**PROBLEM BASED LEARNING QUESTIONS ON CVS AND RESPIRATORY SYSTEM:-**

**CASE1:-**  A 15 year old boy was slim and fit when he collapsed after completing a cross-country run at his school in morning. He was rushed to the hospital emergency ward and pronounced dead in the afternoon. { Professional history-Cricket player}

Family history:- Father has hypercholesterolemia.

On autopsy, patient had lumps over knuckles, Xanthelasma present, and coronary arteries were completely blocked having a pin hole opening.

LAB DATA:-

1.Total cholesterol: 290mg/dL

2.Repeat total cholesterol: 296mg/dL

3.Triglycerides: 108mg/dL

4.HDL cholesterol: 55mg/dL

5.LDL cholesterol: 209mg/dL

Que.1. What is the probable diagnosis?

Ans.1. This is a familial case of hypercholesterolemia.

Que.2. What is the biochemical defect?

Ans.2. It is may be due to the defective LDL receptors.

Que.3. What are the risk factors for atherosclerotic heart disease?

Ans.3. The risk factors for atherosclerotic heart disease are:-

1. Hypercholestrolemia

b.Smoking

c.Hypertension

d.Diabetes mellitus

e.Sedentary life style

f.Family history of CHD

g.Age: males->45 females->55

Que.4. Why should we do screening of children with family history of hypercholesterolemia?

Ans.4. We have to detect hypercholesterolemia so as to prevent premature atherosclerosis.

Que.5. What is the acceptable level of plasma total cholesterol?

Ans.5. The normal range should be 150-200mg/dL.

**CASE 2:-** A 32 yrs old sales man was admitted to an ICU with a history of left sided chest pain radiating to the left shoulder and to the medial side of the left upper limb, tightness in chest, nausea, vomiting and difficulty in breathing and severe sweating.

O/E:- ECG reveals ST segment elevation and T wave inversion.

The blood analysis shows:-

* Serum total CK [CPK] : 780.0 U/L
* CK-MB : 85.0 U/L
* Serum LDH: 920.0 U/L
* Serum AST : 72.0 U/L
* Serum total cholesterol : 415.0 mg/dL
* Serum HDL cholesterol : 25.0 mg/dL
* Serum triglycerides : 280.0 mg/Dl
* Serum LDL cholesterol : 334.0 mg/dL

Que.Comment on the laboratory report and mention the probable diagnosis.

Ans. Serum CPK and LDH levels are very much increased. Serum AST is increased and serum ALT is within normal limits. Serum total cholesterol and triglycerides levels are high whereas serum HDL cholesterol is low. LDL cholesterol is considered as bad cholesterol and HDL cholesterol is considered as good cholesterol. The above signs and symptoms, increased serum CPK, LDH and AST are strongly suggestive of **‘MYOCARDIAL INFARCTION’.**

**CASE 3:-** A 60-year old male known COPD, admitted to the hospital with a history of smoking 40 packs per day for 40 years with chronic dyspnoea, cough and sputum since 4 years.

ABG ANALYSIS:-

pH – 7.29

pCO2 – 60mmHg

HCO3- - 32mmol/L

Que. Interpret the lab data and write the diagnosis.

Ans. INTERPRETATION:-

Step 1: Look at pH which is decreased in this case

Step 2: Is it respiratory or metabolic; since pCO2 is predominantly affected, it is a case of respiratory acidosis.

Step 3: Is it acute or chronic; it is a chronic case.

[For every 10mmHg rise in pCO2 , pH decreases by 0.03 units and HCO3- increases by 4 mmol/L in chronic cases. In this case, pCO2 is increased from 40-60 {change of 20mmHg} and pH decreased from 7.4 to 7.34 {change of 0.06} and HCO3- increased from 24 to 32 {change by 4x2=8mmol/L} ]

Therefore, it is a case of **‘CHRONIC COMPENSATED RESPIRATORY ACIDOSIS’**

**CASE 4:-** A 40 year old known asthmatic case was reported.

ABG:- pH- 7.48

pCO2 – 30 mmHg

HCO3- - 19 mmol/L

Que. Comment on the lab data and write the diagnosis.

Ans. INTERPRETATION:-

Step 1: The pH value is increased. [alkalosis]

Step 2: As CO2 is predominantly affected, it is a case of respiratory alkalosis.

Step 3: It is chronic respiratory alkalosis.

[For every 10mmHg decrease in pCO2, pH increases by 0.03 pH units and HCO3- decreases by 5 mmol/L in chronic cases. In this case, as pCO2 decreased from 40 to 30 {change of 10mmHg} pH increased from 7.45 to 7.48 {change of 0.03 units} and HCO3- decreased from 24 to 19 {change by 5 mmol/L} ]

Therefore, it is a case of **‘CHRONIC COMPENSATED RESPIRATORY ALKALOSIS’.**

**CASE 5:-** A 20 year old patient presented with the diazepam over dose.

ABG results:- pH – 7.24

pCO2  - 60 mmHg

HCO3- - 26 mmol/L

Que. Comment on the lab data and write the diagnosis.

Ans. INTERPRETATION:-

Step 1 : There is a decrease in the pH value indicating acidosis.

Step 2 : It is respiratory acidosis

Step 3 : It is an acute case.

