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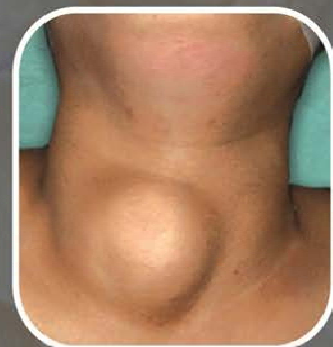
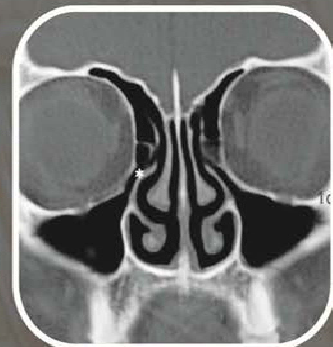
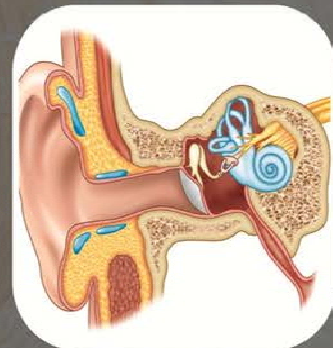


Clinical Atlas of ENT AND HEAD & NECK DISEASES

PS Saharia
Deepti Sinha

Foreword
Prem Kakar

JAYPEE



**Clinical Atlas of
ENT AND HEAD & NECK DISEASES**

Clinical Atlas of ENT AND HEAD & NECK DISEASES

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Foreword

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JAYPEE BROTHERS MEDICAL PUBLISHERS (P) LTD.

New Delhi • London • Philadelphia • Panama



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Clinical Atlas of ENT and Head & Neck Diseases

First Edition : 2013

ISBN 978-93-5025-115-7

Printed at

Dedicated to

*Patients and
All those working towards
the expansion of knowledge and self
and
My parents*

—PS Saharia

*My father and mother for being my guiding light
Raj for being the wind beneath my wings
Adi for my second childhood
Neeti and Preeti for being my friends*

—Deepti Sinha

Foreword

Teaching is an art where few indulge with heart and mind to make it reach the recipient—this is an attempt to achieve the same. This atlas has combined the clinics with a classroom teaching. The atlas constitutes the teaching program for the undergraduates and postgraduates. The atlas shall help teachers, when they use this atlas as a basic and add their experiences to communicate with the students. The atlas is an asset when used with any textbook to get a complete idea of the disease and its management. An accompanying DVD fulfills this aspect as well.



Dr PS Saharia's burning desire to compile this, started at the beginning of career when he had collected invaluable clinical material from busy hospitals. The clinical material has been further supplemented, by an upcoming equally talented young ENT consultant (Dr Deepti Sinha) to bring out a balanced atlas.

The atlas has been well presented in 7 chapters; and it is descriptive for disease affecting the region with photographs of patients, investigations and treatments, etc.

A picture lives up to the adage that it is worth a thousand words.

It represents the main stream of ENT thoughts and covers diseases. Increasing scope of the specialty, where only few can actually be in touch with all its aspects, the authors have not hesitated in including contributions from some distinguished ENT and allied subject physicians.

Some of the internationally known personalities in the specified fields have provided remarks to render the atlas a different status, which shall go a long way in dispersion of the knowledge, far and wide.

Prem Kakar

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Formerly

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Expert Comments

It is with a great pleasure to read the *Clinical Atlas of ENT and Head & Neck Diseases* authored by Dr PS Saharia and Dr Deepti Sinha. It is truly a well-written, easy-to-read and comprehensive atlas covering almost every aspect of various ailments of the diseases of the ear, nose, throat and variety of conditions affecting the head and neck. It is distilled following a wealth of experience of a senior author as a clinician and a teacher over many years.



Although an atlas, it covers across the board relevant aspects of anatomy, physiology, pathology and investigations. I am pleased to see the importance of certain basic clinical examinations in ENT and Head & Neck surgery, where necessary, the authors have used modern equipment with photographs to illustrate the points.

They, where required, make a point to elucidate investigations, such as X-rays, computed tomography (CT), magnetic resonance imaging (MRI) contrast studies; and in the section of otology, various audiograms, tympanograms, otoacoustic emissions, auditory brainstem response (ABR) and electronystagmogram (ENG). The value of tables in all chapters cannot be overemphasized. The illustrations are indeed, plentiful, making it effortless to read and understand the subject.

I have no doubt, the atlas will be an all-embracing, not only to the senior medical students but also a great teaching tool for the novice who has just started ENT residency, as they are likely to come across the different types of patients in practice. The DVD is a welcome addition to the already prolific illustrations and diagrams seen throughout the atlas.

The advent of the atlas will create a clear understanding in the management of patients for the reader.

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Expert Comments

A clear understanding of anatomy and the application of this knowledge is the key to being a successful sinus surgeon. The chapter on Nose, clearly and successfully describes the key concepts of nasal anatomy, basic clinical examinations and proceeds to graphically demonstrate the key pathologies that occur in the nose. It represents a life time of clinical experience, and gives an excellent overview of the myriad of conditions that can afflict the nose. This chapter is a vital description to the neophyte as well as an invaluable resource to the practising surgeon. I would urge the reader to study it well.



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Expert Comments

It has been very satisfying for me to witness the development and refinement of otolaryngological techniques and improved patient care across the globe over the past few decades. This has been accomplished through sharing of knowledge regarding human anatomy and physiology, diagnosis of diseases, and treatment modalities including surgical intervention and techniques. For teachers and students alike, learning is indeed a lifelong process, and nowhere is this more apparent than in the advancing fields of otolaryngology and facial plastic surgery. Dr PS Saharia and Dr Deepti Sinha have provided splendid examples of the diagnosis of severe nasal deformities corrected with the septorhinoplasty approach.



In the atlas, the authors have presented the reader with an array of valuable information, which has been collected over one's professional life-time and has been brought current with insights into recent advances in the field of otolaryngology. The drawings, photographs, and accompanying DVD are critical part of modern-day learning and for the proper understanding of complicated anatomy. Ear, nose, and throat diagnosis and treatment can become second nature and no longer a mystery. Practitioners who follow the dictates of the atlas will have better outcomes in the management of their patients, and this is important in today's healthcare delivery system. It is intended for those desiring to advance their knowledge and clinical experience.

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Preface

Every physician will make and ought to make observations from his own experience, but he will be able to make a better judgment a juster observation by comparing what he reads and what he sees together.

—William Osler (1904)

What our eyes see, our brain registers and provides a lifelong impression. This has been the basis for creating this much-needed *Clinical Atlas of ENT and Head & Neck Diseases*. It is a collection from the busiest hospitals, which are visited by thousands of patients everyday.

The atlas covers ENT curriculum and can be utilized as a companion to ENT textbook. A DVD version of the same has been incorporated with the atlas, for utilizing it in the classroom for teaching.

It is a comprehensive atlas showing the patients charts, histopathology, specimen, instruments, etc. and the diseases affecting ear, nose, throat, head and neck region.

PS Saharia
Deepti Sinha

Acknowledgments

We sincerely acknowledge the contribution of photographs from the experience of ENT surgeons: Dr Ashish K Lahiri, Dr Ashok Vaid, Dr Devinder Rai, Dr Sudhir Bahadur, Dr Anil K Monga, Dr Sowrabh K Arora, Dr Shalabh Sharma, Dr Bhavin Parikh, Dr Mukul Gupta, Dr Nishi Gupta and Dr Nitin Aggarwal. We are also thankful to Dr AK Khurana (Gastroenterologist), Dr Raj Anand (Oculoplastic surgeon) and Dr Nandini Vasdev (Pathologist), for their valuable contributions.

We are thankful to Dr Ashish Shrivastava and Dr Geeta Shrivastava for their help in formulating the slides into digital format.

We take this opportunity to express our thanks to the management of Max Hospitals, where this venture has taken a final shape with special thanks to Ms Sangeeta Jain who supported us with typing work.

Our acknowledgments to Shri Jitendar P Vij (Group Chairman), Mr Ankit Vij (Managing Director), Mr Tarun Duneja (Director-Publishing), Mr KK Raman (Production Manager), Mrs Samina Khan (PA to the Director-Publishing), Mr Sunil Kumar Dogra (Production Executive), Mr Neelambar Pant (Production Coordinator), Mr Bharat Bhushan (Typesetter), Dr Mohd Naved (Senior Proofreader), Mr Manoj Pahuja (Senior Graphic Designer), Mr Santosh Kumar Pandit (Graphic Designer) and other staff of M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India, for their untiring efforts to bring out the clinical experience into atlas.

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Septoplasty and Septorhinoplasty Manual



Dr PS Saharia

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NASAL ANALYSIS

as practiced by
Dr PS Saharia

Better results, after a functional and cosmetic surgery of the nose can be achieved by a proper preoperative anatomical and physiological analysis of the nasal septum and the various constituents of the superstructure of the nose. A way to analyze the nose has been described.

Nasal analysis is the foundation of a successful Septorhinoplasty and thus the technique of analysis should be perfected by all the surgeons undertaking Septorhinoplasty for better results. The surgeon should develop a sense of esthetics which is based on his own experience and this ensures better sense of proportion of the nose on the face, comprising of forehead, nose, cheekbones and the chin. One should be able to visualize the results of surgery on the face after each surgical procedure is done to modify the shape of the nose.

A successful surgeon should understand his personal and the surgical limitation and modify the procedures accordingly to provide better results. An evaluation of the results on several patients will help crystallize one's own technique. It is also important to inform the patient about the surgical procedure and its outcome particularly in case with severely crooked nose or a badly deviated septum. A possibility of revision surgery, which may vary between 5% and 15% of cases, should be mentioned to the patient. Highly sensitive and emotionally imbalanced patients

should better be avoided. It is better to enquire from the patient his expectations out of the surgery and make a note along with your remarks about the expected along with your remarks about the expected results.

The analysis of the nose is necessary to understand the deformity and to envisage a plan for the surgical procedure.

It is a matter of observation that a patient with deviated nasal septum invariably has nasal blockage of the opposite passage and one looks by instinct for hypertrophic turbinate or high septal deviation on the other side. Over a period of time surgeons engaged in doing Septorhinoplasty do develop the clinical sense of correlating the external deformity of the nose with the underlying anatomical placement of the various component of the superstructure of the nose.

The external nose which is covered with skin of variable thickness and quality is thin and mobile over the glabella and rhinion and thick and adherent over the tip. It covers from above downwards, a pair of frontal process of maxilla and nasal bones, upper alar cartilages and lower alar cartilages. If we visualize from right to the left the (R) frontal process of maxilla and nasal bone, upper alar cartilage, lower alar cartilage with its medial crura, followed in the midline is the nasal septum and then the similar components on the other side.

The inside of the nose has cartilaginous nasal septum in the midline and this is in continuity with the upper alar cartilaginous the processus dorsalis. Its inferior border rests in the groove of the maxillary crest and is caudally related to the anterior nasal spine and the medial crura of the lower alar cartilages.

The nasal blockage is caused because of deviation, spur dislocation or fracture of the septum. The septum due to its displacement from the anatomical position could also produce columellar deformity or external nasal deviation.

The nasal blockage may also be because of a narrowed or collapsed nasal valve area. The areas bounded by lateral crura, lateral wall of the vestibule, anterior part of the floor of the vestibule, anterior part of the floor of the nostril, the septum and the septoalar angle contribute to the formation of the nasal valve.

The diagrammatic picture of the nasal superstructure should mentally be adopted to get the clarity of concept, as each component produces deformity by its own shape and thus needs to be dealt with separately.

The lower alar cartilages contribute to the formation of the lobule of the nose, tip and columella. Wider lateral crura adds to the bulk of the lobule and small medial one may get a bifid tip deformity, whereas excessive fibrofatty tissue adds to the build of the lobule. The upper alar cartilages which are in contact with the nasal septum medially are caudally related to the lower alar cartilages and cephalically lie underneath the nasal bones. They contribute to the width of the lateral wall, prominence of the dorsum or a hump.

The nasal bones and the frontal process of the maxilla provide the shape or deformity of the upper one-third of the nose and is

responsible for a broad or humped nose or a variable frontonasal angle.

A bulky procerus or an over acting depressor labii superioris muscle do provide a full frontonasal angle or the depression of the tip of the nose on smiling and should be dealt with accordingly.

Now to analyse, say a commonly seen "C" shaped deformity of the left. The nasal along with the frontal process of maxilla are shifted to the left of the midline, the upper alar cartilages accordingly are curved at the mid point having upper part to the left and lower to right, probably due to curved nasal septum, and lower alar cartilage points to the right side. The caudal margin of the septum lies in the right its body lies prominently in the left nostril.

Thus anatomically analyzing the nose eases the process of Septorhinoplasty.

DISCUSSION

The analysis of the nose, as recommended by Anderson (1985) provides scores for the various landmarks and thus help to organize record for the nasal deformity, surgery and the postoperative result, (Refer chart 1).

Saharia (1988) had developed a scoring chart for the nasal septal surgery which provides details for the nasal septal defects and its surgery. Functional aspect of the nasal airway is also taken care of in the chart, (Refer chart 2). Combining the two charts will ensure better records for the cases of Septorhinoplasty. These charts have particular importance in the era of CPA. The scoring technique would help to evaluate the cases a little better than the visual impact alone. One needs to have an "Anatomical Eye" on the nose to use these chart.

Chart 1: Rhinoplasty scale

Rhinoplasty scale (100)

Name _____ Surgery date _____ Date of study _____

<i>Frontal view</i>		<i>Lateral view</i>	
	<i>Pre</i>	<i>Post</i>	
<i>Nasal dorsum</i>			<i>Nasal dorsum</i>
<i>Deviation from midline</i>			<i>Pre</i>
<i>Bony portion (2)</i>			<i>Post</i>
<i>Cartilaginous portion (2)</i>			<i>Nasal dorsum</i>
<i>Width</i>			<i>Nasofrontal angle depth (2)</i>
<i>Radix area (2)</i>			<i>Nasal dorsum (not lobule)</i>
<i>Bony portion (2)</i>			<i>Convexity</i>
<i>Cartilaginous portion (2)</i>			<i>Bony portion (2)</i>
<i>Depressions</i>			<i>Cartilaginous portion (2)</i>
<i>Bony portion (1)</i>			<i>Soft tissue (1)</i>
<i>Cartilaginous portion (1)</i>			<i>Concavity</i>
Nasal Base			<i>Bony portion (2)</i>
<i>Width of lobule (2)</i>			<i>Cartilaginous portion (2)</i>
<i>Width of alae (2)</i>			<i>Absence supratip break (I)</i>
<i>Alar flare (2)</i>			<i>Projection of pyramid</i>
<i>Deviation of lobule (2)</i>			<i>Excessive (2)</i>
<i>Inequality alar levels (1)</i>			<i>Deficient (2)</i>
<i>Alar cartilages</i>			<i>Length of dorsum (including lobule)</i>
<i>Depression (1)</i>			<i>Excessive (2)</i>
<i>Discrete prominences (1)</i>			<i>Deficient (2)</i>
<i>Skin quality (1)</i>			Nasal Base
Lateral Nasal Walls			<i>Tip projection</i>
<i>Width of nasofacial angles</i>			<i>Excessive (2)</i>
<i>Upper third (2)</i>			<i>Deficient (2)</i>
<i>Middle third (2)</i>			<i>Columella</i>
<i>Lower third [alae not included (2)]</i>			<i>Hanging (2)</i>
<i>Depressions</i>			<i>Retracted (2)</i>
<i>Nasal bones (R-L) (1)</i>			<i>Columella-lip ratio (1)</i>
<i>UL cartilages (R-L) (1)</i>			<i>Level disparity medial-lateral crura (1)</i>
<i>Prominences</i>			<i>Caudal contour of nose</i>
<i>Nasal bones (R-L) (1)</i>			<i>Medial crura angulation (1)</i>
<i>UL cartilages (R-L) (1)</i>			<i>Too straight (1)</i>
Lobule			<i>Webbing nasolabial angle (1)</i>
<i>Height (2)</i>			<i>Notching of ala (1)</i>
<i>Configuration triangle (2)</i>			<i>Length facial plane of pyramid (2)</i>
<i>Dome asymmetries (2)</i>			Basal View
<i>Bifidity (2)</i>			Columella
<i>Knuckling (2)</i>			<i>Length (1)</i>
Nostrils			<i>Lobule height/length ratio (1)</i>
<i>Shape (2)</i>			<i>Deviation (2)</i>
<i>Asymmetries (1)</i>			<i>Caudal septal distortion (2)</i>
<i>Long axis (2)</i>			<i>Splayed crural feet</i>
<i>Double sil (1)</i>			<i>Unilateral (1)</i>
			<i>Bilateral (1)</i>
			<i>Widening base (2)</i>
			<i>Bifidity (1)</i>
			<i>Scarring (1)</i>
			ALA
			<i>Thickened (1)</i>
			<i>Poor scanning (1)</i>

Preoperative score _____ Postoperative score _____

Chart 2: Saharia's septoplasty scale

Saharia's septoplasty scale (10)
(As per examination)

	<i>Preop</i>	<i>Postop</i>		<i>Preop</i>	<i>Postop</i>
A. Septal deformity			B. Septal perforation (Small/large)		
Septal spur (2)			C. Airways		
Septal deviation (2)		 mm right side (1)		
Fracture (2)		 mm left side (1)		
Dislocation (2)					
Total score preoperative/10 _____			Total score postoperative/10 _____		

Lastly a summary of the analysis can be written to organize the surgical procedures.

