
D. Staphylococcus aureus

- Based on information given in the question the organism is an obligate aerobe and is oxidase positive which is suggestive of Pseudomonas.

PSEUDOMONAS AERUGINOSA

- Slender, Gram-negative bacillus
- Actively motile by a polar flagellum
- Non-capsulated but many strains have a mucoid slime layer.

CULTURAL CHARACTERISTICS

- Ordinary media : It grows well producing large, opaque, irregular colonies, with a distinctive, musty, mawkish or earthy smell.
- Nutrient agar : Iridescent patches with a metallic sheen are seen in cultures with crystals beneath the patches.
- MacConkey medium : It forms non-lactose fermenting colonies.
- Blood agar : Many strains are hemolytic on blood agar.
- Broth : It forms a dense turbidity with a surface pellicle.
- Pigment production : P.aeruginosa produces a number of pigments, the best known being pyocyanin and fluorescein.

PATHOGENICITY

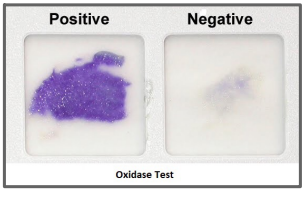
- 'Blue pus'
- Several toxic extracellular products have been identified :
  - i. Exotoxin A :
    - has a mechanism of action similar to that of the diphtheria toxin.
  - ii. Other extracellular enzymes and toxins include proteases, elastases, hemolysins and enterotoxin.
  - iii. The slime layer acts as a capsule in enhancing virulence.

LABORATORY DIAGNOSIS

- Selective media may be necessary such as cetrimide agar for isolation from feces or other samples with mixed flora.
- Species identification is done by biochemical tests.

BIOCHEMICAL REACTIONS

- Metabolism is oxidative and non-fermentative.
- It is catalase positive, oxidase positive and motile.
- Hugh and Leifson's medium :
  - Glucose is used to test an oxidative attack on sugars.
- Nitrates are reduced to nitrites and further to gaseous nitrogen;
- Arginine dihydrolase test is positive.



ESCHERICHIA COLI

- Gram-negative, straight rod arranged singly or in pairs.
- Motile by peritrichate flagella, though some strains may be non-motile.

CULTURAL CHARACTERISTICS

- Grows aerobically and is a facultative anaerobe.
- On blood agar : hemolytic
- On MacConkey medium : colonies are bright pink due to lactose fermentation.
- On selective media, growth is largely inhibited, such as DCA or SS agar used for the isolation of salmonellae and shigellae.

ANTIGENIC STRUCTURE

- Serotyping or antigenic typing of E.coli on three antigens :
  - Somatic antigen O,
  - Flagellar antigen H and
  - Capsular antigen K.
- F antigens : Fimbriae are important virulence factors. They are heat labile

KLEBSIELLA PNEUMONIAE

- a.k.a Friedlander's bacillus, Bacillus mucosus capsulatus
- Non-motile rods, which are capsulated short, plump and straight.
- Mucopolysaccharide capsule is often prominent
- Gram-stained smears as halos around the rods.

CULTURAL CHARACTERISTICS

- Grow well on ordinary media, forming large, dome- shaped, mucoid colonies of varying degrees of stickiness.
- On MacConkey agar, they grow as lactose-forming colonies.

BIOCHEMICAL REACTIONS

- Ferment carbohydrates (glucose, lactose, sucrose and mannitol) - the production of acid and abundant gas.
- IMViC reaction is - -++

STAPHYLOCOCCUS AUREUS

- Spherical cocci
- Arranged characteristically in grape -like clusters.
- They are non motile,non sporing
- They stain readly with anilline dyes and are uniformly Gram positive.

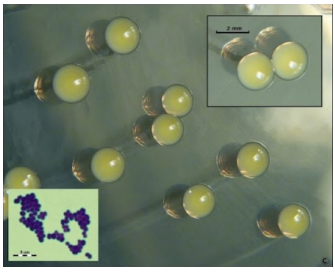
CULTURAL CHARACTERISTICS

- Solid medium :
  - i. On nutrient agar :
    - After incubation for ,t4 hours, the colonies are large (2-4 mm in diameter), circular,convex, smooth,shiny, opaque and easily emulsifiable.

- characteristic 'oil paint' appearance.

ii. On blood agar :
- Most strains are pemolytic, especially when incubated under 20-25% carbon dioxide.
- Hemolysis is marked on rabbit or sheep blood and weak on horse blood agar.

iii. MacConkey agar, they produce smaller colonies that appear pink due to lactose fermentation.



SELECTIVE MEDIA

- For isolating S.aureus from specimens such as feces containing other bacteria.
- These include media containing :
  i. 8 to 10 % NaCl (salt-milk agar, salt broth),
  ii.Lithium chloride and tellurite. (Ludlam's medium) and polymyxin.

BIOCHEMICAL REACTIONS

i. Coagulase test :
- Tube coagulase test detects free coagulase (extra-cellular product) of S.aureus.
- The slide test detects bound coagulase (clumping factor) but can be positive for some of the CONS.

ii. Other biochemical tests :
- Catalase-positive (unlike streptococci) and usually hydrolyse urea,
- Reduce nitrates to nitrites, liquefy gelatin and are MR- and VP-positive but indole-negative.

11. Which among the following is used for Blood Spill management

A. Chlorhexidine
B. Formaldehyde
C. Ethylalcohol
D. Sodium Hypohlorite

BLOOD SPILLAGE

- In this situation, clean up the spillage and record the incident, using the following procedure.

1. Wear a pair of non-sterile gloves.
2. Use tongs or a pan and brush to sweep up as much of the broken glass (or container) as possible. Do not pick up pieces with your hands.
3. Discard the broken glass in a sharps container.
   - If this is not possible due to the size of the broken glass, wrap the glass or container in several layers of paper and discard it carefully in a separate container.
   - Do not place it in the regular waste container.
4. Use disposable paper towels to absorb as much of the body fluids as possible.

Reference :
https://www.ncbi.nlm.nih.gov/books/NBK138660/

5. Wipe the area with water and detergent until it is visibly clean.
6. Saturate the area again with sodium hypochlorite 0.5% (10 000 ppm available chlorine).
   - This is a 1:10 dilution of 5.25% sodium hypochlorite bleach, which should be prepared daily.
7. Rinse off the tongs, brush and pan, under running water and place to dry.
8. Remove gloves and discard them.
9. Wash hands carefully with soap and water, and dry thoroughly with single-use towels.
10. Record the incident in the incident book if a specimen was lost, or persons were exposed to blood and body fluids.

12. The young boy/child presented with bloody stools and cramping pain in the abdomen.Identify the medium used in culturing the causative organism in the given picture :

A. Selenite F Broth Enrichment media
B. Alkaline Peptone Water
C. Brain Heart Infusion
D. Loeffler's serum

- Bloody stools,cramping pain abdomen and give image of culturing media showing Selenite F Broth suggestive of Bacillary Dysentry.

SHIGELLA

- Short, Gram-negative rods.
- Non-motile, non-sporing and non-capsulated.
- Fimbriae may be present.

CULTURAL CHARACTERISTICS

- Aerobes and facultative anaerobes.
- Colonies on MacConkey agar are colourless due to the absence of lactose fermentation.
- Deoxycholate citrate agar (DCA) and xylose lysine deoxycholate (XLD) is a useful selective medium (in which shigella do not have a black centre as against Salmonella which appears red with a black centre).
- Growth is inhibited on Wilson and Blair bismuth sulphite medium.

BIOCHEMICAL REACTIONS

- MR positive and reduce nitrates to nitrites.
- Do not form H S and are inhibited by KCN.

ANTIGENIC STRUCTURE

- Possess one or more 'major' antigens and a large number of 'minor' somatic O antigens.
- Some strains possess K antigens.
- Fimbrial antigens are also present.

- S.dysenteriae type 1 forms a toxin (Shiga toxin - Exotoxin produced by Gram negative bacillus).
- Three types of toxic activity have been demonstrated in shigella culture filtrates :
  - Neurotoxicity
  - Enterotoxicity
  - Cytotoxicity

BACILLARY DYSENTERY

CLINICAL FEATURES

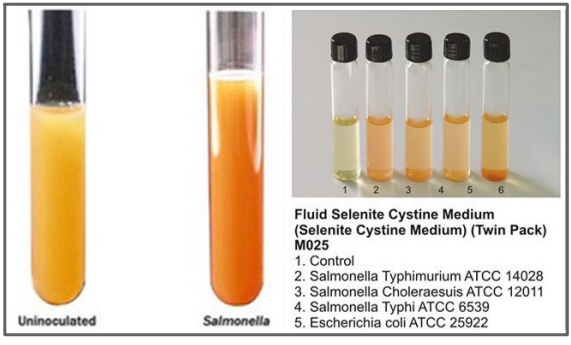
- Main frequent passage of loose, scanty feces containing blood and mucus, along with abdominal cramps and tenesmus.
- Fever and vomiting may be present.
- Hemolytic uremic syndrome may occur.

LABORATORY DIAGNOSIS

- Sachs' buffered glycerol saline or Gram-negative broth
- Selenite F broth or Salmonella Shigella (SS) broth

i. Culture :

- MacConkey and DCA or XLD plates are inoculated.
- Selective medium used for isolation is the SS agar where colonies are colourless.



Pharmacology

1. A patient with bronchial asthma developed glaucoma. Which of the following drug will be used for OAG in this patient ?

A. Lantanoprost
B. Carboprost
C. Alprostadil
D. Gemeprost

- Among the given options only Latanoprost is used for Glaucoma
- Alprostadil is used in Pulmonary hypertension,Erectile dysfunction
- Gemeprost is used in Cervical ripening
- Carboprost is used in Post Partum Hemorrhage

LATANOPROST

- PG F2α derivative has shown efficacy similar to timolol (i.o.t. reduction by 25-35%)
- Effect is well sustained over long-term.
- Once daily application and absence of systemic complications, PG analogues have become the first choice drugs for open angle glaucoma.

SIDE EFFECT

- Blurring of vision,
- Increased iris pigmentation,
- Thickening and darkening of eyelashes
- Macular edema can develop during treatment

ALPROSTADIL

- PGE 1 Analogue injected directly into the corpus cavernosum using a fine needle produces erection lasting 1-2 hours to pemit intercourse.
- Local tenderness may occur.
- Penile fibrosis and priapism are rare.
- Most commonly used drug in patients not responding to PDE-5 inhibitors, such as neurogenic and psychogenic ED.
- A transurethral pellet termed ' medicated urethral system for erection' (MUSE) has been developed which avoids intracavernosal injection

CARBOPROST

- 15-methyl PG F2α
- Used as i.m. is an alternative drug for control of PPH due to uterine atony, especially in patients unresponsive to ergometrine and oxytocin. Q

Reference :
Essentials of Medical Pharmacology by Tripathi 8th Ed.

GEMEPROST

- Dinoprostone (PGE2)
- Applied intravaginally or in the cervical canal, low doses of PGE,which do not affect uterine motility, make the cervix soft and compliant.
- If needed labour may be induced 12 hours later with oxytocin : chances of failure are reduced.

2. Rate of administration of drug is equal to rate of elimination.How will you calculate the dosing rate of that drug to maintain steady state concentration ?

A. Dosing rate = Vd x Target Plasma Concentration
B. Dosing rate = CL x Target Plasma Concentration
C. Dosing rate = Vd/Target Plasma Concentration
D. Dosing rate = CL/Target Plasma Concentration

MAINTENANCE DOSE

- Is one that is to be repeated at specified intervals after the attainment of target Cpss (steady state plasma concentration) so as to maintain the same by balancing elimination.

Maintenance dose = CL x Target Plasma Concentration

LOADING DOSE

- Is a single or few quickly repeated doses given in the beginning to attain target concentration rapidly.
- Loading dose is governed only by V and not by CL or t1/2.

Loading Dose = Vd x Target Plasma Concentration

3. A 45yr old patient being treated with low dose aspirin since 6 months presented with rectal bleeding.Inhibition of which of the following substance is likely to be responsible for the bleeding ?

A. Thromboxane A2
B. Leukotriene
C. Bradykinin
D. PG I2

- TX A2 :
  - Produced locally by platelets
  - Is a potent inducer of aggregation and release reaction.
- PG I2
  - Generated by vascular endothelium.
  - Potent inhibitor of platelet aggregation.
- PG E2 : Has dose dependent pro- and anti-aggregatory effects

- Thromboxane and PG I2 has a mutually antagonistic system.
- Aspirin interferes with haemostasis by inhibiting platelet aggregation.

Reference :
Essentials of Medical Pharmacology by Tripathi 8th Ed. Pg : 200
Notespaedia Pharmacology IB Pg : 16

- TXA2 plays an important role in amplifying aggregation.
- Before it is deacetylated in liver, aspirin acetylates COX-1 in platelets while they are in portal circulation.
- Platelets are unable to regenerate fresh COX-1 (lack nucleus: do not synthesize protein), while vessel wall is able to do so (fresh enzyme is synthesized within hours).
- Thus, at low doses, Aspirin selectively inhibits TXA2 production and has anti thrombotic effect lasting > 3 days.

BRADYKININ

- Is a nonapeptide plasma kinin.
- Kinins are generated by proteolytic reactions triggered by tissue injury, inflammation, allergic reaction, etc., and play important mediator roles.

ACTIONS

i. Blood vessels : more potent vasodilators than ACh and histamine.

ii. Smooth muscles :
- Induce contraction of intestine which is characteristically slow.
- They cause marked bronchoconstriction in guineapigs and in asthmatic patients.
- Some smooth muscles may be relaxed.

iii. Neurons :
- Strongly stimulate nociceptive afferents and produce a burning sensation.
- Increase permeability of the blood-brain barrier.

iv. Kidney :
- Increase renal blood flow as well as facilitate salt and water excretion by action on tubules.

ICATIBANT :
- It is a synthetic decapeptide bradykinin B, receptor antagonist which is resistant lo kinin degrading enzymes
- Has a duration of action upto 6 hour.; after s.c. injection.
- It is used in Hereditary angioedema, Drug induced angioedcma , Burns, Pancreatitis.

4. Tocilizumab is a newer monoclonal antibody for treatment of Rheumatoid arthritis.It acts against :

A. IL-3
B. IL-12
C. IL-6
D. IL-2

THERAPEUTIC MONOCLONAL ANTIBODIES	TARGET; AB TYPE	THERAPEUTIC USE
Abciximab	GPIIb/IIIa; chimeric IgG1 Fab	Prevention of blood clots in angioplasty
Adalimumab	TNF, human IgG1	Rheumatoid arthritis

Reference :
Goodman and Gilmans Pharmacological Basis of Therapeutics 13th Ed. Pg : 661

THERAPEUTIC MONOCLONAL ANTIBODIES	TARGET; AB TYPE	THERAPEUTIC USE
Bevacizumab	VEGF; humanized IgG1	Colorectal cancer
Brodalumab	IL-17RA/human IgG2	Plaque psoriasis
Canakinumab	IL-1β; human IgG1	Muckle-Wells syndrome
Certolizumab pegol	TNF; humanized Fab, pegylated	Crohn disease
Daclizumab	IL-2R; humanized IgG1	Multiple sclerosis
Denosumab	RANK-L; human IgG2	Bone loss
Eculizumab	C5; humanized IgG2/4	Paroxysmal nocturnal hemoglobinuria
Golimumab	TNF; human IgG1	Rheumatoid and psoriatic arthritis, Ankylosing spondylitis
Idarucizumab	Dabigatran; humanized Fab	Dabigatran excess (reversing anticoagulation)
Infliximab	TNF; chimeric IgG1	Crohn disease
Natalizumab	α4 integrin; humanized IgG4	Multiple sclerosis
Ocrelizumab	CD20/human IgG1	Multiple Sclerosis
Omalizumab	IgE; humanized IgG1	Asthma
Palivizumab	RSV; humanized IgG1	Prevention of respiratory syncytial virus infection
Pembrolizumab	PD1; humanized IgG4	Melanoma, non-small cell carcinoma
Reslizumab	IL-5; humanized IgG4	Asthma
Rituximab	CD20; chimeric IgG1	Non-Hodgkin lymphoma
Sarilumab	IL-6R/human IgG1	Rheumatoid arthritis
Secukinumab	IL-17a; human IgG1	Psoriasis
Tocilizumab	IL-6R; humanized IgG1	Rheumatoid arthritis
Trastuzumab	HER2; hIgG1	Breast cancer
Ustekinumab	IL-12/23; human IgG1	Psoriasis

5 .Methotrexate mechanism of action in treatment of osteosarcoma is:

A. Competitive inhibitor of DHFR
B. Analog of UMP
C. Dihydropteroate synthase inhibitor
D. Pyrimidine analogue

METHOTREXATE

- Methotrexate is absorbed orally, 50% plasma protein bound, little metabolized and largely excreted unchanged in urine
- This folic acid analogue is one of the oldest and highly efficacious antineoplastic drugs

MECHANISM OF ACTION

- Acts by inhibiting dihydrofolate reductase (DHFRase)—blocking the conversion of dihydro- folic acid (DHFA) to tetrahydrofolic acid (THFA).
- Utilizing the folate carrier it enters into cells and is transformed to more active polyglutamate form by the enzyme folypolyglutamate synthase (FPGS).
- Tetrahydrofolic acid is an essential coenzyme required for one carbon transfer reactions in de novo purine synthesis and amino acid interconversions.
- The inhibition is pseudo- irreversible because Mtx has 50,000 times higher affinity for the enzyme than the normal substrate.

SIDE EFFECTS

- Methotrexate has cell cycle specific action— kills cells in S phase; In addition to DHFRase it inhibits thymidylate synthase as well so that DNA synthesis is primarily affected.
- However, synthesis of RNA and protein also suffers. It exerts major toxicity on bone marrow—low doses given repeatedly cause megaloblastic anaemia, but high doses produce pancytopenia.
- Mucositis and diarrhoea are common side effects. Desquamation and bleeding may occur in g.i.t.

TOXICITY

- Salicylates, sulfonamides, dicumerol displace it from protein binding sites.
- Aspirin and sulfonamides enhance toxicity of Mtx by decreasing its renal tubular secretion.
- The toxicity of Mtx cannot be overcome by folic acid, because it will not be converted to the active coenzyme form.
- However, Folinic acid (N5 formyl THFA, cirtrovorum factor) rapidly reverses the effects. Thymidine also counteracts Mtx toxicity.

Reference:Essentials of Medical Pharmacology Seventh Edition KD TRIPATHI, Pg 862

6. A patient is on metronidazole. Co - administration of which of the following to be avoided?

A. MAO inhibitor

B. Grape fruit juice

C. Alcohol

D. PHENTOLAMINE

METRONIDAZOLE

- It is the prototype nitroimidazole for trichomoniasis and later found to be a highly active amoebicide.
- It has broad-spectrum cidal activity against anaerobic protozoa, including Giardia lamblia in addition to the above two.
- Many anaerobic and microaerophilic bacteria, such as Bact. fragilis, Fusobacterium, Clostridium perfringens, Cl. difficile, Helico- bacter pylori, Campylobacter, peptococci, spirochetes and anaerobic Streptococci are sensitive.

PHARMACOKINETICS

- Metronidazole is almost completely absorbed from the small intestines; little unabsorbed drug reaches the colon.
- It is widely distributed in the body, attaining therapeutic concentration in vaginal secretion, semen, saliva and CSF.
- Metabolism occurs in liver primarily by oxidation and glucuronide conjugation followed by renal excretion.
- Plasma t1/2 is 8 hrs.

CONTRAINDICATIONS

In neurological disease, blood dyscrasias, first trimester of pregnancy (though no teratogenic effect has yet been demonstrated, its mutagenic potential warrants caution). Cautious use in chronic alcoholics.

INTERACTIONS

- A disulfiram-like intolerance to alcohol occurs in some patients taking metronidazole. Alcohol-metronidazole interaction occurs only in some individuals, while majority of those taking it can consume alcohol without any reaction. There is no convincing evidence of disulfiram-like action of metronidazole, but manufactures advise caution in drinking during metronidazole therapy.
  It passes off with time as alcohol is metabolized.
  Only reassurance and supportive treatment is needed.
- Enzyme inducers (phenobarbitone, rifampin) may reduce its therapeutic effect.
- Cimetidine can reduce metronidazole metabolism: its dose may need to be decreased.
- Metronidazole enhances warfarin action by inhibiting its metabolism.
  It can decrease renal elimination of lithium and precipitate toxicity.

7. A patient with AF was on Digoxin.He was started on KCl,Triamterene,Clarithromycin and Aspirin.Serum level of Digoxin is 2.5mg/dl and it is increased to 3.4mg/dl.Presented with flattening of T waves in ECG.Which of the following drug is responsible for this increase in serum concentration of digoxin ?

A. Triamterene
B. KCl
C. Aspirin
D. Clarithromycin

- Flattening of T waves in ECG is suggestive of Hypokalemia which is caused by Clarithromycin.
- All other drugs mentioned in option causes hyperkalemia

ABC TRANSPORTERS IN DRUG ABSORPTION AND ELIMINATION

- The systemic exposure to orally administered digoxin is decreased by coadministration of rifampin (an MDR1 inducer) and is negatively correlated with the MDR1 protein expression in the human intestine.
- MDR1 is also expressed on the brush border membrane of renal epithelia, and its function can be monitored using digoxin (> 70% excreted in the urine).
- MDR1 inhibitors (e.g., quinidine, verapamil, valspodar, spironolactone, clarithromycin, and ritonavir) all markedly reduce renal excretion of digoxin.

TRIAMTERENE

- Most important effect is to decrease K+ excretion(when it is high due to large K+ intake or use ofa diuretic that enhances K+ loss.)
- Is incompletely absorbed orally, partly bound to plasma proteins, largely metabolized in liver to an active metabolite and excreted in urine.
- Plasma t 1/2 : 4 hours
- Side effects :
  i. Nausea,
  ii.Dizziness, muscle cramps and rise in blood urea.
  iii.Impaired glucose tolerance and photosensitivity (but urate level is not increased.)

CLARITHROMYCIN

- More acid-stable than erythromycin.
- Is rapidly absorbed:
- Oral bioavailability is : 50% due to first pass metabolism; food delays but does not decrease absorption.
- Active metabolite is produced.
- No dose modification is needed in liver disease or in mild-to-moderate kidney failure.
- Inhibits CYP3A4
- First line drug in combination regimens for MAC infection in AIDS patients.
- Second line drug for other atypical mycobacterial diseases as well as leprosy.

8.A girl on sulfonamides developed abdominal pain and presented to emerge with seizures.
The probable cause is ?

A. Acute intermittent porphyria
B. Congenital erythropoietin porphyria
C. Infectious mononucleosis
D. Kawasaki disease

ACUTE INTERMITTENT PORPHYRIA

Acute intermittent porphyria is due to a deficiency of the enzyme porphobilinogen deaminase (also known as hydroxymethylbilane synthase) that leads to accumulation of the porphyrin precursors delta-aminolevulinic acid and porphobilinogen initially in the liver.

SYMPTOMS

AIP manifests after puberty, especially in women (due to hormonal influences). Symptoms usually come as discrete attacks that develop over two or more days. Abdominal pain, which is associated with nausea, can be severe and occurs in most cases.
Other symptoms may include:

- vomiting
- constipation
- pain in the back, arms and legs
- muscle weakness (due to effects on nerves supplying the muscles)
- urinary retention
- palpitations (due to a rapid heart rate and often accompanied by increased blood pressure)
- confusion, hallucinations, seizures, insomnia

Drugs causing acute intermittent porphyria

B	Barbiturates
O	OCPs
G	Griseofulvin
E	Estrogen
S	Sulfonamides
C	Chlordiazepoxide
P	Phenytoin
R	Rifampicin

DIAGNOSIS

Laboratory tests done on samples of urine taken during an attack show increased levels of two porphyrin precursors (delta-aminolevulinic acid and porphobilinogen). Levels of these precursors are very high during attacks and remain high in people who have repeated attacks.
The precursors can form porphyrins which turn the urine red to red-brown.

TREATMENT

- People with severe attacks are treated with heme given by vein. Blood and urine levels of delta-aminolevulinic acid and porphobilinogen are promptly lowered and symptoms subside, usually within several days. If treatment is delayed, recovery takes longer, and some nerve damage may be permanent.
- Dextrose given by mouth (or by vein if people are vomiting) can also be beneficial, particularly in people whose attacks are brought on by a low-calorie, low-carbohydrate diet, but these measures are less effective than heme.

Reference:Essentials of Medical Pharmacology Seventh Edition KD TRIPATHI, Pg 838

9. Being a medical practitioner,how would you correctly prescribe Alprazolam ?

A. Alprazolam 0.5mg one tab OD for 7 days

B. Alprazolam 1/2 mg one tab HS for 7 days

C. Alprazolam 0.5mg tab at bedtime for 7 days

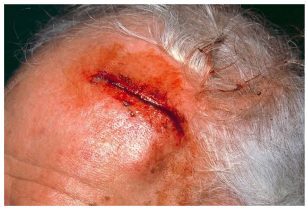
D. Alprazolam 500mcg one tab at bedtime for 7 days.

ALPRAZOLAM

- Primary indication of this potent and intermediate duration of action BZD is anxiety disorder, but it is also being employed as night-time hypnotic due to rapid oral absorption.
- Its active metabolite has a short half-life.
- Discontinuation after regular use has produced relatively marked withdrawal phenomena.
- Useful in anxiety a sociated with depression.
- Its plasma t1/2 is about 12 hours, but an active metabolite is produced.
- Withdrawal symptoms may be more marked on discontinuation than with other BZDs.
- For Panic disorder - Dose: 0.25-1.0 mg TDS; upto 6 mg/day.
- For Sleep disturbances (Insomina) - Alprazolam 1/2mg tab at HS for 7 days

1. Identify the injury :

A. Incised looking laceration
B. Incised wound
C. Lacerated looking incised wound
D. Laceration



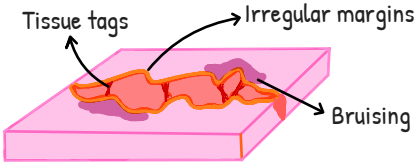
From the given image of the wound the margins appears to be sharp and there is linear splitting of tissue which is an Incised looking laceration.

LACERATIONS

- Also called tears or ruptures.
- Blunt force on areas where the skin is close to bone, and the subcutaneous tissues are scanty, may produce a wound which by linear splitting of the tissues (as the skin is easily stretched during impact), may look like incised wound.
- Lacerations are most common where the skin can be compressed between the applied force and underlying bone (i.e. over the scalp, face, elbows, knees, shins etc).
- If the force produces bleeding into adjacent tissues, the injury is a 'Contused-laceration' or 'bruised-tear'.
- If the margins are abraded, it is called "Abraded laceration" or "Scraped tear".
- If the blunt force produces extensive bruising and laceration of deeper tissues, it is called "crushing" injury

SOME OF THE CHARACTERS OF LACERATED WOUND

- Margins – irregular with pieces of tissues attached in between,called tissue tags or bridges
- Bruising – seen around the margin
- Deeper tissues – unevenly divided with tissue tags
- Hair bulbs – crushed



- Depth of the wound – varies with force, and contains abundant foreign matter such as dust particles, paint material of the vehicle involved, etc
- Laceration of viscera – heavy bleeding may be seen, which ultimately turn fatal.
- Skin laceration – usually one of the margins overhangs the other, which could help in assessing the direction of force
- On healing – it produces permanent scar.
- Tearing at the ends of lacerations, at angles diverging from the main laceration itself, so-called "SWALLOW TAILS", are frequently noted.
- Antemortem lacerations show bruising, eversion, gaping and blood-staining of margins, greater bleeding and vital reaction.

Reference :
Nagesh Kumar Rao forensic medicine 2nd Ed Pg : 226 Notespaedia FMT Image Bank Pg:59
K.S Narayan Reddy The Essentials 33rd Ed Pg : 189

TYPES

1) Split Lacerations :

- Splitting occurs by crushing of the skin between two hard objects.
- Scalp lacerations occur due to the tissues being crushed between skull and some hard object, such as the ground or a blunt instrument.
- Splits are not undermined, but show tissue bridges.

● Incised-like or Incised-looking Wounds :

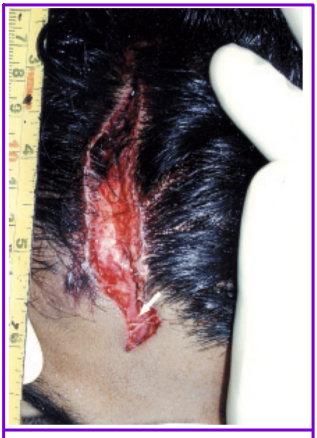
- Lacerations produced without excessive skin crushing may have relatively sharp margins.
- Blunt force on areas where the skin is close to bone, and the subcutaneous tissues are scanty, may produce a wound which by linear splitting of the tissues (as the skin is easily stretched during impact), may look like incised wound.

2) Stretch Lacerations :

- Overstretching of the skin, if it is fixed, will cause laceration.
- There is localised pressure with pull which increases until tearing occurs and produces a flap of skin, which is peeled off the underlying bone or deep fascia.

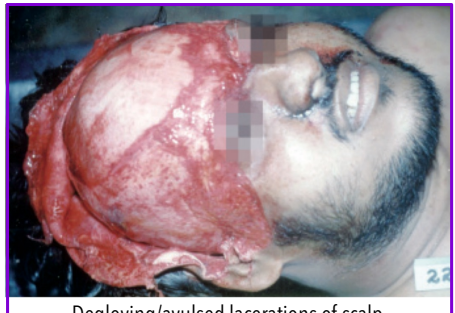
3) Avulsion (shearing laceration) :

- Is a laceration produced by sufficient force (shearing force) delivered at an acute angle to detach (tear off) a portion of a traumatised surface or viscus from its attachments.
- The shearing and grinding force by a weight, such as lorry wheel passing over a limb may produce separation of the skin from the underlying tissues (avulsion) over a relatively large area. This is called "FLAYING".



Incised like laceration on the forehead with contused margins and tissue tags/bridges (arrow), etc.

Avulsed lacerations of left heel and foot



Degloving/avulsed lacerations of scalp

INCISED WOUNDS

- An incised wound (cut, slice) is a clean cut through the tissues, (usually the skin and subcutaneous tissues, including blood vessels), caused by sharp-edged instrument.
- Incised wound is simple when it is superficial and bleeds minimum and heals completely in 15 days.
- The scar will be permanent.
- It is dangerous or fatal when deep and involves the viscera or major blood vessels.

CHARACTERISTIC FEATURES OF INCISED WOUND

- Shape : Spindle-shaped due to gaping by skin elasticity, zigzag where skin is lax, e.g. Axilla
- Edges : Clean, well defined and everted
- Bleeding: Minimum if smaller vessels are cut, more if bigger vessels are cut
- Length : It is greater than width/depth
- Width : It is greater than edge of weapon

GROSS AND MICROSCOPIC CHANGES IN ESTIMATING TIME SINCE INJURY

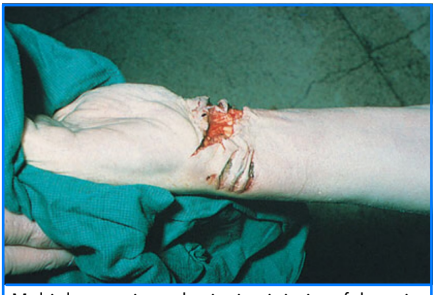
Gross Observation :

- Red and with clotted blood : Fresh wound
- Blood and lymph scab formed : 12 hours

Microscopic Observation

- Epidermal cells fill the gap : 24 hours
- Capillary network formed : 36 hours
- Growth of connective tissue fibroblasts : 2-3 days
- Parallel growth of vessels : 3-5 days
- Scar tissue formed : 7 days

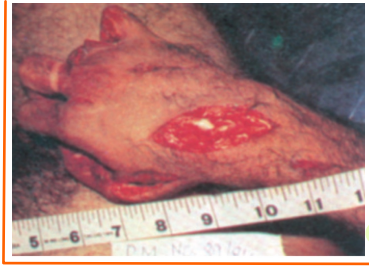
HESITATION CUTS/TENTATIVE CUTS



Multiple tentative or hesitation injuries of the wrist typical of self-infliction.

- Are parallel superficial cuts suggestive of suicidal motive, seen in the vital parts of the body such as neck, chest on left side (precordial area), wrists, etc. and are inflicted by the victim, prior to the final fatal deep cut or stab, e.g. suicidal cut- throat wound.

DEFENSE CUTS



- Are incised wounds suggestive of homicidal motive or assault with sharp weapon, seen on a victim's forearms and hands, while making either an attempt to grab the weapon by its blade resulting in active defense wounds or just taking the weapon on his parts in order to protect himself from the attack resulting in passive defense wound.
- Thus defense wounds are of two types: Active and Passive.

2.While recording evidence in a court of law,the lawyer asked the witness : " Did you see that Mr.'A' Killed Mr.'B'?".Witness answered : "Yes".This type of question is permitted in ?

A. Examination in Chief
B. Direct examination
C. Re-examination
D. Cross examination

- Here in this case lawyer asks a leading question to the witness which is permissible in cross examination.
- Leading quesitions are not permitted in Examination in Chief (a.k.a Direct examination) and Re-examination

LEADING QUESTION

- Is one which suggests to the witness the answer desired, or which includes a material fact, and admits of a conclusive answer by a simple "Yes" or "No".
- Eg :
  i. "Was this injury caused by a sharp weapon?" Ans: Yes or No
  ii. "Was the length of cut 3 cm?" Ans: Yes or No
  iii."Was it on the front of the abdomen?". Ans: Yes or No
  - They are all leading questions, as they suggest the answer "Yes" or "No".

CROSS-EXAMINATION

- In this, the witness is questioned by the lawyer for the opposite party, i.e., lawyer for the accused (defence lawyer) (S.137, I.E.A.).
- In a murder trial, the defence witness is cross- examined by public prosecutor.
- The main objects are :
  (1) to elicit facts favourable to his case,
  (2) to test the accuracy of the statements made by the witness (S.146, I.E.A.),
  (3) to modify, or explain what has been said,
  (4) to develop new or old facts,
  (5) to discredit the witness, and
  (6) to remove any undue or excessive emphasis which may have been given to any of them.
- The witness has to answer any question relevant to the matter in issue, even though the answer will expose or prove his guilt directly or indirectly, e.g., which would reveal that he has committed an illegal operation.
- If a witness is forced to give an answer admitting his guilt, he cannot be arrested or prosecuted for it, and also it is not taken as proof against him in any criminal proceeding.
- Leading questions are permissible during cross-examination (S.143, I.E.A.).

Reference :
K.S Narayan Reddy The Essentials 33rd Ed Pg : 13,14,15
Notespaedia FMT IB Pg : 14

EXAMINATION-IN-CHIEF

- This is the first examination of a witness. It consists of questions put to him by the lawyer (counsel or advocate) for the side which has summoned him.
- In Government prosecution cases. the public prosecutor first examine" the witness.
- If witness is called by private party. he is first examined by the lawyer of that party.
- The object is to elicit all relevant, convincing medical facts and the conclusions which the doctor has drawn from the facts.
- In this leading questions are not allowed, except in those cases, where the Judge is satisfied that a witness is hostile .

RE-EXAMINATION (RE-DIRECT EXAMINATION)

- This is conducted by the lawyer for the side which has called the witness.
- The object is to correct any mistake or to clarify or add details to the statements the witness has made in cross-examination.
- It is an opportunity for the witness to explain more fully some answer which might appear damaging to his direct evidence, because of skilful questioning or tactics by the cross-examiner.
- The witness should not bring in any new matter at this stage.
- The opposing lawyer has right of re-cross-examination on the new point raised.
- Leading questions are not allowed.

QUESTIONS BY JUDGE

- The Judge may ask any question, in any form, about any fact. relevant or irrelevant, at any stage of the examination to clear up doubts.
- The Court is also empowered to recall and re- examine any witness already examined if his evidence appears to the Court to be essential to the just decision of the Court

3.A 25year old female came to the ER and was given the history of Cocaine overdose. What is the least likely symptom seen in this person :

A. Agitation

B. Hyperthermia

C. Bradycardia

D. Myocardial Infarction

COCAINE

- Obtained from the leaves of Erythroxylum coca.
- The leaves contain about 0.5 to 1% cocaine.
- It is a colourless, odourless, crystalline substance with bitter taste.
- It contains alkaloids ECGONINE, HYGRINE and CINNAMYL COCCAINE.
- CRACK : Prepared by combining cocaine with baking soda and water, which is suitable for smoking.
- SPEEDBALL : A combination of cocaine and heroin taken by injection.
- The common names of substances used by addicts are: CRACK,PASTA,BAZOOKA and SPEED BALL.

Reference :
K.S Narayan Reddy The Essentials 33rd Ed Pg : 602

SIGNS AND SYMPTOMS

- Onset of Action :
  - i. Inhaled, the onset of action is within one to three minutes;
  - ii. i.v. or smoked it acts in seconds and peak action is in 3 to 5 minutes;
  - iii. Topically to the nasal mucosa, it peaks in 20 to 30 minutes;
  - iv. Orally it peaks within 60 to 90 minutes.

(1) Stage of Excitement :

- Dryness in the mouth,
- Dysphagia,
- Feeling of well-being and loss of depression and fatigue.
- The patient may be excited, restless and talkative, but this passes into a calm, dull condition.
- The pulse is rapid, respirations rapid and deep, pupils dilated, headache, pallor of the skin, cyanosis, sweating, and the temperature is raised.
- It produces hypertension which may lead to cerebral bleeding.
- The reflexes are exaggerated, and there may be tremors or convulsions.
- Occasionally, the patients may have hallucinations and become maniacal.
- There is often a feeling of tingling or numbness in the hands and feet, and a numb feeling at the place where the drug has touched, e.g. nose and back of throat, when it has been sniffed.

(2) Stage of Depression:

- Within an hour or even less, respirations become feeble, profuse perspiration, collapse, convulsions and death occurs.
- Death is due to respiratory failure, cardiac failure, or vascular collapse.
- Sudden death may occur following i.v. injection, and smoking than snorting, due to cardiac arrhythmias due to direct action on myocardium, and cardiopulmonary arrest.

- Fatal Dose: One gm. orally.
  - Procaine is about half as toxic as cocaine; butacaine is twice and dibucaine five to ten times.
- Fatal Period: Few minutes to few hours.

MAGNAN'S SYMPTOM OR COCAINE BUGS

- Is characteristic, in which there is a feeling as if grains of sand are lying under the skin or some small insects are creeping on the skin giving rise to itching sensation (formication, tactile hallucination) with resultant excoriation, leading to irregular scratches and ulcers

Plant with leaves and fruits (ripe)

Flower

Magnan symptom/ Cocaine bugs is seen in? [NEET'20]

4. The active principle of this poison is :

A. Abrin
B. Ricin
C. Crotin
D. Bhilawanol

- The given seed is black, heart-shaped with rough projection at the base which suggests it is a " MARKING NUT" (a.k.a SEMICARPUS ANACARDIUM)

SEMICARPUS ANACARDIUM

- Marking nuts (bhilawa) are balck, heart-shaped with rough projection at the base.
- They have a thick, cellular pericarp, which contains an irritant juice which is brownish, oily and acrid but turns black on exposure to air.
- The active principles are semecarpol (0.1%) and bhilawanol (15 to 17 %).

SIGNS AND SYMPTOMS

- Applied externally, the juice causes irritation and a painful blister which contains acrid serum, which produces eczematous eruptions of the neighbouring skin with which it comes into contact, and there is itching.
- The lesion resembles a bruise. Later an ulcer is produced, and there may be sloughing.
- Taken by mouth, the juice causes less irritant action.
- In large dose, it produces blisters on throat and severe gastrointestinal irritation, dyspnoea, tachycardia,hypotension,cyanosis,reflexes,delirium,coma and death.
- Fatal Dose : 5 to 10 g.
- Fatal Period : 12 to 24 hours.

ABRUS PRECATORIUS

- It is also known as jequirity,Indian liquorice, rosary bead, gunja or rati.
- The seeds contain an active principle ABRIN, a toxalbumen, which is similar to viperine snake venom; also present are abrine (N-methyltryptophan), an arninoacid, haemoglutinin in the cotyledons; a lipolytic enzyme, and abralin, a glucoside.
- All parts of the plant are poisonous.
- Seeds are tasteless and odourless.
- Abrin inhibits protein synthesis and causes cell death.

Reference :
K.S Narayan Reddy The Essentials 33rd Ed Pg : 556
Notespaedia FMT Image Bank Pg : 98

RICINUS COMMUNIS

- a.k.a The castor plant (arandi).
- Entire plant is poisonous, though seeds are most poisonous, containing toxalbumen RICIN, a water-soluble glycoprotein (highest level in the seeds), and a powerful allergen (CBA).
- The seeds are rich in a purgative oil, which is pale-yellow with a faint odour and acrid taste.
- Ricin blocks protein synthesis through the inhibition of RNA polymerase.
- Ricin is said to be 6000 times more powerful than cyanide.
- The unbroken seeds are non-poisonous when swallowed and also when cooked.

CROTON TIGLIUM

- The seeds of croton (jamalgota or naepala) contain CROTIN, a toxalbumen.
- Seeds are oval, dark- brown with longitudinal lines.
- They have no smell.
- Crotonoside, a glycoside, which is less poisonous is also present.

ERGOT

- Ergot is the dried sclerotinum of the fungus Claviceps purpurea, which grows on cereals like rye, barley, wheat, oats, etc.
- It gradually replaces the grain forming a curved, dark-purple or black compact mass.
- It contains about thirty alkaloids, but ergotoxine, erogotamine and erogometrine (ecbolics) are important.
- They directly stimulate muscle fibres.
- Ergot alkaloids exert their primary effect by stimulating adrenergic receptors, both peripherally and centrally.

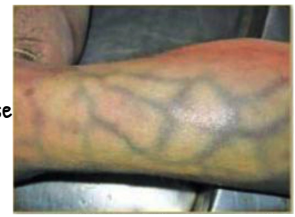
CALOTROPIS

- Calotropis gigantea (akdo, madar) has purple flowers and calotropis procera has white flowers.
- The active principles are uscharin, calotoxin, calactin and calotropin (glycoside).
- The milky juice in addition contains trypsin.
- The leaves and stem when incised yield thick acrid, milky juice.

Reference :
K.S Narayan Reddy The Essentials 33rd Ed Pg : 555,557

5. Which of the following is true regarding the changes given in the image below ?

A. Seen in 24 hours
B. Due to sulphhaemoglobin
C. Due to non bacterial cause
D. Due to Drowning



The first external sign of putrefaction in a body lying in air is usually a greenish discolouration of the skin over the region of the caecum

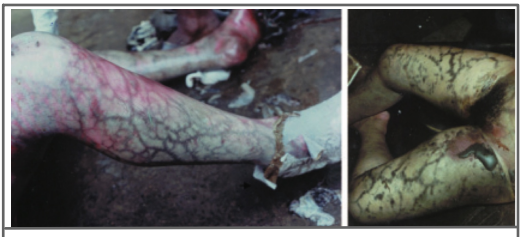
- The given image shows superficial veins stained purplish which is suggestive of MARBLING OF SKIN

MARBLING OF SKIN

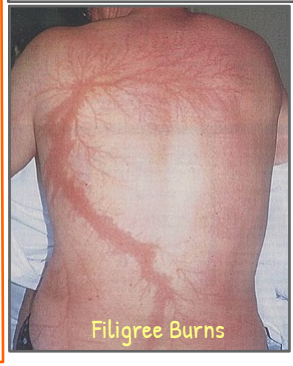
- The superficial veins especially over the roots of the limb, thighs, sides of the abdomen, shoulders, chest and neck are stained greenish-brown or purplish-red depending on the total amount of sulphhaemoglobin formation within the affected vessels (linear branching pattern) due to the haemolysis of red cells, which stains the wall of the vessel and infiltrates into the tissue, giving a marbled appearance (red, then greenish pattern in skin resembling the branches of a tree).
- This starts in 24 hours, but is prominent in 36 to 48 hours.
- Putrefactive effusion of foul-smelling blood stained fluid into the pleural cavities usually starts at about the time when the skin becomes macerated.
- Such effusions usually do not exceed 60 to 100 ml. unless death resulted from drowning, when several hundred ml. of drowning medium which oozed out through the lungs and visceral pleura, may be present in the thoracic cavities.
- The reddish-green colour of the skin may become dark-green or almost black in 3 to 4 days.

ARBORESCENT OR FILIGREE BURNS

- a.k.a Lichtenberg's flowers.
- They are superficial, thin, irregular and tortuous markings on the skin.
- This fern-like pattern of erythema in the skin is usually found over the shoulders or the flanks.
- It appears within few minutes to one hour of accident.
- Mechanism:
  - The mechanism by which this pattern occurs is not known.
  - It may be caused due to the slight staining of the tissues by haemoglobin from lysed red blood cells along the path of the electric current or rupture of smaller blood vessels at several places giving rise to ecchymoses.



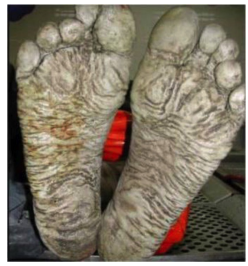
Marbling on left leg and thighs



Reference :
K.S Narayan Reddy The Essentials 33rd Ed Pg : 556
Notespaedia FMT Image Bank Pg : 38

6. The above phenomenon is due to

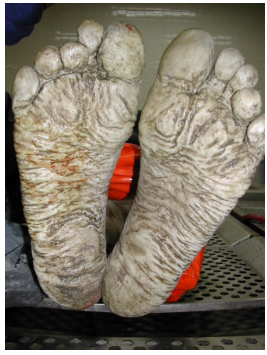
A. Case of torture in hot water
B. Case of Post mortem hanging
C. Case of immersion in water for 36 hours
D. Case of colliquative liquefaction

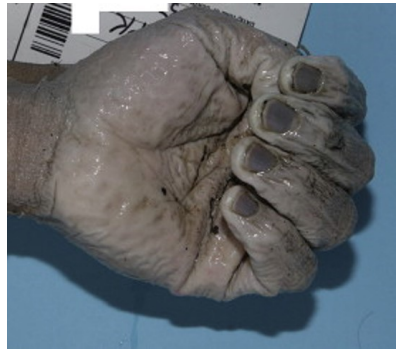


- The given image shows soddening of the skin occurs due to absorption of water into its outer layer, known as "WAHSERWOMAN'S FOOT".

WASHER WOMAN'S HANDS AND FEET

- Soddening of the skin occurs due to absorption of water into its outer layer.
- It is first seen on the fingertips in two to four hours and spreads to the palm and the backs of the fingers, and the back of the hand, in that order in about twentyfour hours.
- Similar progress and changes are seen in the skin of the foot, but when shoes are worn, it takes almost twice as long.
- Wrinkling of the skin begins to appear shortly after immersion, bleaching of epidermis in four to eight hours, and the bleached, wrinkled and sodden appearance is seen in twenty hours.
- The skin becomes sodden, thickened, wrinkled, and white in colour, known as "washerwoman's hands".





CUTIS ANSERINA

- Goose-skin or goose-flesh, in which the skin has granular and puckered appearance may be seen on the anterior surfaces of the body particularly on the thighs.
- It is produced by the spasm of the erector pilae muscles, attached to each hair follicle, and can occur in living when the skin comes in contact with cold water.
- It may occur on submersion of the body in cold water immediately after death, while the muscles were still warm and irritable.
- It is also produced by rigor mortis of the erector muscles.

Reference :
K.S Narayan Reddy The Essentials 33rd Ed Pg : 368
Notespaedia FMT Image Bank Pg : 98

Colliquative putrefaction :
Note the changes body has undergone and thick, semi-fluid black mass fallen at the feet of the victim and body is partially skeletonized

7.A married woman gives birth to twins.The husband doubts he is the father and gets a paternity test done.The test shows that he is the father of one infant but not the other.This is :

A. Superfoetation
B. Superfecundation
C. Papyraceous child
D. Suppositious child

SUPERFECUNDATION	SUPERFOETATION
• Fertilisation of two ova which have been discharged from the ovary at the same period,by two separate acts of coitus committed at short intervals. • Both ova do not always develop to maturity. • One foetus may be aborted early or die and retained until the labour that expels the other. • The dead foetus may be flattened by pressure and may not be recognisable, and is referred to as FOETUS COMPRESSUS or FOETUS PAPYRACEUS. • The spermatozoa causing fertilisation may be from different men.	• Fertilisation of a second ovum in a woman who is already pregnant. • Ovulation may occur during first trimester of pregnancy until decidua vera comes into apposition with decidua reflexa and decidual cavity gets obliterated. • Fertilisation of newly released ovum may occur following coitus. • Later, two foetuses are born either at the same time showing different stages of development, or two fully developed foetuses are born at different periods varying from one to three months.

SUPPOSITITIOUS CHILDREN

- It means fictitious children.
- A woman may pretend pregnancy and delivery and later produce a living child as her own, or she may substitute a male child for female child born of her, or for an abortion.
- This is done for obtaining money or for the purpose of claiming property.

PAPYRACEUS CHILD

- When intrauterine fetal demise of a twin occurs early in pregnancy
- Retention of the foetus for a minimum of 10 weeks
- This results in compression of small foetus
- Loss of fluid
- Resembles PARCHMENT PAPER

8. A person was sitting naked in the balcony facing a crowded park getting sexual gratification.This paraphilia is known as ?

A. Voyeurism
B. Exhibitionism
C. Masochism
D. Fetichism

EXHIBITIONISM

- An exhibitionist is one who, over a period of six months or more, experiences recurrent, intense, sexually arousing fantasies, sexual urges or behaviour, including the exposure of one's genitals to an unsuspecting stranger.
- It usually occurs before adulthood.
- It is done mostly by males, often to children or the persons of the opposite sex.
- Majority of them are psychopathic or suffer from compulsion neurosis and suffer from alcoholism, epilepsy, senile dementia, GPI, etc.
- It is an obscene act punishable with imprisonment up to three months or fine.

VOYEURISM

- a.k.a SCOPTOPHILIA
- Voyeur (the socalled Peeping Tom) is defined as one who, experiences recurrent, intense, sexually arousing fantasies, sexual urges or behaviours involving the act of observing an unsuspecting person who is naked, in the process of disrobing, or engaged in sexual activity.
- Masturbation at the scene or later to the memories of watching the unsuspecting stranger is normally the source of sexual pleasure.

FETICHISM

- A fetish is an abnormal stimulus or object of sexual desire.
- Fetichism means the use of such objects for sexual gratification.
- In this, recurrent, intense sexual fantasies, sexual urges or behaviour involving the use of living or non-living objects occur.
- In this, the person experiences sexual excitement leading to orgasm from part of the body of a woman or some article belonging to her that normally has no sexual influence on the mind, e.g., underclothing. brassiere, petticoat, stocking, shoes, etc. which act as substitute for the female love object.

MASOCHISM

- a.k.a PASSIVE ALGOLAGNIA.
- In masochism, sexual gratification is obtained or increased by the suffering of pain.
- Masochists get pleasure from being beaten, abused, tortured. humiliated, enslaved, degraded or dominated by their sexual partner, and they tend to place themselves repeatedly in self-defeating situations.
- ALGOLAGNIA : includes both sadism and masochism.

9. A child consumed some unknown fruit.After some time,he started symptoms of irritability, restlessness,confusion,inability to pass urine,hot skin and photophobia.Select the poison and antidote ?

A. Datura and Physostigmine
B. Yellow oleander and Physostigmine
C. Datura and Digibond
D. Yellow Oleander and Digibond

- From the given history of consumption of unknown fruit and symptoms occuring following consumption is suggestive of DATURA POISONING,whereas in YELLOW OLEANDER POISONING symptoms produced are slight numbing sensation and feeling of heat in the mouth and purging. Ingestion causes burning pain in the mouth, dryness of throat, tingling and numbness of tongue, vomiting, diarrhoea, headache, giddiness, dilated pupils, loss of muscular power and fainting. Pulse is rapid, weak and irregular, blood pressure low.

DATURA POISONING

- Two varieties of this plant exist :
  (1) Datura alba, a white flowered plant, and
  (2) Datura niger, a deep- purple flowered plant.
- The flowers are bell-shaped.
- All parts of these plants including nectar (honey) are poisonous, especially the seeds and the fruit.
- They contain 0.2 to 1.4% of hyoscine (scopolamine), hyoscyamine, and traces of atropine.

SIGNS AND SYMPTOMS

- Contact with leaves or flowers causes dermatitis in sensitive persons.
- A bitter taste, dryness of mouth and throat, with difficulty in talking, dysphagia, burning pain in the stomach and vomiting are first noticed.
- The face becomes flushed, conjunctivae congested, pupils widely dilated with loss of accommodation for near vision, developing in temporary blindness, photophobia and diplopia.
- Light reflex at first is sluggish and later absent.
- The pollen can cause unilateral mydriasis (cornpicker' s pupil).
- Mental changes include restlessness and agitation and patient cannot recognise relatives or friends.
- Urinary retention and inability to pass urine occurs.
- The patient becomes confused, giddy, staggers as if drunk.
- The skin is dry and hot, the pulse rapid 120 to 140 per minute, full and bounding, but later becomes weak and irregular, and the respirations are increased.
- Muscle tone and deep reflexes are increased, and there may be muscular spasm or convulsions.
- 8 D's of Datura Posionong: Dryness of mouth, dysphagia, dilated pupils, dry; hot skin, drunken gait, delirium, drowsiness, death due to respiratory failure.

TREATMENT

(1) Emetics can be used.
(2) Wash-out the stomach repeatedly with a weak solution of tannic acid.
(3) Give activated charcoal and a cathartic.
(4) Wash-out the lower bowel frequently.
(5) PHYSOSTIGMINE
(6) PILOCARPINE NITRATE
(7) Morphine is to be avoided because of the danger of depressing the respiratory centre.
(8) Symptomatic.

CERBERA THEVETIA POISONING

- a.k.a YELLOW OLEANDER; pila kaner
- All parts of cerbera thevetia are poisonous.
- All active principles are glycosides.
- Milky juice exudes from all parts of the plant.
- The seeds contain four percent of the cardiac glycoside :
  i.THEVETIN (similar to digitalis in action);
  ii.THEVETOXIN is similar to but less toxic than thevetin;
  iii.NERIFOLIN (more potent than thevetin); peruvoside, and ruvoside, cerberin and also a bitter principle that acts on the CNS, and produces tetanoid convulsions.

TREATMENT

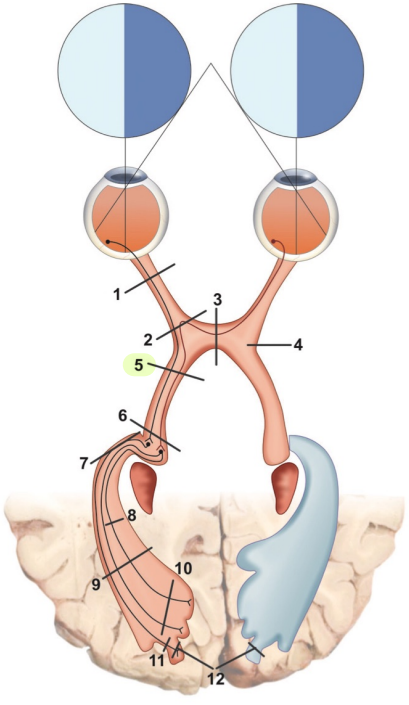
(1) Wash out the stomach.
(2) Sodium molar lactate transfusion with glucose ,Atropine,Adrenaline and Noradrenaline is beneficial.
(3) Symptomatic.

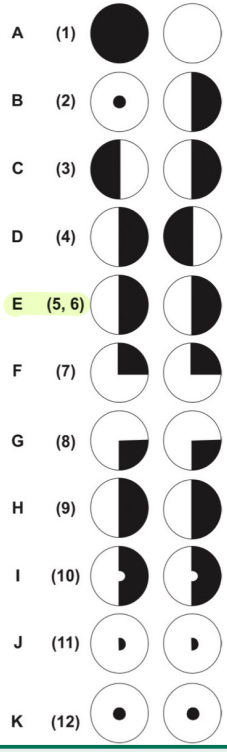
Ophthalmology

1. 33yr female with complaints of diminishing vision on right halves of both eyes. What is the Probably diagnosis ?

A.Left optic tract
B.Right occipital lobe
C.Optic chiasma
D.Right Optic Nerve

LESIONS OF VISUAL PATHWAY





AT THE LEVEL OF

1. optic nerve;
2. proximal part of optic nerve;
3. central chiasma;
4. lateral chiasma (both sides);
5. optic tract;
6. geniculate body;
7. part of optic radiations in temporal lobe;
8. part of optic radiations in parietal lobe;
9. optic radiations;
10. visual cortex sparing the macula;
11. visual cortex, only macula;
12. bilateral visual cortex only macula

Reference; Comprehensive Ophthalmology, AK Khurana (Pg 310)

Reference; Notespaedia ophthalmology image bank pg 109

Site of lesion	Salient features	Common causes
• Optic nerve	– Ipsilateral blindness, – Absent ipsilateral direct light reflex,	– Optic atrophy, – Traumatic avulsion of optic nerve
	– Absent contra-lateral consensual light reflex	– Acute optic neuritis
• Saggital (central) lesions of the chiasma	– Bitemporal hemianopia	– Supra-sellar aneurysms, – Pituitary tumours, – Craniophary-ngioma, – Glioma of third ventricle
• Lateral chiasmal lesions	– Binasal hemianopia	– Distention of third ventricle, – Atheroma of posterior communicating arteries
• Optic tract	– Homonymous heminanopia – Wernick's hemianopic pupillary response	– Syphlitic meningitis – Tuberculosis and tumours of optic thalamus – Aneurysms of superior cerebellar or posterior cerebral arteries
• Lateral geniculate body	– Homonymous hemianopia with sparing of pupillary reflexes	– Syphlitic meningitis – Tuberculosis and tumours of optic thalamus
• Optic radiations – Total fibres – Lower fibres only – Upper fibres only	– Homonymous hemianopia (sometimes sparing the macula) – Homonymous upper – quadrantinopia (pie-in the sky) – Homonymous lower quadrantinopia (Pie-in the floor)	– Vascular occlusion – Primary and secondary tumours, and – Trauma – Temporal lobe lesions – Anterior parietal lobe lesion
Visual cortex	– Homonymous hemianopia (usually sparing the macula)	– Vascular occlusion – Primary and secondary tumour – Trauma

2. Transparency of corneal endothelium is maintained by

A. Heparan Sulphate

B. Chondroitin Sulphate

C. Keratin Sulphate

D. Hyaluronic Acid

- Glycosaminoglycans (GAGs) previously called as mucopolysaccharides, mucous proteins, or proteoglycan complexes are the most abundant heteropolysaccharides present in the human cornea and they help to maintain the structural integrity of the corneal extracellular matrix.
- In cornea, there are two main types of GAGs: chondroitin sulphate/dermatan sulphate (CS/DS) and keratan sulphate (KS), the latter being predominantly found in the adult human corneal stroma
- Keratan sulfate (KS), also called keratosulfate, is any of several sulfated glycosaminoglycans (structural carbohydrates) that have been found especially in the cornea, cartilage, and bone.
  - The amount of KS found in the cornea is 10 fold higher than it is in cartilage and 2-4 times higher than it is in other tissues.
  - It is produced by corneal keratocytes and is thought to play a role of a dynamic buffer of corneal hydration.
  - In a rare progressive disorder called macular corneal dystrophy (MCDC), the synthesis of keratan sulfate is either absent (MCDC type I) or abnormal (MCDC type II)

CORNEAL TRANSPARENCY

ANATOMICAL FACTORS

- Corneal epithelium and tear film. Homogeneity of the refractive index throughout the epithelitun.
- Peculiar arrangement of corneal lamellae (lattice theory of Maurice).
- Peculiar refractive index of corneal lamellae with variation less Lhan 200 μm (Goldmann and Benedek theory).
- Avascularity of cornea.

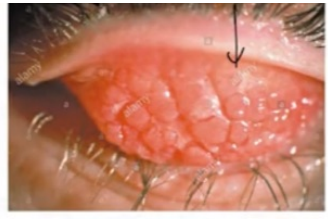
PHYSIOLOGICAL FACTORS

Keep the cornea in a relative state of dehydration (78% water content). These factors are:

- Barrier function of limiting layers (epithelium and endothelium).
- Endothelial pumps. Corneal endothelium plays a predominant role in controlling fluid transport due to several enzyme systems: Na/K• ATPase pump, Bicarbonate-dependent ATPase, Na• /H' pump, and is thus the most important factor in maintaining corneal dehydration.
- Evaporation from corneal surface.
- Normal intraocular pressure (IOP).
- Swelling pressure (SP) of the stroma which counrers the imbibition effect of intraocular pressure (IOP).
- Corneal crystallins, i.e. waler soluble proteins of keratocytes (transketolase and aldehyde dehydrogenase class lAl) also connibute to corneal transparency at the cellular level.

3.A female comes with H/O contact lens use comes with following. What is the diagnosis?

A .Trachoma
B.GPC
C.Spring Cataract
D.Acute follicular conjunctivitis



GIANT PAPILLARY CONJUNCTIVITIS (GPC)

- GPC is the inflammation of conjunctiva with formation of very large sized papillae greater than 1 mm in size (currently defined as papillae greater than 0.3 mm in diameter).
- Etiopathogenesis GPC, also known as mechanically-induced papillary conjunctivitis, is a localised allergic response to a physically rough or deposited surface (contact lens, prosthesis, exposed nylon sutures and scleral buckle).
- Exact pathogenesis of GPC is not clear, but is most frequently attributed to the combined effect of mechanical trauma and the subsequent immune response to antigens in the form of surface deposits or envirorunemal factors.

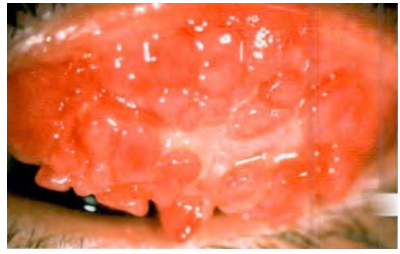
CLINICAL FEATURES

- Symptoms

mild irritation and itching, stringy discharge and reduced wearing time of contact lens or prosthetic shell.

- Signs.

Papillary hypertrophy (often ranging between 0.6 mm to 1.75 mm in diameter) of the upper tarsal conjunctiva, similar to that seen in palpebral form of VKC with hyperaemia are the main sign

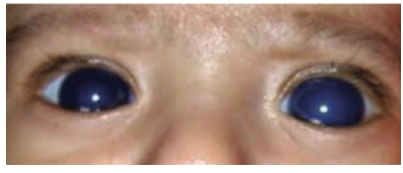


TREATMENT

1. Offending cause should be removed. After discontinuation of contact lens or artificial eye or removal of nylon sutures, the papillae resolve over a period of one month.
2. Mast cell stabilizer such as sodium cromoglycate and nedocromil are known to relieve the symptoms and enhance the rate of resolution.
3. Combined antihistamines and mast cell stabilizers like azelastine and olopatadine are very effective.
4. Steroids may be required in resistant cases.

4. A one month baby comes with watering and photophobia,diagnosis is :

A.Congenital Glaucoma
B.Hurler syndrome
C.Opthalmia neonatorum
D.Galactosemia



The image shows congenital glaucoma.

CONGENITAL/ DEVELOPMENTAL GLAUCOMAS

PRIMARY CONGENITAL GLAUCOMA (PCG) refers to abnormally high IOP which results due to developmental anomaly of the angle of the anterior chamber, not associated with any other ocular or systemic anomaly.

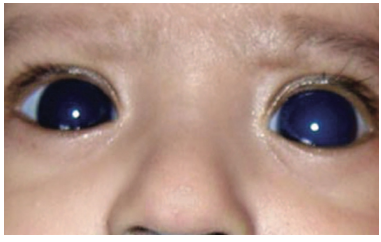
1. Newborn glaucoma, True congenital glaucoma, is labelled when IOP is raised during intrauterine life and child is born with ocular enlargement.
2. Infantile glaucoma is labelled when the disease manifests prior to the child's third birthday.
3. Juvenile glaucoma is labelled in who develop pressure rise after 3 years but before adulthood. Juvenile POAG have a genetic anomaly on the long arm of chromosome 21

Reference; Notespaedia ophthalmology image bank pg 81
Reference; Comprehensive Ophthalmology, AK Khurana (Pg 219)

CLINICAL FEATURES

1. Lacrimation, photoplzobia and blepharospasm often occur together and form the classic triad of symptoms of congenital glaucoma.
Often there is history of eye rubbing. These are thought to be caused by irritation of corneal nerves, which occurs as a result of the elevated IOP.
2. Corneal signs. Corneal signs include its oedema, enlargement and Descemet's breaks.
- Corneal oedema. It is frequently the first sign which arouses suspicion and is often associated with marked photophobia and lacrimation.
- Corneal enlargement. It occurs along with enlargement of globe-buphthalmos , especially when the onset is before the age of 3 years.
- Haah's striae Tears and breaks in Descemet's membrane occur because Descemet's membrane is less elastic than the corneal stroma. Tears are usually peripheral, concentric with the limbus and appear as opaque lines with double contour.
- Hazy cornea with frosted glass appearance may results over the time.

3. Sclera Becomes thin and appears blue due to underlying uveal tissue.
4. Anterior chamber becomes deep.
5. Iris may show iridodonesis and atrophic patches in late stage.
6. Lens becomes antero-posteriorly flat due to stretching of zonules and may even subluxate backward.
7. Optic disc may show variable cupping and atrophy especially after third year.
8. IOP is raised which is neither marked nor acute.
9. Axial myopia may occur because of increase in axial length which may give rise to anisometropic amblyopia.
10. Large eyeball (buphthalmos) is seen when developmental glaucoma occurs early (before 3 years of age).



• **BUPHTHALMOS**
(occurs with onset before the age of 3 years)
Enlarged eyeball, corneal diameter more than 13 mm, corneal oedema (first sign), Haab's striae (healed splits in Descemet's membrane), deep anterior chamber, raised IOP, and variable optic disc cupping. Eye becomes myopic.

TREATMENT

- IOP must be lowered by medical treatment with hyperosmolic agents, carbonic anhydrase inhibitors abs B blockers till surgery
- Incision angle surgery
- Filteration surgery
- Glaucoma drainage devices (GDD)

5. A 15 yr old girl with myopic astigmatism shows non acceptance to spectacle . what can be prescribed :

A.LASIK
B.FEMTO LASIK
C.ICI
D.Spherical alternative correction

LASIK and FEMTO LASIK can be done only in 18 yrs and above.

