The Tamilnadu Dr MGR Medical University MBBS Prefinal Otolaryngology march 2007 question paper with solution

By

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Drtbalu's otolaryngology online

I. Write Essay:

1. Enumerate the causes of conductive hearing loss with intact tympanic membrane. Discuss the clinical features and management of otitis media with effusion in a 4 year old child. (15 marks)

Introduction:
Conductive deafness is defined as deafness caused due to defects involving the conducting mechanism of the ear. The components of conducting mechanism include:

External ear – Pinna, external auditory canal, ear drum
Middle ear – Medial wall of ear drum, the three ossicles, air in the middle ear cavity.

Any derangement in the above mentioned components responsible for the conduction of sound could cause conductive deafness.

Causes of conductive hearing loss in a patient with intact ear drum:

External ear causes:
Impacted cerumen. Impacted cerumen can cause conductive hearing loss to the extent of 30%. The hearing loss is more if the cerumen is closely adherent to the ear drum thereby preventing its effective vibration in response to sound.

Osteomas involving bony portion of external canal. These are benign lesions involving the bony portion of the external auditory canal. This is common in swimmers. Presence of these masses in the external canal prevents effective sound transmission to the ear drum.

Ear drum causes:
Tympanosclerosis (extensive) involving the ear drum can cause ineffecive sound transmission to the middle ear ossicular chain. Extensive tympanosclerosis can prevent ear drum from vibrating normally in response to sound.

Middle ear causes:

Ossicular chain defects:
Fusion of head of the malleus and incus.
Disruption of incus
Fixation of foot plate of stapes (otosclerosis)

Accumulation of fluid in the middle ear cavity:

Secretory otitis media – In this condition middle ear cavity is filled with fluid. This causes ineffective sound coupling between the ear drum and the ossicular chain. This condition is common in children and chronic adenotonsillitis has been implicated as the potential cause for this condition.

Acute otitis media – Purulent material gets accumulated in the middle ear cavity. These patients have intense ear ache in addition of conductive deafness.

Adhesive otitis media – Eustachean tube dysfunction may cause blockage in the communication between the middle ear cavity and

the nasopharynx. This in turn prevents normal ventilation of the middle ear. Absence of regular gas exchange via the eustachean tube causes vacuum in the middle ear cavity. This vacuum causes the ear drum to prolapse into the middle ear cavity. It may be so retracted that it could be in direct contact with the promontory. In this condition there is ineffective sound coupling between the ear drum and the ossicular chain.

Clinical features and management of otitis media with effution in a 4 year old child:

Age of occurrence: OME shows classically a bimodal distribution. The first peak occurs around 2 years of age, and the second peak occurs at about 5 years of age. This distribution occurs roughly around the ages when the child goes to preschool and primary school.

Clinical features: A high index of suspicion is necessary to identify this condition. Every child with upper respiratory infection must be otoscopically examined. These children have a tendency to keep fiddling with the ear using their fingers / ear buds. This could be the direct result of conductive deafness. Otoscopic findings: The tympanic membrane may be bulging, or retracted with a distorted cone of light. The ear drum may appear yellow, blue or simply clear white. Pneumatic otoscopy will reveal a ear drum which has a restricted mobility.

Management:

1. Antibiotics: Amoxycillin is the drug of choice followed by cephalosporins.

2. Nasal decongestants like oxymetazoline / xylometazoline may help in some cases.

3. Topical nasal steroids can be used in resistant cases.

4. Autoinflation of eustachean tube by performing valsalva maneuver. Balloon blowing may also help.

Surgical management:

1. Adenotonsillectomy

2. Myringotomy and insertion of ventilation tubes – This is usually performed in the anteroinferior quadrant. It should be borne in mind that antero inferior quadrant is closest to the tympanic end of the eustachean tube. Insertion of ventilation tube in this area would ensure drainage of middle ear contents and ventilation via the external auditory canal.

2. Enumerate the causes of epistaxis. Describe the management of epistaxis.
(5 + 5 = 10 marks)

Definition: Epistaxis is defined as bleeding from the nasal cavity. This is a Greek word meaning nose bleed. Since it is a very common problem its true incidence is very difficult to predict.

History: Hippocrates said that pinching the nose for sometime and asking the patient to breath through the mouth stopped bleeding from the nose. Carl Michel and James Little were the first to identify the vascular plexus in the anterior part of the nasal septum as the common area from which nasal bleeding occurs. Pilz was the first person to treat epistaxis by surgically ligating the external carotid artery, seiffert ligated the internal maxillary artery through the maxillary antrum via caldwelluc approach.

The nose has a rich supply of blood vessels with good contribution from both external and internal carotid systems. The general rule of the thumb is that the area of nasal cavity below the level of middle turbinate has rich blood supply from the external carotid system, where as the area above the middle turbinate receives extensive supply from the internal carotid artery. Anastomosis occur between the external and internal carotid system throughout the nasal cavity.