[For every 10 mmHg increase in pCO2, pH decreases by 0.08 pH unit and HCO3- increases by 6 mmol/L in acute cases]. In this case, as pCO2 has increased from 40 to 60 {change of 20mmHg} pH decreased from 7.4 to 7.24 {change of 0.08x2 =0.16 pH units} and HCO3- has slightly increased from 24 to 26 {change by 2mmol/L}. Therefore, it is a case of **‘ACUTE RESPIRATORY ACIDOSIS’.**

**CASE NO 6:**  A teenager came to the emergency and trauma department at AIIMS hospital. He had suffered a lacerated wound of the left lower leg in a road traffic accident that had cut the tibial artery. About an estimated 1.6 liters of blood i.e. 30 % of blood volume had been lost. An attempt to control the bleeding by pressure was made. He had received 2 liters of dextran in the ambulance.On Examination: Pulse 130/min BP 84/61 mm Hg, Resp. Rate 24/min

1. **What is the diagnosis?**

Hemorrhagic shock

1. **Define shock. Explain the compensatory mechanisms in shock.**

Ans. Shock is a syndrome characterized by low cardiac output which is inadequate to maintain normal tissue perfusion.

The types of shock are Hypovolemic, Hemorrhagic, Traumatic, surgical and dehydration shock

**Rapid Compensatory mechanisms**

Haemorrhage, decreases blood volume, venous return, cardiac output, and blood pressure. Decrease in blood pressure decreases baroreceptor activity and increases chemoreceptor. This reflexly increases sympathetic discharge.

Tachycardia (**rapid thready pulse)**, generalised vasoconstriction, increased secretion and release of catecholamines from the adrenal medulla, rapid breathing (**rapid shallow breathing)**

Renal ischemia produces release of angiotensin II that stimulates **thirst,** and decrease in urine output (**acute renal**

**failure)**

These mechanisms retain water and try to bring the ECF volume back to normal.

**Long term Compensatory Mechanisms**

Restoration of plasma volume within 12‐72 hours by retention of water by kidneys and capillary shift mechanisms

Restoration of plasma proteins over a period of 3‐4 days due to increased protein synthesis in liver

Restoration of RBC mass in 4‐8 weeks due to increase in erythropoietin secretion by the kidneys.

Restoration of B.P over several months via kidneys by long term regulatory mechanisms.

1. What is the course of treatment?

About 2 Litres of blood transfusion would restore vitals to normal. Emergency surgery will need to be done to repair the tibial artery.

**Case No 7** A 56 year old patient who has smoked three packs /day of cigarettes 7 years ago presents to the physician complaining of sharp cramps and pains especially while exercising. It is relieved by rest.

On examination : The ankle-brachial index is 0.7(normal: 0.95 to 1.20). Duplex ultrasound imaging shows an increased lumen blood flow velocity in both calves, indicating areas of stenosis. Toes on both feet show signs of cyanosis. The lower legs appear shiny and feel cool to the touch. Elevation of the calf causes the skin color to become pale.

Question 1) What is your diagnosis?

Intermittent claudication

Buergers disease is a small vessel disease that affects the arteries due to habit of smoking. The patient suffers from pain while walking, gangrene and amputation of limbs.

Intermittent claudication is a clinical symptom of pain in calf muscles that comes and goes. Thrombophelbitis is inflammation of the veins caused by thrombus. It is due to venous occlusion and fatigue sets in early.

Q1. What is fatigue?

Ans1. Fatigue refers to a temporary reduction of the working capacity of a cell, a organ or an organism as a whole resulting from prolonged exertion. It is a reversible phenomenon and passes off after rest.

Q2. What is the site of fatigue?

Ans 2. There possible sites of fatigue are the nerve fibers, the neuromuscular junction and the muscle fiber.

In man the first site of fatigue is the “synapse in the brain”

In amphibians, the neuromuscular junction seems to be the first site of fatigue. This can be shown indirectly as follows-

To prove the muscle is not the site of fatigue, the muscle is stimulated directly after fatigue sets in. It is seen that the muscle contracts vigorously.

To prove the nerve is not the site of fatigue, the two nerve muscle preparations are crossed and a block of ice kept over one of it, it prevents the transmission of nerve impulse.

Q3. How does motivation improve physical performance?

Ans 3. Motivation helps improve physical performance. Motivation can be described as an individual inner will and desire to succeed. Motivation can influence decisions, learning and performance in sport. Motivation can be inner driven or an external stimulus e.g cheer leaders or home crowd support. Cerebral cortex plays an important role as thoughts become things.

They are examples of sleepless saints who stay in the physiological state of suspended animation their body battery being recharged by the cosmic energy. Their joy of meditation is equated to a cocktail of the joy of millions of sleep. They do not feel fatigue. The energy in a 2 gram of tissue can light the state of California for two days. There is 1000 years of energy in the spine. The moment we say we are fatigued the mind gives up. The power of mind over matter is gaining importance as wisdom is the greatest cleanser.

This can be proved by giving rest to the subject for 15 minutes and asking him to perform the procedure again, telling him he can do better.

What are the other factors affecting fatigue?

The other factors effecting fatigue are weight to be lifted, frequency of contractions, motivation, training, temperature of environment, blood supply to contracting muscles

Q4. Why fatigue occurs earlier in arterial occlusion?

Ans 4. On raising the blood pressure to about 160-170 mmHg arterial blood flow to the arm is stopped. Fatigue sets in much earlier now because there is not only an accumulation of waste products in exercising muscle but also decreased oxygen supply and other nutrients.

Q5. Why does fatigue sets in earlier in venous occlusion as compared to normal?

Ans 5. Fatigue sets in when the blood pressure cuff is raised to 40 mm Hg as this causes accumulation of waste products in the exercising muscles.

Q6. What is the treatment of our patient?

Ans 6. Angioplasty of the lesions need to be performed, resulting in improved leg blood flow and restoring the ability to exercise.