Intra corneal lens implantation is preferred in this case.

ASTIGMATISM

Refraction varies in different meridia of the eye

Regular astigmatism - refractive power changes uniformly from one meridian to another

Depending upon the axis and the angle between the two principal meridia

- With-the-rule astigmatism

  The vertical meridian is more curved than the horizontal
  The two principal meridia are at right angle to each other.

- Against-the-rule astigmatism

  Reverse of with-the-rule astigmatism,
  The horizontal meridian is more curved than the vertical.

- Oblique astigmatism

  Two principal meridia are at right angle to each other
  but these are not the horizontal and vertical

- Bioblique astigmatism

  Two principal meridia are not at right angle to each other

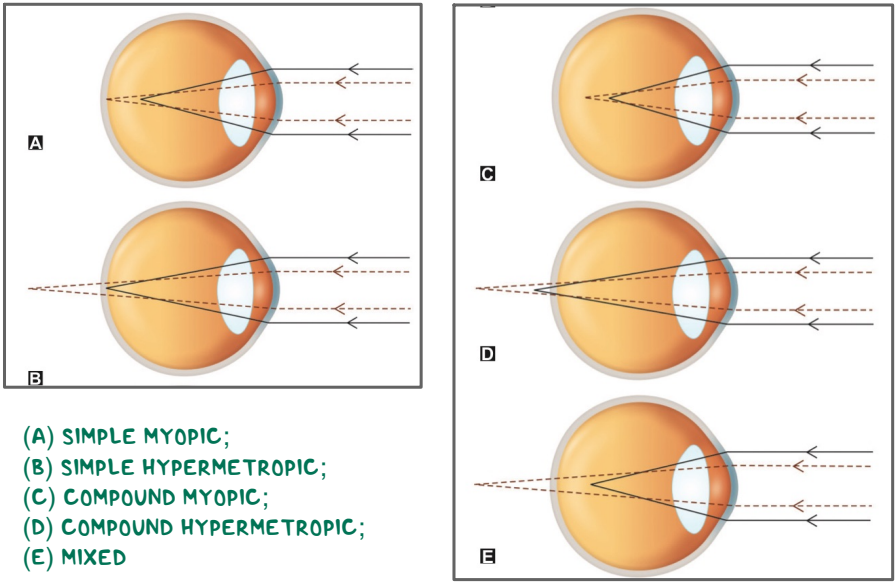
IRREGULAR ASTIGMATISM

1. Curvatural irregular astigmatism
   - Found in patients with extensive corneal scars or keratoconus.
2. Index irregular astigmatism
   - due to variable refractive index in different parts of the crystalline lens.

FEMTO-LASIK

Also known as 'ALL laser LASIK' or 'No blade LASIK' or iLASIK refers to the technique in which the corneal flap is made with the help of femtosecond laser (rather than the micro keratome) for greater precision and consistency.

REFRACTIVE TYPES OF REGULAR ASTIGMATISM



(A) SIMPLE MYOPIC;
(B) SIMPLE HYPERMETROPIC;
(C) COMPOUND MYOPIC;
(D) COMPOUND HYPERMETROPIC;
(E) MIXED

Surgery	Range of Myopia	Possible Complications
Methods to induce flattening of the central cornea		
Radial keratotomy*	1 to 6 D	Perforation, infection, surgically induced astigmatism, corneal scarring, late rupture with trivial trauma
Intracorneal rings or segments (ICR or INTACS)†	1 to 6 D	Unpredictable results, keratitis
Excimer laser photorefractive keratotomy (PRK)‡	1 to 4 D	Delayed visual recovery, faint corneal haze, glare and loss of contrast sensitivity
Excimer laser-assisted in situ keratomileusis (LASIK)§	1 to 10 D	Dry eye, infection, diffuse lamellar keratitis, interface debris and epithelial ingrowth, flap displacement, surgically induced astigmatism or aberrations, regression, iatrogenic thinning and ectasia, glare, difficulty with night driving and retinal detachment
Laser assisted epithelial keratomileusis (LASEK)††	1 to 6 D	Same as LASIK except there are no microkeratome-related complications
Femtosecond laser-assisted LASIK‡‡	1 to 10 D	Diffuse lamellar keratitis, incomplete flap and interstitial corneal stromal haze are still potential problems
Methods to reduce the overall refractive power of the eye		
Clear lens extraction	> 15 D	Retinal detachment, endophthalmitis
Phakic intraocular lens‖	5 to 20 D	Endophthalmitis, cataract

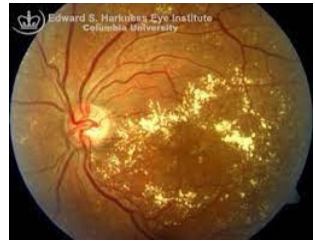
6. An elderly female with gradual painless DOV fundus findings image :

A.Hard Exudates in DR

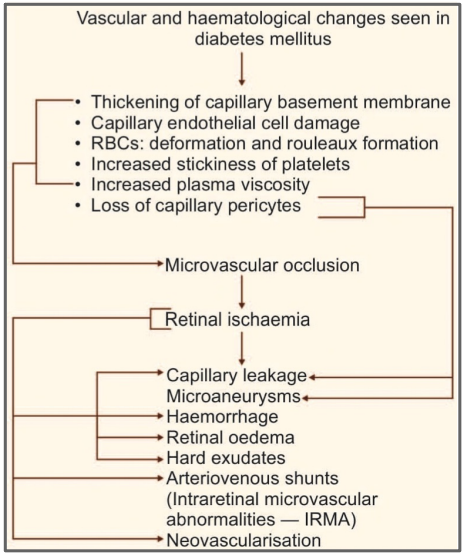
B.Flame haemorrhages in HTN

C.Soft exudates in HTN

D.CRVO



DIABETIC RETINOPATHY

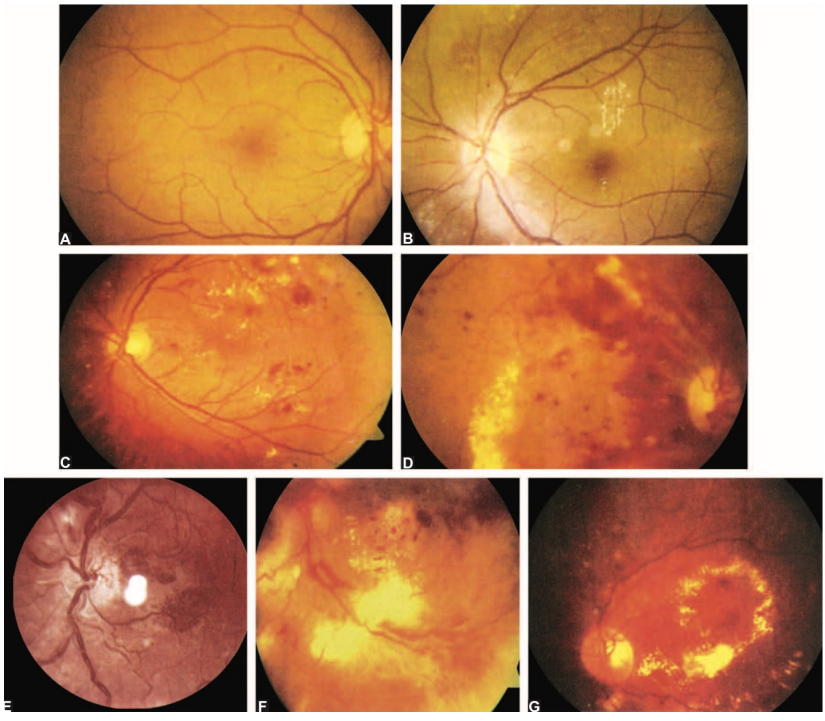


Pathogenesis of diabetic retinopathy

• After 10 years, 20% of type I and 25% of type II diabetics develop retinopathy.
• After 20 years, 90% of type I and 60% of type II diabetics develop retinopathy.
• After 30 years, 95% of both type I and type II diabetics develop retinopathy.

CLASSIFICATION

1. Mild NPDR
• At least one microaneurysm must be present.
2. Moderate NPDR
• Microaneurysms/intraretinal haemorrhage in 2 or 3 quadrants.
• Early mild IRMA.
• Hard/soft exudates may or may not present.
3. Severe NPDR. Any one of the following (4-2-1 Rule)
• Four quadrants of microaneurysms and extensive intraretinal haemorrhages. • Two quadrants of venous beading.
• One quadrant of IRMA changes.
4. Very severe NPDR. Any two of the following(4-2-1 Rule)
• Four quadrants of microaneurysms and extensive intraretinal haemorrhages.
• Two quadrants of venous beading.
• One quadrant of IRMA changes.



A, mild NPDR; B, moderate NPDR; C, severe NPDR; D, very severe NPDR; E, early PDR; F, highrisk PDR; G, focal exudative diabetic maculopathy

NON-PROLIFERATIVE DIABETIC RETINOPATHY (NPDR)

- Microaneurysms are seen in the macular area (the earliest detectable lesion) and elsewhere in relation to area of capillary nonperf usion.
- Retinal haemorrhages. Both deep (dot and blot haemorrhages which are more common) and superficial haemorrhages (flame-shaped), occur from capillary leakage.
- Retinal oedema characterized hy retinal thickening is caused by capillary leakage.
- Hard exudates-yellowish-white waxy-looking patches are arranged in clumps or in circinate pattern. These are commonly seen in the macular area. These occur due to chronic localised oedema and are composed of leaked lipoproteins and lipid filled macrophages.
- Cotton-woof spots, are small whitish fluffy superficial lesions. These represent areas of nerve fibre infarcts.
- Venous abnormalities (beading, looping and dilatation) occur adjacent to area of capillary non-perfusion.
- Intraretinal microvascular abnormalities (IRMA) seen as fine irregular red lines connecting arterioles with venules, represent arteriovenular shunts.
- Dark-blot haemorrhages representing haemorrhagic retinal infarcts.

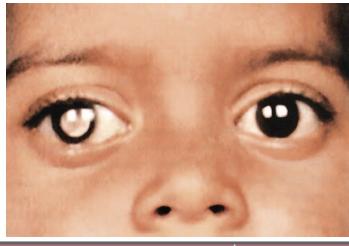
7.A child with whitish pupillary reflex has undergone enucleation and shows Flexner Winter Steiner Rosette.What is your diagnosis ?

A.Retinoblastoma
B.Rhabdomyosarcoma
C.Medulloblastoma
D.Astrocytoma

Flexner-Wintersteiner rosettes are found in retinoblastoma.

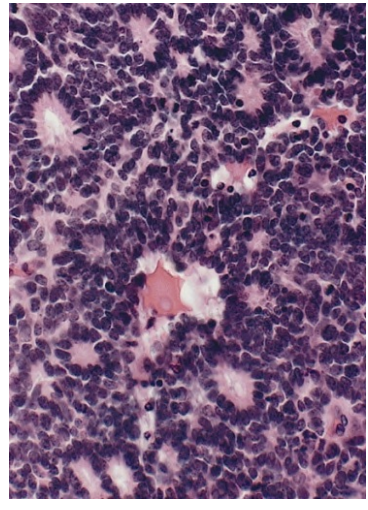
RETINOBLASTOMA

- Common malignant tumour arising from the neurosensory retina in one or both eyes.
- Retinoblastoma (RB) gene has been identified as 14 band on the long arm of chromosome 13 (13q 14) and is a 'cancer suppressor' or 'antioncogenic' gene.
- Deletion or inactivation of RB gene by two mutations (Knudson's two-hit hypothesis) results in occurrence of retinoblastoma.



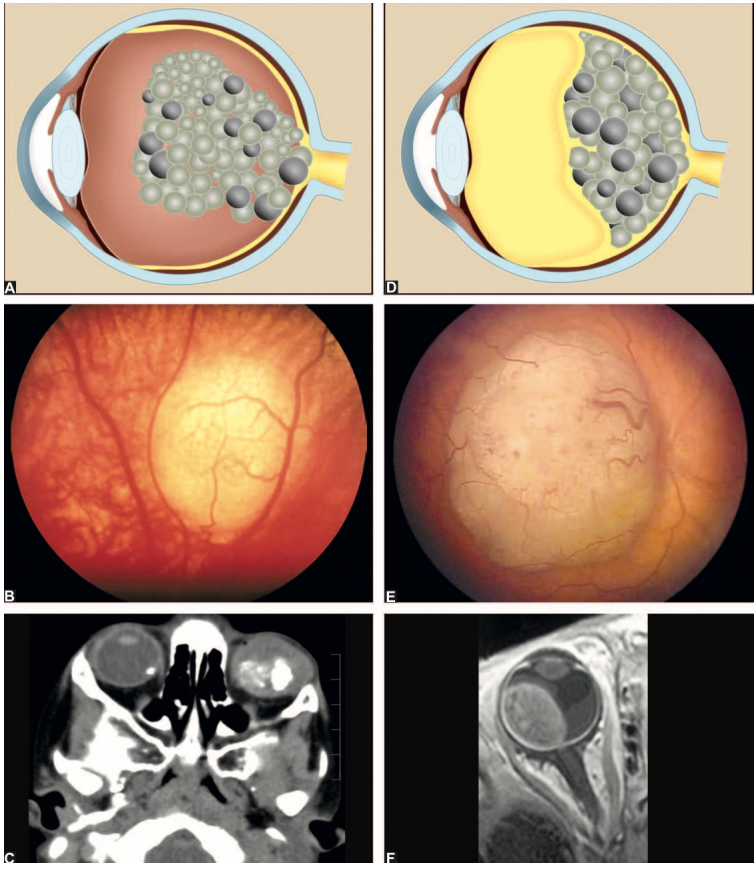
Leukocoria or yellowish-white pupillary reflex (amaurotic cat's eye appearance) is the commonest presenting feature

Presenting features	Percentages
Leucocoria	60%
Strabismus	20%
Painful red eye	07%
Poor vision	05%
Asymptomatic	03%
Orbital cellulitis	03%
Unilateral mydriasis	02%
Heterochormia iridis	01%
Hyphema	01%



Flexner-Wintersteiner rosettes in Retinoblastoma.

- Flexner-Wintersteiner rosettes are found in retinoblastoma. They are composed of tumor cells surrounding a lumen of cytoplasmic extensions.
- The presence of Flexner-Wintersteiner rosette is characteristic for retinoblastoma but is also seen in pineoblastoma and medulloepitheliomas.



Endophytic retinoblastoma Grows inwards and is white or pearly pink in colour. Fine blood vessels may be present on its surface. In the presence of calcification, it gives the typical 'cottage-cheese' appearance.

Exophytic retinoblastoma Grows outwards and causes exudative retinal detachment.

8. DOV in dim light along with dry eyes and roughening of corneal surface.Which deficiency can be associated ?

A.Iron
B.Protein
C.Niacin
D.Retinoic acid

Retinoic acid is a metabolite of vitamin A_1 that mediates the functions of vitamin A_1 required for growth and development.

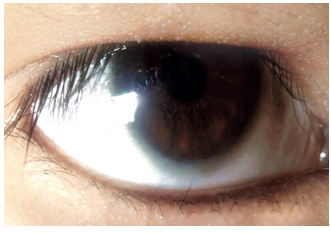
XEROPHTHALMIA

Ocular manifestations of vitamin A deficiency

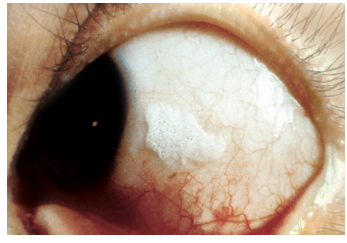
Due to dietary deficiency of vitamin A or its defective absorption from the gut.

WHO CLASSIFICATION (1982)

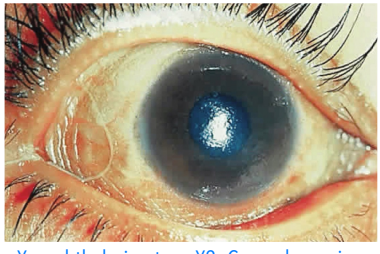
XN.	NIGHT BLINDNESS
X1A	CONJUNCTIVAL XEROSIS
X1B	BITOT'S SPOTS
X2	CORNEAL XEROSIS
X3A	CORNEAL ULCERATION/KERATOMALACIA AFFECTING LESS THAN ONE-THIRD CORNEAL SURFACE
X3B	CORNEAL ULCERATION/KERATOMALACIA AFFECTING MORE THAN ONE-THIRD CORNEAL SURFACE
XS	CORNEAL SCAR DUE TO XEROPHTHALMIA
XF	XEROPHTHALMIC FUNDUS.



Xerophthalmia, stage X1A: Conjunctival xerosis

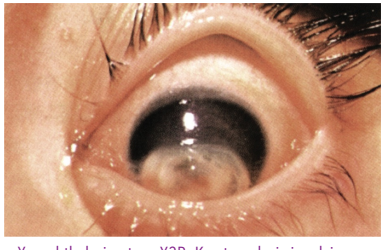


Xerophthalmia, stage X1B: Bitot spots

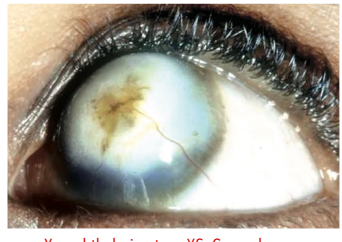


Xerophthalmia, stage X2: Corneal xerosis

Xerophthalmia, stage X3A: Keratomalacia involving less than one-third of corneal surface



Xerophthalmia, stage X3B: Keratomalacia involving more than one-third of corneal surface



Xerophthalmia, stage XS: Corneal scars

XN: Night blindness

It is the earliest symptom of xerophthalmia in children.

X1 A: Conjunctival xerosis.

It consists of one or more patches of dry, lustreless, non wettable conjunctiva. These patches almost always involve the interpalpebral area of the temporal quadrants and often the nasal quadrants as well.

XIB: Bitot's spots.

The Bitot's spot is a raised, silvery white, foamy, triangular patch ofkeratinised epithelium, situated on the bulbar conjunctiva in the interpalpebral area .

It is usually bilateral and temporal, and less frequently nasal.

X2: Corneal xerosis.

The earliest change in the cornea is punctuate keratopathy which begins in the lower nasal quadrant, followed by haziness and/or granular pebbly dryness

X3A aml X3B: Corneal ulceration/keratomalacia.

Stromal defects occur in the lace stage due to colliquative necrosis and take several forms. Small ulcers (1-3 mm) occur peripherally; they are characteristically circular, with steep margins and are sharply demarcated. Large ulcers and areas of necrosis may extend centrally or involve the entire cornea.

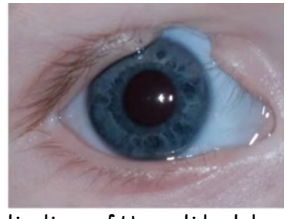
XS: Cornealscars.

Healing of stromal defects results in corneal scars of different densities and sizes which may or may nor cover the pupillary area.

XFC: xerophtlwlmic fundus

It is characterized by typical seed-like, raised, whitish lesions scattered uniformly over the pan of the fundus at the level of optic disc

9. Upper lid coloboma most likely complication ?



A.Cataract

B.Exposure keratitis and 2° bacterial infections

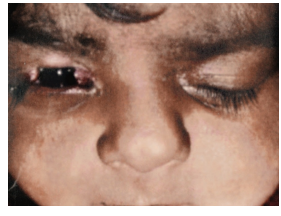
C.Difficulty in eye movement

D.Glaucoma

Exposure keratitis and 2° bacterial infections are the complications of Upper lid coloboma

CONGENITAL COLOBOMA UPPER EYELID

- Rare condition characterised by a full thickness triangular gap in the tissues of the lids .
- The anomaly usually occurs near the nasal side and involves the upper lid more frequently than the lower lid.
- Treatment consists of plastic repair of the defect.



Reference; Notespaedia ophthalmology image bank pg 124

Reference; Comprehensive Ophthalmology, AK Khurana (Pg 362)

COMPLICATIONS

- Dryness and thickening of conjunctiva and corneal ulceration (exposure keratitis) may occur due to prolonged exposure.
- Eczema and dermatitis of the lower lid skin may occur due to prolonged epiphora.

MEASURES TO PREVENT EXPOSURE KERATITIS

- Artificial tear drops should be instilled frequently and the open palpebral fissure should be filled with an antibiotic eye ointment during sleep and in comatosed patients.
- Soft bandage contact lens may be used to prevent exposure keratitis.
- Tarsorrhaphy may be performed to cover the exposed cornea when indicated.

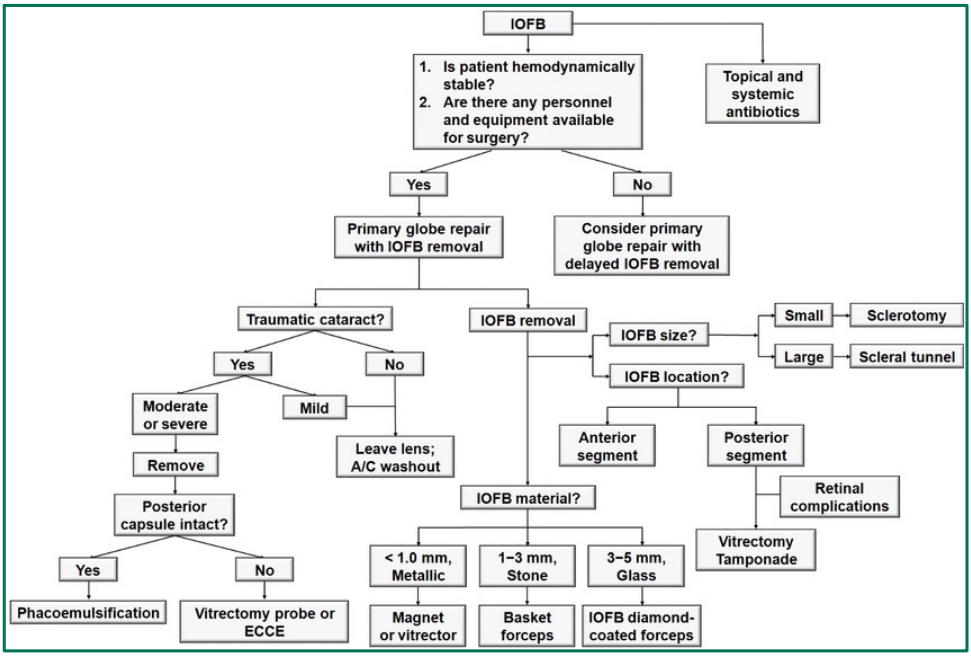
10. A worker who was working with hammer and chisel while he was trying to break a hard stone admitted to hospital after injury to eye. Which among the following investigations done would be detrimental?

A. CT scan
B. MRI
C. X-ray
D. B mode scan

MRI is the worst choice of investigation as the foreign body involved is Magnetic (Iron)

INTRAOCULAR FOREIGN BODY

- Localization of the IOFB is the prime aspect of management.
- Plain X-ray is useful in radio-opaque foreign bodies only and will not detect radiolucent IOFBs such as wood or glass.
- Computed tomography provides much more reliable information regarding the size, shape, and localization of the foreign body and is the preferred imaging modality.
- MRI is not used for metallic IOFB given the potential to dislodge the foreign body and cause further destruction and should never be the initial imaging modality of choice after trauma. MRI may be more effective in localizing nonmetallic IOFB such as wood, but should only be used if there is 100% certainty that the object is non-metallic.
- Ultrasound can be a useful adjunct in localizing IOFB and to determine if the object is metallic. It should be performed carefully in cases of an open globe injury.
  Ultrasonography is useful to determine the extent of the intraocular damage, retinal detachment, double perforation, as well as foreign bodies not seen on x-ray studies particularly in cases with intraocular blood.
  Ultrasonography can assist in identifying the nature (air, glass,etc) and shape of the IOFB.
- Ultrasound biomicroscopy and/or gonioscopy can be used if FB in the angle is suspected.
  These tests should be performed with caution to prevent expulsion of intraocular contents.



11. A thin built boy comes with long fingers with subluxation of lens shows deficiency of cystathionine synthase. Which Amino acid should be supplemented?

A. Serine

B. Tyrosine

C. Methionine

D. Cysteine

CLASSIC HOMOCYSTINURIA

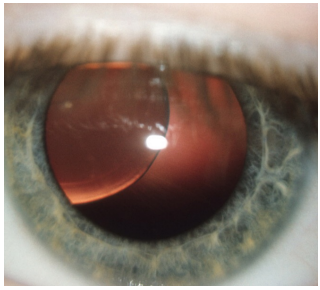
Most common inborn error of methionine metabolism.

Autosomal Recessive trait

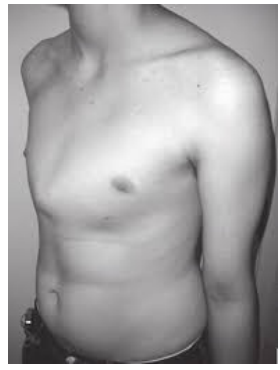
Deficient Cystathionine Beta synthase (Chromosome 21)

CLINICAL FEATURES

- Failure to thrive
- Developmental delay
- Ectopic lentis (Subluxation of Ocular lens)
- Severe Myopia and iridodonesis
- Progressive intellectual disability - common
- Psychiatric and behavioural disorders
- Convulsions
- Skeletal deformities resembling Marfan syndrome
- Scoliosis,Pectus excavatum or carinatum
- Arachnodactyly
- Thromboembolic episodes



Ectopia lentis



Pectus Carinatum

DIAGNOSIS

i. Methionine and Homocystine levels are elevated in body fluids.(Diagnostic lab findings)
ii.Cystine level is low or absent in plasma
iii. Enzyme assay

TREATMENT

i. Vitamin B6
ii. Betaine - lowers homocysteine levels in body fluids.

A normal dose of folic acid supplement and occasionally adding cysteine to the diet can be helpful, as glutathione is synthesized from cysteine (so adding cysteine can be important to reduce oxidative stress).

Reference : Nelson Testbook of Pediatrics 20th Ed. Pg 644

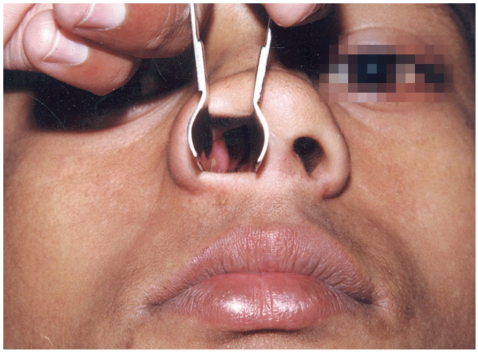
1. A woman is presented to ENT OPD with complaints of nasal obstruction. O/E greenish black crust seen in nasal cavity covering turbinate and septum. She also had merciful anosmia. What other sign will you find in this case on examination.

A. Roomy nose
B. Nasal polyp
C. Foreign body
D. Inferior turbinate hypertrophy

The patient is having Atrophic Rhinitis.

ATROPHIC RHINITIS (OZAENA)

- Chronic inflammation of nose characterized by atrophy of nasal mucosa and turbinate bones
- Wide nasal cavity in atrophic rhinitis
- Roomy nose



SYMPTOMS

It is a bilateral condition mostly in females present with:

- Nasal obstruction
- Nose bleed
- Headache
- Merciful anosmia—foul smell, which the patient herself is unable to perceive due to atrophy of olfactory nerves and excessive crusting of nose.

SIGNS

- Grayish black crusts in the nose
- Nasal passages are roomy
- Turbinates are shrivelled and detachment of crusts- bleeding and ulcerated mucosa
- In late cases, septal perforation and saddle nose may be present
- Eustachian tube catarrh is present and paranasal sinuses and pharynx may be affected.

Reference; Notespaedia ENT image bank pg 82
Reference: Diseases of Ear, Nose and Throat, Dhingra (Pg 171)
Textbook of Ear, Nose and Throat, BS Tuli (Pg 167)

TREATMENT

- Medical treatment includes:

– Regular nasal cleaning

– Antibiotics – ANTIOZAENA solution

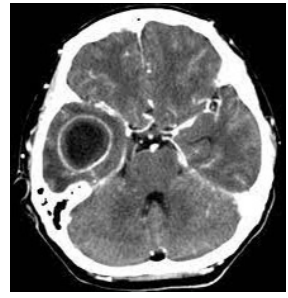
– Application of 25 percent glucose in glycerine which inhibits the growth of proteolytic organisms.

- Surgical treatment

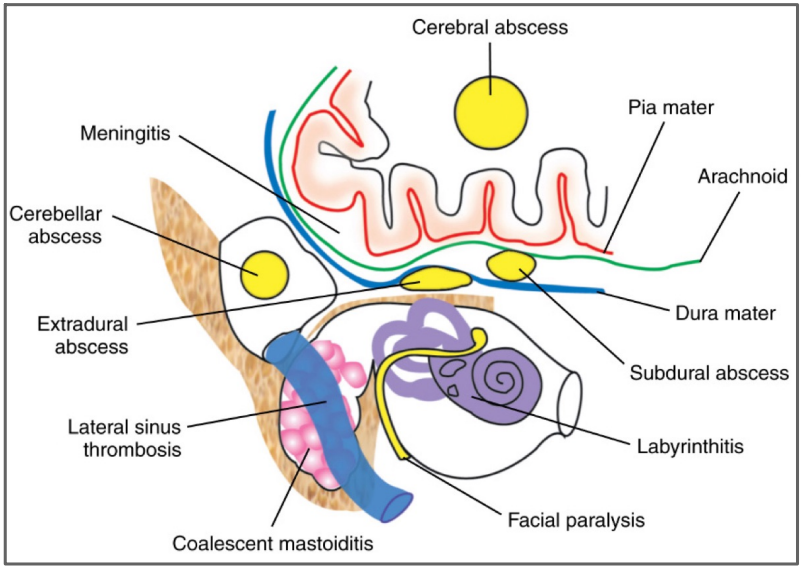
Young's (complete closure of nostril)or modified Young's operation(partial closure)

2. Patient with history of chronic mild ear infection now presents with neurological manifestation, headache and vomiting. CT brain is shown. Probable diagnosis is.

A. Extra dural abscess
B. Cerebral abscess
C. Temporal lobe abscess
D. Meningitis



BRAIN ABSCESS



COMPLICATIONS

INTRATEMPORAL

1. Mastoiditis
2. Petrositis
3. Facial paralysis
4. Labyrinthitis

INTRACRANIAL

1. Extradural abscess
2. Subdural abscess
3. Meningitis
4. Brain abscess
5. Lateral sinus thrombophlebitis
6. Otitic hydrocephalus.

BRAIN ABSCESS

- Cerebral abscess develops as a result of direct extension of middle ear infection through the tegmen or by retrograde thrombophlebitis, in which case the tegmen will be in- tact. Often it is associated with extradural abscess.
- Cerebellar abscess also develops as a direct extension through the Trautmann's triangle or by retrograde throm- bophlebitis.

CLINICAL FEATURES

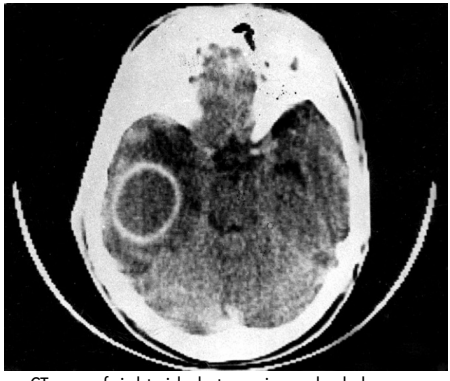
(a) Headache. Often severe and generalized, worse in the morning.
(b) Nausea and vomiting. The latter is usually projectile. Seen more often in cerebellar lesions.
(c) Level of consciousness. Lethargy, which progresses to drowsiness, confusion, stupor and finally coma.
(d) Papilloedema is absent in early cases. Appears late when raised intracranial tension has persisted for 2-3 weeks. Appears early in cerebellar abscess.
(e) Slow pulse and subnormal temperature.

TEMPORAL LOBE ABSCESS

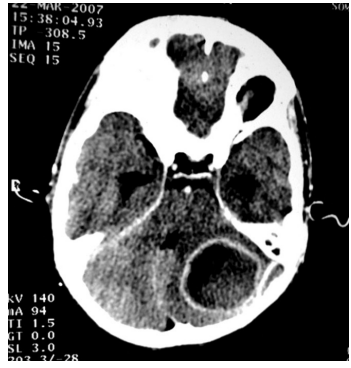
(i) Nominal aphasia. If abscess involves dominant hemisphere, i.e. left hemisphere in right-handed persons, patient fails to tell the names of common objects such as key, pen, etc. but can demonstrate their use.
(ii) Homonymous hemianopia. This is due to pressure on the optic radiations. Visual field, opposite to the side of lesion, is lost.
(iii) Contralateral motor paralysis. In the usual upward spread of abscess, face is involved first followed by the arm and leg. Inward spread, towards internal capsule, involves the leg first followed by the arm and the face.
(iv) Epileptic fits. Involvement of uncinate gyrus causes hallucinations of taste, and smell and involuntary smacking movements of lips and tongue. Generalized fits may occur.

INVESTIGATIONS

1. Skull x-rays. are useful to see midline shift, if pineal gland is calcified, and also reveals gas in the abscess cavity. They have been replaced by CT scan.
2. CT scan. is the single most important means of investigation and helps to find the site and size of an abscess . It also reveals associated complications such as extradural abscess, sigmoid sinus thrombosis, etc. MRI has further improved the diagnosis.
3. X-ray-ray mastoids or ct scan. of the temporal bone for evaluation of associated ear disease.
4. Lumbar puncture. CSF will show some rise in pressure, increase in protein content but normal glucose level. White cell count of CSF is raised but is much less than seen in cases of meningitis. CSF contains polymorphs or lymphocytes depending on the acuteness of lesion.



CT scan of right-sided otogenic cerebral abscess.



CT scan showing left-sided cerebellar abscess.

TREATMENT

- CHLORAMPHENICOL and third generation CEPHALOSPORINS are usually effective.
- Culture of discharge from the ear may be helpful in the choice of antibiotic.
- Raised intracranial tension can be lowered by DEXAMETHASONE or MANNITOL
- Discharge from the ear should be treated by suction clearance and use of topical ear drops.
- Abscess is approached through a sterile field.
  Options include:
  (i) repeated aspiration through a burr hole,
  (ii) excision of abscess and
  (iii) open incision of the abscess and evacuation of pus.
- Chronic otitis media would require radical mastoidectomy to remove the irreversible disease and to exteriorize the infected area.
- Surgery of the ear is undertaken only after the abscess has been controlled by antibiotics and neurosurgical treatment.

3. A 35 year old male presents with epistaxis. Conservative management was done to stop the bleeding but it failed which will be the next step of management.

A. Endoscopic sphenopalatine artery ligation
B. Maxillary artery ligation
C. ICA ligation
D. ECA ligation

EPISTAXIS

Bleeding from inside the nose

Nosebleed

1. **Spontaneous epistaxis:**
   - Common in children and young adults
   - It arises from Little's area
   - It may be precipitated by infection or minor trauma
   - It is easy to stop and tends to recur.
2. **Hypertensive epistaxis:**
   - Hypertensive epistaxis affects an older age group
   - It arises far back or high up in the nose
   - It is often difficult to stop, and it may recur.

SITES OF EPISTAXIS

1. Little's area(90% cases)
2. Above the level of middle turbinate (anterior and posterior ethmoidal vessels)
3. Below the level of middle turbinate. (branches of sphenopalatine artery)
4. Posterior part of nasal cavity- directly into the pharynx.
5. Diffuse. Both from septum and lateral nasal wall.
6. Nasopharynx.

	Anterior epistaxis	Posterior epistaxis
Incidence	More common	Less common
Site	Mostly from Little's area or anterior part of lateral wall	Mostly from posterosuperior part of nasal cavity; often difficult to localize the bleeding point
Age	Mostly occurs in children or young adults	After 40 years of age
Cause	Mostly trauma	Spontaneous; often due to hypertension or arteriosclerosis
Bleeding	Usually mild, can be easily controlled by local pressure or anterior pack	Bleeding is severe, requires hospitalization; postnasal pack often required

Reference; Notespaedia ENT image bank pg 87
Reference: Diseases of Ear, Nose and Throat, Dhingra (Pg 197)
Textbook of Ear, Nose and Throat, BS Tuli (Pg 185)

MANAGEMENT OF NOSEBLEED

- Nasal packing: Anterior and posterior nasal packing or nasopharyngeal balloon
- Cauterization of bleeding area
- Pterygopalatine fossa block
- Laser photocoagulation
- Pharmacologic treatment
- Arterial ligation
- Embolization

LIGATION OF VESSELS

1. External carotid. When bleeding is from the external carotid system and the conservative measures have failed, ligation of external carotid artery above the origin of superior thyroid artery should be done. It is avoided these days in favour of embolization or ligation of more peripheral branches of sphenopalatine artery.

2. Maxillary artery. Ligation of this artery is done in uncontrollable posterior epistaxis. Approach is via Caldwell-Luc operation. Posterior wall of maxillary sinus is removed and the maxillary artery or its branches are blocked by applying clips. This procedure is now superceded by transnasal endoscopic sphenopalatine artery ligation.

3. Ethmoidal arteries. In anterosuperior bleeding above the middle turbinate, not controlled by packing, anterior and posterior ethmoidal arteries, which supply this area, can be ligated. The vessels are exposed in the medial wall of the orbit by an external ethmoid (Lynch) incision.

TRANSNASAL ENDOSCOPIC SPHENOPALATINE ARTERY LIGATION (TESPAL)

- The procedure can be done with rigid endoscopes under topical anaesthesia with sedation or under a general anaesthesia.
- A mucosal flap is lifted in posterior part of lateral nasal wall, sphenopalatine artery (SPA)is localized as it exits the foramen and closed with a vascular clip.
- Distal branches of the artery can be additionally cauterized and the flap then reposited.
- Anterior ethmoidal artery can also be ligated by Lynch incision as an adjunctive procedure.

SPA ligation gives high success in control of refractory posterior bleed.

COMPLICATIONS

Death in 0.6 percent cases may be due to:

- Cerebral hemorrhages
- Aspiration
- Shock
- Septicemia
- Pneumonia
- Coronarythrombosis
- Intestinal infarction.

4. A patient of thyroidectomy was being extubated. The anaesthesiologist realised that when he removes the tube; the patient beings to have recurrent cyanotic spell. Which of the following could be the cause:

A. B/L RLN palsy
B. B/L SLN palsy
C. U/L RLN palsy
D. Hemorrhage

LARYNGEAL PARALYSIS

Laryngeal paralysis may be unilateral or bilateral, and may involve:

1. Recurrent laryngeal nerve.
2. Superior laryngeal nerve.
3. Both recurrent and superior laryngeal nerves (combined or complete paralysis).

CAUSES OF RECURRENT LARYNGEAL NERVE PARALYSIS

Right	Left	Both
• Neck trauma • Benign or malignant thyroid disease • Thyroid surgery • Carcinoma cervical oesophagus • Cervical lymphadenopathy	I. *Neck* • Accidental trauma • Thyroid disease (benign or malignant) • Thyroid surgery • Carcinoma cervical oesophagus • Cervical lymphadenopathy	• Thyroid surgery • Carcinoma thyroid • Cancer cervical oesophagus • Cervical lymphadenopathy
• Aneurysm of subclavian artery • Carcinoma apex right lung • Tuberculosis of cervical pleura • Idiopathic	II. *Mediastinum* • Bronchogenic cancer • Carcinoma thoracic oesophagus • Aortic aneurysm • Mediastinal lymphadenopathy • Enlarged left auricle • Intrathoracic surgery • Idiopathic	

UNILATERAL RECURRENT LARYNGEAL PARALYSIS

- May pass undetected as about one-third of the patients are asymptomatic.
- Others have some change in voice but no problems of aspiration or airway obstruction.
- The voice in unilateral paralysis gradually improves due to compensation by the healthy cord which crosses the midline to meet the paralyzed one.

BILATERAL SUPERIOR LARYNGEAL NERVE PARALYSIS

- Presence of both paralysis and bilateral anaesthesia causes inhalation of food and pharyngeal secretions giving rise to cough and choking fits.
- Voice is weak and husky.

Reference: Disease of ENT, Dhingra pg 337

BILATERAL (BILATERAL ABDUCTOR PARALYSIS)

- Neuritis or surgical trauma (thyroidectomy) are the most important causes.
- The condition is often acute.
- As all the intrinsic muscles of larynx are paralyzed, the vocal cords lie in median or paramedian position due to unopposed action of cricothyroid muscles
- As both the cords lie in median or paramedian position, the airway is inadequate causing dyspnoea and stridor but the voice is good.
- Dyspnoea and stridor become worse on exertion or during an attack of acute laryngitis.

COMBINED PARALYSIS (RECURRENT AND SUPERIOR LARYNGEAL NERVE PARALYSIS)

BILATERAL

1. Aphonia. As cords do not meet at all.
2. Aspiration. This is due to incompetent glottis and laryngeal anaesthesia.
3. Inability to cough. This is due to inability of the cords to meet. This results in retention of secretions in the chest.
4. Bronchopneumonia. This is due to repeated aspira- tions and retention of secretions.

5. Which of the following is the topical use of the medicine shown in the image:

A. Subglottic stenosis
B. Indy type 1 myringoplasty
C. Post- adenoidectomy to control bleeding
D. Rhino- cerebral mucormycosis

MITOMYCIN C

- Antineoplastic antibiotic isolated from the bacterium Streptomyces caespitosus
- Bioreduced mitomycin C generates oxygen radicals
- Alkylates DNA
- Preferentially toxic to hypoxic cells
- Used in treatment regime, Palliative regime of various CA and also as Intravesical injection in Bladder CA

IN ENT

- Antiproliferative agent- Inhibit fibroblastic proliferation and decrease Scar formation
- Because of these properties used as topical solution in various ENT surgeries viz.,
  - FESS
  - Endoscopic DCR
  - Subglottic Stenosis
  - Ventilation tube in OME
  - Choanal atresia
  - Oesophageal stenosis
  - Hypopharyngeal stenosis
  - Tracheal stenosis

6. A post tonsillectomy child was lying in the ward. He started bleeding in the ward. Which of the following should be done:

A. Take to OT, remove the clot and re-ligation
B. Take to OT and pressure packing
C. Cautery
D. Conservative management

The child is having reactionary haemorrhage . Removal of clot and re ligation is the next step.

COMPLICATIONS OF TONSILLECTOMY

IMMEDIATE

1. Primary haemorrhage
Occurs at the time of operation.
It can be controlled by pressure, ligation or electrocoagulation of the bleeding vessels.
2. Reactionary haemorrhage
Occurs within a period of 24 h and can be controlled by simple measures such as removal of the clot, application of pressure or vasoconstrictor.
Presence of a clot prevents the clipping action of the superior constrictor muscle on the vessels which pass through it (compare postpartum uterine bleeding).
If above measures fail, ligation or electrocoagulation of the bleeding vessels can be done under general anaesthesia.
3. Injury to tonsillar pillars, uvula, soft palate, tongue or superior constrictor muscle due to bad surgical technique.
4. Injury to teeth.
5. Aspiration of blood.
6. Facial oedema. Some patients get oedema of the face particularly of the eyelids.
7. Surgical emphysema. Rarely occurs due to injury to superior constrictor muscle.

DELAYED

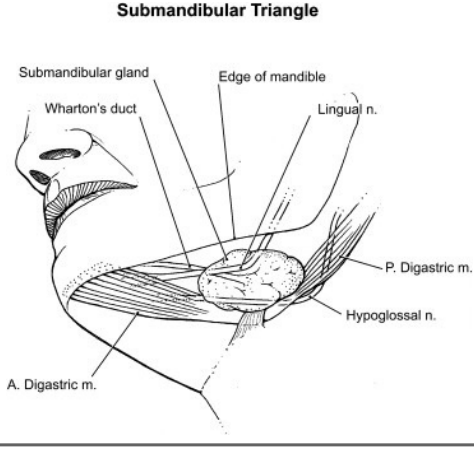
1. Secondary haemorrhage. Usually seen between the fifth to tenth postoperative day. It is the result of sepsis and premature separation of the membrane. Usually, it is heralded by bloodstained sputum but may be profuse.
2. Infection. Infection of tonsillar fossa may lead to parapharyngeal abscess or otitis media.
3. Lung complications. Aspiration of blood, mucus or tissue fragments may cause atelectasis or lung abscess.
4. Scarring in soft palate and pillars.
5. Tonsillar remnants. Tonsil tags or tissue, left due to inadequate surgery, may get repeatedly infected.
6. Hypertrophy of lingual tonsil. This is a late com- plication and is compensatory to loss of palatine tonsils.

7. A patient had undergone submandibular gland excision and the Wharton's duct was ligated in the process. Which of the following nerves is most likely to be damaged.

A. Inferior alveolar nerve
B. Hypoglossal nerve
C. Lingual nerve
D. Nerve to mylehyoid

SUBMANDIBULAR GLAND

- Surgical excision of the submandibular gland (SMG) is commonly indicated in patients with neoplasms, and non-neoplastic conditions such as chronic sialadenitis, sialolithiasis, ranula and drooling.
- The submandibular duct, or "Wharton's duct", is approximately 5 cm in length and arises from the anterior portion of the gland.
- It traverses the floor of mouth between the mylohyoid and genioglossus muscles alongside the tongue.
- Its opening is found at the base of the frenulum of the tongue just posterior to the inferior incisors on the submandibular caruncle via one to three orifaces.
- The sublingual space is an important anatomical region that lies between the tongue and the mandibular ramus containing the deep portion of the sublingual gland, the anterior portion of the Wharton's duct, and the lingual nerve which carries parasympathetic nerve fibers to the gland.
- During its course, the lingual nerve loops under the duct running a lateral to medial course and travels deep and superior to the SMG

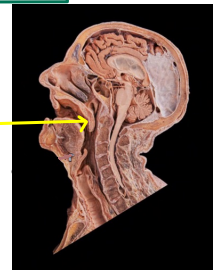


Lateral Transcervical approach. The lingual nerve loops under the duct running a lateral to medial course and the travels deep and superior to the submandibular gland. The hypoglossal nerve lies inferior and medial to the lower third of the gland under the posterior belly of the digastric muscle.

NERVE INJURIES

- **Lingual nerve:**
  Immediate post-operative ipsilateral parathesia and loss of taste from the anterior two-thirds of the tongue, which is rarely permanent.
- **Hypoglossal nerve:**
  Ipsilateral paresis or paralysis of the intrinsic muscles of the tongue leading to dysarthria and deviation of tongue to side of the lesion. This nerve is rarely injured in this procedure to an extent to produce noticeable disability.
- **Facial nerve (marginal mandibular branch):**
  Ipsilateral paresis or paralysis of the muscles supplying the lower lip and chin, including depressor labii inferioris, which characteristically presents as drooping of the lower lip. This is usually temporary, lasting for 6-12 weeks.

8. A patient was admitted with skull base trauma. The doctor was testing the marked structure. Which of the following nerve is being tested.



A. Vagus
B. Facial
C. Glossopharyngeal
D. Trigeminal

The posterior part of soft palate is sensory and is innervated by the pharyngeal branches of the glossopharyngeal nerve (CN IX)

SOFT PALATE

Surfaces and borders	Surfaces: Oral, nasal Borders: Anterosuperior, lateral, posteroinferior
Features	Uvula, palatoglossal arch, palatopharyngeal arch
Muscles	Musculus uvulae, tensor veli palatini, levator veli palatini, palatopharyngeus and palatoglossus muscles
Innervation	General sensory: Maxillary nerve (anterior part), glossopharyngeal nerve (posterior part) Special sensory/taste buds: Facial nerve Visceromotor/mucous glands: Glossopharyngeal nerve
Blood supply	Ascending palatine artery of facial artery
Function	Speech, breathing, and swallowing

Community Medicine

1. In a 10 year school child under school health program, which vaccine has to be given?

A. BCG

B. MMR

C. TT, Td

D. DPT

NATIONAL IMMUNIZATION SCHEDULE

Vaccine	When to give	Dose	Route	Site
For Pregnant Women				
Tetanus Toxoid (TT)-1	Early in pregnancy	0.5 ml	Intra-muscular	Upper Arm
TT-2	4 weeks after TT-1*	0.5 ml	Intra-muscular	Upper Arm
TT-Booster	If received 2 TT doses in a pregnancy within the last 3 yrs*	0.5 ml	Intra-muscular	Upper Arm
For Infants				
BCG	At birth or as early as possible till one year of age	0.1ml (0.05ml until 1 month age)	Intra-dermal	Left Upper Arm
Hepatitis B-birth dose	At birth or as early as possible within 24 hours	0.5 ml	Intra-muscular	Antero-lateral side of mid-thigh
OPV-0	At birth or as early as possible within the first 15 days	2 drops	Oral	Oral
OPV 1, 2 and 3	At 6 weeks, 10 weeks and 14 weeks (OPV can be given till 5 years of age)	2 drops	Oral	Oral
DPT 1, 2 and 3	At 6 weeks, 10 weeks and 14 weeks (DPT can be given up to 7 yrs of age)	0.5 ml	Intra-muscular	Antero-lateral side of mid thigh
Hepatitis B 1, 2 and 3	At 6 weeks, 10 weeks and 14 weeks (can be given till one year of age)	0.5 ml	Intra-muscular	Antero-lateral side of mid-thigh
Pentavalent** **1, 2 and 3**	At 6 weeks, 10 weeks and 14 weeks (can be given till one year of age)	0.5 ml	Intra-muscular	Antero-lateral side of mid-thigh
Measles-1	9 completed months–12 months. (Measles can be given till 5 years of age)	0.5 ml	Sub-cutaneous	Right Upper Arm
Japanese Encephalitis JE-1**	9 completed months–12 months.	0.5 ml	Sub-cutaneous	Left Upper Arm
Vitamin A (1st dose)	At 9 completed months with measles	1 ml (1 lakh IU)	Oral	Oral
For Children				
DPT booster-1	16–24 months	0.5 ml	Intra-muscular	Antero-lateral side of mid-thigh
Measles 2nd dose	16–24 months	0.5 ml	Sub-cutaneous	Right upper Arm
OPV Booster	16–24 months	2 drops	Oral	Oral
Japanese Encephalitis**	16–24 months	0.5 ml	Sub-cutaneous	Left upper Arm
Vitamin A* (2nd to 9th dose)**	16 months. Then, one dose every 6 months up to the age of 5 years.	2 ml (2 lakh IU)	Oral	Oral
DPT Booster-2	5–6 years	0.5 ml.	Intra-muscular	Upper Arm
TT	10 years and 16 years	0.5 ml	Intra-muscular	Upper Arm

2. A girl child has recurrent yeast and respiratory virus infection since she was 3 months old. Now considering studies for immune status, which vaccine is contraindicated?

A. TT/Td

B. Measles/ MMR

C. DPT

D. Killed IPV

- Live vaccines should not be administered to persons with immune deficiency diseases.
- Pregnancy is a nother contra indication unless the risk of infection exceeds the risk of harm to the foetus of some live vaccines.

TYPES OF VACCINES

Live vaccines	Live Attenuated vaccines	Killed Inactivated vaccines	Toxoids	Cellular fraction vaccines	Recombinant vaccines
•Small pox variola vaccine	•BCG •Typhoid oral •Plague •Oral polio •Yellow fever •Measles •Mumps •Rubella •Intranasal Influenza •Typhus	•Typhoid •Cholera •Pertussis •Plague •Rabies •Salk polio •Intra-muscular influenza •Japanise encephalitis	•Diphtheria •Tetanus	•Meningococcal polysaccharide vaccine •Pneumococcal polysaccharide vaccine •Hepatitis B polypeptide vaccine	•Hepatitis B vaccine

Vaccine	Contraindication
Human papillomavirus (HPV)	• History of hypersensitivity to yeast
Tetravalent meningococcal conjugate vaccine (MCV4)	• History of severe allergic reaction to diphtheria toxoid or to dry natural rubber latex • History of group B *Streptococcus* (GBS) is a relative contraindication; therefore, clinicians should discuss this with patients prior to administration
Tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis (Tdap)	• History of encephalopathy within 7 days of administration of prior dose of pertussis vaccine not attributable to another cause • Boostrix® (GlaxoSmithKline Biologicals) should not be given if there is a history of anaphylaxis to latex because the tip and plunger of the needleless syringe contain latex • Adacel® (Sanofi Pasteur, Inc) preparations have no latex
Trivalent inactivated influenza vaccine (TIV)	• History of anaphylaxis to eggs
Live-attenuated influenza vaccine (LAIV)	• History of anaphylaxis to eggs • Chronic conditions that put the individual at higher risk for complications from influenza (eg, asthma, chronic heart or lung disease, kidney disease, diabetes) • Concomitant aspirin therapy • Possible immunodeficiency • History of GBS
Measle, mumps, rubella (MMR)	• History of anaphylaxis to gelatin or neomycin • Known immunodeficiency • Pregnancy
Varicella	• History of anaphylaxis to gelatin or neomycin • Known immunodeficiency • Pregnancy
Inactivated poliovirus vaccine (IPV)	• History of anaphylaxis to streptomycin, neomycin, or polymyxin B

*A vaccine should not be given if there was a severe allergic reaction after a previous dose or to any component to the vaccine. The *AAP Red Book* contains additional information on contraindications or precautions.

3. Throat swab was taken from a three year old girl who had fever, cough and sore throat and was sent for culture and sensitivity. The swab will be disposed to which coloured biomedical waste bags ?

A. Yellow

B. Red

C. Black

D. White translucent box

Throat swab contaminated with body fluids should be disposed of in yellow bag.

Schematic of the BMW/hospital waste generation to disinfection and disposal practices.

SEGREGATION GUIDELINES*	
COLOUR	**WASTE DESCRIPTION**
YELLOW*	Human tissues, organs, body parts, items contaminated by blood/body fluids, soiled cotton & dressing, soiled plaster casts etc.
RED*	Catheters, tubes, cannulae, syringes, plastic IV bottles & sets, used gloves, infected plastics, specimen containers, lab waste, microbiology cultures, used or discarded bags of blood/blood products, vaccines etc.
BLUE*	Glass items, needles, syringes, scalpels, blades, used and unused sharps etc.
BLACK*	Discarded medicines, discarded cytotoxic drugs etc.
GREEN	General waste, non-infected plastic materials & papers, disposables, cardboards, metal containers, office waste, food waste etc.
***Recommended by CPCB**	

CATEGORY OF WASTE	COLOR CODE (NON-CHLORINATED BAGS)	PRE-TREATMENT REQUIRED OR NOT	FINAL DISPOSAL OPTION AT CBMWTF
Soiled waste like items contaminated with blood (excluding blood bags) & body fluids, cotton swabs, etc	YELLOW	Not required	Incineration
Chemical used in the labs, Liquid waste generated in Labs, used or disinfectants to be discarded, infected secretions, aspirated body fluids, floor washings & other housekeeping & disinfecting activities	YELLOW	Separate Collection system leading to Effluent Treatment Plant for pre-treatment & neutralization	The pre-treated liquid waste shall conform to the discharge norms and then is lead to the general drain.
Personal Protective Materials like face mask, gown, caps, etc (made of fibre material or others except those made of disposable plastics)	YELLOW	Not required	Incineration
Discarded Linen, beddings contaminated with blood or body fluid	YELLOW	Not Required	Non-chlorinated Chemical Disinfection followed by incineration.
Microbiology & Laboratory waste like cultures, stocks, specimens, vaccines, dishes and devices used for cultures, blood bags, etc.	YELLOW	Pre-treatment with non-chlorinated chemicals or autoclave, microwave or hydro-clave in safe plastic bags or containers.	Pre-treated and followed by incineration.
Gloves even if contaminated with blood & body fluids	RED	Not required	Autoclaved, shredding followed by recycling.
IV tubing, bottles, sets, catheters, urine bags, vaccutainers, etc	RED	Not Required	Autoclaved, shredding followed by recycling.
Broken or intact glassware's, medicine vials, ampoules except those contaminated with Cytotoxic Waste.	BLUE Puncture proof, leak proof box or container with blue marking	Not required	Disinfection then recycling.
Metallic Sharp waste like needles, scalpels, blades, syringes with fixed needles.	WHITE Puncture proof, leak proof, tamper proof containers	Not Required	Autoclaving, shredding followed by encapsulation or disposed in iron foundries.

Common Bio-Medical Waste Treatment Facility (CBMWTF)

https://health.uk.gov.in/files/Corona/13._Biomedical_Waste_Management_for_COVID_19_1.pdf

4. Following admission of a RTA case, there is spillage of blood on hospital floor.
Which disinfectant will you use to clean the floor?

A. Ethyl alcohol
B. Chlorhexidine
C. Formaldehyde
D. Sodium hypochlorite

Sodium hypochlorite is used as a disinfectant in case of blood spillage on hospital floor

DISINFECTION

Thermal or chemical destruction of pathogen and other types of microorganisms.
Disinfection is less lethal than sterilization because it destroys most recognized pathogenic microorganisms but not necessarily all microbial forms (e.g., bacterial spores).

- **NATURAL AGENTS:** Sunlight, Air
- **PHYSICAL AGENTS:** Burning, Hot air, Boiling, Autoclaving, Radiation
- **CHEMICAL AGENTS**
  - Phenol and related compounds: Phenol, Crude phenol, Cresol, Cresol emulsions, Chlorhexidine (hibitane), Hexachlorphane, dettol
  - Quaternary ammonia compounds: Cetrimide, Savlon
  - Halogens and their compounds: Bleaching powder, Hypochlorites, Iodine
  - Alcohols: Ethyl and isopropyl alcohols
  - Formaldehyde
  - Oxidizing agents
  - Metals as microbicides
  - Lime

HYPOCHLORITES

- Hypochlorites are the most widely used chlorine disinfectant, available as liquid (e.g. sodium hypochlorite) or solid (e.g. calcium hypochlorite).
- The most prevalent chlorine products are aqueous solutions of 5.25– 6.15 per cent of sodium hypochlorite, usually called household bleach.
- They have a broad spectrum of antimicrobial activity, do not leave toxic residues, are unaffected by water hardness, are inexpensive and fast acting, remove dried or fixed organisms and biofilms from surfaces

ALCOHOL

- Ethyl alcohol in the form of industrial methylated spirit is the alcohol most commonly used for skin disinfection and hand washing.
- Pure alcohol has no powers of disinfection but when diluted with water, it is potent bactericidal, fungicidal, virucidal and tuberculocidal, but does not destroy bacterial spores

FORMALDEHYDE

- Formaldehyde is a highly toxic and irritant gas which precipitates and destroys protein.
- It is effective against vegetative bacteria, fungi and many viruses but only slowly effective against bacterial spores (e.g., tetanus spores) and acid-fast bacteria.
- It does not injure fabrics and metals.
  Formaldehyde gas is most commonly used for disinfection of rooms.

5. A 16 month old child, 8 kg weight. On assessing in a growth chart, child falls below -3SD. What should be done next for management?

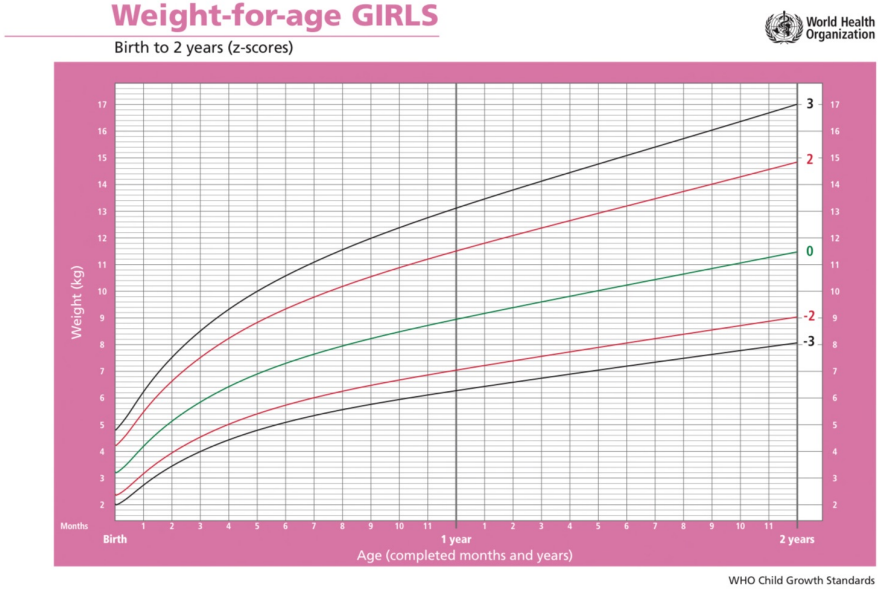
A. Assure mother that no malnutrition
B. Mild malnutrition- home treatment
C. Moderate malnutrition- teach mother on how to feed
D. Severe malnutrition: refer to NRC

GROWTH CHART

"ROAD-TO-HEALTH"

Normal: 90 - 110%
Grade I (mild malnutrition): 75 - 89%
Grade II (moderate malnutrition): 60 - 74%
Grade III (severe malnutrition): <60%

Percentile	z-score
>99th	3
97th	2
85th	1
50th	0
15th	-1
3rd	-2
<1st	-3

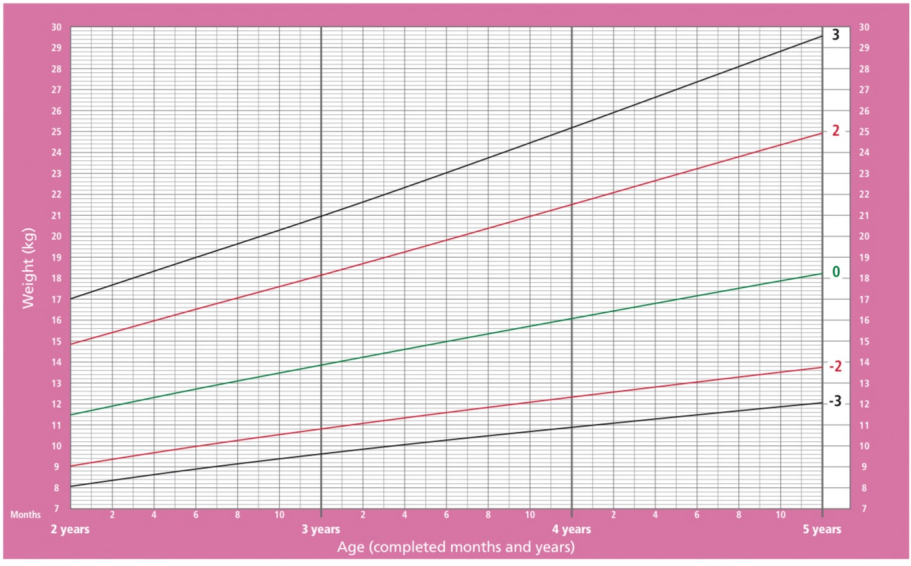


WHO Child Growth Standards

Weight-for-age GIRLS

2 to 5 years (z-scores)

World Health Organization

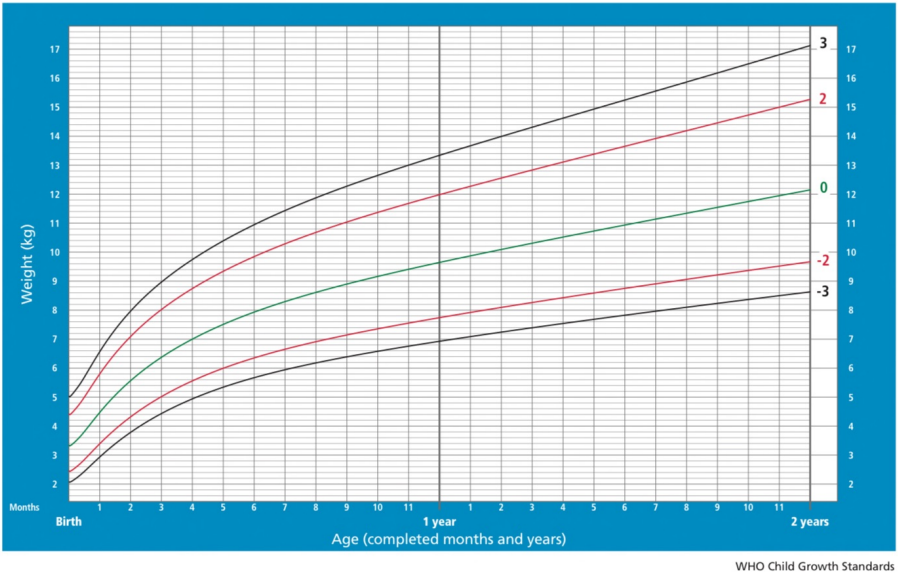


WHO Child Growth Standards

Weight-for-age BOYS

Birth to 2 years (z-scores)

World Health Organization

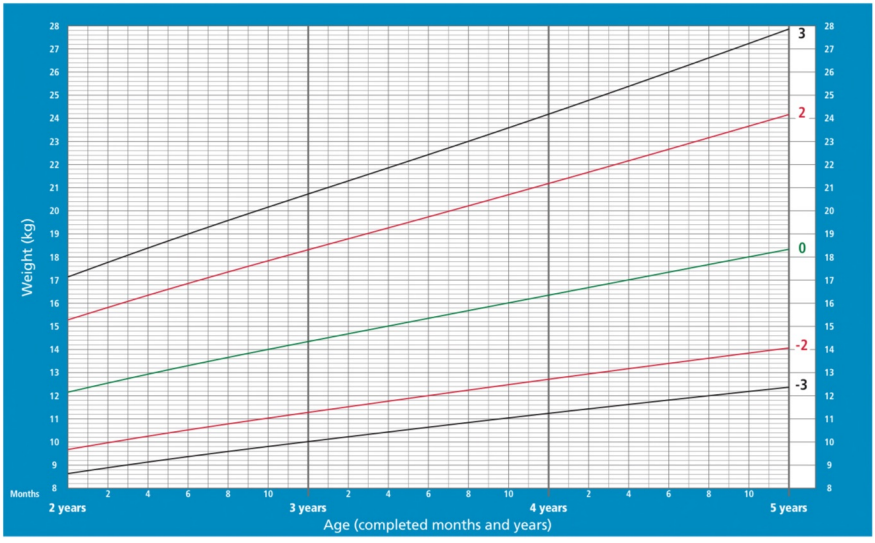


WHO Child Growth Standards

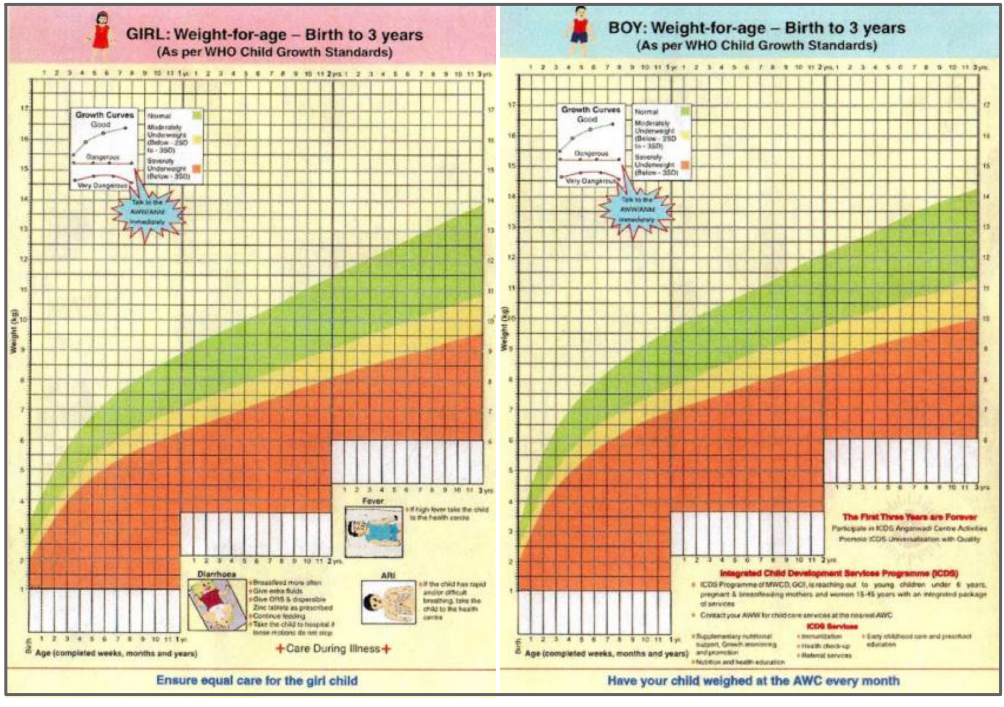
Weight-for-age BOYS

2 to 5 years (z-scores)

World Health Organization



WHO Child Growth Standards



GREEN COLOUR –> Reassure the mother
YELLOW COLOUR –> Refer to PHC (MBBS)
RED COLOUR –> Refer to CHC, District hospital (Paediatrician)

6. An anganwadi teacher takes weight and height of a 4 year old and finds out that height for age < - 2SD, likely cause is?