External carotid system: Blood from the external carotid system reaches the nasal cavity via the facial and the internal maxillary arteries which are branches of the external carotid artery. The artery of epistaxis is the sphenopalatine branch of internal maxillary artery. This is called so because this vessels supplies the major portion of the nasal cavity. It enters the nasal cavity at the posterior end of the middle turbinate to supply the lateral nasal wall, it also gives off a septal branch which supplies the nasal septum.

Facial artery: the superior labial branch of the facial artery is one of its terminal branches. It supplies the anterior nasal floor and anterior portion of the nasal septum through its septal branch.

Internal maxillary artery: after entering into the pterygopalatine fossa this vessel gives rise to 6 branches. These branches are posterior superior aleveolar artery, descending palatine artery, infra orbital artery, sphenopalatine artery, pterygoid artery, and pharyngeal artery. The descending palatine artery enters the nasal cavity through the greater palatine canal to supply the lateral wall of the nose, it also contributes blood supply to the nasal septum through its septal branch.

Internal carotid system: the internal carotid artery supplies the nasal cavity via its ophthalmic artery. It enters the orbit via the superior orbital fissure and divides into many branches. The posterior ethmoiod artery one of the branches of ophthalmic artery

exits the orbit via the posterior ethmoidal foramen located 2-9 mm anterior to the optic canal. The anterior ethmoidal artery which is larger leaves the orbit through the anterior ethmoidal foramen. Both these vessels cross the roof of the ethmoid and descends into the nasal cavity through the cribriform plate. It is here that these vessels divide into lateral and septal branches to supply the nose.

Little's area: This area is located in the anterior part of the cartilagenous portion of the nasal septum. Here there is extensive submucous anastomosis of blood vessels both from the external and the internal carotid systems. Bleeding commonly occurs from this area since it is highly vascular and is also exposed to the exterior. Anastomosis occur between the septal branches of sphenopalatine artery, greater palatine artery, superior labial artery and the anterior ethmoidal artery. This plexus is also known as Keisselbach's plexus. Bleeding from this area is common because mucosal drying occurs commonly here and this area is easily accessible to nose picking. Among the vessels taking part in the anastomosis the anterior ethmoidal artery is from the internal carotid system while the other vessels are from the external carotid system. Bleeding from this area is clearly seen and easily accessible and flows through the anterior nasal cavity hence it is known as anterior bleed.

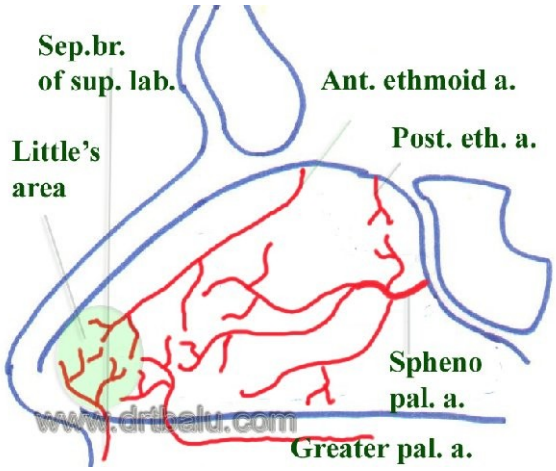

Figure showing little's area

Woodruff's plexus: is responsible for posterior bleeds. This area is located over the posterior end of the middle turbinate. The anastomosis here is made up of branches from the internal maxillary artery namely its sphenopalatine and ascending pharyngeal branches. The maxillary sinus ostium forms the dividing line between the anterior and posterior nasal bleeds. Posterior nasal bleeds are difficult to treat because bleeding area is not easily accessible. Bleeding from Woodruff's plexus commonly occur in patients with extremely high blood pressure. Infact this plexus acts as a safety valve in reducing the blood pressure in these patients, lest they will bleed intracranially causing more problems. In patients with posterior bleeds it is difficult to access the amount of blood loss because most of the blood is swallowed by the patient.

Etiology: The etiology of epistaxis is not just simple or straight forward. It is commonly multifactorial, needing careful history taking and physical examination skill to identify the cause. For purposes of clear understanding the etiology of epistaxis can be classified under two broad heads, i.e. local and systemic causes.

Local factors causing epistaxis: include vascular anamolies, infections and inflammatory states of the nasal cavity, trauma, iatrogenic injuries, neoplasms and foreign bodies. Among these causes the commonest local factors involved in epistaxis is infection and inflammation. Infections and inflammation of the nasal mucous membrane may damage the mucosa leading on to bleeding from the underlying exposed plexus of blood vessels. Chronic granulomatous lesions like rhinosporidiosis can cause extensive epistaxis.

Aneurysms involving the internal carotid artery may occur following head injury, injury sustained during surgical procedures. These extradural aneuryms and aneurysms involving the cavernous sinus may extend into the sphenoid sinus wait for the opportune moment to rupture. It can cause sudden fatal epistaxis, or blindness. Urgent embolisation is the preferred mode of management of this condition.