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**PROBLEM BASED LEARNING IN HAEMATOLOGY**

A 5 year old boy has complaints of excessive bruises around pressure points of knees and elbows. The parents noted that he bleeds easily when the skin surface is scratched. His maternal grandmother has a history of hemophilia

On Examination: Platelets 2,50,000 /mm3 (normal 1,50,000-4,00,000/ mm3). Bleeding time: 6 min (normal 2-8min). Factor VIII levels : low , vWF levels: normal

1. **What is Hemophilia and purpura?** Hemophiliais congenital deficiency of

Factor VIII: Hemophilia A

Factor IX: Hemophilia B

Hemophilia A is inherited as sex linked. The subject has a normal bleeding time but a prolonged clotting time. The patient is treated by Factor VIII rich preparation prepared from plasma during hemorrhage. The patient needs to be careful when travelling by two wheelers. Role of factor VIII is shown below in coagulation pathway.

1. **Intrinsic**

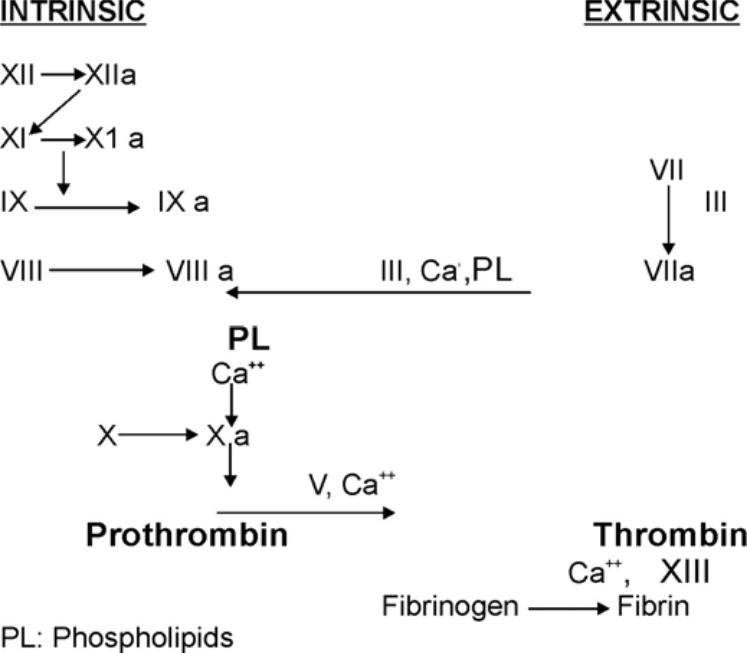
XII to XII a occurs when collagen is exposed in blood vessels (in vivo)

Or in vitro when bood comes in contact with negatively charged blood.

1. **Extrinsic**

Injury to blood vessel or tissue causes activation of

thromboplastin or factor III



**Treatment:** Desmopressin triggers factor VIII released from endothelium. Factor VIII rich plasma /cryoprecipitate or purified recombinant factor VIII may be given.

**Purpura:** It is characterized by spontaneous superficial bleedingand accumulation of blood below the skin and mucus membrane. This is due to deficiency of platelet (thrombocytopenia or Idiopathic thrombocytopenic purpura or ITP and Thrombotic Thrombocytopenic purpura or TTP), lazy platelets or dysfunctional platelets (thrombasthenia). Purpura may be palpable which is vasculative purpura e.g. Henoch Scholein purpura.

**Problem based learning in Gastro Intestinal Tract**

1. A computer call center operator visits the medical officer complaining of epigastric pain that begins after eating since the last few months. He has a history of working night shifts and smokes one pack of cigarettes for the last 10years.

On examination: Endoscopy : ulcer of 2 cm detected, Biopsy of gastric mucosa is positive for *Helicobacter pylori* infection

1. What is the diagnosis?

Peptic Ulcer

**2.Describe how acid is secreted by the cells of the gastric mucosa and explain the mechanism of regulation of gastric juice. Write composition of gastric juice. What are the functions of HCL.**

**Ans. Mechanism of acid secretion**

In the apical cells of gastric parietal cell there exist H+ K + ATPase that pumps H+ out of the parietal cells in exchange for K

H+ is formed from H2CO3 that is formed by hydration of CO2, catalyzed by carbonic anhydrase. HCO3‐ formed by the dissociation of H2CO3 is extruded out of the basolateral membrane of parietal cells in exchange for Cl‐.

**Regulation of acid secretion**

Acid secretion is stimulated by Histamine via H2 receptors, by acetylcholine via M3 muscarinic receptors, and probably by gastrin via gastrin receptors, in the membrane of parietal cells. Prostaglandins E2 inhibit acid secretion by activating Gi. This explains the increase incidence of ulcers in patients taking aspirin (inhibits PGE).

**Gastric juice composition**

Cations : Na, K, Mg, H pH =1

Anions: Cl, SO4, HPO4

Pepsin

Lipase

Mucus

Intrinsic factors

**Functions of HCl**

HCl kills many gastric bacteria, aids protein digestion, provides the necessary pH to provide pepsin for necessary protein digestion, and stimulates the flow of bile and pancreatic juice.

1. A 43-year-old man comes to the clinic complaining of abdominal cramps.

The patient has a 15-year history of alcohol abuse. He is complaining over the past 6 months of passing pale and malodorous stools that are difficult to flush.

Q)What is your diagnosis?

Ans. Malabsorption

Q) What is malabsorption syndrome?

When 50% of small intestine is resected or bypasses the absorption of vitamins and nutrients are so compromised that it is difficult to prevent malnutrition and wasting. This is called malabsorption.