A. Chronic malnutrition
B. Acute malnutrition
C. Recent malnutrition
D. No malnutrition

Low height for age reflects past or chronic malnutrition.

Nutritional status	Age: birth to 5 years Indicator and cut-off value compared to the median of the *WHO child growth standards*[a]
Obese	Weight-for-length/height[b] or BMI-for-age >3 standard deviations (SD) of the median
Overweight	Weight-for-length/height[b] or BMI-for-age >2 SD and ≤3 SD of the median
Moderately underweight	Weight-for-age <−2 SD and ≥−3 SD of the median
Severely underweight	Weight-for-age <−3 SD of the median
Moderate acute malnutrition	Weight-for-length/height[b] or BMI-for-age ≤−2 SD and ≥−3 SD of the median, or mid-upper arm circumference ≥115 mm and <125 mm
Severe acute malnutrition	Weight-for-length/height[b] or BMI-for-age <− 3 SD of the median or mid-upper arm circumference <115 mm, or bilateral pitting oedema
Moderately stunted (moderate chronic malnutrition)	Length/height-for-age[b] ≤−2 SD and ≥−3 SD of the median
Severely stunted (severe chronic malnutrition)	Length/height-for-age[b] <−3 SD of the median
Moderately wasted	Weight-for-length/height ≤−2 SD and ≥−3 SD of the median
Severely wasted	Weight-for-length/height <−3 SD of the median

WEIGHT-FOR-AGE

- Measurement of weight and rate of gain in weight are the best single parameters for assessing physical growth.
- A single weight record only indicates the child's size at the moment; it does not give any information about whether a child's weight is increasing, stationary or declining.
- This is because, normal variation in weight at a given age is wide.

HEIGHT (LENGTH)-FOR-AGE

- Height is a stable measurement of growth as opposed to body weight.
- Whereas weight reflects only the present health status of the child, height indicates the events in the past also.
- The use of growth (height) centile chart is particularly valuable in studying the trend of height curve.
- Low height for age : This is also known as nutritional stunting or dwarfing. It reflects past or chronic malnutrition.

WEIGHT-FOR-HEIGHT

- It helps to determine whether a child is within range of "normal" weight for his height.
- Low weight for height : This is also known as nutritional wasting or emaciation (acute malnutrition). It is associated with an increased risk of mortality and morbidity

HEAD AND CHEST CIRCUMFERENCE

- At birth the head circumference is about 34 cm.
- It is about 2 cm more than the chest circumference. By 6 to 9 months, the two measurements become equal, after which the chest circumference overtakes the head circumference.
- In severely malnourished children, this overtaking may be delayed by 3 to 4 years due to poor development of the thoracic cage.

7. Global hunger index does not include

A. IMR

B. Child mortality

C. Child underweight

D. Child undernourishment

GLOBAL HUNGER INDEX (GHI)

- The Global Hunger Index (GHI) is a tool designed to comprehensively measure and track hunger globally, by region and country.
- It highlights successes and failures in hunger reduction.
- It is calculated each year by the International Food Policy Research Institute.
- GHI combines three equally weighted indicators into one index:

1. UNDERNOURISHMENT : the proportion of undernourished people as a percentage of the population (reflecting the share of the population with insufficient calorie intake);

Reference: Preventive and Social Medicine, PARK pg 757

2. **CHILD UNDERWEIGHT** : the proportion of children under the age of five who are underweight (that is, have low weight for their age, reflecting wasting, stunted growth, or both), which is one indicator of child undernutrition;

3. **CHILD MORTALITY** : the mortality rate of children under the age of five (partially reflecting the fatal synergy of inadequate food intake and unhealthy environments).

$$\text{GHI} = \frac{\text{Proportion of undernourished population (PNU)} + \text{Children under weight (CUW)} + \text{Child mortality in percentage (CM)}}{3}$$

- The calculations result in a 100-point scale on which zero is the best score (no hunger) and 100 the worst

8. In a community attacked with an outbreak of multiple cases of encephalitis. According to universal immunisation schedule, the route of administration of vaccine for the likely infection is?

A. Live subcutaneous
B. Killed subcutaneous
C. Live intra-dermal/ intra - muscular
D. Killed intra- dermal/ intramuscular

JAPANESE ENCEPHALITIS VACCINE

- Currently, the three types of JE vaccines in large-scale use are :
  (i) the mouse brain-derived, purified and inactivated vaccine, which is based on either the Nakayama or Beijing strains of the JE virus and produced in several Asian countries;
  (ii) the cell culture- derived, inactivated JE vaccine based on the Beijing P-3 strain,
  (iii) the cell culture-derived, live attenuated vaccine based on the SA 14-14-2 strain of the JE virus.
- The immunization schedules of the 3 licensed JE vaccines that are currenly in large-scale use vary with the profile of the respective vaccines and depend on local epidemiological circumstances and recommended schedules of other childhood vaccines.
- When immunizing children 1- 3 years of age the mouse brain-derived vaccine provides adequate protection throughout childhood following 2 primary doses 4 weeks apart, and boosters after 1 year and subsequently at 3-yearly intervals until the age of 10- 15 years.
- The vaccine is given subcutaneously in doses of 0.5 ml for children under 3 years and one ml for children more than 3 years of age.
- Protective immunity develops in about one month time after the second dose. The vaccine is best used in the interepidemic period. It should be offered to the most vulnerable and high-risk groups.

- Equally good childhood protection is obtained by a single dose of the cell-culture based, live attenuated vaccine (SA-14-14-2) followed by a single booster given at an interval of about 1 year.
- This vaccine is available in India and is an integral part of Universal Immunization Programme in 83 endemic districts in Uttar Pradesh, Assam, West Bengal and Karnataka targeting children in age group 1-15 years .
- The importance of achieving long-term protection is underlined by the observation that in some areas an increasing proportion of the JE cases occur in individuals older than 10 years of age

9. Which thermometer is used to assess velocity of air and not the cooling power?

A.Kata

B.Globe

C.Silvered

D.Wet globe

The Kata thermometer provides a good general measure of air velocity

KATA THERMOMETER

- The Kata thermometer is an alcohol thermometer with a glass bulb 4 cm long and 1.8 cm in diameter. The readings on the stem are marked from 100 deg. to 95 deg.F.
- The Kata thermometer is now largely used as an anemometer for recording low air velocities rather than the cooling power of the air

  (1) The standard Kata - cooling range between 100 deg. - 95 deg.F.
  (2) The High Temperature Kata - cooling range between 130 deg. - 125 deg.F.
  (3) The Extra High Temperature Kata - cooling range between 150 deg. - 145 deg.

- The standard Kata is coloured red; the high temperature instrument dark blue; and the extra high instrument magenta.
- Kata thermometers have silvered bulbs to reduce the errors due to radiation.

SILVERED THERMOMETER

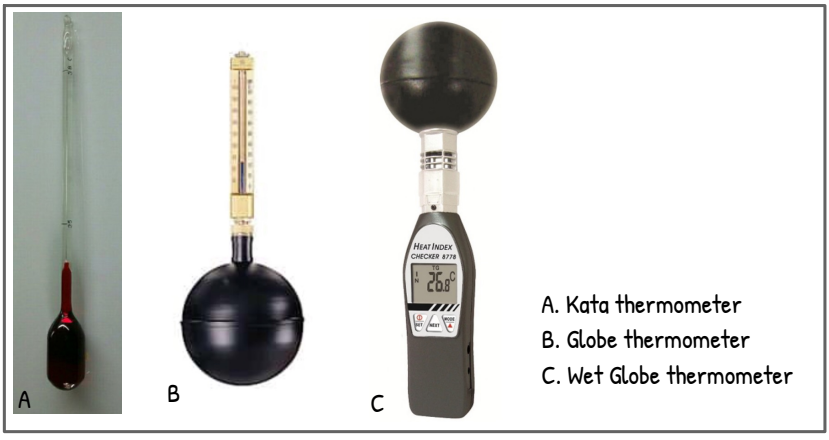
- The bright metallic surface reflects as much of the incident radiant heat as possible. This gives a more accurate reading of the air temperature.

WET GLOBE THERMOMETER

- This instrument is designed for environmental heat measurement.

GLOBE THERMOMETER

- The globe thermometer is used for the direct measurement of the mean radiant temperature of the surroundings.



10. You were given the incharge of health service of nearby school. To whom should you provide reports of the school health service ?

a. District Hospital

b. Sub Centre

c. Primary Health Centre

d. Sub divisional health centre

SCHOOL HEALTH SERVICE

- The primary health centres are charged with the responsibility of administering school health service within their jurisdiction.
- It requires a whole-time, medical officer to cover 5,000 to 6,000 children a year.
- The School Health Committee (1961) has therefore recommended that the staff of the primary health centres should be augmented by additional staff to carry out effectively the school health programme.
- The School Health Committee set up by the Government of India in 1960 recommended that school health service should be an integral part of the general health services.
- The general health services in India are administered largely through the primary health centres in the rural areas, where the bulk of India's population lives.
- School health service is therefore an important function of the primary health centres.

OBJECTIVES OF SCHOOL HEALTH SERVICE

1. the promotion of positive health;
2. the prevention of diseases;
3. early diagnosis. treatment and follow-up of defects;
4. awakening health consciousness in children; and
5. the provision of healthful environment.

11. SI unit of brightness of light?

A. Lux

B. Candela

C. Lumen

D. Luminance

MEASURES OF LIGHT

THE FOUR MEASURES ARE :

(1) Luminous intensity, which is the "power" of a light source considered as a point radiating in all directions; this is measured as candela or candle power.

(2) Luminous flux, which is the flow of light related to a unit of solid angle measured in lumen.

(3) Illumination or illuminance, which is the amount of light reaching a surface measured in lux per unit area; and

(4) Brightness or luminance which is the amount of light reflected from a surface measured in /amberts.

Description	Quantity measured Name	Recommended Unit*	Other Units
1. Brightness of point source	Luminous intensity	Candela	Candle power
2. Flow of light	Luminous flux	Lumen	
3. Amount of light reaching surface	Illumination Illuminance	Lux (Phot)	Foot candle Lumen/cm²
4. Amount of light re-emitted by surface	Brightness Luminance	Lambert	Foot lambert Candles/cm²

12. Post Diwali air pollution index chart was created. Air quality index values on 4 different days were plotted. A AQI value of 407 indicates which level of air pollution?

A. Poor

B. Very poor

C. Severe

D. Moderate

INDICATORS OF AIR POLLUTION

The best indicators of air pollution are sulphur dioxide, smoke and suspended particles. These are monitored on a daily basis over several sites. The results are then collected by a central agency.

(a) Sulphur dioxide : This gas is a major contaminant in many urban and industrial areas. Its concentration is estimated in all air pollution surveys.

(b) Smoke or soiling index: A known volume of air is filtered through a white filter paper under specified conditions and the stain is measured by photoelectric meter. Smoke concentration is estimated and expressed as micrograms/ cubic metre of air as an average level over a period of time.

(c) Grit and dust measurement : Deposit gauges collect grit dust and other solids. These are analyzed monthly.

(d) Coefficient of haze : A factor used, particularly in the USA in assessing the amount of smoke or other aerosol in air.

(e) Air pollution index : It is an arbitrary index which takes into account one or more pollutants as a measure of the severity of pollution.

AIR QUALITY INDEX (AQI)	CATEGORY
0-50	Good
51-100	Satisfactory
101-200	Moderate
201-300	Poor
301-400	Very Poor
401-500	Severe

The AQI is calculated based on the average concentration of a particular pollutant measured over a standard time interval (24 hours for most pollutants, 8 hours for carbon monoxide and ozone).

13. A researcher wants to know whether there is an association of CRP values with risk of MI/ Cancer. They started to take CRP readings. 4 values of RR was plotted (1, 1.5,1.7, 1.8) with respect to CRP:

A. CRP increases Disease/ cancer risk
B. CRP has no relationship
C. CRP decreases and disease decreases
D. No association

RELATIVE RISK

- Relative risk (RR) is the ratio of the incidence of the disease (or death) among exposed and the incidence among non-exposed.

$$RR = \frac{\text{Incidence of disease (or death) among exposed}}{\text{Incidence of disease (or death) among non-exposed}}$$

Reference: Preventive and Social Medicine, PARK pg 81

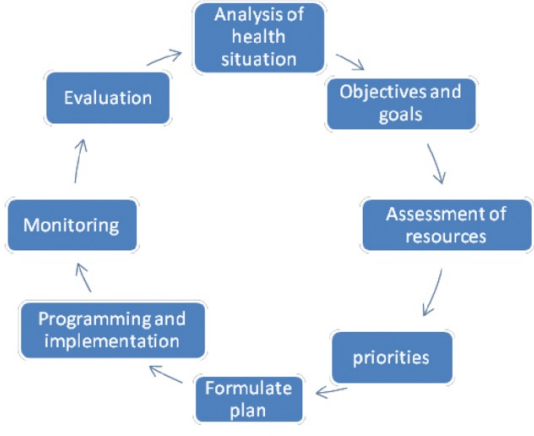
- Estimation of relative risk (RR) is important in aetiological enquiries.
- It is a direct measure (or index) of the "strength" of the association between suspected cause and effect.
- A relative risk of one indicates no association; relative risk greater than one suggests "positive" association between exposure and the disease under study.
- A relative risk of 2 indicates that the incidence rate of disease is 2 times higher in the exposed group as compared with the unexposed.
- Equivalently, this represents a 100 per cent increase in risk.
- A relative risk of 0.25 indicates a 75% reduction in the incidence rate in exposed individuals as compared with the unexposed .
- It is often useful to consider the 95 per cent confidence interval of a relative risk since it provides an indication of the likely and maximum levels of risk.
- The larger the RR, the greater the "strength" of the association between the suspected factor and disease.
- It may be noted that risk does not necessarily imply causal association.

14. For functioning of a health Centre with reference to evaluation, which is the most important determinant for assessing clinical management

A. Process
B. Outcome
C. Input
D. Structure

PLANNING CYCLE

Planning may be defined as a process of analysing a system, or defining a problem, assessing the extent to which the problem exists as a need, formulating goals and objectives to alleviate or ameliorate those identified needs, examining and choosing from among alternative intervention strategies, initiating the necessary action for its implementation and monitoring the system to ensure proper implementation of the plan and evaluating the results of intervention in the light of stated objectives.



Reference: Preventive and Social Medicine, PARK pg 932

EVALUATION

- The purpose of evaluation is to assess the achievement of the stated objectives of a programme, its adequacy, its efficiency and its acceptance by all parties involved.
- While monitoring is confined to day-to-day or ongoing operations, evaluation is mostly concerned with the final outcome and with factors associated with it.
- Good planning will have a built-in evaluation to measure the performance and effectiveness and for feed-back to correct deficiencies or fill up gaps discovered during implementation.
- In the words of the WHO Expert Committee on National Health Planning in Developing Countries, evaluation

"measures the degree to which objectives and targets are fulfilled and the quality of the results obtained.
It measures the productivity of available resources in achieving clearly defined objectives.
It measures how much output or cost-effectiveness is achieved.
It makes possible the reallocation of priorities and of resources on the basis of changing health needs"

Monitoring	Evaluation
• Routine/continuous. Entails data collection and analysis throughout the life of the project • Provides early indications of progress and achievement of goals • Measures project inputs, process, and outputs. • Performed throughout the life of a project	• Episodic/intermittent. Data collection at the start of a programme (to provide a baseline) and again at the end, rather than at repeated intervals during programme implementation • Systematic data collection process • Capable of linking programme activities to behaviors and health outcomes as direct results • Identifies the outcome and impact of a project with the aim of informing the design of future projects and determining if the interventions carried out were effective • Examines longer-term outcomes or results • Identifies how and why activities succeeded, failed, or where changes are needed.

15. In kerala,after flood, People's Complaining of fever, doxycycline prophylatically distributed,along it which insecticide preferred.
A.MALATHION
B.LINDANE
C.PARIS GREEN
D.ZINC PHOSPHIDE

ZINC PHOSPHIDE is a rodenticide and is the preferred insecticide to be given during flood.

RODENTICIDES

- Rodenticides are of two main types - single-dose (acute) and multiple-dose (cumulative) .
- The former are lethal to the rat after a single feeding, while the latter require repeated feedings over a period of 3 more days
- An Expert Committee of the WHO grouped the "acute" rodenticides as below :

- Those requiring ordinary care
  - Red squill
  - Norbromide
  - Zinc phosphide
- Those requiring maximal precaution
  - Sodium fluorocetate
  - Fluoroacetamide
  - Strychnine
- Too dangerous for use
  - Arsenic trioxide
  - Phosphorus
  - Thallium sulphate
  - ANTU
  - Gophacide.

ZINC PHOSPHIDE

- Zinc phosphide is an efficient rodenticide.
- When moist, the chemical slowly gives off phosphine whose garlic odour is repellent to man and domestic animals, but seems to have no adverse effect on rats.
- Zinc phosphide is now extensively used in India.
- It is used in the ratio of 1 part to 10 parts of wheat or rice flour and mixed with a few drops of edible oil in order to render it more attractive to rats.
- Rats are killed in about three hours.
- The use of rubber gloves is recommended in handling zinc phosphide as it is highly poisonous.
- Special bait boxes have been designed for the administration of very toxic compounds such as zinc phosphide to eliminate the risk to man and domestic animals.
- Because of its good safety record, low cost and reasonably high effectiveness, Zinc phosphide is recommended for large scale use against rats

Reference: Preventive and Social Medicine, PARK pg 847

16. Malnutrition is a man made and has many ecological factors playing a role. Which of the following ecological factor has a conditioning effect?

A. Food habits

B. Infections

C. Rearing of child

D. Socioeconomic status

MALNUTRITION

- Malnutrition is truly a man-made disease.
- Malnutrition is like an iceberg; most people in the developing countries live under the burden of malnutrition.
- Pregnant women, nursing mothers and children are particularly vulnerable to the effects of malnutrition.
- The adverse effects of maternal malnutrition have been well documented-maternal depletion, low birth weight. anaemia, toxemias of pregnancy, postpartum haemorrhage, all leading to high mortality and morbidity.
- The ecological factors related to malnutrition are as follows-

1. SOCIO-ECONOMIC FACTORS:

a) Capital family income is very low
b)Lack of knowledge regarding food values
c) Bottle fed baby
d)Young and malnourished mother
e) Inadequate sanitary environment etc.

2. CONDITIONING INFLUENCES :

Infectious disease are an important conditioning factor responsible for malnutrition, diarrhoea ,measles, whooping cough, malaria, tuberculosis-all contribute to malnutrition.

3. SOCIO –CULTURAL FACTORS:

Religious trend- Hindus do not eat beef, and muslims .some orthodox, Hindus and Janis do not eat meat, fish, eggs and certain vegetables like onion.
Shortage of food:
a. less food production due to- - natural disaster e.g., flood ,storm
b. destruction of food due to lack of proper storage

Measures to improve the nutritional status of mothers and children may be broadly divided into direct and indirect nutrition interventions .

- Direct interventions - supplementary feeding programmes. distribution of iron and folic acid tablets, fortification and enrichment of foods, nutrition education, etc.
- Indirect nutrition interventions - measures such as control of communicable diseases through immunization, improvement of environmental sanitation, provision of clean drinking water, family planning, food hygiene, education and primary health care.

Reference: Preventive and Social Medicine, PARK pg 574

1. In operation theatre, a hernia surgery is going on. The sac is found medial to inferior epigastric artery. What is the hernia and what is the treatment for it?

A. Direct, Basini repair
B. Indirect, Basini repair
C. Direct, Lichtenstein
D. Indirect, Lichtenstein

INGUINAL HERNIA

- Most common hernia in men and women but much more common in men.
- Congenital inguinal hernia is known as indirect, oblique or lateral
- Acquired hernia is called direct or medial.
- Sliding hernia that is acquired but is lateral in position
- Occasionally, both lateral and medial hernias are present in the same patient (pantaloon hernia)

ANATOMICAL CLASSIFICATION

INDIRECT HERNIA

- It comes out through internal ring along with the cord.
- It is lateral to the inferior epigastric artery

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

INDIRECT INGUINAL HERNIA

- Can occur in any age from childhood to adult
- Occurs in a pre-existing sac
- Protrusion through the deep ring; herniation occurs later
- Pyriform/oval in shape; descends obliquely and downwards.
- Can become complete by descending down into the scrotum.
- Sac is anterolateral to the cord.
- Ring occlusion test does not show any impulse after occluding the deep ring.
- 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/strangulation are common.
- Sac should be opened during surgery

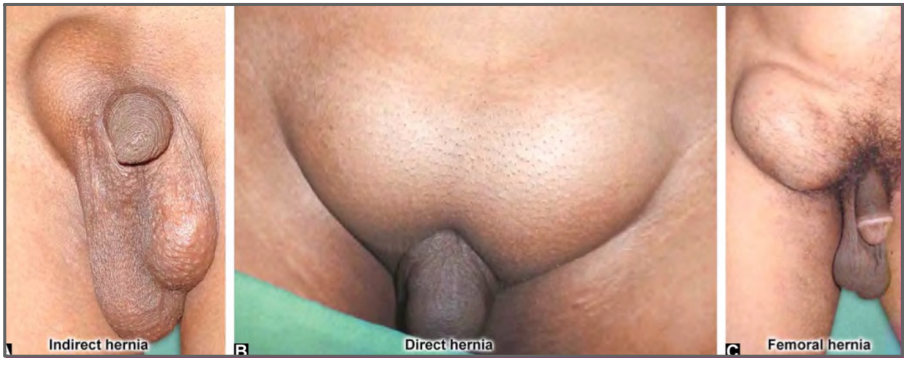
Reference :
Notespaedia Surgery High Yield Pg : 71
Bailey and Love's Short Practice of Surgery 27th Ed

DIRECT INGUINAL HERNIA

- Common in elderly
- Always acquired
- Herniation through posterior wall of the inguinal canal
- Globular/round in shape; descends directly forward bulge
- Descent down into the scrotum is rare
- Sac is posterior to the cord
- Test shows impulse even after occluding the deep ring
- Impulse is felt over the pulp of the little finger
- Test shows impulse on the middle finger
- Commonly bilateral
- Rare but can occur
- Sac is not necessarily opened unless obstruction is present

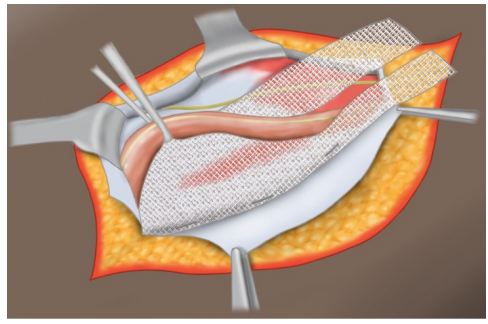
OPERATIONS FOR INGUINAL HERNIA

- Herniotomy
- Open suture repair
  - Bassini
  - Shouldice
  - Desarda
- Open flat mesh repair
  - Lichtenstein
- Open complex mesh repair
  - Plugs
  - Hernia systems
- Open preperitoneal repair
  - Stoppa
- Laparoscopic repair
  - TEP (Totally Extraperitoneal Repair Repair)
  - TAPP (Transabdominal Preperitoneal Mesh Repair)

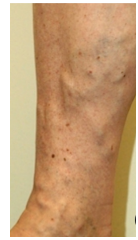


LICHTENSTEIN REPAIR

- Lichtenstein tension-free mesh inguinal hernia repair is a simple, safe, comfortable, effective method, with extremely low early and late morbidity and remarkably low recurrence rate and therefore it is our preferred method for hernia repair
- A Lichtenstein repair of a direct hernia is a tension-free mesh repair.
- The direct hernia is isolated from the cord structures, and then invaginated back into the abdominal cavity.
- The defect in the wall is then closed by suture, after which the mesh is fixed to bridge the defect.
- With large defects, an initial closure by suture allows better positioning of the mesh.
- The closure of the defect is not performed under tension, and should approximate only the transversalis fascia.
- The mesh is placed over the defect, and secured to the inguinal ligament and the internal oblique aponeurosis by running and interrupted sutures.
- The use of a mesh reinforces the back wall and is said to reduce postoperative pain and recurrence.



2. A patient with no complaints presents with dilated veins. What is the CEAP staging for the image shown below?



A. C2a
B. C2b
C. C3a
D. C3b

VARICOSE VEIN

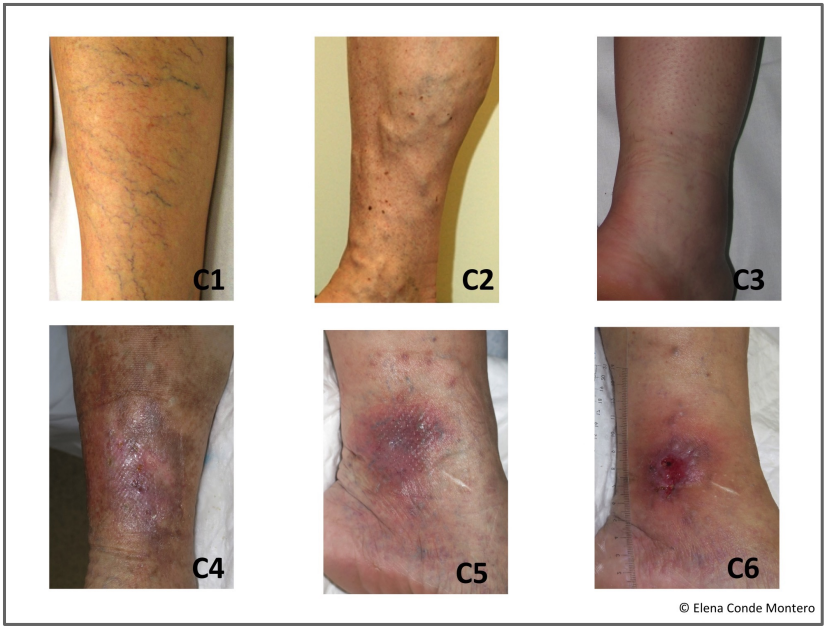
- Subcutaneous dilated vein 3mm in diameter or larger. They are frequently elongated and tortuous, with intermittent 'blowouts', but are defined by the presence of reflux and may be straight and uniform tubes morphologically.
- Aching, heaviness, throbbing, burning or bursting over affected areas, Itching
- Symptoms typically increase throughout the day or with prolonged standing, and are relieved by elevation or compression hosiery.
  - medial thigh and calf varicosities suggest GSV incompetence
  - posterolateral calf varicosities are suggestive of SSV incompetence
  - anterolateral thigh and calf varicosities may indicate isolated incompetence of the ASV

Reference :
Notespaedia Surgery High Yield Pg : 127
Bailey and Love's Short Practice of Surgery 27th Ed

CEAP CLASSIFICATION

Clinical Classification (C)		Etiologic Classification (E)	
C_0	No visible/palpable signs of venous disease	E_c	Congenital
		E_p	Primary
C_1	Telangiectasias or reticular veins	E_s	Secondary (postthrombotic)
C_2	Varicose veins	E_n	No venous etiology identified
C_3	Edema	**Anatomic Classification (A)**	
C_{4a}	Pigmentation and/or eczema	A_s	Superficial veins
		A_p	Perforator veins
C_{4b}	Lipodermatosclerosis and/or atrophy	A_d	Deep veins
C_5	Healed venous ulcer	A_n	No venous location identified
C_6	Open venous ulcer	**Pathophysiologic Classification (P)**	
		P_r	Reflux
	Subscript	P_o	Obstruction
A	Asymptomatic	$P_{r,o}$	Reflux and obstruction
S	Symptomatic	P_n	No venous pathophysiology identifiable

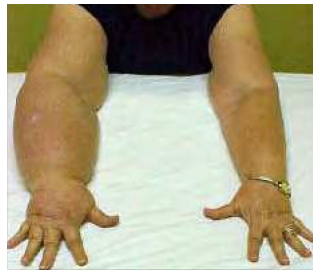
CEAP (Clinical-aEtiology-Anatomy-Pathophysiology) classification for chronic venous disorders



C2a - without symptoms
C2b - with symptoms

3. A patient underwent mastectomy for carcinoma breast with upper limb swelling after 3 months. What is the diagnosis?

A. Upper limb lymphangiosarcoma
B. Upper limb lymphedema
C. Wound infection
D. Seroma



Algorithm for management of operable breast cancer

- Achieve local control
- Appropriate surgery
  - Wide local excision (clear margins) and radiotherapy, *or*
  - Mastectomy ± radiotherapy (offer reconstruction – immediate or delayed)
  - Combined with axillary procedure (see text)
  - Await final pathology and receptor measurements
  - Use risk assessment tool; stage if appropriate
- Treat risk of systemic disease
  - Offer chemotherapy if prognostic factors poor; include Herceptin if Her-2 positive
  - Radiotherapy as decided above
  - Hormone therapy if oestrogen receptor or progesterone receptor positive

MASTECTOMY

- Mastectomy is indicated for large tumours (in relation to the size of the breast), central tumours beneath or involving the nipple, multifocal disease, local recurrence or patient preference.
- Radical Halsted mastectomy, which included excision of the breast, axillary lymph nodes and pectoralis major and minor muscles, is no longer indicated.
- Simple mastectomy involves removal of only the breast with no dissection of the axilla, except for the region of the axillary tail of the breast, which usually has attached to it a few nodes low in the anterior group.
- The modified radical (Patey) mastectomy
  - more commonly performed
  - excised mass is composed of the whole breast,a large portion of skin, the centre of which overlies the tumour but which always includes the nipple;all of the fat, fascia and lymph nodes of the axilla.
- Radiotherapy to the chest wall after mastectomy is indicated in selected patients in whom the risks of local recurrence are high.
- Tamoxifen has been the most widely used 'hormonal' treatment in breast cancer.
- Chemotherapy using a first-generation regime such as a 6-monthly cycle of cyclophosphamide, methotrexate and 5-fluorouracil (CMF) will achieve a 25% reduction in the risk of relapse over a 10–15-year period.

COMPLICATIONS OF MASTECTOMY

- Seroma
  - –MC complication,beneath skin flaps and axilla, occurs in 30% cases
  - –Catheter is retained until drainage is <30 ml/day
- Wound infection
  - –Majority are due to skin flap necrosis
- Lymphedema
  - –Occurs less frequently with the standard axillary dissections.
  - –Extensive LN dissection, radiation therapy, presence of positive LNs, obesity are predisposing factors.
- Injury to Long Thoracic (Motor) Nerve
  - –Seen in 10% of all cases.
  - –Result in a palsy of the Serratus anterior muscle (classical winged scapula)
- Injury to Thoracodorsal Nerve:
  - –Leads to palsy of the latissimus dorsi muscle.
- Redundant Axillary Fat Pad

LYMPHOEDEMA

- Lymphoedema of the arm is a troublesome complication of breast cancer treatment, fortunately seen less often now that radical axillary dissection and radiotherapy are rarely combined.
- However, it does still occur occasionally after either mode of treatment alone and appears at any time from months to years after treatment.
- There is usually no precipitating cause but recurrent tumour should be excluded because neoplastic infiltration of the axilla can cause arm swelling as a result of both lymphatic and venous blockage.
- This neoplastic infiltration is often painful because of brachial plexus nerve involvement.
- An oedematous limb is susceptible to bacterial infection following quite minor trauma and this requires vigorous antibiotic treatment.
- Antibiotics may need to be given for much longer than is normal and patients at risk of infection should have antibiotics readily available to enable treatment to be started promptly.
- Treatment of late oedema is difficult but limb elevation, elastic arm stockings and pneumatic compression devices can be useful.

LYMPHANGIOSARCOMA

- Rare complication of lymphoedema with an onset many years after the original treatment.
- It takes the form of multiple subcutaneous nodules in the upper limb and must be distinguished from recurrent carcinoma of the breast.
- The prognosis is poor but some cases respond to cytotoxic therapy or irradiation.

4. A 60 yr old female presents with increased bowel sounds. X-ray shows dilated bowel loops, with air in biliary tree. She gives history of open hysterectomy done 2 years back. What is the diagnosis?

A. Adhesions
B. Gall stone ileus
C. Mesenteric ischemia
D. Large bowel obstruction

EFFECTS AND COMPLICATIONS OF GALLSTONES

In gallbladder	In Bile duct	In Intestine
• Silent stones	• Obstructive jaundice	• Gallstone ileus
• Acute cholecystitis	• Cholangitis	
• Chronic cholecystitis	• Acute pancreatitis	
• Mucocele		
• Empyema		
• Perforation		
• Gangrene		
• Carcinoma		

GALLSTONE ILEUS

- Passage of a stone through a spontaneous biliary-enteric fistula leading to a mechanical bowel obstruction
- MC site of fistula: Between the gallbladder and duodenum
- 2nd MC site: Between gallbladder and transverse colon

Rigler's triad: The classic plain abdominal film triad of small bowel obstruction, pneumobilia, and ectopic gallstone is considered pathognomonic

Bouveret's Syndrome

- Duodenal obstruction due to gallstones, usually in the bulb is known as Bouveret's syndrome
- It is treated by duodenostomy or pyloroplasty

CLINICAL FEATURES

- Occurs most commonly in the elderly (>70 years)
- Nausea, vomiting, and abdominal pain, signs and symptoms of intestinal obstruction,
- A history of gallstonerelated symptoms may be present in only 50% of patients.
- Pain may be episodic and recurrent as the impacted stone temporarily obstructs the bowel lumen and then dislodges and moves distally, known as tumbling obstruction.
- The MC site of obstruction is the ileumQ (60%); followed by the jejunum (15%); stomach (15%); colon (5%, sigmoid colon); duodenum

Reference:Bailey & Love's short practice of surgery, pg 1199
Reference: Notespaedia Surgery high yield , pg 185

DIAGNOSIS

- Abdominal Xray: Evidence of an intestinal obstruction with pneumobilia or a calcified stone distant from the gallbladder.
- MC site of obstruction is the terminal ileum because of its narrow lumen.

TREATMENT

- It is a surgical emergency without a period of waiting in the hope that stone will pass
- In case of obstruction in the ileum calculus can be manipulated proximally to a healthy jejunum where a safe enterotomy and stone removal may be executed.
- Stable patients: Takedown of the biliary-enteric fistula and cholecystectomy during the same procedure is warranted because recurrent cholecystitis and cholangitis are common.
- Unstable patients or a significant inflammation in RUQ: Unstable to withstand a prolonged operative procedure, the fistula can be addressed at a second laparotomy

5. A 80yr old man with prostate lesion of 2x3 cm presented to the opd. PSA is 8. Gleason score is 6. What is the next management for him?

A. Radical prostatectomy
B. Active surveillance
C. Brachytherapy
D. Hormone therapy

PROSTATE CARCINOMA

- Most common malignant tumour in men over the age of 65 years.
- About 10–15% of younger men who develop prostate cancer have a positive family history of the disease.

TREATMENT OF CA PROSTATE

- T1a
  - Incidentally found tumors at TURP, by definition low volume (≤5%), usually well differentiated associated with very slow growth rate.
  - Managed by watchful waiting (Regular follow up with DRE and PSA)
- T1b, T1c and T2
  - Management depends on patient's age, life expectancy, performance status and patient's preference.
  - In younger, fitter men (<70 years): Radical prostatectomy or radiotherapy, if surgery is contraindicated
  - Elderly (>70 years) with life expectancy <10 years: Watchful waiting (Progress rate is very slow, 10% at 10 years)

Reference:Bailey & Love's short practice of surgery, pg 1469
Reference: Notespaedia Surgery high yield , pg 253

●Advanced disease (T3, T4 or any metastasis)

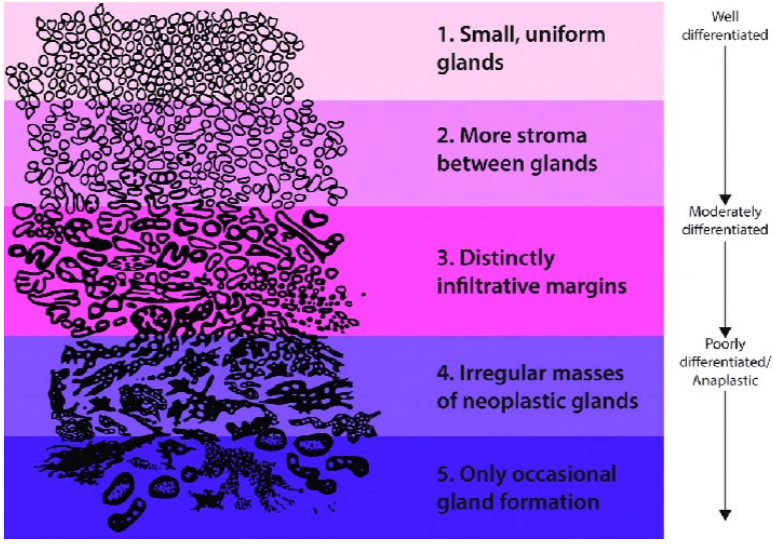
- Palliative treatment, androgen ablation or palliative radiotherapy
- Androgen ablation (Hormone therapy) is first line of treatment:
Orchiectomy + Flutamide or LHRH + Flutamide
- Palliative radiotherapy

GLEASON SCORE

Gleason score is the MC used histological grading system for prostate cancer.

- The two most predominant histological patterns of the prostate cancer are assigned a Gleason grade ranging from 1 – 5.
- Primary grade is assigned to the pattern of cancer that is most commonly observed in the histological slides of the specimen.
- Secondarygrade is assigned to the second most commonly observed pattern in the specimen.
- Gleason score is the sum of the two grades.Thus it is also known as Gleason sum.
- If the entire specimen has only one. pattern present, then both the primary and secondary grades are reported as the same grade.
- The Gleason grade ranges from 1 to 5, with 5 having the worst prognosis.
- The Gleason score ranges from 2 to 10.
- The Gleason score is used to help evaluate the prognosis of men with prostate cancer.

Together with other parameters, the Gleason score is incorporated into a strategy of prostate cancer staging which predicts prognosis and help guidetherapy.



PROSTATE SPECIFIC ANTIGEN (PSA)

- Normal value:≤4ng/mlQ(in>50years);
  Value > 20 ng/ml is diagnostic of CA prostate
- PSA is the single test with highest positive predictive value for CA prostate.
- PSA is prostate specific, not the cancer specific
- Level of PSA is directly related to tumor burden

6. What surgery is shown in the image?

A. Theirsch wiring
B. Altemier procedure
C. Hemorrhoidectomy
D. Well's operation

RECTAL PROLAPSE

- Mucous membrane and submucosa of the rectum protrude outside the anus for approximately 1–4 cm.
- It may be mucosal or full thickness (whole wall of the rectum is included)
- Commences as a rectal intussusception
- In children, the prolapse is usually mucosal and should be treated conservatively
- In the adult, the prolapse is often full thickness and is frequently associated with incontinence
- Surgery is necessary for full-thickness rectal prolapse

CLINICAL FEATURES

Children:

- Mucosal prolapse often commences after an attack of diarrhea, or from loss of weight and consequent loss of fat in the ischiorectal fossae.
- It may also be associated with fibrocystic disease, neurological causes and maldevelopment of the pelvis

Adults:

- Often associated with third-degree hemorrhoids.
- In the female a torn perineum, and in the male straining from urethral obstruction, predisposes to mucosal prolapse.
- In old age, both mucosal and full-thickness prolapse are associated with atony of the sphincter mechanism

DIAGNOSIS

- Before operative intervention, a careful history, physical examination, and colonoscopy should be performed.
- Manometry should be done in cases associated with incontinence

Reference:Bailey & Love's short practice of surgery, pg 1323

MANAGEMENT

ABDOMINAL PROCEDURES

- Considered the surgical procedures of choice for young and fit individuals
- Not suitable for elderly and infirm patients
- Are most likely to improve continence
- Have least recurrence rates
- Postoperative constipation is the MC side effect
- Abdominal Procedures
- Abdominal rectopexy
  - –Suture Rectopexy
  - –Mesh Rectopexy
    - –Posterior (Well's lvalon's)
    - –Anterior (Ripstein's)Q
    - –Lateral (Orr-Loygue)
    - –Ventral
  - –Resection Rectopexy (Frykman and Goldberg)
- Anterior resection

PERINEAL PROCEDURES

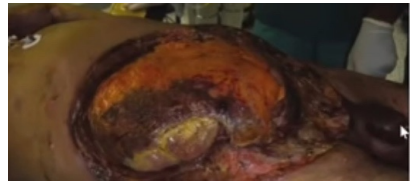
- Relatively minor procedures that may be performed under local or regional anaesthesia
- Well tolerated by elderly, frail and unfit patients
- Less likely to improve continence
- Recurrence rates varying from 5–35% higher than following abdominal rectopexy
- Postoperative constipation is infrequent
  - Delorme's muscosectomy
  - Thiersh and encirclement
  - Altemeier rectosigmoidectomy

THIERSCH PROCEDURE

- Thiersch procedure (anal encirclement) is performed frequently in patients with old age or high risks with rectal prolapse.
- It is a simple procedure using a suture or prosthesis that narrows the anus.
- When it was reported for the first time by Thiersch a silver wire was used as a prosthesis.
- Presently, because of ulcers and other complications, instead of wires, sutures and nylon, dacron, silastic, teflon and silicon rubber materials are used
- It has become largely obsolete owing to problems with chronic perineal sepsis, anal stenosis and obstructed defaecation.

7. A male patient presents as shown in the image. Which of the following is correct?

A. Orchidectomy done
B. Urinary diversion done
C. Mixed aerobic and anaerobic infection is seen
D. Anti Gas gangrene serum given



NECROTISING FASCITISIS

- Necrotising fascitisis is a rapidly spreading infection that produces necrosis of the subcutaneous tissues and overlying skin.
- It is caused by β-haemolytic streptococci and, occasionally, Staphylococcus aureus but may take the form of a polymicrobial infection associated with other aerobic and anaerobic pathogens, including Bacteroides, Clostridium, Proteus, Pseudomonas and Klebsiella.
- It is termed Fournier's gangrene when it affects the perineal area and Meleney's gangrene when it involves the abdominal wall.

FOURNIER'S GANGRENE

- Polymicrobial infection of the soft tissues of the perineum, external genitalia and perianal region.
- It is a form of necrotising fasciitis.
- There is rapid onset of gangrene leading to exposure of the scrotal contents.
- It can arise following minor injuries or procedures in the perineal area, such as a bruise, scratch, urethral dilatation, injection of haemorrhoids or opening of a periurethral abscess.
- Many patients have concurrent illnesses that diminish their defences, most notably diabetes mellitus and alcoholism.
- There is a mixed infection of aerobic and anaerobic bacteria in a fulminating inflammation of the subcutaneous tissues, which results in an obliterative arteritis of the arterioles to the scrotal skin that in turn results in gangrene.
- The condition can spread rapidly to involve the fascia and skin of the penis, perineum and abdominal wall

CLINICAL FEATURES

- There is sudden pain in the scrotum associated with prostration, pallor and pyrexia.
- Cellulitis spreads rapidly (within hours) with small necrotic areas of skin which, if untreated, coalesce to involve the entire scrotal and penile coverings, which may then slough, leaving the testes exposed but healthy.
- There may be crepitus and a foul-smelling exudate.

The patient typically becomes septic and severely unwell in a short period of time

Reference:Bailey & Love's short practice of surgery, pg 419

TREATMENT

- Fournier's gangrene requires early and aggressive treatment if the patient is to survive
- Treatment involves urgent surgical debridement of necrotic tissue in combination with early use of intravenous broad spectrum antibiotics

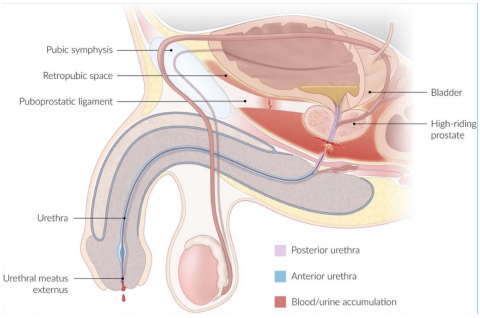
8. Mr Ramu faced an accident and had straddle injury and presents with blood at urethral meatus.What the next best step in management ?

A. Do SPC and drain the urine
B. Foley's catheter inserted
C. Do Retrograde urethrogram
D. CECT Pelvis is taken

- Patient with history of straddle injury and presenting with blood at urethral meatus most probably there is Rupture of Membranous Urethra

RUPTURE OF MEMBRANOUS URETHRA

- a.k.a Posterior Urethral Rupture, Prostatic Urethral Rupture
- Occurs in association with a fractured pelvis and may be associated with an extraperitoneal rupture of the bladder.
- Most common causes of pelvic fracture are road traffic accidents, severe crush injuries and falls.



CLINICAL FEATURES

- Blood in external meatus.
- Failure or difficulty in passing urine.
- Extravasation of urinetoscrotum,perineum and abdominal wall.
- Shock with pallor, tachycardia, hypotension.
- On PR examination, prostate may be felt high or may not be palpable at all. marked bruising of the pubic area, scrotum and penis.
- Vermooten's sign – Prostate is attached to pubis by puboprostatic ligament and disruption of puboprostatic ligament with complete rupture of urethra can lead to floating prostate.

INVESTIGATIONS

- X-ray pelvis to see for fracture.
- Ultrasound abdomen to see pelvis and other injuries.
- Urethrogram is done to see the site and type of tear

Reference :
Notespaedia Surgery High Yield Pg: 234
Bailey & Love's Short Practice of Surgery 27th Ed.

TREATMENT

- If patient is in shock and have associated injuries are treated.
- Suprapubic catheter should be inserted as soon as practicable using the Seldinger technique.
- In the presence of a coexisting extraperitoneal bladder injury :
  - no bladder will be apparent on ultrasound examination
  - surgical exploration, bladder repair, suprapubic catheter placement and drainage of the retropubic space is needed.
- In a patient with a pelvic fracture who does not have blood at the urethral meatus but who has not yet passed urine :
  - A single, gentle attempt at catheterization, by an experienced doctor,is permissible.
  - In adults a 16F soft, silicone catheter should be used.
- Delayed anastomotic urethroplasty is the preferred definitive management

A man was brought to the emergency department after he fell into a manhole and injured perineum.He gets an urge to micturate but is unable to pass urine.On examination,blood was seen at the tip of urethra and swelling of the penis and scrotum was seen.What is the site of injury ? [NEET '20]

9. What is the metabolic finding seen in a baby with Congenital Hypertrophic Pyloric Stenosis ?

A. Hyperchloremic Hypernatremic Metabolic Alkalosis
B. Hypochloremic Hyponatremic Metabolic Acidosis
C. Hypochloremic Hyponatremic Metabolic Alkalosis
D. Hyperchloremic Hyponatremic Metabolic Alkalosis

CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

- Males (especially firstborns) are affected approximately 4-6 times as often as females.
- Increased in infants with B and O blood groups.
- Occasionally associated with :
  - tracheoesophageal fistula and hypoplasia or
  - agenesis of the inferior labial frenulum.

ETIOLOGY

- Unknown
- Associated with :
  - eosinophilic gastroenteritis
  - Apert syndrome,
  - Zellweger syndrome,
  - trisomy 18
  - Smith-Lemli-Opitz syndrome, and Cornelia de Lange synd.
- Also associated with the use of erythromycin in neonates (highest risk if the medication is given within the 1st 2 wk of life.)
- Reduced nitric oxide might contribute to the pathogenesis of pyloric stenosis.

Reference :
Notespaedia Paediatrics High Yield Pg: 109
Nelson TEXTBOOK of PEDIATRICS 20th Ed Pg: 179

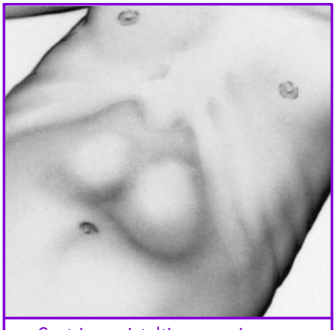
- Hyperchloremic , Hypokalemia metabolic Alkalosis with PARADOXICAL ACIDURIA occurs due to continuos vomiting
- Unconjugated hyperbilirubinemia is more common than conjugated and usually resolves with surgical correction.
- Approximately 5% of affected infants; mutations in the bilirubin uridine diphosphate glucuronosyl- transferase gene (UGT1A1)

CLINICAL MANIFESTATIONS

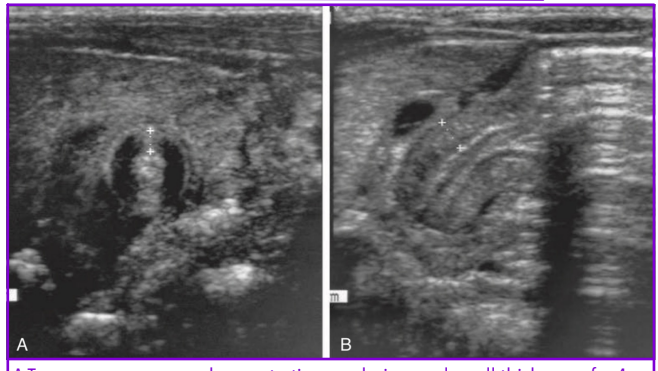
- Nonbilious vomiting is the initial symptom (may or may not be projectile, progressive, occurring immediately after a feeding.)
- Firm, movable, approximately 2 cm in length, olive shaped, hard mass(best palpated from the left side, and located above and to the right of the umbilicus in the midepigastrium beneath the liver's edge.) The olive is easiest palpated after an episode of vomiting.
- After feeding, there may be a visible gastric peristaltic wave that progresses across the abdomen.

INVESTIGATIONS

- USG Criteria for diagnosis include :
  - Pyloric thickness 3-4 mm
  - An overall pyloric length 15-19 mm
  - Pyloric diameter of 10-14 mm.
- Contrast studies :
  - i. String sign - demonstrate an elongated pyloric channel
  - ii. Shoulder sign - a bulge of the pyloric muscle into the antrum
  - iii. Double tract sign - parallel streaks of barium seen in the narrowed channel.



Gastric peristaltic wave in an infant with pyloric stenosis.



A.Transverse sonogram demonstrating a pyloric muscle wall thickness of >4 mm (distance between crosses).
B.Horizontal image demonstrating a pyloric channel length >14 mm (wall thickness outlined between crosses) in an infant with pyloric stenosis.

TREATMENT

- Correction of the alkalosis is essential to prevent postoperative apnea.
- The surgical procedure of choice is pyloromyotomy.
- Ramstedt procedure is performed through a short transverse skin incision.
  - feedings can be initiated within 12–24 hr after surgery and advanced to maintenance oral feedings within 36–48 hr after surgery.
- Endoscopic balloon dilation done in infants with persistent vomiting secondary to incomplete pyloromyotomy.
- Conservative management with nasoduodenal feedings is advisable in patients who are not good surgical candidates.
- Oral feeding is started at a volume of 10 mL formula, 6 times a day.
- The volume is increased day by day until patients tolerate 150 mL/kg/day unless vomiting occurs more than twice a day.

10. A 40yr old male presents to emergency with acute abdominal pain and distension.A fluid filled lesion is present in epigastric region.What is the abnormal investigation seen ?

A. Lipase
B. CGT
C. Bilirubin
D. CEA

- Acute abdominal pain and distension with fluid filled lesion present in epigastric region,most probable diagnosis is Acute Pancreatitis with pseudocyst.

ACUTE PANCREATITIS

- Inflammation of the pancreatic parenchyma.
- Defined as an acute condition presenting with abdominal pain, a threefold or greater rise in the serum levels of the pancreatic enzymes amylase or lipase, and/ or characteristic findings of pancreatic inflammation on contrast-enhanced CT.
- Premature activation of pancreatic enzymes within the pancreas, leading to a process of autodigestion.
- May be categorised :
  - mild (interstitial oedematous pancreatitis)
  - severe (necrotising pancreatitis).

CAUSES

- Gallstones
- Alcoholism
- Malnutrition
- Post ERCP
- Ampullary tumour
- Drugs (corticosteroids, azathioprine, asparaginase, valproic acid, thiazides, oestrogens)
- Hyperparathyroidism : Hypercalcaemia
- Pancreas divisum Autoimmune pancreatitis

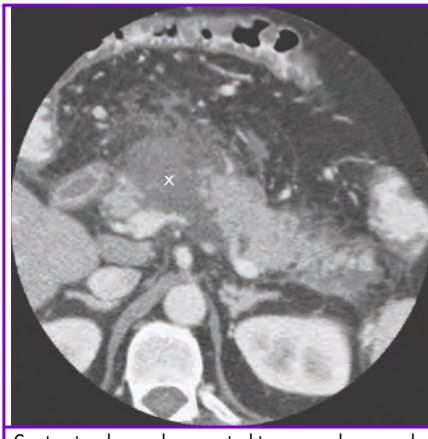
- Abdominal trauma
- Hereditary pancreatitis
- Viral infections (mumps, coxsackie B)
- Idiopathic

CLINICAL FEATURES

- Pain is the cardinal symptom.
  - Develops quickly, reaching maximum intensity within minutes rather than hours and persists for hours or even days.
  - Severe, constant and refractory to the usual doses of analgesics.
  - Usually experienced first in the epigastrium but may be localised to either upper quadrant or felt diffusely throughout the abdomen.
  - There is radiation to the back in about 50% of patients
  - Some patients may gain relief by sitting or leaning forwards.
- Nausea
- Repeated vomiting
- Hiccoughs
- Tachypnoea is common
- Tachycardia
- Hypotension may be present.
- Non-Pathognomic Signs :
  - Grey Turner's sign : bluish discolouration of the flanks
  - Cullen's sign : bluish discolouration of the umbilicus
- Distended abdomen - ascites with shifting dullness may be present

INVESTIGATIONS

- A serum amylase level three times above normal is indicative of the disease.
- Serum lipase level (provides a slightly more sensitive and specific test than amylase.)
- Contrast-enhanced CT is probably the best single imaging investigation
- Chest radiograph
- Cross-sectional MRI
- Serum Urea
- Serum Calcium
- Serum Albumin
- Blood Glucose



Contrast-enhanced computed tomography scan showing acute necrotising pancreatitis. Note the area of reduced enhancement in the pancreas (marked X), the peripancreatic oedema and stranding of the fatty tissues

The Ranson and Glasgow 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 score
On admission	**Within 48 hours**
Age >55 years	Age >55 years
White blood cell count >16 × 10^9/L	White blood cell count >15 × 10^9/L
Blood glucose >1.1 mmol/L (>200 mg/dL)	Blood glucose >10 mmol/L (no history of diabetes)
LDH >350 units/L	LDH > 600 units/L
AST >250 units/L	Serum urea >16 mmol/L (no response to intravenous fluids)
Within 48 hours	Arterial oxygen saturation (PaO_2) <8 kPa (60 mmHg)
Haematocrit fall of 10% or greater	Serum calcium <2.0 mmol/L
Blood urea nitrogen rise >5 mg/dL (1.8 mmol/L) despite fluids	Serum albumin <32 g/L
Arterial oxygen saturation (PaO_2) <8 kPa (60 mmHg)	
Serum calcium <2.0 mmol/L	
Base deficit >4 mmol/L	
Fluid sequestration >6 litres	

AST, aspartate aminotransferase; LDH, lactate dehydrogenase; PaO_2, arterial oxygen tension.

- A serum C-reactive protein level >150 mg/L at 48 hours after the onset of symptoms is also an indicator of severity.

ATLANTA CLASSIFICATION OF ACUTE PANCREATITIS

- Patients with acute pancreatitis be stratified into 3 groups :
  - i. Mild acute pancreatitis :
    - No organ failure
    - No local or systemic complications
  - ii. Moderately severe acute pancreatitis :
    - organ failure that resolves within 48 hours (transient organ failure); and/or
    - local or systemic complications without persistent organ failure.
  - iii. Severe acute pancreatitis :
    - Persistent organ failure (>48 hours);
    - Single organ failure;
    - Multiple organ failure.

MANAGEMENT OF ACUTE PANCREATITIS

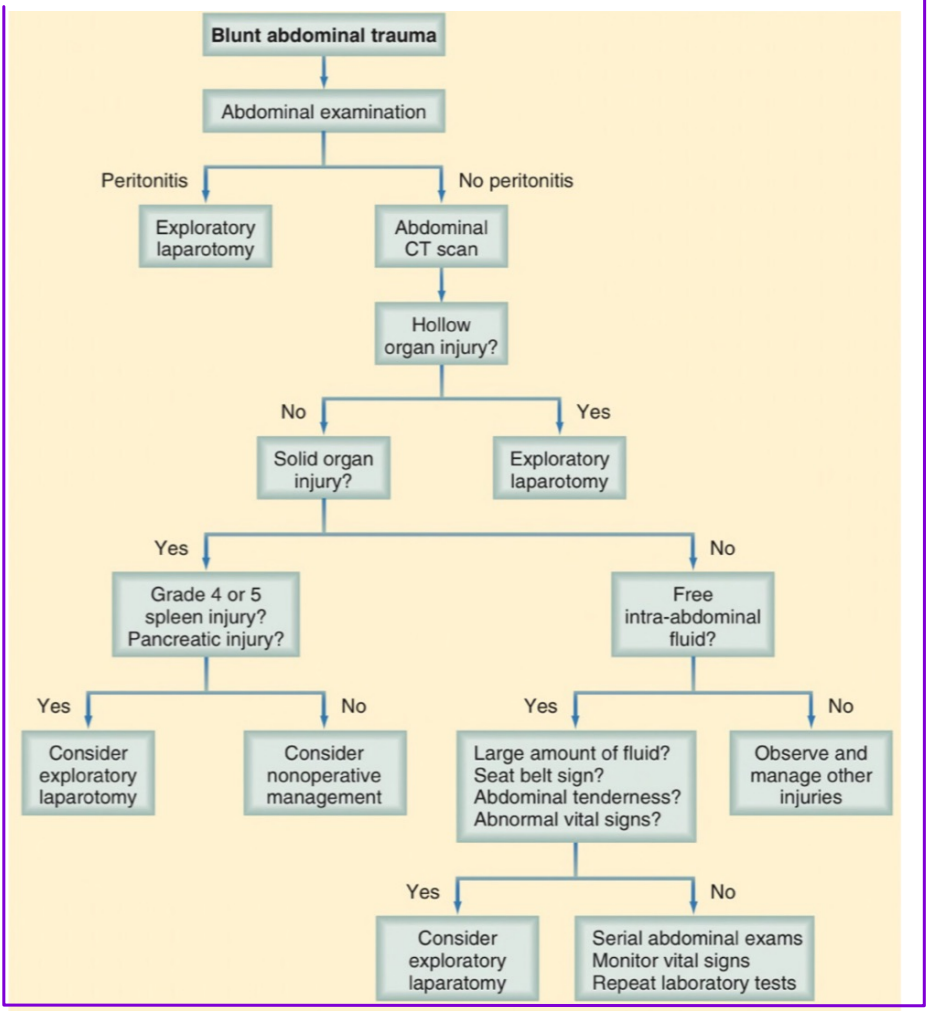
- Admission to ICU
- Analgesia
- Aggressive fluid rehydration
- Supplemental oxygen
- Invasive monitoring of vital signs, central venous pressure, urine output, blood gases
- Frequent monitoring of haematological and biochemical parameters (including liver and renal function, clotting, serum calcium, blood glucose)
- Nasogastric drainage (only initially)
- Antibiotics if cholangitis suspected; prophylactic antibiotics can be considered
- CT scan essential if organ failure, clinical deterioration or signs of sepsis develop

- ERCP within 72 hours for severe gallstone pancreatitis or signs of cholangitis
- Supportive therapy for organ failure if it develops (inotropes,ventilatory support,haemofiltration, etc.)
- If nutritional support is required, consider enteral (nasogastric) feeding

11. A patient with Blunt injury abdomen presents with flank ecchymoses.His vitals are stable.Which of the following investigation is best to localise the site of bleeding ?

A. CECT Abdomen
B. USG Abdomen
C. RGU
D. X Ray

ALGORITHM FOR THE EVALUATION AND MANAGEMENT OF BLUNT ABDOMINAL TRAUMA



Reference :
Notespaedia Surgery High Yield Pg: 37
Sabiston Textbook of Surgery 20th Ed.

12. A patient presents with vomiting and features of Gastric outlet obstruction. He has tumour infiltrating from stomach to pancreas into duodenum and having multiple liver mets in right lobe liver. What is the ideal treatment for him?

A. Palliative gastro jejunostomy
B. Right hepatectomy with radical gastrectomy
C. Whipple procedure
D. Palliative care

CARCINOMA PANCREAS

- MC type is pancreatic ductal adenocarcinoma (PDAC)
- More common in Men, African Americans, mean age at diagnosis is 72 years
- Overall, <5% of individuals will survive 5 years beyond their diagnosis.

CLINICAL FEATURES

- MC symptom for patients with PDACs in the periampullary region is jaundice.
- Pain typically arising in the epigastrium and radiating to the back.
- Weight loss affecting more than 50% of individuals.
- For tumors of the body and tail of the pancreas, pain and weight loss become more common at presentation.
- A palpable distended gallbladder in 1/3rd of patients with periampullary PDAC (Courvoisier Law)
- With widespread disease, a left supraclavicular node (Virchow's node) may be palpable. Periumbilical lymphadenopathy may be palpable (Sister Mary Joseph's node).
- In cases of peritoneal dissemination, perirectal tumor involvement may be palpable via digital rectal examination, referred to as Blumer's shelf.

DIAGNOSIS

- Tumor markers: CA19-9 (most sensitive) and CEA.
- MDCT is investigation of choice for the evaluation of lesions arising in the pancreas.
- For suspected periampullary pathology, a three-phase (noncontrast, arterial, and portal venous) CT scan with 3-mm slices and coronal and three-dimensional reconstruction should be routine.
- ERCP: Reserved for cases requiring therapeutic or palliative intervention
- Double duct sign on ERCP is highly suggestive of pancreatic head cancer
- EUS: For identifying lesions <2 cmQ that do not appear on CT scans

K-RAS2 oncogene is activated (by point mutation) in >95% of pancreatic cancers (MC gene mutation)

TREATMENT

- Surgical resection remains the only potentially curative treatment of pancreas cancer.
  - Tumors of head of the pancreas → Pylorus preserving pancreaticoduodenectomy or Longmire-Traverso procedure is preferred
  - Tumors of body and tail of the pancreas → Distal pancreatectomy and en-bloc splenectomy
- MC complication of pancreaticoduodenectomy is delayed gastric emptying

MC cause of death following pancreaticoduodenectomy is cardiopulmonary complications.
Most important predictor of post-operative survival is R0 resection.
Most important margin in pancreaticoduodenectomy is retroperitoneal or uncinate margin.

PALLIATIVE THERAPY FOR PANCREATIC CANCER

Biliary obstruction	• ERCP with metal stent placement (Best) • Roux-en-Y hepaticojejunostomy
Gastric outlet obstruction	• Endoscopic stenting (Preferred) • Double bypass (Roux-en-Y hepaticojejunostomy + gastrojejunostomy)
Pain	• NSAIDs or opiates • Celiac nerve block

13. A patient underwent cholecystectomy and presents to hospital with bile duct stones after 2 years . What is the name of such stones?

A. Primary stone
B. Secondary stone
C. Retained stone
D. Missed stone

CHOLEDOCHOLITHIASIS

- CBD stones are classified by point of origin
- Found in 6-12% of patients with GB stones
- Retained stones discovered within 2 years of cholecystectomy
- Recurrent stones detected >2 years following cholecystectomy

Recurrent stones are type of primary stones as it is formed within biliary tract

PRIMARY CBD STONE	SECONDARY CBD STONE
• Formed within the biliary tract • Associated with biliary Stasis and infection • More commonly seen in Asian populations • Soft, friable, light-brown stones or sludge in the common duct	• Formed initially in the GB • Migrate through the cystic duct into CBD • Most common bile duct stones in Western countries • Usually cholesterol stones

CLINICAL FEATURES

- CBD stones may be silent and are often discovered incidentally.
- In these patients, biliary obstruction is transient, and laboratory tests may be normal.
- Clinical features suspicious for biliary obstruction due to CBD stones include biliary colic, jaundice, clay colored stools, and darkening of the urine.
- Fever and chills may be present in patients with choledocholithiasis and cholangitis.
- Serum bilirubin (>3.0 mg/dL), aminotransferases, and ALP are commonly elevated in patients with biliary obstruction but are neither sensitive nor specific for the presence of common duct stones.
- Among these, serum bilirubin has the highest positive predictive value (28%–50%) for the presence of choledocholithiasis.
- Laboratory values may be normal in one third of patients with choledocholithiasis.