Nasal superstructure which rests on the face over the central pillar, the nasal septum, is so formed that each component carries its own importance in the modification of the shape of the nose. One has to get familiar with the detailed anatomy to undertake the surgery of Septorhinoplasty and for that a frequent visit to the departments of anatomy/mortuary is recommended.

Saharia's septoplasty chart provides two information, viz. type of septal deformity, and the status of the nasal airway (Septal deformity count for 8 marks, while 4 marks are for the nasal airways which can be measured by the size of the fog formation on a spatula). One should plan the surgical procedure and be ready for any consequence. It is good to develop one standard technique which can be modified accordingly for

each situation. Start the work on the nasal septum and correct its deformity. By making bilateral marginal incisions and intercartilaginous incisions the lower alar cartilages are delivered and the tip of the nose is modified. The tip is taken as the main landmark in modifying the shape of the nose. Thereafter work on the upper alar cartilages to narrow the nose or to reduce the hump. Bony hump is also reduced. Finally perform the osteotomies to narrow the upper one-third of the nose.

Septorhinoplasty means correction of the nasal septum, to clear the nasal blockage and the correction of the superstructure nasal deformity, to make the nose look more normal than fashionable on the face of the person and does not mean that all above said procedures are mandatory. Thus Septorhinoplasty is a procedure which is modern and scientific and is no less esthetic.

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SEPTOPLASTY

Technique as practiced by
Dr PS Saharia

Patients with deviated nasal septal defects may present with problems of nasal functions or nasal deformity and require surgical correction. A study of pathoanatomy of the nasal septum is necessary to understand the various septal defects and their surgical correction.

SURGICAL ANATOMY

Nasal septum is a partition and is mainly constituted by a septal cartilage, vomer and perpendicular plate of ethmoid. The mucoperichondrium covers the cartilage and supplies its' nutritional needs. Inferiorly the septal cartilage rests in the maxillary groove and they are separately covered by perichondrium and periosteum.

SEPTAL DEFECTS

They are classified as: (a) Septal spur: it has sharp lateral edge and superior and inferior surfaces. (b) Septal dislocation: the septum is dislocated from the maxillary groove and or peeps out of the columellar pocket (Fig. 1). (c) septal deformity of 'C' or S shapes (d) Fractured Septum: may cause the septum to be thick or get absorbed to enhance the membranous septum or even result in sagging of the tip of the nose (Fig. 2).

SEPTAL SURGERY

Principle of septoplasty has changed the concept of septal surgery. The septum no more acts as an autograft in its own bed. The important perichondrium which supplies the nutrition to the septum is preserved to the maximum extent.

SURGICAL PROCEDURE

Patients may be operated under local anesthesia with sedation or under general anesthesia. The nose is sprayed and lightly packed with 4% Xylocaine/Adrenaline lotion. The site is injected with 2% Xylocaine and 1:200,000 Adrenaline solution.

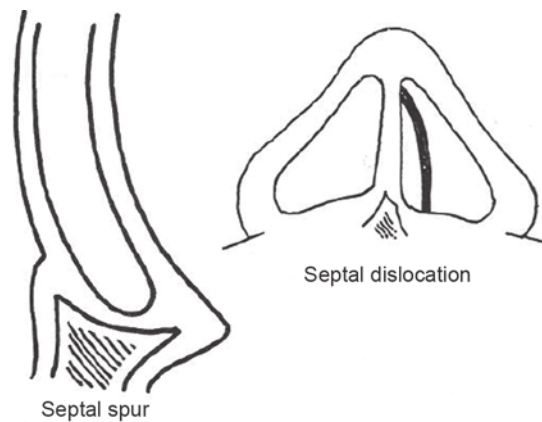


Fig. 1: Septal spur and septal dislocation

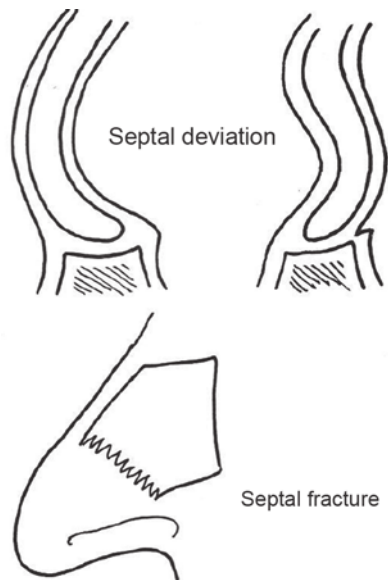


Fig. 2: Septal fracture

SPUR: A Bard Parker knife with 15 No. blade is used to make an incision along the margin or superior surface of the spur. The incision is extended from behind forward along the spur. The incision is extended upwards just behind the mucocutaneous junction. The superior border (or say lateral border) of the cartilage which is found lying out of the maxillary groove is incised along the length and the septum is pushed medially to the midline to reduce the dislocation. The inferior mucoperiosteal flap is reflected inferiorly, off the maxillary crest. The bony crest which forms the part of spur is chiseled out/or fractured to the midline to complete the procedure of spurectomy (Fig. 3).

If the nasal septum is also deviated, the mucoperichondrium is further reflected off the septal cartilage. The septal cartilage is incised at its posterior attachments with vomer and

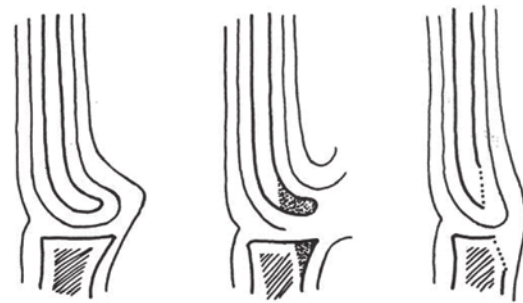


Fig. 3: Spurectomy

ethmoid right up to the nasal dorsum, with the help of the sharp edge of a curved perichondrial elevator. In cases where the septal deformity is associated with external deformity of the cartilaginous dorsum, bilateral extra mucosal paramedian incisions on the processus dorsalis of the nasal septum are made.

The overlying soft tissue is lifted off the dorsum through these incisions. This releases the septal cartilage from all its attachments except the mucoperichondrial cover of the other side and ensures the septum to be in the midline.

The bony septum is either resected submucosally or fractured extramucosally to ensure patency of the airway. The mucosal flaps are replaced with ease and if required one plain 3-zero catgut suture is applied to keep the flaps in position.

CAUDAL DISLOCATION

An incision is made over the mucocutaneous covering over the delicate caudal margin of the septum, keep the corresponding medial crura of the lower alar cartilage away from the incision. A pocket is created in the columella

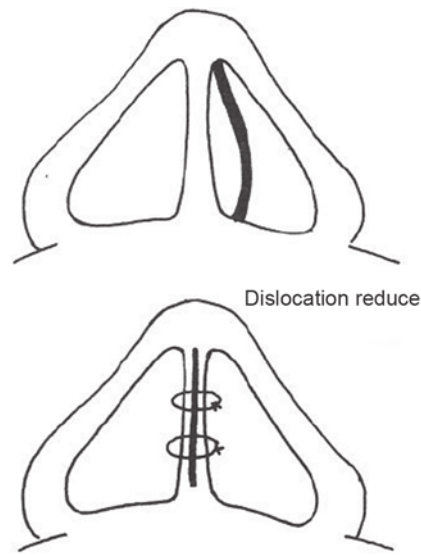


Fig. 4: Correction of caudal dislocation

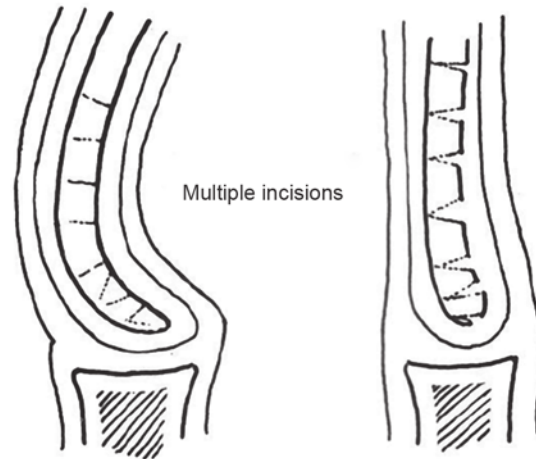


Fig. 5: Multiple incisions are made on concave side to break cartilage memory

by dissecting through the membranous septum and the two medial crurae. The caudal part of the septum is denuded of the perichondrium on either side. The inferior margin is separated by sharp dissection. The anterior nasal spine is fractured to the midline only if it interferes with the septal cartilage to come to the midline. To ensure its new position one 3-zero catgut stitch is applied (Fig. 4).

TRUE SEPTAL DEVIATION

As incision preferably on the concave side of the septal deviation, just behind mucocutaneous junction is made and extended posteriorly. The septal cartilage is exposed and multiple cartilage incision are made to break the spring. If the cartilage is large and does not lie in the maxillary groove, it could be trimmed along its inferior border or to break

the spring action a portion of the septal cartilage between the multiple incisions is taken out (Fig. 5).

FRACTURED OR DUPLICATED CARTILAGE

The blunted caudal margin of the fractured and duplicated septum is felt and incision is made over it. Bilateral mucoperichondrial flaps are carefully reflected to expose and release the fractured cartilage. The fracture is reduced by snipping the fibrous adhesions or by making necessary cuts on the cartilage. The mucoperichondrium flaps are reflected and columella is dissected to create a pocket. The pocket will keep the repaired septum in position, an additional, through and through, catgut stitch may be applied to ensure this. If it is not feasible then the fractured part of the

septum may be cut, trimmed or removed and replaced by a corresponding partial septal cartilage homograft (Fig. 6).

The nasal cavities are packed with antibiotics impregnated pack for 24 hours it could be replaced by applying a multiple through and through 3-zero catgut stitch which will keep the flaps/cartilage in position to provide a patent airway.

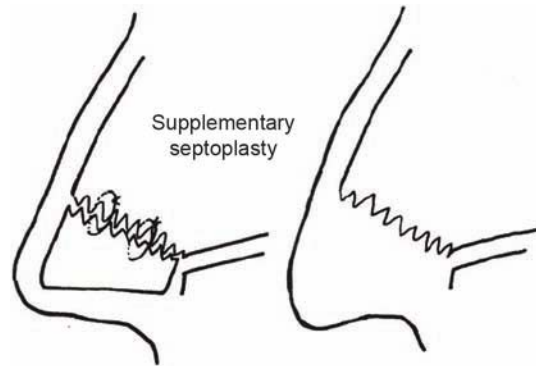


Fig. 6: Supplementary septoplasty

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SEPTORHINOPLASTY BASIC PROCEDURE

as practiced by
Dr PS Saharia

Rhinoplasty has reference in *Sushtra Samhita* 700 BC where a forehead flap was utilized for cosmetic reconstruction of the nose. Plastic reconstruction has since improved with the changing society, medical sciences and the modern techniques.

Septorhinoplasty is a procedure to improve the function and the shape of the nose according to the patient's desire and surgical feasibility. The patient must mention, as what is his problem, and what he would like to have and what he does not like of his nose. The surgeon should explain as to what is possible and what is not feasible.

PREOPERATIVE ASSESSMENT

Discuss the presenting problem, the deformity and the procedure with the patient.

Anterior rhinoscopy is done to assess the position of the septum, airway and the nasal mucosa.

External examination is done to ascertain the deformity of the nose and to define the anatomical defects for correction. This may be done according to a plan - (Anderson / Saharia Chart)

Preoperative photograph in profile, front and extended neck views, (Photograph) are

shot. One may utilize a digital camera and a computer to record the preoperative and postoperative results. This also helps to discuss the procedure with the patient (Figs 7A to D).

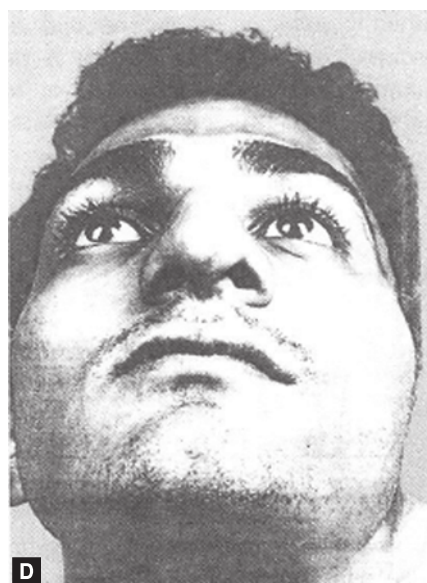
- A preanesthetic check-up is done
- A psychiatry check-up may also be required for some of the cases.
- Informed preoperative consent for Septorhinoplasty is mandatory.

A detailed proforma as suggested by Anderson/ Saharia to know the functional and physical deformities, is filled. The facial analysis and the nasal measurements may also be done.

SURGERY

Anesthesia

The operation is done either under local or general anesthesia. Premedicate the patient for general anesthesia and keep scope to use hypotensive drugs as well. It is important to fix the endotracheal tube in midline. This will avoid a pull to the angle of the mouth and distortion of the nose. An endolaryngeal packing is necessary. The patient should preferably be monitored during the course of surgery.



Figs 7A to D: Patient photographs

For local anesthesia, the patient is sedated about half an hour before the surgery. Initially 2% Xylocaine ribbon packing of the nose is done, then it is infiltrated with 2% Xylocaine with 1:80,000 adrenaline. Local infiltration is made at the points, demonstrated in the diagrams (Fig. 8). The best time to operate the case is after fifteen minutes of the infiltration. The vibrissae are trimmed from inside the nostrils with the help of the blunt ended scissors smeared with vaseline/or an ointment.

Positioning

Patient is operated in supine position with head raised by 15 degree. The head should rest on head ring on a head ring. Use a headlight and a toplight. Drape the patient's head to keep the triangle of interest exposed (Fig. 9).