The defective intestinal function in tropical sprue is a apparently due to folic acid deficiency, due to gliaden induced immune response present in wheat, rye, barley, oats , rye or due to alcohol ingestion as in the above case. This produces villi deficiency. These foods when eliminated from diet and patient treated by tetracycline the villi restore to normal.

**Causes**

Abnormalities in the digestion in intestinal lumen Glucose‐galactose malabsorption.

Inadequate lipolysis e.g. due to pancreatic insufficiency or excess secretion of gastric acid. Decreased conjugated bile salts e.g. due to ileal resection or overgrowth.

Abnormalities in mucosal cell transport. Nonspecific due to tropical sprue, celiac disease etc. Specific due to various disacharidase deficiencies.

Abnormalities of fat transport in intestinal lymphatics

**Symptoms**

Wasting, Malnutrition , Hypocalcemia, Arthritis, Hyperuricemia, fatty infiltration of liver causing cirrhosis. Diarrhea is also caused.

Lowers plasma cholesterol (gastrectomy done in morbid obesity) **Steatorrhea:** the amount of fat and protein in the stoolsincreased. The stool becomes bulky, pale, foul smelling and greasy

Vitamins that are fat soluble are lost in the stool, producing vitamin deficiencies.

**Problem Based Learning in Respiratory Physiology**

A 60 year old man makes a pilgrimage to Kedarnath. He complains of dyspnea, headache, dizziness and insomnia. He was short of breath while sitting down also. On Examination Fingernails show slight cyanosis. Pulse oximeter shows 68% saturation.

Q) What is your diagnosis?

Ans) Acute mountain sickness

Q) What is **HAPO:** High altitude pulmonary edema Pulmonaryvasoconstriction in response to hypoxia is associated by hypertension and causes edema. The person suffers from dyspnea. He needs rest, calcium channel blockers, oxygen and return to lower altitude. This may occur even in acclimatised individuals who exercise and is usually seen in unacclimatised individuals. It is also associated with nausea, vomiting, headache and cerebral edema.

**Explain the physiological basis of the pilgrims symptoms due to High Altitude**

Hyperventilation occurs due to hypoxic hypoxia, which stimulates the peripheral chemoreceptors. The concentration of 2,3 DPG increases, because of increase of pH in the cells(as deoxyhemoglobin binds more hydrogen ions than oxyhemoglobin). The person feels lazy, sleepy and may get a headache. These are due to central CNS depression by hypoxia.

Q) Enumerate changes that occur during Acclimatisation of High Altitude

**Acclimatisation of High Altitude**

1. Larger chest size

2. Increase in Red blood cells, circulation improves

3. Increased vascularity and improved oxygen utilisation in tissues

A 28-year-old nun is brought to the emergency department complaining of headache, vertigo, dizziness, and confusion. It was a cold winter evening. The patient lives in the ashram basement room. A truck unloading construction material had its exhaust stuck by mistake in her basement room all night. She did not come for break fast next morning and was found with cherry red discoloration of skin.

**LABORATORY STUDIES reveal**

Arterial blood gases: PO2 95 mm Hg, PCO2 40 mm Hg, pH 7.4

Mixed venous blood gases: PO2 22 mm Hg, PCO2 43 mm Hg, pH 7.37

Carboxyhemoglobin level: 40%

Question 1) What is your diagnosis?

Answer 1. Carbon monoxide poisoning

Question 2) What form of hypoxia is she suffering from?

Answer 2) Hypoxia is defined as deficiency of oxygen at the tissue level

**Ischemic or anemic hypoxia:** This occurs when hemoglobinconcentration is low. The decrease in hemoglobin concentration in carbon monoxide poisoning occurs due to formation of carboxyhemoglobin. The C0‐Hb affinity is twenty times more than that of oxygen‐Hb. The individual is cherry red in colour. The other causes are methemoglobinemia.

Question 3) Enumerate the factors affecting the shift of oxygen dissociation curve to left.

Ans3) **Shift to Left**

Carbonmonoxide poisoning

Alkalosis

Decrease in body temperature

Fetal haemoglobin

Myoglobin

Question 4) What would be the treatment for the sister?

Restoration of oxygen delivery to the brain will result in a diminishing of this patient’s symptoms. The defect in oxygen-carrying capacity, however, represents a significant barrier. Breathing 100% oxygen would increase fivefold the amount of oxygen dissolved in the plasma. This increase alone, however, would not be sufficient to support metabolism. Patients with severe

carbon monoxide poisoning, where carboxyhemoglo-bin levels exceed 70%, are treated in a hyperbaric oxy-gen chamber. At the higher total barometric pressure, the dissolved oxygen can be sufficient to support basal metabolism. Given time, carbon monoxide will disassociate from hemoglobin and hemoglobin-carrying capability can be restored. Alternatively, transfusion can be used to introduce normal hemoglobin into the patient to enhance the blood oxygen-carrying capacity.

**Problem Based Learning in General Physiology**

1. Calculate the E.C.F. volume and its % of T.B.W. Given the following data: from the following data:

Amount of thiosulphate injected 590mg.

Plasma concentration of substance at the end of 1 hour :3 mg/100 cc.

Amount of substance excreted in 1 hour =20 mg

Assume 45 liters of T.B.W. as 100%

Using the formula concentration=Amount/ Volume

and Amount = (amount injected- amount excreted)

Volume of ECF =(590-20)/0.03

= 570/0.03

=19000mL or 19L

Percentage 19/45 X100=42%

2. Calculate the I.C.F. Volume from the data:

a. Amount of Amino pyrine injected= 430 mg.