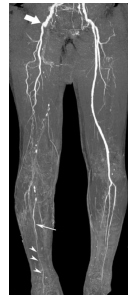
DIAGNOSIS

- USG: First test, can document GB stones and estimate the CBD diameter
- A dilated bile duct (>8 mm in diameter) in a patient with gallstones, jaundice and biliary pain is highly suggestive of choledocholithiasis.
- MRCP: Provides excellent anatomic detail, with sensitivity and specificity of 95% and 98%, respectively, for CBD stones.
- ERCP: Diagnostic and therapeutic test of choice for patients with suspected CBD stones.

TREATMENT

- Treatment options are ERCP, laparoscopic or open CBD Exploration.

14. A male smoker presents with Claudication pain right leg. Angiography is shown here. What is the next step in management?



A. Aorto femoral graft
B. Ileofemoral graft
C. Endovascular stenting
D. β-blockers and statins

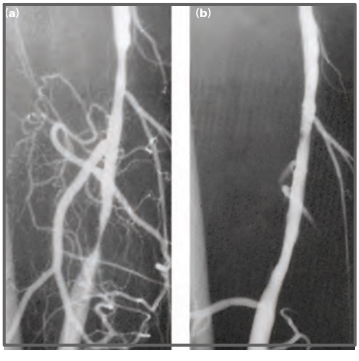
CLAUDICATION

- Claudication is often a marker of silent coronary arterial disease, whose extent correlates with the ABPI; a 0.1 decrease in ABPI below 0.9 is associated with a 10% increase in the relative risk of a major cardiovascular event.
- The common modifiable risk factors for peripheral arterial disease mirror those for coronary artery disease: smoking, diabetes mellitus, hypertension and hyperlipidaemia.
- Therefore, the two main aims when treating claudication are prevention of major cardiovascular morbidity through risk factor modification and symptom relief/improvement.
- **ANKLE–BRACHIAL PRESSURE INDEX (ABPI)**

  The ratio of systolic pressure at the ankle to that in the ipsilateral arm.
  The normal resting ABPI is 0.9–1.3; values below 0.9 indicate some degree of arterial obstruction (claudication), <0.5 suggests rest pain and <0.3 indicates imminent necrosis.

TRANSLUMINAL ANGIOPLASTY AND STENTING

- Arterial occlusive disease may be treated by inserting a balloon catheter into an artery and inflating it within a narrowed or blocked area.
- This technique is suitable for patients with claudication, rest pain or tissue necrosis.
- Following percutaneous femoral artery puncture under local anaesthetic, a guidewire is inserted and negotiated through the stenosis or occlusion under fluoroscopic control.
- A balloon catheter is then inserted over the guidewire and positioned within the lesion.
- The balloon is then inflated at high pressure for approximately 30 seconds and deflated.
- Satisfactory dilation of the lesion is confirmed by performing an angiogram.
- Percutaneous transluminal angioplasty (PTA) has proved very successful in dilating the iliac and femoropopliteal segments; the results below the knee are less successful.
- Long occlusions may be treated by the technique of subintimal angioplasty, where the guidewire crosses the lesion in the subintimal space (in the arterial wall) and a new lumen is created by inflation of the balloon.
- If the vessel fails to stay adequately dilated (often caused by elastic recoil of the artery), it may be possible to hold the lumen open using a metal stent



Narrowed superficial femoral artery before and after transluminal angioplasty. The advantage of this technique is that it can be carried out under local anaesthesia using the Seldinger technique of percutaneous arterial puncture, and is therefore especially useful in the treatment of patients who are medically unfit for major surgery.

Reference: Bailey & Love's short practice of surgery, pg 968

15. A patient presented with Road traffic accident,c/o breathlessness and decreased air entry into right lung and patient is hypotensive.Which among the following is the best step ?

A. Wide bore needle insertion followed by resuscitation
B. Needle inserted in the 5th ICS in Mid Axillary line
C. Fluid resuscitation
D. Chest Ray

- Based on given history most probable diagnosis is Tension Pneumothorax

TENSION PNEUMOTHORAX

- Develops when a 'oneway valve' air leak occurs either from the lung or through the chest wall.
- Mediastinum is displaced to the opposite side, decreasing venous return and compressing the opposite lung.

CAUSES

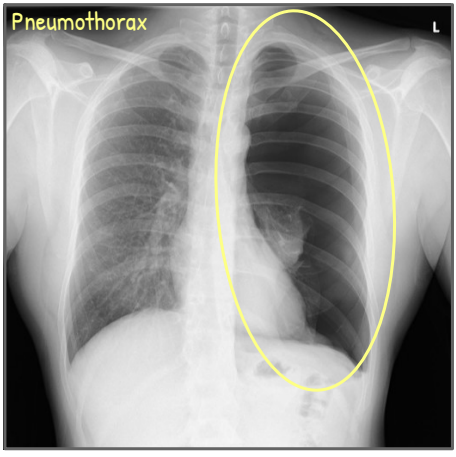
- Penetrating chest trauma,
- Blunt chest trauma with a parenchymal lung injury
- Iatrogenic lung injury (e.g. due to central venepuncture)
- Mechanical positive pressure ventilation.

CLINICAL PRESENTATION

- Patient increasingly restless with tachypnoea, dyspnoea and distended neck veins (similar to pericardial tamponade).
- On examination :
  - Tracheal deviation (a late finding)
- Hyperresonance and decreased or absent breath sounds over the affected hemithorax.

DIAGNOSIS

- Diagnosis is Clinical
- Chest X Ray findings :
  - Visible visceral pleural edge is seen as a very thin, sharp white line
  - No lung markings are seen peripheral to this line
  - Peripheral space is radiolucent compared to the adjacent lung
  - Lung may completely collapse
  - Mediastinum should not shift away from the pneumothorax unless a tension pneumothorax is present (discussed separately)
  subcutaneous emphysema and
  - Pneumomediastinum may also be present
- CT Chest



Reference :
Notespaedia Surgery High Yield Pg: 34
Bailey & Love's Short Practice of Surgery 27th Ed.

TREATMENT

- Immediate decompression, initially by rapid insertion of a largebore cannula into the second intercostal space in the midclavicular line of the affected side, then followed by insertion of a chest tube through the fifth intercostal space in the anterior axillary line.

1. You are doing the lumbar puncture.Which is the last structure to be encountered :

A. Ligamentum lavum

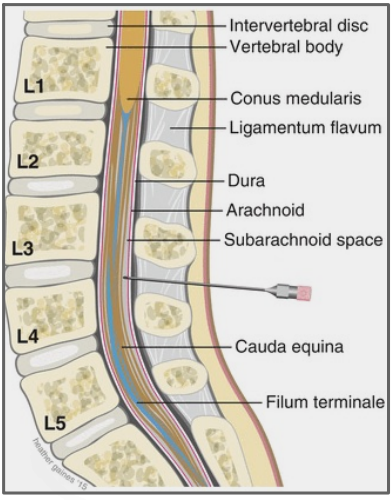
B. Dura

C. Arachnoid

D. Pia

LUMBRA PUNCTURE

- Lumbar puncture (LP) with examination of cerebrospinal fluid (CSF) is an important diagnostic tool for a variety of infectious and noninfectious neurologic conditions.

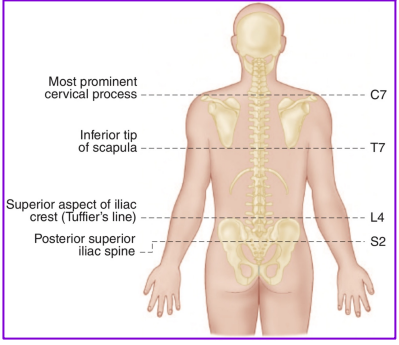


INDICATIONS

- Useful in the diagnosis of :
  - Bacterial, fungal, mycobacterial, and viral central nervous system (CNS) infections.
- Helpful in diagnosis of :
  - Subarachnoid hemorrhage (SAH),
  - CNS malignancies,
  - Demyelinating diseases, and
  - Guillain-Barré syndrome.

- The Lumbar Puncture needle pierces in order :
  - skin,
  - subcutaneous tissue,
  - supraspinous ligament,
  - interspinous ligament,
  - ligamentum flavum,
  - epidural space containing the internal vertebral venous plexus,
  - dura,
  - arachnoid, and
  - finally the subarachnoid space.

Surface landmarks for identifying spinal levels.



2. A patient is undergoing surgery where anesthesia patient developed severe hyperthermia and muscle rigidity.Which of the following agent would have also contributed to this condition ?

A. D-Tubucurare

B. Succcinylcholine

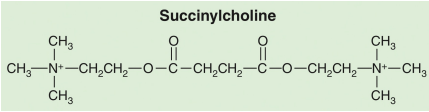
C. Cis-Atracurium

D. Rocuronium

- Based on the given symptoms it is suggestive of Malignant Hyperthermia

SUCCINYLCHOLINE

- also called Suxamethonium—consists of two joined ACh molecules



METABOLISM & EXCRETION

- Rapid onset of action (30–60 s) and short duration of action (typically less than 10 min)
- Most of it is rapidly metabolized by pseudocholinesterase into succinylmonocholine.
- Action can be prolonged by high doses,infusion of succinylcholine, or abnormal metabolism.
- The latter may result from hypothermia(decreases the rate of hydrolysis),reduced pseudocholinesterase levels, or a genetically aberrant enzyme.
- Reduced levels of pseudocholinesterase accompany pregnancy, liver disease, kidney failure, and certain drug therapies.

SIDE EFFECTS & CLINICAL CONSIDERATIONS

A. Cardiovascular :
- Stimulation of nicotinic receptors in parasympathetic and sympathetic ganglia, and muscarinic receptors in the sinoatrial node of the heart, can increase or decrease blood pressure and heart rate.

B. Fasciculations
C. Hyperkalemia
D. Muscle Pains
E. Intragastric Pressure Elevation
F. Intraocular Pressure Elevation
H. Malignant Hyperthermia
J. Prolonged Paralysis
K. Intracranial Pressure :
- Slight increases in cerebral blood flow and intracranial pressure in some patients.
- The increase in intracranial pressure can be attenuated by maintaining good airway control and instituting hyperventilation.

L. Histamine Release

Drugs known to trigger malignant hyperthermia :
i. Inhaled general anesthetics :
- Ether,Halothane,Methoxyflurane,Enflurane,Isoflurane, Desflurane,Sevoflurane

ii.Depolarizing muscle relaxant : Succinylcholine

Q.22yr old male was brought to casualty following RTA.On assessing GCS was 8/15. He needs to be intubated.Which among the following muscle relaxants is used ?
A.Mivacurium
B.Rocuronium
C.Suxamethonium
D.Gantacurium

Reference :
Notespaedia Anesthesia High Yield Pg : 29
Morgan &Mikhail Clinical Anesthesiology 6th Ed. Pg : 205

USES

- Rapid sequence intubation
- Emergency surgeries (with full stomach)
- Difficult intubation

CISATRACURIUM

- Stereoisomer of atracurium that is four times more potent.
- Undergoes degradation in plasma at physiological pH and temperature by organ-independent Hofmann elimination.
- The resulting metabolites (a monoquaternary acrylate and laudanosine) have no neuromuscular blocking effects.
- The amount of laudanosine produced for the same extent and duration of neuromuscular blockade is much less than with atracurium.
- Metabolism and elimination are independent of kidney or liver failure.
- Side Effects & Clinical Considerations :
  - Cisatracurium does not produce a consistent, dose-dependent increase in plasma histamine levels following administration.
  - Cisatracurium does not alter heart rate or blood pressure, nor does it produce autonomic effects, even at doses as high as eight times ED95.
  - Like in atracurium there is the production of laudanosine, pH and temperature sensitivity, and chemical incompatibility.

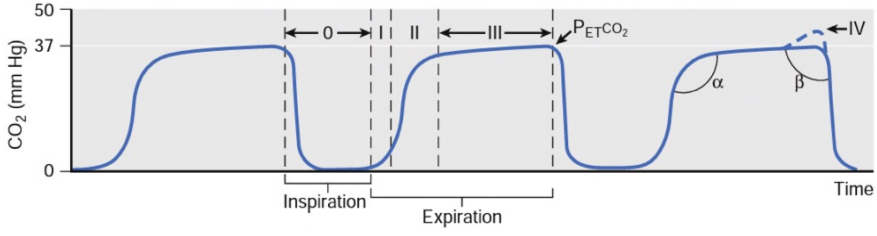
ROCURONIUM

- Monoquaternary steroid analogue of vecuronium.
- Undergoes no metabolism and is eliminated primarily by the liver and slightly by the kidneys.
- Duration of action is modestly prolonged by severe liver failure and pregnancy.
- Does not have active metabolites
- Elderly patients may experience a prolonged duration of action due to decreased liver mass
- Is less potent than most other steroidal muscle relaxants (potency seems to be inversely related to speed of onset).
- Side Effects & Clinical Considerations :
  i. Has an onset of action that approaches succinylcholine (60-90 s), making it a suitable alternative for rapid-sequence inductions
  ii. Sugammadex permits rapid reversal of dense rocuronium-induced NM blockade.
  iii. Rocuronium has been shown to be a rapid (90 s) and effective agent (decreased fasciculations and postoperative myalgias) for precurarization prior to administration of succinylcholine. It has slight vagolytic tendencies.

3. A 30yr old male was intubated for surgery.The best method to confirm the position of Endotracheal tube is ?

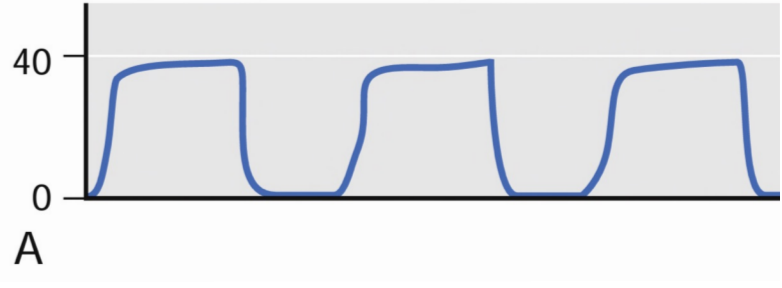
A. X Ray Chest
B. Auscultation
C. CO 2 level estimation
D. Chest Expansion

- In an incubated patient best method to confirm the position of Endotracheal tube is Capnography (refers not only to the method of CO2 measurement,but also to its graphic display as a function of time or volume).

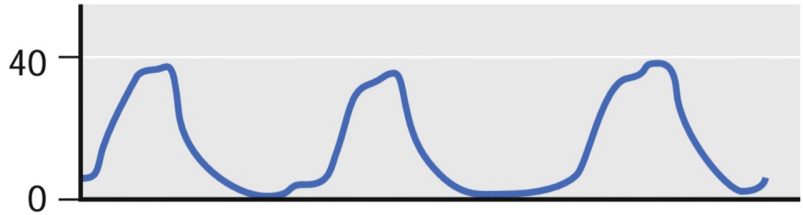


-Representative Time Capnogram for Three Breaths.
-The expiratory segment is divided into phases I, II, III, and IV.
α denotes the angle between phases II and III,
β denotes the angle between phase III and the descending limb of phase 0.
-Phase I corresponds to the exhalation of dead space gas from the central conducting airways or any equipment distal to the sampling site, which ideally should have no detectable CO2 (i.e., partial pressure of CO2, PCO2~0).
-During phase II, a sharp rise in PCO2 to a plateau indicates the sampling of transitional gas between the airways and alveoli.
- The plateau region of the capnogram, phase III, corresponds to the PCO2 in the alveolar compartment.
- Phase 0 : a sharp downstroke of PCO2 occurs as fresh inspired gas moves past the sampling site and washes out the remaining CO2.

TIME CAPNOGRAM

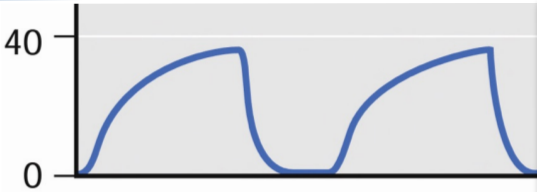


Normal capnogram during controlled mechanical ventilation



B

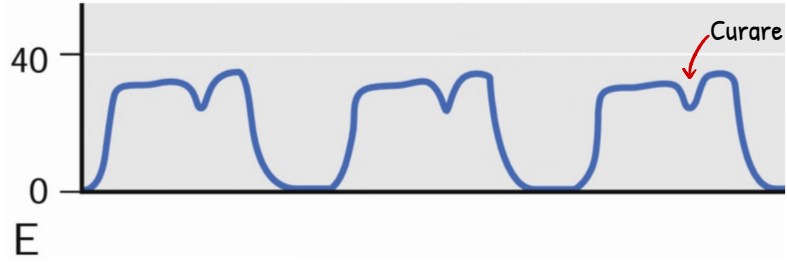
Normal capnogram during spontaneous breathing



C

While monitoring patient in OT room.You see similar capnogram as shown below on monitor.What could be the possible cause ?
A.Normal capnogram
B.Esophageal intubation
C.Bronchospasm
D.Spontaneous rebreathing

Increased upslope of phase III, as may occur during bronchospasm (asthma, chronic obstructive pulmonary disease), or partially obstructed endotracheal tube/breathing circuit.

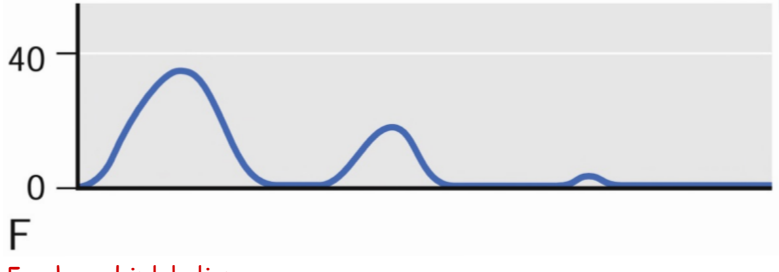


E

Clefts during phase III indicating spontaneous breathing efforts during controlled mechanical ventilation

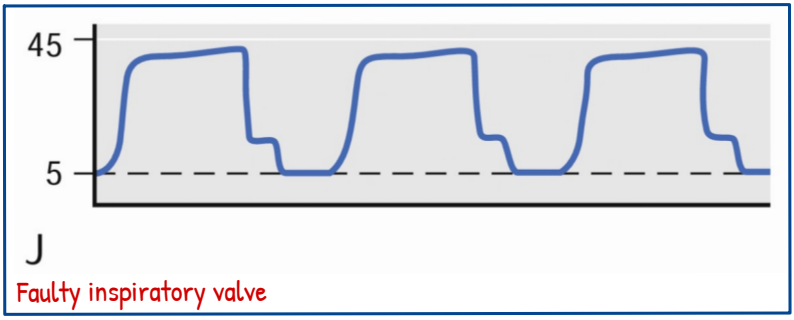
NEET '20

Capnograph shown below is due to ?



F

Esophageal intubation



Faulty inspiratory valve

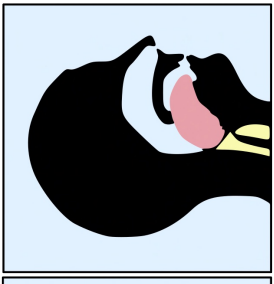
4. The image given below,done for airway management includes :

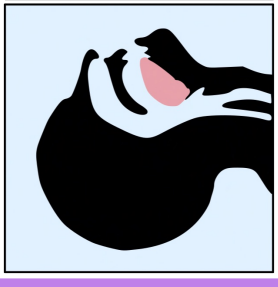
A. Chin lift
B. In line manual stabilisation
C. Jaw thrust
D. Head extension

- The given image shows Head tilt/Chin lift which is part of Triple manoeuver.

HEAD TILT/CHIN LIFT

- Is a procedure used to prevent the tongue obstructing the upper airways.
- The maneuver is performed by tilting the head backwards in unconscious patients, often by applying pressure to the forehead and the chin.
- The maneuver is used in any patient in whom cervical spine injury is not a concern
- This maneuver and the jaw-thrust maneuver are two of the main tools of basic airway management.
- If cervical spine injury is a concern and/or the patient is immobilized on a long spine board and/or with cervical collar; the jaw-thrust maneuver can be used instead.
- If the patient is in danger of aspirating; he or she should be placed in the recovery position or advanced airway management should be used.

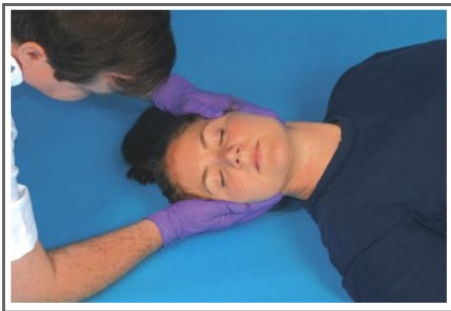




MANUAL IN LINE STABILISATION (MILS)

- Provides a degree of stability to the cervical spine prior to the application of a cervical collar.
- Should be used in conjunction with a cervical collar to assist continued spine management while :
  - Extricating or moving the patient
  - Performing a log roll
  - Transferring the patient to and from a stretcher.

Reference :
https://www.ambulance.qld.gov.au/docs/clinical/cpp/CPP_Manual%20inline%20stabilisation.pdf



1. Kneel behind the patient and place your hands firmly around the base of the skull on either side.

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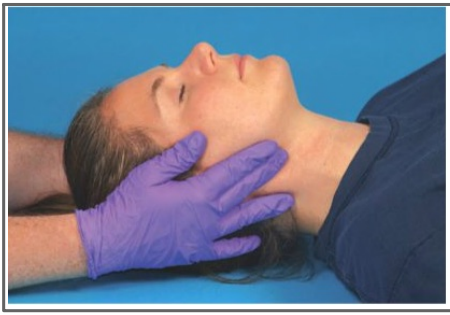
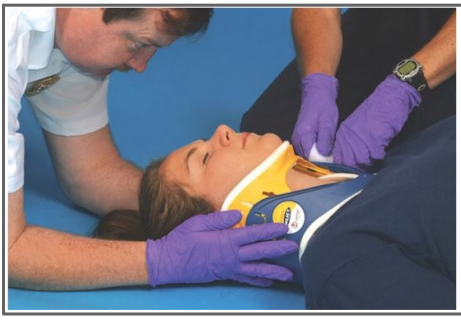


2. Support the lower jaw with your index and long fingers and the head with your palms. Gently lift the head into a neutral,eyes-forward position,aligned with the torso.Donot move the head or neck excessively,forcefully or rapidly.

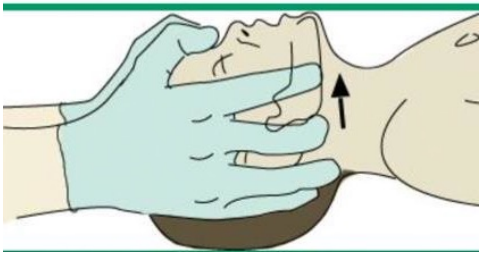
↓



3. Continue to support the head manually while your partner places a rigid cervical collor around the neck.Maintain manual support until you have completely secured the patient to a backboard.

JAW THRUST MANEUVER

- The rescuer uses two or three fingers of each hand to lift the jaw upward and outward so that the lower central incisors are anterior to the upper central incisors.
- In children with traumatic injuries,the cervical spine must be maintained in a neutral position during this maneuver



Reference :
https://www.researchgate.net/figure/Jaw-Thrust-maneuver_fig5_343793910

5. A young male was given regional anesthesia with 0.25% Bupivacaine.The patient became unresponsive and pulse became unrecordable.The best management would have been :

A. CPCR with 20% intralipid
B. CPCR with Sodabicarb
C. CPCR with Dobutamine
D. CPCR with Calcium

- Bupivacaine is highly cardiotoxic and it can manifest as Dysarrhythmia.
- Patient became unresponsive and pulse unrecordable suggests he is going into cardiac arrest.
- For the above case CPCR should be started and Intralipid is antidote for Bupivacaine.

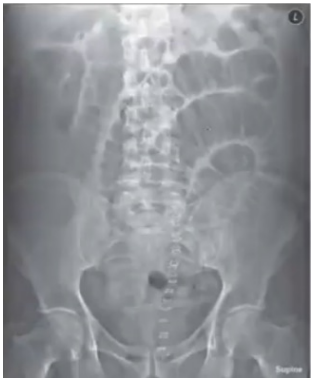
LIPID RESUSCITATION

- Intravenous infusions of lipid emulsions have become a standard treatment of local anesthetic systemic toxicity.
- The mechanism by which lipid is effective is not clear but is likely related to its ability to extract bupivacaine (or other lipophilic drugs) from aqueous plasma or tissue targets, thus reducing their effective free concentration ("lipid sink").
- According to the ASRA guidelines :
  - An intravenous bolus dose of lipid emulsion starts with a bolus of 1.5 mL/kg (100 mg in adults) followed by a continuous infusion at 0.25 mL/kg/min.
  - ASRA guidelines recommend additional modifications of standard advanced cardiac life support (ACLS) protocols, including avoidance of vasopressin, calcium channel blockers, β-adrenergic blockers, or other local anesthetics (lidocaine, amiodarone).

Radiology

1. A patient presents with abdominal pain and distension.On X ray abdomen we can see dilated bowel loops,these loops are. ?

A. Jejunum
B. Duodenum
C. Ileum
D. Transverse colon



The dilated bowel loops in the Abdomen X-ray are Jejunum

BOWEL OBSTRUCTION

Distiniguishing features of small and large bowel dilatation		
	Small bowel dilatation	**Large bowel dilatation**
Number of bowel loops	Usually numerous	Fewer loops
Distribution of bowel	Central abdomen	Peripheral abdomen
Size of bowel	Rarely > 5 cm	Often > 5 cm
Fold pattern	Valvulae conniventes: thin complete bands across the small bowel ▶ the folds are closer together than colonic haustra ▶ they are most prominent in the jejunum	Colonic haustra: these are usually thick incomplete bands ▶ they may be absent from the descending and sigmoid colon
Bowel contents	Fluid and gas	Faeces and gas

RADIOLOGICAL FEATURES OF OBSTRUCTION (ON PLAIN X-RAY)

- The obstructed small bowel is characterised by straight segments that are generally central and lie transversely.
- No/ minimal gas is seen in the colon
- Jejunum is characterised by its valvulae conniventes, which completely pass across the width of the bowel and are regularly spaced, giving a 'concertina' or ladder effect
- Ileum – the distal ileum has been piquantly described by Wangensteen as featureless
- Caecum – a distended caecum is shown by a rounded gas shadow in the right iliac fossa
- Large bowel, except for the caecum, shows haustral folds, which, unlike valvulae conniventes, are spaced irregularly, do not cross the whole diameter of the bowel and do not have indentations placed opposite one another

CLINICAL FEATURES

Depending on the level of obstruction, bowel obstruction can present with

- Abdominal pain,
- Swollen abdomen,
- Abdominal distension,
- Constipation.

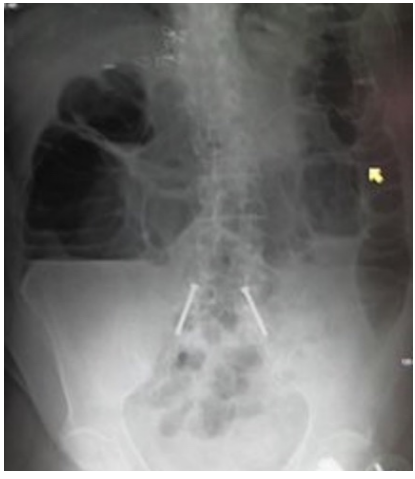
Bowel obstruction may be complicated by dehydration and electrolyte abnormalities due to vomiting; respiratory compromise from pressure on the diaphragm by a distended abdomen, or aspiration of vomitus; bowel ischemia or perforation from prolonged distension or pressure from a foreign body.

In small bowel obstruction,

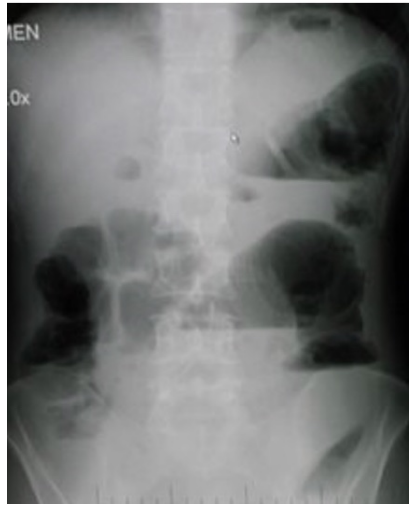
The pain tends to be colicky (cramping and intermittent) in nature, with spasms lasting a few minutes.
The pain tends to be central and mid-abdominal.
Vomiting may occur before constipation.

In large bowel obstruction,

The pain is felt lower in the abdomen and the spasms last longer.
Constipation occurs earlier and vomiting may be less prominent.
Proximal obstruction of the large bowel may present as small bowel obstruction.



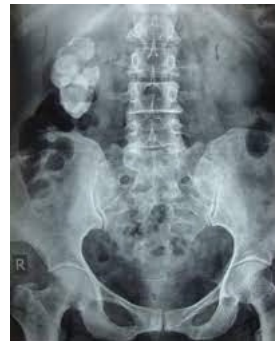
Upright abdominal X-ray of a person with a large bowel obstruction showing multiple air fluid levels and dilated loops of bowel.



Upright abdominal X-ray demonstrating a small bowel obstruction. Note multiple air fluid levels.

2. A 30yr old female present with dull aching pain in the back and sterile pyuria. Radiograph is shown.WHat is the most probable diagnosis ?

A.Nephrocalcinosis
B.Putty kidney
C.Staghorn calculus
D.Caldified Psoas Abscess



The Patient is having Renal TB and the image shows Putty Kidney

GENITOURINARY TUBERCULOSIS (GUTB)

- Accounts for 14% of non-pulmonary cases of TB.
- It is caused by dissemination of the organism through the bloodstream and is thus always secondary TB.
- There is either reinfection or reactivation of old TB.

GUTB is a cause of sterile pyuria

- CT is the most sensitive modality for visualising renal calcifications and CT urography is more sensitive at identifying all manifestations of renal tuberculosis:

EARLY:

- Papillary necrosis (single or multiple) resulting in uneven caliectasis.

PROGRESSIVE:

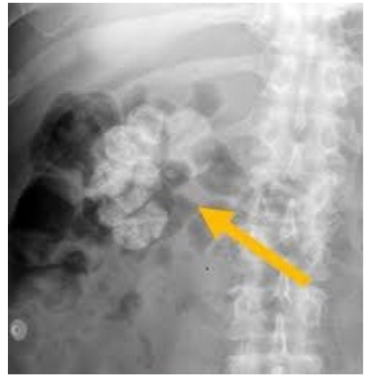
Multifocal strictures can affect any part of the collecting system;

- generalised or focal hydronephrosis;
- mural thickening and enhancement;
- poorly enhancing renal parenchyma, either due to direct involvement or due to hydronephrosis.

END-STAGE:

- Progressive hydronephrosis results in very thin parenchyma, mimicking multiple thin-walled cysts;
- Amorphous dystrophic calcification eventually involves the entire kidney

(known as PUTTY KIDNEY).



A putty kidney refers to a pattern of renal calcification associated with renal tuberculosis conventionally described on plain radiography. The calcification is characteristically very homogeneous and ground glass-like, representing calcified caseous tissue

Reference:Grainger & Allison's Diagnostic Radiology
Reference: Notespaedia Radiology image bank pg 126

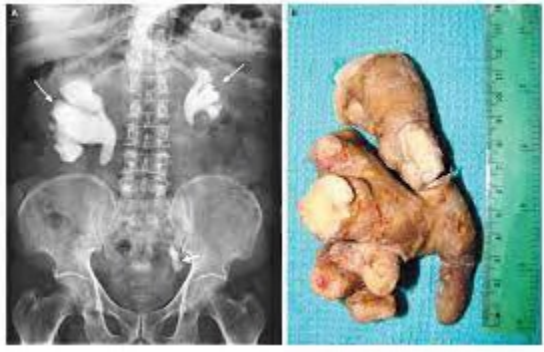
STAGHORN CALCULI

- Coral calculi, are renal calculi that obtain their characteristic shape by forming a cast of the renal pelvis and calyces, thus resembling the horns of a stag.
- They refer to struvite calculi involving the renal pelvis and extending into at least two calyces
- The majority of staghorn calculi are symptomatic, presenting with fever, hematuria, flank pain and potentially septicemia and abscess formation.
- **PLAIN RADIOGRAPH**

  The vast majority of staghorn calculi are radiopaque and appear as branching calcific densities overlying the renal outline and may mimic an excretory phase intravenous pyelogram. Lamination within the stone is common.
- **ULTRASOUND**

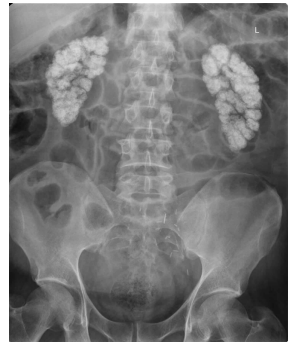
  The collecting system is filled with a densely calcified mass, producing marked posterior acoustic shadowing.
- **CT**

  Staghorn calculi are radiopaque and conform to the renal pelvis and calyces, which are often to some degree dilated. When viewed on bone windows they have a laminated appearance, due to alternating bands of magnesium ammonium phosphate and calcium phosphate



NEPHROCALCINOSIS

- Anderson-Carr kidney or Albright calcinosis, refers to the deposition of calcium salts in the parenchyma of the kidney.
- It may cause acute kidney injury. It is now more commonly used to describe diffuse, fine, renal parenchymal calcification in radiology.
- During its early stages, nephrocalcinosis is visible on x-ray, and appears as a fine granular mottling over the renal outlines.
- It may be severe enough to cause (as well as be caused by) renal tubular acidosis or even end stage kidney disease, due to disruption of the kidney tissue by the deposited calcium.



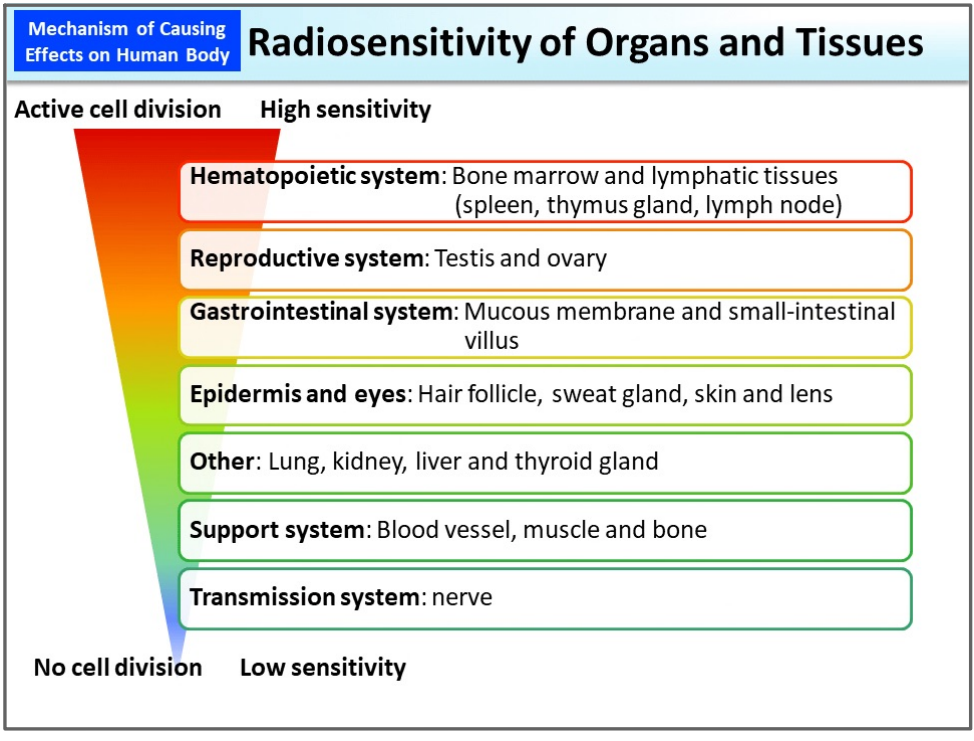
3. A child is suffering from Acute lymphoblastic leukaemia. He undergoes prophylactic cranial irradiation as preparation for Bone Marrow Transplant for treatment of ALL. Which of the following will be least affected by radiation exposure?

A. Spermatogonia
B. Intestinal Epithelial Cells
C. Neurons in Brain
D. Bone marrow

The neurons in the brain are the least affected by radiation exposure.

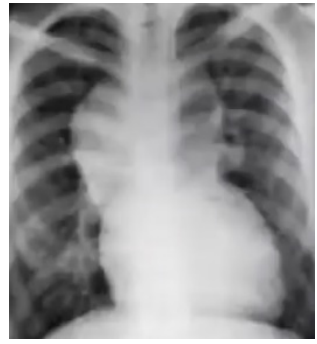
RADIOSENSITIVITY

- The law of Bergonie and Tribondeau is that the radiosensitivity of a biological tissue is directly proportional to the mitotic activity and inversely proportional to the degree of differentiation of its cells.
- Usually neoplastic cells are more radiosensitive than the cells from which they originate; in fact, they reproduce much faster than healthy cells.
- Cellular differentiation describes the extent to which a tumor resembles the normal tissue from which it derives. The more it differs from healthy tissue, the greater its radiosensitivity will be.



4. A 12 year old child presents with cyanosis.Based on given chest X ray finding. What is the most probable diagnosis ?

A. Truncus Arteriosus
B. Supracardiac TAPVC
C. TGA
D. TOF



The X-ray shows snowman sign seen in Supracardiac TAPVC

TOTAL ANOMALOUS PULMONARY VENOUS RETURN

- All the pulmonary veins instead of joining the left atrium are connected anomalously to result in the total pulmonary venous blood reaching the right atrium.
- **CLASSIFICATION** :
  - i. supracardia - most common
  - ii.cardia
  - iii.infracardia
  - iv.mixed varieties.
- Obstructed TAPVC is a pediatric cardiac surgical emergency because prostaglandin therapy is usually not effective.
- Non-obstructive TAPVC is commoner than the obstructive type.

CLINICAL MANIFESTATIONS

- severe obstruction to pulmonary venous return :
  - most prevalent in the infracardiac group
  - present with severe cyanosis and respiratory distress.
  - Murmurs may not be present.
  - Infants are severely ill and fail to respond to mechanical ventilation.
- mild or no obstruction to pulmonary venous return :
  - characterized by the development of heart failure as the pulmonary vascular resistance falls, with mild to moderate degrees of desaturation.
  - Systolic murmurs may be audible along the left sternal border, and a gallop rhythm may be present.

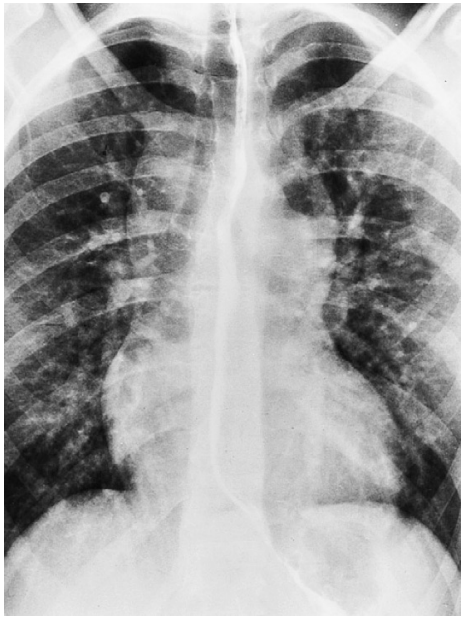
INVESTIGATIONS

ECG : right ventricular hypertrophy (usually a qR pattern in V3R and V1, and the P waves are frequently tall and spiked).

Chest X Ray :

- In neonates perihilar pattern of pulmonary edema and a small heart.
- In older children, if the anomalous pulmonary veins enter the innominate vein and persistent left superior vena cava, a large supracardiac shadow can be seen, which together with the normal cardiac shadow forms a "snowman" appearance.

On ECHO : a large right ventricle and usually identifies the pattern of abnormal pulmonary venous connections



CXR IN SUPRACARDIAC TYPE OF TAPVD.

The pulmonary vessels are large, consistent with increased pulmonary blood flow.

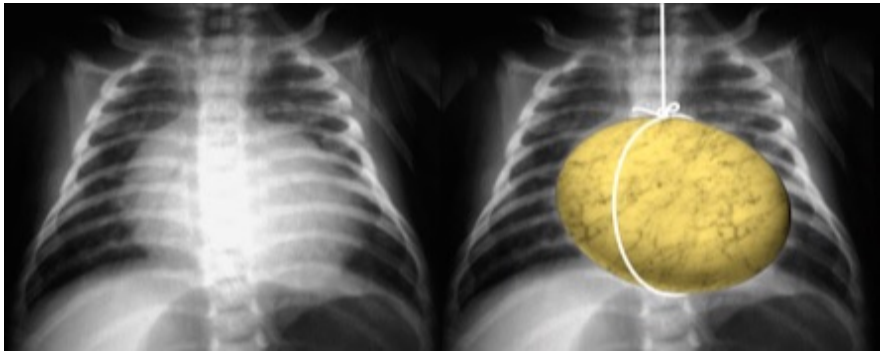
The abnormal mediastinum (the 'snowman') is caused by the dilated right superior vena cava on the right side and by the anomalous vertical vein draining both lungs on the left side.

TREATMENT

- Surgical correction of TAPVR is indicated during infancy, with emergency repair performed for those patients with venous obstruction.
- If surgery can't be performed then extracorporeal membrane oxygenation may be required to maintain oxygenation.
- Those with aggressive veno-occlusive disease, heart-lung transplantation may be the only option

TRANSPOSITION OF THE GREAT ARTERIES

A ventriculo-arterial discordance with an anterior aorta arising from the anterior right ventricle and the pulmonary artery arising from the posterior left ventricle



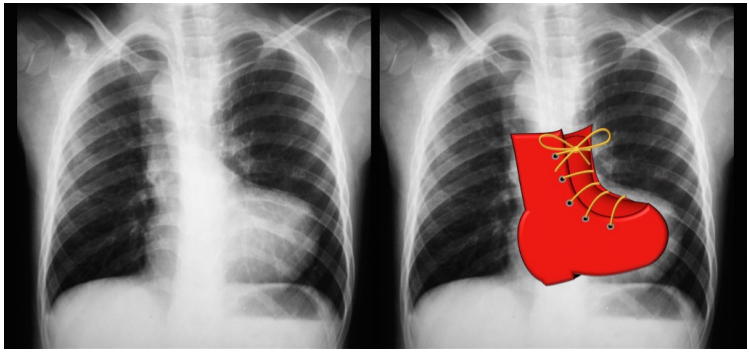
An 'egg on side' appearance: this is as a result of a narrow superior mediastinum (the main pulmonary artery is located directly behind the aorta) together with a slightly enlarged, rounded heart

TETRALOGY OF FALLOT

- It is the most common congenital cyanotic heart disease in adults.
  - VSD (almost always large)
  - Pulmonary stenosis
  - Right ventricular hypertrophy
  - Over riding aorta

INVESTIGATIONS

CXR

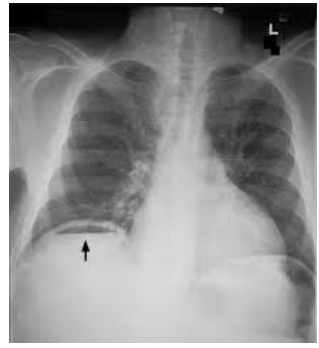


- 'Boot'-shaped heart: this appearance is partly as a result of a concavity in the left heart border (hypoplastic main pulmonary artery) and an upward prominence of the cardiac apex (enlarged right ventricle)
- Pulmonary oligaemia

TRANSTHORACIC ECHOCARDIOGRAPHY: The investigation of choice

5. A 35 year old male presented to the ED with fever and acute pain in the abdomen for past 4 days. X ray abdomen is given below. What is the most likely diagnosis?

A. Hollow viscus perforation
B. Liver abscess
C. Thoracic Empyema
D. Gastric volvulus



The given X-ray shows Air under the diaphragm

PNEUMOPERITONEUM

- Pneumoperitoneum is the presence of air or gas in the abdominal (peritoneal) cavity.
- The most common cause of a pneumoperitoneum is a perforation/disruption of the wall of a hollow viscus.

RADIOLOGICAL FEATURES

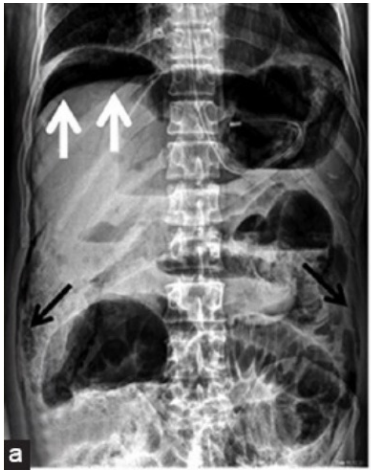
An erect chest x-ray is the most sensitive plain radiograph for the detection of free intraperitoneal gas. On chest x-ray, any subdiaphragmatic free gas can be seen

- Erect CXR
  Gas is seen under the diaphragm (this can detect as little as 1ml of free gas)
- Left lateral decubitus AXR
  Gas is seen between the liver and abdominal wall
- Supine AXR
  - Gas is seen within the RUQ- particularly within the subhepatic space and hepatorenalfossa (Morrison'spouch)
  - Triangular collections of air are seen within the abdomen (outlining the visceral contents)
  - Gas is seen on either side of the falciform ligament
  - Scrotal air can be seen in children
- CT(lung window settings)
  This is the most sensitive technique for detecting small amounts of calcium free gas(look anterior to the liver, anteriorly within the central abdomen, and within the peritoneal recesses)

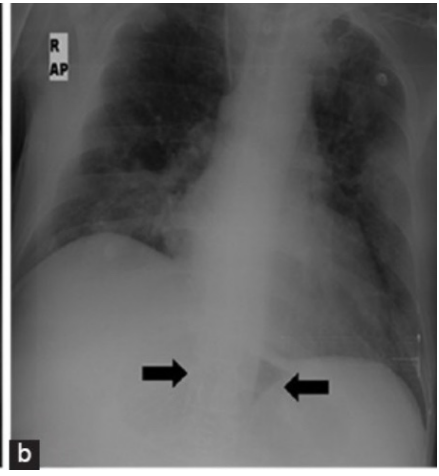
RADIOLOGICAL SIGNS

- Cupola/saddlebag/mustache sign – Seen on a supine radiograph, refers to air accumulation underneath the central tendon of the diaphragm in the midline.
- Rigler's sign – Air outlining both sides of the bowel wall.
- Lucent liver sign - Reduction of liver opacity due to air located anterior to the liver
- Football sign – Seen in massive pneumoperitoneum, where the abdominal cavity is outlined by gas.
- Silver's sign – Also called a falciform ligament sign, where air outlines the falciform ligament.

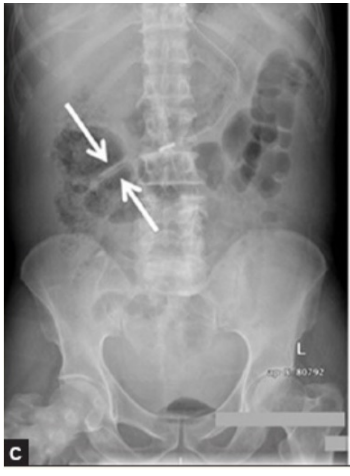
- Inverted V sign – Air outlining lateral umbilical ligaments (inferior epigastric vessels).
- Doge's cap sign – Triangular collection of gas in Morison pouch.
- Telltale triangle sign – Triangular air pocket between three loops of bowel.
- Urachus sign – Outline of middle umbilical ligament.



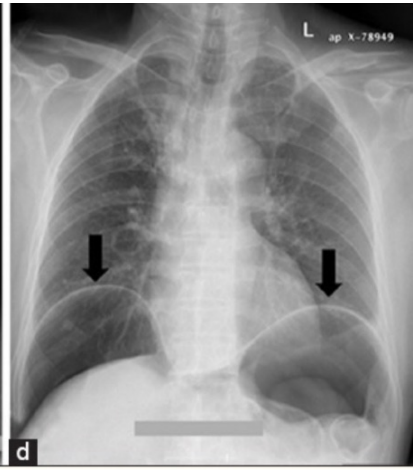
(a) X-ray of abdomen, erect view, showing subdiaphragmatic free air (white arrows), air outlining the properitoneal fat stripe (black arrows)



(b) Cupola sign (arrowheads)



(c) Rigler's sign (arrow)



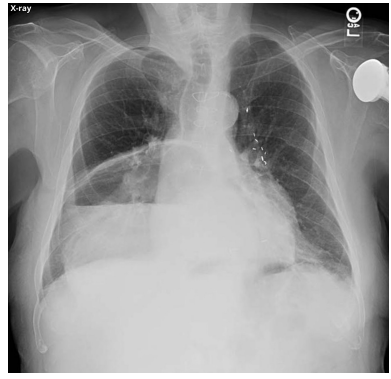
(d) Chest radiograph showing free air under the diaphragms (arrowhead)

GASTRIC VOLVULUS

- Specific type of volvulus that occurs when the stomach twists on its mesentery. It should be at least 180° and cause bowel obstruction to be called gastric volvulus.
- Organo-axial volvulus is more common in adults, responsible for 60% of presentations. Mesentero-axial volvulus is more common in children.

Patients may present with the classic triad of Borchardt:

- severe sudden epigastric pain
- intractable retching without vomiting
- inability to pass a nasogastric tube



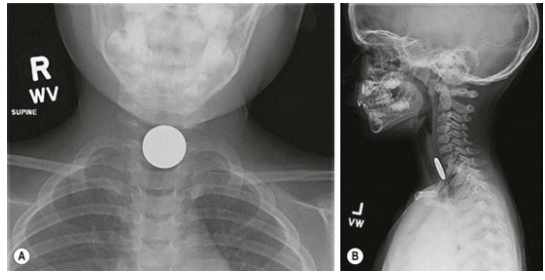
Organo-axial volvulus

RADIOGRAPHIC FEATURES

- Chest radiograph
  intrathoracic; upside-down stomach
  mediastinal or retrocardiac air-fluid level
- Abdominal radiograph;
  when performed with the patient upright
  - unexpected location of the gastric bubble
  - double air-fluid level
  - large, distended stomach
  - collapsed small bowel

6. A 9 year old child was brought to emergency by the mother with difficulty in swallowing for past few hours. He was noted to play unsupervised. X-ray was taken . Identify the location of foreign body.

A. Oesophagus
B. Trachea
C. Soft tissue of neck
D. Artifact of X-ray



The given X-ray shows foreign body in oesophagus

FOREIGN BODY INGESTION AND ASPIRATION

- Most aerodigestive foreign bodies are esophageal—85%
- The highest incidence occurs between 1 and 3 years of age
- Most esophageal FB impact in cervical esophagus just below cricopharyngeus muscle
- Another 4% to 5% of esophageal foreign bodies become lodged at midesophagus
- distal esophagus often caused by extraluminal compression --aortic arch or left main bronchus

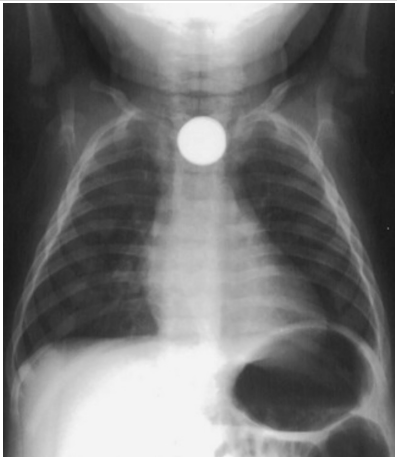
CLINICAL FEATURES

OESOPHAGEAL FOREIGN BODY

- Vomiting, odynophagia, dysphagia, ptyalism
- A large FB may cause symptoms of airway obstruction and cough (compression or irritation of upper airway)
- In long-standing impaction, fever and other symptoms of respiratory infection may be present

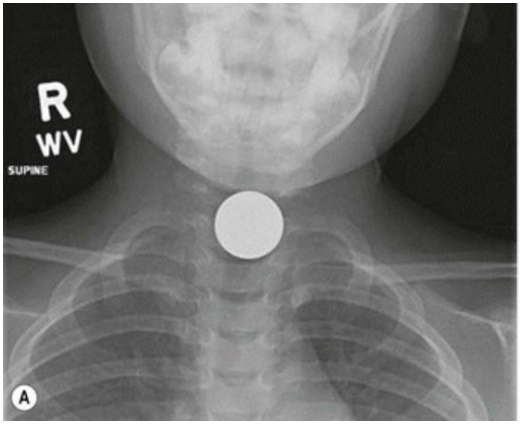
TRACHEAL FOREIGN BODY

- Asthmatoid wheeze, audible slap and palpable thud

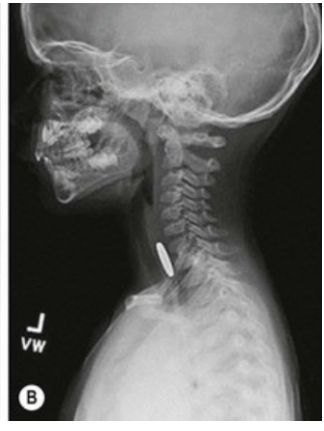


Posteroanterior radiograph of an esophageal foreign body (coin) lodged at the level of the cricopharyngeus muscle. This is the most common area of the esophagus to harbor a coin in children. Coins remaining in the upper tract are usually removed unless there is steady progression with observation. This coin would probably be symptomatic in an infant and cause respiratory distress, drooling, wheezing, and perhaps stridor. Note: The chance of spontaneous passage is about 20% to 25%.

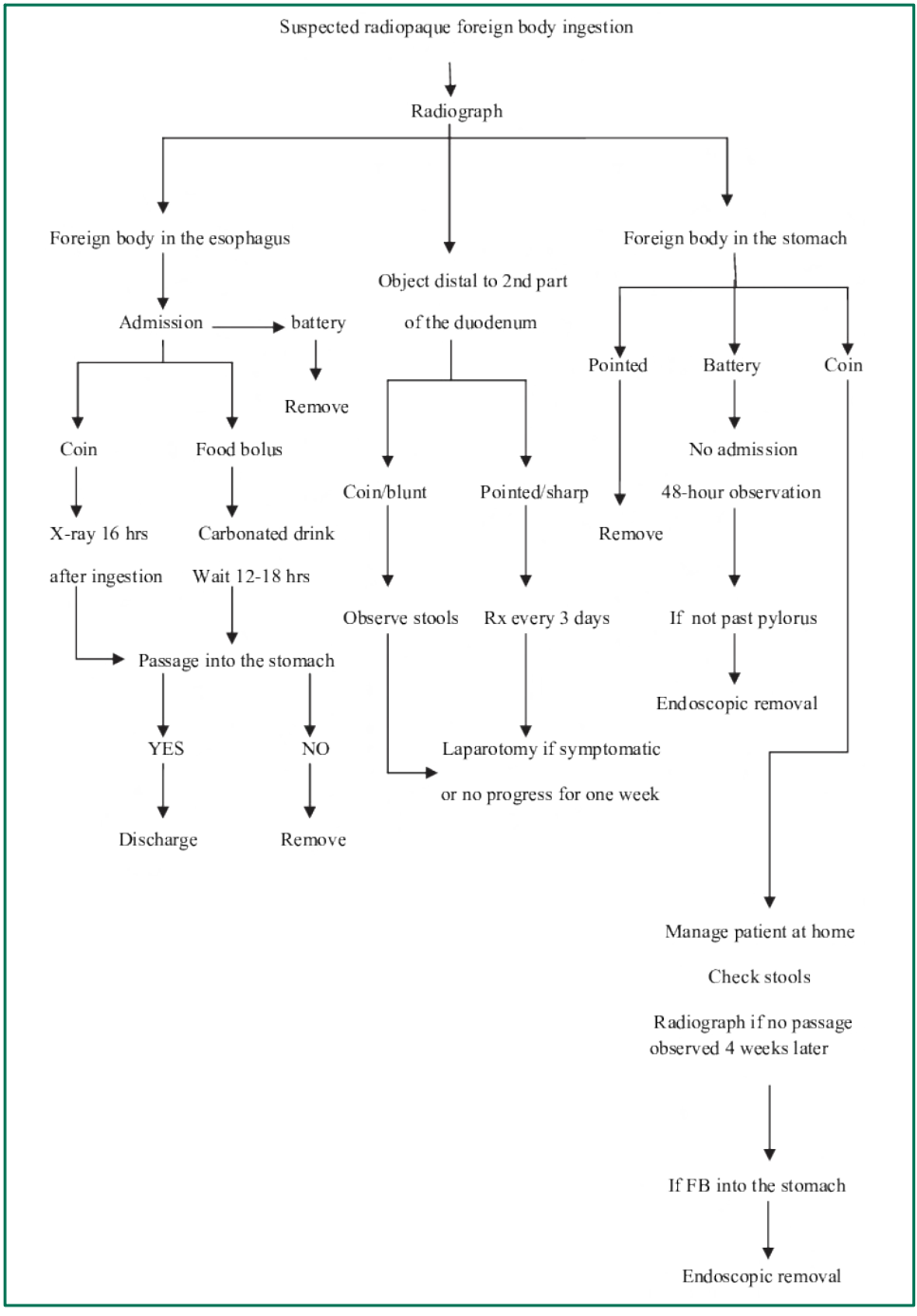
This 3-year-old child presented with dysphagia and drooling.



(A) The anteroposterior radiograph shows a coin that appears en face inthe upper esophagus.



(B) The lateral view shows that the coin is posterior to the trachea, confirming its esophageal location.


Suspected radiopaque foreign body ingestion
Radiograph
Foreign body in the esophagus
Admission
battery
Remove
Coin
Food bolus
X-ray 16 hrs
after ingestion
Carbonated drink
Wait 12-18 hrs
Passage into the stomach
YES
NO
Discharge
Remove
Object distal to 2nd part
of the duodenum
Coin/blunt
Pointed/sharp
Observe stools
Rx every 3 days
Laparotomy if symptomatic
or no progress for one week
Foreign body in the stomach
Pointed
Battery
Coin
Remove
No admission
48-hour observation
If not past pylorus
Endoscopic removal
Manage patient at home
Check stools
Radiograph if no passage
observed 4 weeks later
If FB into the stomach
Endoscopic removal

7. A patient met with RTA was brought to casualty. He had a feeble pulse with pulse rate of 110 bpm. There is reduced air entry on the left side of thorax. Systolic BP is 70. On examination there is bruising with left hypochondrium ecchymosis.What is the next best step in management ?

A. CECT Abdomen
B. Abdominal paracentesis
C. E FAST
D. X-ray Abdomen

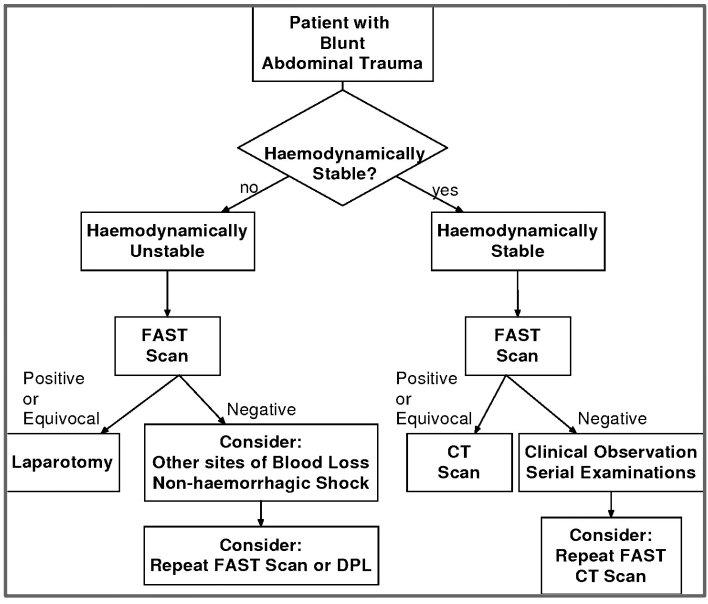
The patient is unstable. The next best step in management is E FAST.

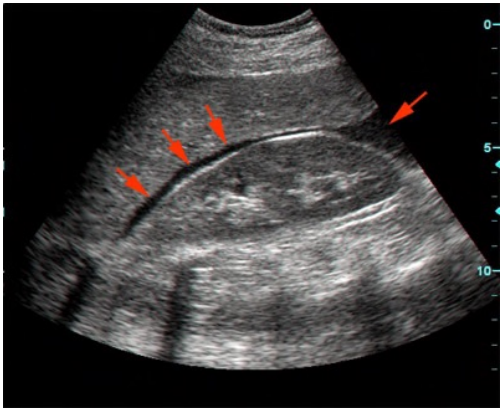
FAST

The FAST (Focused Assessment with Sonography in Trauma) examination looks for the presence of fluid—presumed to be blood in the appropriate clinical setting—visualizing 10 structures or spaces in four areas:

- Pericardial
- Perihepatic
- Perisplenic
- Pelvic

- The E-FAST (Extended-FAST) additionally surveys the anterior and lateral pleural spaces (thoracic view) to evaluate for a pneumothorax or pleural effusion, assumed to be a hemothorax in trauma patients.
- E-FAST has high sensitivity and specificity (especially in the setting of hypotension), can be done quickly, noninvasively, and without radiation exposure, and may be repeated. Due to these benefits, ultrasound has essentially replaced diagnostic peritoneal lavage (DPL) in evaluation of trauma patients.
- In a hemodynamically unstable patient, a positive E-FAST may indicate immediate intervention (eg, tube thoracostomy, pericardial window, diagnostic laparotomy).
- In a hemodynamically stable patient, the E-FAST can direct further diagnostic testing.
- The E-FAST examination should be completed in < 5 minutes
- The pericardial sac is evaluated first, especially after penetrating trauma, because pericardial fluid after trauma can be immediately life-threatening and supersede treatment of other injuries.
- The E-FAST examination maximizes sensitivity by imaging dependent positions in the peritoneal cavity where fluid preferentially accumulates. This fluid appears as anechoic (black) areas filling the potential spaces. The examination also focuses on interfaces between solid organs in order to maximize fluid visibility.





A positive FAST - fluid (black stripe, indicated by red arrows) within Morison's pouch.

- FAST is most useful in trauma patients who are hemodynamically unstable.
- A positive FAST result is defined as the appearance of a dark ("anechoic") strip in the dependent areas of the peritoneum.
- In the right upper quadrant this typically appears in Morison's Pouch (between the liver and kidney).
- This location is most useful as it is the place where fluid will collect with a supine patient.
- In the left upper quadrant, blood may collect anywhere around the spleen (perisplenic space).
- In the pelvis, blood generally pools behind the bladder (in the rectovesicular space).

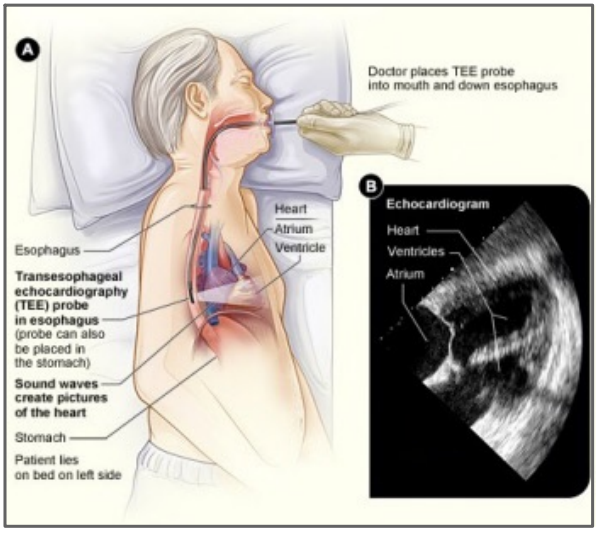
8. A 63 year old hypertensive patient presented with chest pain and diaphoresis. On examination he has unequal pulses in both arms. Which of the following is the most useful imaging investigation in emergency in this case?

A. Cardiac enzymes
B. TEE
C. TTE
D. D dimer

The patient is having aortic dissection. The useful imaging investigation for this patient is TEE as he is unstable.

TRANSESOPHAGEAL ECHOCARDIOGRAM

- A transesophageal echocardiogram (TEE) uses echocardiography to assess the structure and function of the heart.
- During the procedure, a transducer (like a microphone) sends out ultrasonic sound waves.
- When the transducer is placed at certain locations and angles, the ultrasonic sound waves move through the skin and other body tissues to the heart tissues, where the waves bounce or "echo" off of the heart structures.
- The transducer picks up the reflected waves and sends them to a computer. The computer displays the echoes as images of the heart walls and valves.
- A transesophageal echocardiogram is done by inserting a probe with a transducer down the esophagus. This provides a clearer image of the heart because the sound waves do not have to pass through skin, muscle, or bone tissue.
- The TEE probe is much closer to the heart since the esophagus and heart are right next to each other.

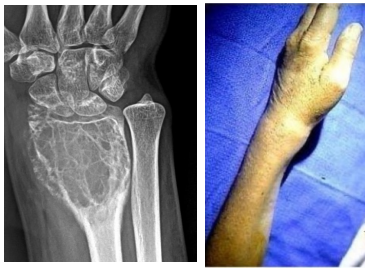


D-dimer is a fibrin degradation product that is often used to measure and assess clot formation.

Reference:Grainger & Allison's Diagnostic Radiology

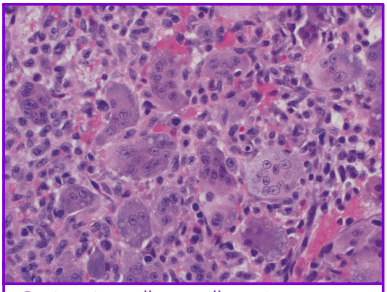
1. 30 yr old male complains of gradual swelling around wrist for 3 months,clinical photo and X Ray is given below.What is the most likely diagnosis ?

A. Ewing's sarcoma
B. Osteoclastoma
C. Pycnodysostosis
D. Osteochondroma



- From the given history,age of the person and site of lesion being epiphysis and the lesion being expansile with septations most probable diagnosis is Osteoclastoma

GIANT CELL TUMOR



Benign giant cell tumor illustrating an abundance of multinucleated giant cells with background mononuclear stromal cells.