Trauma is one of the common local causes of epistaxis. It is commonly caused by the act of nose picking in the Little's area of the nose. This is commonly seen in young children. Acute facial trauma may also lead to epistaxis. Patients undergoing nasal surgeries may have temporary episodes of epistaxis.

Irritation of the nasal mucous membrane: any disruption of normal nasal physiology can cause intense drying and irritation to the nasal mucosa causing epistaxis. These episodes are common during extremes of temperature when the nasal mucosa is stressed to perform its airconditioning role of the inspired air. In these conditions there is extensive drying of nasal mucosa causes oedema of the nasal mucous membrane. This oedema is caused due to venous stasis. Ultimately the mucosa breaches exposing the underlying plexus of blood vessels casuing epistaxis.

Anatomical abnormalities: Common anatomical abnormality causing epistaxis is gross septal deviation. Gross deviations of nasal septum causes disruption to the normal nasal airflow. This disruption leads to dessication / drying of the local mucosa. The dry mucosa cracks and bleeds.

Septal perforations: Chronic non healing septal perforations can cause bleeding from the granulation tissue around the perforation.

Neoplasms: involving the nose and paranasal sinuses can cause epistaxis. Neoplasms include benign vascular tumors like hemangioma, juvenile nasopharyngeal angiofibroma, and malignant neoplasms like squamous cell carcinoma. If epistaxis

occurs along with secretory otitis media then nasopharyngeal carcinoma should be the prime suspect.

Systemic causes for epistaxis:

Hypertension is one of the common systemic causes of epistaxis. Accumulation of atheroscerotic plaques in the blood vessels of these patients replaces the muscular wall. This replacement of muscular wall reduces the ability of the blood vessels to constrict facilitating epistaxis. This is one of the common causes of posterior nasal bleeds. It commonly arises from the Woodruff's plexus found close the posterior end of the middle turbinate.

Hereditary hemarrhagic telengectasia is another systemic disorder known to affect the blood vessels of the nose. This disease causes loss of contractile elements within the blood vessels causing dilated venules, capillaries and small arteriovenous malformations known as telengectasia. These changes can occur in the skin, mucosal lining the whole of the respiratory passage and urogenital passage. Bleeding from these telengectasia is difficult to control. Bleeding invariably starts when the patient reaches puberty. Common cause of mortality in these patients is gastrointestinal bleed.

Systemic diseases like syphilis, tuberculosis & wegner's granulomatosis cause epistaxis because of their propensity to cause ulceration of the nasal mucous membrane.

Blood dyscrasias can also cause epistaxis. A low platelet count is one common cause of nasal bleed in this category. In thrombocytopenia the platelet count is less than 1 lakh. Epistaxis can start when the platelet count reduces to 50,000. Platelet deficiency can be caused by ingestion of drugs like aspirin, indomethacin etc. Hyperspenism can cause thrombocytopenia in idiopathic thrombocytopenic purpura. These patients need to be transfused fresh blood in adequate quantities. Only when thee platelet count increases will the nasal bleed stop.

Incidence: The incidence of epistaxis is known to be slightly higher in males. It also has a bimodal distribution affecting young children and old people.

Evaluation: While evaluating a patient with epistaxis it is absolutely necessary to assess the quantum of blood loss. The blood pressure and pulse rate of these patients must be constantly monitored. These patients will have tachycardia. Infusion of fluid must be started immediatly. Initially ringer lactate solution will suffice. If the patient has suffered blood loss of more than 30% of their blood volume (about 1.5 liters) then blood transfusion becomes a must. Further examination should be started only after the patient's general condition stabilises.

History: Careful history taking is a must. History taking should cover the following points:

1. History regarding the freqency, severity and side of the nasal bleed.

2. Aggravating and relieving factors must be carefully sought.

3. History of drug intaken must be sought.

4. History of systemic disorders like hypertension and diabetes mellitus must be sought.

Physical examination:

The nasal pack if any must be removed. Anterior nasal examination should be done, first attempted without the use of nasal decongestants. If visualisation is difficult due to oedema of the nasal mucosa then nasal decongestants can be used to shrink the nasal mucosa. The solution used for anesthetising the decongesting the nose is a mixture of 4% xylocaine and xylometazoline.

Nasal endoscopy can be performed under local anesthesia to localise posterior bleeds.

Investigations:

If bleeding is minimal no investigation is necessary.

If bleeding is more then a complete blood work up to rule out blood dyscrasias is a must. It includes bleeding time, clotting time, platelet count and partial thromboplastin time.

Imaging studies like CT scan of the para nasal sinuses must be done to rule out local nasal conditions of epistaxis. Imaging must be done only after 24 hours of removing the nasal packing. Scans done with the nasal pack or immediatly after removing the nasal pack may not be informative.

In diffiucult and intractable cases angiography can be done and the internal maxillary artery can be embolised in the same sitting. This procedure should be reserved only for cases of intractable nasal bleeding.