Amount of substance excreted during 30 minutes= 10 mg.

Conc. Of substance in plasma is 1 mg %

b. Amount of inulin injected 3.2 mg.

Amount of substance excreted during 1 hour 0.2 mg

Conc. Of Inulin is 0.02 mg % in plasma

Ans: Using the formula concentration=Amount/ Volume

and Amount = (amount injected- amount excreted)

ICF Volume cannot be calculated directly.

TBW volume= (430-10)/0.01=42L

ECF Volume= (3.2-0.2)/0.02=15L

ICF Volume= TBW-ECF

= 42-15= 27L

1. **Enumerate the different body fluid compartments**

Ans.

Plasma 5%

Interstitial 15%

ICF 40%

ECF = Plasma + Interstitial

Extracellular fluid (ECF)

Intracellular fluid (ICF)

Transcellular fluids e.g. CSF, aqueous humor, synovial fluid

The body water constitutes 60% of body weight (40+15+5%)

Remaining 40% is protein, carbohydrates, fat and minerals.

3. Calculate the Blood volume given the following data:

Amount of Evan’s Blue injected=5 mg.

Conc. Of dye in the plasma after 20 minutes 0.002 mg/ml.

P.C.V. 50 %

Ans 3. Evans Blue dye binds to plasma protein.

Volume of plasma= 5/0.002= 2.5L

Volume of PCV is 50%

Volume of plasma is also 50 %

So the volume of Blood Volume =2.5+2.5L= 5 L

**Problem based learning in Special senses and ENT**

**Q1. What is deafness? What are the types of deafness?**

Ans.

**Deafness** is defined aspartial or complete loss of hearing.

**Types:**

**1. Conductive deafness**: When the cause of deafness is present in external ear or middle ear.

External ear causes:

**a. Congenital:**

Atresia

Microtia

  Treacher-Collins syndrome

**b. Impacted wax or cerumen**

**c. Impacted foreign body**

**d. Otitis externa:**

Diffuse otitis externa (when the auditory canal is obstructed)

Otomycosis (when mycotic plug is formed)

**e. Neoplasm:**

(i)Benign - Osteoma, chondroma, exostosis.

(ii)Malignant - Osteosarcoma, chondrosarcoma.

Middle ear causes:

**a. Congenital:**

oOssicular chain deformity

oFused ossicles

oIncudo-stapedial joint separation

oCongenital otosclerosis

**b.Traumatic**

oHaematomas

oOssicular dislocation

**c.Inflammatory**

oAcute : ASOM

oChronic:

-Non-specific: CSOM, Adhesive otitis media, secondary otitis media

-Specific: Tubercular and syphilitic.

**d.Neoplastic**

oGlomus jugularae

oCarcinoma.

**2. Sensory-neural deafness**

Causes of sensory neural deafness are-

Congenital **(a)Hereditary group (Genetic):**

Pendred syndrome

Waardenburg‟s syndrome

  Klippel Fiel syndrome

**(b)Pregnancy group:**

Rubella

Rh-factor

Congenital syphilis

Severe viral infection of the mother

**(c)Birth group or prenatal group:**

Prolonged labour

Anoxia or hypoxia

Premature birth

Birth trauma

Phenylketonuria

**I.Cause in cochlea or inner ear:**

i.Traumatic:

Fracture temporal bone (\*)

Head injury

Blast injury (\*)

ii.Operative:

Post-stapedectomy (\*)

Labyrynthectomy

iii. Infective:

Bacterial: Labyrinthitis (\*)

Viral: Measles, mumps, influenza,pox, etc.

iv.Vascular:

Spasm

Thrombosis

v.Toxic:

Streptomycin, quinine, gentamycin,and other ototoxic drugs. (\*)

vi. Degenerative:

Senile deafness or presbyacusis

vii. Noise induced:

Acute noise trauma

Chronic noise trauma

viii. Miscellaneous:

Diabetes

Meniere‟s disease

Ramsay Hunt syndrome

**II.Causes in internal auditory canal and C.P.angle:**

i.Acuostic neuroma (\*)

ii.Meningioma

iii.Cholesteatoma

iv.Tuberculoma

v.Basal meningitis

**III.Cause in central nervous system:**

Dessiminated sclerosis

Vascular accidents

Tumours

**3.Mixed deafness**

**4.Psychogenic deafness.**

**List the tests of hearing and explain any one of them.**

**Ans.** Tests of hearing

A watch may be used to test hearing. The distance at which the tick tock disappears is compared with both ears and the ears of the examiner.

The normal ear can hear the rubbing (not snapping) of the index and the thumb done when feeling for money or cloth texture

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Bone

conduction

better than

normal

Bone

conduction

worse than

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Audiometry measures the auditory acuity when the subject is subjected to pure tones of various frequencies

**What are cochlear microphonic potentials**?

**Ans.** If an electrode is placed in any cochlear compartment oreven near the cochlea, a electrical potentials an be recorded in response to acoustic stimuli. These follow the wave form of the stimuli and are known as cochlear microphonics.

It arises in the outer hair cells. It is inhibited by antibiotic kanamycin.

These are generated by the piezoelectric effect. They are present even after birth. They are the electricity generated by the mechanical movement of hair cells.