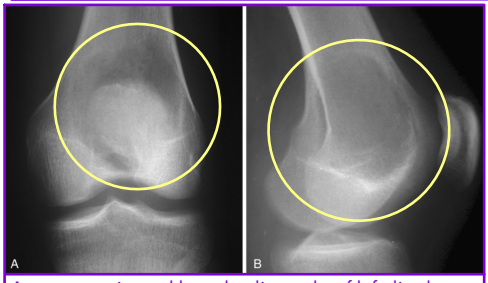
- Typically occur in patients 20 to 40 years old, and there is a slight female predominance.
- Site :
  - -Most common location for this tumor is the distal femur > proximal tibia > In the distal radius
  - - Spinal involvement, other than the sacrum, is rare.
- Malignant giant cell tumors
  - - represent less than 5% of cases.
  - - classified as primary or secondary.
- Usually are solitary lesions but can be multicentric.
- Although these tumors typically are benign, pulmonary metastases occur in approximately 3% of patients.
- Occasionally be seen in conjunction with Paget's disease of bone and can arise in association with focal dermal hypoplasia (Goltz syndrome).
- Frequently are locally aggressive.

CLINICAL FEATURES

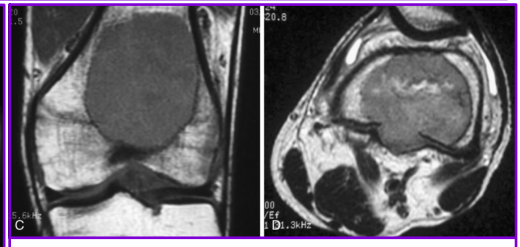
- Progressive pain :
  - - related to activity initially and only later becomes evident at rest.
  - - severe, unless a pathologic fracture has occurred.

INVESTIGATIONS

- Radiographic findings often are diagnostic.
  - The lesions are eccentrically located in the epiphyses of long bones and usually abut the subchondral bone (often with a multilocular appearance "soap bubbles").
  - Lesions are purely lytic,frequently expands or breaks through the cortex
- On MRI, the lesion usually is dark on T1 weighted images and bright on T2 weighted images.



Anteroposterior and lateral radiographs of left distal femur show lytic lesion with extension to articular surface.



Coronal and axial MR images show extent of lesion within bone and soft tissue.

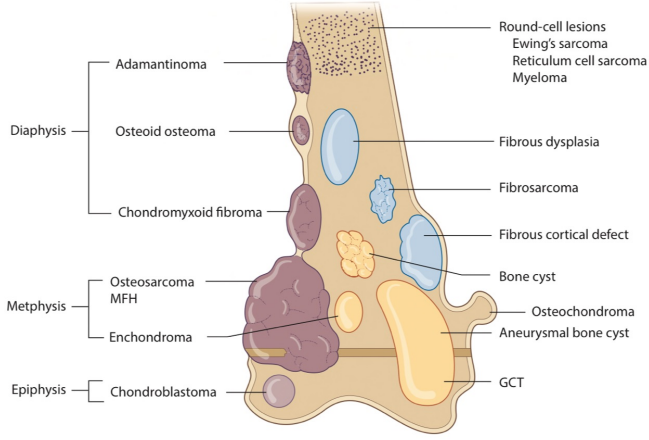
GCTs are staged according to the classification system described by Campanacci

Stage 1 - Completely intraosseous
Stage 2 - Demonstrates cortical erosion without destruction
Stage 3 - Characterized by cortical destruction with a soft-tissue component

TREATMENT

- Stage 1 and 2 lesions :
  - extended intralesional curettage with a detailed debridement of the lesional wall will be effective.
- Stage 3 disease :
  - thorough curettage though in some cases en-bloc resection.
  - radiotherapy was used following curettage and still has a role in difficult locations such as the spine.
- Anti-RANKL antibodies : Denosumab (stop the osteolytic process and switch the balance towards bone formation.) Q
- Use of adjuvants, such as liquid nitrogen, phenol, bone cement, electrocautery, or an argon beam coagulator, theoretically helps kill any remaining tumor cells.
- Bisphosphonates (administered systemi cally or locally) might help prevent recurrence.
- For inoperable lesions in the spine or pelvis, irradiation or embolization (or both) may be used.
- In patients with pulmonary metastases, resection should be attempted.

Potential diagnoses : The location of the lesion within the bone is significant.



Possible diagnosis based on the radiographic appearances, divided by age group

Age (years)	Well-circumscribed lesion	Ill-defined lesions	Sclerotic lesions
0–10	Eosinophilic granuloma Simple bone cyst	Eosinophilic granuloma Ewing's sarcoma Leukaemia	Osteosarcoma
10–20	Non-ossifying fibroma Osteoblastoma Fibrous dysplasia Eosinophilic granuloma Simple bone cyst Aneurysmal bone cyst Chondroblastoma Chondromyxoid fibroma	Ewing's sarcoma Eosinophilic granuloma Osteosarcoma	Osteosarcoma Fibrous dysplasia Eosinophilic granuloma Osteoid osteoma Osteoblastoma
20–40	Giant-cell tumour Enchondroma Low-grade chondrosarcoma Brown tumour Osteoblastoma	Giant-cell tumour	Enchondroma Bone island Parosteal osteosarcoma Burnt-out lesion: • Non-ossifying fibroma • Eosinophilic granuloma • Simple bone cyst • Aneurysmal bone cyst • Chondroblastoma
40+	Metastases Myeloma Geode	Metastases Myeloma High-grade chondrosarcoma	Metastases Bone island
All ages	Infection	Infection	Infection

PYCNODYSOSTOSIS

- Is a lysosomal storage disease of the bone caused by a mutation in the gene that codes the enzyme Cathepsin K.
- Causes the bones to be abnormally dense ; the last bones of the fingers (the distal phalanges) to be unusually short; and delays the normal closure of the connections (sutures) of the skull bones in infancy, so that the "soft spot" (fontanelle) on top of the head remains widely open.
- Other abnormalities involve the head and face, teeth, collar bones, skin, and nails.
- The front and back of the head are prominent.
- Within the open sutures of the skull, there may be many small bones (called wormian bones).
- Treatment : Symptomatic management

2. 20 yr old patient presenting with chronic low backache and early morning stiffness since last two years.Since last 6 months there are bilateral heel pains also.Most likely diagnosis for this patient is ?

A. TB Spine
B. Ankylosis spondylitis
C. Mechanical pain
D. Disc prolapse

3. A 26 yr old male with backache,morning stiffness and reddening of eyes with an X-Ray presented in OPD.Most likely diagnosis for this patient is ?

A. Rheumatoid Arthritis
B. Ankylosis spondylitis
C. Psoriatic arthritis
D. Osteoporosis

- From the above questions features present are suggestive of Ankylosing Spondylitis.

ANKYLOSING SPONDYLITIS

- a.k.a Marie strumpell disease,chronic disease characterised by a progressive inflammatory stiffening of the joints.
- Affects axial skeleton, especially the sacro- iliac joints.
- Disease commonly seen in young adults
- More common in males

AETIOPATHOLOGY

- Exact aetiology is not known
- Strong association has been found between a genetic marker—HLA-B27.
- Sacro-iliac joints are usually the first to get involved; followed by the spine from the lumbar region upwards.
- The hip, the knee and the manubrio-sternal joints are also involved frequently.

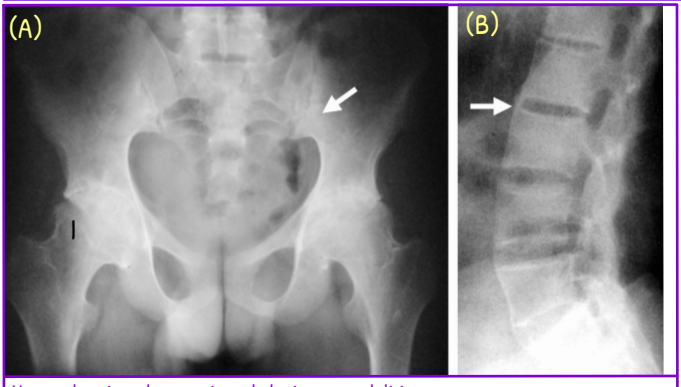
CLINICAL FEATURES

- Gradual onset of pain and stiffness of the lower back.
- Pain tends to be worst at night or early morning, awakening the patient from sleep.
- Pain in the heel, pubic symphysis, manubrium sterni and costo-sternal joints.
- Kyphotic deformity of spine and deformity of the hips may be prominent features.
- May occasionally present with involvement of peripheral joints such as the shoulders, hips and knees.
- Smaller joints are rarely involved.

DIAGNOSIS

- Stiff spine :
  - There may be a loss of lumbar lordosis.
- Tests for detecting sacro-iliac involvement :
  - i. Gaenslen's test:
    - The hip and the knee joints of the opposite side are flexed to fix the pelvis, and the hip joint of the side under test is hyperextended over the edge of the table.
    - This will exert a rotational strain over the sacro-iliac joint and give rise to pain
  - ii. Straight leg raising test
  - iii. Pump-handle test
- Tests for cervical spine involvement :
  - i. The Fle'che test
- Thoracic spine involvement : A chest expansion less than 5 cm indicates involvement of the costo-vertebral joints.
- X-rays of the pelvis (AP), and dorso-lumbar spine (AP and lateral) are required.
- Oblique views of sacro-iliac joints may be required in early stages to appreciate their involvement.
- Following changes may be seen on X-ray of the pelvis :
  - Haziness of the sacro-iliac joints
  - Irregular subchondral erosions in SI joints
  - Sclerosis of the articulating surfaces of SI joints
  - Widening of the sacro-iliac joint space
  - Bony ankylosis of the sacro-iliac joints
  - Calcification of the sacro-iliac ligament and sacro-tuberous ligaments
  - Evidence of enthesopathy

- X-ray of the lumbar spine may show the following :
  - Squaring of vertebrae
  - Loss of the lumbar lordosis.
  - Bridging 'osteophytes' (syndesmophytes).
  - Bamboo spine appearance.
- ESR: elevated
- Hb: mild anaemia
- HLA-B27:positive



X-rays showing changes in ankylosing spondylitis.
(a) X-ray of the pelvis, AP view, showing bilateral SI joint and hip involvement
(b) X-ray of the lumbar spine, Lateral view, showing calcification of the ligaments

TREATMENT

- No specific therapy is available.
- Structured exercise programme.
- Conservative methods :
  (i) Drugs—NSAIDs are given for pain relief; Indomethacin is effective in most cases;
  (ii) Physiotherapy
  (iii)Radiotherapy – in some resistant cases;
  (iv) Yoga therapy.
- Operative methods :
  - Spinal osteotomy, and joint replacement for cases with hip or knee joint ankylosis.

POTT'S PARAPLEGIA

- a.k.a TB Spine with Neurological Involvement.
- It occurs most commonly in tuberculosis of the dorsal spine (because the spinal canal is narrowest in this part, and even a small compromise can lead to a neurological deficit.)

CLINICAL FEATURES

- **Muscleweakness,spasticity and in-coordination** due to pressure on the corticospinal tracts which are placed anteriorly in the cord and are probably more sensitive to pressure.
- **Paraplegia in extension** : Tone of the muscles is increased due to absence of normal corticospinal inhibition, resulting in paraplegia in extension.
- **Paraplegia in flexion** : Absence of paraspinal tract functions in addition to the corticospinal functions leads to paraplegia in flexion.
- **Complete flaccid paraplegia** : Paraplegia becomes completely flaccid once all transmission across the cord stops.

INVESTIGATIONS

- **MRI scan (Investigation of choice) :**
  (i) type of vertebral destruction;
  (ii) presence of para-vertebral soft tissue abscess; and
  (iii) cause of paraplegia i.e., whether it is pus, sequestra etc.
- **CT scan :** To evaluate the vertebral canal.

TREATMENT

- **Principles of treatment :**
  a) To promote recovery of the affected neural tissues, by reversing the cause responsible for compression, either by drugs or by operation.
  b) To achieve healing of the vertebral lesion, and to support the spine till the diseased segment becomes stable.
  c) To undertake rehabilitative measures to prevent contractures, and to regain strength in the affected part.
- **Conservative treatment :**
  - Anti-tubercular chemotherapy forms the mainstay of treatment.
  - All patients are started on 4-drugs anti-tubercular chemotherapy as soon as the diagnosis is made.
  - Absolute rest to spine
  - If paraplegia improves, conservative treatment is continued.
  - Patient is allowed to sit in the bed with the help of a brace as soon as the spine has gained sufficient strength.
  - Bracing is continued for a period of about 6 to 12 months.
- **Operative treatment :**
  a) Costo-transversectomy
  b) **Antero-lateral decompression (ALD)**
  c) **Radical debridement and arthrodesis (Hongkong operation)**
  d) Laminectomy :
    - It is indicated in cases of spinal tumour syndrome

4. A 60yr old female with previous history of Colle's fracture is complaining of backache.DEXA Score is - 2.6.Which of the following given statement is wrong in relation to treatment of this patient. ?

A. Teriparatide should be started before supplementing bisphosphonates
B. Bisphosphonates not given for more than a year.
C. Calcium required is 1200mg per day
D. Oral Vit D3 given along with Oral calcium

- From the given history most probable diagnosis is Osteoporosis.
- A T-score within 1 SD (+1 or -1) of the young adult mean indicates normal bone density.
- A T-score of 1 to 2.5 SD below the young adult mean (-1 to -2.5 SD) indicates low bone mass.
- A T-score of 2.5 SD or more below the young adult mean (more than -2.5 SD) indicates the presence of osteoporosis.

OSTEOPOROSIS

- Characterized by an abnormally low bone mass and defects in bone structure, a combination which renders the bone unusually fragile and at greater than normal risk of fracture in a person of that age, sex and race.
- Bone depletion may be due to :
  - bone resorption,
  - decreased bone formation or
  - a combination of the two.

RISK FACTORS FOR OSTEOPOROSIS

- Age
- Female
- Previous fragility fracture
- Current use or frequent recent use of oral or systemic glucocorticoids
- Family history of hip fracture
- Low body mass index (BMI) (less than 18.5 kg/m2)
- Smoking
- Alcohol intake of > 14 units per wk for women and > 21 units per wk for men

CLINICAL FEATURES

- An asymptomatic disorder unless complications (predominantly fractures) occur.
- Dorso-lumbar spine is the most frequent site.
- Pain from fractures

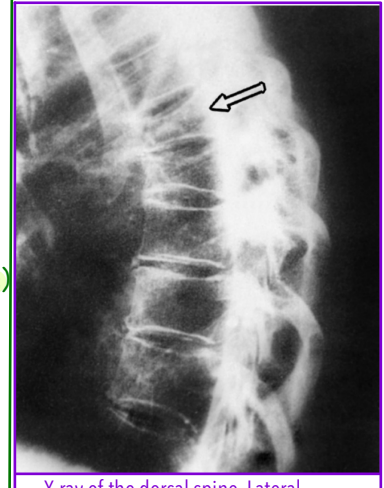
Reference :
Notespaedia Orthopedics High Yield Pg : 112
Notespaedia Orthopedics IB Pg : 118

INVESTIGATIONS

- Serum calcium, phosphates and alkaline phosphatase are within normal limits.
- Total plasma proteins and plasma albumin may be low.
- Densitometry:
  - Method to quantify osteoporosis.
  - Two types of bone densitometry are available – ultrasound based and X-ray based.
- DEXA scan is an X-ray based bone densitometry, and is the gold standard in the quantification of bone mass.Q
- Neutron activation analysis
- Bone biopsy.
- On X-rays:
  - Loss of vertical height of a vertebra.
  - Cod fish appearance: The disc bulges into the adjacent vertebral bodies so that the disc becomes biconvex.
  - Ground glass appearance of the bones.
  - Metacarpal index and vertebral index

TREATMENT

- Medical treatment: consists of
  - High protein diet
  - Calcium supplementation
  - Androgens
  - Estrogens: halt the progressive loss of bone mass in post- menopausal osteoporosis.
  - Vitamin D : increase calcium absorption from gut is given alongwith Calcium.
  - Alandronate
  - Calcitonin
  - Teriparatide: Anabolic agent increasing osteoblastic new bone formation.(Started before Bisphosphonates)
  - Denosumab, Strontium: Antiresorptive agents.
- Orthopaedic treatment : consists of
  - Exercises
  - Bracing: Prophylactic bracing of the spine by using an ASH brace or Taylor brace may be useful in prevention of pathological fractures in a severely m osteoporotic spine.



X-ray of the dorsal spine, Lateral view, showing marked osteoporosis

5. A patient fell down from bicycle and started having pain around hip,shortening of limb and attitude was flexion,addiction,IR of Hip ?

A. Anterior dislocation
B. Transcervical fracture
C. Posterior dislocation
D. Intertrochanteric fracture

- From the given history and there is shortening of hip and attitude being flexion,addiction and IR most probable diagnosis : Posterior dislocation

POSTERIOR DISLOCATION OF THE HIP

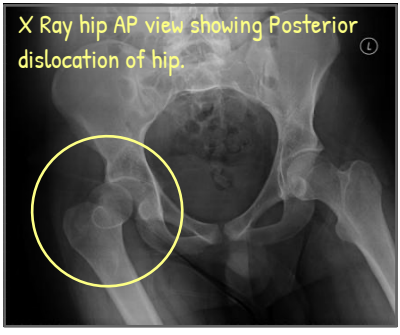
- The head of the femur is pushed out of the acetabulum posteriorly.

MECHANISM OF INJURY

- Injury is sustained by violence directed along the shaft of the femur, with the hip flexed.

CLINICAL FEATURES

- The patient presents with a history of severe trauma followed by pain, swelling and deformity (flexion, adduction and internal rotation).
- Shortening of the leg.
- One may be able to feel the head of the femur in the gluteal region.


X Ray hip AP view showing Posterior dislocation of hip.

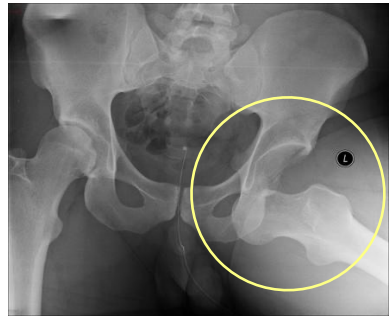
DIAGNOSIS

- Radiological features :
  - The femoral head is out of the acetabulum.
  - The thigh is internally rotated so that the lesser trochanter is not seen.
  - Shenton's line* is broken.

TREATMENT

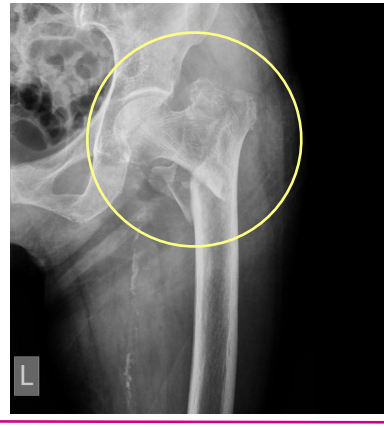
- Hip should be reduced as soon as possible as long as head remains out of more chances it becoming a avascular.
- Open reduction may be required in cases where :
  (i) closed reduction fails, usually in those presenting late;
  (ii) if there is intra-articular loose fragment not allowing accurate reduction; and
  (iii) if the acetabular fragment is large and is from the weight bearing part of the acetabulum.

ANTERIOR DISLOCATION OF THE HIP



- Usually sustained when the legs are forcibly abducted and externally rotated.
- This may occur in a fall from a tree when the foot gets stuck and the hip abducts excessively, or in a road accident.
- Clinically,the limb is in an attitude of external rotation.
- There may be true lengthening, with the head palpable in the groin.

INTER-TROCHANTERIC FRACTURES



- Fractures in the inter trochanteric region of the proximal femur, involving either the greater or the lesser trochanter or both.
- Fracture sustained in :
  i. Elderly people : sustained by a sideway fall or a blow over the greater trochanter.
  ii. Young : occurs following violent trauma, as in a road traffic accident.
- The distal fragment rides up so that the femoral neck-shaft angle is reduced (coxa vara).
- The fracture is generally comminuted and displaced.

OBGY

1. 25 yr primigravida is on indomethacin (25 mg TDS) for polyhydramnios till 35 weeks. What abnormality can the fetus have, if she goes into labor now?

A. Flap closure of foramen ovale
B. Patent ductus arteriosus
C. Premature closure of the ductus arteriosus
D. Premature closure of the ductus venosus

HUMAN TERATOGENIC DRUGS

Drug	*Abnormality*
Thalidomide	phocomelia, multiple defects of internal organs
Anticancer drugs (methotrexate)	cleft palate, hydrocephalus, multiple defects, foetal death
Androgens	virilization; limb, esophageal, cardiac defects
Progestins	virilization of female foetus
Stilboestrol	vaginal carcinoma in teenage female offspring
Tetracyclines	discoloured and deformed teeth, retarded bone growth
Warfarin	depressed nose; eye and hand defects, growth retardation
Phenytoin	hypoplastic phalanges, cleft lip/palate, microcephaly
Phenobarbitone	various malformations
Carbamazepine	neural tube defects, assorted abnormalities
Valproate sod.	spina bifida and other neural tube defects, heart and limb abnormalities
Alcohol	low IQ baby, growth retardation, foetal alcohol syndrome
ACE inhibitors	hypoplasia of organs, growth retardation, foetal loss
Lithium	foetal goiter, cardiac and other abnormalities
Antithyroid drugs	foetal goiter and hypothyroidism
Indomethacin/aspirin	premature closure of ductus arteriosus
Isotretinoin	craniofacial, heart and CNS defects, hydrocephalus

RISK CATEGORY OF DRUGS DURING PREGNANCY*

Category		*Examples*
A No risk	Adequate studies in pregnant women have failed to demonstrate a risk to the foetus	Inj. Mag. sulfate, thyroxine
B No evidence of risk in humans	Adequate human studies are lacking, but animal studies have failed to demonstrate a risk to the foetus or Adequate studies in pregnant women have failed to demonstrate a risk to the foetus, but animal studies have shown an adverse effect on the foetus	Penicillin V, amoxicillin, cefactor, erythromycin, paracetamol, lidocaine
C Risk cannot be ruled old	No adequate studies in pregnant women, and animal studies are lacking or have shown an adverse effect on foetus, but potential benefit may warrant use of the drug in pregnant women despite potential risk	Morphine, codeine, atropine, corticosteroids, adrenaline, thiopentone, bupivacaine
D Benefit may out weigh potential risk	There is evidence of human foetal risk, but the potential benefits from use of the drug may be acceptable despite the potential risk	Aspirin, phenytoin, carbamazepine, valproate, lorazepam, methotrexate
X Contra-indicated	Studies in animals or humans have demonstrated foetal abnormalities, and potential risk clearly outweighs possible benefit	Estrogens, isotretinoin, ergometrine thalidomide

2. Deoxygenated blood is returned to the placenta from the fetus through ?

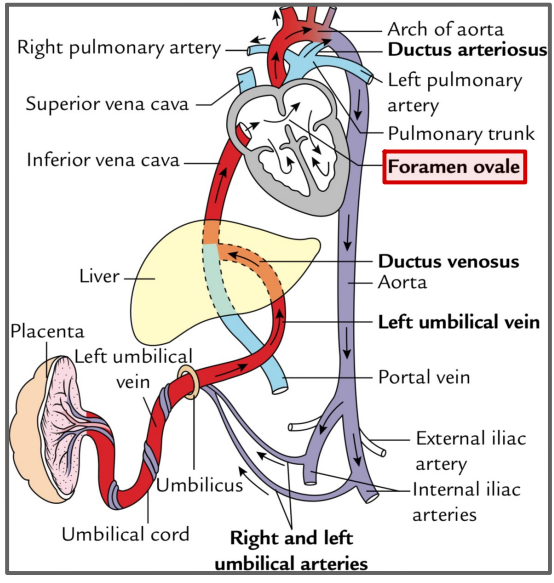
A. Umbilical artery

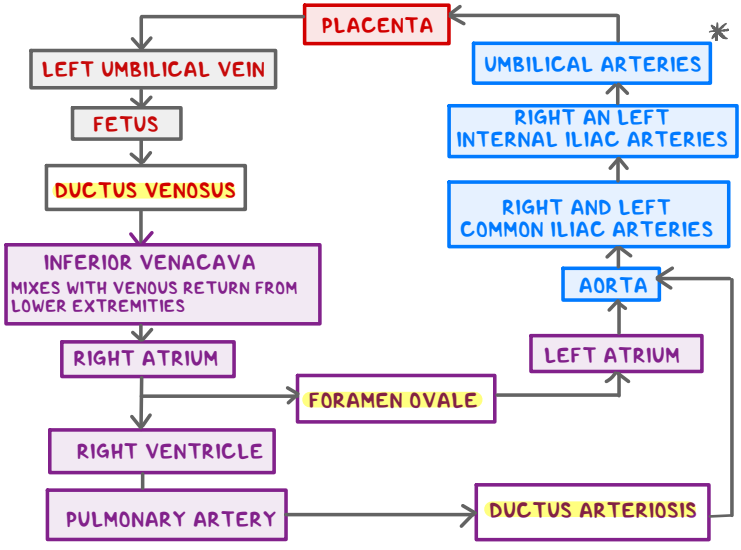
B. Umbilical vein

C. Ductus arteriosus

D. Ductus venosus

FETAL CIRCULATION





3. A married woman gives birth to twins. The husband doubts he is the father and gets a paternity test done. The test shows that he is the father of one infant but not the other. This is a case of?

A. Superfetation
B. One infant is atavistic
C. Superfecundation
D. One infant is suppositious

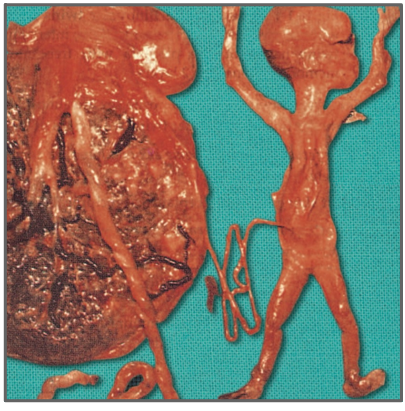
SUPERFECUNDATION

- Fertilization of two different ova released in the same cycle, by separate acts of coitus within a short period of time.

Homopaternal superfecundation refers to the fertilization of two separate ova from the same father, leading to fraternal twins; Heteropaternal superfecundation is referred to as a form of atypical twinning where, genetically, the twins are half siblings – sharing the same mother, but with different fathers.

- **SUPERFETATION** is the fertilization of two ova released in different menstrual cycles. The nidation and development of one fetus over another fetus is theoretically possible until the decidual space is obliterated by 12 weeks of pregnancy.

FETUS PAPYRACEOUS



Fetus papyraceous or compressus is a state which occurs if one of the fetuses dies early. The dead fetus is flattened, mummified and compressed between the membranes of the living fetus and the uterine wall. It may occur in both varieties of twins, but is more common in monozygotic twins and is discovered at delivery or earlier by sonography

Reference:DC DUTTA's TEXTBOOK OF OBSTETRICS (pg 233)
Reference: Williams Obstetrics 24th edition(pg 893)

4. A 28 yr primigravida who is a known case of Mitral valve replacement presents at 36wks to the antenatal OPD. She is on warfarin 4mg. Which is correct regarding anticoagulant therapy?

A. Discontinue warfarin and start heparin
B. Discontinue warfarin and start heparin and aspirin
C. Discontinue warfarin and start aspirin
D. Continue warfarin and start heparin

ANTICOAGULANTS IN PREGNANCY

Anticoagulants are indicated in cases with:

(a) Congenital heart disease,
(b) pulmonary hypertension,
(c) mechanical heart valve,
(d) atrial fibrillation.

- The patient taking warfarin should discontinue it as soon as pregnancy is diagnosed and to replace it by heparin 5,000 units twice daily subcutaneously up to 12th week.
- Low molecular weight heparin (LMWH) can also be used.
- This is then replaced by warfarin tablet 3 mg. daily to be taken at the same time each day and continued up to 36 weeks.
- Thereafter it is replaced by heparin up to 7 days postpartum. Warfarin is then to be continued.
- UFH, LMWH and Warfarin therapy do not contraindicate breast-feeding.

Drug	Mode of Action	Dose	Side Effects
Heparin ▪ (unfractionated heparin) ▪ **Low-molecular-weight heparin (LMWH)**	Inhibits action of thrombin, it also enhances the activity of antithrombin III, increases factor Xa inhibitor **Antidote:** Protamine sulfate	5,000–10,000 IU to be administered parenterally. **DVT and pulmonary embolism:** loading dose 5,000 units IV followed by continuous infusion of 18 units/kg/hr. **Pregnancy:** 5,000-10,000 SC every 12 hrs (with monitoring). **LMWH:** Enoxaparin 1 mg/kg twice daily SC, less antithrombotic effect	**Maternal:** Hemorrhage, urticaria with long-term use thrombocytopenia and osteopenia, hyperkalemia **Fetal:** It does not cross the placenta, not teratogenic **Low-molecular-weight heparins**—as effective and safe as unfractionated heparin. Longer half-life and once daily dose is convenient. Standard dose does not require monitoring
Warfarin	**Interferes with synthesis of vitamin K dependent factors** (II, VII, IX, X)	5–10 mg orally daily for initial 2 days then 3–9 mg daily (taken at the same time each day) depending upon the prothrombin time (INR) (INR – 2.5–3.5)	**Maternal:** Hemorrhage **Fetal:** Warfarin embryopathy (5%), nasal hypoplasia, bone stiplings, optic atrophy, mental retardation, microcephaly, chondrodysplasia punctata. Women with mechanical heart valves, warfarin is preferred. To avoid first trimester.

5. A 25 yr woman presents to the antenatal OPD. This is her 2nd pregnancy and her 1 st pregnancy was 4 years earlier where she delivered twins at term. Her parity index is?

A. G2P1
B. G2P2
C. G3P1
D. G3P2

PARITY INDEX

- **GRAVIDA** denotes a pregnant state both present and past, irrespective of the period of gestation.
- **PARITY** denotes a state of previous pregnancy beyond the period of viability.
- **ABORTION** o the total number of induced abortion or miscarriage or ectopic pregnancy before 20 weeks
- **L** represents number of living children

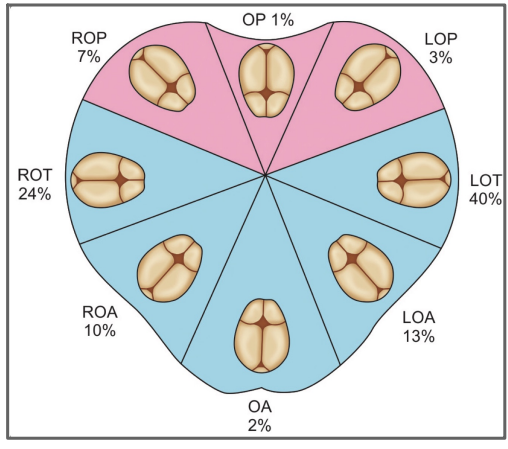
▪ **A nullipara** is one who has never completed a pregnancy to the stage of viability. She may or may not have aborted previously.	▪ **A nulligravida** is one who is not now and never has been pregnant.
▪ **A primipara** is one who has delivered one viable child. Parity is not increased even if the fetuses are many (twins, triplets).	▪ **A primigravida** is one who is pregnant for the first time.
▪ **A multigravida** is one who has previously been pregnant. She may have aborted or have delivered a viable baby.	▪ **Multipara** is one who has completed two or more pregnancies to the stage of viability or more.
▪ **A parturient** is a women in labor.	▪ **A puerpera** is a woman who has just given birth.

6. A 28 yr primigravida is in labor. She has a repeated urge to pass urine and has premature bearing down. On examination, there is infra umbilical flattening , and the fetal heart is heard on the lateral side. What is the most likely presentation/ position?

A. Knee
B. Occipital- Posterior
C. Brow
D. Right dorso- anterior

FETAL POSITION

- It is the relation of the denominator to the different quadrants of the pelvis.
- Most common:
  left occipitoanterior (LOA)



OCCIPUT-POSTERIOR POSITION (OP)

In a vertex presentation where the occiput is placed posteriorly over the sacroiliac joint or directly over the sacrum, it is called an occiput-posterior position.

RIGHT OCCIPITOPOSTERIOR (ROP)

- When the occiput is placed over the right sacroiliac joint
- Third position of the vertex

LEFT OCCIPITOPOSTERIOR (LOP)

- When placed over the left sacroiliac joint
- Fourth position of the vertex

DIRECT OCCIPITOPOSTERIOR

- When it points toward the sacrum.

In more than 50%, the occipitoposterior position is associated with either an anthropoid or android pelvis.

FETAL FACTORS

Marked deflection of the fetal head, too often favors posterior position of the vertex.

The causes of deflexion are:

(1) High pelvic inclination.

(2) Attachment of the placenta on the anterior wall of the uterus

(3) primary brachycephaly

ABDOMINAL EXAMINATION

- Inspection: The abdomen looks flat, below the umbilicus.
- Umbilical grip: The findings are:
  (1) The fetal limbs are more easily felt near the midline on either side.
  (2) The fetal back is felt far away from the midline on the flank and often difficult to outline clearly.
  (3) The anterior shoulder lies far away from the midline.
- Pelvic grips: The findings are:
  (1) The head is not engaged.
  (2) The cephalic prominence (sinciput) is not felt so prominent as found in well-flexed occipitoanterior. In direct occipitoposterior, the small sinciput is confused with breech.
- Auscultation: The maximum intensity of the fetal heart sounds is heard on the flank and often difficult to locate especially in LOP. However, in direct occipitoposterior, the FHS is distinctly felt in the midline.

VAGINAL EXAMINATION

The findings in early labor are:

(1) Elongated bag of membranes which is likely to rupture during examination.

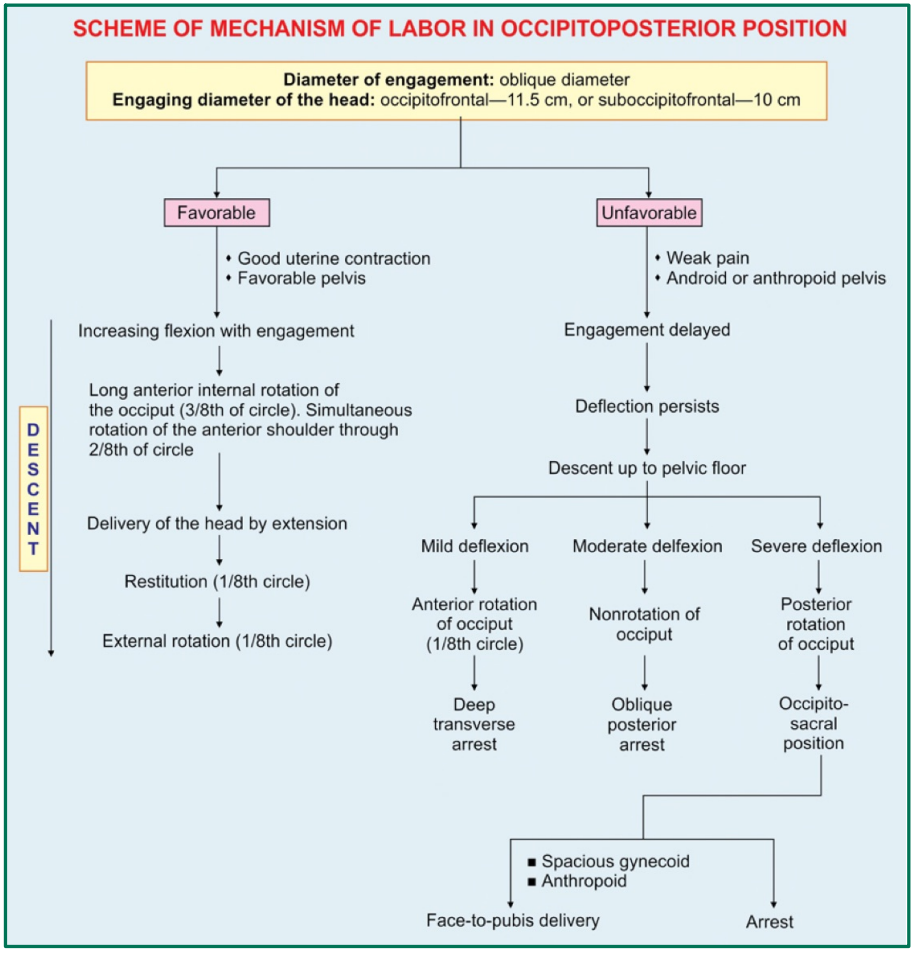
(2) The sagittal suture occupies any of the oblique diameters of the pelvis.

(3) Posterior fontanel is felt near the sacroiliac joint.

(4) The anterior fontanel is felt more easily

In late labor, the diagnosis is often difficult because of caput formation which obliterates the sutures and fontanels.

Because of deflexion, engagement is delayed.



7. Following delivery, a woman has a tonic PPH. Despite conservative measures, the bleeding persists. She was taken to the OT where the surgeon proceeds to do a devascularization procedure. Which vessels are ligated?

A. Uterine, ovarian, internal iliac
B. Uterine, ovarian, external iliac
C. Uterine, vaginal, pudendal
D. Uterine, internal iliac, obturator

POSTPARTUM HEMORRHAGE

Any amount of bleeding from or into the genital tract following birth of the baby up to the end of the puerperium, which adversely affects the general condition of the patient evidenced by rise in pulse rate and falling blood pressure is called postpartum hemorrhage

- Primary: Hemorrhage occurs within 24 hours following the birth of the baby.
  These are of two types:
  Bleeding occurs before expulsion of placenta.
  Bleeding occurs subsequent to expulsion of placenta (majority).
- Secondary: Hemorrhage occurs beyond 24 hours and within puerperium, also called delayed or late puerperal hemorrhage.

CAUSES OF PPH

- Atonic PPH (Tone)
  It is the most common cause of primary PPH accounting for 90% of cases.
  The bleeding occurs as the blood vessels are not obliterated by contraction and retraction of uterine muscle fibres
- Traumatic PPH (Trauma)
  Genital tract injuries like: Lacerations of the cervix, vagina and perineum; Colporrhexis and Rupture uterus
- Coagulopathy (Thrombin)
  Disseminated intravascular coagulation (DIC) and hypofibrinogenemia are rare causes of PPH
- Other causes (Tissue)
  Retained products of conception

SURGICAL METHODS TO CONTROL PPH

(a) B-Lynch compression suture (1997) and multiple square sutures: Both these surgical methods work by tamponade (like bimanual compression) of the uterus
(b) Ligation of uterine arteries—the ascending branch of the uterine artery is ligated at the lateral border between upper and lower uterine segment. In atonic hemorrhage, bilateral ligation is effective in about 75% of cases.
(c) Ligation of the ovarian and uterine artery anastomosis, if bleeding continues, is done just below the ovarian ligament.
(d) Ligation of anterior division of internal iliac artery (unilateral or bilateral)—reduces the distal blood flow. It helps stable clot formation by reducing the pulse pressure up to 85%. Due to extensive collateral circulation, there is no pelvic tissue necrosis.
(e) Angiographic selective arterial embolization (bleeding vessel) under fluoroscopy (interventional radiology) can be done using gel foam.

SCHEME OF MANAGEMENT OF TRUE PPH

Immediate measures

- Call for extra help (communication)
- Commence IV line with two wide bore cannulas
- Send blood for cross matching tests, coagulation screening including fibrinogen level and ask blood for 2 units (at least)
- Rapidly infuse normal saline/haemaccel 2 liters till blood is available
- To catheterize the bladder
- To monitor pulse, BP, temperature, output, oximeter every 15–30 minutes

Skill drill for PPH using mannequins for all labor attendants

To Feel the Uterus by Abdominal Palpation

Uterus atonic

- Massage the uterus to make it hard
- To add oxytocin 10–20 units in 500 mL of Normal saline, at the rate of 40 drops per minute
- Injection methergine 0.2 mg IV (slowly)
- To examine the expelled placenta

Uterus remains atonic

- Exploration of the uterus
- Blood transfusion
- To continue oxytocin drip

Uterus atonic

- 15 methyl $PGF_{2\alpha}$ 250 µg IM/intramyometrial
  OR
- Misoprostol 1000 µg per rectum
  OR
- Carbetocin 100 µg IM / IV

Uterus atonic

Uterine tamponade

(any method)

- Balloon tamponade (Fig. 28.4)
- Bimanual compression (Fig. 28.3)
- Tight intrauterine packing under anesthesia

Uterus atonic

Surgical methods

(any method)

Stepwise uterine devascularization procedure

- B-Lynch compression and multiple square sutures (Fig. 28.6)
- Ligation of uterine artery and utero-ovarian anastomotic vessels unilateral or bilateral (Fig. 28.5)
- Ligation of anterior division of internal iliac artery (unilateral or bilateral)
- Angiographic arterial embolization with gelatin sponge

Hysterectomy (rarely)

Uterus hard and contracted

Traumatic

Exploration

Hemostatic sutures on the tear sites

Commonly Used Oxytocics in the Management of PPH

Drug	Dose	Route	Dose frequency	Side effects	Contra-indications
Oxytocin	10–40 units in 1 L of crystalloid solution	First line: IV;Second line: IM (10 units)	Continuous IV	• Nausea • Water intoxication	Not as IV bolus, otherwise none
Methergine	0.2 mg	First line IM/IV; Second line PO	Every 2–4 hours	• Nausea • Vomiting • Hypertension	• Hypertension • Pre-eclampsia
15 methyl $PGF_{2\alpha}$	0.25 mg	First line IM; Second line intrauterine	Every 15–90 min. (8 doses maximum)	• Nausea • Vomiting • Diarrhea • Chills	• Bronchial asthma • Active cardiac, renal or hapatic disease
Misoprostol (PGE_1)	600–1000 mcg	First line PR second line PO	Single dose	• Fever, • Tachycardia	None

Bleeding controlled

- Continuous observation in high dependency unit/ICU
- Documentation of procedures adopted in respect to time

Guidelines for Management of PPH : RCOG, FIGO and ACOG

COMPLICATIONS OF 3RD STAGE OF LABOR

a) PPH,
b) Retained placenta,
c) Shock,
d) Pulmonary embolism
e) Inversion of uterus.

8. A 21 yr old primigravida presents to the antenatal OPD. Her school going nephew who lives in the same house has contracted varicella. A blood sample is taken for antibodies against varicella. The report is negative. What does this signify?

A. Susceptible to chicken pox
B. Immune to chicken pox
C. Susceptible to zoster
D. Immune to zoster

INTRAUTERINE INFECTIONS

T	TOXOPLASMOSIS
O	SYPHILIS, VARICELLA-ZOSTER, PARVOVIRUS B19, ZIKA VIRUS
R	RUBELLA
C	CYTOMEGALOVIRUS
H	HERPES

VARICELLA

Congenital- Intrauterine transmission

Pregnant female gets chicken pox in Ist trimester- TERATOGENICITY
Most severe form , limb atrophy/ hypoplasia, cortical atrophy, cicatrise (scars)

Neonatal - Perinatal transmission

Most common
Risk period- 5 days before and 2 days after delivery

Clinical features: Necrotising pneumonia
Vesicular skin rash

Prevention : VZIG varicella zoster immunoglobin

Treatment : acyclovir to both mother and baby

VARICELLA IN PREGNANCY

- Varicella zoster virus (DNA) does cross the placenta and may cause congenital or neonatal chickenpox.
- Maternal mortality is high due to varicella pneumonia.
  Other maternal complications are: encephalitis and bacterial superinfection.
- Congenital varicella syndrome (CVS) is characterized by: hypoplasia of limb, psychomotor retardation, IUGR, chorioretinal scarring, cataracts, microcephaly and cutaneous scarring.
- The risk of congenital malformation is nearly absent when maternal infection occurs after 20 weeks. Varicella (live attenuated virus) vaccine is not recommended in pregnancy.

- Varicella PCR can identify VZV specific DNA from vesicular fluid.
- ELISA can detect VZV specific IgG and IgM.
- Varicella zoster immunoglobulin (VZIG) should be given to exposed non-immune patients as it reduces the morbidity.
- VZIG should also be given to newborn exposed within 5 days of delivery.
- Oral acyclovir, valacyclovir is safe in pregnancy and reduce the duration of illness when given within 24 hours of the rash. However, it cannot prevent congenital infection.

9. A 28 yr primigravida presents at 36 wks with labor pain and vulval ulcers. She does not give history of similar lesions ever in the past. On examination, there are multiple painful vesicular lesions. Which is the best treatment option?

A. Acyclovir and elective LSCS
B. Analgesics and antibiotics
C. Antivirals and spontaneous vaginal delivery
D. Acyclovir and induction of labor

HERPES SIMPLEX VIRUS

- Usually transmitted sexually by an infected partner but may possibly be transmitted by orogenital contact.
- The incubation period is 2-14 days.

CLINICAL FEATURES

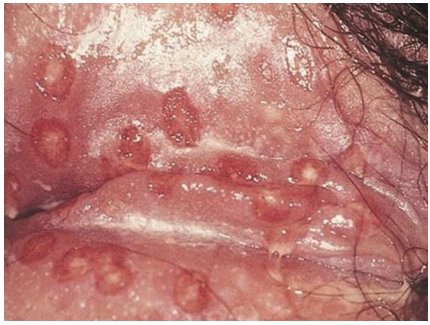
- Symptoms of the first attack usually appear less than 7 days after sexual contact. Initially, red painful inflammatory area appears commonly on the clitoris, labia, vestibule, vagina, perineum ,and cervix.
- Multiple vesicles appear which progress into multiple shallow ulcers and ultimately heal up spontaneously by crusting.
- It takes about 3 weeks to complete the process. Inguinal lymphadenopathy occurs.
- Constitutional symptoms include fever, malaise, and headache.
- There may be vulvar burning, pruritus, dysuria, or retention of urine.

DIAGNOSIS

- Virus tissue culture and isolation – confirmatory.
- Detection of virus antigen by ELISA or immuno- fluorescent method.
- PCR test to identify the HSV DNA is the rapid, specific, and most accurate test.

TREATMENT

- Acyclovir which inhibits the intracellular synthesis of DNA by the virus, reduces the symptoms, duration of viral shedding, and helps in rapid healing.
- Its prophylactic use can reduce the episodes of recurrence.
- Saline bath may relieve local pain.



Recurrent STD infection caused by the double- stranded DNA of herpes simplex virus (almost 80% are type-II infections).

The incubation period is 3-7 days. Herpes simplex virus type I affects only 30% vulval lesions.

It mostly affects women between 20 and 30 years.

EFFECT ON PREGNANCY

- Increased risk of miscarriage is inconclusive.
- If the primary infection is acquired in the last trimester there is chance of premature labor or IUGR.
- Transplacental infection is not usual.
- The fetus becomes affected by virus shed from the cervix or lower genital tract during vaginal delivery.
- The baby may be affected in utero from the contaminated liquor following rupture of the membranes.
- Risk of fetal infection is high in primary genital HSV at term due to high virus shedding compared to a recurrent infection.
- Cesarean delivery is indicated (ACOG) in an active primary genital HSV infection where the membranes are intact or recently ruptured.
- Acyclovir 400 mg three times daily for five days is the drug of choice when virus culture is positive.

NEONATAL INFECTION

- Neonatal infection may be disseminated (fatal) or localized or it may be asymptomatic.
- Diagnosis is made by detection of the viral DNA by PCR.
- It is manifested as chorioretinitis, microcephaly, mental retardation, seizures and deaths.
- Neonatal HSV infection is treated with intravenous acyclovir.
- Neonatal mortality is high.
- Prophylactic acyclovir (400 mg twice daily) or valacyclovir (1 gm twice daily) can reduce HSV shedding, neonatal transmission and cesarean delivery.
- Breastfeeding is allowed provided the mother avoids any contact between her lesions, her hands and the baby.

10. A 35 yr woman is a chronic hypertensive. She visits the clinic for preconception counselling. Which of the following anti hypertensives need to be stopped prior to conception?

A. Calcium channel blockers
B. Alpha methyl dopa
C. ACE inhibitors
D. Labetalol

ANTIHYPERTENSIVE DRUGS

- Antihypertensive drugs are essential when the BP is 160/110 mm Hg to protect the mother from eclampsia, cerebral hemorrhage, cardiac failure and placental abruption.
- Aim is to reduce BP to a mean less than 125 mm Hg.
- Their benefit in mild or moderate hypertension is not yet known.
- If there is any risk of target organ damage (kidney) antihypertensives are given to maintain BP $\leqslant$ 140 mm Hg.
- First line therapy is either methyldopa or labetalol.
- Second line drug is nifedipine.
- ACE inhibitors are avoided in pregnancy.

Mechanism of Action	Dose	Side Effects	Contraindications and Precautions
ACE inhibitors: Inhibit formation of angiotensin II from angiotensin I. ARB–Blocks Angiotensin-II receptors	▪ Captopril Orally 6.25 mg bid ▪ Telmisartan orally 20–40 mg a day	▪ Maternal: Hypotension, headache, asthenia, arrhythmias ▪ Fetal: Oligohydramnios, IUGR, fetal renal tubular dysgenesis, neonatal renal failure, pulmonary hypoplasia	**Should be avoided in pregnancy.** Suitable for chronic hypertension in nonpregnant state or postpartum

ACE inhibitors causes Renal hyperfusion —> Renal agenesis

11. A 24 yr Primi with a history of infertility for 3 years presents with 6 weeks amenorrhea. She has mild abdominal pain and spotting PV. Her UPT is weakly positive. On examination, she is hemodynamically stable. There is a 3 x 2.5 cm left sided adnexal lesion. B HCG : 2800 Ultrasound reveals a left sided tubal gestational sac with no cardiac activity. Which is the best management option?

A. Expectant management
B. Salpingectomy
C. Milking the tube
D. Single dose of Methotrexate

ECTOPIC PREGNANCY

Fertilized ovum is implanted and developed outside the normal endometrial cavity.

CLINICAL FEATURES

- Amenorrhea (seen in 75% cases)
- Abdominal pain (seen in 100% cases, it is the most consistent symptom)
- Appearance of vaginal bleeding

The above triad may be accompanied by nausea, vomiting, fainting attacks or syncope.
Patient may present in shock with pallor, tachycardia, hypotension and cold clammy extremities, if ectopic pregnancy has ruptured

INVESTIGATIONS

Transvaginal/ultrasound is the investigation most commonly done to diagnose ectopic pregnancy.
Diagnostic features are:

- Absence of intrauterine pregnancy with positive pregnancy test.
- Fluid in the pouch of Douglas (Cul-de-sac).
- Adnexal mass clearly separated from the ovary.
- Gestational sac in the adnexa surrounded by a hyperechoic ring (Bagel sign/ tubal ring sign).
- Rarely cardiac motion may be seen in an unruptured tubal ectopic pregnancy.

INDICATIONS

EXPECTANT

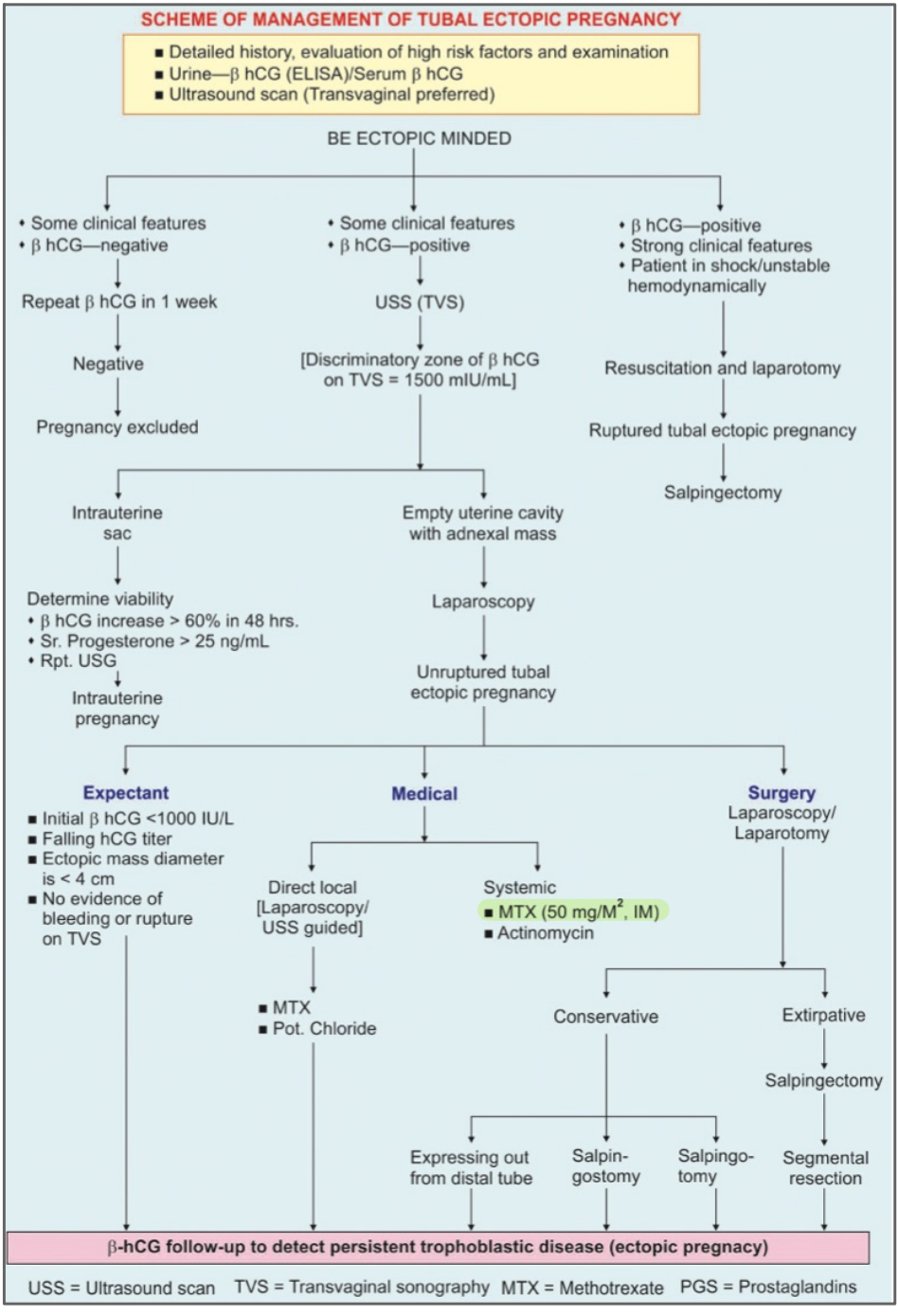
(i) Initial serum hCG level less than 1,000 IU/L and the subsequent levels are falling.
(ii) Gestation sac size less than 4 cm.
(iii) No fetal heart beat on TVS.
(v) No evidence of bleeding or rupture on TVS.

MEDICAL

(i) Hemodynamically stable.
(ii) Serum hCG level should be less than 3,000 IU/L.
(iii) Tubal diameter should be less than 4 cm without any fetal cardiac activity.
(iv) There should be no intra-abdominal hemorrhage.
For systemic therapy, a single dose of methotrexate (MTX) 50 mg/M2 is given intramuscularly.

SURGICAL

(a) Cases not fulfilling the criteria of medical therapy.
(b) Cases where



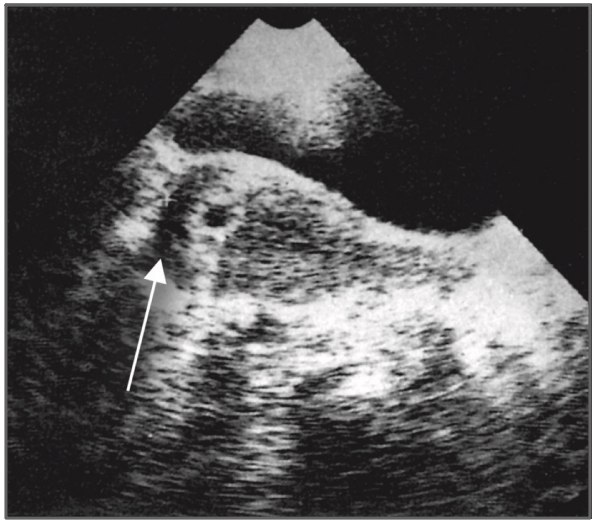
12. A 28 yr woman has been on OCPs for 5 months. She presents to the OPD with 6 weeks amenorrhea and her UPT is positive. Which is the most accurate method to determine gestational age in this woman?

A. Counting 280 days from LMP
B. Counting 256 days from UPT positive
C. CRL on ultrasound
D. Examination of uterine size

BEST PARAMETERS FOR ESTIMATION OF FETAL AGE

1ST TRIMESTER	CROWN RUMP LENGTH
2ND TRIMESTER	BIPARIETAL DIAMETER
3RD TRIMESTER	FEMUR LENGTH
OVERALL	CROWN RUMP LENGTH

TRANSVAGINAL ULTRASONOGRAPHY



- Gestational sac and yolk sac by 5 menstrual weeks
- Fetal pole and cardiac activity – 6 weeks;
- Embryonic movements by 7 weeks.
- Fetal gestational age is best determined by measuring the CRL between 7 and 12 weeks (variation ± 5 days).
- Doppler effect of ultrasound can pick up the fetal heart rate reliably by 10th week.

13. 16 yr old girl with a partial transverse vaginal septum presents with dysmenorrhea and chronic pelvic pain. What is the most common complication?

A. Endometriosis
B. Tuboovarian abscess
C. Dermoid cyst
D. Theca luteal cyst

TRANSVERSE VAGINAL SEPTA

- Due to faulty fusion or canalization of the urogenital sinus and the Müllerian ducts.
- About 45% occur in the upper vagina, 40% in mid vagina and 15% in the lower vagina.
- Septum located in the lower vagina is often complete and the signs and symptoms are similar to that of imperforate hymen.
- Ultrasonography is a useful investigation to detect hematometra, hematocolpos, and also urinary tract malformations.

CRYPTOMENORRHEA

Condition where the menstrual blood fails to come out from the genital tract due to obstruction in the passage.

- Causes:

The commonest cause is congenital due to imperforate hymen.
Other congenital causes are Transverse vaginal septum and Atresia of upper-third of vagina and cervix.
The acquired cause is rare due to cervical stenosis following amputation, conization or deep cauterization.

- Pathophysiology

If the site of obstruction is
- Low down in the vagina: hematocolpos → hematometra → hematosalpinx.
- At the cervix, hematometra → hematosalpinx.

Hematocolpos produces marked elongation of the urethra → retention of urine.

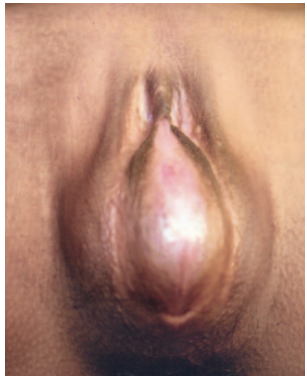
- Clinical features

The patient aged about 13–15 (congenital type) complains of periodic pain lower abdomen.
Hematocolpos is usually associated with urinary problems to the extent of retention of urine.
Abdominal examination reveals an uniform globular mass in the hypogastrium.
Vulval inspection reveals the bulging hymen.
Rectal examination confirms the fullness of the vagina and uterine mass.

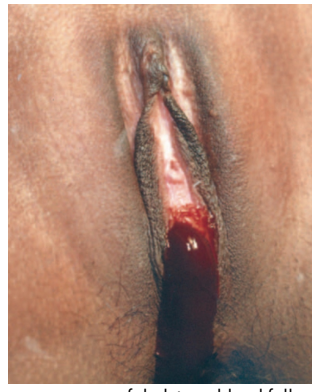
- Management

(i) cruciate incision of the hymen and drainage of blood
(ii) Dilatation of the cervix in stenosis.

- If untreated -> Hematometra and endometriosis

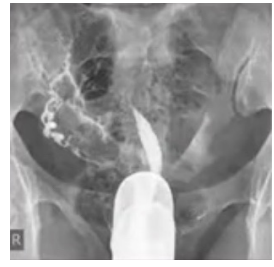


Tense bulging of the hymen in hematocolpos



Spontaneous escape of dark tarry blood following incision

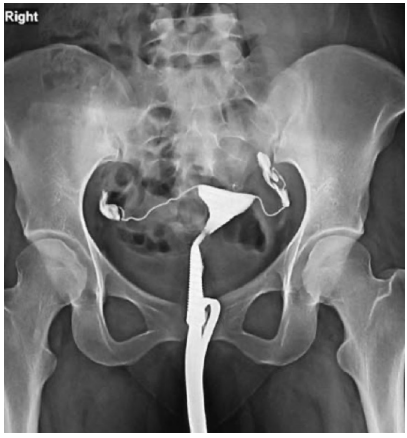
14. What is the name of the investigation shown below done in a female who presented with recurrent miscarriage?



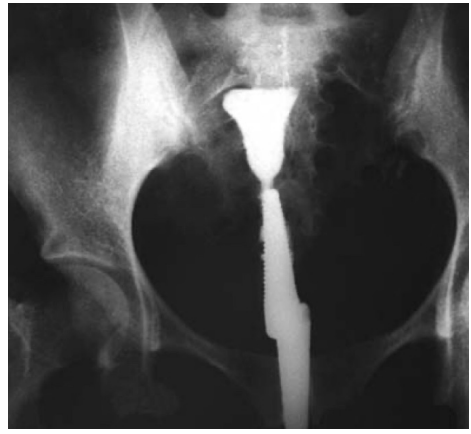
A. Genitogram
B. Saline infusion sonography
C. Laparoscopy
D. Hysterosalpinography

HYSTEROSALPINGOGRAPHY

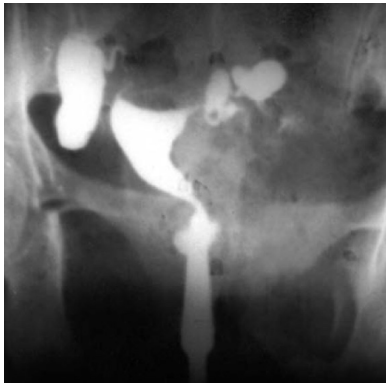
- Hysterosalpingography (HSG), also known as uterosalpingography, is a radiologic procedure to investigate the shape of the uterine cavity and the shape and patency of the fallopian tubes.
- This means it is a special x-ray using dye to look at the womb (uterus) and Fallopian tubes.
- It injects a radio-opaque material into the cervical canal and usually fluoroscopy with image intensification.
- A normal result shows the filling of the uterine cavity and the bilateral filling of the fallopian tube with the injection material.
- To demonstrate tubal rupture, spillage of the material into the peritoneal cavity needs to be observed.
- Indications
  - Infertility to assess uterine morphology and tubal patency.
- Contraindications
  - pregnancy
  - active pelvic infection
  - recent uterine or tubal surgery



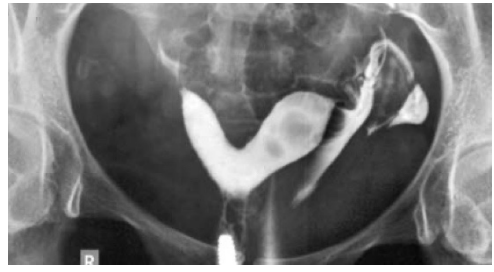
Normal hysterosalpingogram (normal cavity) with bilateral patent tubes (free peritoneal spill).



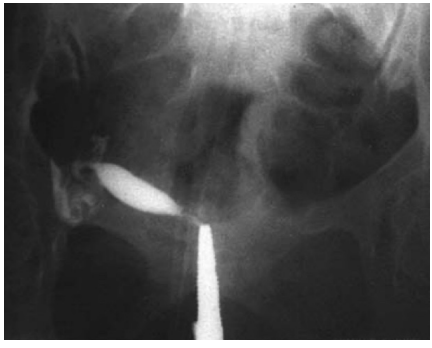
Hysterosalpingogram showing bilateral cornual block.



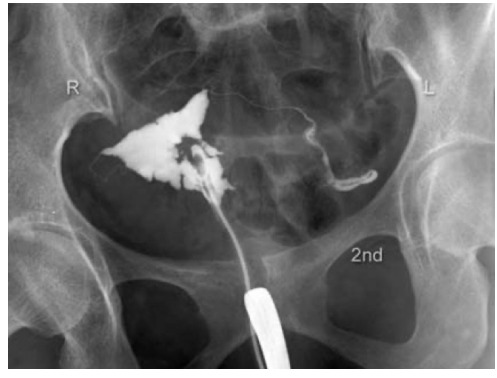
Hysterosalpingogram showing bilateral hydrosalpinx (fimbrial block).



Hysterosalpingogram showing bicornuate uterus.



Hysterosalpingogram showing a unicornuate uterus



Hysterosalpingogram showing intrauterine adhesion.

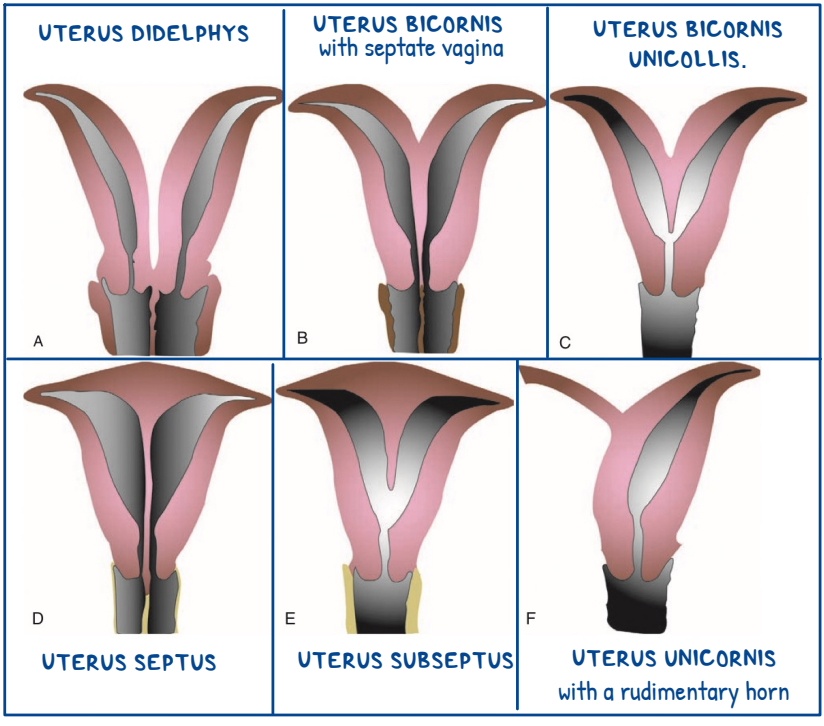
15. A 28 yr old woman is undergoing evaluation for successive recurrent pregnancy losses. On ultrasound, a mullerian anomaly is suspected. What is the best way to confirm this?