Management:

Conservative:

Nasal packing: Anterior nasal packing using roller gauze impregnated with liquid paraffin is sufficient to manage a majority of anterior nasal bleeds. The liquid paraffin acts as a lubricant, and as a moistoning agent. The tamponoding effect of a nasal pack is sufficient to stop nasal bleeding. This type of roller gauzes can be kept inside the nasal cavity only up to 48 hours after which it has to be removed and changed. The newer packs like the BIPP (Bismuth Iodine paraffin paste) packs can be left safely in place for more than a week.

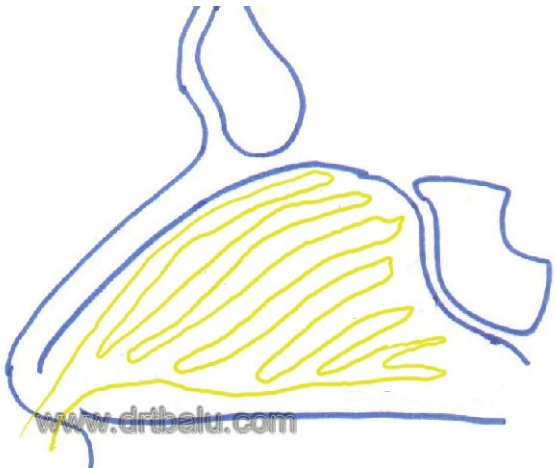

Figure showing anterior nasal packing

To manage post nasal bleed a post nasal pack is a must. Post nasal packing can be done in 2 ways:

Post nasal packing (conventional): A gauze roll about the size of the patient's naso pharynx is used here. Three silk threads must be tied to the gauze roll. One at each end and the other one at the middle. The patient should be in a recumbent position. After anesthetising the nasal cavity with 4% xylocaine the mouth is held open. Two nasal catheters are passed through the nasal cavities till they reach just below the soft palate. These lower ends of the catheters are grasped with forceps and pulled out through the mouth. The silk tied to the ends of the gauze is tied to the nasal catheters. The post nasal pack is introduced through the mouth and gradually pushed into the nasopharynx, at the same time the nasal catheters on both sides of the nose must be pulled out. When the pack snugly sits inside the nasopharynx, the two silk threads tied to its end would have reached the anaterior nares along with the free end of the nasal suction catheter.

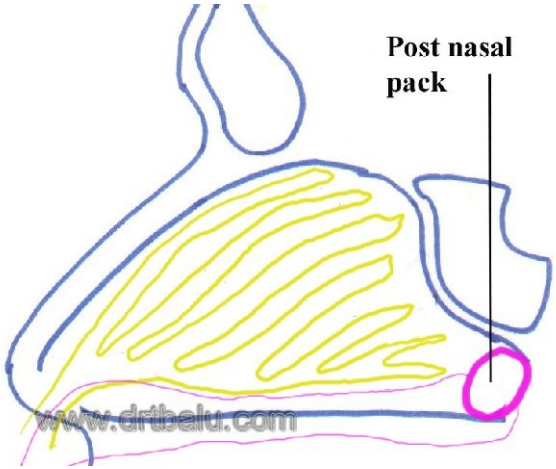

Figure showing postnasal pack in situ

The two silk threads tied to the suction catheters are untied. The catheters are removed from the nose. The silk thread is used to secure the pack in place by tying both the ends to the columella of the nose. The silk tied to the middle portion of the gauze pack is delivered out through the oral cavity and taped to the angle of the cheek. This middle portion silk will help in removal of the nasal pack. In addition to the postnasal pack anterior nasal packing must also be done in these patients.

Postnasal pack using baloon catheters: Specially designed baloon catheters are available. This can be used to perform the post nasal pack. Foleys catheter can be used to pack the post nasal space. Foley's catheter is introduced through the nose and slid up to the nasopharynx. The bulb of the catheter is inflated using air through the side portal of the catheter. Air is used to inflate the bulb because even if the bulb ruptures accidentally there is absolutely no danger of aspiration into the lungs. After the foleys catheter is inflated the free end is knotted and anchored at the level of the anterior nares.

Problems of nasal packing:

1. Epiphora (watering of eyes) occur due to blocking of the nasal end of the nasolacrimal duct.

2. Heaviness /headache due to blocking of the normal sinus ostium.

3. Prolonged post nasal pack can cause eustachean tube block and secretory otitis media.

4. Prolonged nasal packing can cause secondary sinusitis due to blockage of sinus ostium.

Newer packing materials: Newer packing materials made of silicone are available. The advantages of these material are that they are not irritating, patient can breath through the nose with the pack on through the vent provided, these packs can be retained inside the nasal cavity for more than 2 weeks. They can be removed and repositioned if necessory. The only disadvantage is that they are expensive.

Surgical management:

Endoscopic cauterisation can be tried if the bleeders are localised and accessible. If not accessible, ligation of the internal maxillary artery can be done through caldwelluc approach. Spenopalatine artery clipping can be done endoscopically. It is accessible close to the posterior end of the middle turbinate. In rare cases external carotid artery ligation at the neck can be resorted to. External carotid artery is differentiated from the internal carotid in the neck by the fact that internal carotid artery does not give rise to branches in the neck, while the external carotid artery does so.