1. **What is organ of Corti? What are travelling waves? How are they generated in the inner ear? State the role of travelling waves in the mechanism of hearing. Give an account of the mechanism of hearing.**

**Ans.** The sensory cells responsible for hearing are located on thebasilar structure known as organ of corti. This organ extends from the apex to the base of the cochlea and has a spiral shape. It includes **reticular lamina** supported by **rods of Corti.** They contain **outer and inner hair cells.**

**Travelling waves theory**

The movements of the footplate of the stapes set up a series of travelling waves in the perilymph of the scala vestibuli. The height of this wave increases as it moves upward and then dies down. The frequency of the vibration initiating the wave determines the distance to the point of maximum height. Frequency is inversely proportional to this distance. Low pitch sound waves peak nears the apex and high waves sound peak at the apex. These sound waves produce distortion of basilar membrane. The site at which distortion is maximum determines by the frequency.

**Mechanism of hearing**

**Role of external ear**

**Pinna:** collects sound waves and localises the source of sound

**External auditory meatus:** transports sound to middle ear

**Role of middle ear**

**Tympanic membrane:** exerts as pressure receiver, and resonatorwhen sound waves strikes. It critically dampens the sound waves and stops vibrating when the sound waves stop stretching the tympanic membrane.

**Auditory ossicles:** function as lever system that converts theresonant vibration of the tympanic membrane into movements of stapes against the perilymph filled scala vestibuli of cochlea. Its here that sound is converted to fluid. Sound is magnified by 1.5 to 2 times. The sound pressure increases in the middle ear (**Impedence matching)**

**Eustachian tube:** Equilibrates the air on either side of middle earand outer ear (two sides of tympanic membrane)

**Role of internal ear**

**Vibration of basilar membrane**

The basilar membrane vibrates into the scala tympani causing transmission of pressure to oval window. This too and fro rocking motion sets up wave motion in membranous labyrinth.

**Stimulation of hair cells**

The cilia are embedded in tectorial membrane. The movement of basilar membrane moves the hair cells and generates action potential in auditory nerve

**Functions of hair cells**

Inner hair cells are the sensory cells for auditory discrimination. Outer hair cells are helpful in improving hearing by influencing vibration pattern of basilar membrane.

**Pitch discrimination mechanism**

**Place theory** is that frequency inversely to the distance from thehelicotrema as explained above.

**Volley theory** is that the discharge of action potential in thenerve determines loudness. A loud sound produces more rate of firing of action potentials

**Duplex theory :**combines Volley and Place

**Electrophysiology of hearing**

Sound is conducted by air. The ear receives it, the nerve transmits it and the brain interprets and understands it. **Electrical activity in cochlea:** The potential difference betweenthe endolymph and perilymph.

**Action potential of auditory nerve**:

Auditory cortex The anterior part of the temporal cortex receives apex cochlea low frequencies. Posterior receives the base of cochlea high frequency tones. This is tonotopic organisation.

**Q 2. Sudden pain in the ear of a child after upper respiratory tract infections. What is your diagnosis? How is it diagnosed?**

Ans. Child is suffering from acute suppurative otitis media, its clinical features are-

**1. Stage of tubal occlusion:**

**Symptoms:**

i)Acute coryza

ii)Mild earache

iii)Fullness in the ear

iv)Mild conductive deafness

**Signs:**

i)Tympanic membrane retracted & lusterless.

**2. Stage of exudation or pre-suppuration:**

**Symptoms:**

i)Severe earache (sharp & stabbing)

ii)Deafness increases

iii)Bubbling sound (due to serous exudates)

iv)General illness: Rise of temperature, Malaise, Vomiting.

**Signs:**

i) Tympanic membrane: Red & congested, dilated vessels radiating from the handle of the malleus gives Cart-wheel appearance.

**3. Stage of suppuration:**

**Pre-perforation:**

**Symptoms:**

i) Pain is more acute (throbbing)

ii) Deafness is more marked

iii) High rise of temperature (101˚-103˚F)

**Signs:**

i) Bulged, congested & yellow spot on the tympanic membrane

ii) Mastoid tenderness

**Perforation:**

**Symptoms:**

i) Ottorhea (pus or mucopus or may be blood stained)

ii) Pain diminishes

iii)Temperature comes down

iv) Conductive deafness is more marked

**Signs:**

i) Perforation on the tympanic membrane

ii) Pulsating discharge reflect light intermittently called “Light-house sign”

 iii) Mastoid tenderness disappears

**4. Stage of resolution:**

i) In early cases or in mild infection: Resolution occurs without perforation

ii) In cases of perforation: Discharge subsides and perforation heals up or dry small perforation is left behind

**5. State of complication:**

i)Persistence of otorrhoea & deafness

ii)Vertigo & headache

iii)Increase temperature

iv)Facial paralysis.

**Q 3. What are the ototoxic drugs? Mention five.**

Ans.Ototoxic drugs are-

1)Aminoglycoside antibiotics:

Streptomycin & gentamycin - mainly vestibulotoxic,

Neomycin, kanamycin, vancomycin & tobramycin - mainly cochleotoxic.

2)Diuretics: Ethacrynic acid, frusemide, etc.

3)Anti-malarial drug: Quinine, Chloroquine, etc.

4)NSAID: Salicylate, Aspirin, etc.

5)Tobacco and alcohol.

**Q 4. Enumerate causes of pain in ear (otalgia).**

Ans.

**Local cA.External ear**

Furuncle

Perichondritis

Otitis externa

Impacted wax & FB

Herpetic lesions including bullous myringitis

Traumatic rupture of TM & myringitis

Malignant growth

**1.Via Vth cranial nerve**

(a)Dental:

Caries tooth

Apical abscess

Impacted molar

Malocclusion

(b)Oral cavity:

Benign or malignant ulcerative lesions of oral cavity or tongue

(c)Temperomandibular joint disorder:

Bruxism

Osteoarthritis

Recurrent dislocation

Ill-fitting denture

(d)Sphenopalatine neuralgia.