PROCEDURE

Mark The Surgical Anatomy

It is a good habit to study the surgical anatomy of each case and mark the landmarks like

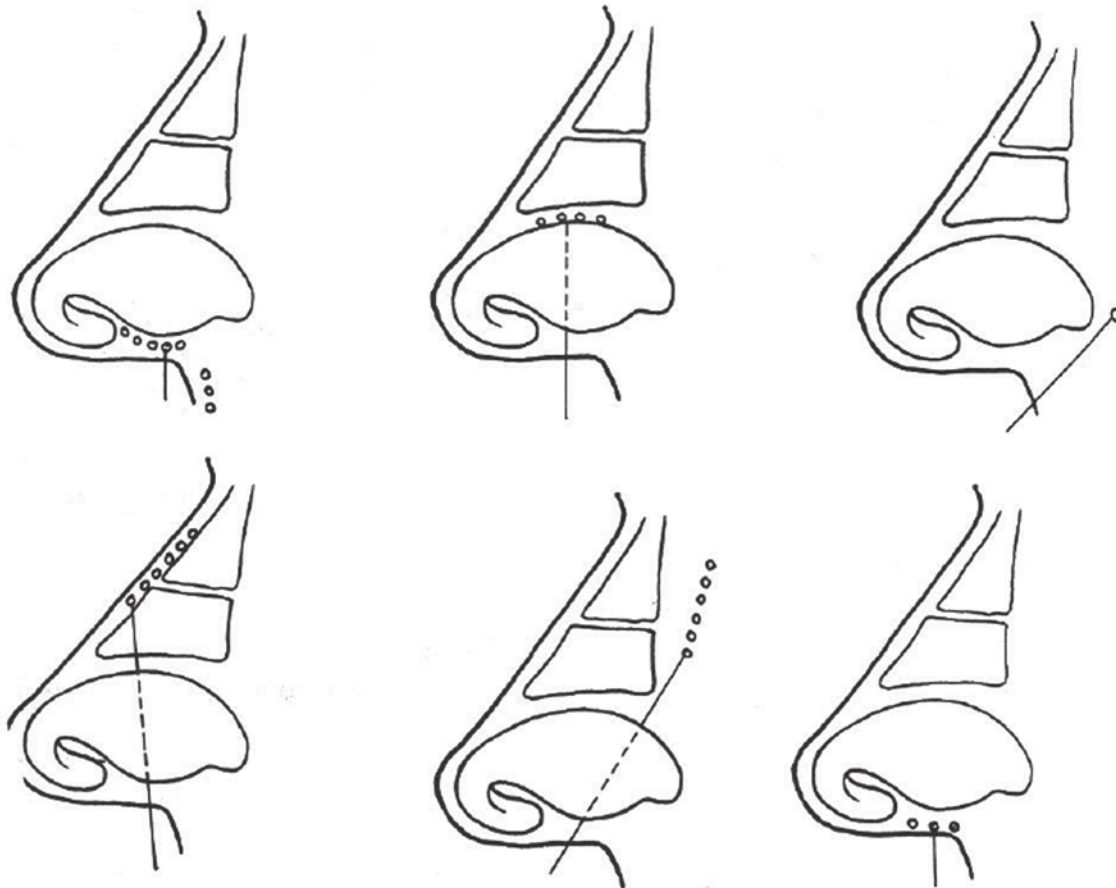


Fig. 8: Local infiltration of the anesthesia is done at the marked sites

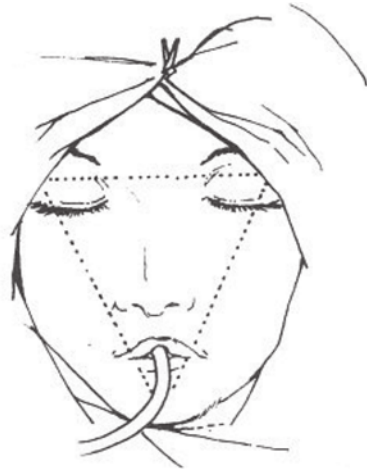


Fig. 9: The triangle of interest

lower alar cartilage, septal dorsum, upper alar cartilage, nasal bones, glabella and the portions of the cartilages and the bones to be trimmed to modify the shape of the nose, along with the dorsal margin of the nasal septum (Fig. 10).

CORRECT THE SEPTAL DEFORMITY

The septal defects which cause functional problem and/or the nasal deformity should be corrected first. It is freed from all the attachment, inferiorly from the maxillary groove, posteriorly from the attachment to the bony nasal septum, and dorsally from the upper alar cartilage. The caudal margin lies free and helps to provide the tip columellar angulation. This angle can be increased or decreased by modifying the caudal margin.

INITIAL INCISIONS

A marginal incision along the caudal border of the lower alar cartilage and medial crura is

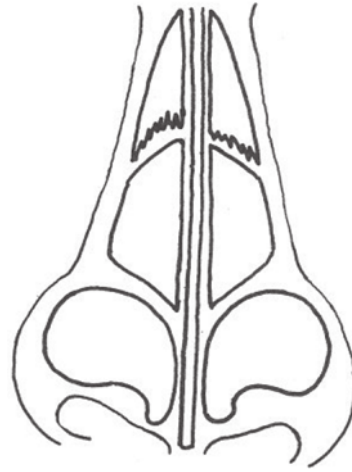


Fig. 10: Surface anatomy of nasal bone, ULC, LAC and septum are marked

made on either side. The subcutaneous tissue and the skin over the lower alar cartilages is elevated. An incision is made between the upper and the lower alar cartilage with No. 15 blade. It is continued medially along the caudal margin of the septum into the membranous septum and if combined on either side, it is a complete transfixion incision (Fig. 11).

LOWER ALAR CARTILAGE

After incisions along the caudal border (marginal incision) and cephalic border of the lower alar cartilage (intercartilagenous incision), the skin over the dorsal surface of the nose is elevated. The lower cartilage on both sides can now be delivered out with the help of a flat metallic instrument like perichondrial elevator (Fig. 12). To narrow the lobule and to rotate the tip the cephalic part of the lower alar cartilage is symmetrically removed on both side, leaving a 5 mm intact strip of caudal margin (Fig. 13). The two medial crura may be brought together and stitched with each other

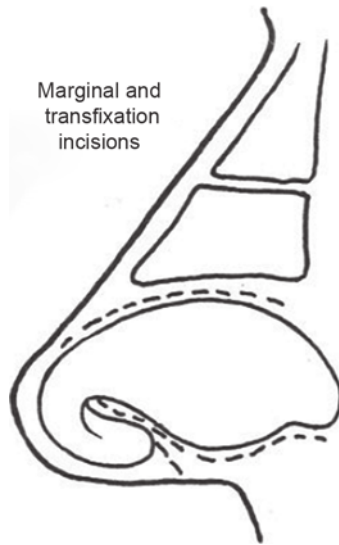


Fig. 11: Marginal and transfixation incisions

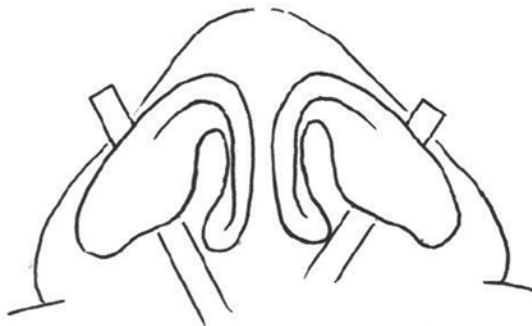


Fig. 12: Bucket handle delivery

to sharpen the tip (Figs 14A and B). To rotate the tip the caudal margin of the septum may be trimmed by a few millimeters, in an inverted equilateral triangle shape.

The surgery to modify the tip of the nose is based on the camera tripod concept. The lateral crurae of the lower alar cartilages from the two legs while combined two medial crurae form the third limb of the tripod. Reduction of

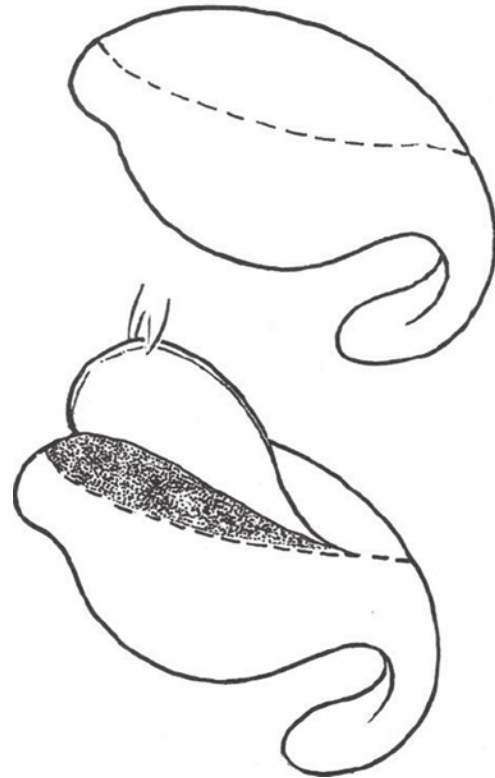


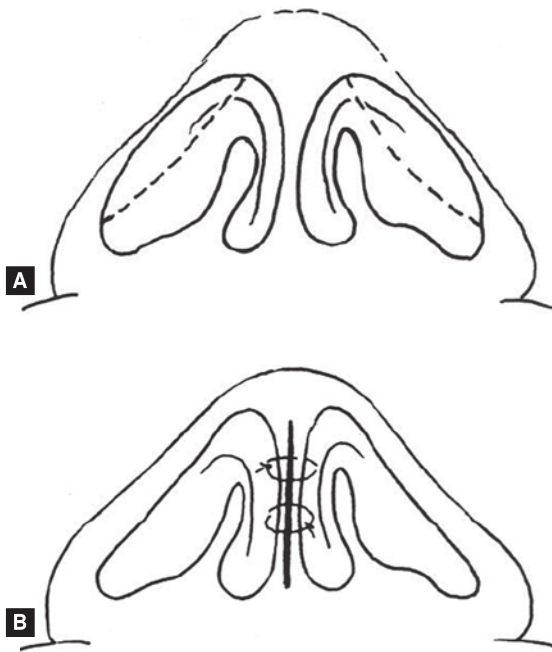
Fig. 13: Lower alar cartilage is rolled off the skin

enhancement in the size of any of the limbs will change the projection and rotation of the nasal tip.

Procedures which are commonly done to modify the nasal tip are complete strip removal, rim strip removal, dome morselization and Goldman tip procedures.

UPPER ALAR CARTILAGE

The upper alar cartilages are separated from the septum by fine scissors or by a knife. Once separated, appropriate size of the medial border of the upper lateral cartilage is cut to reduce the hump. The nasal septum may also be reduced to the same height (Fig. 15).



Figs 14A and B: (A) Complete strip technique for tip-plasty is done and the medial crurae are stitched together; (B) A support for medial crurae with cartilage

Examining the nose at this stage, one will realize that the cartilaginous part of the nose comprising of the lower alar cartilage, nasal septum and the upper alar cartilages has been narrowed and a distinct broad bony part of the nose is left (Fig. 16).

BONY HUMP—OSTEOTOMY

The bony hump is reduced with a rasp. All the bony chips or bone dust is sucked out with the help of a suction tip (Fig. 17). Lateral osteotomy is undertaken with a curved Silverman osteotome, after giving a small incision in the vestibule of the nose along the sharp pyriform aperture. It is felt with a finger in the lateral part of the vestibule (Fig. 18). Lateral osteotomy can also be done by external

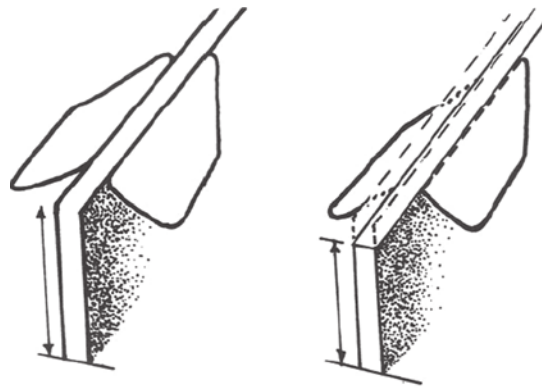


Fig. 15: Upper alar cartilages and nasal septum are cut to the same level



Fig. 16: Bony 1/3 appears broader

approach by making two stab incisions of 2 mm, size along the lateral bony wall of the nose and above the medial canthus. A 2 mm, osteotome is used to make 5/6 bony cuts along the lateral bony wall (Fig. 19).

The osteotomy should be done as close to the face as possible to avoid a stepdown deformity. The nasal bones and the frontal process of maxilla are infrafractured.

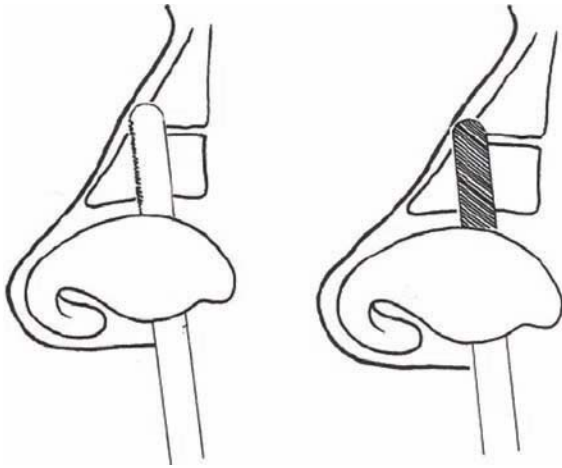


Fig. 17: Bony hump is reduced with a rasp or a saw

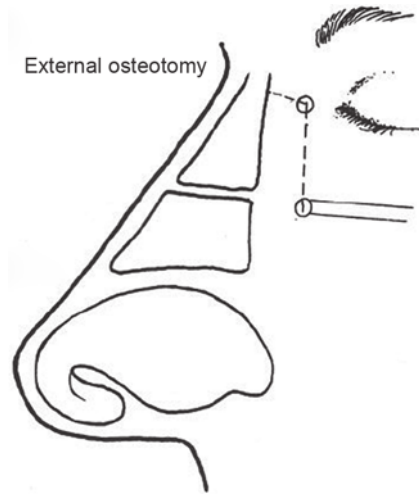


Fig. 19: External osteotomy

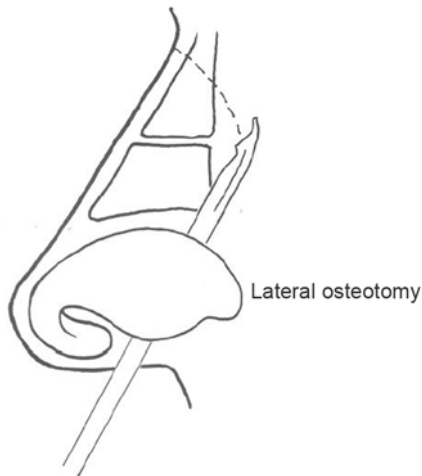


Fig. 18: Lateral osteotomy



Fig. 20: Median and paramedian osteotomies are performed

Paramedian osteotomies are performed by an osteotome. Starting from the caudal margin of the nasal bone on either side. Once this bony cut joins the horizontal osteotomy, the whole bony component comprising of frontal process

of maxilla and nasal bone is free and should be able to move the bony 1/3rd in any position, this infact is the "end point" of all good osteotomies, otherwise one may need to revise it (Fig. 20).

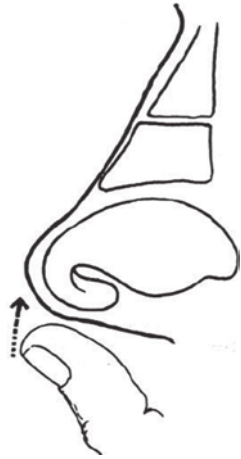


Fig. 21: Check for pollybeak by pushing the columella upwards

FINAL CLOSURE

At this stage, squeeze out and suck out the collected blood, bony chips or dust from the subcutaneous plane and look for the final shape of the nose.

Press the tip of the nose at the nasolabial angle and look for any pollybeak deformity caused due to the under reduced position of the nasal septum, or the untrimmed upper alar cartilage and it should be reduced accordingly. Look at the level of the septum and upper alar cartilage and whether they have been corrected to the desired profile level (Fig. 21).

The marginal incision is closed with four zero catgut. Close the transfixion incision. The nose is packed with a small antibiotic impregnated ribbon gauze. The bridge of the nose and the tip is strapped with adhesive tape. POP nasal cast of 6 layers is cut and soaked in warm water and is placed over the bridge and secured in place with adhesive tapes. Two minutes are taken for the POP to dry and set. A bolster is applied.

POSTOPERATIVE CARE

Keep a few gauze packs with the patient for cleaning the secretion and the blood. Patient is given antibiotics, anti-inflammatory drugs along with cold sponging of the eyes to lessen the subcutaneous edema. After forty-eight hours, the nasal packs are taken out. Bleeding may occur but is seldom troublesome. Patient is fit to home after that. The patient is briefed about the “black eye” which occurs after osteotomy. It disappears in due course. The POP cast and the strapping are removed after one week.

Postoperative photographs after six weeks and six months will show results in comparison to the preoperative photographs. The photographs are of help in medicolegal consequences, if any.

AUGMENTATION RHINOPLASTY

If the nasal dorsum is saddled, one may use a variety of grafts like autologous, homologous or alloplastic grafts. Depending on the extent of the deformity the size of the graft may vary. The commonly accepted grafts is the iliac crest autograft. This is achieved by bilateral intercartilagenous incisions or even in a case with open rhinoplasty procedure.

ADDED MEASURES TO IMPROVE THE RESULT

The results of cosmetic rhinoplasty can also be improved by certain adjuvant technique by modifying the facial proportions by mentoplasty, malarplasty, facial liposuction, or just by facial make-up.

TIP PLASTY

as practiced by
Dr PS Saharia

The cosmetic rhinoplasty as initiated by Jack Joseph in the part of the last century has stood the test of time. It is only in the last quarter that the functional aspect has been combined to make it a single stage procedure which deals with cosmetic and functional aspects and is called Septorhinoplasty, varying demands for a hump, upturned nose or pinched nostrils and the dynamic character of the tip, make this surgery a very "live" procedure.