Ethmoidal artery ligation: If epistaxis occur high in the nasal vault, anterior and posterior ethmoidal arteries may be ligated using ligaclips. These arteries can be accessed using an external ethmoidectomy incision. The anterior ethmoidal artery is usually found 22mm from the anterior lacrimal crest. If ligation of the anterior ethmoidal artery does not stop bleeding then posterior ethmoidal artery should also be ligated. The posterior ethmoidal artery can be found 12mm posterior to the anterior ethmoidal vessel.

Epistaxis caused by the presence of tumors both benign and malignant calls for definitive treatment of the tumor perse.

3. What are the causes of stridor in children? Discuss the types, procedure and complications of Tracheostomy. (3 + 2 + 3 + 2 = 10 marks)

Causes of stridor in children can be classified according to the anatomy of the lesion:

1. Supralaryngeal causes:

a. Nose - choanal atresia

Obstruction due to infection / truama / tubes

b. Cranio facial anamolies:

These patients have narrowing of oropharynx, nasopharynx and nasal cavities. The may also additionally manifest with macroglossia. The various anamolies associated with respiratory difficulties are:

Pierre Robin syndrome

Treacher collin syndrome

Apert's syndrome

Cruzon's syndrome

Mobieus syndrome

c. Macroglossia :

Beckwith Wiedemann syndrome

Down's syndrome

d. Tumors:

Hemangioma

Neuroblastoma

e. Laryngomalacia : Is caused by an excessiely elastic cartilagenous support to the airway seen in infants. This commonly affects the glottic and supra glottic airway of infants. This excessively soft and elastic cartilage causes inspiratory collapse of the arytenoid, aryepiglottic folds and epiglottis during inspiration. The omega shaped epiglottis seen often in the infants adds to the problem. This causes occlusion of the laryngeal inlet. These patients have inspiratory stridor which becomes better on prone position or when the child is calm. Stridor is worsened if the child is restless or excited.

The cry of the child is usually normal. The child may also have aspiration and feeding difficulties. It is commonly seen during the first few months of life.

Direct laryngoscopy shows indrawing and falling forwards of the arytenoid and the aryepiglottic folds. The epiglottis may be infolded.

This condition may be managed conservatively, as the cartilage in infants tend to become stiffer as the child grows. In difficult cases the patient may be subjected to tracheostomy to secure the airway and to prevent aspiration, and feeding gastrostomy to maintain the nutritional status of the child. Epiglottoplasty may be considered in resistant cases.

2. Glottic causes:

a. Vocal cord palsy : Is one of the commonest cause of airway obstruction. In 80% of patients it is unilateral.

Etiology: Could be caused due to injury to vagus nerve at the level of Nucleus ambiguus - it is often bilateral.

Injury to the left recurrent laryngeal nerve due to cardio vascular causes and thoracic causes.

It could be caused due to increased intracranial pressure - i.e. Meningomyelocoele with Arnold Chiari malformation.

Clinical features: Inspiratory stridor at birth

Weak, hoarse cry or aphonia.

If unilateral the patient feel better when placed on the side of the lesion.

Investigation:

Direct laryngoscopy under local anaesthesia.

Conservative management.

Reduction of elevagted intracranial tension.

In bilateral palsy tracheostomy is indicated.

b. Tumors :

Papilloma

Hemangioma

Cystic hygroma

Laryngoceles

c. Atresia

d. Webs: are caused due to failure of recanalisation of the larynx . It can range between a complete occlusion by mucosa and submucous tissue or partial occlusion by a thin membranous web. It can occur in supraglottis, glottis and subglottis area. Commonly it is seen in the glottic area. It occurs in one in 10,000 live births.

Stridor is inspiratory in nature and is present from birth. The degree of airway obstruction depends on the extent of the web. The cry is weak or absent because of fixity of the cord. Symptoms are not positional in nature.

Treatment:

Tracheostomy may be life saving.

Perforation of the web.

Dilatation of the web.

Cryo surgery.

Laser surgery

e. Trauma

3. Subglottic causes:

Stenosis - congenital / acquired : is the most common cause of neonatal airway obstruction. It may either be congenital or acquired. Subglottic area is the narrowest portion of neonatal airway. Even a 1mm mucosal oedema in this area is sufficient to reduced the circumference by 1/3 compromising the airway. Congenital stenosis is more common in male children. Acquired subglottic stenosis is due to prolonged intubation, insertion of a large endotracheal tube etc. These patients present with inspiratory and expiratory stridor (to & fro stridor).

This condition is diagnosed by performing an endoscopy.

Treatment:

Tracheostomy may be life saving.

Dilatation

Laser luminisation

Anterior cricoid split

Resection of the stenotic segment with reconstruction

Webs

Atresia

Tumors like hemangioma & cystic hygroma.