A. Transvaginal ultrasound
B. Hysterosalpingography
C. CECT
D. Hysteroscopy + laparoscopy

MULLERIAN DUCT ANOMALIES

WHO CLASSIFICATION OF MULLERIAN ANOMALIES

Class I	Mullerian agenesis (MRKH syndrome)
Class II	Unicornuate uterus
Class III	Didelphys uterus
Class IV	Bicornuate uterus
Class V	Septate uterus
Class VI	Arcuate uterus
Class VII	DES related abnormalities/T shaped uterus



DIAGNOSIS

- Internal examination reveals septate vagina and two cervices.
- Passage of a sound can diagnose two separate cavities.
- For exact diagnosis of the malformation, internal as well as external architecture of the uterus must be visualized.
- For this reason several investigations in different combinations are done, such as hysterography , hysteroscopy, laparoscopy , ultrasonography (vaginal probe) and magnetic resonance imaging (MRI).
- Direct visualisation by Hysteroscopy + laparoscopy is the best way to confirm.
- Ultrasonography and MRI are noninvasive procedures.
- Urological tract is also evaluated at the same time.
- The renal tract abnormality in association with Müllerian abnormality is about 40%. Skeletal system anomaly (12%) is also associated

16. A 28 yr P0A3 with recurrent 2nd trimester abortions was found to have a uterine septa on sono- salpingography. What is the best management option?

A. Dilatation and curettage
B. Laparoscopic metro Plast
C. Hysteroscopic septal resection
D. Laparotomy and metroplasty

MULLERIAN AGENESIS

Complete failure in the development of the mullerian ducts, resulting in absence of the fallopian tubes, uterus, and most of vagina (as 1/3 rd of vagina is formed by Mullerian duct).

Karyotype : 46XX
Phenotype : Female

CLINICAL FEATURES

- Primary amenorrhoea
- Secondary sexual characteristics are normal as ovaries are normal (because ovaries do not develop from mullerian duct but from genital ridge, so ovulation is also normal)
- P/V- Vagina is felt like a blind pouch and uterus is absent

INVESTIGATIONS

- Pelvis and abdomen ultrasound
- MRI gives more precise definition of pelvic viscera.
- Karyotype.
- Radiology—descending pyelography to delineate urinary tract anomalies.

Mullerian agenesis is associated with renal anomalies and skeletal anomalies-
Mayer Rokitansky kuster Hauser syndrome.

MANAGEMENT

- Repair of vaginal agenesis is done either by frank dilatation or vaginoplasty.
- Surgical management: Vaginoplasty either by McIndoe reed procedure or Williams vaginoplasty or amnion vaginoplasty.
- Hysteroscopic metroplasty is more commonly done.
  Resection of the septum can be done either by a resectoscope or by laser.
  Advantages are:
  (a) High success rate (80–89%),
  (b) Short hospital stay,
  (c) Reduced postoperative morbidity (infection or adhesions), and
  (d) Subsequent chance of vaginal delivery is high compared to abdominal metroplasty where cesarean section is mandatory.

16. A 28 yr woman being evaluated for infertility was found to have a uterine didelphys on 3D ultrasound.All are possible complications except?

A. Transverse lie
B. Endometriosis
C. Repeated abortion
D. Premature labor

UTERUS DIDELPHYS

There is complete lack of fusion of the Müllerian ducts with a double uterus, double cervix and a double vagina

CLINICAL FEATURES

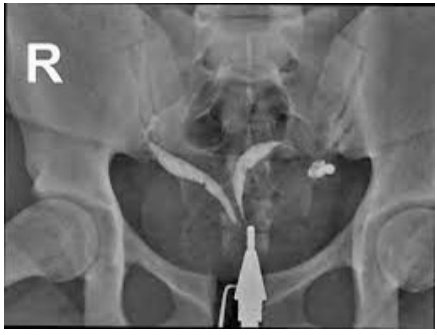
Gynecological

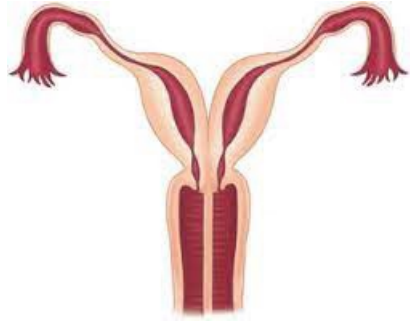
i. Infertility and dyspareunia are often related in association with vaginal septum.
ii. Dysmenorrhea in bicornuate uterus or due to cryptomenorrhea (pent up menstrual blood in rudimentary horn).
iii. Menstrual disorders (menorrhagia, cryptomenorrhea) are seen. Menorrhagia is due to increased surface area.

Obstetrical

i. Midtrimester abortion which may be recurrent.
ii. Rudimentary horn pregnancy may occur due to transperitoneal migration of sperm or ovum from the opposite side. Cornual pregnancy (ectopic) inevitably ends in rupture around 16th week.
iii. Cervical incompetence.
iv. Increased incidence of malpresentation– transverse lie in arcuate or subseptate, breech in bicornuate, unicornuate or complete septate uterus.
v. Preterm labor, IUGR, IUD.

vi. Prolonged labor—due to incoordinate uterine action.
vii. Obstructed labor—obstruction by the nongravid horn of the bicornuate uterus or rudimentary horn.
viii. Retained placenta and postpartum hemorrhage where the placenta is implanted over the uterine septum.





17. Reema Devi , a 28 yr newly married woman presents to your sub center for contraceptive advice. She is started on oral contraceptive pills. She presents after 2 weeks with a history of missing 4 tablets on different days in the first 2 weeks of cycle. What will you advise her?

A. Discontinue the packet and start an alternate method of contraception
B. Take 4 tablets the next day, continue the remaining packet, use additional contraception (condom) and give emergency pill if H/o intercourse in the last 72 hrs.
C. Take the next pill as soon as possible, continue the remaining packet, use additional contraception (condom) and give emergency pill if h/o intercourse in the last 72hrs.
D. Take the next pill as soon as possible and continue the remaining tablets

COMBINED ORAL CONTRACEPTIVES (PILLS)

- New users should normally start their pill packet on day one of their cycle.
- One tablet is to be taken daily preferably at bed time for consecutive 21 days.
- It is continued for 21 days and then have a 7 days break, with this routine there is contraceptive protection from the first pill.
- Next pack should be started on the eighth day, irrespective of bleeding (same day of the week, the pill finished).
- Thus, a simple regime of "3 weeks on and 1 week off" is to be followed. Packing of 28 tablets, there should be no break between packs. Seven of the pills are dummies and contain either iron or vitamin preparations.
- However, a woman can start the pill up to day 5 of the bleeding. In that case she is advised to use a condom for the next 7 days.

MISSED PILLS

- Normally there is return of pituitary and ovarian follicular activity during the pill-free interval (PFI) of 7 days.
- Breakthrough ovulation may occur in about 20 percent cases during the time. Lengthening of PFI due to omissions, malabsorption, or vomiting either at the start or at the end of a packet, increases the risk of breakthrough ovulation and therefore pregnancy.

MANAGEMENT

- When a woman forgets to take one pill (late up to 24 hours), she should take the missed pill at once and continue the rest as schedule. There is nothing to worry.
- When she misses two pills in the first week (days 1–7), she should take 2 pills on each of the next 2 days and then continue the rest as schedule.
  Extra precaution has to be taken for next 7 days either by using a condom or by avoiding sex.
- If 2 pills are missed in the third week (days 15–21) or if more than two active pills are missed at any time, another form of contraception should be used as back up for next 7 days as mentioned above. She should start the next pack without a break.
- If she misses any of the 7 inactive pills (in a 28-day pack only) she should throw away the missed pills. She should take the remaining pills one a day and start the new pack as usual.

18. A woman was on OCP. Later she contracted TB for which she was prescribed ATT according to NTEP and she was also advised to use additional contraceptive methods. This is because

a. Rifampicin induces metabolism of OCPs
b. Rifampicin is teratogenic
c. Isoniazid is teratogenic
d. OCP increases the metabolism of Rifampicin

DRUG INTERACTIONS OF OCP

- Effectiveness of some drugs (Aspirin, oral anticoagulants, oral hypoglycemics) are decreased and that for some other drugs (beta blockers, corticosteroids, diazepam, aminophylline) are increased by oral contraceptives.
- ADDITIONAL CONTRACEPTION: To ensure cent percent efficacy, additional mechanical contraceptives (usually condom) are to be used in the following circumstances :
  (1) When broad spectrum antibiotics like ampicillin, amoxicillin, tetracycline, doxycycline are used – as they impair the absorption of ethinyl estradiol.
  (2) When enzyme inducing drugs are used, e.g.
  (a) barbiturates
  (b) all antiepileptic drugs except sodium valproate and clonazepam
  (c) rifampicin
  (d) ketoconazole
  (e) griseofulvin
  (f) ritonavir
  (g) nevirapine
- Under such circumstances high dose preparations (ethinyl estradiol of 50 µg or more) are to be used to counter balance the increased liver metabolism.

19. A POA3 presents with a history of 3 abortions. The first at 8 weeks , 2nd at 11 weeks and the 3rd at 24 weeks with a history of early onset preeclampsia. Which of the following is the most likely cause of her abortions?

A. Syphilis
B. APLA
C. TORCH
D. GDM

ANTI PHOSPHOLIPID ANTIBODY SYNDROME(APAS)

- Anti phospholipid antibody syndrome(APAS)—is due to the presence of antiphospholipid antibodies.
- These are: lupus anticoagulant (LAC),
  Anticardiolipin antibodies (ACAs)
  B glycoprotein 1 antibodies (B - GPA1)
- Mechanisms of pregnancy loss in women with APAS are:
  (a) inhibition of trophoblast function and dierentiation,
  (b) activation of complement pathway,
  (c) release of local inflammatory mediators (cytokines, interleukins) and
  (d) thrombosis of uteroplacental vascular bed.
- Ultimate pathology is fetal hypoxia.
- Other pathological changes like placental vascular atherosis, intervillous and spiral artery thrombosis and decidual vasculopathy with fibrinoid necrosis lead to inadequate maternal blood supply to fetus.
- Common obstetric complications associated with antiphospholipid syndrome are:
  (a) recurrent fetal loss (10 weeks), preterm birth;
  (b) IUGR;
  (c) IUFD;
  (d) severe preeclampsia;
  (e) HELLP syndrome;
  (f) placental abruption;
  (g) recurrent thrombotic events and
  (h) thrombocytopenia.

Risk Factors for Preeclampsia	Etiopathological Factors for Preeclampsia
▪ **Primigravida**: Young or elderly (first time exposure to chorionic villi) ▪ **Family history**: Hypertension, preeclampsia ▪ **Placental abnormalities**: • Hyperplacentosis: Excessive exposure to chorionic villi—(molar pregnancy twins, diabetes) • Placental ischemia. ▪ **Obesity**: BMI >35 kg/m^2, Insulin resistance. ▪ **Pre-existing vascular disease** (p. 255). ▪ **New paternity** ▪ **Thrombophilias** [antiphospholipid syndrome, protein C, S deficiency, Factor V Leiden (p. 319)].	♦ Failure of trophoblast invasion (abnormal placentation) p. 37 ♦ Vascular endothelial damage ♦ Inflammatory mediators (cytokines) ♦ Immunological intolerance between maternal and fetal tissues (p. 720) • Coagulation abnormalities (p. 711) ♦ Increased oxygen free radicals ♦ Imbalance of angiogenic and antiangiogenic proteins (see p 257) ♦ Genetic predisposition (polygenic disorder) ♦ Dietary deficiency or excess

CAUSES OF INTRAUTERINE FETAL DEATH

A. Maternal (5–10%)

- Hypertensive disorders in pregnancy (p. 255)
- Diabetes in pregnancy (p. 330)
- Maternal infections (malaria, hepatitis, influenza, toxoplasma, syphilis) (p. 343)
- Hyperpyrexia (temp > 39.4°C)
- Antiphospholipid syndromes (APS) (p. 196) presence of lupus anticoagulant (LA), anticardiolipin antibodies (ACA) → decidual vasculopathy with fibrinoid necrosis, placental vascular atherosis and intervillous thrombosis → IUFD (p. 399)
- Thrombophilias: Factor V Leiden, protein C, protein S-deficiency, hyperhomocysteinemia (p. 399) Mechanism of IUFD is similar to (APS)
- Abnormal labor (p. 463) (prolonged or obstructed labor, ruptured uterus)
- Post-term pregnancy (p. 371)
- Systemic lupus erythematosus (p. 340)

B. Fetal (25–40%)

- Chromosomal abnormalities (p. 127)
- Major structural anomalies (p. 567)
- Infections (virus p. 349, bacteria p. 341, chorioamnionitis).
- Rh-incompatibility (p. 388)
- Non-immune hydrops (p. 571)
- Growth restriction

C. Placental (20–35%)

- Antepartum hemorrhage p. 282 : Both placenta previa and abruptio placentae can cause fetal death by producing acute placental insufficiency (p. 289, 299).
- Cord accident (prolapse, true knot, cord round the neck) p. 460
- Twin transfusion syndrome (TTTS) (p. 240).
- Placental insufficiency (p. 533).

D. Iatrogenic

- External cephalic version (p. 663)
- Drugs (quinine beyond therapeutic doses)

E. Idiopathic (25–35%)

- Cause remains unknown even with thorough clinical examination and investigations

CAUSES OF MISCARRIAGE IN APAS ARE:

(a) inhibition of trophoblast proliteration and function,
(b) release of local inflammatory mediators (cytokines) through complement pathway,
(c) spiral artery and placental intervillous thrombosis and
(d) decidual vasculopathy with "fibrinoid necrosis "

TREATMENT

- Women are treated with low-dose aspirin (50 mg/day) and heparin (5,000 units SC twice daily) up to 34 weeks.
- Unfractionated heparin and low molecular weight heparin (LMWH) are equally eective and safe.

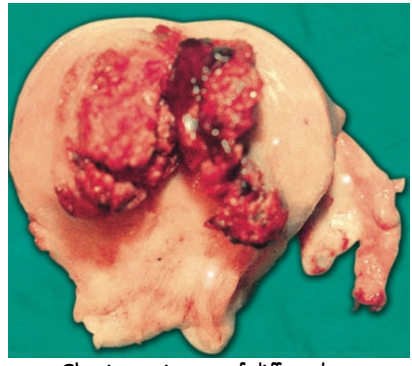
20. A 25 yr woman had evacuation of molar pregnancy done 6 months earlier. She now presents with General ill health, breathlessness, cough and irregular vaginal bleeding. On chest X-ray, there are canon ball metastasis. Her beta hCG levels are high. Which is the best management option?

A. Multidose inj Methotrexate and Inj folinic acid
B. Hysterectomy
C. Single dose inj methotrexate
D. Multiple drug regime EMA - CO

The patient is having Stage 1 choriocarcinoma and the treatment is Multidose inj Methotrexate and Inj folinic acid

CHORIOCARCINOMA

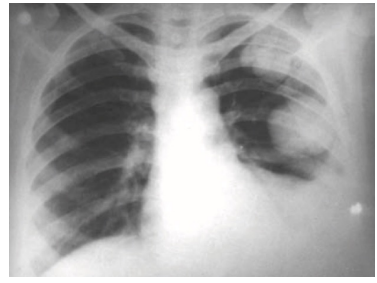
- Highly malignant tumor arising from the chorionic epithelium.
- The primary site is usually anywhere in the uterus.
- The common sites of metastases are lungs (80%), anterior vaginal wall (30%), brain (10%), liver (10%) and others.
- Trophoblastic disease following a normal pregnancy is either choriocarcinoma or PSTT and not a benign or invasive mole.



Choriocarcinoma of diffuse type

CLINICAL FEATURES

- Persistent ill health.
- Irregular vaginal bleeding, at times brisk.
- Continued amenorrhea.
- Other symptoms due to metastatic lesions are:
  Lung:Cough, breathlessness, hemoptysis.
  Vaginal: Irregular and at times brisk hemorrhage.
  Cerebral: Headache, convulsion, paralysis or coma.
  Liver: Epigastric pain, jaundice.
- Patient looks ill.
- Pallor of varying degrees.
- Physical signs are evident according to the organ involved.
- Bimanual examination reveals subinvolution of the uterus. There may be a purplish red nodule in the lower-third of the anterior vaginal wall. Unilateral or bilateral enlarged ovaries may be palpable through lateral fornices.



Cannon ball shadow in the left apical and mid region of the lung with pleural effusion in choriocarcinoma

DIAGNOSTIC CRITERIA FOR POST-MOLAR GTN (FIGO)

levels of serum βhcG are followed up.

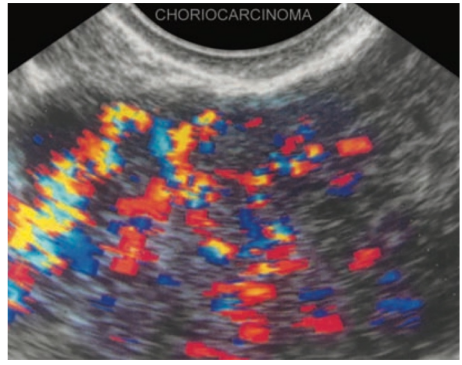
1. ≥ four values of plateaued hcG (± 10%) over at least 3 weeks time.
2. A rise of hcG of > 10% for > 3 values over at least 2 weeks time.
3. Histologic diagnosis of choriocarcinoma.
4. Persistence of hcG beyond 6 months of mole evacuation.

FIGO ANATOMIC STAGING FOR GTT	
Stage I	The lesion is confined to the uterus.
Stage II	The lesion spreads outside the uterus but is confined to the genital organs.
Stage III	The lesion metastasizes to the lungs.
Stage IV	The lesion metastasizes to sites such as brain, liver or gastrointestinal tract.

WHO PROGNOSTIC SCORING SYSTEM OF GESTATIONAL TROPHOPLASTIC DISEASE AS MODIFIED BY FIGO (2000)

PARAMETER	SCORE			
	0	*1*	*2*	*4*
Age (years)	< 40	> 40	—	—
Antecedent pregnancy	Mole	Abortion	Term	—
Interval (month)‡	< 4	4–6	7–12	≥ 13
Pretreatment hCG (IU/L)	$< 10^3$	10^3–10^4	10^4–10^5	$> 10^5$
Largest tumor (cm)	< 3	3 – 5	> 5	—
Site of metastases	Lung Pelvis	Spleen Kidney	GI tract Liver	Brain
No. of metastases detected	—	1–4	5–8	> 8
Prior chemotherapy	—	—	Single drug	Multiple drugs

Total score: < 6 is low risk and a total score ≥ 7 is high risk.
‡ **Interval:** Time between antecedent pregnancy and start of chemotherapy.



Transvaginal color Doppler scan of chorio-carcinoma showing randomly dispersed vessels

MANAGEMENT OF GTN	
A. Stage I Low risk GTN	• Single agent chemotherapy.
High risk or Resistant	• Combination therapy • Family completed→ hysterectomy.
B. Stage II and III Low risk GTN	• Single agent chemotherapy. • Hysterectomy (family completed) to reduce tumor mass.
High risk or Resistant	• Combination therapy • Hysterectomy — to reduce (trophoblastic) tumor mass.
C. Stage IV	• Combination chemotherapy. • Surgery (hepatic resection, craniotomy). • Radiation (cerebral metastasis).

TREATMENT

- Chemotherapy is now the mainstay in the treatment.
- Whether a single agent or multidrug regimen is to be used, depends on the risk factors present.
- In general, patients with non-metastatic (low risk) and good prognosis disease are treated effectively with single agent therapy (Methotrexate or Actinomycin).
- The patients with poor prognosis metastatic disease should be treated with combination drug regimen (EMACO regimen).

TABLE 23.19 SINGLE DRUG REGIMEN IN LOW-RISK CASES

Methotrexate	1–1.5 mg/kg	IM/IV	Days 1, 3, 5 and 7
Folinic acid	0.1–0.15 mg/kg	IM	Days 2, 4, 6 and 8

The courses are to be repeated at interval of 7 days

21. A 55 Yr old lady with 5 children presents with leakage of urine on coughing. On examination, there is second degree uterine prolapse and cystocele. What is the most likely urinary abnormality?

A. Overflow incontinence
B. Urge incontinence
C. Stress incontinence
D. Neurogenic bladder

STRESS INCONTINENCE	URGE INCONTINENCE (SENSORY)	DETRUSOR INSTABILITY
• leakage of urine coincides with stress • no prior urge to void • amount – small • Patient – fully aware • micturition – normal	• Unable to control the escape of urine, once there is urge to void • amount – large • Patient – aware of the urge • Urgency and frequency	• May occur abruptly even without a full bladder • amount – large • Patient – not aware • frequency and nocturia

GENUINE STRESS INCONTINENCE (GSI)

Involuntary urethral loss of urine when the intravesical pressure exceeds the maximum urethral pressure in the absence of detrusor activity according to the international continence society (ICS)

ETIOPATHOGENESIS

- Bladder Neck Descent (Including urethral hypermobility) (75-80%)

  Due to loss of integrity of the fibromuscular tissue that supports the bladder neck & urethra

- Intrinsic Sphincter Defect (20-25%)

  When the sphincter mechanism is compromised and fails to close the urethro vesical junction.

RISK FACTORS

- Gender - UI is more common in women than men
- Hypoestrogenism
- Parity - Higher incidence of UI in multiparous females
- Repeated child birth
- Underlying medical conditions like diabetes, obesity parkinsonism, and multiple sclerosis
- Previous pelvic surgery with resultant scar formation
- Pharmacological agents like diuretics, caffeine, and anticholinergics
- Chronically increased intra- abdominal pressure as in COPD

CLINICAL FEATURES

Escape of urine with coughing, sneezing or laughing.

The loss of urine has got the following features:

- Brief and coincides precisely to the period of raised intra-abdominal pressure.
- Unassociated with a desire to pass urine.
- Rarely, occurs in supine position or during sleep.
- Patients are fully aware of it.
- The amount of loss is small.

It is the most common variety of urinary incontinence.

Grade I	Incontinence with cough or sneeze
Grade II	With mild exercise
Grade III	Even with change of posture

DIAGNOSIS OF GSI

- Clinical stress test—positive.
- Pad test—positive.
- Midstream urine analysis—normal.
- Urinary diary (volume frequency chart).
- Uroflowmetry—normal.
- Cystometry—normal.
- Significant lowering of urethral closure pressure during strain.
- Leak-point pressure (Valsalva) test—positive.
- Cystourethroscopy—negative finding.
- Videocystourethrography—bladder neck funneling.
- Transvaginal endosonography—altered anatomical relationship (descent) of urethrovesical junction and bladder base.

TEST FOR DETECTING STRESS INCONTINENCE

- **Bonney's test:** In this test the patient is asked to insert 2 fingers , in the paraurethral region causing lifting of the bladder neck and then the patient is asked to cough. If SUI gets corrected, then it is due to bladder neck desent. If SUI persists, it is due to sphincter defect.
- **Marchetti test:** is same as Bonney's test, except that instead of fingers, two Allis forceps are used.
- **Q tip test:** A sterile cotton swab is introduced into the level of bladder neck. Then the patient is asked to strain. Marked upward elevation of cotton tip (>30) indicates urethra hypermobility. Goniometer is used to measure the urethero - vesicle angle.

MANAGEMENT

CONSERVATIVE

1st line of treatment

- Young woman
- Frail, old woman
- Postpartum, previous failed surgery

1. Kegel pelvic floor exercises x 4-6 months
2. Electric/magnetic stimulation for nerve damage, magnetic stimulation
3. Artificial urinary sphincter in neurological condition
4. Vaginal cones

DRUGS

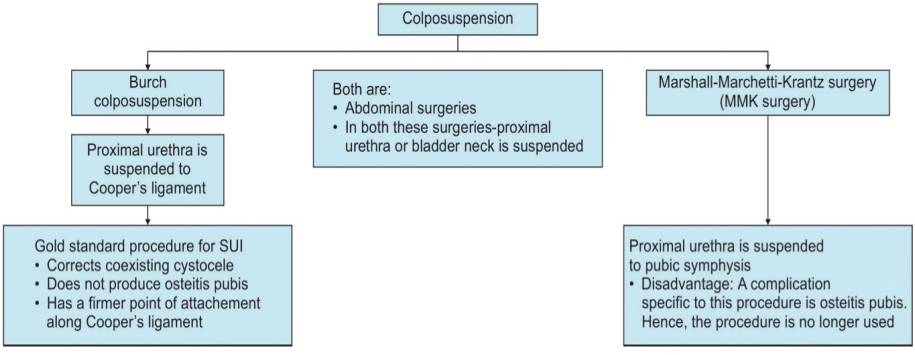
- Oestrogen cream in menopausal woman
- Venlafaxine 75 mg daily
- Imipramine 10-20 mg BD

MINIMALLY INVASIVE SYNTHETIC MIDURETHRAL SLINGS

- Tension-free vaginal table (TVT)
- Tension free obturator tape (TOT)

Both are vaginal surgeries
Both are day care surgeries
In both, midurethra is suspended

SURGERY



22. A 25yr married female who is Anxious about conception comes to the opd with C/o profuse vaginal discharge. There is no H/o itching and her menstrual cycles are regular. The most likely diagnosis is.

A. Candida
B. Trichimonas
C. Bacterial vaginosis
D. Physiological

LEUCORRHEA

Leucorrhea is strictly defined as an excessive normal vaginal discharge.

- The excess secretion is evident from persistent vulval moistness or staining of the undergarments (brownish yellow on drying) or need to wear a vulval pad.
- It is nonpurulent and nonoffensive.
- It is nonirritant and never causes pruritus.

PHYSIOLOGIC DISCHARGE

The normal secretion is expected to increase in conditions when the estrogen levels become high. Such conditions are :

- During puberty—Increased levels of endogenous estrogen lead to marked overgrowth of the endocervical epithelium which may encroach onto the ectocervix producing congenital ectopy (erosion) → increased secretion.
- During menstrual cycle

  Around ovulation—Peak rise of estrogen → increase in secretory activity of the cervical glands. Premenstrual pelvic congestion and increased mucus secretion from the hypertrophied endometrial glands.
- Pregnancy—There is hyperestrinism with increased vascularity. This leads to increased vaginal transudate and cervical gland secretion.
- During sexual excitement, when there is abundant secretion from the Bartholin's glands.

	BACTERIAL	CANDIDIASIS	TRICHOMONAS
Organism	Gardenella vaginalis (Mycoplasma mobilincus, Ureaplasma, PeptoStreptococcus)	Candida albicans > Candida galbrata > Candida tropicalis	Trichomonas vaginalis (flagellated protozoa)
pH	> 4.5	< 4.5	5-6
Complaints	Foul smelling dirty white discharge, No inflammation, no itching	Intense pruritis, Curdy white discharge (cottage cheese like)	Profuse frothy greenish yellow discharge, Dysuria, Dyspareunia (Strawberry vagina)
IOC (Saline microscopy)	'Clue cells', i.e. vaginal epithelial cells to which bacteria are adhered seen	Pseudohyphae seen	Typical motile flagellated trichomonas
Gold standard investigation	Gram stain, Nugent score is calculated by the presence of lactobacillus	Culture on sabouraud's medium or Nickerson medium	Culture on—Feinberg-whittington media or Diamond media
Amine test/ Whiff test 10% KOH added to discharge	Positive, fishy odour or amine like odour obtained.	Negative	Maybe positive/or negative
T/t Nonpregnant females	Metronidazole (500 mg BD x 7 days) or clindamycin	Fluconazole/miconazole topically or orally (150 mg, single dose)	Metronidazole (2 gm single dose oral)
T/t Pregnancy	Metronidazole (250 mg TDS x 7 days) avoided in first trimester	Topical azole antifungals avoided in first trimester	Metronidazole (250 mg TDS x 7days)
Rx of male partner	Not needed as BV is not an STD	If partner has symptoms then treatment needed	Always done as TV is an STD

23. A 28 yr woman with infertility presents to you. On ultrasound there is an intramural fibroid measuring 7 x 5 cm near the right Cornua and another intramural fibroid measuring 5 x 5 cm near the left cornua. HSG reveals bilateral tubal block at the region of the tubal Ostia. Semen parameters are normal and there is no ovulatory disturbance. What is the best management for this woman?

A. GnRH analogues
B. Laparoscopic myomectomy
C. ART
D. Uterine artery embolization

FIBROIDS

Fibroid is the commonest benign tumor of the uterus and also the commonest benign solid tumor in female.

CLINICAL FEATURES

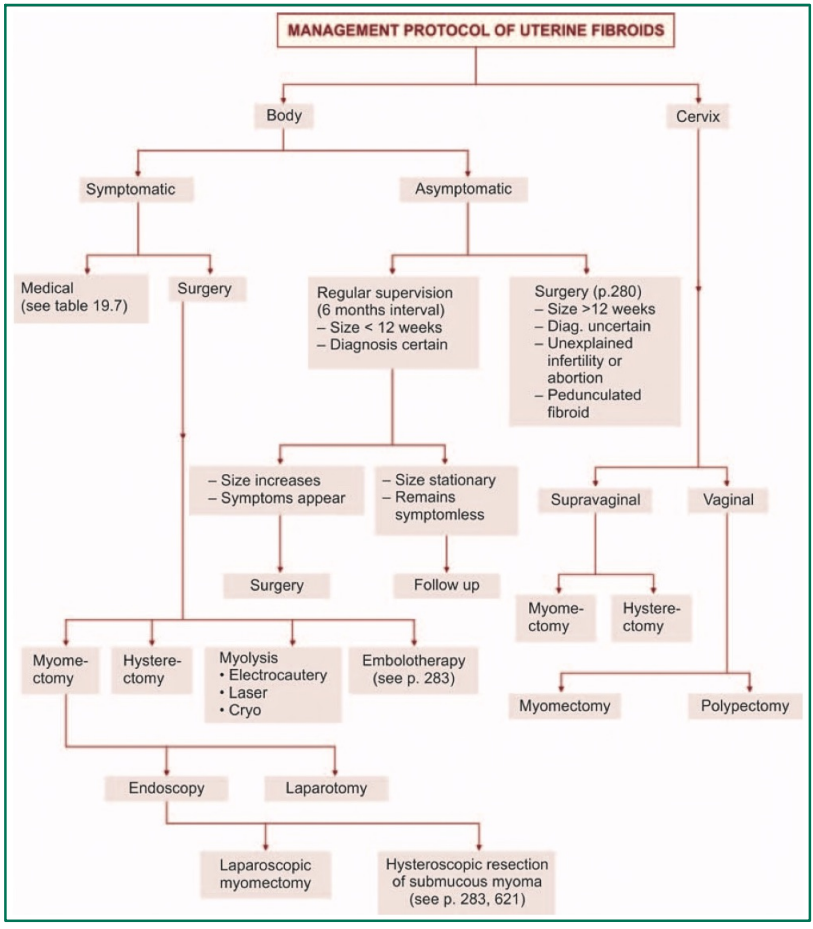
- Asymptomatic—majority (75%)
- Menstrual abnormality: Menorrhagia,metrorrhagia.
- Dysmenorrhea
- Dyspareunia
- Infertility
- Pressure symptoms
- Recurrent pregnancy loss (miscarriage, preterm labor)
- Lower abdominal or pelvic pain
- Abdominal enlargement.

SECONDARY CHANGES AND COMPLICATIONS

- Hyaline change, cystic degeneration and atrophy
- Calcareous degeneration, osseous degeneration
- Red degeneration
- Sarcomatous change
- Torsion, haemorrhage
- Infection/ulceration
- Inversion of the uterus
- Endometrial carcinoma associated with fibromyoma
- Endometrial and myohyperplasia
- Accompanying adenomyosis
- Parasitic fibroid

INVESTIGATIONS

- M/c investigation done in fibroids or IOC is USG. (It is most readily available, least cost-effective but not as accurate as MRI at determining the precise location or size of fibroids especially in larger uteri or those with multiple fibroids)
- Best investigation to detect a small submucous fibroid is hysteroscopy.



MYOMECTOMY is the enucleation of myomata from the uterus leaving behind a potentially functioning organ capable of future reproduction.

INDICATIONS	CONTRAINDICATIONS
• Persistent uterine bleeding despite medical therapy. • Excessive pain or pressure symptoms. Size >2 weeks, woman desirous to have a baby. • Unexplained infertility with distortion of the uterine cavity. • Recurrent pregnancy wastage due to fibroid. • Rapidly growing myoma during follow-up. Subserous pedunculated fibroid.	• Infected fibroid. • Growth of myoma after menopause. • Suspected malignant change (sarcoma). • Parous women where hysterectomy is safer and is a definitive treatment • Function less fallopian tubes (bilateral hydrosalpinx, tubo-ovarian mass) • Pelvic or endometrial tuberculosis. • During pregnancy or during cesarean section.

24. A 39 yr woman presents to the medicine opd with complaints of fatigue and lethargy. She gives a history of delivering a 3.5 kg baby 5 years earlier following which she received multiple blood transfusions. She never resumed menstruation following delivery and also had failure of lactation. Which is the most likely diagnosis?

A. Euthyroid sick syndrome
B. Hypothyroid
C. Sheehan syndrome
D. Late onset blood transfusion reaction

SHEEHAN'S SYNDROME

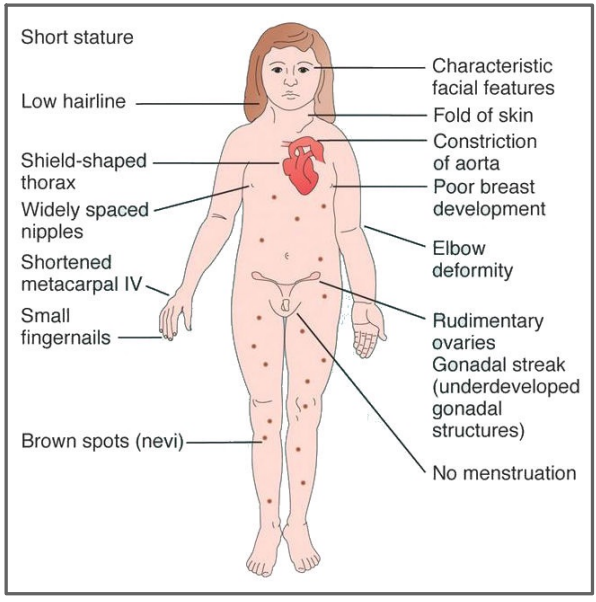
- Post-partum pituitary infarction occurs because the enlarged pituitary gland of pregnancy is more vulnerable to ischaemia of post-partum haemorrhage and systemic hypotension.
- Failure to lactate is the earliest symptom, failure to regain menstruation after delivery and breast involution.
- Uncommonly, it can present acutely with circulatory collapse, severe hyponatraemia, diabetes insipidus, hypoglycae mia, congestive cardiac failure, psychosis, finally leading to coma and death.
- MRI may show hypertrophic pituitary in early stages, but later, atrophic pituitary and empty sella develop. It also excludes a pituitary mass.
- Treatment is substitution of deficient hormones.

CAUSES OF SECONDARY AMENORRHEA		
	DISORDERS OF OUTFLOW TRACT/ UTERUS	Acquired obstruction (gynatresia) of cervical canal causing severe stenosis or atresia following electrocauterization, chemical burns, cervical amputation in Fothergill's repair, conization, CIN, or genital tuberculosis. Ashermann's syndrome Following excessive curettage or endometrial tuberculosis
	DISORDERS OF OVARY	Ovary Tumor – Masculinizing tumors/PCOD Trauma – Surgical extipiration/Radiotherapy Infections – Mumps, Tuberculosis rarely pyogenic infections Premature ovarian failure
	PITUITARY	Hyperprolactinemia/prolactin tumor/prolactinoma Insufficiency as in Simmond's disease, Sheehan syndrome. Empty sella syndrome , Infiltrative disease
	HYPOTHALAMUS	GnRH deficiency , Vigorous exercise/excessive stress Weight loss, Eating disorders – anorexia and Bulimia Tumor(includingbcraniopharyngioma, germinoma, endodermal sinus tumor, eosinophilic granuloma, and glioma) Radiation, Pseudocyesis Infection (TB), Infiltrative disease– sarcoidosis

25. A 12 yr girl is brought to the OPD by her mother. She is concerned that she is shorter than her peers. On examination there is ptosis on the right side , shield like chest and a webbed neck. On evaluation, which of the following would you expect to find?

A. Ultrasound showing streak ovaries and a small uterus
B. ECHO showing tricuspid stenosis
C. Ultrasound showing hepatomegaly and altered echo texture
D. Ultrasound showing single kidney

TURNERS SYNDROME



TURNER SYNDROME	NOONAN SYNDROME
• No Ptosis • Heart disease associated - Coarctation of aorta - Bicuspid aortic valve (most common) • IQ normal • Amenorrhea , streak ovary	• Ptosis is common in Noonan • Heart disease associated - Pulmonary stenosis • IQ low • Males- cryptotchidis • females are relatively normal

CLINICAL FEATURES

The syndrome is characterized by:

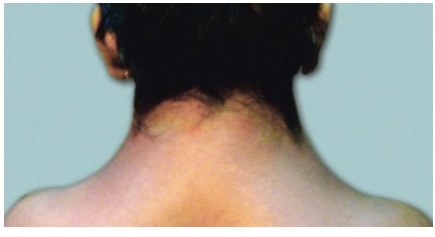
- short stature (height < 150 cm),
- webbing of the neck,
- cubitus valgus,
- broad shield chest,
- low hair line on the neck,
- lymphedema,
- short fourth metatarsals
- poor development of secondary sex characters
- mentally retarded and often associated with coarctation of aorta

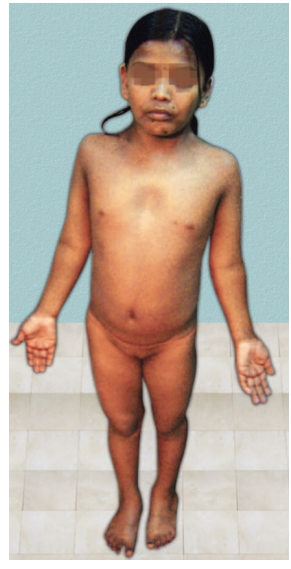
- Cardiac (coarctation of aorta). Renal anomalies (horseshoe kidneys) and multiglandular autoimmune disorders are common.
  45X is the commonest chromosomal abnormality and 99% of fetuses are aborted.
- Vagina, uterus and fallopian tubes are present.
- The uterus is small but is responsive to exogenous estrogen.
- Gonads are 'streaks' (fibrous tissue) without any follicle nor any potentiality to produce hormone (chromosomally incompetent ovarian failure).
- Associated autoimmune disorders like Hashimoto's thyroiditis, Addison's disease, hypothyroidism are common

INVESTIGATIONS

- Sex chromatin study is negative.
- Karyotype is 45, XO.
- Serum E2 is very low.
- Serum FSH and LH are elevated.
- Autoantibodies maybe present

Thus, there is hypergonadotrophic hypogonadism state.





17 Years old girl with Turner's syndrome (45, X). Characteristic somatic abnormalities seen are: short stature (130 cm), shield chest, widely apart nipples, cubitus vulgus, short neck and absent secondary sex characters.

1. All are increased in Iron Deficiency Anemia except ?

A. TIBC

B. Soluble Tranferrin Receptor

C. Transferrin Saturation

D. Iron Protopophyrin

IRON DEFICIENCY ANEMIA

PATHOPHYSIOLOGY

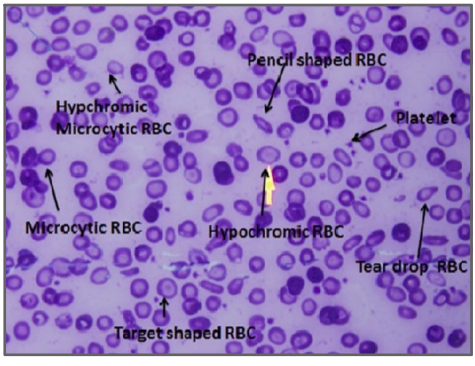
- Diminished dietary iron absorption in the proximal small intestine or excessive loss of body iron result in iron deficiency.
- Iron is essential for multiole metabolic processes, including oxygen transport, DNA synthesis, and electron transport.
- Iron defidencv anemia can result in diminished growth and leantlng and have serious consequences in children.

CLINICAL FEATURES

- Behavioral symptoms,such as irritability and anorexia, precede weakness, fatigue, leg cramps, breathlessness and tachycardia.
- Congestive heart failure and splenomegaly may occur with severe persistent,untreated iron deficiency
- Angular stomatitis, glossitis, koilonychia and platynychia are seen in severe iron deficiency .

LABORATORY DIAGNOSIS

- Peripheral smear shows that red cells are microcytic and hypochromic with anisocytosis poikilocytosis and increased red cell distribution width (RDW).
- MCV and MCHC are reduced.
- Red cell number is reduced.
- Serum iron is reduced, total iron binding capacity (TIBC) is increased and transferrin is reduced to less than 16% (normal 25-50%).
- Serum ferritin : Reduction in occurs early.
- High free erythroprotoporphyrin(FEP) is seen before anemia develops.



TREATMENT

- Oral iron preparations : taken on an empty stomach or in between meals for best absorption
  - Oral preparation is Ferrous sulfate (20% elemental iron).
- The dose for treatment of is 3-6 mg/kg/day.
- Reticulocyte count increases 72hrs - 96hrs after initiating therapy
- After correction of anemia, oral iron should be continued for 4-6 months to replenish iron stores.

Reference :
Nelson Textbook of Pediatrics 20th Ed

2. A 10 yr old child with generalised edema,Mother complains of child passing frothy urine. Serum cholesterol 238mg/dl.On Urine analysis : Protein 3+ and Urine microscopy revealed fat droplets.What is the most probable diagnosis ?

A. Good pasture syndrome
B. Urinary Tract Infection
C. Nephrotic syndrome
D. Nephritic syndrome

- From the given history (child passing frothy urine)and urine examination findings most probable diagnosis is Nephrotic syndrome.

NEPHROTIC SYNDROME

- The triad of clinical findings
  i. Arising from the large urinary losses of protein are hypoalbuminemia (≤2.5 g/dL),
  ii. Edema, and
  iii.Hyperlipidemia (cholesterol >200 mg/dL).

ETIOLOGY

- Nephrotic syndrome have a form of primary or idiopathic nephrotic syndrome.
- Glomerular lesions associated with idiopathic nephrotic syndrome include minimal change disease (the most common), focal segmental glomerulosclerosis, membranoproliferative glomerulonephritis, C3 glomerulopathy, and membranous nephropathy.
- Secondary to systemic diseases such as :
  - Systemic lupus erythematosus,
  - Henoch-Schönlein purpura, malignancy (lymphoma and leukemia), and
  - Infections (hepatitis, HIV, and malaria)

CLINICAL CONSEQUENCES

- Edema : Most common presenting symptom of children
- Hyperlipidemia :
  - Increase in cholesterol, triglycerides, low-density lipoprotein, and very-low-density lipoproteins.
- Increased Susceptibility to Infections :
  - Such as cellulitis, spontaneous bacterial peritonitis, and bacteremia.
  - Spontaneous bacterial peritonitis presents with fever, abdominal pain, and peritoneal signs.
  - Pneumococcus is the most frequent cause of peritonitis, Gram-negative bacteria also are associated with a significant number of cases.
  - Fever or other signs of infection must be evaluated aggressively, with appropriate cultures drawn, and should be treated promptly and empirically with antibiotics.
  - Peritoneal leukocyte counts >250 are highly suggestive of spontaneous bacterial peritonitis.
- Hypercoagulability

Reference :
Notespaedia Pediatrics High Yield Pg : 234
Nelson Textbook of Pediatrics 20th Ed.

DIAGNOSIS

- Confirmed by urinalysis with first morning urine protein :
  - Creatinine ratio and serum electrolytes, blood urea nitrogen, creatinine, albumin, and cholesterol levels; evaluation to rule out secondary forms of nephrotic syndrome (children ≥10 yr): complement C3 level, antinuclear antibody, double-stranded DNA and hepatitides B and C, and HIV in high-risk populations; and kidney biopsy (for children ≥12 yr, who are less likely to have MCNS).
- The urinalysis reveals 3+ or 4+ proteinuria, and microscopic hematuria is present in 20% of children.
- A spot urine protein : creatinine ratio should be >2.0.
- The serum creatinine value is usually normal.
- The serum albumin level is <2.5 g/dL, and serum cholesterol and triglyceride levels are elevated.
- Serum complement levels are normal.
- A renal biopsy is not routinely performed if the patient fits the standard clinical picture of MCNS.

TREATMENT

- Corticosteroids are the mainstay of therapy for MCNS.
- For Initial Episode of Nephrotic Syndrome :
  - Prednisone or prednisolone should be administered as a single daily dose of 60 mg/m2/day or 2 mg/kg/day to a maximum of 60 mg daily for 4-6 wk followed by alternate-day prednisone (starting at 40 mg/m2 qod or 1.5 mg/kg qod) for a period ranging from 8 wk to 5 mo, with tapering of the dose.
- Response : Defined as the attainment of remission within the initial 4 wk of corticosteroid therapy.
- Remission : Consists of a urine protein : creatinine ratio of < 0.2 or < 1+ protein on urine dipstick for 3 consecutive days.
- Alternative Therapies to Corticosteroids :
  - Cyclophosphamide prolongs the duration of remission and reduces the number of relapses in children with frequently relapsing and steroid-dependent nephrotic syndrome.
  - Calcineurin inhibitors (cyclosporine or tacrolimus) are recommended as initial therapy for children with steroid-resistant nephrotic syndrome.
  - Mycophenolate can maintain remission in children with steroid-dependent or frequently relapsing nephrotic syndrome.
  - Levamisole, an antihelmintic agent with immunomodulating effects that has been shown to reduce the risk of relapse
- Immunizations :
  - Full pneumococcal vaccination (with the 13-valent conjugant vaccine and 23-valent polysaccharide vaccine) and influenza vaccination annually to the child and their household contacts; defer vaccination with live vaccines until the prednisone dose is below either 1 mg/kg daily or 2 mg/kg on alternate days.
  - Live virus vaccines are contraindicated in children receiving corticosteroid- sparing agents

Typical Features	Nephrotic Syndrome	Nephritic Syndrome
Onset	Insidious	Abrupt
Edema	++++	++
Blood Pressure	Normal	Raised
Jugular Venous Pressure	Normal/Low	Raised
Proteinuria	++++	++
Hematuria	May/may not occur	+++
Red blood cell casts	Absent	Present
Serum Albumin	Low	Normal/Slightly reduced

GOODPASTURE DISEASE

Characterized by pulmonary hemorrhage and glomerulonephritis.

Disease results from :

- Attack on these organs by antibodies directed against certain epitopes of type IV collagen, located within the alveolar basement membrane in the lung and glomerular basement membrane (GBM) in the kidney.

CLINICAL MANIFESTATIONS

- Hemoptysis
- Concomitant renal manifestations include :
  - Acute glomerulonephritis with hematuria,
  - Proteinuria, and
  - Hypertension

DIAGNOSIS

- Serum antiGBM antibody is present and complement C3 level is normal.
- Antineutrophilic cytoplasmic antibody levels can be found to be elevated along with the anti GBM antibody.
- PBS : normocytic and normochromic anemia.
- Urinalysis : Reveal hematuria and proteinuria
- Elevated blood urea nitrogen and creatinine.
- Chest radiography : Scattered patches of pulmonary infiltrates.
- PFT : Reveals a restrictive defect with reduction in FVC and FEV at 1-second.
- Bronchoscopy and Bronchoalveolar Lavage :
- Lung biopsy :
  - Reveals capillaritis from neutrophils, hemosiderin-laden macrophages, type II pneumocyte hyperplasia, and interstitial thickening at the level of the alveolus.
- Immunofluorescence : along the basement membrane in a linear pattern.

TREATMENT

- Corticosteroids coupled with oral cyclophosphamide.
- Daily plasmapheresis

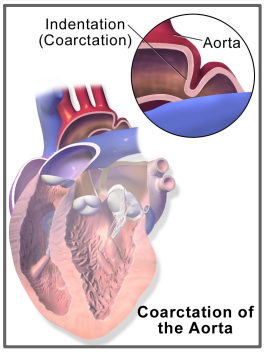
Reference :
Nelson Textbook of Pediatrics 20th Ed. Pg : 2507

3. A 10yr old child presents with upper limb hypertension,lower limb pulses are not palpable.Chest X rays showed Notching of Ribs.What is the most probable diagnosis ?

A. Atrial Septal Defect
B. Bicuspid Aortic Valve
C. Patent Ductus Arteriosus
D. Coarctation of Aorta

- With the given signs and Chest X ray showing Rib notching is suggestive of Coarctation of Aorta

COARCTATION OF THE AORTA



- Located at the junction of the arch with the descending aorta.
- It is a sharp indentation involving the anterior, lateral and posterior wall of the aorta,medial wall is spared.
- Site : May be distal or proximal to the ligamentum arteriosus and also left subclavian artery.
- Common association : Bicuspid aortic valve

CLINICAL FEATURES

- Present with left ventricular dysfunction and heart failure.
- The only symptoms in uncomplicated coarctation :
  i. Intermittent claudication,
  ii. Pain and weakness of legs and
  iii. Dyspnea on running.
- Examination shows delayed and weak femorals and strong brachial arteries.
- Heart size remains normal with a forcible or heaving left ventricular apex.
- A systolic thrill may be palpable in the suprasternal notch.
- Prominent arterial pulsations in the suprastemal notch and the carotid vessels.
- S 1 is accentuated (sometimes followed by a constant ejection click).
- S 2 is normally split with a loud aortic component.
- Continuous murmurs : Audible over collaterals in the chest wall but are uncommon.
- Aortic ejection systolic murmur and/or a regurgitation murmur may be present because of the commonly associated bicuspid aortic valve.

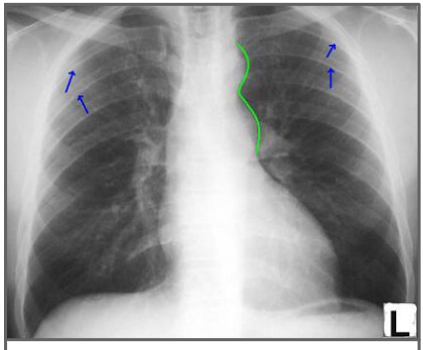
COMPLICATIONS

- Rupture of berry intracranial aneurysm and dissection of aorta.
- Infective endarteritis may occur.

Reference :
Nelson TEXTBOOK of PEDIATRICS 20th Ed Pg: 2205 , Notespaedia Pediatrics High Yield Pg : 149
GHAI Essential Pediatrics 9th Ed Pg : 428

INVESTIAGTIONS

- ECG : shows left ventricular hypertrophy.
- Chest X-ray :
  - Normal sized heart with prominent ascending aorta and the aortic knuckle.
  - The characteristic notching of the lower borders of ribs is seen beyond the age of 10years.



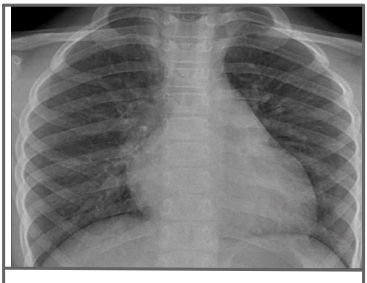
Green : Aortic knuckle
Blue Arrow : Notching of Ribs

TREATMENT

- In newborns and infants : Surgery is preferred
- In older children, adolescents and adults : Balloon dilation with or without stenting
- Prostaglandin E1 : Used to maintain ductal patency prior to surgery in first few weeks of life.

ATRIAL SEPTAL DEFECT

- ASD is classified as follows :
  i. Fossa ovalis ASD
  ii. Sinus venosus ASD
  iii. Ostium primum ASD
  iv. Coronary Sinus ASD



Chest X-ray of a child with large ASD and hemodynamically significant left-to-right shunt.

FINDINGS

- Pretricuspid shunt.
- Enlarged right ventricle results in a parasternal impulse.
- Ejection systolic murmur
- ECG of ostium secundum ASD : Right axis deviation and right ventricular hypertrophy.
- Chest X-ray :
  - Shows mild to moderate cardiomegaly, Right Atrial and Right Ventricular enlargement, prominent main pulmonary artery) segment, a relatively small aortic shadow and plethoric lung fields.

TREATMENT

- Closed percutaneously in the catheterization laboratory with occlusive devices.
- Closure is recommended before school entry to prevent late complications.

PATENT DUCTUS ARTERIOSUS

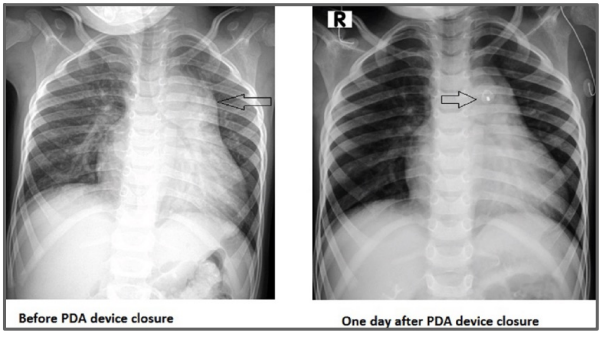
- Is a communication between the pulmonary artery and the aorta.
- The aortic attachment of the ductus arteriosus is just distal to the left subclavian artery.

CLINICAL FEATURES

- Results in a left-to-right shunt from the aorta to the pulmonary artery.
- Continuous murmur
- Late A2 (S2 may be paradoxically split)
- History of effort intolerance, palpitation and frequent chest infections.
- The cardiac impulse is hyperkinetic with a left ventricular type of apex.
- A systolic or a continuous thrill may be palpable at the second left intercostal space.
- 3rd sound at the apex, followed by a delayed diastolic murmur in large shunts.

INVESTIGATIONS

- ECG :
  - Shows normal axis with left ventricular dominance or hypertrophy.
  - Deep Q waves in left chest leads with tall T waves
- Chest X Ray :
  - Exhibits cardiac enlargement with a left ventricular silhouette; cardiac size depends on the size of the left-to-right shunt.
  - Ascending aorta and the aortic knuckle are prominent; pulmonary vasculature is plethoric.



TREATMENT

- Indomethacin or ibuprofen is likely to be effective before the age of 2 weeks in preterm newborns.
- Newborns not responding to these agents require surgical ligation.
- PDA in term infants may close spontaneously as late as one month after birth.
- Catheter-based treatment (occlusive devices or coils).

4. 10yr old child presents with myoclonus jerk,decrease performance in school.There is history of fever at the age of 1 yr with rash.Above features are suggestive of :

A. Subacute Sclerosis Panencephalitis
B. Medial Temporal Sclerosis
C. Absence seizures
D. JME

- History of fever at 1 yr of age with rash most probably due to Measles and following infections after many years child presenting with myoclonus jerk,decreased performance in school is chronic complication of measles suggestive of SSPE(Subacute Sclerosis Panencephalitis)

SUBACUTE SCLEROSING PANENCEPHALITIS

- Chronic complication of measles with a delayed onset and an outcome that is nearly always fatal.
- It may be result of persistent infection with an altered measles virus that is harbored intracellularly in the central nervous system for several years.

PATHOGENESIS

- Include defective measles virus and interaction with a defective or immature immune system.

CLINICAL MANIFESTATIONS

- Begin insidiously 7-13 yr after primary measles infection.
- Subtle changes in behavior or school performance appear, including irritability, reduced attention span, and temper outbursts.

i. Stage I may at times be missed due to mildness of the symptoms.

ii.Stage II :

- Is massive myoclonus, which coincides with extension of the inflammatory process site to deeper structures in the brain, including the basal ganglia.

iii.Stage III :

- Involuntary movements disappear and are replaced by choreoathetosis, immobility, dystonia, and lead pipe rigidity that result from destruction of deeper centers in the basal ganglia.
- The sensorium deteriorates into dementia, stupor, and then coma.

iv. Stage IV :

- Is characterized by loss of critical centers that support breathing, heart rate, and blood pressure.

DIAGNOSIS

- Documentation of a compatible clinical course and at least 1 of the following supporting findings :

(1) Measles antibody detected in cerebrospinal fluid (Elevated IgG and IgM antibody titers in dilutions >1 : 8).

(2) Characteristic electroencephalographic findings, and

(3) Typical histologic findings in and/or isolation of virus or viral antigen from brain tissue obtained by biopsy or postmortem examination.

- Brain biopsy is no longer routinely indicated for diagnosis of SSPE.

MANAGEMENT

- Primarily supportive
- Carbamazepine : Control of myoclonic jerks in the early stages of the illness.
- Virtually all patients eventually succumb to SSPE.
- Most die within 1-3 yr of onset from infection or loss of autonomic control mechanisms.

Reference :
Nelson TEXTBOOK of PEDIATRICS 20th Ed
Notespaedia Pediatrics High Yield Pg : 66

ABSENCE SEIZURES

- Typical absence seizures usually start at 5-8 yr of age and are often, owing to their brevity, overlooked by parents for many months even though they can occur up to hundreds of times per day.
- Precipitated by Hyperventilation for 3-5 min.
- Early onset absence seizures (before the age of 4 yr) should trigger evaluation for glucose transporter defect that is often associated with low CSF glucose levels and an abnormal sequencing test of the transporter gene.

CLINICAL FEATURES

- Do not have an aura, usually last for only a few seconds, and are accompanied by eye lid flutter or upward rolling of the eyes.
- Can have simple automatisms like lip-smacking or picking at clothing and the head can minimally fall forward.
- Do not have a postictal period and are characterized by immediate resumption of what the patient was doing before the seizure.

TREATMENT

- Most often initially treated with Ethosuximide.
- Alternative drugs of first choice are lamotrigine and valproate, especially if generalized tonic-clonic seizures coexist with absence seizures, as these 2 medications are effective against the latter seizures whereas ethosuximide is not.
- Patients resistant to ethosuximide : Respond to Valproate or to Lamotrigine.
- Other medications that could be used for absence seizures include Acetazolamide, Zonisamide, or Clonazepam.

JUVENILE MYOCLONIC EPILEPSY

- a.k.a Janz syndrome.
- Most common generalized epilepsy in young adults.
- Linked to mutations in many genes including CACNB4; CLNC2; EJM2, 3, 4, 5, 6, 7, 9; GABRA1; GABRD ; and Myoclonin1/ EFHC1

MANIFESTATIONS

- Myoclonic jerks in the morning, often causing the patient to drop things;
- Generalized tonic- clonic or clonic-tonic-clonic seizures upon awakening; and juvenile absences.
- Sleep deprivation, alcohol (in older patients), and photic stimulation

INVESTIGATIONS

- EEG :
  - Generalized 4-5 Hz polyspike-and-slow-wave discharges.
  - There are other forms of generalized epilepsies such as photoparoxysmal epilepsy, in which generalized tonic-clonic, absence or myoclonic generalized seizures are precipitated by photic stimuli such as strobe lights, flipping through TV channels and viewing video games.

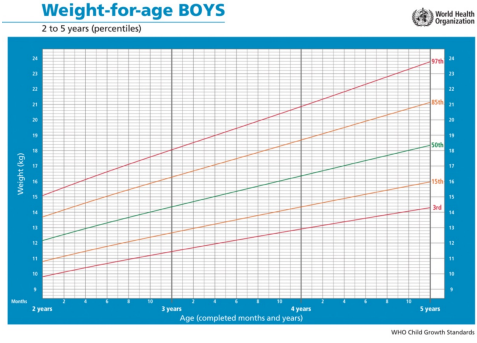
- Other forms of reflex (i.e., stimulus-provoked) epilepsy can occur; associated seizures are usually generalized, although some may be focal

MEDIAL TEMPORAL SCLEROSIS

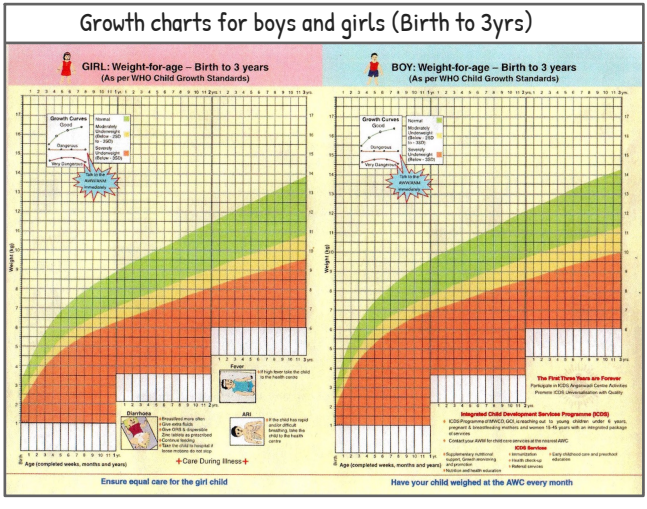
- a.k.a Temporal lobe epilepsy.
- Often preceded by febrile seizures
- It is the most common cause of surgically remediable partial epilepsy in adolescents and adults.
- Activation of frontal and secondary generalized discharges in sleep leads to more global delay secondary to the syndrome of continuous spike waves in slow-wave sleep
- Syndrome of Rasmussen encephalitis is a form of chronic encephalitis that manifests with unilateral intractable partial seizures, epilepsia partialis continua, and progressive hemiparesis of the affected side, with progressive atrophy of the contralateral hemisphere.

5. 16 month old boy is weighing 8kg.Assess his growth with the growth chart given.What needs to be done next ?

A. Assure mother that no malnutrition
B. Mild malnutrition - Home treatment
C. Moderate malnutrition - Teach mother on how to feed
D. Severe malnutrition - refer to nutrition rehabilitation center

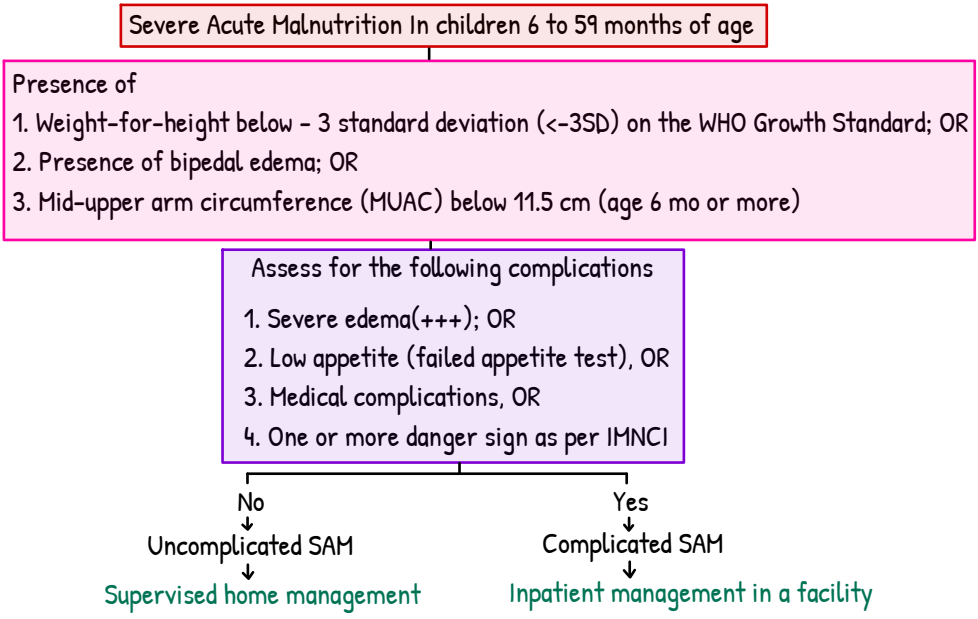


- By assessing growth chart for 16 month old boy weighing 8kg : he is below 3rd percentile which is suggestive of Severe Malnutrition.



MANAGEMENT OF MALNUTRITION

- **Mild and Moderate Malnutrition :**
  - The mainstay of treatment is provision of adequate amounts of :
    i. Energy – at least 150 kcal/kg/day should be given.
    ii. Protein intake of 3g/kg/day is sufficient.
  - Nutritious home food is recommended.
  - Milk is the most frequent source of the protein used in therapeutic diets, though other sources, including vegetable protein mixtures, have been used successfully.
  - The best measure ofthe efficacy oftreatment ofmild and moderate malnutrition is weight gain.
- **Severe Acute Malnutrition (SAM) :**
  - Complicated SAM, and the child is referred for inpatient management.
  - Nutritional rehabilitation of a child with SAM would require 3-5 months.



6. 13 yr old child with recurrent respiratory tract infections and passage of bulky greasy stools.Fat percentage in stools was high.Which of the following is true about this condition ?

A. Hypernatremia
B. Protein losing enteropathy
C. Small intestine obstruction
D. Decreased sweat chloride

- Based on history and clinical manifestation most probable diagnosis is Cystic fibrosis where Sweat testing for chloride is decreased.

CYSTIC FIBROSIS

- Is an inherited multisystem disorder of children and adults;
- It is the most common life-limiting recessive genetic trait among whites.
- Dysfunction of the cystic fibrosis transmembrane con- ductance regulator protein (CFTR), the primary defect.
- CF is inherited as an Autosomal Recessive trait.
- The CF gene codes for the CFTR protein
- CFTR is expressed largely in epithelial cells of airways, the gastrointestinal tract (including the pancreas and biliary system), the sweat glands, and the genitourinary system.

CLINICAL MANIFESTATIONS

- Respiratory Tract :
  - Cough is the most constant symptom of pulmonary involvement.
  - Expectorated mucus is usually purulent.
  - Early physical findings include increased AP diameter of the chest, generalized hyperresonance, scattered or localized coarse crackles, and digital clubbing.
  - Cyanosis is a late sign.
  - Nasal obstruction and rhinorrhea are common, caused by inflamed, swollen mucous membranes or, in some cases, nasal polyposis.
- Intestinal Tract :
  - Ileum is completely obstructed by meconium (meconium ileus).
  - Abdominal distention, emesis, and failure to pass meconium appear in the 1st 24-48 hr of life.
  - Protein and fat malabsorption from exocrine pancreatic insufficiency.
  - Symptoms include frequent, bulky, greasy stools and failure to gain weight even when food intake appears to be large.
  - Less-common gastrointestinal manifestations include intussusception, fecal impaction of the cecum with an asymptomatic right lower quadrant mass, and epigastric pain owing to duodenal inflammation.
  - Due to Vit. E deficiency : Neurologic dysfunction (dementia, peripheral neuropathy) and hemolytic anemia may occur.
  - Due to Vit.K deficiency : Hypoprothrombinemia result in bleeding diathesis.
- Biliary Tract :
  - Manifestations can include icterus, ascites, hematemesis from esophageal varices, and evidence of hypersplenism.
- Pancreas :
  - Hyperglycemia and glucosuria, including polyuria and weight loss, may appear, especially in the 2nd decade of life.
- Genitourinary Tract :
  - Sexual development is often delayed but only by an average of 2 yr.
  - The female fertility rate is diminished.