Today the desire of an Indian patient is to have a functional, well formed straight nose with a prominent tip. The steps involved to produce such results are septoplasty, tip plasty, hump and upper alar cartilage reduction, osteotomies, splinting and dressing.

The tip of the nose is the referral point for adjusting the height and the shape of the nose. It is the highest point on the amorphous lobule of the nose. A supratip dip just cephalic to the lobule provides the tip an extra prominence. The lobule of the nose is covered with thick skin and ample amount of subcutaneous tissue which lies over the perichondrium covering the lateral crura and the dome. The medial crura is covered with thin skin with minimal subcutaneous tissue.

Tardy, 1990 has described major and minor support mechanisms of the tip of the nose. The three major factors include size, shape and the resilience of the lower alar cartilage, medial cural attachment to the inferior angle of the

nasal septum and thirdly the attachments between the upper and lower alar cartilages. These are supported by minor factors such as ligamentous swing between the lower alar cartilage and the nasal septum, sesamoid cartilages supporting the lateral crura attachment to the pyriform aperture; the subcutaneous and muscular tissues covering the alar cartilages, the anterior nasal spine and the membranous septum.

The tip has two dimensions, its projection on the face and its rotation in relation to the upper lip. Both aspects are inter-related like three limbs of a camera tripod. The tripod concept of the tip projection is helpful in understanding the dynamics of the tip modification (Fig. 22).



Fig. 22: Tripod concept

It is always important to analyze the nasal deformity of the septum, nasal dorsum, lobule, tip of the nose and the columellar, Saharia, 1998 Anderson, 1985.

SURGICAL TECHNIQUE FOR OPEN RHINOPLASTY

This mode of rhinoplasty provides a good exposure of the nasal superstructure and thus it is good for the beginners or for severely deformed noses, revision rhinoplasty or for congenitally deformed noses.

It involves a transcolumellar incision and thus external. It joins the two marginal incisions and opens up the nasal superstructure, which is modified as in the closed technique.

The transcolumellar incision is closed with 4-5 Vicryl and stitches are removed on the 6th day.

COMPLICATIONS ASSOCIATED WITH SEPTORHINOPLASTY

- Local infections.
- Irregularity of the nasal contour by projecting grafts, or by over or under correction.
- Columellar necrosis particularly after open rhinoplasty.
- Pollybeak deformity.
- Functional 'problem of nasal blockage due to uncorrected nasal septum or due to synechiae in the area of the nasal valve.

Once the problem is analyzed and recognized. It is 90% solved, McCollough 1987. Factors such as patient's height, facial features, ethnic background, skin texture need to be

considered in the operative plans. Only 5% patients have thin skin with visible outline of the nasal frame work while 20% have thick dark skin according to Baser, 1995.

If Septorhinoplasty is considered as the king of all the cosmetic surgical procedures, tip plasty is no less the queen. The tip sets the stage for rest the stage for the Septorhinoplasty. The height and the width of the nose have to be modified according to the nasal tip.

An adequate exposure is required to repair the nasal tip and it is provided either by delivery technique achieved through marginal and intercartilage incisions or by an open rhinoplasty technique. The first technique preserves the anatomical relationship during the surgery. The open rhinoplasty technique bares open the lower alar cartilage and the subcutaneous tissue well, but in an unnatural relationship and it is difficult to visualize the normal contour of the nasal tip while operating.

One should recognize one's own limitations in dealing with cases for tip - plasty and may develop one's own technique and master it for better results.

Once the exposure is achieved, tip rotation, projection, bifidity, columellar show, columellar lip relationship are checked and corrected. Procedures to modify the tip cartilage as described by McCollough are suitable for different shapes of the lobule. One who has developed his own technique can keep other methods to tackle different situations. Various techniques to modify the shape of the tip as advocated by the author are complete strip, rim strip, lateral crural flap, dome morselization, dome division and Goldman tip procedure (Fig. 23).

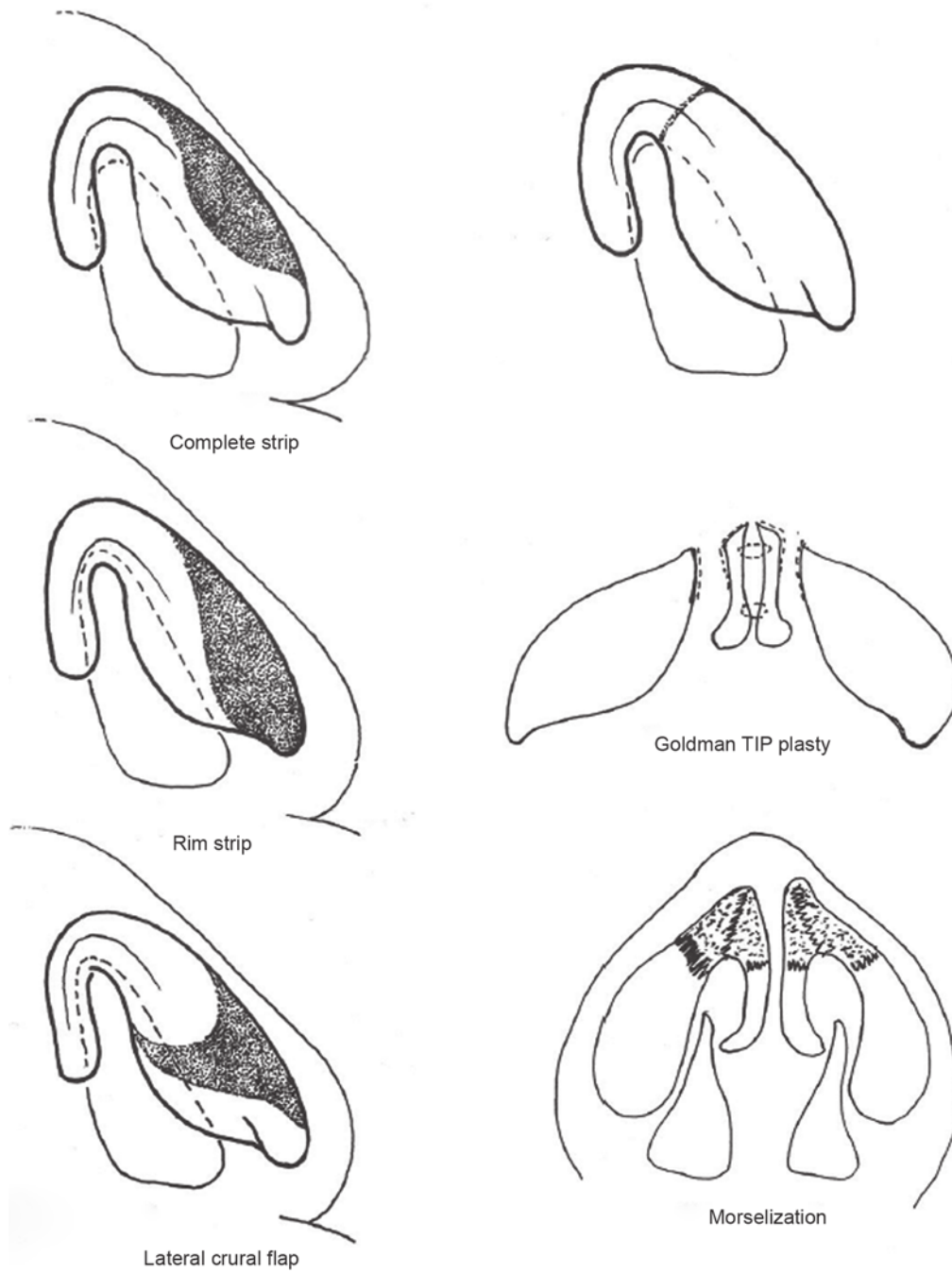


Fig. 23: Different techniques for tip remodelling

Procedures that shorten the lateral crura while lengthening the medial crura will affect rotation and increase projection. In order to achieve reduction in bulbosity in conjunction with rotation dome suturing is very effective. When deprojection is the goal then one may divide the dome and shorten the medial and lateral crurae to the desired length, Farris 1999.

I make marginal and intercartilagenous incisions and deliver the lower alar cartilage and symmetrical cephalic strips from the lower alar cartilage are removed, leaving behind at least 5 mm of its caudal margin. It provides good contour to the lobule and the tip of the nose. It also causes the lower alar cartilage to rotate and project. It is helped by the removal of a medial triangular piece from the upper alar cartilage after it is separated from the nasal septum.

If the tip needs more projection, steal parts of the lateral crura and suture them together and form a new dome. If the tip needs further rotation one may remove an inverted

triangular piece from the caudal margin of the nasal septum. If the medial crurae are found weak, I incorporate a cartilage strut between the two and stitch the three together. The position of the columella improves by this technique. The columella has direct impact on the esthetics of the nasal tip.

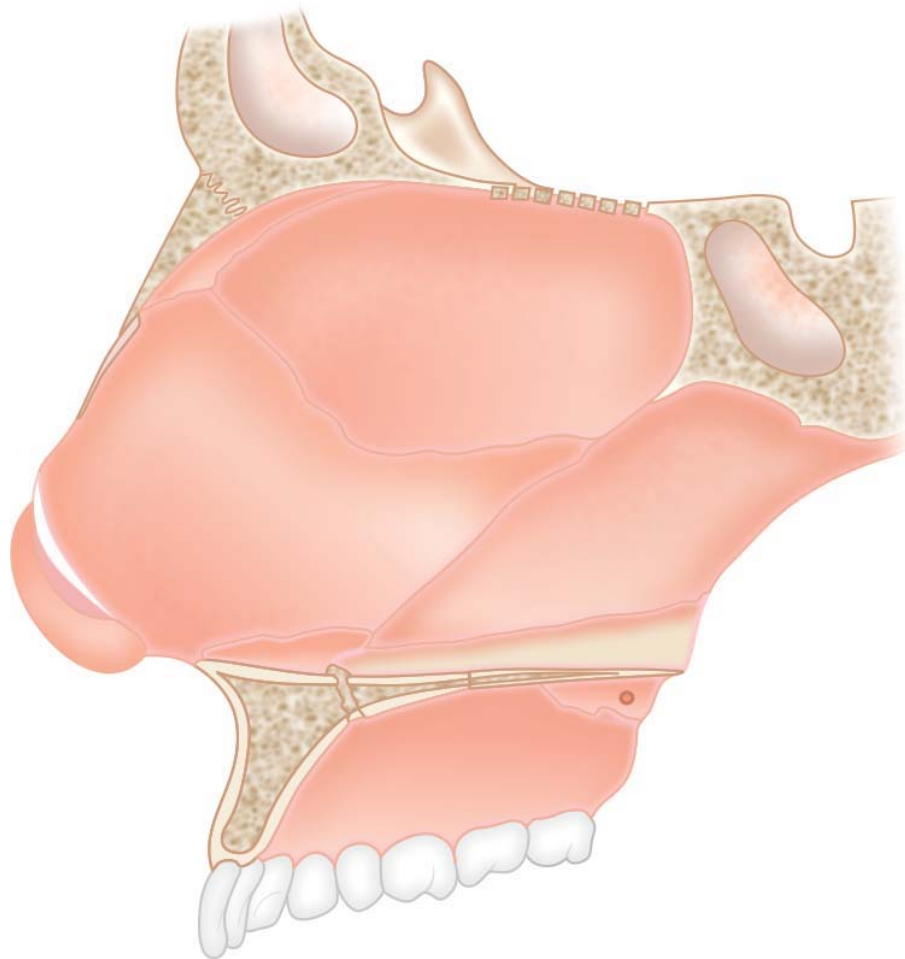
The dorsal margin of the nasal septum along with the processus dorsalis is may be reduced to avoid any pollybeak deformity.

The marginal incision and the incision to expose the septum are sutured with 3 zero chromic catgut. A light medicated pack is kept in the nasal cavity, 5 mm Micropore tape is applied to tip of the nose to keep the skin in close proximity of the underlying structures. A plaster of Paris cast is applied to the nasal dorsum. Postoperative treatment is provided with anti-inflammatory drugs, antibiotics and pain killers. The POP is kept in position for a week. A set of pre- and postoperative photographs of these patients are essential for comparing the results.

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Nose



Nose is a prominent midline structure on the face. When viewed from lateral aspect, various landmarks are revealed. These landmarks are produced as a result of underlying bones, cartilages and soft tissues. The upper one-third of the nose is formed by bones and lower two-third which is mobile is formed by cartilages.

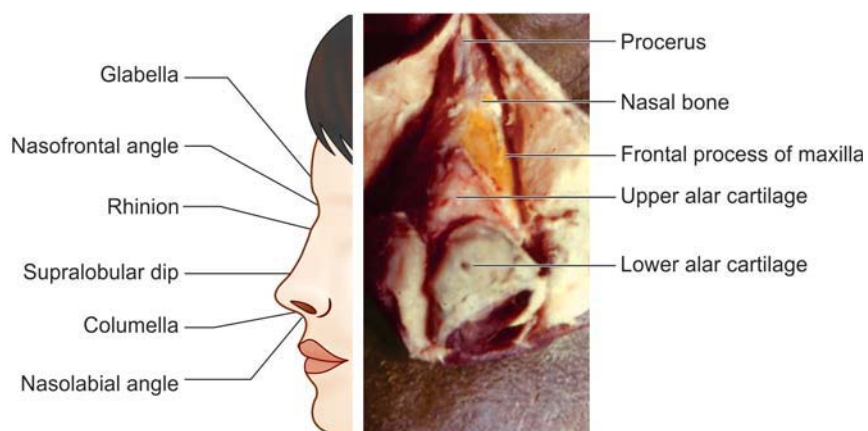


Fig. 1.1: External anatomy of nose

The nasal septum is formed by various cartilages and bones as shown.

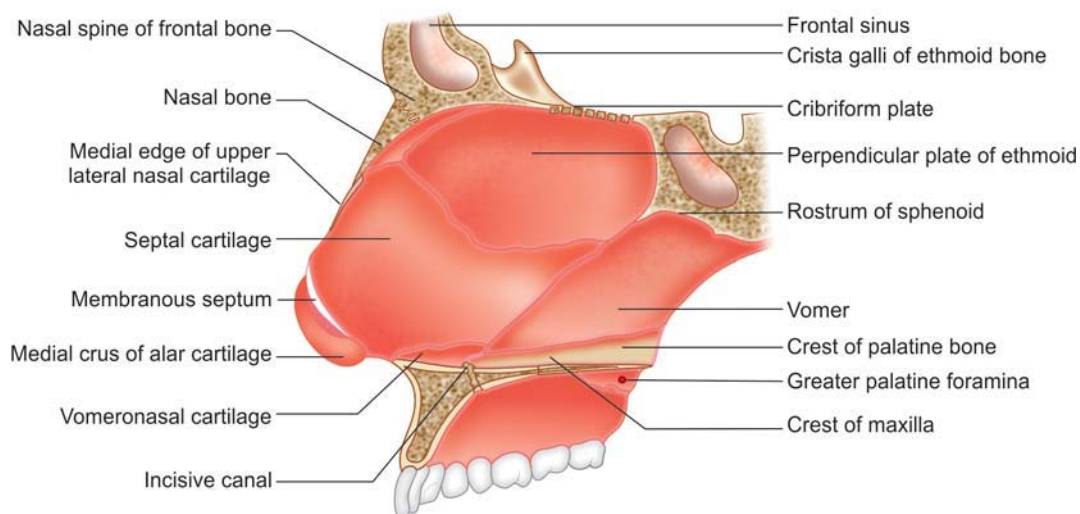


Fig. 1.2: Anatomy of nasal septum

The lateral wall of the nose has three curved projections called turbinates or concha. Various paranasal sinuses open into the meati as seen in the diagram. The nasolacrimal duct opens under the inferior turbinate.