Subglottic hemangioma is may be asymptomatic at birth. As it grows it may produce symptoms at a later date. These children

manifest with stridor, change in voice is possible if the tumor involves the under surface of the vocal cord. These children become symptomatic by 6 months. Female child is more commonly affected than males. Subcutaneous hemangiomas may also be seen in 50% of these children.

Xray soft tissue lateral view may show an eccentric swelling in the subglottic region. Endoscopy is diagnostic.

Management:

Tracheostomy is indicated to tide over acute crisis

Steroids may be prescribed to reduce subglottic oedema.

Laser excision

Cryotherapy

4. Tracheal causes:

Tracheomalacia : More or less similar to laryngomalacia. The stridor produced is expiratory in nature.

Stenosis

Cyst

Atresia : Often fatal at birth. It is also associated with other congenital abnormalities.

5. Extrinsic causes:

Thyroid swelling

Vascular rings : Compression of trachea and oesophagus due to abnormalities in the development of great vessels. It could be due to right sided aortic arch, double aortic arch, anomalous right subclavian artery, or aberrent left pulmonary artery.

Dyspnoea is present at birth, becomes worse on neck extension. Stridor is expiratory in nature.

Diagnosis is by:

Endoscopy

Arteriograms

Contrast CT scans

Hemangioma

Cystic hygroma

Teratoma

Mediastinal masses

Types of tracheostomy:

Permanent: Also known as end tracheostomy. This is performed by exteriorizing the trachea. This procedure is performed in patients undergoing total laryngectomy.

Temporary tracheostomy: As the name suggests this procedure is purely temporary, usually performed to enable the patient to get over the acute respiratory crisis. Depending on the position of tracheal opening this procedure can be classified into High and Low trachestomy.

Percutaneous tracheostomy: This procedure is performed using specially designed kit. A needle is used to perforate the anterior wall of trachea, this opening is dilated and the tracheostomy tube is introduced via the dilated opening. This procedure is usually performed in Intensive care unit settings.

Minitracheostomy: Here a small canula is passed through an opening made in the cricothryoid membrane. A separate mini tracheostomy kit is available for performing this procedure.

The kit contains:

1. A special scalpel
2. Canula

3. Obturator
4. Suction tube
5. A tape to anchor the tube

Cricothyrotomy: Is not performed commonly nowadays because of high incidence of postoperative subglottic stenosis. This procedure is indicated only under extremely emergency conditions.
Cricothyroid membrane is incised through vertical incision and tracheostomy tube is inserted through it. Ideally these patients must be converted into a regular tracheostomy within 48 hours.

Surgical procedure:

Anaesthesia: Under emergency situations it is performed under local infiltration anaesthesia. Under elective conditions it is performed under general anaesthesia.

Position: Supine with neck hyperextended.

Incision: Emergency tracheostomy is performed with a vertical incision extending from the lower border of cricoid cartilage up to 2cm above supra sternal notch. This area is also known as the Burn's space and is devoid of deep cervical fascia.

Elective tracheostomy is performed through a horizontal incision at 2cm above the sternal notch.

If performed under emergency settings local anaesthesia is prefered. The drug used is 2 % xylocaine with 1 in 100000 adrenaline. 2 ml of this solution is infiltrated in to the Burns space.

Through a vertical incision extending from the lower border of cricoid cartilage up to 2cm above the sternal notch theskin, platysma, and cervical fascia are incised. Branches of anterior jugular vein if any are ligated and divided. Sternohyoid and Sternothyroid muscles are retracted using langenbachs retractors. The anterior wall of trachea is exposed after splitting the pretracheal fascia. The tracheal rings are clearly identified. Few drops of 2% xylocaine is instilled into the trachea through a syringe. This process serves to desensitise the tracheal mucosa while it is being incised. Incision over the trachea is sited between the second and the third tracheal rings. If the tracheostome is planned for a long duration then it is better to excise a portion of the tracheal ring completely. If tracheostomy is planned for a short duration of less than a month then the cartilage is not completely removed but partially excised creating a flap based either superiorly or inferiorly. This is known as the Bjork flap. This flap can be sutured to the skin to keep the tracheostome open. Trahceostomy tube is inserted into the opening and the wound is closed with silk.

A wet gauze is placed over the tracheostome in order to moisturise the inspired air.

If the patient is to be connected to a ventilator, then a portex tube is used. If the tracheostomy is performed to releive acute airway obstruction then a metal tracheostomy tube like the Fuller or Jackson is preferred.

Complications of tracheostomy:

1. Injury to thryoid isthumus causing troublesome bleeding
2. Too lateral dissection may cause extensive bleeding and possible injury to recurrent laryngeal nerve.
3. Injury to the apex of the lung (right)

4. Sudden apnoea when the trachea is opened, due to loss of hypoxic respiratory drive. This can be prevented by slow opening of the trachea, or by subjecting the patient to inhage carbogen a mixture of carbondioxide and oxygen.

5. Subcutaneous emphysema if pretracheal fascia is not dissected properly, or too small a tube is introduced into the tracheostome.