Reference :
Notespaedia Pediatrics High Yield Pg : 99
Nelson TEXTBOOK of PEDIATRICS 20th Ed

- Sweat Glands :
  - Excessive loss of salt in the sweat predisposes young children to salt depletion episodes.
  - These children present with hypochloremic alkalosis.

DIAGNOSIS

- Sweat Testing :
  - More than 60 mEq/L of chloride in sweat is diagnostic of CF when 1 or more other criteria are present.
  - Threshold levels of 30-40 mEq/L for infants have been suggested.
  - Borderline (or intermediate) values of 40-60 mEq/L.
- DNA Testing :
  - Most common CFTR mutations.
  - This testing identifies ≥90% of individuals who carry 2 CF mutations.
- Other Diagnostic Tests :
  - i. Finding of increased potential differences across nasal epithelium :
    - Increased voltage response to topical amiloride application, followed by the absence of a voltage response to a β-adrenergic agonist.
    - Used to confirm the diagnosis of CF in patients with equivocal or frankly normal sweat chloride values.

DIAGNOSTIC CRITERIA FOR CYSTIC FIBROSIS (CF)

- Presence of typical clinical features (respiratory, gastrointestinal, or genitourinary) or
- A history of CF in a sibling or
- A positive newborn screening test

  plus
- Laboratory evidence for CFTR (CF transmembrane regulator) dysfunction :
  - Two elevated sweat chloride concentrations obtained on separate days or
  - Identification of two CF mutations or
  - An abnormal nasal potential difference measurement

TREATMENT

- Pulmonary Therapy :
  - i. Inhalation Therapy :
    - Aerosol therapy is used to deliver medications and hydrate the lower respiratory tract.
    - Human recombinant DNase (2.5 mg), given as a single daily aerosol dose.
    - Nebulized hypertonic saline
  - ii. Airway Clearance Therapy :
    - Chest physical therapy (PT)
  - iii. Antibiotic Therapy
  - iv. Bronchodilator Therapy

v. Anti inflammatory Agents :
- Corticosteroids

vi. Emerging Therapies :
- Ivacaftor activates the CFTR-G551D mutant protein, a class III CFTR mutation that results in protein localized to the plasma membrane but loss of chloride channel function.
- CFTR mutation F508del

7. A 4yr old boy presented with developmental delay;recurrent chest infections and worsening bone pain.His serum PTH was High,Calcium was normal.Phosphate was low.The probable diagnosis is ?

A. Vitamin D dependent rickets Type 1
B. Vitamin D dependent rickets type 2
C. X linked hypophosphatemic rickets
D. Nutritional rickets

- Based on given history and laboratory findings most probable diagnosis is Nutritional rickets.

NUTRITIONAL VITAMIN D DEFICIENCY RICKETS

- Most common cause of rickets globally

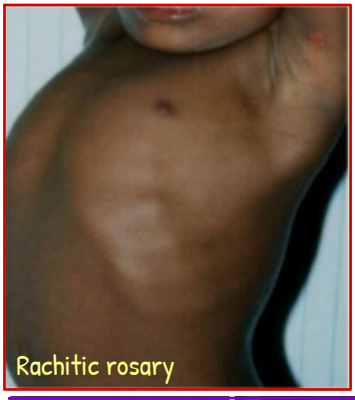
ETIOLOGY

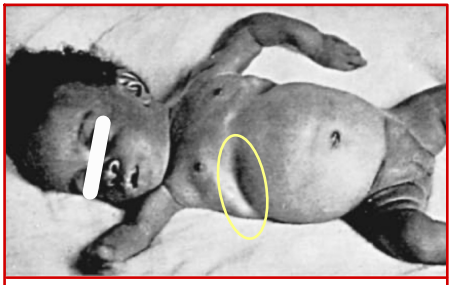
- Most commonly occurs in infancy because of a combination of poor intake and inadequate cutaneous synthesis.

CLINICAL MANIFESTATIONS

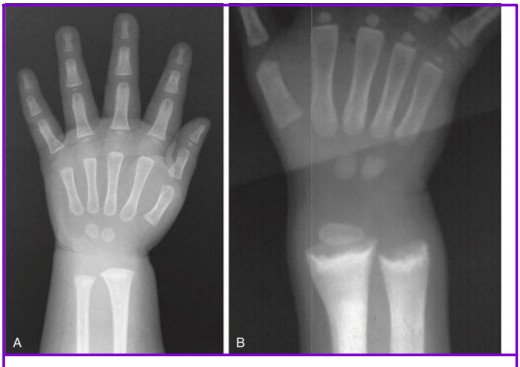
- GENERAL :
  - Failure to thrive
  - Listlessness
  - Protruding abdomen
- HEAD :
  - Craniotabes
  - Frontal bossing
- CHEST
  - Rachitic rosary
  - Harrison groove
- BACK
  - Scoliosis
  - Kyphosis
  - Lordosis
- HYPOCALCEMIC SYMPTOMS
  - Tetany
  - Seizures
  - Stridor due to laryngeal spasm
- Muscle weakness (especially proximal)
- Fractures
- Delayed fontanel closure
- Delayed dentition; caries Craniosynostosis
- Respiratory infections and atelectasis*
- EXTREMITIES
  - Enlargement of wrists and ankles
  - Valgus or varus deformities
  - Windswept deformity (combination of valgus deformity of 1 leg with varus deformity of the other leg)
  - Anterior bowing of the tibia and femur Coxa vara
  - Leg pain

Reference :
Notespaedia Pediatrics High Yield Pg : 99 , Notespaedia Pediatrics IB Pg : 68
Nelson TEXTBOOK of PEDIATRICS 20th Ed

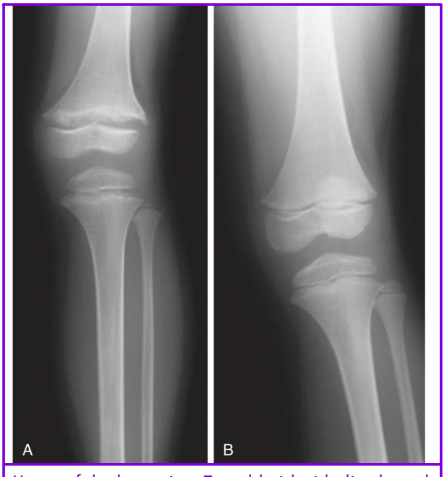




Deformities in rickets showing curvature of the limbs, potbelly, and Harrison groove.



Wrist x-rays in a normal child (A) and in a child with rickets (B). The child with rickets has metaphyseal fraying and cupping of the distal radius and ulna.



X-rays of the knees in a 7 yr old girl with distal renal tubular acidosis and rickets. A, At initial presentation, there is widening of the growth plate and metaphysical fraying. B, Dramatic improvement after 4 mo of therapy with alkali.

INVESTIGATIONS

Laboratory Findings in Various Disorders Causing Rickets

DISORDER	Ca	Pi	PTH	25-(OH)D	1,25-$(OH)_2$D	Alk Phos	URINE Ca	URINE Pi
Vitamin D deficiency	N, ↓	↓	↑	↓	↓, N, ↑	↑	↓	↑
Chronic kidney disease	N, ↓	↑	↑	N	↓	↑	N, ↓	↓
Dietary Pi deficiency	N	↓	N, ↓	N	↑	↑	↑	↓
Tumor-induced rickets	N	↓	N	N	RD	↑	↓	↑
Fanconi syndrome	N	↓	N	N	RD or ↑	↑	↓ or ↑	↑
Dietary Ca deficiency	N, ↓	↓	↑	N	↑	↑	↓	↑

↓, decreased; ↑, increased; ↑ ↑, extremely increased; 1,25-(OH)2D, 1,25-dihydroxyvitamin D; 25-OHD, 25-hydroxyvitamin D; ADHR, autosomal dominant hypophosphatemic rickets; Alk Phos, alkaline phosphatase; ARHR, autosomal recessive hypophosphatemic rickets; Ca, calcium; HHRH, hereditary hypophosphatemic rickets with hypercalciuria; N, normal; Pi, inorganic phosphorus; PTH, parathyroid hormone; RD, relatively decreased (because it should be increased given the concurrent hypophosphatemia); VDDR, vitamin D–dependent rickets; XLH, X-linked hypophosphatemic rickets.

Biochemical Changes in Genetic causes of Rickets

	SERUM BIOCHEMISTRY							URINE BIOCHEMISTRY		
	Phosphate	Calcium	PTH	25OHD	1,25H_2D	FGF23	Alk Phos	Phosphate	Calcium	OTHER FEATURES
HYPOCALCEMIC VITAMIN D PATHWAY DEFECTS										
Vitamin D deficiency	Low	Variable	High	Low	Might be increased	NA	Increased	Increased	Low	Variable aminoaciduria
VDDR1B	Low	Low	High	Very low	Variable	NA	Increased	Increased	Low	25OHD does not increase after vitamin D dosing
VDDR1A	Low	Low	High	Normal or high	Very low or ND	NA	Increased	Increased	Low	25OHD does increase after vitamin D dosing
VDDR2A	Low	Low	High	Normal or high	High	NA	Increased	Increased	Low	—
VDDR2B	Low	Low	High	Normal or high	High	NA	Increased	Increased	Low	—
HYPOPHOSPHATEMIC RICKETS WITH RAISED FGF23										
XLH	Low	Normal	Normal or slightly high	Normal	Low	High	Increased	Increased	Variable	Urine calcium:creatinine used in monitoring therapy
ADHR	Low	Normal	Normal	Normal	Low	High	Increased	Increased	Variable	—
ARHR1	Low	Normal	Normal	Normal	Low	High	Increased	Increased	Variable	—
ARHR2	Low	Normal	Normal	Normal	Low	High	Increased	Increased	Variable	—
HYPOPHOSPHATEMIC RICKETS WITHOUT RAISED FGF23										
Dent's disease*	Low	Normal	Normal	Normal	Normal	Normal	Increased	Increased	High	Low molecular weight proteinuria
HHRH	Low	Normal	Normal	Normal	Normal	Normal	Increased	Increased	High	No loss of low molecular weight protein
αKlotho mutation	Low	Normal	Normal	Normal	Normal	Normal	Increased	Increased	Variable	—
OTHER INHERITED RACHITIC DISORDERS										
HPP (severe)	High	High	Low	Normal	Normal	Normal	Very low	Normal or high	High	Raised concentrations of mineralization inhibitors
HPP (mild)	Normal or high	Normal or high	Low or normal	Normal	Normal	Normal	Low	Normal	Variable	Raised concentrations of mineralization inhibitors

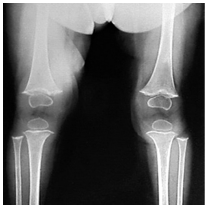
TREATMENT

- For nutritional vitamin D deficiency :
  - Should receive vitamin D and adequate nutritional intake of calcium and phosphorus.
  - There are 2 strategies for administration of vitamin D.
    - i. With stoss therapy, 300,000-600,000 IU of vitamin D are administered orally or i.m as 2-4 doses over 1 day.
    - ii. The alternative is daily, high-dose vitamin D, with doses ranging from 2,000-5,000 IU/day over 4-6 wk.
  - Either strategy should be followed by daily vitamin D intake of 400 IU/day if <1 yr old or 600 IU/ day if >1 yr old.
- For congenital rickets includes :
  - Vitamin D supplementation and adequate intake of calcium and phosphorus.
  - Use of prenatal vitamins containing vitamin D.
- For Vitamin D-Dependent Rickets, Type 1 : long-term treatment with 1,25-D (calcitriol).
- For Vitamin D-Dependent Rickets, Type 2 :
  - respond to extremely high doses of vitamin D2, 25-D or 1,25-D
  - Calcium doses are 1,000-3,000 mg/day.
- For Chronic Kidney Disease :
  - use of a form of vitamin D that can act without 1-hydroxylation by the kidney (calcitriol), which both permits adequate absorption of calcium and directly suppresses the parathyroid gland.

- For Calcium deficiency :
  - providing adequate calcium, typically as a dietary supplement (doses of 700 [1-3 yr age], 1,000 [4-8 yr age], 1,300 [9-18 yr age] mg/day of elemental calcium are effective).
- For X-Linked Hypophosphatemic Rickets :
  - combination of oral phosphorus and 1,25-D (calcitriol).
  - The daily need for phosphorus supplementation is 1-3 g of elemental phosphorus divided into 4-5 doses.
  - Calcitrol is administered 30-70 ng/kg/day divided into 2 doses.
- For Hereditary Hypophosphatemic Rickets with Hypercalciuria :
  - oral phosphorus replacement (1-2.5 g/day of elemental phosphorus in 5 divided oral doses).

8. X Ray of a 5yr old child was given for knee joint.There were dense white lines on X Rays :

A. Rickets
B. Scurvy
C. Osteomalacia
D. Osteoporosis



SCURVY

- Due to deficiency of Vit.C

CLINICAL FEATURES

- Early features includes :
  - Irritability, anorexia, anemia and appearance of petechiae due to increased capillary fragility.
  - Gingival swelling, bleeding gums,generalized tenderness of the limbs,painful joint swellings and peripheral edema are seen.
  - The child may present with inability to move limbs (pseudoparalysis) due to pain and assume a frog-like posture, with semiflexion at hips and knees.
  - Characteristic angular swellings at the costochondral junction, known as Scorbutic rosary
  - Scurvy can result in cerebral hemorrhage or hemopericardium and is potentially fatal, if left untreated.

DIAGNOSIS

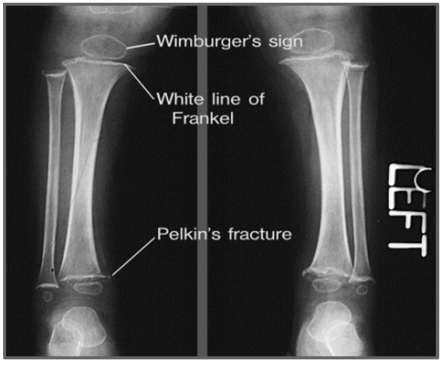
- Characteristic physical findings and history of inadequate dietary intake of vitamin C.
- X-rays of long bones : A ground glass appearance with thinning of cortex (pencil thin cortex).
- An irregular thickened white line appears at the metaphysis (White line of Frankel), representing the zone of well-calcified cartilage.

Reference :
Ghai Essential Pediatrics 9th Ed.

- There is a zone of rarefaction proximal to this line, which represents poorly formed trabeculae (Triimmerfeld zone).
- The lateral part of the rarefaction appears as a triangular defect : Pelken spurs.
- The epiphyses are surrounded by a thin white line (Wimberger ring sign)

TREATMENT

- 100 - 200 mg of vitamin C orally or parenterally prompts rapid improvement m symptoms and resolution of the radiological signs.
- Daily intake of 100 mL of orange juice or tomato pulp has the same effect.



9. A 4-year-old child who has ventricular shunt for hydrocephalus got it replaced because of shunt malfunction. The child now presents with irritability and loss of appetite for 3 days and no other symptoms rectal temperature was 38.7 C.What will be the next step in management :

A. Blood culture to be taken
B. Blood culture and CSF from shunt tap
C. Start IV antibiotics
D. Shunt patency study

HYDROCEPHALUS

- Is not a specific disease; it represents a diverse group of conditions that result from impaired circulation and/or absorption of CSF or, in rare circumstances, from increased production of CSF by a choroid plexus papilloma

CAUSES OF HYDROCEPHALUS

i. COMMUNICATING

- Achondroplasia
- Basilar impression
- Benign enlargement of subarachnoid space
- Choroid plexus papilloma
- Meningeal malignancy
- Meningitis
- Posthemorrhagic

ii.HYDRANENCEPHALY

- Holoprosencephaly
- Massive hydrocephalus
- Porencephaly

ii. NONCOMMUNICATING

- Aqueductal stenosis Infectious*
- X-linked
- Mitochondrial
- Autosomal recessive
- Autosomal dominant
- L1CAM mutations
- Chiari malformation
- Dandy-Walker malformation
- Klippel-Feil syndrome
- Mass lesions
- Abscess
- Hematoma
- Tumors and neurocutaneous disorders
- Vein of Galen malformation
- Walker-Warburg syndrome

CLINICAL MANIFESTATIONS

- In an infant, an accelerated rate of enlargement of the head is the most prominent sign.
- The anterior fontanel is wide open and bulging, and the scalp veins are dilated.
- The forehead is broad, and the eyes might deviate downward because of impingement of the dilated suprapineal recess on the brainstem tectum : Setting-Sun Eye Sign.
- Long-tract signs, including :
  - Brisk tendon reflexes,
  - Spasticity,
  - Clonus (particularly in the lower extremities), and
  - Babinski sign, are common.
- Irritability, lethargy, poor appetite, and vomiting are common to both age groups.
- Headache is a prominent in older patients.
- A gradual change in personality and deterioration in academic productivity.
- Percussion of the skull might produce a cracked pot sound or Macewen sign

DIAGNOSIS

- A cranial bruit is audible in association with many cases of vein of Galen arteriovenous malformation.
- Transillumination of the skull is positive with massive dilation of the ventricular system.
- Plain skull films :
  - Shows separation of the sutures, erosion of the posterior clinoids in an older child, and an increase in convolutional markings (beaten-silver appearance) on the inside of the skull
- CT scan and/or MRI Scan along with ultrasonography in an infant are the most important.

TREATMENT

- Medical management, including the use of Acetazolamide and Furosemide.
- Extracranial shunts : Ventriculoperitoneal shunt.
- Endoscopic third ventriculostomy
- Major complications of shunting :
  - Occlusion (characterized by : headache, papilledema, emesis, mental status changes) and
  - Bacterial infection usually caused by Staphylococcus epidermidis.

SHUNT MALFUNCTION OR SHUNT FAILURE

- Referred to as shunt failure, is a partial or complete blockage (obstruction) of the shunt that causes it to function intermittently or not at all.
- A shunt blockage from blood cells, tissue, or bacteria can occur in any part of the shunt. Both the ventricular catheter (the portion of the tubing placed in the brain) and the distal part of the catheter (the tubing that drains fluid to another part of the body) can become blocked by tissue.
- Blood culture and CSF from shunt tap should be taken if patient is irritable,c/o fever,loss of appetite.
- When a shunt infection occurs, the standard treatment is the surgical removal of all of the shunt hardware.

OTHER SHUNT COMPLICATIONS

- Over drainage causes the ventricles to decrease in size and may create slit-like ventricles
- Under drainage causes the ventricles to increase in size and can fail to relieve the symptoms of hydrocephalus.
- Adjustment to accommodate patient growth.
- Subdural hematoma occurs if blood from broken vessels becomes trapped between the brain and skull.
- Multiloculated hydrocephalus is a loculated (isolated) CSF compartment in the brain that is enlarged but not connected to the ventricular system.
- Material degradation
- Seizures sometimes occur in people with hydrocephalus.
- Abdominal complications in the abdomen can occur in people with hydrocephalus treated with a shunt.

MOST COMMON TESTS TO ASSESS POSSIBLE SHUNT MALFUNCTION OR SHUNT FAILURE

- Ultrasound (US) is used in infants with open soft spots or fontanels.
- Computed tomography (CT Scan) is a reliable procedure for diagnosing and assisting in the management of hydrocephalus.
- Magnetic Resonance Imaging (MRI), like the CT scan, is a diagnostic technique that produces images of the brain.
- X-ray of the shunt system often called a shunt series.
- Shunt flow studies, which also may be referred to as a shunt patency study or shuntogram.
- Shunt Tap is a diagnostic test to screen for infection and confirm that the shunt is still functioning.
- External Ventricular Drain (EVD) is a treatment that allows the temporary drainage of CSF from the lateral ventricles of the brain, or lumbar space of the spine, into an external collection bag. Intracranial Pressure Monitoring (ICP) is a diagnostic test that helps your doctors determine if high or low CSF pressure is causing your symptoms.

Reference :
https://www.hydroassoc.org/complications-of-shunt-systems/

10. A 4-year female with Presented with arthritis, palpable purpura abdominal pain and hematuria. Likely diagnosis is

A. Henoch Schonlein Purpura

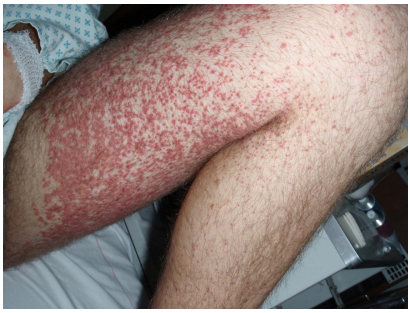
B. SLE

C. Takayasu arteritis

D. Rheumatoid arthritis

HENOCH-SCHONLELN PURPURA

- IgA vasculitis is a common vasculitic disorder of childhood and is characterized by presence of a non- thrombocytopenic and usually palpable purpura, transient arthralgia (occasionally, arthritis) and abdominal symptoms.



Classification criteria for childhood HENOCH-SCHONLELN PURPURA
• Palpable purpura with at least one of the following: - Diffuse abdominal pain - Any biopsy showing predominant lgA deposition Arthritis or arthralgia - Renal involvement (any hematuria and/or proteinuria)

LABORATORY INVESTIGATIONS

- Is a clinical diagnosis
- There may be nonspecific increase in total sErum IgA levels.
- Microscopic hematuria and proteinuria may be present.
- Skin biopsy shows leukocytoclastic vasculitis.
- On indirect immunofluorescence :
  - There are deposits of IgA in skin as well as renal biopsies.
- USG examinations may be needed.

TREATMENT

- Generally supportive with maintenance of hydration and pain relief.
- Prednisolone usually continued for 2-3 weeks (in gradually tapering doses) depending on the clinical response.
- Nephritis may need immunosuppressants (Prednisolone and azathioprine)

1. A 45 yr old patient presents with complaints of chest pain.Angiogram was done and it revealed Left circumflex artery occlusion.Stenting is done and patient was started on Aspirin 75mg,Atorvastatin 20mg and Metoprolol 50mg.Which of the follwoing statement is correct regarding further management of this patient ?

A. Increased dose of Aspirin

B. Add Nifedipine

C. Add P2Y12 Inhibitor

D. Increased dose of Metoprolol to 100mg

ANTIPLATELET AND ANTICOAGULATION TREATMENT SUMMARY ACS ACCORDING TO THE AMERICAN HEART ASSOCIATION/AMERICAN COLLEGE OF CARDIOLOGY PRACTICE GUIDELINE.

- Initial Treatment :
  - i. DAPT and Anticoagulant therapy :
    1. Aspirin
    2. P2Y 12 inhibitor : Clopidogrel or Ticagrelor
    3. Anticoagulant : Enoxaparin or UFH or Fondaparinux or Bivalirudin
    4. Can consider GP IIb/IIIa receptor inhibitors in high-risk patients stratified to early invasive strategy(Eptifibatide or Tirofiban.)
- During Hospitalization :
  - i. Medically treated patients :
    1. Aspirin.
    2. P2Y 12 inhibitor : either Ticagrelor or Clopidogrel.
    3. Anticoagulant : Enoxaparin or UFH or Fondaparinux
  - ii. PCI treated patients :
    1. Aspirin.
    2. P2Y 12 inhibitor : Clopidogrel or Ticagrelor or Prasugrel.
    3. Anticoagulant : Enoxaparin or UFH or Fondaparinux* or Bivalirudin.
    4. Can consider GP IIb/IIIa receptor inhibitors in high- risk patients not adequately per-treated with clopidogrel or in high-risk patients adequately pre-treated with clopidogrel.
- Long-term :
  - i. Medically treated patients :
    1. Aspirin indefinitely.
    2. P2Y 12 inhibitor : Clopidogrel or Ticagrelor for up to 12 months.
  - ii. PCI treated patients :
    1. Aspirin indefinitely.
    2. P2Y 12 inhibitor : Clopidogrel or Ticagrelor or Prasugrel for at least 12 months.

Reference :
Notespaedia Medicine High Yield Pg : 20
Harrison's Principles of Internal Medicine 20th Ed Pg :1870

2. A patient present with fever,nocturnal cough,breathlessness and wheezing for 4 wks.Absolute eosinophil count > 5000.Chest X ray shows Miliary Pattern.What is the most probable diagnosis ?

A. Bronchial asthma
B. Miliary TB
C. Tropical Pulmonary Eosinophilia
D. Hypersensitivity Pneumonitis

TROPICAL PULMONARY EOSINOPHILIA

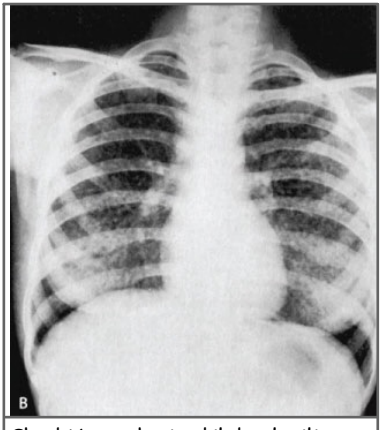
- Develops in some individuals infected with the lymphatic- dwelling filarial species.

CLINICAL FEATURES

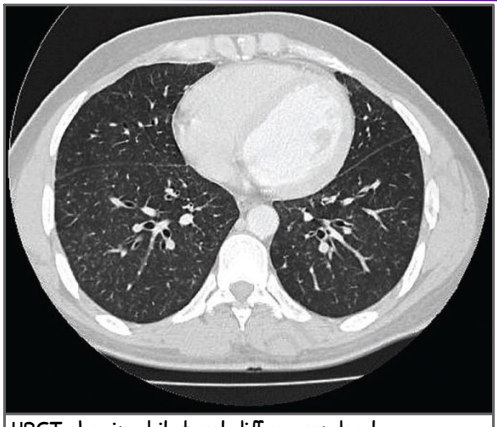
- Paroxysmal cough and wheezing (usually nocturnal and probably related to the nocturnal periodicity of microfilariae)
- Weight loss,
- Low-grade fever
- Lymphadenopathy

INVESTIGATIONS

- Blood eosinophilia (>3000 eosinophils/μL).
- Chest x-rays or CT scans :
  - May be normal, but generally show increased bronchovascular markings.
  - Diffuse miliary lesions or mottled opacities may be present in the middle and lower lung fields.
- Tests of pulmonary function show restrictive abnormalities in most cases and obstructive defects in half.
- Total serum IgE levels (4-40 KIU/mL)
- Antifilarial antibody levels are markedly elevated.



Chest X-ray showing bilateral miliary nodules



HRCT showing bilateral diffuse randomly distributed micronodules shadows

Reference :
Harrison's Principles of Internal Medicine 20th Ed Pg :1632

TREATMENT

- DEC is used at a daily rate for 14 days.
- Symptoms usually resolve within 3–7 days after the initiation of therapy.
- Relapse, which occurs in 12–25% of cases (sometimes after an interval of several years),requires re-treatment.

MILIARY OR DISSEMINATED TB

- Clinical manifestations are nonspecific.
- Fever, night sweats, anorexia, weakness, and weight loss are presenting symptoms.
- Cough and other respiratory symptoms due to pulmonary involvement as well as abdominal symptoms.
- Physical findings include hepatomegaly, splenomegaly, and lymphadenopathy.
- Chest X Ray :
  - Reveals a miliary reticulonodular pattern (more easily seen on underpenetrated film),although no radiographic abnormality may be evident early in the course and among HIV- infected patients.

3. A 35yr old female presented with claudication in forearm,transient loss of vision and abdominal pain. She also had weak femoral pulses.Fundus also showed retinal hemorrhages.What is the most probable diagnosis ?

A. Takayasu arteritis
B. PAN
C. Microscopic polyangitis
D. Thromboangitis obliterans

TAKAYASU ARTERITIS

- Is an inflammatory and stenotic disease of medium- and large-sized arteries characterized by a strong predilection for the aortic arch and its branches.
- Most commonly affected arteries : Subclavian artery > Common Carotid > Abdominal aorta > Renal

CLINICAL MANIFESTATIONS

- Generalized symptoms include malaise, fever, night sweats, arthralgias, anorexia, and weight loss.
- Pulses are commonly absent in the involved vessels, particularly the subclavian artery.
- Subclavian if affected : Arm claudication, Raynaud's phenomenon
- Common carotid : Visual changes, syncope, transient ischemic attacks, stroke
- Abdominal aorta : Abdominal pain, nausea, vomiting
- Renal : Hypertension, renal failure

LABORATORY MANIFESTATIONS

- Elevated ESR
- Mild anemia, and
- Elevated immunoglobulin levels.

Reference :
Notespaedia Medicine High Yield Pg : 166
Harrison's Principles of Internal Medicine 20th Ed Pg : 2585

- Diagnosis is confirmed by the characteristic pattern on arteriography :
  - Irregular vessel walls, stenosis, poststenotic dilation, aneurysm formation, occlusion, and evidence of increased collateral circulation.
  - Complete aortic arteriography by catheter-directed dye arteriography or magnetic resonance arteriography should be obtained
  - Histopathologic demonstration of vessel wall inflammation that is predominantly lymphocytic with granuloma formation and giant cells involving the media and adventitia

TREATMENT

- Takayasu arteritis is most often chronic and relapsing.
- Glucocorticoid therapy : Prednisone per day alleviates symptoms
- In individuals who are refractory to or unable to taper glucocorticoids : Methotrexate is given

MICROSCOPIC POLYANGITIS

- Necrotizing vasculitis with few or no immune complexes affecting small vessels (capillaries, venules, or arterioles).
- Absence of granulomatous inflammation.
- Mean age of onset is 57 years.
- Affected : Males more affected than females.

CLINICAL MANIFESTATIONS

- Disease onset may be gradual.
- Initial symptoms of fever, weight loss, and musculoskeletal pain.
- Glomerulonephritis occurs in at least 79% of patients
  - Rapidly progressive, leading to renal failure.
- Hemoptysis (First symptom of alveolar hemorrhage)
- Other manifestations include mononeuritis multiplex and gastrointestinal tract and cutaneous vasculitis.

TREATMENT

- A steroid (usually prednisone) in combination with a cyclophosphamide (CYC) or rituximab is typically the first combination of medications to be prescribed.
- After control of the disease – usually around 4 – 6 months of treatment maintenance therapy will be used to keep the disease in remission.

THROMBOANGIITIS OBLITERANS

- a.k.a Buerger's disease
- Is an inflammatory occlusive vascular disorder involving small and medium-size arteries and veins in the distal upper and lower extremities.

CLINICAL FEATURES

- Triad of :
  - i. Claudication of the affected extremity
  - ii. Raynaud's phenomenon, and
  - iii. Migratory superficial vein thrombophlebitis.

- Claudication usually is confined to the calves and feet or the forearms and hands.
- The physical examination : normal brachial and popliteal pulses but reduced or absent radial, ulnar, and/or tibial pulses.

TREATMENT

- No specific treatment except abstention from tobacco.
- Arterial bypass of the larger vessels may be used in selected instances, as well as local debridement, depending on the symptoms and severity of ischemia.
- Antibiotics may be useful

4. A Labourer came to emergency department, with complain of dizziness and excessive fatigue while taking history by doctor he told that he was working in hot climate, temperature noted that day was 42° C, his skin showed poor turgor all of the following symptoms can be present except

A. Sweating
B. Hypotension
C. Tachypnea
D. High volume pulse

- Based on the given history it is suggestive of Heat Stroke.

HEATSTROKE

- There is a a total loss of thermoregulatory function.

CLINICAL FEATURES

- Typical vital-sign abnormalities include tachypnea, various tachycardias, hypotension, and a widened pulse pressure.
- Weakness, dizziness, disorientation, ataxia, and gastrointestinal or psychiatric symptoms.
- Two forms of heatstroke :
  - i. Classic (epidemic) heatstroke (CHS)
  - ii. Exertional heatstroke (EHS)

Typical Manifestations of Heatstroke	
CLASSIC	EXERTIONAL
Older patient	Younger patient
Predisposing health factors/medications	Healthy condition
Sedentary	Sporadic cases
Heat waves	Exercising
Anhidrosis (if possible)	Diaphoresis (common)
Central nervous system dysfunction	Myocardial/hepatic injury
Oliguria	Acute renal failure
Coagulopathy (mild)	Disseminated intravascular coagulation
Mild lactic acidosis	Marked lactic acidosis

CLASSIC	EXERTIONAL
Mild creatine kinase elevation	Rhabdomyolysis
Normoglycemia/calcemia	Hypoglycemia/calcemia
Normokalemia/Normonatremia	Hyperkalemia/Hyponatremia

- Historical and physical triad of exposure to a heat stress, CNS dysfunction, and a core temperature >40.5°C helps establish the preliminary diagnosis.

COOLING STRATEGIES

- Before cooling is initiated, endotracheal intubation and continuous core-temperature monitoring should be considered.
- Hypoglycemia : glucose infusion.
- Evaporative cooling is frequently the most practical and effective technique.
- Cool water (15°C [60°F]) is sprayed on the exposed skin while fans direct continuous airflow over the moistened skin.
- Immersion cooling in ice-cold water is an alternative option in EHS.
- To avoid hypothermic afterdrop ,active cooling should be terminated ~ 38°-39°C (100.4°F-102.2°F).

RESUSCITATION

- Aspiration commonly occurs in heatstroke, and endotracheal intubation is usually necessary.
- Seizures are common, and can occur during therapeutic cooling.
- Cold induced tonic-clonic muscular rigidity mimics seizure activity.
- The hypotension that is initially common among patients with heat- stroke results from both dehydration and high-output cardiac failure caused by peripheral vasodilation.
- Vasoactive catecholamines such as Dopamine or Dobutamine may be necessary (if the cardiac output remains depressed despite an elevated CVP)
- Anticholinergic medications (including atropine) inhibit sweating and should be avoided.

5. Which among the following is an indicator of poor prognosis in case of Multiple myeloma :

A. Serum β2 Microglobulin
B. Serum Creatinine kinase
C. Serum Calcium
D. Serum Glucose

MULTIPLE MYELOMA

- A malignant proliferation of plasma cells derived from a single clone.

CLINICAL MANIFESTATIONS

- Bone pain is the most common symptom in myeloma, affecting nearly 70% of patients.
- Persistent localized pain usually signifies a pathologic fracture.
- The most common infections are pneumonias and pyelonephritis (Most frequent pathogens are Streptococcus pneumoniae, Staphylococcus aureus, and Klebsiella pneumoniae in the lungs and Escherichia coli and other gram-negative organisms in the urinary tract.)

Reference :
Harrison's Principles of Internal Medicine 20th Ed.

- Renal failure occurs in nearly 25% of myeloma patients
- Normocytic and normochromic anemia occurs in ~80% of myeloma patients.
- Deep venous thrombosis is also observed with use of thalidomide, lenalidomide, or pomalidomide in combination with dexamethasone.

Diagnostic Criteria for Multiple Myeloma, Myeloma Variants

1. Smoldering Multiple Myeloma (Asymptomatic Myeloma)

- Both criteria must be met:
  - Serum monoclonal protein (IgG or IgA) ≥30 g/L or urinary monoclonal protein ≥500 mg per 24 h and/or clonal bone marrow plasma cells 10–60%
  - Absence of myeloma defining events or amyloidosis

2. Symptomatic Multiple Myeloma

- Clonal bone marrow plasma cells or biopsy-proven bony or extramedullary plasmacytomaa and any one or more of the following myeloma defining events :
  - Evidence of one or more end-organ damage that can be attributed to the underlying plasma cell proliferative disorder, specifically:
  - Hypercalcemia: serum calcium >0.25 mmol/L (>1 mg/dL) higher than the upper limit of normal or >2.75 mmol/L (>11 mg/dL)
  - Renal insufficiency : creatinine clearance <40 mL per minb or serum creatinine >177 μmol/L (>2 mg/dL)
  - Anemia : hemoglobin value of >20 g/L below the lower limit of normal, or a hemoglobin value <100 g/L
  - Bone lesions : one or more osteolytic lesions on skeletal radiography, CT, or PET-CTc
- Any one or more of the following biomarkers of malignancy:
  - i. Clonal bone marrow plasma cell percentagea ≥60%
  - ii.Involved: uninvolved serum free light chain ratiod ≥100
  - iii. >1 focal lesions on MRI studiese

3. Nonsecretory Myeloma

- No M protein in serum and/or urine with immunofixationf
- Bone marrow clonal plasmacytosis ≥10% or plasmacytomaa
- Myeloma-related organ or tissue impairment.

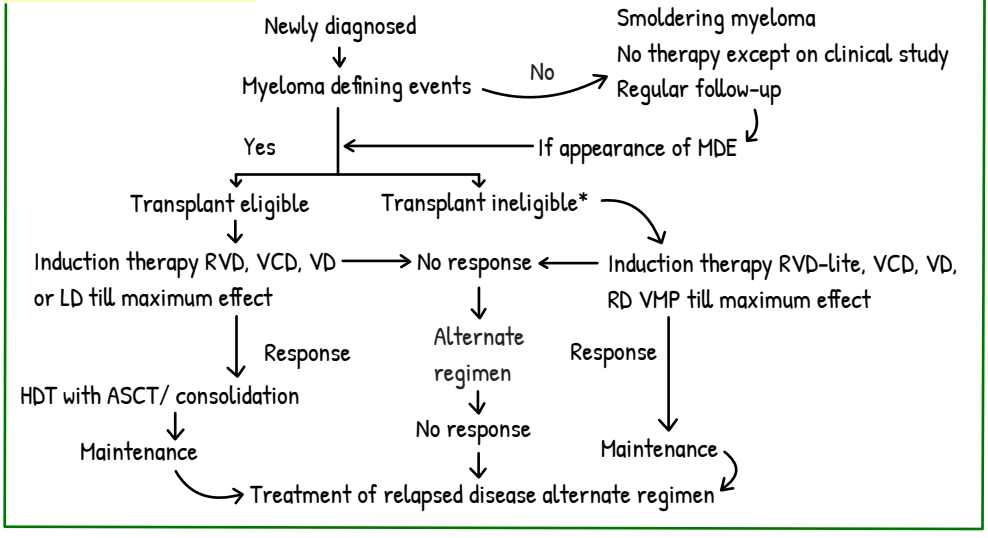
PROGNOSIS

- Serum β2-microglobulin is the single most powerful predictor of survival and can substitute for staging.
- Combination of serum β2-microglobulin and albumin levels forms the basis for a three-stage International Staging System (ISS)

STANDARD INVESTIGATIVE WORKUP

1. Investigations to Evaluate for Clonal Plasma Cells :
   - Bone marrow aspirate and biopsy (fine needle aspiration of plasmacytoma if indicated)
     - Histology
     - Clonality by kappa/lambda immunostaining by flow cytometry or immunohistochemistry
2. Investigations to Evaluate Clonal Paraprotein :
   - Serum protein electrophoresis and immunofixation
   - Quantitative serum immunoglobulin levels (IgG, IgA, and IgM)
   - 24-h Urine protein electrophoresis and immunofixation
   - Serum free light chain and ratio
   - Immunofixation for IgD or IgE in select cases
3. Investigation to Evaluate End-Organ Damage :
   - Hemogram to assess for anemia
   - Chemistry panel for renal function and calcium
   - Skeletal survey to evaluate bone lesions
     - PET/CT or MRI if SMM or solitary plasmacytoma with no other MDE or extramedullary disease
4. Investigation for Risk Stratification :
   - β-2 microglobulin and serum albumin for ISS stage
   - Fluorescent in situ hybridization for hyperdipoidy, del17p, t(4;14); t(14;16),amp1q34, and del 13 on bone marrow sample - LDH
5. Specialized Investigation in Selected Cases :
   - Abdominal fat pad for amyloid
   - Serum viscosity if IgM component or high IgA levels or serum M-component >7 g/dL

TREATMENT ALGORITHM



C, cyclophosphamide; D, dexamethasone; M, melphalan; P, prednisone; R, lenalidomide; RVD-lite, weekly regimen; V, bortezomib. Alternate regimen-combinations including daratumumab; elotuzumab; panobinostat; carfilzomib; ixazomib, pomalidomide or agents; ASCT, autologous stem cell transplantation; HDT, high-dose therapy; MDE, myeloma defining events.

6. Which among the following is false statement ?

A. Diagnosed by urine VMA and Catecholamines
B. Treatment of choice is surgical excision
C. Propranolol is given initially to treat hypertension
D. Can present as Hypertension alone,sometime with abdominal pain and vomiting.

PHEOCHROMOCYTOMA

- Catecholamine- producing tumors derived from the sympathetic or parasympathetic nervous system.
- These tumors may arise sporadically or be inherited as features of multiple endocrine neoplasia type 2 (MEN 2), von Hippel- Lindau (VHL) disease.
- The classic "rule of tens" for pheochromocytomas states that :
  i. ~ 10% are bilateral,
  ii. 10% are extra- adrenal, and
  iii. 10% are malignant.

CLINICAL FEATURES

- Headaches
- Profuse sweating
- Palpitations and tachycardia
- Hypertension, sustained or paroxysmal
- Anxiety and panic attacks
- Pallor
- Nausea
- Abdominal pain
- Weakness
- Weight loss
- Paradoxical response to antihypertensive drugs
- Polyuria and polydipsia
- Constipation
- Orthostatic hypotension
- Dilated cardiomyopathy
- Erythrocytosis
- Elevated blood sugar
- Hypercalcemia

INVESTIGATIONS

- 24-h urinary tests :
  - Catecholamines
  - Fractionated metanephrines
  - Total metanephrines
- Plasma tests :
  - Catecholamines
  - Free metanephrines
- Imaging :
  - CT
  - MRI
- MIBG scintigraphy
- 18 Fluoro-DOPA PET/CT
- Somatostatin Receptor Scintigraphy

TREATMENT

- Surgical removal is the treatment of choice.
- Minimally invasive techniques (laparoscopy or retroperitoneoscopy) have become the standard approaches in pheochromocytoma surgery.
- Classically, blood pressure has been controlled by α-adrenergic blockers (oral phenoxybenzamine).
- Oral Prazosin or intravenous phentolamine can be used to manage paroxysms while adequate alpha blockade is awaited.
- Beta blockers can then be added.(Not an initial treatment of choice)
- An adrenocorticotropic hormone (ACTH) test (Used to exclude cortisol deficiency when bilateral adrenal cortex sparing surgery has been performed.)

7. Patient with history of recurrent retrosternal chest pain,each episode lasting 3-5 min and subsiding with sublingual nitrate.Now presented in emergency with chest pain.He is k/c/o HTN, DM, Hypercholesterolemia.He is on Lovastatin,Aspirin,Atenolol and Metformin.ECG shows LVH with Flat T wave.What is the next step in management ?

A. i.v GTN Infusion

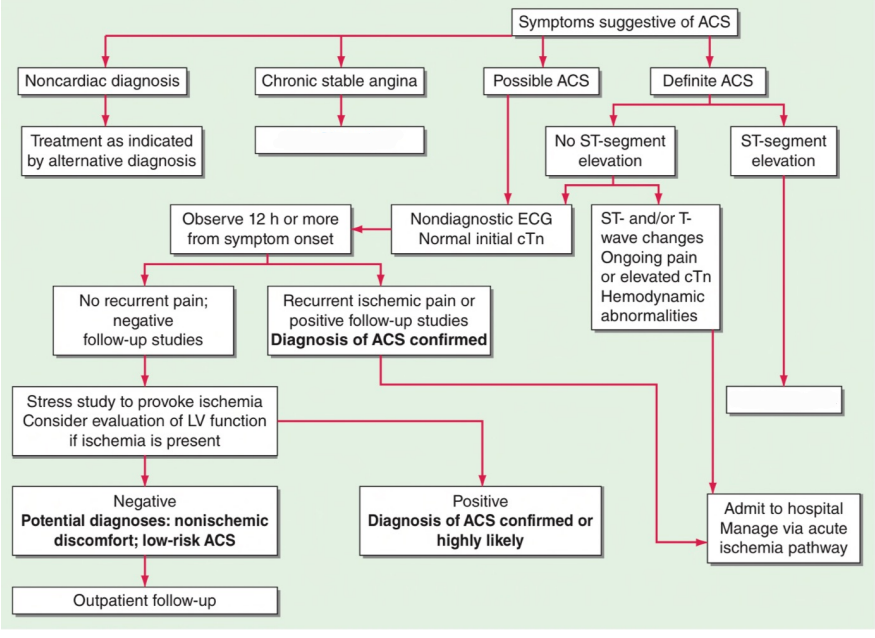
B. Injection Enoxaparin

C. Add Clopidogrel

D. Increase the dose of beta blocker

- Based on the given history it is suggestive of Acute Coronary Syndrome.Since ECG is not showing any ST segment elevation ,most probable diagnosis is ACS-NSTEMI

NON-ST-SEGMENT ELEVATION ACUTE CORONARY SYNDROME



Reference :
Notespaedia Medicine High Yield Pg : 14
Harrison's Principles of Internal Medicine 20th Ed.

MANAGEMENT

Initial Treatment

DAPT and Anticoagulant therapy:
1. Aspirin (COR I, LOE A).
2. P2Y 12 inhibitor: clopidogrel or ticagrelor (COR I, LOE B).
3. Anticoagulant:
   Enoxaparin (COR I, LOE A) or UFH (COR I, LOE B) or fondaparinux (COR I, LOE B) or bivalirudin (for early invasive strategy, COR I, LOE B).
4. Can consider GP IIb/IIIa receptor inhibitors in high-risk patients stratified to early invasive strategy (eptifibatide or tirofiban; COR IIb, LOE B).

During Hospitalization

Medically treated patients:
1. Aspirin (COR I, LOE A).
2. P2Y 12 inhibitor: either ticagrelor or clopidogrel (COR I, LOE B).
3. Anticoagulant:
   Enoxaparin (COR I, LOE A) or UFH (COR I, LOE B) or fondaparinux (COR I, LOE B).

PCI treated patients:
1. Aspirin (COR I. LOE A).
2. P2Y 12: inhibitor: clopidogrel or ticagrelor or prasugrel (COR I, LOE B).
3. Anticoagulant:
   Enoxaparin (COR I, LOE A) or UFH (COR I, LOE B) or fondaparinux* (COR I, LOE B) or bivalirudin (COR I, LOE B).
4. Can consider GP IIb/IIIa receptor inhibitors in high-risk patients not adequately per-treated with clopidogrel (COR I, LOE A) or in high-risk patients adequately pre-treated with clopidogrel (COR IIa, LOE B).

Long-term

Medically treated patients:
1. Aspirin indefinitely (COR I, LOE A).
2. P2Y 12 inhibitor: clopidogrel or ticagrelor for up to 12 months (COR I, LOE B)

PCI treated patients:
1. Aspirin indefinitely (COR I, LOE A).
2. P2Y 12 inhibitor: clopidogrel or ticagrelor or prasugrel for at least 12 months (COR I, LOE B).

COR, classes of recommendation; DAPT, dual antiplatelet therapy; GP IIb/IIIa, glycoprotein IIb/IIIa; LOE, levels of evidence; NSTE-ACS, non-ST-segment elevation acute coronary syndrome; PCI, percutaneous coronary intervention; UFH, unfractionated heparin.

8. A patient complains of headache for past 2 years for which she took analgesics regularly.There is no history of nausea,vomiting or blurred vision or photophobia.However from the past 6 months the headache was not relieved by NSAID's and was worsening.Now on stopping the analgesics the headaches have improved.Likely diagnosis is ?

A. Chronic migraine
B. Tension headache
C. Medication overuse headache
D. New onset persistent headache

MEDICATION-OVERUSE HEADACHE

- Overuse of analgesic medication for headache can aggravate headache frequency, markedly impair the effect of preventive medicines, and induce a state of refractory daily or near-daily headache.
- Even after cessation of analgesic use, many patients continue to have headache, although they may feel clinically improved in some way, especially if they have been using opioids or barbiturates regularly.

Reference :
Harrison's Principles of Internal Medicine 20th Ed.

DIAGNOSIS

A. Headaches occuring on ≥ 15days/month in a patient with a pre-existing headache disorder.
B. Regular overuse for ≥ 3 month of one or more drugs that can be etaken fro acute and/or symptomatic treatment of headache.
C. Not better accounted for by another ICHD-3 Diagnosis

MEDICATIONS THAT CAN TRIGGER

- Barbiturates : ≥ 10days/month
- Opiates : ≥ 10 days/month
- Triptans : ≥ 10days/month
- OTC Analgesics

MANAGEMENT

i. Outpatients :

- Reduce the medication dose by 10% every 1-2 weeks.
- A small dose of a nonsteroidal anti-inflammatory drug (NSAID) such as naproxen, if tolerated, will help relieve residual pain as analgesic use is reduced.

ii. Inpatients :

- Antiemetics and fluids are administered as required.
- Clonidine is used for opioid withdrawal symptoms.
- For acute intolerable pain during the waking hours, Aspirin.
- IM chlorpromazine can be helpful at night; patients must be adequately hydrated.
- Serotonin 5-HT3 receptor antagonists, such as Ondansetron or Granisetron, or the neurokinin receptor antagonist, Aprepitant, may be required with DHE to prevent significant nausea, and domperidone orally or by suppository can be very helpful.

Classification of Daily or Near-Daily Headache

- Primary
  - >4 H DAILY
    - Chronic migraine
    - Chronic tension-type headache
    - Hemicrania continuaa
    - New daily persistent headache
  - <4 H DAILY
    - Chronic cluster headache
    - Chronic paroxysmal hemicrania
    - SUNCT/SUNA
    - Hypnic headache
- Secondary
  - Posttraumatic
    - Head injury
    - Iatrogenic
    - Postinfectious
  - Inflammatory, such as :
    - Giant cell arteritis
    - Sarcoidosis
    - Behçet's syndrome
  - Chronic CNS infection
  - Medication-overuse headache

9. 68yr old male with cough,sputum.On Ausculation showed bronchial breath sounds.No confusion.RR 20/min,Urea 44/min,BP 110/70mm Hg.What is the next best step in management ?

A. Home treatment with antibiotics
B. Admit in ICU and a manage without Invasive mechanical ventilation
C. Admit in ICU and manage with Invasive mechanical ventilation
D. Consider hospital admission in non ICU setting

- Although a number of prediction rules exist, the two most frequently used are :
  - Pneumonia Severity Index (PSI), a prognostic model used to identify patients at low risk of dying,
  - CURB-65 criteria, a severity-of-illness score.
- The CURB-65 criteria include five variables :
  - Confusion (C);
  - Urea >7 mmol/ (U);
  - Respiratory rate ≥30/min (R);
  - Blood pressure, systolic ≤90 mmHg or diastolic ≤60 mmHg (B); and
  - Age ≥65 years.
- Patients with :
  - Score of 0 can be treated outside the hospital.
  - Score of 1 or 2, the patient should be hospitalized unless the score is entirely or in part attributable to an age of ≥65 years (In such cases, hospitalization may not be necessary).
  - Scores of ≥3, mortality rates are 22% overall; these patients may require ICU admission.

Treatment Options Based on CURB-65 Score		
0 or 1	Group 1 : Mortality low (1.5%)	Low Risk Consider Home Treatment
2	Group 2 : Mortality Intermediate (9.2%)	Consider Hospital supervised treatment (either short stay inpatient or hospital supervised outpatient)
≥ 3	Group 3 : Mortality high (22%)	Manage in hospital as severe pneumonia;consider admission to intensive care unit,especially with CURB-65 score of 4 or 5

- Here in this clinical scenario age is ≥ 65 , Urea level is high : Score is 2

↓

Patient requires hospital admission but not in ICU

Reference :
Notespaedia Medicine High Yield Pg : 63
Harrison's Principles of Internal Medicine 20th Ed.

10. A patient presented with severe chest pain to the emergency.He had an episode of syncope with transient loss of consciousness.On examination patient was diaphorectic and appeared grossly unstable.Bilateral pulses were unequal.ECG showed tachycardia with non specific ST-T changes.Which of the following investigations will guide further management ?

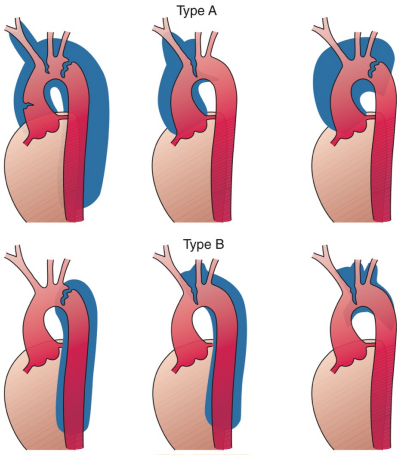
A. TEE
B. Cardiac enzymes
C. Chest CT
D. MRI

- Based given history and ECG findings most probable diagnosis is Aortic Dissection

AORTIC DISSECTION

- Is caused by a circumferential or, less frequently, transverse tear of the intima. It often occurs along the right lateral wall of the ascending aorta where the hydraulic shear stress is high.
- Another common site is the descending thoracic aorta just below the ligamentum arteriosum.

CLASSIFICATION OF AORTIC DISSECTIONS



- Stanford classification :
  - Type A dissections (top) involve the ascending aorta independent of site of tear and distal extension;
  - Type B dissections (bottom) involve transverse and/or descending aorta without involvement of the ascending aorta.
- DeBakey classification :
  - Type I dissection involves ascending to descending aorta (top left);
  - Type II dissection is limited to ascending or transverse aorta, without descending aorta (top center + top right);
  - Type III dissection involves descending aorta only (bottom left).

CLINICAL MANIFESTATIONS

- Men are more affected than women.
- Sudden onset of pain :
  - Described as very severe and tearing and is associated with diaphoresis.
  - May be localized to the front or back of the chest, often the interscapular region, and typically migrates with propagation of the dissection.
- Syncope,
- Dyspnea,
- Weakness.

Reference :
Notespaedia Medicine High Yield Pg : 35
Harrison's Principles of Internal Medicine 20th Ed.

- Hypertension or hypotension,
- Loss of pulses,
- Aortic regurgitation,
- Pulmonary edema, and neurologic findings due to carotid artery obstruction (hemiplegia, hemianesthesia) or spinal cord ischemia (paraplegia).
- Bowel ischemia, hematuria, and myocardial ischemia

DIAGNOSIS

- Noninvasive techniques such as Echocardiography, CT, and MRI.
- Transthoracic echocardiography can be performed simply and rapidly and has an overall sensitivity of 60–85% for aortic dissection.
- CT and MRI : both highly accurate in identifying the intimal flap and the extent of the dissection.
- Contrast Enhanced CT is preferred if patient is stable otherwise TEE.

TREATMENT

- Medical therapy :
  - Unless contraindicated, β-adrenergic blockers should be administered parenterally, using intravenous propranolol, metoprolol, or the short-acting esmolol to achieve a heart rate of ~60 beats/min.
  - Accompanied by sodium nitroprusside infusion to lower systolic blood pressure to ≤120 mmHg.
  - Labetalol a drug with both β- and α-adrenergic blocking properties,may be used as a parenteral agent in acute therapy for dissection.
  - Calcium channel antagonists Verapamil and Diltiazem may be used intravenously if Nitroprusside or β-adrenergic blockers cannot be employed.
- Emergent or urgent surgical correction is the preferred treatment for acute ascending aortic dissections and intramural hematomas (type A).
- Aortic valve repair or a composite valve-graft conduit is used if the aortic valve is disrupted.
- Thoracic endovascular aortic repair with an endoluminal stent graft is indicated for complicated type B dissections

11. A 30yr old male weighing 70kg has a serum Sodium of 120mEq/L.What is the the total sodium deficit ?

A. 280 mEq C. 840 mEq
B. 480 mEq D. 1400mEq

- Total Sodium deficit = TBW x (Desired Sodium - Measured Sodium)
  = 60% x 70 x (140 -120)
  = 0.6 x 70 x 20
  = 42 x 20
  = 840 mEq

Total Sodium deficit = TBW x (Desired Sodium - Measured Sodium)

12. 50yr old male presented with fatiguability for one year.Evaluation showed mild anemia,massive splenomegaly.Hb 10%,TLC 120x10(3),Platelet 500x10(3),Neutrophils 60%,Basophil 4%,Eosinophil 6%,Lymphocytes 15%.Also seen were myeloblasts,myelocytes,metamyelocytes and bands.M:E ratio of 18: 1.What is the most sensitive investigation ?

A. Leucocyte Alkaline Phosphatase
B. FISH with PCR
C. Immunophenotyping
D. Whole body PET CT

- Based on symptoms and lab findings most probable diagnosis is Chronic Myeloid Leukemia.

CHRONIC MYELOID LEUKEMIA

- Is a clonal hematopoietic stem cell disorder.
- BCR-ABL1 chimeric gene product, that codes for a constitutively active tyrosine kinase, resulting from a reciprocal balanced translocation between the long arms of chromosomes 9 and 22, t(9;22)(q34.1;q11.2), known as the Philadelphia chromosome (Ph)

CLINICAL PRESENTATION

- Most patients with CML (90%) present in the indolent or chronic phase.
- Fatigue, malaise, weight loss (if high leukemia burden), or early satiety and left upper quadrant pain or masses (from splenomegaly).
- Common symptoms, when present, are manifestations of anemia and splenomegaly.
- Splenomegaly is the most common physical finding.
- Other less common findings include hepatomegaly (5–10%), lymphadenopathy (5–10%).

INVESTIGATIONS

- In untreated CML, leukocytosis ranging from 10–500 × 109/L is common.
- Peripheral blood :
  - Predominance of neutrophils and the presence of bands, myelocytes, metamyelocytes, promyelocytes, and blasts (usually ≤5%).
  - Basophils and/or eosinophils are frequently increased.
  - Thrombocytosis is common.
  - Anemia
- Bone marrow is hypercellular with marked myeloid hyperplasia and a high myeloid-to-erythroid ratio of 15–20:1.
- Marrow blasts are 5% or less.
- Diagnosis depends on documenting the t(9;22)(q34.1;q11.2).
- Techniques such as FISH and PCR are now used to aid in the diagnosis of CML.

Ways to detect Philadelphia chromosome : Cytogenetics > FISH > RT-PCR

Reference :
Harrison's Principles of Internal Medicine 20th Ed.

13. A patient k/c/o Cirhhosis presents with distended abdomen and jaundice.He now comes with decreased urine output that does not respond to fluids.BP is normal.USG shows cirrhosis with ascites.BUN 42 and Creatinine 1.8.Which of the following is used in treatment ?

A. Torsemide
B. Albumin with Octreotide
C. Liberal Saline Infusion
D. i.v Dobutamine

- Based on given information,USG findings,BUN/Creatinine ratio is more than 20 most probable diagnosis is Hepatorenal Syndrome(HRS).

HEPATORENAL SYNDROME

- Is a form of functional renal failure without renal pathology that occurs in patients with advanced cirrhosis or acute liver failure.
- The diagnosis is made usually in the presence of a large amount of ascites in patients who have a stepwise progressive increase in creatinine.

Types of HRS

Type 1 HRS

Characterized by a progressive impairment in renal function and a significant reduction in creatinine clearance within 1–2 weeks of presentation.

Type 2 HRS

Characterized by a reduction in glomerular filtration rate with an elevation of serum creatinine level, but it is fairly stable and is associated with a better outcome

CRITERIA FOR DIAGNOSIS

- Cirrhosis with Ascites
- S.Creatinine > 1.5mg/dl
- No improvement in Serum Creatinine after 2 days of diuretic withdrawal.
- Absence of Shock
- No current/Recent use of Nephrotoxic drugs
- Absence of Parenchymal kidney disease

TREATMENT

- Current treatment : Midodrine, an α-agonist, along with octreotide and intravenous albumin.
- The best therapy for HRS is liver transplantation
- Stop diuretics
- Stop Anti Hypertensives
- Avoid Nephrotoxic drugs

Reference :
Harrison's Principles of Internal Medicine 20th Ed.

14. A 68yr old female patient presented with Atrial fibrillation with Mitral stenosis.ECHO does not show any thrombus.Which among the following is the best to prevent stroke ?

A. Dabigatran
B. Aspirin
C. Warfarin
D. LMWH

RECOMMENDATIONS FOR THE PREVENTION OF THROMBOEMBOLIC EVENTS IN AF

VKAs - Vitamin K Antagonist

Recommendations	Class	Level
For stroke prevention in AF patients who are eligible for OAC, NOACs are recommended in preference to VKAs (excluding patients with mechanical heart valves or moderate-to-severe mitral stenosis).	I	A
For stroke risk assessment, a risk-factor–based approach is recommended, using the CHA_2DS_2-VASc clinical stroke risk score to initially identify patients at 'low stroke risk' (CHA_2DS_2-VASc score = 0 in men, or 1 in women) who should not be offered antithrombotic therapy.	I	A
OAC is recommended for stroke prevention in AF patients with CHA_2DS_2-VASc score ≥2 in men or ≥3 in women.	I	A

Recommendations	Class	Level
OAC should be considered for stroke prevention in AF patients with a CHA_2DS_2-VASc score of 1 in men or 2 in women. Treatment should be individualized based on net clinical benefit and consideration of patient values and preferences.	IIa	B
For bleeding risk assessment, a formal structured risk-score–based bleeding risk assessment is recommended to help identify non-modifiable and address modifiable bleeding risk factors in all AF patients, and to identify patients potentially at high risk of bleeding who should be scheduled for early and more frequent clinical review and follow-up.	I	B
For a formal risk-score–based assessment of bleeding risk, the HAS-BLED score should be considered to help address modifiable bleeding risk factors, and to identify patients at high risk of bleeding (HAS-BLED score ≥3) for early and more frequent clinical review and follow-up.	IIa	B

Recommendations	Class	Level
Stroke and bleeding risk reassessment at periodic intervals is recommended to inform treatment decisions (e.g. initiation of OAC in patients no longer at low risk of stroke) and address potentially modifiable bleeding risk factors.[a]	I	B
In patients with AF initially at low risk of stroke, first reassessment of stroke risk should be made at 4–6 months after the index evaluation.	IIa	B
If a VKA is used, a target INR of 2.0–3.0 is recommended, with individual TTR ≥70%.	I	B

[a]Including uncontrolled BP; labile INRs (in a patient taking VKA); alcohol excess; concomitant use of NSAIDs or aspirin in an anticoagulated patient; bleeding tendency or predisposition (e.g. treat gastric ulcer, optimize renal or liver function etc.).

Recommendations	Class	Level
In patients on VKAs with low time in INR therapeutic range (e.g. TTR <70%), recommended options are: • Switching to a NOAC but ensuring good adherence and persistence with therapy; or	I	B
• Efforts to improve TTR (e.g. education/counselling and more frequent INR checks).	IIa	B
Antiplatelet therapy alone (monotherapy or aspirin in combination with clopidogrel) is not recommended for stroke prevention in AF.	III	A
Estimated bleeding risk, in the absence of absolute contraindications to OAC, should not in itself guide treatment decisions to use OAC for stroke prevention.	III	A
Clinical pattern of AF (i.e. first detected, paroxysmal, persistent, long-standing persistent, permanent) should not condition the indication to thromboprophylaxis.	III	B

Recommendations for occlusion or exclusion of the LAA	Class	Level
LAA occlusion may be considered for stroke prevention in patients with AF and contraindications for long-term anticoagulant treatment (e.g. intracranial bleeding without a reversible cause).	IIb	B
Surgical occlusion or exclusion of the LAA may be considered for stroke prevention in patients with AF undergoing cardiac surgery.	IIb	C

15. Patient presented with Seizure,urine output 650ml/24hrs,Urine Osmolality 1000 mOsm/kg,Serum Osmolality 270mOsm/Kg.On further evaluation what electrolyte abnormality is expected ?

A. Hyponatremia
B. Hypokalemia
C. Hypernatremia
D. Hyperkalemia

- This is a case of SIADH.

HYPONATREMIA DUE TO INAPPROPRIATE ANTIDIURESIS

- Decrease in plasma osmolarity/sodium below the normal range (hypotonic hyponatremia) can be due to any of three different types :
  (1) an increase in total body water that exceeds the increase in total body sodium (hypervolemic hyponatremia);
  (2) a decrease in body sodium greater than the decrease in body water (hypovolemic hyponatremia); or
  (3) an increase in body water with little or no change in body sodium (euvolemic hyponatremia)

CLINICAL CHARACTERISTICS

- Antidiuresis of any cause decreases the volume and increases the concentration of urine.
- Acute cases : accompanied by symptoms and signs of water intoxication that may include mild headache, confusion, anorexia, nausea, vomiting, coma, and convulsions.
- Severe acute hyponatremia may be lethal.
- The hypervolemic form : Characterized by generalized edema and other signs of marked volume expansion.

CAUSES

- Neoplasms
- Carcinomas
  - Lung
  - Duodenum
  - Pancreas
  - Ovary
  - Bladder, ureter
- Head trauma (closed and penetrating)
- Infections
  - Pneumonia, bacterial or viral
  - Abscess, lung or brain
  - Cavitation (aspergillosis)
  - Tuberculosis, lung or brain
  - Meningitis, bacterial or viral
  - Encephalitis
  - AIDS
- Neurologic
  - Guillain-Barré syndrome
  - Multiple sclerosis
  - Delirium tremens
  - Amyotrophic lateral sclerosis
  - Hydrocephalus
  - Psychosis
  - Peripheral neuropathy

- Drugs
  - Vasopressin or desmopressin
  - Serotonin reuptake inhibitors
  - Oxytocin,
  - High dose Vincristine
  - Carbamazepine
  - Nicotine
  - Phenothiazines
  - Cyclophosphamide
  - Tricyclic antidepressants
  - Monoamine oxidase inhibitors
- Metabolic
  - Acute intermittent porphyria
  - Pulmonary
  - Asthma
  - Pneumothorax Positive-pressure respiration

TREATMENT

- In acute symptomatic SIADH : raise plasma osmolarity and/ or plasma sodium at a rate ~1 % an hour until they reach levels of ~270 mosmol/L or 130 meq/L, respectively.
- Reduce body water by giving an AVP receptor-2 antagonist (vaptan) to block the antidiuretic effect of AVP and increase urine output
- A combined V2/V1a antagonist (Conivaptan), has been approved for short-term, in- hospital IV treatment.
- Fluid intake should be restricted to less than urine output.

16. A female patient feels numb on the fingertips.Her facial skin was tightened.ANA was found to be positive.Immunofluorescence showed nucleolar pattern.What is most probable diagnosis ?