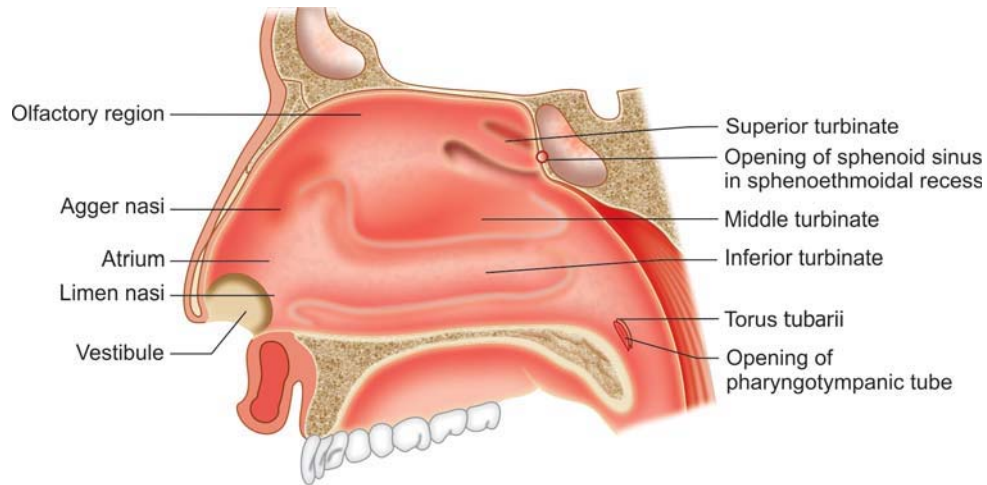


Fig. 1.3: Lateral wall of nose

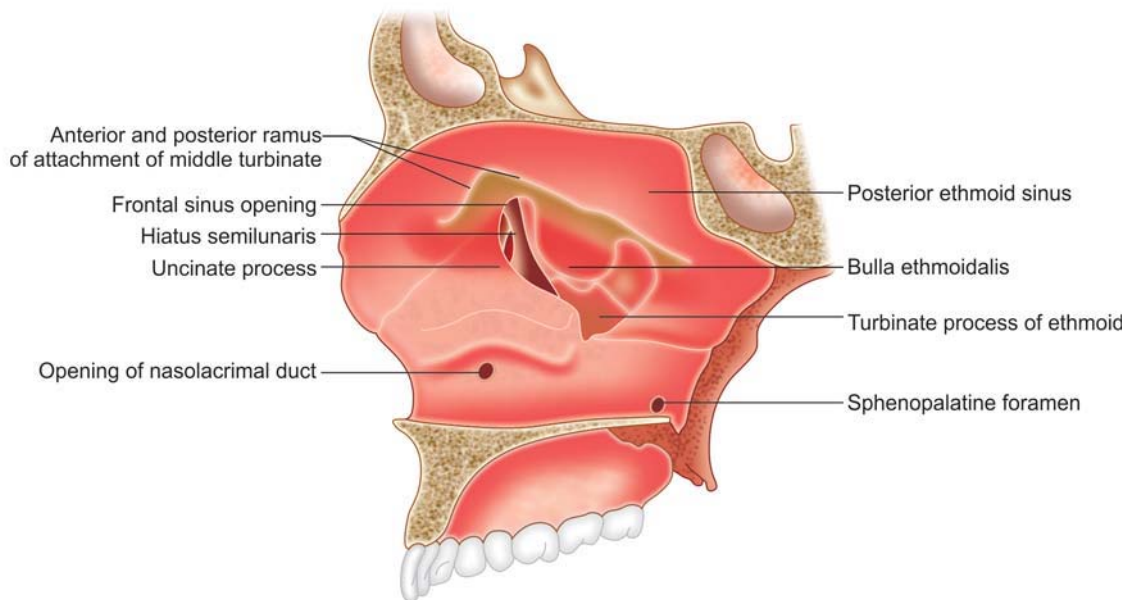


Fig. 1.4: Lateral wall of nose with turbinates removed

The nose has rich blood supply through the anterior and posterior ethmoidal arteries (internal carotid) and by the superior labial, sphenopalatine and greater palatine arteries (external carotid artery). The area of the vascular anastomosis between them is called Little's area. The Little's area is a common site for anterior epistaxis. This area is easily traumatized by nose picking.

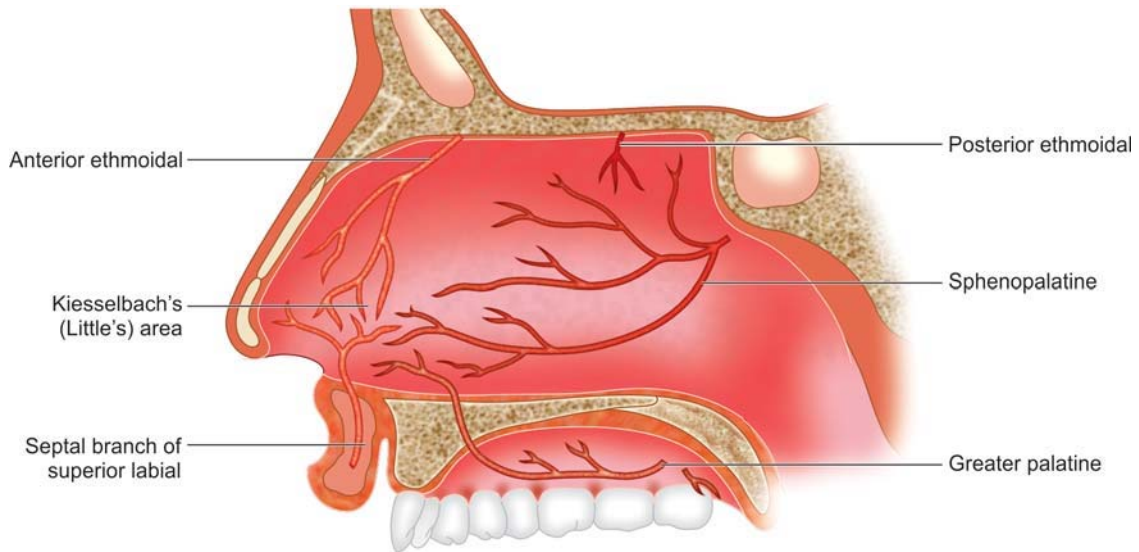


Fig. 1.5: Arterial supply of the nasal septum

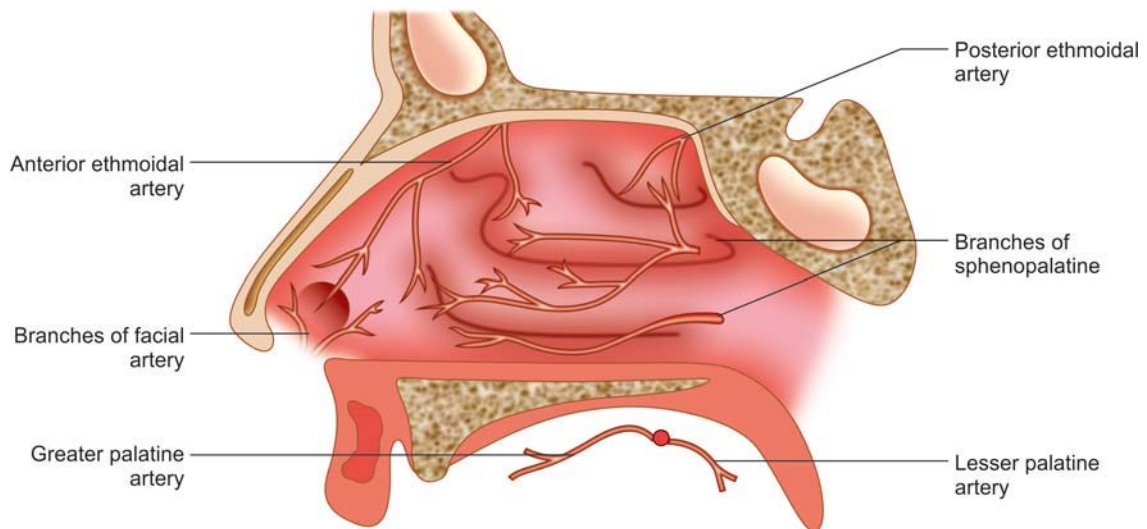


Fig. 1.6: Blood supply of lateral wall of nose

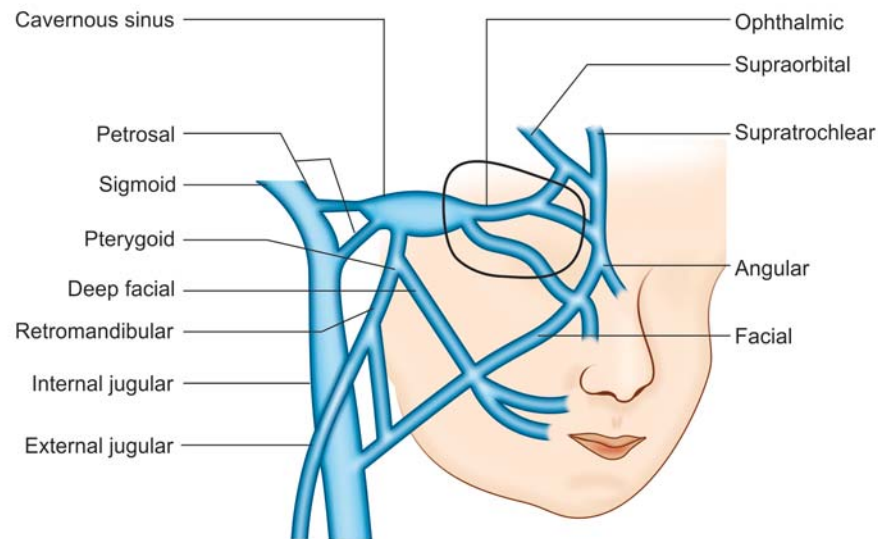
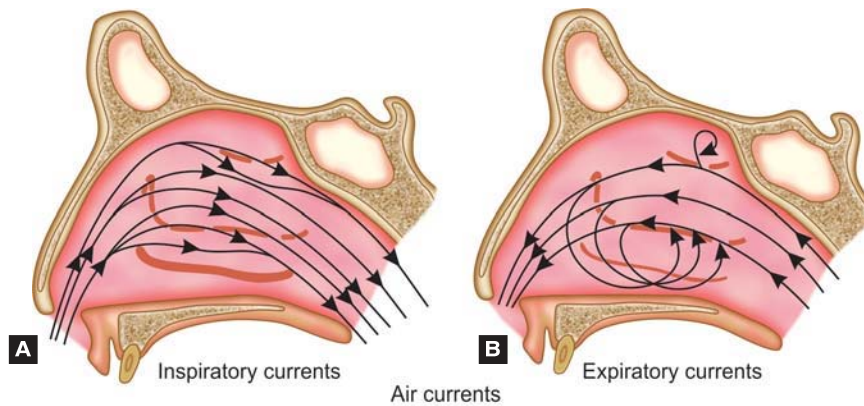


Fig. 1.7: Venous drainage of nose and face

Venous drainage of the nose is shown in Figure 1.7. Nasal infection can lead to cavernous sinus thrombosis which may get involved due to ascending infection from the nose and surrounding area. Thus, it should be treated with broad spectrum antibiotics.



Figs 1.8A and B: Nasal physiology

The inspiratory currents pass directly into the nasopharynx while the expiratory currents aerate the paranasal sinuses.

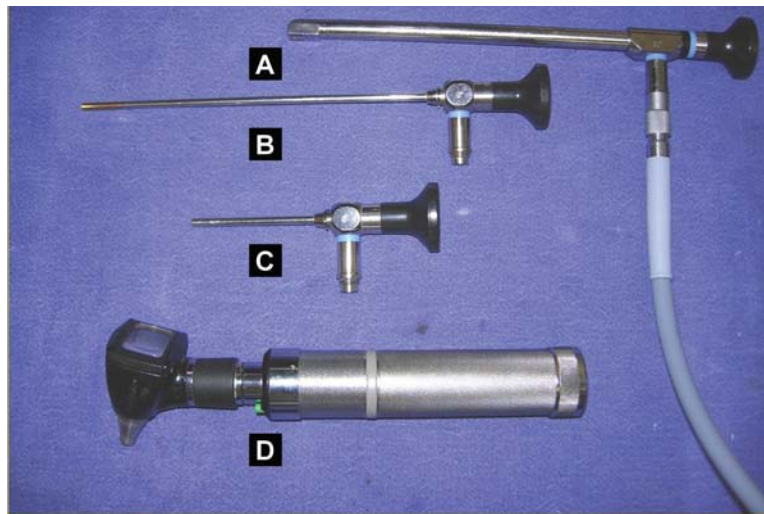


Fig. 1.9: ENT examination: ENT treatment unit



Fig. 1.10: ENT OPD equipment

Endoscopes required for examining the ear, nose and throat are shown.



Figs 1.11A to D: A-90° Laryngoscope, B-30° Nasal endoscope, C-Otoendoscope, D-Otoscope

Table 1.1: Functions of nose

- Respiratory airway
- Humidification and warming of air
- Protection by mucociliary blanket
- Olfaction
- Vocal resonance
- Protective nasal reflexes.



Fig. 1.12: Examination of nose: The vestibule of the nose is examined for fissure, vestibulitis or boil

External examination of the nose is performed to ascertain the symmetry, structural defect and other lesions. Vestibule is everted and examined for fissures or vestibulitis.

Anterior rhinoscopy is done by reflecting deep penetrating light through a head mirror. The various turbinates, meati and nasal septum are checked.



Fig. 1.13: Anterior rhinoscopy

Nasal endoscopy is now a standard OPD procedure for complete examination of nasal cavity as well as nasopharynx.

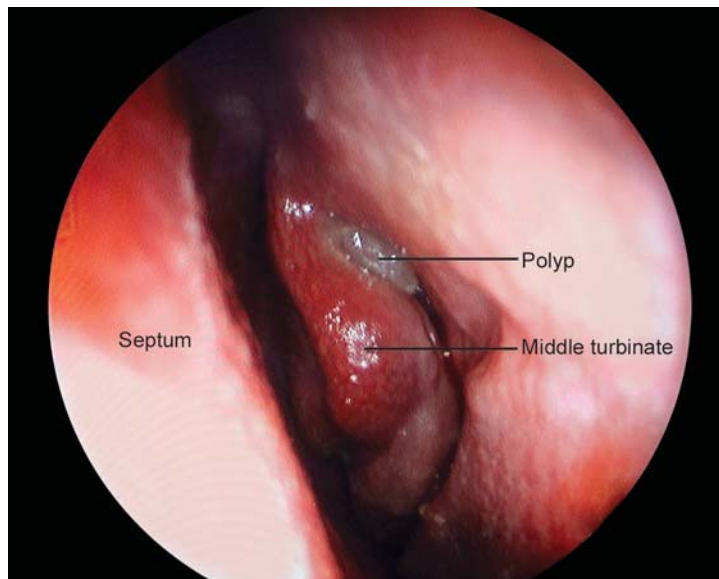


Fig. 1.14: Nasal endoscopy

Compare the patency of the nasal passages by allowing the fog to settle on a shining surface.



Fig. 1.15: Test for nasal patency: Fog test

When necessary, the nasal cavities are examined after applying local anesthetic and decongestant.



Fig. 1.16: Examine nose after decongestion with adrenaline or xylometazoline

Posterior rhinoscopy reveals posterior edge of the nasal septum, posterior choanae and posterior ends of the turbinates. Eustachian tubes open in the lateral wall while the fossae of Rosenmuller lie posterosuperiorly. The adenoids are situated in the roof and posterior wall.

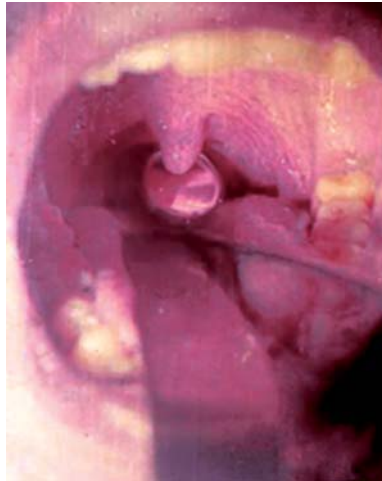


Fig. 1.17: Postnasal examination

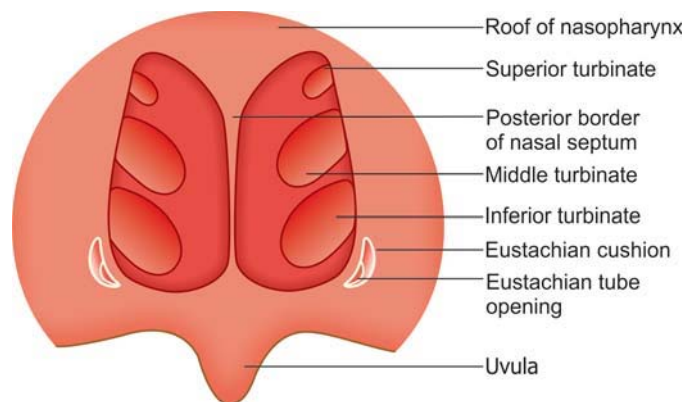


Fig. 1.18: Diagrammatic view of structures seen in posterior rhinoscopy
(Nasopharyngoscopy can be done with nasal endoscope)

Common symptoms of the nasal disease are blockage, discharge and disorder of smell. Since the nose is vascular, bleeding is a common complaint. Causes and symptoms of nasal blockage and discharge are illustrated.