6. Injury to great vessels. This can occur in children.

II. Write briefly on:

a. Submucous fibrosis: Oral Submucosal fibrosis is a chronic debilitating potentially malignant condition of the oral cavity associated with betel nut chewing. It is characterized by fibrosis of the oral soft tissue, resulting in marked rigidity and inability to open the mouth. The inability to open the mouth is slowly progressive in nature.

Pathophysiology:

Buccal mucosa was the most commonly involved site, but no part of the oral cavity is immune to this condition. Almost all the patients were pan chewers. Pathophysiology of this disease is not well established. A number of factors trigger the disease process by causing a juxtaepithelial inflammatory reaction in the oral mucosa. Factors such as areca nut chewing, ingestion of chilies, genetic and immunologic processes, nutritional deficiencies can lead to this condition.

Betel nut chewing: The areca nut component used along with betel leaf has been implicated. A recent study has clearly demonstrated

the dose and frequency relationship of areca nut chewing in the pathogenesis of this disorder. Arecoline the active ingredient of areca nut is known to stimulate fibroblasts to increase its production of collagen by 150%. Flavinoids, catechin and tannin present in betel nuts cause collagen to cross link making them less susceptible to collagenase enzyme degradation. The disease process of Submucosal fibrosis is active even after cessation of betel nut chewing suggesting that arecoline not only affects the fibroblasts it also affects gene expression in fibroblasts causing them to produce increased amount of collagen with intense cross linkages.

Studies have also shown that arecoline inhibits metalloproteinases (particularly metalloproteinase 2) thus decreasing the overall breakdown of tissue collagen. Studies have also shown that keratinocyte growth factor-1, insulin like growth factor-1, and interleukin 6 expression, which have all been implicated in tissue fibrogenesis, were also significantly up-regulated by arecoline. Areca nuts are also rich in copper content. Chewing areca nuts increases the amount of copper in the oral cavity fluids. Copper is known to stimulate fibrinogenesis by activating copper dependent lysyl oxidase activity (suggested by Trivedi).

Ingestion of chillies in the pathophysiology has been controversial. Capsaicin the active ingredient of chillies have been demonstrated to increase the level of fibrosis in rats.

Immunologic process: have also been implicated in the pathophysiology of submucosal fibrosis. Increased levels of CD4 components have been demonstrated in these patients. The number of langerhan giant cells in the site have shown an increase.

Nutritional deficiencies: Iron deficiency, vitamin B complex deficiency, and zinc deficiency have also been postulated as

predisposing factors. Infact these nutritional elements are necessary for normal repair mechanism to repair the oral mucosa which is constantly traumatized.

The morbidity and mortality of this condition is due to the fact that the patient is unable to open the mouth and consume normal quantities of food.

In Indian subcontinent females out number males in the incidence of this disease. No age group is immune to this condition. In Indian conditions in addition to the irritant effects of arecoline the nutritional deficiencies also play an important role in the pathogenesis of this disorder.

Clinical features:

1. Progressive inability to open the mouth due to fibrosis and scarring

2. Oral pain and burning sensation when the patient consumes spicy food

3. Increased salivation

4. Change in taste

5. Secretary otitis media due to stenosis of the pharyngeal end of Eustachian tube

6. Dryness of mouth

7. Dysphagia when consuming solids if esophagus is involved

Treatment:

Medical: Weekly Submucosal intralesional injections of steroids may help in prevention of progression of the disease. Cessation of betel nut chewing is a must. Placental extracts can be injected intra lesionally to reduce the inflammatory effects of the disease. Use of topical hyaluronidase in doses of 150 units in association with steroids has proved beneficial.

Intralesional injection of IFN-gamma has a role due to the immuno regulatory effect of the molecule.

Surgical management is reserved only for advanced cases with severe trismus. This include excision of fibrous bands, with split thickness skin grafting.

b. Cochlear implant: This is a surgically implanted electronic device that provides a sense of sound to a person who is suffering from profound sensorineural hearing loss. Cochlear implant is also known as the Bionic ear.

Parts of cochlear implant:

External unit – contains microphones, speech processor and a transmitter.

Internal unit contains receiver stimulator and 24 micro electrodes which is introduced into the cochlea via the round window membrane. These electrodes coil inside the cochlea. Processed sound signals from the transmitter reaches the electrodes placed in the inner ear via electromagentic induction.

Patient selection criteria:

1. Congenital deafness
2. Acquired profound deafness above 80dB sensorineural
3. Postlingual profound deafness following meningitis

   Surgical steps:

   Creation of wide post auricular flap for safe placement of implant over mastoid bone. A bed is created in the temporo-occipital bone for revceiver stimulation.

   Posterior tympanotomy to approach the round window niche.

   Widening the round window opening (cochleostomy).

   Insertion of electrode array through cochleostomy into the basal turn of cochlea.

Closure of cochleostomy with fibrofatty tissue to prevent CSF leak.

c. Vocal nodule:

Synonyms: Singer's nodule, Teacher's nodule.