**2.Via IXth cranial nerve**

(a)Oropharynx:

Acute tonsillitis

Peritonsillar abscess

Tonsillectomy

Benign or malignant ulcer of soft palate, tonsil and its pillars

(b)Base of tongue:

Tuberculosis or malignancy

(c)Elongated styloid process.

**3.Via Xth cranial nerve**

Malignancy or ulcerative lesion of—Vallecula, Epiglottis,Larynx or laryngopharynx,Oesophagus.

**4.Via C2** **and C3** **spinal nerves**

Cervical spondylosis

Injuries to cervical spine

Caries spine

**B.Middle ear**

Acute otitis media

Acute salpingitis

Acute mastoiditis

Barotraumatic otitis media

Haemotympanum

Unsafe variety of CSOM withthreatening complications

Malignant growth

.

**Q 5. 48 years female patient complaining of 15-20 sneezes at a time since 10 years and history of intermittent nasal obstruction and watery nasal discharge since last 1 year. What is the likely diagnosis and management.**

Ans.

This patient is having allergic rhinitis

**Symptoms:**

 (1)Paroxysmal sneezing followed by watery nasal discharge

(2)Nasal obstruction

(3)Nasal irritation

(4)Anosmia

(5)Heaviness of head & headache

(6)Irritation and congestion of eyes, respiratory distress, and broncho-spasm.

**Medical Management.**

**Prophylactic:**

(a)Avoidance of allergen

(b)A course of desensitizing vaccine based on result of skin sensitivity test.

(c)Hyposensitisation by vaccine

(d)Immuno-therapy by gamma-globulin or immunoglobulin injection.

**Curative management:**

1.Oral antihistamines e.g., Pheniramine maleate, Promethazine, Cetrizine, Terfanadine, Loratidine, Fexofenadine etc.

2.Symapthomimetic drugs:Topical use of sympathomimetic drugs causes nasal decongestion e.g., Phenylephrine, Oxymetazoline, Xylometazoline et.

3.Steroid:Can be used as spray (e.g., Beclomethasone, Fluticasone) or as submucosal injection.

4.Sodium chromoglycate as nasal spray.5.

General body nutrition is to be improved. Vitamin C and B-complex is to be administered. Bowel is to be kept regular.

**Surgical management:**

(a) Minor surgery:

  Reduction of nasal turbinate‟s (inferior):

  (i)Surface electro-cautery

(ii)Submucosal diathermy (S.M.D)

(b)Other nasal surgery:

(i)Submucosal resection (S.M.R)

(ii)Fiber-optic endoscopic sinus surgery (FESS).

**Q. 6 Enumerate Epistaxis in a child of 3/4 years - cause and management.**

Ans.

**Cause:**

In children commonest cause is epistaxis from Little‟s area either spontaneous or

due to-

1.Picking of the nose

2.Injury to the nose

3.Exanthematous fever (e.g., measles, pox)

4.Foreign body nose

5.Diphtheric rhinitis

6.Enlarged adenoids, etc.

**Management:**

1. Pinching of nose for 10-15 minutes, as pressure on nostril from outside compresses the vessels on the Little’s area and stops bleeding.

 2. Traumatic bleeding is often controlled by application of ice on bridge of the nose which causes reflex vaso-constriction.

3.In cases of persistent bleeding, the blood is sucked out including clots with suction machine under direct vision and spraying the nose with 4% xylocaine.

- If the actual bleeding point is found out, may be cauterized by chemical or electro-cautery under G/A.

- If bleeding point cannot be seen, then thick cotton wool pledget soaked in 4% Xylocaine solution should be inserted into the nasal cavity and it is removed after several minutes.

**Q 7. What you know about achalasia cardia?**

Ans.

**Achalasia cardia:**

Achalasia Cardia is a primary oesophageal motility disorder, characterized by a hypertensive lower oesophageal sphincter (LOS) which fails to relax on swallowing, and by aperistalsis of the body of the oesophagus.

**Incidence:**

The incidence of the disease is 1-2 per 200,000 per year, with both sexes equally affected.Onset of the disease is typically between the ages of 20 and 50.

**Aetiology:**

Exact aetiology is unknown. Some theories are-

1.Loss of ganglionic cells in the myenteric (Auerbach‟s) plexus

2.Abnormal pinch-cock action of right crus of diaphragm

3.Vagal disturbance

4.Aerophagy

5.Primary dilatation

6.Lack of integrated parasympathetic stimulation and non-propulsive motility in the body of the oesophagus.

**Pathology:**

Marked dilatation of the lower two- third of the oesophagus

Lumen (diameter) 7.5 cm.

Muscular walls are hypertrophied

No hypertrophy of the cardiac sphincter

Histopathology of muscle specimens generally shows a reduction in the number of ganglion cells (and mainly inhibitory neurons) with a variable degree of chronic inflammation.

**Clinical features:**

Age- Young person of both sexes. Onset of disease is insidious.

Symptoms:

1.Dysphagia

  –more liquid then solid.

2.Regurgitation of undigested food.

3.Discomfort or pain in the retrosternal or epigastric region.

4.Loss of weight.

5.Fullness after meal in retrosternal or epigastric area.

6.Night time cough.

**Differential diagnosis:**

1.Carcinoma of oesophagus

2.Stricture

3.Hiatus hernia.

**Investigations:**

1.Endoscopic examination shows a tight cardia and food residue in the oesophagus.