Table 1.2: Structural causes of nasal blockage

Congenital

- Choanal atresia
 - Unilateral/Bilateral

Acquired

- Deviated nasal septum
- Nasal polyps
- Tumor

Nasal atresia can either be anterior or posterior. Acquired anterior nasal atresia case is shown.



Fig. 1.19: Anterior acquired nasal atresia secondary to cancerum oris

Choanal atresia can be unilateral or bilateral. Bilateral congenital atresia is an emergency requiring urgent intervention.



Fig. 1.20: Unilateral congenital posterior atresia. It can be demonstrated by a plain X-ray examination with a cotton ball soaked in radiopaque dye (arrow)

Unilateral cases may remain undiagnosed until adulthood. Bilateral cases present as acute respiratory distress in neonates. It can be demonstrated by an axial CT scan of paranasal sinuses as shown in Figure 1.21. In this patient, a nasal cannula was inserted through the partial atresia on right side. Complete bony atresia is seen on the left.



Fig. 1.21: CT scan of congenital choanal atresia



Fig. 1.22: Acute vestibulitis

Vestibulitis is caused by staphylococci. It should be treated promptly as it may result in cavernous sinus thrombosis (refer to Figure 1.7 venous drainage of the nose).

New growths of the external nose are frequently seen. Nasal warts and rhinophyma cases are managed by radiofrequency ablation or laser.

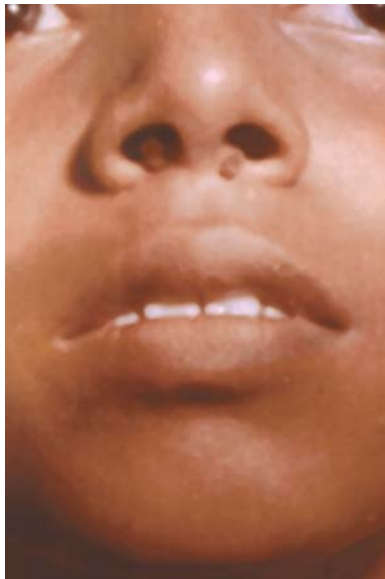


Fig. 1.23: Nasal warts



Fig. 1.24: Rhinophyma

A case of rodent ulcer of the nose.



Fig. 1.25: Basal cell carcinoma presenting as rodent ulcer involving nose and lower eyelid with cicatrization

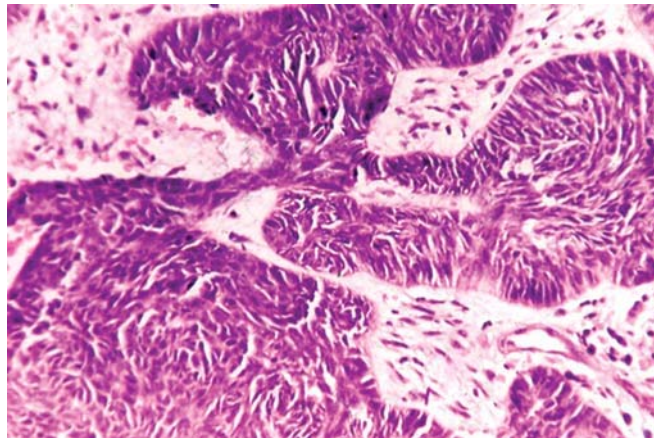


Fig. 1.26: Histopathology of basal cell carcinoma: Microscopic picture of basal cell carcinoma (rodent ulcer) showing anastomosing cords of elongated tumor cells

Injury to the nose may cause loss of tissue or distortion of the shape of the nose as shown. The lost tissue is transplanted while distortion is corrected by remoulding the underlying nasal cartilages and bones.

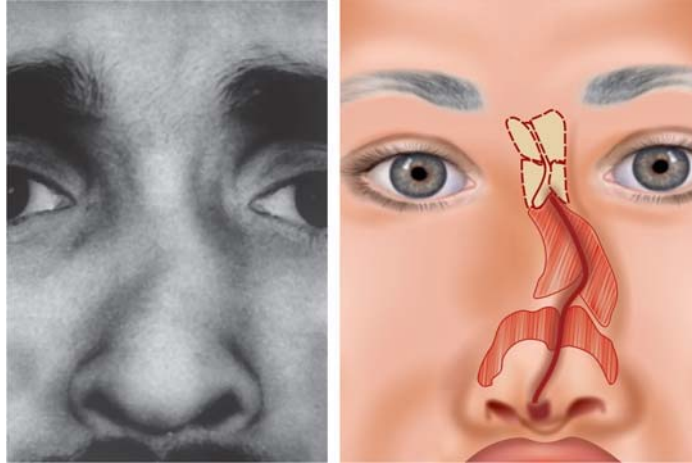


Fig. 1.27: Photograph and diagrammatic representation of fractured nose



Fig. 1.28: Deviated nasal septum with external deformity

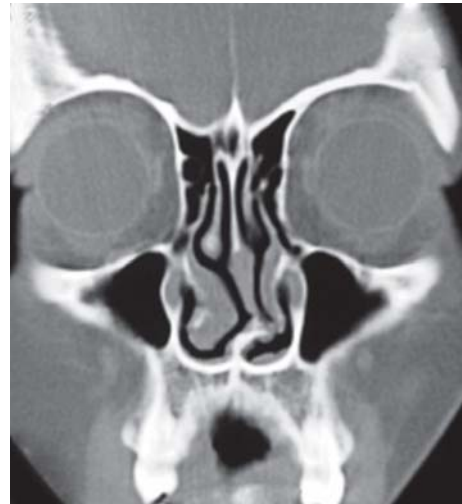


Fig. 1.29: Coronal CT: Deviated nasal septum left and spur is in contact with inferior turbinate. Such a spur may cause contact headache. Also note right inferior turbinate hypertrophy on opposite side

Deviated nasal septum may be asymptomatic or may produce nasal obstruction and its consequent symptoms. It may also contribute to the external deformity of the nose. Symptomatic deviations need surgical correction, i.e. septoplasty or septorhinoplasty.



Fig. 1.30: Septal perforation has varied etiology. Its presence can be confirmed by a probe as demonstrated

Table 1.3: Causes of septal perforation

1. Trauma
 - Operative
 - Tribal customs
 - Nose picking.
2. Chronic specific rhinitis
 - TB
 - Syphilis
 - Lupus
 - Leprosy.
3. Nonspecific rhinitis
 - Rhinitis caseosa
 - Midline granulomas.
4. Neoplasms.
5. Septal infection.
6. Heavy metal poisons
 - Chromium
 - Lead
 - Mercury
 - Arsenic.
7. Cocaine abuse.
8. Idiopathic.



Figs 1.31A and B: Bilateral septal swelling: Septal abscess should be drained urgently to prevent complications like septal perforation or saddle deformity

Acute inflammation of the mucous membrane of the nose is called acute rhinitis or coryza. It results in blockage of the nose and rhinorrhea. Etiology of coryza is varied as charted below: The viruses are the chief causative organisms.

Table 1.4: Etiology of coryza	
<i>Climate:</i>	Environmental temperature and humidity changes
	Fatigue
	Low nutritional intake
	Vitamin A, C, and D deficiency
<i>Nasal obstruction</i>	Chronic upper respiratory tract infections
	Change in nasal pH
<i>Viral infections</i>	<ul style="list-style-type: none"> • Influenza virus • Rhinovirus • Coxsackie virus • Respiratory syncytial virus • Echovirus • Parainfluenza virus • Rheovirus

Allergic rhinitis is the manifestation of an antigen-antibody reaction in the nasal mucosa. A variety of allergens are known to cause this. Vasomotor rhinitis is due to autonomic instability and clinically presents with similar symptoms.

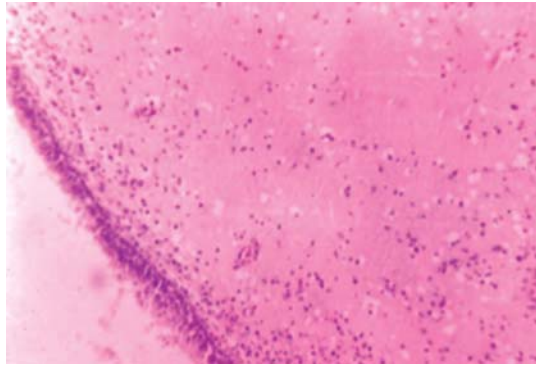


Fig. 1.32: HPE of allergic rhinitis slide showing characteristic edema and infiltration of the mucosa predominantly by eosinophils

Table 1.5: Treatment of allergic rhinitis

- Allergen avoidance
- Antihistamines
- Sprays
 - Azelastine
 - Fluticasone
 - Combination: Fluticasone + Azelastine
- Immunotherapy.

Table 1.6: Vasomotor rhinitis

Presentation: In young with high strung personality with autonomic instability

Symptoms: Rhinorrhea, sneezing and nasal blockage

Treatment:

- Lifestyle changes
- Antihistamines
- Decongestants
- Nasal sprays: Antihistaminic-cortisone

Table 1.7: Causes of atrophic rhinitis*Heredity*

- Hormonal imbalance—more in females
- Chronic sinusitis
- Excessive surgical removal of nasal mucosa
- Malnutrition
- Iron deficiency anemia
- Idiopathic

**Fig. 1.33:** Patient with saddle nose secondary to atrophic rhinitis

Atrophic rhinitis usually presents with nasal obstruction, epistaxis and depressed bridge of nose. A variety of causes are responsible for the same. The changes in the nasal cavity can be minimal to marked crusting along with a fetid odor (ozena), which at times is repulsive to others, but patient being anosmic is unaware of it. Alkaline nasal douching is recommended to keep the nasal cavity clean.

Table 1.8: Treatment of atrophic rhinitis

- Alkaline nasal douching
- Kemicetene antiozena solution
- Anhydrous glucose in glycerine
- Vitamin, iron, antioxidants supplementation
- Surgery (Youngs' operation).



Fig. 1.34: Rhinolith specimen from right nasal cavity of an eleven-year-girl who presented with intermittent right side epistaxis

Table 1.9: Rhinolith

Complaints

Unilateral nasal obstruction. Foul smelling blood stained rhinorrhea

On examination

Brown-black stony hard mass in nasal cavity

Investigation

X-ray PNS (Waters' view), CT Paranasal sinus

Treatment

Removal under GA



Fig. 1.35: Fungal rhinosinusitis: Repeated polyp formation may result in expansion of the nasal bridge or even proptosis when associated with rhinitis caseosa as seen in this patient



Fig. 1.36: Antrochoanal polyp: Pedunculated mass of the nasal mucosa as sequelae of allergy and/or infection is termed as nasal polyp. Ethmoidal polyps are multiple while antrochoanal polyp is usually single as shown



Fig. 1.37: Coronal CT: Left antrochoanal polyp: Nasotracheal component in scan above and choanal component in scan below

Axial scan clearly shows the entire extent of the polyp.

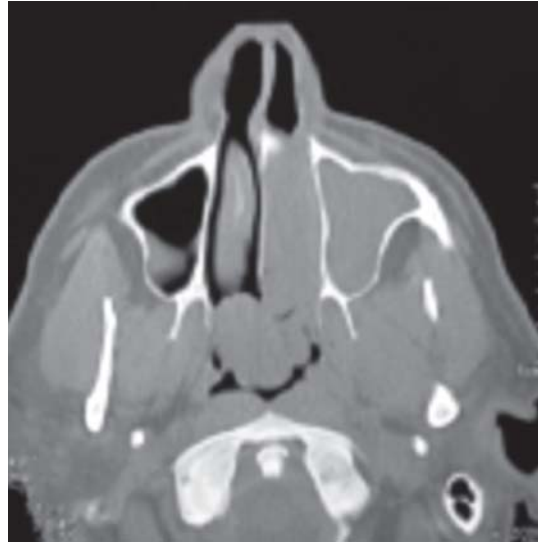


Fig. 1.38: Axial CT: Left antrochoanal polyp of same patient as in Figure 1.37



Fig. 1.39: Antrochoanal polyp specimen: The antrochoanal polyp has small antral, narrow elongated nasal and rounded nasopharyngeal parts

Microscopic picture shows polyp covered with metaplastic squamous epithelium.

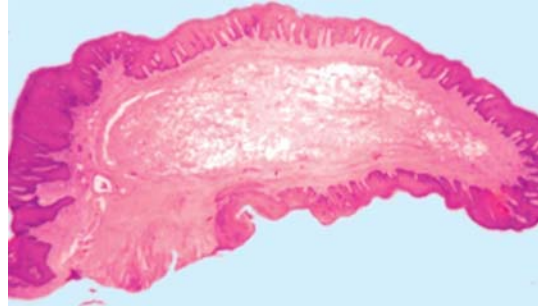


Fig. 1.40: Cut section: Nasal polyp

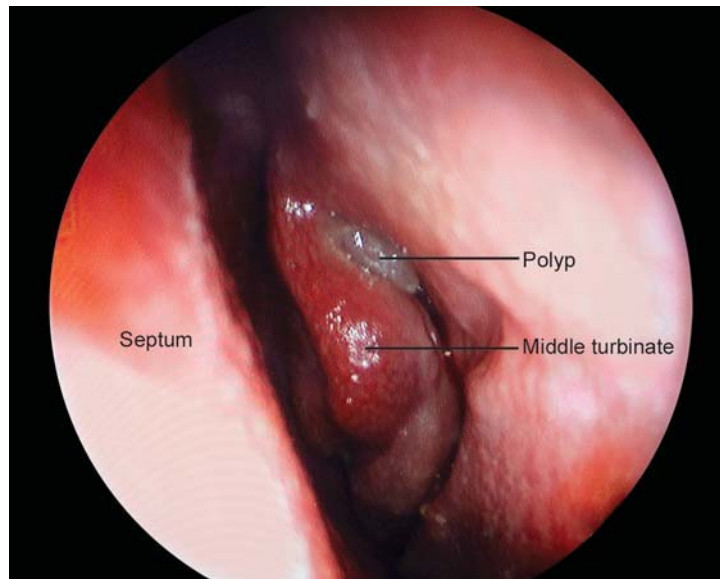


Fig. 1.41: Ethmoid polyps

Nasal polyps are removed by endoscopic sinus surgery.

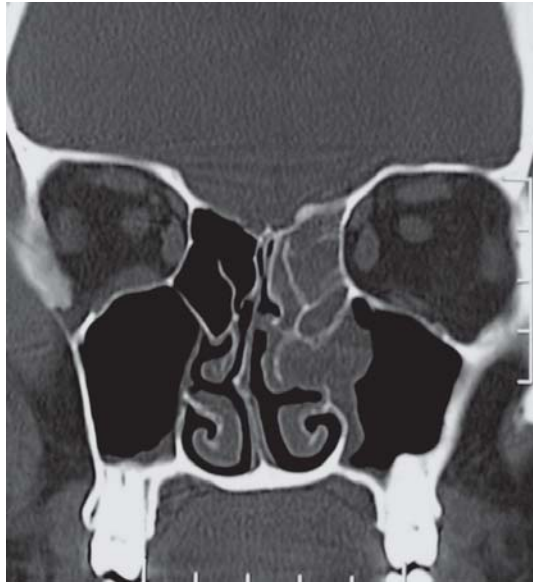


Fig. 1.42: Plain CT PNS coronal cut at osteomeatal complex showing left ethmoid polyps

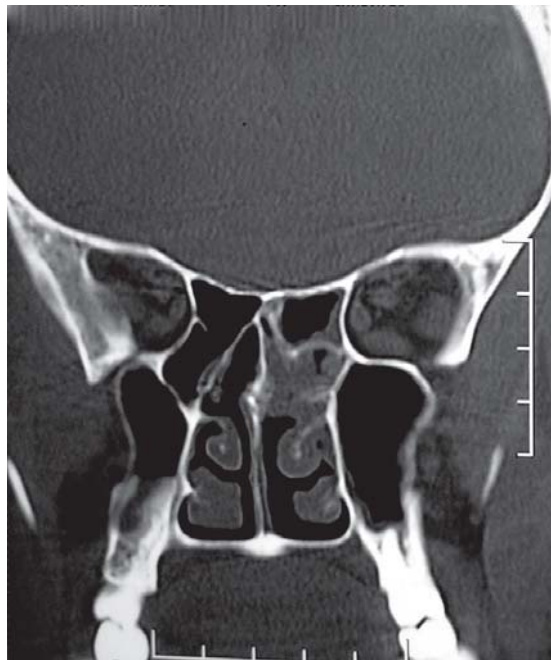


Fig. 1.43: Plain CT PNS coronal cut at level of posterior ethmoids showing polyps



Fig. 1.44: Postoperative ethmoid cavity



Fig. 1.45: Instruments for endoscopic sinus surgery (ESS). Microdebrider assisted ESS preserves nasal mucosa and is less traumatic

Nasal foreign bodies can be removed by a curved probe (Eustachian catheter) in the OPD itself.