This disorder frequently affects children and adults. In children it appears as spindle shaped thickenings of the edges of the vocal cords, whereas in adults they appear as more localised thickenings, varying from small points - nodules. These nodules typically appear at the junction of the anterior and middle 1/3 of the vocal cords. They appear almost aways symmetrically.

Pathophysiology:

Are caused by a combination of overtaxing and incorrect use of the voice. This is also aggravated by the presence of infections in the para nasal sinuses, tonsils, and adenoids. Patients with habitual dysphonia frequently encounter this condition. This condition can be effectively prevented or cured by voice rest or by using the voice properly. Infact the nodules can appear and disappear in a matter of weeks. If the aggravating factors persist for a long time then these nodules become permanent.

Stages of vocal nodule formation:

Stage of transudation:

Oedema occurs in the submucosal plane in this stage. This occur during the acute phase of the disorder. This stage is reversible in nature and may become normal on giving voice rest.

Stage of ingrowth of vessels:

In this stage neovascularisation of the area occur. This phase is also reversible, but takes a long time to become normal.

Stage of fibrous organisation:

In this stage the transudate in the submucosal area is replaced by fibrinous material. This stage is more or less resistant to conservative line of management.

These stages can be clearly observed by laryngoscopy under stroboscopic light. Local oedematous swelling of recent onset vibrates in phase with the whole vocal fold, whereas an older and more fibrous swelling can impede the vibrations so much that only a part of the cord is seen to vibrate. The improvement in the vibration pattern or signs of recovery are picked up early during stroboscopic examination.

Clinical features:

1. Change in voice

2. Fatiguability of voice

3. Decreased pitch range

Management:

1. Voice rest plays a sheetanchor role in the management of vocal nodule. This may range from complete voice rest to partial rest.

2. Speech therapy will help patients with habitual dysphonia from developing vocal nodule.

3. Treatment of sinus infections, tonsillitis and adenoiditis must not be ovelooked.

4. If the vocal nodule bceome permanent then microlaryngeal removal is advocated.

d. Rinne's test: is a tuning fork test used to clinically test hearing deficiencies in patients. It is designed to compare air conduction with bone conduction thresholds. Under normal circumstances, air conduction is better than bone conduction. Ideally 512 tuning fork is used. It should be struck against the elbow or knee of the patient to vibrate. While striking care must be taken that the strike is made at the junction of the upper 1/3 and lower 2/3 of the fork. This is the maximum vibratory area of the tuning fork. It should not be struck against metallic object because it can cause overtones. As soon as the fork starts to vibrate it is placed at the mastoid process of the patient. The patient is advised to signal when he stops hearing the sound. As soon as the patient signals that he is unable to hear the fork anymore the vibrating fork is transferred immediatly just close to the external auditory canal and is held in such a way that the vibratory prongs vibrate parallel to the acoustic axis. In patients with normal hearing he should be able to hear the fork as soon as it is transferred to the front of the ear. This result is known as Positive rinne test. (Air conduction is better than bone conduction). In case of conductive deafness the patient will not be able to hear the fork as soon as it is transferred to the front of the ear (Bone conduction is better than air conduction). This is known as negative Rinne. It occurs in conductive deafness. This test is performed in both the ears.

If the patient is suffering from profound unilateral deafness then the sound will still be heard through the opposite ear this condition leads to a false positive rinne.

e. Rhinoscleroma: This is a chronic granulomatous condition of the nose and upper airway. This condition is caused by infection by the bacilli Klebsiella rhinoscleromatis.

It is also known as Firsch bacilli (as an honor to von Firsh who described this organism).

Pathophysiology: This condition is contracted by direct inhalation of droplets / contaminated material. This infection usually prefers transitional epithelial zones (like vestibule of nose where the squamous epithelium changes in to ciliated columnar epithelium and supraglottic area of larynx).

Clinical features:

1. Nasal obstruction
2. Rhinorrhoea
3. Epistaxis
4. Nasal deformity
5. Anesthesia of palate
6. Anosmia

Classically this condition passes through three stages:

Catarrhal stage: This initial stage begins with non specific rhinitis progressing on to formation of foul smelling thick discharge and crusting of the nasal mucosa.

Granulomatous stage: This stage is also known as hyperplastic stage. Nasal mucosa shows rubbery nodules / polypoidal lesion. Epistaxis may be present. The nasal cavity appears enlarged with destruction of nasal cartilages. Involvement of maxillary antrum is common in this stage and may form the reservoir.

The soft palate appears to be thickened at its junction with that of hard palate. If the lesion presents like a tumor with evidence of tissue destruction it is known as pseudotumerous rhinoscleroma.

Sclerotic stage: This stage is characterised by sclerosis and fibrosis. In this stage the nodules present in the nasal mucosa

gets replaced by fibrous tissue.

Management: Antibiotics. Tetracycline happens to be the drug of choice. Ciprofloxacillin and rifampicin have also found to be effective.

Steroids can be administered inorder to prevent excessive scarring of tissues.

Surgery is indicated in patients with extensive scarring of the nose.

(5 x 5 = 25 marks)