2. Barium swallow X-ray of the oesophagus usually shows a "bird‟s beak" narrowing at the GO junction and oesophageal dilatation proximal to the narrowing.-

 3.Oesophagoscopy

shows the dilated oesophagus with smooth narrowing of cardiac end containing undigested food.

4.Oesophageal Manometry :

In this test, a thin tube is passed into the esophagus to measure the pressure exerted by the esophageal sphincter.)

Typical manometrical findings are the absence of oesophageal peristalsis and a hypertensive LOS which fails to relax completely in response to swallowing.

**Treatment:**

1.Medical treatment: Before meal- Nifedipine

2.Botulinum toxin injection. Injected into the sphincter, botulinum toxin paralyzes themuscle and allows it to relax.

3.Forceful dilatation of cardia under general anaesthesia: Oesophagoscopy & dilatation by-

Plastic balloons and Hydrostatic bag

4.Surgical treatments:

Hellar‟s myotomy under general anaesthesia

Anastomic operation– anastomosis between stomach & oesophagus.

**Q.8. A woman of 45 years of age with anaemia complains dysphagia. What is your diagnosis ?**

Ans.

 The woman is probably suffering from Plummer-Vinson syndrome (Paterson-Brown Kelly syndrome).This is a precancerous lesion, commonly seen in woman whom there is chronic superficial pharyngo-oesophagitis.

**Aetiology:**

Iron deficiency anaemia

Vitamin deficiency

Auto-immune disease.

**Clinical features:**

1.Dysphagia – more to solids

2.Feeling of lump in the throat

3.Features of iron deficiency anaemia

4.Angular stomatitis, glossitis, and koilonychias.

5.Web formation or cicatrisation in post-carotid region.

**Diagnosis:**

(1) By clinical features, signs of vitamin deficiency

(2) Hypochromic microcytic anaemia

(3) Barium swallow X-ray shows a web at the post-cricoid region.

(4) Hypopharyngoscopy and oesophagoscopy to confirm.

(5) Serum iron and iron binding capacity to see prognosis after treatment.

**Treatment:**

1.Iron and vitamins are given in large doses.

2.Endoscopic examination and dilatation relieves dysphagia.

3.Follow-up.

**Q.9. Enumerate causes of thyroid swelling. Name the investigations of thyroid enlargement.**

Ans.

Classification of thyroid swelling:

**Simple goiter (euthyroid)**

Diffuse hyperplastic Physiological Pubertal Pregnancy Multinodular goiter

**Toxic**

Diffuse

Graves‟ disease

 MultinodularToxic adenoma

**NeoplasticBenign**

Follicular adenoma

**Malignant**

  Papillary carcinoma

Follicular carcinoma

    Anaplastic carcinoma

Medullary carcinoma

Lymphoma

Metastatic Local infiltration

**Inflammatory**

Autoimmune Chronic lymphocytic thyroiditis

Hashimoto’s thyroiditis

  Granulomatous De Quervain‟s thyroiditis

 Fibrosing Riedels thyroiditis

Infective Acute (bacterial thyroiditis, viral thyroiditis, „subacute thyroiditis‟)

 Chronic (tuberculous, syphilitic)

**Investigations:**

**1.Thyroid function tests**

T3,T4,TSH

**Euthyroid, Thyrotoxic, Myxoedema**

**2.** **Autoantibody titres**

  Serum level of antibodies against thyroid peroxidase (TPO) and thyroglobulin are useful in determining the cause of thyroid dysfunction and swelling. Levels above 25

Units for TPO antibody and titres of greater than 1:100 for anti-thyroglobulin are considered significant.

**3.** **Isotope scan**

The uptake by the thyroid of a low dose of either radiolabelled iodine (132 I) or the cheaper technetium (99mTc) will demonstrate the distribution of activity in the whole gland. In hyperthyroidism both the proportion of the tracer dose taken up and the rate at which this takes place are increased.

**4.** **Ultrasonography**

Ultrasongrapphy is used in determining the physical characteristics of thyroid swellings and to demonstrate subclinical nodularity and cyst formation.

**5.** **Fine-needle aspiration cytology**

FNAC is the choice of investigation in discrete thyroid swellings. Thyroid conditions that can be diagnosed by FNAC include colloid nodules, thyroiditis, papillary carcinoma, medullary carcinoma, anaplastic carcinoma and lymphoma.

**6.Radiology**

Chest and thoracic inlet radiograph may confirm the presence of significant retrosternal goitre and tracheal deviation, compression or retrosternal extension and are required when either clinical suspicion or FNAC indicates malignancy

.

**7.Ultrasound scan**

High-frequency ultrasound gives good anatomical images of the thyroid and surrounding structures.

**8.Other scans**

Computed topography (CT), magnetic resonance imaging (MRI) and positron emission topography (PET) are used for the assessment of known malignancy and to assess the extent of retrosternal and, occasionally, recurrent goitres.

**9.Laryngoscopy**

Flexible laryngoscopy is used preoperatively to determine the mobility of the vocal cord.

**10.Core biopsy**

Core biopsy gives a strip of tissue for histological assessment. It is applied in assessment of locally advanced, surgically unresectable malignancy.

**What is the role of skeletal muscles of middle ear?**

**Ans.** Tensor tympani: increases the tension of tympanicmembrane by pulling the handle of malleus medially. Thus it keeps the tympanic membrane attached.

Stapedius: It pulls the footplate of the stapes out of the oval window on contraction. It is supplied by facial nerve. They protects the internal ear from loud sounds.

**What is Anosmia?**

**Ans.** Anosmia is complete absence eof sense of smell due todamage of olfactory mucosa or olfactory pathway by disease or trauma.