Fig. 1.46: Foreign body nose and its removal



Fig. 1.47: X-ray showing button battery in nose of a 3-year-old



Fig. 1.48: Button battery removed with Eustachian catheter in OPD

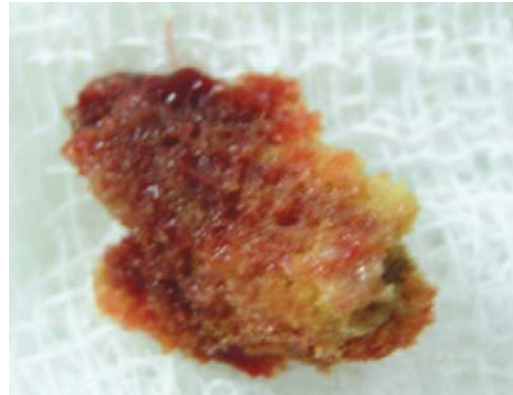


Fig. 1.49: Foam removed from nose

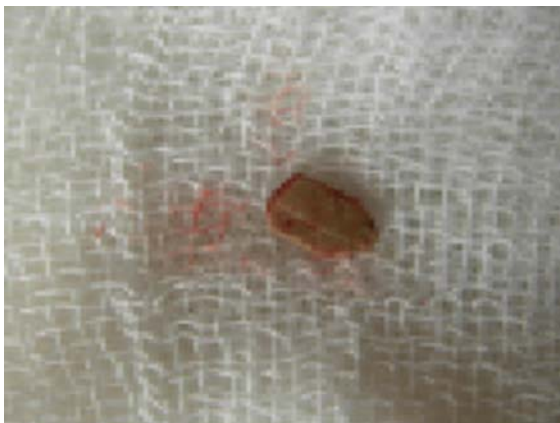


Fig. 1.50: Chalk removed from nose



Fig. 1.51: Various foreign bodies removed from ear, nose, throat and bronchus

Table 1.10: Causes of epistaxis

Epistaxis
<p><i>Local</i></p> <ul style="list-style-type: none"> • Congenital <ul style="list-style-type: none"> – Multiple telangiectasia.
<ul style="list-style-type: none"> • <i>Acquired</i> <ul style="list-style-type: none"> • Trauma • Foreign body • Inflammation • Angioma • Nasopharyngeal angiofibroma • Nasopharyngeal carcinoma.
<p><i>Drugs</i></p> <ul style="list-style-type: none"> • Salicylates • Arsenic.
<p><i>Infectious fevers</i></p> <ul style="list-style-type: none"> • Measles • Chickenpox • Typhoid.
<p><i>Systemic</i></p> <ul style="list-style-type: none"> • Hematological <ul style="list-style-type: none"> – Hemophilia – Christmas disease – Leukemia – Kala-azar – Malaria. • Conditions with raised intra-arterial pressure <ul style="list-style-type: none"> – Arteriosclerosis – Hypertension – Nephritis. • Conditions with raised intravenous pressure <ul style="list-style-type: none"> – Mitral stenosis – Emphysema.

Epistaxis is a symptom and may result from variety of causes. The common site for epistaxis is Little's area. Epistaxis may vary from just spotting to severe bout of bleeding.

An outdoor procedure to stop epistaxis is to pinch the nostril for three minutes as shown.



Fig. 1.52: First aid: Pinch the nostrils for three minutes by watch



Fig. 1.53: Anterior nasal packing: Epistaxis is controlled by anterior nasal packing as shown or by endoscopic cauterization of bleeding points or by ligating the artery in severe refractory cases

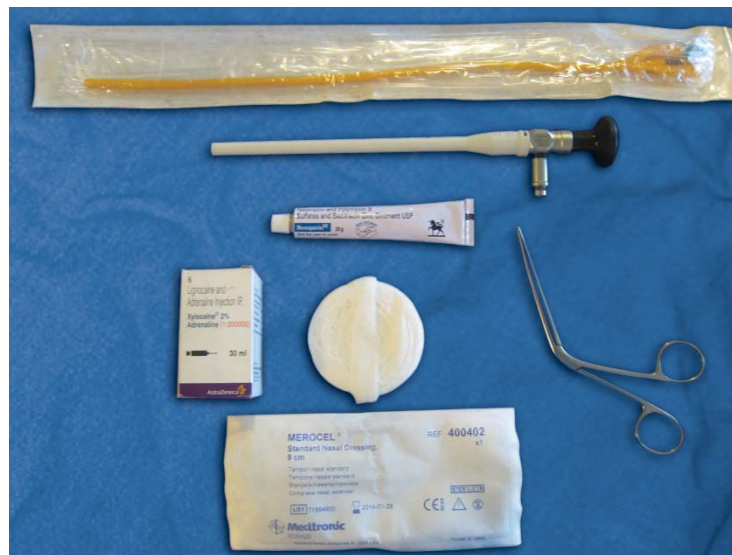


Fig. 1.54: Materials for nasal packing

Table 1.11: Nasal granulomas

1. Scleroma
2. Rhinosporidiosis
3. Tuberculous
4. Lupus vulgaris
5. Syphilitic
6. Leprosy
7. Boeck's sarcoid
8. Diphtheria
9. Histoplasmosis
10. Sporotrichosis
11. Leishmaniasis
12. Moniliasis
13. Yaws
14. Glanders.

Rhinoscleroma can be presented as an infiltrative lesion causing granulomatous lesion of the external nose.



Fig. 1.55: Infiltrative nasal scleroma

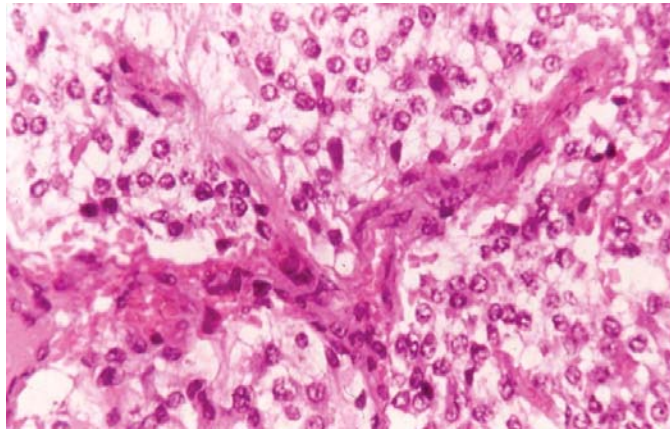


Fig. 1.56: Histopathology showing Mikulicz cells and macrophage with vacuolated cytoplasm. Microscopically, this lesion consists of infiltration of connective tissue by inflammatory cells. The characteristic Mikulicz cell of scleroma is a large macrophage with vacuolated cytoplasm



Fig. 1.57: Fungal granuloma in left nasal cavity: Rhinosporidiosis is a fungal granuloma affecting the nasal mucosa and produces a polypoidal granular mass which bleeds on touch

Microscopically, the fungal spores in various stages of maturation can be seen in the mucosa.

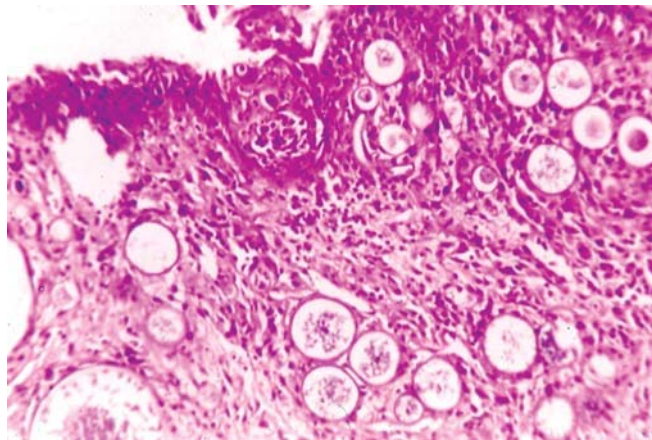


Fig. 1.58: Histopathology showing fungal spores



Fig. 1.59: Lupus external nose. Lupus is an uncommon granuloma affecting the nose

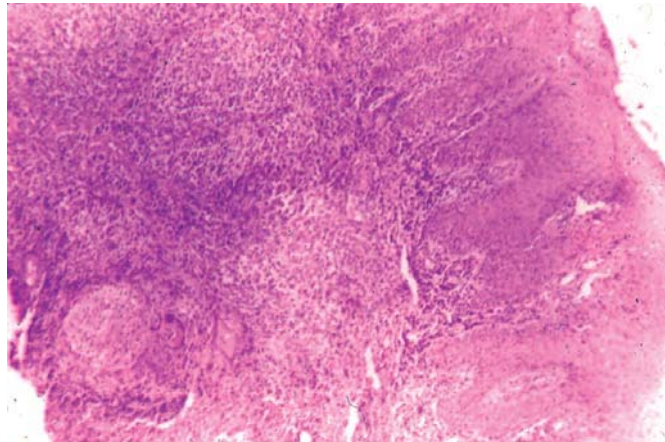


Fig. 1.60: Histopathology of lupus nose: Microscopically, granulomas consist of lymphocytes, epithelioid cells and Langhans giant cells and they are present in the dermis



Fig. 1.61: Nasal tumor. Nasal tumors produce blockage, epistaxis and distortion of the nasal anatomy. Photograph shows a large nasal tumor occupying the nostril. The most common nasal tumor is squamous cell carcinoma

Table 1.12: Tumors of the nose and paranasal sinuses

Nasal tumors are uncommon and can be classified into ectodermal, mesenchymal, neurogenic or odontomes as charted

<i>Ectodermal</i>	
<i>Benign</i> Papilloma Adenoma	<i>Malignant</i> Squamous cell carcinoma Adenocystic carcinoma Adenocarcinoma
<i>Neurogenic</i>	
Esthesioneuroma	Neurofibroma
<i>Mesodermal</i>	
Fibroma Osteoma Chondroma	Lymphosarcoma Fibrosarcoma Chondrosarcoma Myxosarcoma Rhabdo sarcoma Malignant melanoma
<i>Odontomes</i>	

Symptomatic cases of deviated nasal septum are corrected by septoplasty. Those cases of DNS with external deformity undergo septorhinoplasty.



Fig. 1.62: Markings for septorhinoplasty: Rhinoplasty may be reconstructive or cosmetic. Esthetic realignment and restructuring the components of the nose constitutes cosmetic rhinoplasty



Figs 1.63A to D: Septorhinoplasty: Preoperative and postoperative photographs

Photographs of a patient before and after septorhinoplasty shown.



A Crooked nose

B Postoperative

Figs 1.64A and B



C Preoperative basal view

D Postoperative basal view

Figs 1.65C and D



E Hump

F Postoperative profile corrected

Figs 1.66E and F

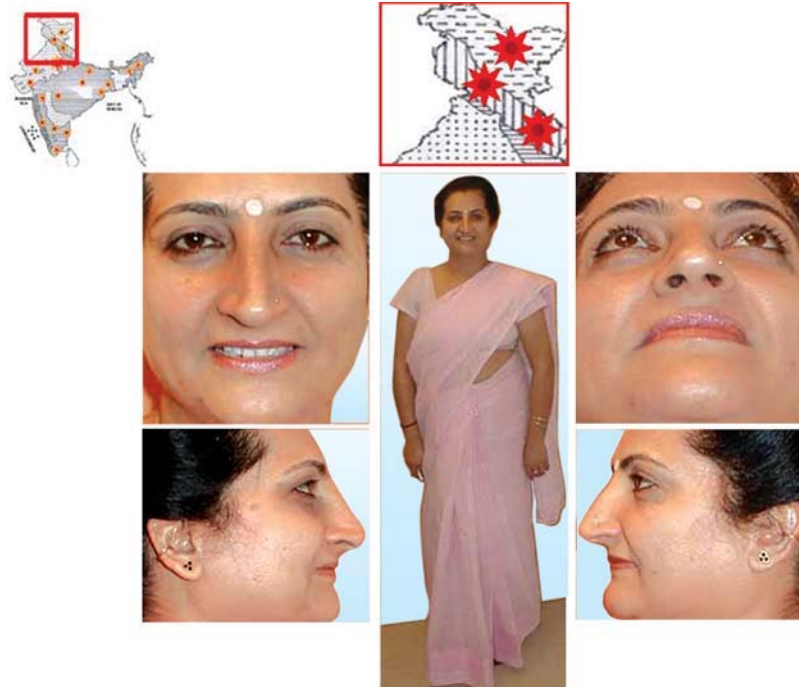


Fig. 1.67: Indian nose: In northern India, people have long, pointed and well-defined nose with large dorsoventral and anteroposterior dimensions of the nasal septum

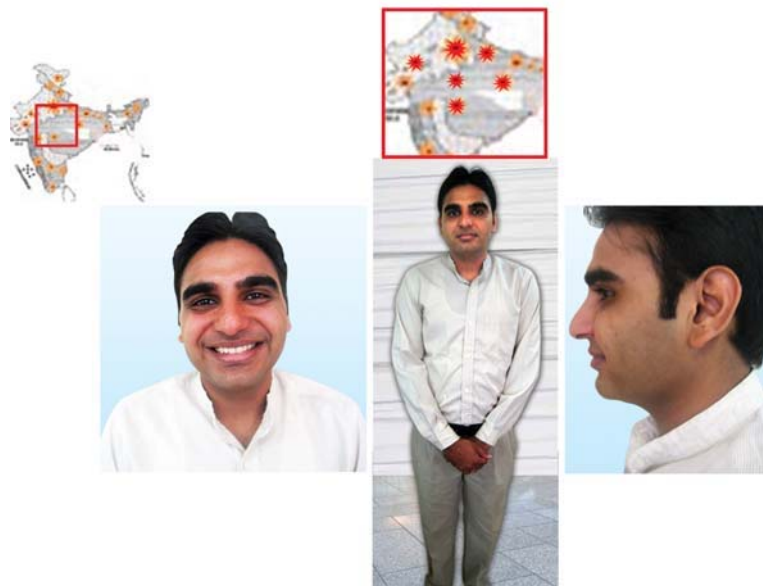


Fig. 1.68: Indian nose: In central India, people have prominent nose with amorphous dorsum and wider septoalar angle and thicker subcutaneous covering



Fig. 1.69: Indian nose: In eastern India, the nose of people is rather flat and small with short septum, smaller alar cartilages and short columella

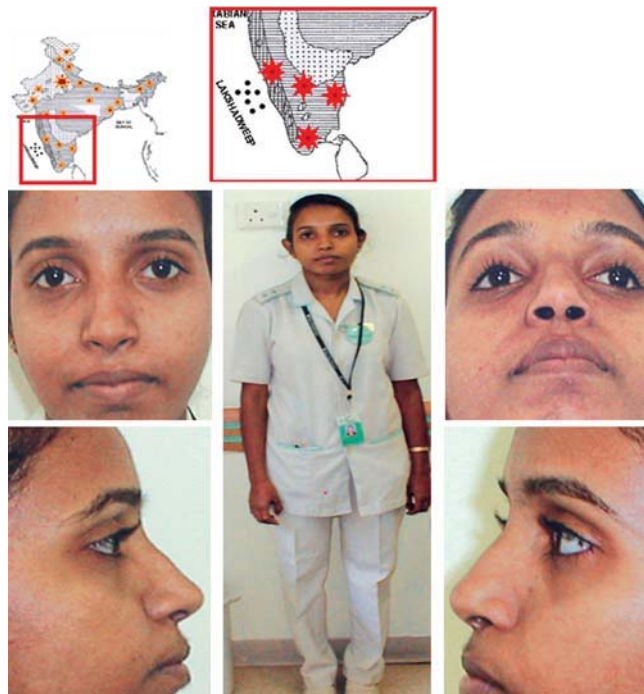
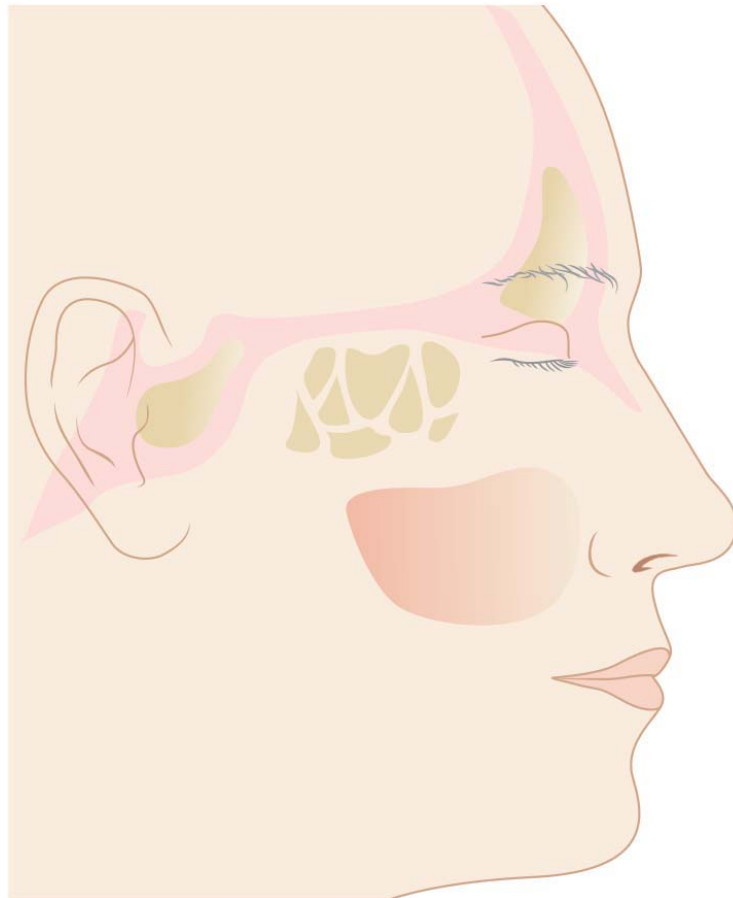


Fig. 1.70: Indian nose: In south India, people have small noses, with smaller alar cartilages and short columella with short dorsum

2

PS Saharia, Deepti Sinha

Paranasal Sinuses



Paranasal sinuses, the air filled spaces, are the parameres of the nasal cavity. There are four pairs of sinuses which are classified into anterior and posterior groups. Anterior group consisting of frontal sinus, maxillary and anterior ethmoid sinus, open in the middle meatus. Posterior group consisting of posterior ethmoids and sphenoid open in superior meatus. They are in close relation to the cranium, pituitary fossa and orbit (Fig. 2.1).