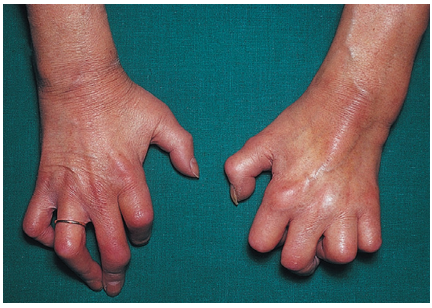
A. Systemic sclerosis
B. Sjogren's syndrome
C. SLE
D. Rheumatoid arthritis

SYSTEMIC SCLEROSIS

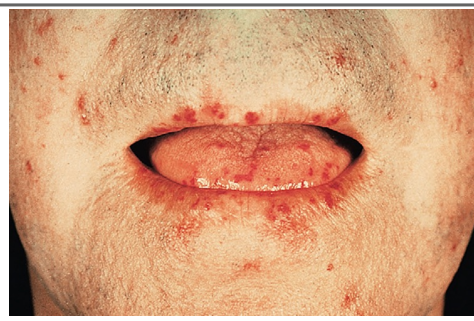
- Multisystem disorder characterized by thickening of the skin (scleroderma) and distinctive involvement of multiple internal organs

DIFFUSE CUTANEOUS SSC	LIMITED CUTANEOUS SSC
• Rapid development of symmetric skin thickening of proximal and distal extremity, face, and trunk. • At high risk for development of visceral disease early in course.	• Long-standing Raynaud's phenomenon • Skin involvement limited to fingers (sclerodactyly), extremity distal to elbows, and face • May have 'CREST' syndrome (Calcinosis, Raynaud's, oEsophageal involvement, Sclerodactyly and Telangiectasia).

Reference :
Notespaedia Medicine High Yield Pg : 171
Harrison's Principles of Internal Medicine 20th Ed



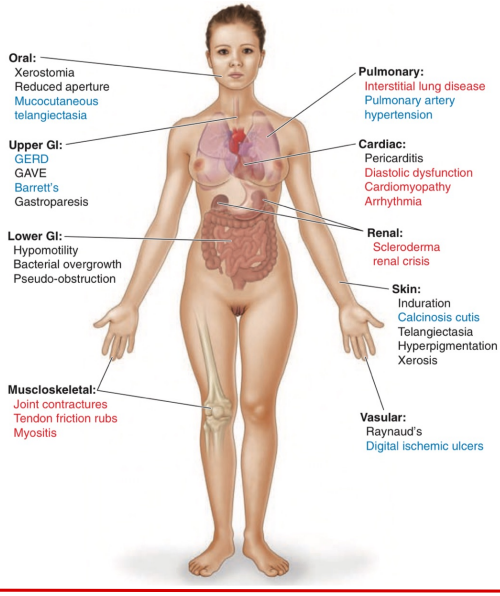
Hands showing tight, shiny skin, sclerodactyly, flexion contractures of the fingers and thickening of the left middle finger extensor tendon sheath.



Typical facial appearance in the CREST syndrome

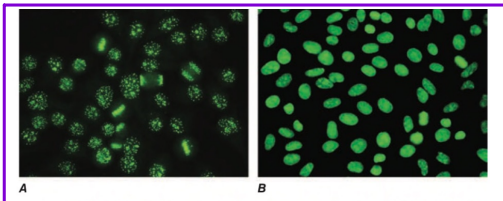
CLINICAL MANIFESTATIONS

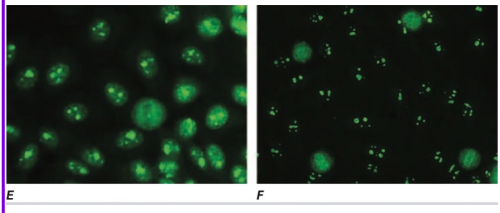
- Multi-organ involvement in systemic sclerosis



LABORATORY INVESTIGATIONS

- ESR is usually elevated and raised levels of IgG,
- CRP values - increased in severe organ involvement or coexisting infection.
- ANA is positive in about 70%,
- 30% of patients with DCSS have antibodies to topoisomerase 1 (Scl70).
- 60% of patients with CREST syndrome have anticentromere antibodies





Indirect immunofluorescence on HEp-2 substrate shows distinct patterns: A. anti- centromere; B. anti-Scl-70/ topoisomerase I; C. anti-PM/Scl; D. anti-Th/To; E. anti-RNA polymerase III; F. anti-fibrillarin/U3RNP antibodies. Except for anti-centromere (discrete dots in metaphase nucleus), variations of nucleolar staining are clues to autoantibody specificity. However, immunoassays employing purified autoantigens are recommended to confirm specificity of these autoantibodies.

SSc-associated autoantibodies: immunofluorescence patterns.

MANAGEMENT

- Raynaud's should be treated by avoidance of cold exposure and calcium antagonists
- Oesophageal reflux should be treated with proton pump inhibitors and antireflux agents.
- Hypertension should be treated with ACE inhibitors
- Joint involvement may be treated with analgesics and/or NSAID.
- Pulmonary hypertension may be treated with bosentan.

SJÖGREN'S SYNDROME

- Is a chronic, slowly progressing autoimmune dis- ease characterized by lymphocytic infiltration of the exocrine glands resulting in xerostomia and dry eyes (keratoconjunctivitis sicca).

ASSOCIATION WITH OTHER AUTOIMMUNE DISEASES

- Rheumatoid arthritis
- Systemic lupus erythematosus
- Scleroderma
- Mixed connective tissue disease
- Primary biliary cirrhosis
- Autoimmune thyroid disease
- Chronic active hepatitis

CLINICAL MANIFESTATIONS

- Non-Specific :
  - Fatigability/Myalgias : Fibromyalgia
  - Arthralgias/Arthritis : Usually non-erosive, leading to Jaccoud's arthropathy
  - Raynaud's phenomenon
- Peri-Epithelial :
  - Lung involvement : Small airway disease/lymphocyte interstitial pneumonitis
  - Kidney involvement : Interstitial kidney disease is usually asymptomatic
  - Liver involvement : Primary biliary cirrhosis stage I

- Immune-Complex mediated :
  - Small vessel vasculitis : Purpura, urticarial lesions
  - Peripheral neuropathy : Polyneuropathy, either sensory or sensorimotor
  - Glomerulonephritis : Membranoproliferative
- Lymphoma : Glandular MALT lymphoma is most common

Revised International Classification Criteria for SjÖgren's Syndrome

I. Ocular symptoms: a positive response to at least one of three validated questions.
   1. Have you had daily, persistent, troublesome dry eyes for >3 months?
   2. Do you have a recurrent sensation of sand or gravel in the eyes?
   3. Do you use tear substitutes more than three times a day?

II. Oral symptoms: a positive response to at least one of three validated questions.
   1. Have you had a daily feeling of dry mouth for >3 months?
   2. Have you had recurrent or persistently swollen salivary glands as an adult?
   3. Do you frequently drink liquids to aid in swallowing dry foods?

III. Ocular signs: objective evidence of ocular involvement defined as a positive result to at least one of the following two tests:
   1. Shirmer's I test, performed without anesthesia (≤5 mm in 5 min)
   2. Rose Bengal score or other ocular dye score (≥4 according to van Bijsterveld's scoring system)

IV. Histopathology: In minor salivary glands focal lymphocytic sialoadenitis, with a focus score ≥1.

V. Salivary gland involvement: objective evidence of salivary gland involvement defined by a positive result to at least one of the following diagnostic tests:
   1. Unstimulated whole salivary flow (≤1.5 mL in 15 min)
   2. Parotid sialography
   3. Salivary scintigraphy

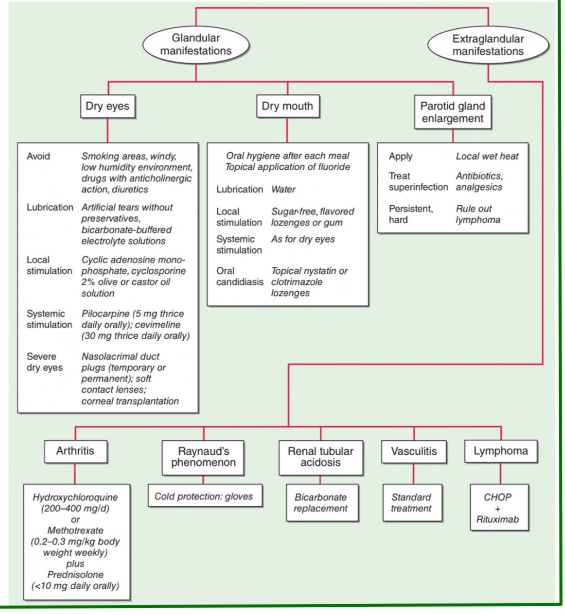
VI. Antibodies in the serum to Ro/SS-A or La/SS-B antigens, or both.

aExclusion criteria: past head and neck radiation treatment, hepatitis C infection, AIDS, preexisting lymphoma, sarcoidosis, graft-versus-host disease, use of anticholinergic drugs. bPrimary Sjögren's syndrome: any four of the six items, as long as item IV (histopathology) or VI (serology) is positive; or any three of the four objective-criteria items (III, IV, V, VI). cIn patients with a potentially associated disease (e.g., another well-defined connective tissue disease), the presence of item I or item II plus any two from among items III, IV, and V may be considered indicative of secondary Sjögren's syndrome.

DIAGNOSIS

- Diagnosed if :
  (1) the patient presents with eye and/ or mouth dryness,
  (2) eye tests disclose keratoconjunctivitis sicca,
  (3) mouth evaluation reveals dry oral mucosa, and/or
  (4) the patient's serum reacts with immunoglobulins (rheumatoid factors), Ro/SS-A, and/or La/ SS-B autoantigens.

TREATMENT ALGORITHM



17. Patient with vomiting,was treated with anti-emetics.Patient was relieved of symptoms,but then later develop abnormal movements.What is the drug to be prescribed to reduce the movements ?

A. Scopolamine/Hyoscine
B. Methyldopa
C. Benzhexol
D. Cyproheptadine

- This is a case of Acute Drug Induced Dystonia.

DRUG INDUCED DYSTONIA

- Terms used to describe drug-induced dystonia include: tardive dystonia; tardive dyskinesias; acute dystonic reaction.

SYMPTOMS

- May include intermittent spasmodic or sustained involuntary contractions of muscles in the face, neck, trunk, pelvis, and extremities.
- Symptoms are usually transient
- Dyskinesias are usually characterized by quick, jerking movements that may include grimacing, tongue protrusion, lip smacking, puckering, and eye blinking.
- The arms, legs, and trunk may also be involved.
- Movements of the fingers may appear as though the individual is playing an invisible guitar or piano.
- The frequency and pattern of movements may fluctuate.

CAUSE

- Drugs belonging to this class of neuroleptics include (trade name listed in parenthesis): Acetohenazine , Amoxapine, Chlorpromazine,Fluphenazine, Haloperidol, Mesoridazine, metaclopramide , Molinndone, Perphanzine , Piperacetazine, prochlorperzine , Promazine, promethazine , Thiethylperazine ,Thioridazine, Thiothixene,Trifluoperazine,Triflupromazine and Trimeprazine .

TREATMENT

- Gradual withdrawal from the offending medication.
- A class of newer, "atypical" neuroleptics (such as clozapine, olanzapine, and quetiapine) may be a suitable substitute.
- Anticholinergics (such as trihexyphenidyl and benztropine) and muscle relaxers used to treat other forms of dystonia may also be helpful.
- Baclofen and clonazepam are also sometimes used to treat tardive dystonia.
- Botulinum toxin injections to a particular muscle group are an additional option for treatment.

18. A 54 yr old male patients from Chattisgarh having progressive motor paresis symptoms and leg stiffness.What history will you enquire for making a diagnosis ?

A. Diet history
B. Vaccination history
C. History of Fever
D. Past history of similar illness

- Based on given history this is suggestive of Neurolathyrism so dietary history is important in making diagnosis.

NEUROLATHYRISM

- Is a neurological disease of humans, caused by eating certain legumes of the genus Lathyrus.
- This disease is mainly associated with the consumption of Lathyrus sativus (also known as grass pea, chickling pea, kesari dal, or almorta) and to a lesser degree with Lathyrus cicera, Lathyrus ochrus and Lathyrus clymenum containing the toxin ODAP.
- Two free amino acids : BOAA (Beta -N- Oxalylamino-L Alanine and Beta Diaminopropionic acid have found to be toxic.

SIGNS AND SYMPTOMS

- Causes paralysis, characterized by lack of strength in or inability to move the lower limbs, and may involve pyramidal tracts producing signs of upper motor neuron damage.
- The toxin may also cause aortic aneurysm.
- A unique symptom of lathyrism is the atrophy of gluteal muscles (buttocks).
- Children can additionally develop bone deformity and reduced brain development.

CAUSES

- Toxicological cause of the disease has been attributed to the neurotoxin ODAP which acts as a structural analogue of the neurotransmitter glutamate.

MANAGEMENT

- Disease is usually nonprogressive but irreversible.
- Tolperisone, a centrally acting muscle relaxant, has been shown to produce significant reduction in the spasticity in neurolathyrism patients.

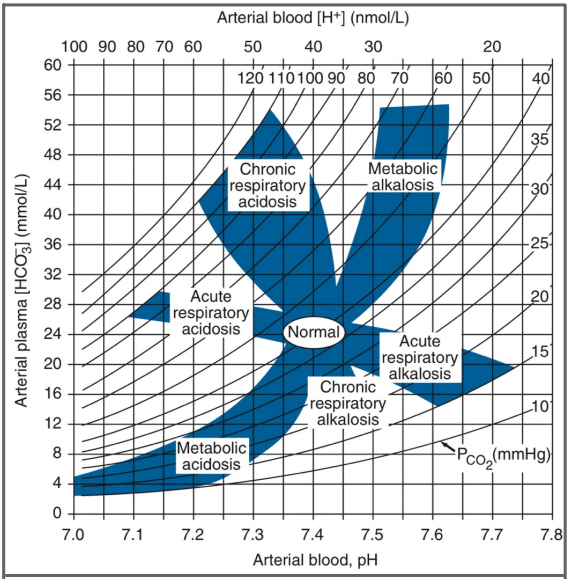
PREVENTION

- Food preparation measures can help:
  - i. Boiling in water or repeated steeping in hot water and discarding the extracts can detoxify seeds.
  - ii. Roasting the seeds at 140 C for 15 to 20 minutes results in 80-90% destruction of neurotoxins.
  - iii. Soaking the seeds or dhal overnight and decanting the water before cooking eliminates about 90% of the toxin.

19. A female patient with hysteria was hyperventilating.She developed carpopedal spasm.On ABG pH =7.52,pCO2 =20,HCO3 - = 24.What is the acid base abnormality ?

A. Respiratory alkalosis
B. Metabolic alkalosis
C. Respiratory acidosis
D. Metabolic acidosis

- Based on the given values and patient hyperventilating most probable diagnosis is Respiratory alkalosis.



Acid-base nomogram.

NORMAL VALUES

- pH (7.35-7.45)
- PaO2 (75-100 mmHg)
- PaCO2 (35-45 mmHg)
- HCO3 (22-26 meq/L)
- Base excess/deficit (-4 to +2)
- SaO2 (95-100%)

TREATMENT OF RESPIRATORY ALKALOSIS

- The management of respiratory alkalosis is directed toward alleviation of the underlying disorder.
- Patients with the hyperventilation syndrome may benefit from reassurance, rebreathing from a paper bag during symptomatic attacks, and attention to underlying psychological stress.
- Antidepressants and sedatives are not recommended.
- β blockers may ameliorate peripheral manifestations of the hyperadrenergic state.

Prediction of Compensatory Responses to Simple Acid-Base Disturbances and Pattern of Changes

DISORDER	PREDICTION OF COMPENSATION	RANGE OF VALUES		
		pH	HCO_3^-	$Paco_2$
Metabolic acidosis	$Paco_2 = (1.5 \times HCO_3^-) + 8 \pm 2$ *or* $Paco_2$ will ↓ 1.25 mmHg per mmol/L ↓ in $[HCO_3^-]$ *or* $Paco_2 = [HCO_3^-] + 15$	Low	Low	Low
Metabolic alkalosis	$Paco_2$ will ↑ 0.75 mmHg per mmol/L ↑ in $[HCO_3^-]$ *or* $Paco_2$ will ↑ 6 mmHg per 10 mmol/L ↑ in $[HCO_3^-]$ *or* $Paco_2 = [HCO_3^-] + 15$	High	High	High
Respiratory alkalosis		High	Low	Low
Acute	$[HCO_3^-]$ will ↓ 0.2 mmol/L per mmHg ↓ in $Paco_2$			
Chronic	$[HCO_3^-]$ will ↓ 0.4 mmol/L per mmHg ↓ in $Paco_2$			
Respiratory acidosis		Low	High	High
Acute	$[HCO_3^-]$ will ↑ 0.1 mmol/L per mmHg ↑ in $Paco_2$			
Chronic	$[HCO_3^-]$ will ↑ 0.4 mmol/L per mmHg ↑ in $Paco_2$			

Reference :
Notespaedia Medicine High Yield Pg : 120
Harrison's Principles of Internal Medicine 20th Ed.

20 .A hypertensive patient presented with ESRD and pedal edema.Pateint is not having urine output.On investigations,Serum Creatinine was found to be 1.9mg/dl.Which of the following drug should be used for management of Hypertension in this patient ?

A. Prazosin

B. Aliskiren

C. Chlorthalidone

D. Amlodipine

- As patient is already having pedal edema Amlodipine cannot be used.
- Since Patient is in ESRD and having not urine output Chlorthalidone can't be used.
- Aliskiren can't be used in this patient because it is not actively metabolized in liver and is primarily excreted in urine.
- Based on given history Prazosin is drug used in management of hypertension in this patient because it undergoes hepatic clearance.

PRAZOSIN

- Is the prototypical α1-selective antagonist.

PHARMACOLOGICAL EFFECTS

- Major effects of prazosin result from its blockade of α1 receptors in arterioles and veins.
- This leads to a fall in peripheral vascular resistance and in venous return to the heart.
- Does not increase heart rate.

ADME

- Prazosin is well absorbed after oral administration, and bioavailability is about 50%–70%.
- Extensively metabolized in the liver, and little unchanged drug is excreted by the kidneys.
- The plasma t 1/2 is about 3 h (may be prolonged to 6–8 h in congestive heart failure).
- The duration of action is approximately 7–10 h in the treatment of hypertension.
- The dose is titrated upward depending on the blood pressure.
- In the off-label treatment of BPH, doses from 1 to 5 mg twice daily typically are used.

HYPERTENSIVE VASCULAR DISEASE

Blood Pressure Classification

BLOOD PRESSURE CLASSIFICATION	SYSTOLIC, mmHg	DIASTOLIC, mmHg
Normal	<120	*and* <80
Prehypertension	120–139	*or* 80–89
Stage 1 hypertension	140–159	*or* 90–99
Stage 2 hypertension	≥160	*or* ≥100
Isolated systolic hypertension	≥140	*and* <90

BASIC LABORATORY TESTS FOR INITIAL EVALUATION

SYSTEM	TEST
Renal	Microscopic urinalysis, albumin excretion, serum BUN and/or creatinine

SYSTEM	TEST
Endocrine	Serum sodium, potassium, calcium, TSH
Metabolic	Fasting blood glucose,Total cholesterol,HDL and LDL(often computed) cholesterol,TG's
Other	Hematocrit,Electrocardiogram

LIFESTYLE MODIFICATIONS TO MANAGE HYPERTENSION

- Weight reduction – Attain and maintain BMI <25 kg/m2
- Dietary salt reduction – <6 g NaCl/d
- Adapt DASH-type dietary plan – Diet rich in fruits, vegetables, and low-fat dairy products with reduced content of saturated and total fat
- Moderation of alcohol consumption – For those who drink alcohol, consume ≤2 drinks/d in men and ≤1 drink/d in women.
- Physical activity – Regular aerobic activity, e.g., brisk walking for 30 min/d

Oral Drugs Used in Treatment of Hypertension

DRUG CLASS	EXAMPLES	USUAL TOTAL DAILY DOSE[a] (DOSING FREQUENCY/DAY)	OTHER INDICATIONS	CONTRAINDICATIONS/CAUTIONS
Diuretics				
Thiazides	Hydrochlorothiazide	6.25–50 mg (1–2)		Diabetes, dyslipidemia, hyperuricemia, gout, hypokalemia
	Chlorthalidone	25–50 mg (1)		
Loop diuretics	Furosemide	40–80 mg (2–3)	CHF due to systolic dysfunction, renal failure	Diabetes, dyslipidemia, hyperuricemia, gout, hypokalemia
	Ethacrynic acid	50–100 mg (2–3)		
Aldosterone antagonists	Spironolactone	25–100 mg (1–2)	CHF due to systolic dysfunction, primary aldosteronism	Renal failure, hyperkalemia
	Eplerenone	50–100 mg (1–2)		
K^+ retaining	Amiloride	5–10 mg (1–2)		Renal failure, hyperkalemia
	Triamterene	50–100 mg (1–2)		
Beta blockers				Asthma, COPD, 2nd- or 3rd-degree heart block, sick-sinus syndrome
Cardioselective	Atenolol	25–100 mg (1)	Angina, CHF due to systolic dysfunction, post-MI, sinus tachycardia, ventricular tachyarrhythmias	
	Metoprolol	25–100 mg (1–2)		
Nonselective	Propranolol	40–160 mg (2)		
	Propranolol LA	60–180 (1)		
Combined alpha/beta	Labetalol	200–800 mg (2)		
	Carvedilol	12.5–50 mg (2)		
Alpha antagonists				
Selective	Prazosin	2–20 mg (2–3)	Prostatism	
	Doxazosin	1–16 mg (1)		
	Terazosin	1–10 mg (1–2)		
Nonselective	Phenoxybenzamine	20–120 mg (2–3)	Pheochromocytoma	
Sympatholytics				
Central	Clonidine	0.1–0.6 mg (2)		
	Clonidine patch	0.1–0.3 mg (1/week)		
	Methyldopa	250–1000 mg (2)		
	Reserpine	0.05–0.25 mg (1)		
	Guanfacine	0.5–2 mg (1)		
ACE inhibitors	Captopril	25–200 mg (2)	Post-MI, coronary syndromes, CHF with low ejection fraction, nephropathy	Acute renal failure, bilateral renal artery stenosis, pregnancy, hyperkalemia
	Lisinopril	10–40 mg (1)		
	Ramipril	2.5–20 mg (1–2)		
Angiotensin II antagonists	Losartan	25–100 mg (1–2)	CHF with low ejection fraction, nephropathy, ACE inhibitor cough	Renal failure, bilateral renal artery stenosis, pregnancy, hyperkalemia
	Valsartan	80–320 mg (1)		
	Candesartan	2–32 mg (1–2)		
Renin inhibitors	Aliskiren	150–300 mg (1)	Diabetic nephropathy	Pregnancy
Calcium antagonists				
Dihydropyridines	Nifedipine (long-acting)	30–60 mg (1)		
Nondihydropyridines	Verapamil (long-acting)	120–360 mg (1–2)	Post-MI, supraventricular tachycardias, angina	2nd- or 3rd-degree heart block
	Diltiazem (long-acting)	180–420 mg (1)		
Direct vasodilators	Hydralazine	25–100 mg (2)		Severe coronary artery disease
	Minoxidil	2.5–80 mg (1–2)		

[a]At the initiation of therapy, lower doses may be preferable for elderly patients and for select combinations of antihypertensive agents.

21. A CKD Patient undergoing pyeloplasty.Which among the following is the best suited Post Op Analgesics ?

A. Diclofenac
B. Naproxen
C. Indomethacin
D. Acetaminophen

- Among the following except Acetaminophen rest all are Prostaglandin inhibitors which causes Afferent Arteriolar Constriction can precipitate Acute on Chronic Kidney Disease.
- Acetaminophen is safe in CKD patient.

CHRONIC KIDNEY DISEASE

- Encompasses a spectrum of pathophysiologic processes associated with abnormal kidney function and a progressive decline in glomerular filtration rate (GFR).

ETIOLOGIES OF CKD

- Diabetic nephropathy
- Glomerulonephritis
- Hypertension-associated CKD (includes vascular and ischemic kidney disease and primary glomerular disease with associated hypertension)
- Autosomal dominant polycystic kidney disease
- Other cystic and tubulointerstitial nephropathy

TREATMENT

- Dietary salt restriction and the use of loop diuretics, occasionally in combination with Metolazone. Water restriction is indicated only if there is a problem with hyponatremia.
- Hyperkalemia often responds to dietary restriction of potassium, the use of kaliuretic diuretics, and avoidance of both potassium supplements (including occult sources, such as dietary salt substitutes) and dose reduction or avoidance of potassium-retaining medications (especially angiotensin-converting enzyme [ACE] inhibitors or angiotensin receptor blockers [ARBs]).
- Kaliuretic diuretics promote urinary potassium excretion.
- Potassium-binding resins, such as calcium resonium, sodium polystyrene or patiromer can promote potassium loss through the GI tract and may reduce the incidence of hyperkalemia.
- Intractable hyperkalemia is an indication (although uncommon) to consider institution of dialysis in a CKD patient.
- The renal tubular acidosis and subsequent anion-gap metabolic acidosis in progressive CKD will respond to alkali supplementation, typically with sodium bicarbonate.

ACETAMINOPHEN

- Raises the threshold to painful stimuli, thus exerting an analgesic effect against pain due to a variety of etiologies.

MECHANISM OF ACTION

- Has analgesic and antipyretic effects similar to those of aspirin, but only weak anti-inflammatory effects.
- It is a nonselective COX inhibitor, which acts at the peroxide site of the enzyme.
- The presence of high concentrations of peroxides, as occur at sites of inflammation, reduces its COX-inhibitory activity.

ADME

- Has excellent bioavailability.
- Peak plasma concentrations occur within 30–60 min, and the t1/2 in plasma is about 2 h.
- A small proportion of acetaminophen undergoes CYP-mediated N-hydroxylation to form NAPQI

THERAPEUTIC USES

- Suitable for analgesic or antipyretic uses; it is particularly valuable for patients in whom aspirin is contraindicated (e.g., those with aspirin hypersensitivity, children with a febrile illness, patients with bleeding disorders).

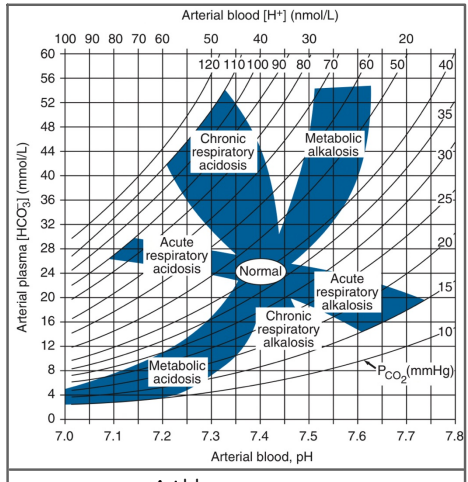
ADVERSE EFFECTS AND TOXICITY

- Usually is well tolerated.
- The most serious acute adverse effect of overdosage of acetaminophen is a potentially fatal hepatic necrosis.

22. A diabetic female patient admitted with persistent vomiting with history of consuming food from outside. BP – 90/60 mm Hg. pH: 7.52. HCO 3- 26. PaCO 2: 20+. Electrolytes: Na- 123, Potassium – 3.2 Chloride – 67 meq/l.What is the cause of her ABG findings?

A. DKA
B. Persistent vomiting
C. Septic chock
D. Renal tubular acidosis

- Based values of pH,Bicarbonate,PaCO2 it is Metabolic alkalosis.



Acid-base nomogram.

NORMAL VALUES

- pH (7.35-7.45)
- PaO2 (75-100 mmHg)
- PaCO2 (35-45 mmHg)
- HCO3 (22-26 meq/L)
- Base excess/deficit (-4 to +2)
- SaO2 (95-100%)

Prediction of Compensatory Responses to Simple Acid-Base Disturbances and Pattern of Changes

DISORDER	PREDICTION OF COMPENSATION	RANGE OF VALUES		
		pH	HCO_3^-	$Paco_2$
Metabolic acidosis	$Paco_2 = (1.5 \times HCO_3^-) + 8 \pm 2$ *or* $Paco_2$ will ↓ 1.25 mmHg per mmol/L ↓ in $[HCO_3^-]$ *or* $Paco_2 = [HCO_3^-] + 15$	Low	Low	Low
Metabolic alkalosis	$Paco_2$ will ↑ 0.75 mmHg per mmol/L ↑ in $[HCO_3^-]$ *or* $Paco_2$ will ↑ 6 mmHg per 10 mmol/L ↑ in $[HCO_3^-]$ *or* $Paco_2 = [HCO_3^-] + 15$	High	High	High
Respiratory alkalosis		High	Low	Low
Acute	$[HCO_3^-]$ will ↓ 0.2 mmol/L per mmHg ↓ in $Paco_2$			
Chronic	$[HCO_3^-]$ will ↓ 0.4 mmol/L per mmHg ↓ in $Paco_2$			
Respiratory acidosis		Low	High	High
Acute	$[HCO_3^-]$ will ↑ 0.1 mmol/L per mmHg ↑ in $Paco_2$			
Chronic	$[HCO_3^-]$ will ↑ 0.4 mmol/L per mmHg ↑ in $Paco_2$			

METABOLIC ALKALOSIS

- Established by an elevated arterial pH, an increase in the serum [HCO3−], and an increase in Paco2 as a result of compensatory alveolar hypoventilation.
- It is often accompanied by hypochloremia and hypokalemia.
- Frequently occurs as a mixed acid base disorder in association with either respiratory acidosis, respiratory alkalosis, or metabolic acidosis.

PATHOGENESIS

- Occurs as a result of net gain of [HCO3−] or loss of nonvolatile acid (usually HCl by vomiting) from the extracellular fluid.
- When vomiting causes loss of HCl from the stomach, HCO3− secretion cannot be initiated in the small bowel and thus HCO3 is added to the extracellular fluid.

CAUSES

I. Exogenous HCO – loads 3
  A. Acute alkali administration
  B. Milk-alkali syndrome
II. Effective ECFV contraction, normotension, K+ deficiency, and secondary hyperreninemic hyperaldosteronism
  A. Gastrointestinal origin
    1. Vomiting
    2. Gastric aspiration
    3. Congenital chloridorrhea
    4. Gastrocystoplasty
    5. Villous adenoma

B. Renal origin :

1. Diuretics
2. Posthypercapnic state
3. Hypercalcemia/hypoparathyroidism
4. Recovery from lactic acidosis or ketoacidosis
5. Nonreabsorbable anions including penicillin, carbenicillin
6. Mg2+ deficiency
7. K+ depletion
8. Bartter's syndrome
9. Gitelman's syndrome

III. ECFV expansion, hypertension, K+ deficiency, and mineralocorticoid excess

A. High renin :

1. Renal artery stenosis
2. Accelerated hypertension
3. Renin-secreting tumor
4. Estrogen therapy

B. Low renin

1. Primary aldosteronism
   a. Adenoma b. Hyperplasia c. Carcinoma
2. Adrenal enzyme defects :
   a. 11β-Hydroxylase deficiency b. 17α-Hydroxylase deficiency
3. Cushing's syndrome or disease
4. Other :
   a. Licorice
   b. Carbenoxolone
   c. Chewer's tobacco

IV. Gain-of-function mutation of sodium channel in DCT with ECFV expansion, hypertension, K+ deficiency, and hyporeninemic-hypoaldosteronism

A. Liddle's syndrome

TREATMENT

- If Primary aldosteronism or Cushing's syndrome is present, correction of the underlying cause.
- Isotonic saline is recommended to reverse the alkalosis when ECFV contraction is present.
- Acetazolamide : Renal HCO3- loss can be accelerated

23. Patient with giant cell arteritis presents with history of headache, Jaw claudication, polymyalgia rheumatica and mononeuritis multiplex. What should be the first line treatment?

A. Prednisolone

B. Tocilizumab

C. Opioids

GIANT CELL ARTERITIS

- Giant cell arteritis (GCA) is a granulomatous arteritis mainly affecting medium-sized arteries in the head and neck.
- It is commonly associated with polymyalgia rheumatica (PMR), which causes pain and stiffness in the shoulders and hips.

CLINICAL FEATURES

- The cardinal symptom of GCA is temporal or occipital headache, which may be accompanied by scalp tenderness.
- Jaw pain develops in some patients, brought on by chewing or talking, due to ischaemia of the masseter muscles.
- Visual disturbance can occur and GCA may present with blindness in one eye due to occlusion of the posterior ciliary artery.
- On fundoscopy, the optic disc may appear pale and swollen with haemorrhages, but these changes take 24–36 hrs to develop and the fundi may initially appear normal.
- Other visual symptoms include loss of visual acuity, reduced colour perception and papillary defects.
- Rarely, transient ischaemic attacks, brainstem infarcts and hemiparesis may occur.

INVESTIGATIONS

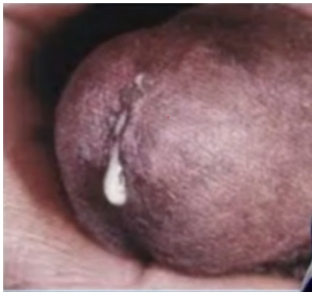
- Raised ESR and CRP.
- Normochromic, normocytic anaemia.
- Diagnosis is based on a combination of the typical clinical features, raised ESR and prompt response to steroid.
- If doubt persists, however, a temporal artery biopsy may reveal characteristic inflammatory changes.
- While a positive biopsy is helpful, a negative biopsy does not exclude the diagnosis because the lesions are focal.
- USS or arteriography may be used to help guide the biopsy.

TREATMENT

- Corticosteroids are the treatment of choice and should be commenced urgently in suspected
- GCA to prevent visual loss.
- Symptoms will completely resolve within 48–72 hrs of starting corticosteroids in virtually all patients.
- The prednisolone dose should be reduced progressively, guided by symptoms and ESR, until an acceptable dose is achieved (5–7.5 mg daily).
- Prophylaxis against osteoporosis should be given in patients with low bone mineral density.

1.A migrant worker came with complains of urethral discharge after a week of unprotected sexual intercourse and complains of burning micturition.Which among the following is the most probable causative organism ?

A. E.coli
B. Gonorrhea
C.Ureaplasma
D.Trichomoniasis



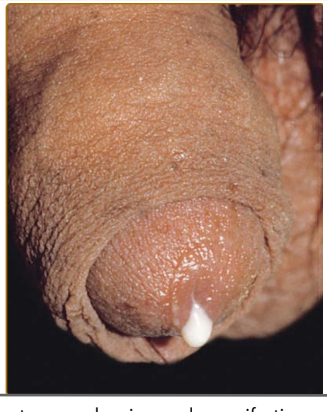
- Urethral discharge after a week of unprotected sexual intercourse and burning micturition is suggestive of Gonorrhoea.

GONORRHEA

- Bacterial infection by Neisseria gonorrhoeae, gram-negative, aerobic coccus-shaped bacterium found in pairs.

CLINICAL FEATURES

- Cutaneous Findings :
  - i. In Men :
    - Most common manifestation is Urethritis,
    - Characterized by a spontaneous, often profuse, cloudy or purulent discharge from the penile meatus
    - Mucosal membrane inflammation in the anterior urethra leads to pain or burning upon urination and meatal erythema and swelling
  - ii.In Women :
    - Endocervix is a common site of local infection.
    - Symptoms includes mucopurulent discharge, vaginal pruritus, and dysuria.
- Proctitis is a manifestation of gonococcal infection
- Non cutaneous findings :
  - i. Pharyngitis
  - ii. Pelvic Inflammatory Disease :
    - Fitz-Hugh-Curtis syndrome, involving inflammation of the liver capsule, is associated with genitourinary tract infection



Acute gonorrhea in a male manifesting as creamy purulent discharge from the urethra.

Reference :
Fitzpatrick's Dermatology 9th Ed. Pg : 3207

RISK FACTORS

- Multiple sex partners;
- Sex partners with known sexually transmitted infections (STIs);
- Engaging in unprotected oral, vaginal, or anal intercourse

ETIOPATHOGENESIS

- Bacterial attachment to columnar epithelial cells via pili or fimbriae.
- The most common sites of attachment include the mucosal cells of the male and female urogenital tracts.
- Inside the cell, the organism undergoes replication and can grow in both aerobic and anaerobic environments.
- After cellular invasion, the organism replicates and proliferates locally, inducing an inflammatory response.
- Humans are the only natural hosts.

DIAGNOSIS

- Vaginal specimens are never recommended for diagnostic purposes because the vaginal mucosa resists gonococcal invasion.
- Bacterial culture has been the "gold standard" diagnostic test.
  - Culture can be performed on modified Thayer-Martin medium.
    i. Men : secretions or urethral swabs.
    ii.Women : Endocervical and endourethral specimens.
- Nucleic acid amplification tests (NAATs) to provide more rapid diagnosis.

TREATMENT

- Prophylaxis for Gonococcal Infection in Neonates :
  - Erythromycin Or Ceftriaxone.

Treatment of Localized, Uncomplicated Gonococcal Infection of the Cervix, Rectum, Pharynx, or

- Urethra :
  i. First-Line Treatment : Ceftriaxone + Azithromycin
  ii. Alternative Therapy : Cefixime + Azithromycin

Treatment of Disseminated Gonococcal Infection :

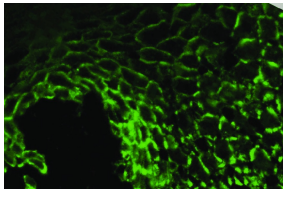
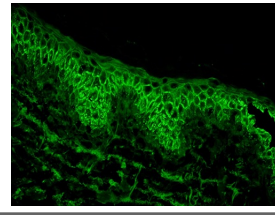
- i. Treatment of Arthritis and Arthritis-Dermatitis Syndrome : Ceftriaxone + Azithromycin
  ii. Treatment of Meningitis and Endocarditis : Ceftriaxone + Azithromycin

2. A 30yr old patient came with flaccid bullae on her skin which are easy to rupture.Biopsy revealed suprabasal split.What is the most probable diagnosis ?

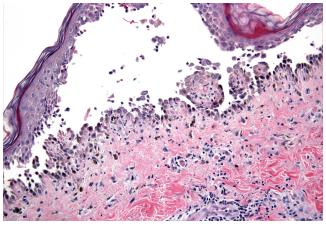
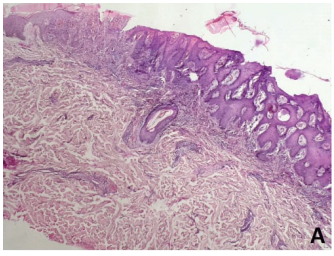
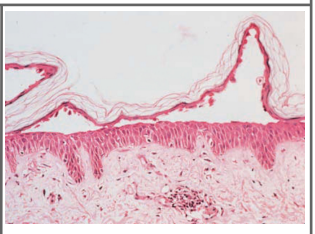
A.Pemphigus vulgaris
B.P.vegetans
C.P.foliaceous
D.Erythema multiforme

- Flaccid bullae easy to rupture and on biopsy suprabasal split is suggestive of Pemphigus vulgaris

- Pemphigus : It is a is a blistering disorder caused by autoantibodies that result in the dissolution of intercellular attachments within the epidermis and mucosal epithelium.

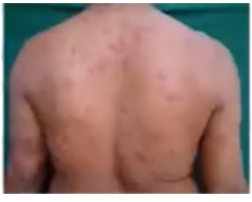
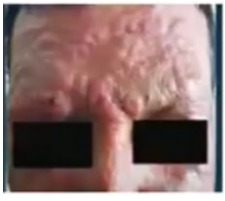
PEMPHIGUS VULGARIS	PEMPHIGUS VEGETANS	PEMPHIGUS FOLIACEUS
PATHOLOGY		
• Suprabasal blister with acantholysis "row of tombstones" appearance	• Suprabasal acantholysis, with papillomatosis of the dermal papillae and downward growth of epidermal strands into the dermis; presence of hyperkeratosis and scale-crust; eosinophilic or neutrophilic intraepidermal abscesses	• Early lesions show eosinophilic spongiosis; histopathology demonstrates acantholysis below stratum corneum; epidermis beneath the granular layer remains intact; subcorneal pustules containing neutrophils and acantholytic epidermal cells in the blister cavity
CLINICAL FINDINGS		
• Flaccid blisters, typically sparing palmoplantar surface. Large erosions are common presentations (+) Nikolsky sign	• Erosions develop excessive papillomatosis tissue and crusting; intertriginous areas, scalp, or face	• Scaly, crusted lesions on erythematous base; seborrheic distribution (face, scalp, upper trunk); small flaccid blisters are transient primary lesions
MUCOSAL INVOLVEMENT		
• Oral and nasal mucous membranes most commonly affected. Others : Esophageal, Vulvar, Cervical, Vaginal, Ocular	• Oral involvement is common	• Very rare
DIAGNOSIS		
• Direct Immunofluorescence :		
- IgG bound to surface of keratinocytes (intercellular pattern) 	- IgG on cell surface of keratinocytes	- IgG bound to surface of keratinocytes (intercellular pattern) 

Reference :
Notespaedia Dermatology IB Pg : 37
Fitzpatrick's Dermatology 9th Ed

PEMPHIGUS VULGARIS	PEMPHIGUS VEGETANS	PEMPHIGUS FOLIACEUS
• ELISA : (QUANTITATIVE)		
- Desmogleins 1, 3 (mucosal and skin involvement) - Desmoglein 3 (mucosal dominant)	- Desmoglein 3, sometimes desmoglein 1	- Desmoglein 1
• Disease Associations : Autoimmune disease, thymoma		
TREATMENT		
• Steroids • Cytotoxic immunosuppressants • Rituximab	• Steroids • Cytotoxic immunosuppressants • Acitretin • Rituximab	• Steroids • Cytotoxic immunosuppressants • Rituximab
HISTOLOGY		
 • Suprabasal acantholysis. • The row of tombstones.	 A	 • Acantholysis in the granular layer

3. A leprosy patient in his multibacillary state and on multidrug therapy presents with severe rashes on the already existing lesions.He shows neurological involvement too.What is the next best ?

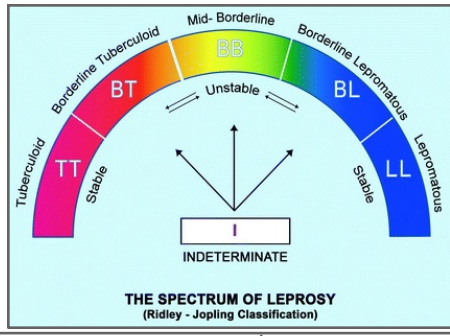
A. Stop ALT and Start steroids
B. Stop ALT and Start Thalidomide
C. Continue ALT and Start Steroids
D. Continue ALT and Start Thalidomide

- Patient on multidrug therapy and in multibacillary state presenting with severe rashes and neurological involvement is suggestive of Type 1 Reaction
  So in this case continue ALT and Start Steroids.

LEPROSY

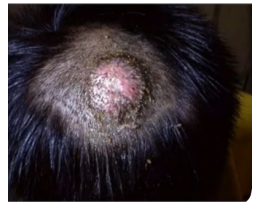
- Leprosy is a chronic granulomatous infection caused by Mycobacterium leprae, that infects mucous cutaneous tissues and peripheral nerves, leading to loss of sensation on the skin—with or without dermatologic lesions—and the development of incapacities during the progression of the disease.



	REVERSAL REACTION (TYPE I)	ERYTHEMA NODOSUM LEPROSUM (TYPE II)
Predominant leprosy subtype	Borderline-tuberculoid Borderline-borderline Borderline lepromatous Lepromatous leprosy	Lepromatous leprosy
Signs and symptoms	Acute worsening of skin lesions Acute worsening of nerve function impairment No apparent systemic symptoms	Erythema nodosum Erythema polymorphous Severe cutaneous necrotizing vasculitis Fever, edema, malaise
Extracutaneous involvement	Neuritis	Neuritis,Panniculitis Glomerulonephritis,Arthralgia Epididymitis,Orchitis Eye inflammation,Osteitis Lymphadenitis
Treatment	Prednisone	Thalidomide and Prednisone Or Pentoxifylline

5. A10yr old male child came to OPD with C/O mild painful swelling in the scalp for past 3 months.She has a dog as pet.What is the most probable diagnosis ?

A. Folliculitis
B. Abscess
C. Kerion
D. Epidermoid cyst



- The given shows a inflamed small boggy swelling in scalp this reaction is usually caused by one of the zoophilic species, typically T. verrucosum or T. mentagrophytes, occasionally a geophilic organism will be isolated and most probable diagnosis is Kerion.

Reference :
Fitzpatrick's Dermatology 9th Ed. Pg : 2914
Notespaedia Dermatology IB Pg : 99

KERION

- It is a painful, inflammatory mass in which such hairs as remain are loose.

ETIOLOGY

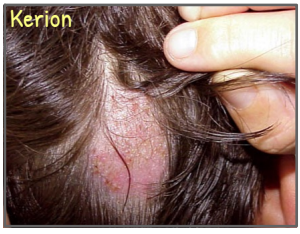
- Caused by one of the zoophilic species, typically T. verrucosum or T. mentagrophytes, occasionally a geophilic organism will be isolated.

CLINCIAL FEATURES

- Follicles may be seen discharging pus, there may be sinus formation.
- Kerion in a patient with Trichophyton tonsurans infection of the scalp.
- Rarely mycetoma-like grains may be found.
- Thick crusting with matting of adjacent hairs is common.
- The area affected may be limited and occasionally a large confluent lesion may involve much of scalp.
- Lymphadenopathy is frequent.

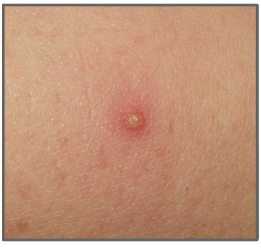
TREATMENT

- First Line :
  - Terbinafine
  - Itraconazole
  - Griseofulvin
- Second line :
  - Itraconazole



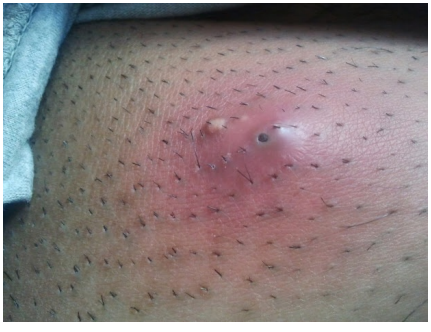
FOLLICULITIS

- Is a pyoderma that begins within the hair follicle, and is classified according to the depth of invasion (superficial and deep), and microbial etiology.
- Superficial folliculitis also has been termed follicular or Bockhart impetigo.
- A small, fragile, dome-shaped pustule occurs at the infundibulum (ostium or opening) of a hair follicle, often on the scalps of children and in the beard area, axillae, extremities, and buttocks of adults.
- Isolated staphylococcal folliculitis is particularly common on the buttocks of adults.



ABSCESSES

- S. aureus dermal and subcutaneous abscesses commonly occur in folliculocentric infections
- Abscesses can also occur at sites of trauma, foreign bodies, burns, or sites of insertion of i.v catheters.
- The initial lesion is an erythematous nodule. If untreated, the lesion often enlarges, with the formation of a pus- filled cavity.

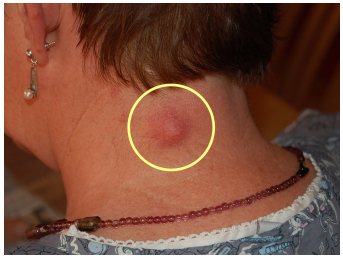


EPIDERMOID CYST

- Follicular cyst-infundibular type, keratin cyst, epidermal cyst, epidermal inclusion cyst, or epithelial cyst are most commonly the result of plugged pilosebaceous units.
- Cysts are lined by epithelium resembling the infundibulum of the hair follicle and express the same cytokeratin profile
- Rarely, BCC, SCC, epithelioid carcinoma, and other malignancies have been reported to occur in conjunction with these cysts.
- Predisposing Genetic disorders :
  i. Gorlin syndrome (nevoid BCC syndrome),
  ii. Pachyonychia congenita type 2 (Jackson-Lawler type),
  iii. Gardner syndrome

CLINICAL FEATURES

- Dermal or subcutaneous mobile nodules with a central punctum.
- The punctum, when present, represents the plugged pilosebaceous unit from which foul- smelling cheesy debris may be expressed.
- Most commonly found on the upper chest, upper back, neck, or head.



TREATMENT

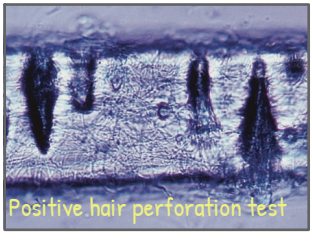
- Complete excision or destruction of the cyst lining is the definitive treatment.
- If a cyst becomes inflamed, painful, or purulent, incision and drainage should be performed, and infection must be considered.
- For small, inflamed, symptomatic lesions, intralesional steroids may be considered.

6. Hair Perforation Test is positive in ?

A. Trichophyton mentagrophytes
B. Microsporangia audouinii
C. Epidermophyton floccosum
D. Microsporum gypseum

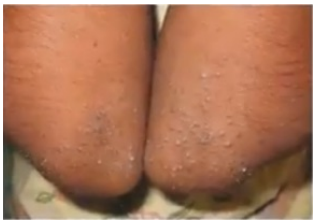
HAIR PERFORATION TEST

- a.k.a in vitro hair perforation test.
- It is a laboratory test used to help distinguish the isolates of dermatophytes, such as Trichophyton mentagrophytes and its variants.
- The test is performed by placing an organism into a Petri dish containing water, yeast extract, and hair.



Reference :
https://mycology.adelaide.edu.au/laboratory/hair/

7. A girl child presented with rough surfaced lesions over elbows,knee(extensor surface), dry eyes and night blindness.What is the dermatological finding shown in the pic?



A. Eczema
B. Icthyosis
C. Keratosis Pilaris Rubro
D. Phrynoderma

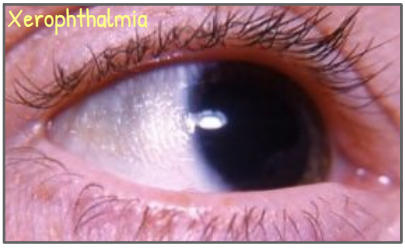
- Based on the given and skin manifestation the most probable diagnosis is Vitamin A deficiency.
- Given image is that of Phrynoderma.

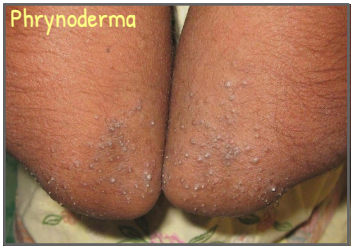
VITAMIN A DEFICIENCY

- Can result in both cutaneous and ocular complications.
- It is, in fact, the most common cause of preventable blindness in children, according to the World Health Organization.
- VAD also is associated with defects in immune regulation.

CLINICAL MANIFESTAIONS

- Cutaneous, Mucocutaneous :
  - Xerosis
  - Skin fissuring (dermatomalacia)
  - Phrynoderma
  - Mucosa
  - Xerostomia
  - Hypotonia
  - Hypogeusia
- Ocular :
  - Impaired dark adaption
  - Xerophthalmia
  - Corneal xerosis, ulceration, keratomalacia
  - Corneal perforation, blindness





DIAGNOSIS

- Serum vitamin A levels/plasma retinol : <20 mcg/dL (<0.70 µmol/L) deficiency
- Relative dose-response test :
  - Serum retinoic acid in serum after oral retinoyl administration glucuronide
  - Serum retinoic acid increase of at least 20% indicates low liver vitamin A reserves

Reference :
Notespaedia Dermatology IB Pg : 90
Fitzpatrick's Dermatology 9th Ed

TREATMENT

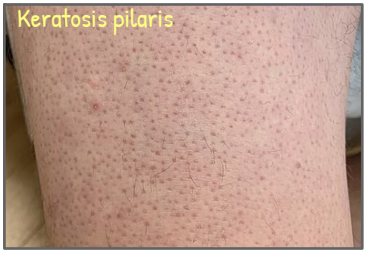
- The recommended daily allowance (RDA) of vitamin A is between 700 mcg (adult females) to 900 mcg (adult males), with younger individuals requiring a lower intake of vitamin A.
- Recommended treatment of VAD is 600 to 3000 mcg of oral vitamin A daily until symptoms resolve and serum levels normalize.
- Repletion dosages depend on the age of the patient, and require conversion to the appropriate food-related equivalent.

KERATOSIS PILARIS

- Keratosis pilaris rubra (KPR, also called keratosis follicularis rubra) is a variant of KP in which erythema is markedly noticeable, extending beyond the peri-follicular skin.
- Findings are usually limited to the cheeks,forehead, and neck.

CLINICAL FEATURES

- Primary lesions of KP are small (typically 1-mm), keratotic, follicular papules with varying degrees of perifollicular erythema.
- Affected areas include the lateral cheeks, extensor aspects of the upper arms ,thighs, and buttocks, and it rarely may be more extensive, extending to the distal limbs and the trunk.

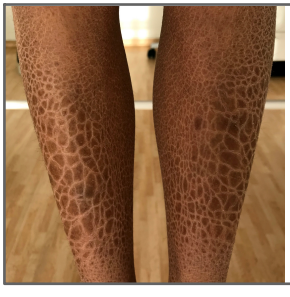


DIAGNOSIS

- It's a clinical diagnosis based on lesional morphology and distribution.
- Dermoscopic findings of thin, short hair shafts that are coiled or twisted within the follicular ostia are supportive.

MANAGEMENT

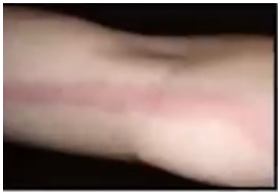
- Tends to improve by adolescence or early adulthood.
- Keratolytic preparations containing urea, lactic acid, or salicylic acid may soften and smooth KP.
- Topical retinoids may also be tried.
- Short courses of low-potency topical corticosteroids
- Treatment with vascular- or pigment-specific lasers modestly improves the erythema or hyperpigmentation associated with KP.



- Ichthyosis is a condition that causes widespread and persistent thick, dry, "fish-scale" skin.

7. A patient presents with pain and swelling in finger and linear wheals can be seen in right upper limb. Which among the following is the suitable antibiotic for treatment ?

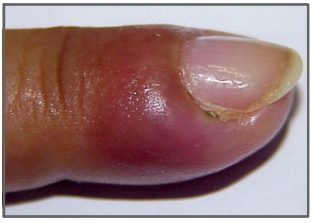
A. Amoxiclav
B. Amikacin
C. Norfloxacin
D. Metronidazole



- Pain and swelling in finger most probable due to paronychia and there is extensive linear wheals in right limb which is linear lymphangitis like lesion.
- Paronychia is of 2 types :
  i. Acute
  ii. Chronic
- For any abscess most commonly used drug is Amoxicillin combination or Cephalosporins.

PARONYCHIA

- Is a skin infection around the fingernails or toenails.
- It usually affects the skin at the base (cuticle) or up the sides of the nail.
- Acute paronychia : comes on suddenly and may not last long; it usually occurs on fingers.
- Chronic paronychia :
  - Lasts longer and may occur on your fingers or toes.
  - It either doesn't get better or keeps coming back.



CLINICAL FEATURES

- Pain, swelling and redness around the base or the sides of the nail.
- Acute paronychia can cause pus-filled pockets (abscesses) to form.
- Chronic paronychia may cause the cuticle to break down.

TREATMENT

- Treatment of choice depends on the extent of the infection.
- Anti-staphylococcal penicillin or first generation cephalosporins is generally effective.
- Clindamycin and Amoxicillin-clavulanate are also appropriate

Acute lymphangitis of forearm due to Staphylococcus aureus. There is a tender linear streak extending proximally from a small area of cellulitis on the volar wrist.

1. While therapy session a therapist developed unconscious and conscious feelings towards the patient . what is it called?

A. Transference

B. Free association

C. Abreaction

D. Countertransference

Countertransference is defined as redirection of a psychotherapist's feelings toward a client or, more generally, as a therapist's emotional entanglement with a client.

COUNTERTRANSFERENCE AND TRANSFERENCE

TRANSFERENCE

- Transference is redirection of a client's feelings from a significant person to a therapist

There are three stages in dealing and using transference in social casework. these stages are:

1. Understanding the Transference
2. Utilizing the transference.
3. Interpreting the transference.

- Understanding of the transference is essential for the worker as it helps to understand the behaviour of the client and to recognize its significance in his development process. It also explains the present unconscious needs of the client.
- Utilization of the transference depends on the understanding of the social case worker of the phenomena. It explains many cures or treatments of emotional disturbance by life situations and by fortune relationships with other problems
- Interpretation of the transference, that is, confronting the individual with the awareness that his behaviour is the repetition of a specific unconscious infantile is definitely part of psychoanalytical therapy and requires preparation of the individual by the careful analysis of his unconscious defense.

COUNTER-TRANSFERENCE

- Counter-transference is defined as redirection of a therapist's feelings toward a client
- It's a therapist's emotional entanglement with a client
- It is a two way process.
- Social case worker has also unconscious tendency to transfer out the client.
- As in the case of transference, these counter transference feelings, both positive an negative, are unconscious but operate with force.
- Therefore, it is the job of case worker to recognize his feelings and must control them.

2. Which is included in form of thought disorder ?

A. Derailment

B. Obsession

C. Somatic delusion

D. Thought insertion

Derailment is a form of thought disorder
(Jumping into new topic)

HEALTHY THINKING

- CONSTANCY- Persistence of a completed thought whether simple or complex.
- ORGANISATION- Contents are related but do not blend with each other but organised
- CONTINUITY- Thoughts or ideas are arranged in order

DISORDERS OF THOUGHT

STREAM	CONTENT
Flow of ideas 1. Disorders of tempo- i. Flight of ideas ii. Inhibition of thinking 2. Disorders of Continuing- i. Perseveration ii. Thought blocking	1. Overvalued idea 2. Magical thinking 3. Superstition 4. Delusion - Fixed, firm belief in something that is not a fact
POSSESION	**FORM**
1. Thought block 2. Obsession— Repeated intrusive thoughts eg:- OCD 3. Thought alienation- Controlled by someone eg:- Schizophrenia i. Thought insertion ii. Thought withdrawal iii. Thought broadcasting	The way thoughts are put in. Characteristic of Schizophrenia i. Loosening of association ii. Verbigeration- no connection b/w words iii. Neologism - coining new word iv. Tangentiality- loss of connection v. Circumstantiality- unnecessary details vi. Derailment- jumping to new topic

3. 16 year old female patient presented with overfamiliarity, flight of ideas, elevated mood, increased sexual desires, pseudo hallucinations. What is the diagnosis?

A. Mania

B. Schizomania

C. Hypomania

D. Cyclothymia

MANIA

- Also known as manic syndrome, is a mental and behavioral disorder defined as a state of abnormally elevated arousal, affect, and energy level, or "a state of heightened overall activation with enhanced affective expression together with lability of affect.
- During a manic episode, an individual will experience rapidly changing emotions and moods, highly influenced by surrounding stimuli.
- Although mania is often conceived as a "mirror image" to depression, the heightened mood can be either euphoric or dysphoric.
- As the mania intensifies, irritability can be more pronounced and result in anxiety or anger.
- The symptoms of mania include :
  - elevated mood (either euphoric or irritable),
  - flight of ideas
  - pressure of speech,
  - increased energy,
  - decreased need and desire for sleep,
  - hyperactivity.
- To be classified as a manic episode, while the disturbed mood and an increase in goal-directed activity or energy is present, at least three (or four, if only irritability is present) of the following must have been consistently present:
  1. Inflated self-esteem or grandiosity.
  2. Decreased need for sleep (e.g., feels rested after 3 hours of sleep).
  3. More talkative than usual, or acts pressured to keep talking.
  4. Flights of ideas or subjective experience that thoughts are racing.
  5. Increase in goal-directed activity, or psychomotor acceleration.
  6. Distractibility (too easily drawn to unimportant or irrelevant external stimuli).
  7. Excessive involvement in activities with a high likelihood of painful consequences. (e.g., extravagant shopping, improbable commercial schemes, hypersexuality).[18]

TREATMENT

The acute treatment of a manic episode of bipolar disorder involves the utilization of either

Mood stabilizer

- Carbamazepine,
- lithium,
- valproate,
- lamotrigine

Atypical antipsychotic

- olanzapine,
- risperidone,
- quetiapine,
- aripiprazole

When the manic behaviours have gone, long-term treatment then focuses on prophylactic treatment to try to stabilize the patient's mood, typically through a combination of pharmacotherapy and psychotherapy.

4. A 53 year male, who is a chronic alcoholic, tried to stop using alcohol after several requests by his family members. He started feeling uneasy and on day 3, he was brought to the hospital with disorientation, irritability, paranoid delusions , visual hallucinations and altered sensorium. Which is your probable diagnosis?

A. Wernicke's encephalopathy

B. Delirium tremens

C. Korsakoff psychosis

D. Anxiety disorder

DELIRIUM TREMENS

- Delirium tremens (DTs) is a rapid onset of confusion usually caused by withdrawal from alcohol.
- When it occurs, it is often three days into the withdrawal symptoms and lasts for two to three days.
- Physical effects may include shaking, shivering, irregular heart rate, and sweating.
- People may also hallucinate.
- Occasionally, a very high body temperature or seizures may result in death.

SYMPTOMS

- nightmares,
- agitation,
- global confusion,
- disorientation,
- visual and auditory hallucinations,
- tactile hallucinations,
- fever,
- high blood pressure,
- heavy sweating,
- other signs of autonomic hyperactivity (fast heart rate and high blood pressure).

These symptoms may appear suddenly but typically develop two to three days after the stopping of heavy drinking, being worst on the fourth or fifth day. These symptoms are characteristically worse at night

TREATMENT

- Benzodiazepines, such as lorazepam (Ativan), diazepam (Valium), or chlordiazepoxide (Librium), which can effectively manage the majority of alcohol withdrawal symptoms, including delirium tremens, and reduce the risk of seizures. These are the most commonly used medications, and large doses may be required.
- Barbiturates, like phenobarbital, although these tend to be added on when benzodiazepines alone haven't been sufficient in managing symptoms.
- Antipsychotics, such as haloperidol (Haldol), may be used in low doses to help reduce problematic behaviors such as agitation, manage psychotic symptoms like hallucinations, and help you think more clearly, although this type of medication has been associated with negative side effects.

WERNICKE ENCEPHALOPATHY (WE)

- Acute neurological condition characterized by a clinical triad of ophthalmoparesis with nystagmus, ataxia, and confusion.
- This is a life-threatening illness caused by thiamine deficiency, which primarily affects the peripheral and central nervous systems.
- Characterized by three main clinical symptoms: confusion, the inability to coordinate voluntary movement (ataxia) and eye (ocular) abnormalities.

KORSAKOFF PSYCHOSIS

- A late complication of persistent Wernicke encephalopathy and results in memory deficits, confusion, and behavioral changes.
- Korsakoff psychosis occurs in 80% of untreated patients with Wernicke encephalopathy.
- Korsakoff syndrome is often accompanied by Wernicke encephalopathy; this combination is called Wernicke-Korsakoff syndrome.
- Symptoms
  1. anterograde amnesia, memory loss for events after the onset of the syndrome
  2. retrograde amnesia, memory loss extends back for some time before the onset of the syndrome
  3. amnesia of fixation, also known as fixation amnesia (loss of immediate memory, a person being unable to remember events of the past few minutes)
  4. confabulation, that is, invented memories which are then taken as true, due to gaps in memory, with such gaps sometimes associated with blackouts
  5. minimal content in conversation
  6. lack of insight
  7. apathy – interest in things is quickly lost, and there is an indifference to change

5. A teacher taught steps of hand washing. Students learned and repeated at home.
What domain of learning does it fall under?

A. Psychomotor

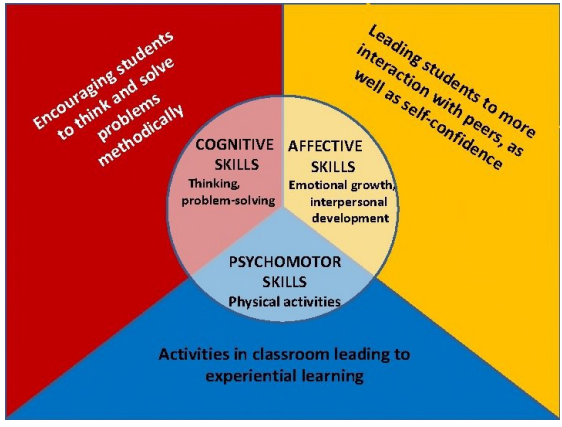
B. Cognitive

C. Cognitiveandaffective

D. Affective

- Learning is not an event. It is a process.
- It is the continual growth and change in the brain's architecture that results from the many ways we take in information, process it, connect it, catalogue it, and use it (and sometimes get rid of it).
- Learning can generally be categorized into three domains: cognitive, affective, and psychomotor.
- Within each domain are multiple levels of learning that progress from more basic, surface-level learning to more complex, deeper-level learning.

Cognitive	Affective	Psychomotor
knowledge	**attitude**	**skills**
1. Recall data	1. Receive (awareness)	1. Imitation (copy)
2. Understand	2. Respond (react)	2. Manipulation (follow instructions)
3. Apply (use)	3. Value (understand and act)	3. Develop Precision
4. Analyse (structure/elements)	4. Organise personal value system	4. Articulation (combine, integrate related skills)
5. Synthesize (create/build)	5. Internalize value system (adopt behaviour)	5. Naturalization (automate, become expert)
6. Evaluate (assess, judge in relational terms)		



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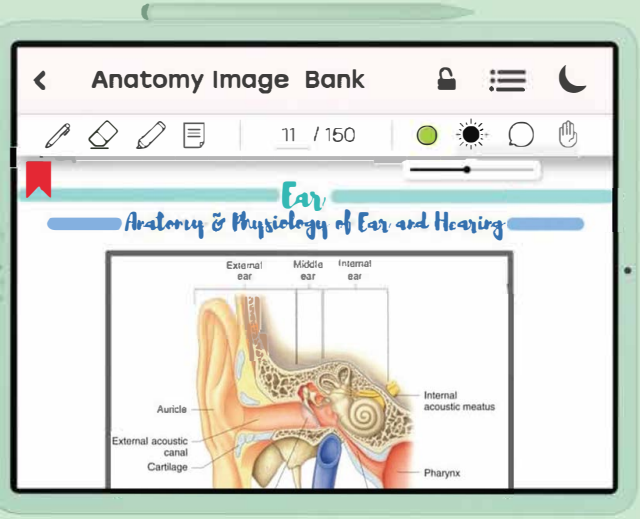

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