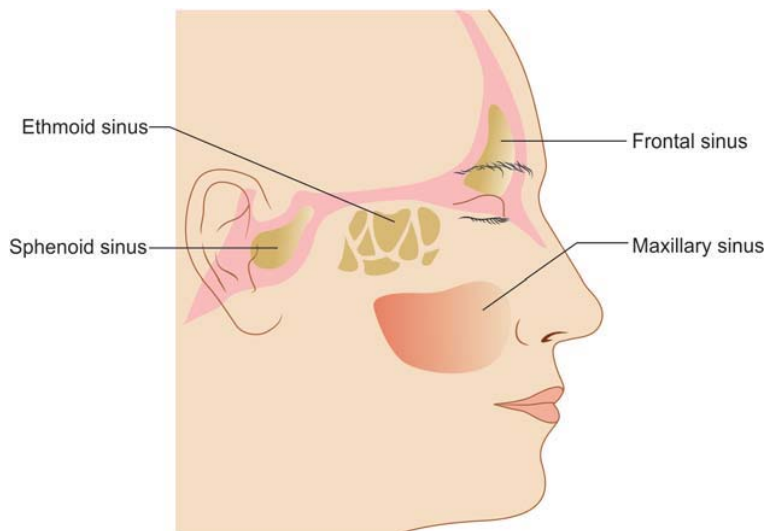


Fig. 2.1: Anatomy of paranasal sinuses (PNS)

Examination of paranasal sinuses involves examination of the nose through anterior rhinoscopy and a look into the lateral wall for polyps or discharge. Epiphora, displacement of the eyeball, swelling of the cheek or fullness of the medial canthus point to the inflammation of the sinuses. Palpate the frontal and maxillary sinus to exclude acute inflammation as demonstrated (Figs 2.2 and 2.3).



Fig. 2.2: Palpation of frontal sinus

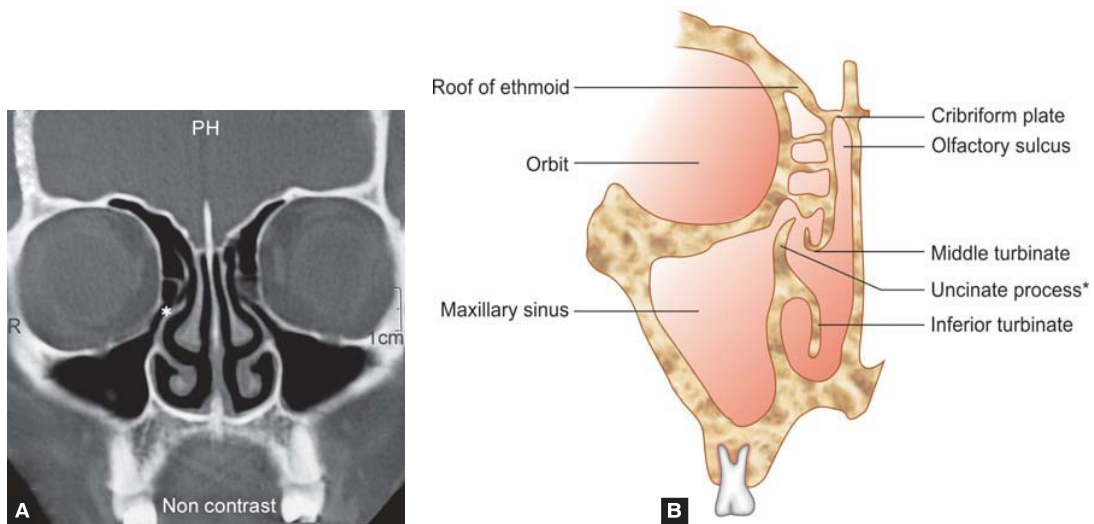


Fig. 2.3: Palpation of maxillary sinus



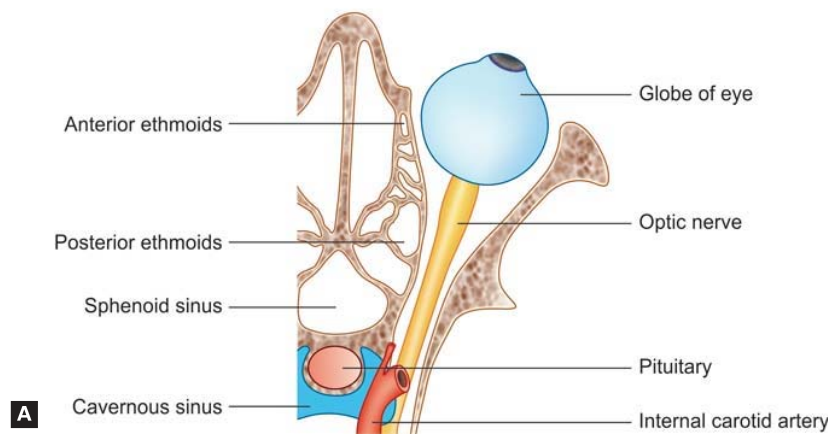
Fig. 2.4: X-ray PNS (Water's view). In this X-ray, there is an air-fluid level in left maxillary sinus

The paranasal sinuses can be examined by X-ray Occipito-mental (Water's) view, Occipito-frontal (Caldwell) view, Submento-vertical (Hirtz position), Lateral view, Oblique view (Rhes position).



Figs 2.5A and B: Coronal CT of PNS

Figure 2.5A shows plain CT scan paranasal sinus coronal section, at the level of osteomeatal complex. The uncinate process(*), middle and inferior turbinates and maxillary sinus are seen clearly on both sides.



A



B

Figs 2.6A and B: Axial section of PNS at the level of sphenoid sinus (A) Diagram; (B) CT scan
Note: Left side ethmoid polyps

Computerized tomography of the paranasal sinus has changed the concept of management of sinus disease. Coronal sections are in the same plane as the osteomeatal complex which has been acknowledged universally as the key area in pathophysiology of sinus disease. The CT scans also serve as a road map for sinus surgery.

The CT scans (see Figure 2.5) are best done after an adequate course of antibiotics and decongestants so that acute inflammation is not mistaken for chronic mucosal disease. Patient should also blow his nose prior to CT scan to expel loose mucus in the nasal cavity.

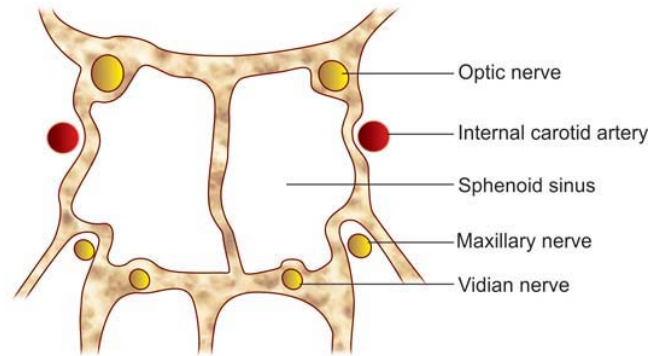


Fig. 2.7: Coronal section of PNS at the level of sphenoid sinus

Vital structures in relation to sphenoid sinus make the surgery in the region dangerous particularly when there is dehiscence of bony covering of optic nerve or carotid artery.

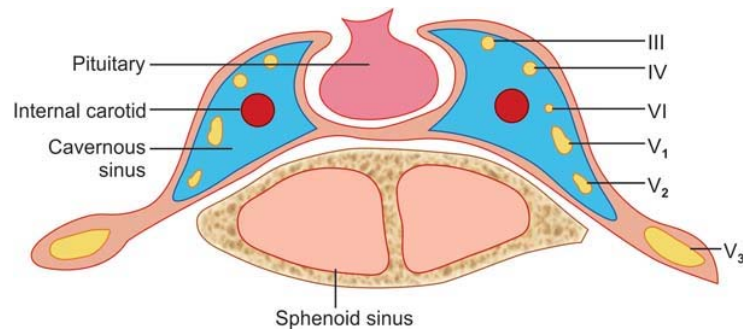


Fig. 2.8: Relations of sphenoid sinus

Endoscopic trans-sphenoidal route is being used by ENT surgeons to remove pituitary adenomas.

Sinusitis may be acute or chronic in nature. Presence of pus in the maxillary sinus is detected by decongesting the middle meatus and tilting the patient's head for the dependent drainage, or by nasal endoscopy.

SINUSITIS

Predisposing Factors

- Temperature changes
- Poor general health
- Nasal obstruction
- Deviated nasal septum
- Bronchitis.

Causes

- Acute rhinitis
- Dental infections
- Swimming and diving
- Trauma.

Causative Organisms

- *Rhinovirus*
- *Parainfluenza virus*
- *Echo virus*
- *Coxsackie virus*
- *Respiratory syncytial virus*
- *Pneumococcus*
- *Streptococcus*
- *Staphylococcus*
- *H. influenzae*
- *E. coli*.

ACUTE SINUSITIS (TABLES 2.1 AND 2.2)

Table 2.1: Signs and symptoms of acute sinusitis

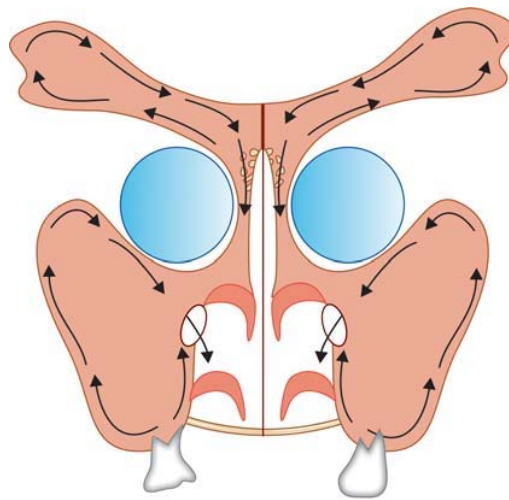
<i>Symptoms</i>	<i>Signs</i>
Fever	Swollen eyelids
Malaise	Flushed cheek
Headache	Tenderness
Nasal discharge	Pus in nasal cavity
Epistaxis	
Anosmia/Cacosmia	
Flat voice	
Pain	
Unpleasant taste	

Headache, fever, nasal blockage and nasal discharge are the common symptoms. Position of the head and time of headache should be enquired from every patient. Presence of pus in the nose and meati confirms the diagnosis. Treatment for acute sinusitis is mainly medical.

Rarely surgical treatment may be required for acute sinusitis with complications.

Table 2.2: Treatment of acute sinusitis

<i>Medical</i>	<i>Surgical</i>
Bed rest	Drainage of sinus
Decongestant nose drops	Endoscopic sinus surgery
Steam inhalation	
Analgesic	
Local fomentation	
Antibiotics (amoxicillin or amoxycillin clavulanate)	

**Fig. 2.9:** Mucociliary flow in the sinuses

Revival of the ciliary activity and provision of adequate drainage through the natural ostium is the keystone to the surgical treatment of sinusitis. A mucociliary flow in various sinuses is shown in Figure 2.9.

Table 2.3: Modes of spread and complications of sinusitis

<i>Sinusitis: Modes of spread</i>	<i>Complications of sinusitis</i>
Direct	Laryngitis
Veins	Pharyngitis
Lymphatic	Osteomyelitis
Perineural	Bronchitis
	Orbital cellulitis
	Polyarthritits

FUNGAL SINUSITIS



Fig. 2.10: Coronal CT scan of a patient with allergic fungal sinusitis



Fig. 2.11: Coronal CT scan of a patient with invasive fungal sinusitis due to mucormycosis.
Note: The breach of lamina papyracea and extension of disease into left orbit

Fungal rhinosinusitis expresses itself in distinct forms that are related to the immunocompetency of the host. The most important distinction is whether the fungus is invasive or noninvasive. Allergic fungal rhinosinusitis (AFRS) is a noninvasive manifestation that occurs because of a hypersensitivity response by the host to the fungus and paradoxically responds to systemic steroids, however, in the invasive fungal sinusitis steroids may be one of the precipitating factors.

Any patient, who is immunosuppressed is at risk for invasive fungal sinusitis. Symptoms of invasive fungal sinusitis may be subtle. Purulent secretion is variable depending on host neutropenia. A wide variety of fungi can cause invasive sinusitis, most commonly *Aspergillus* species and Mucorales (causing mucormycosis). One must assess for palatal erosion, impairment of vision, reduced extraocular movement in cases of invasive mucormycosis. Anesthesia of the face or intranasal area frequently precedes necrosis in mucormycosis. Mottling of turbinates is also an early feature. Frequently nasal mucosa and turbinates are necrotic, insensate and do not bleed on touch or biopsy.



Fig. 2.12: Left frontal mucocele

Mucocele is distention of the sinus with mucoid fluid. Egg shell crackling is characteristic of a thinned out bony capsule. It requires surgical removal with proper drainage of the sinus, by endoscopic sinus surgery or by external or combined approach.



Fig. 2.13: Dental cyst

A dental cyst occurs in relation with an infected tooth while a dentigerous cyst originates in a unerupted tooth. These constitute a group of paranasal cysts and may produce external swelling as well. The treatment is surgical removal.



Fig. 2.14: Left nasolabial cyst.
Note: The fullness in left nasolabial groove

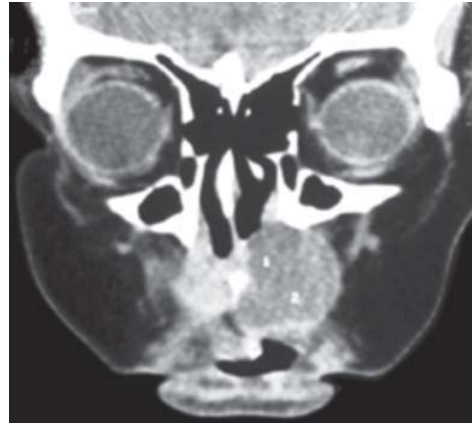


Fig. 2.15: Coronal CT scan of nasolabial cyst (same patient as in Fig. 2.14). Note the homogeneous mass on the left side



Fig. 2.16: Cementifying fibroma (*Source:* Photo by Dr Ashok Vaid)

A young girl presented with a slow progressive protrusion of left eye following a longstanding history of nasal blockage and headache (Figs 2.16 to 2.19).



Fig. 2.17: Note the exophthalmos in same patient as in Figs 2.16 to 2.19 (Source: Photo by Dr Ashok Vaid)

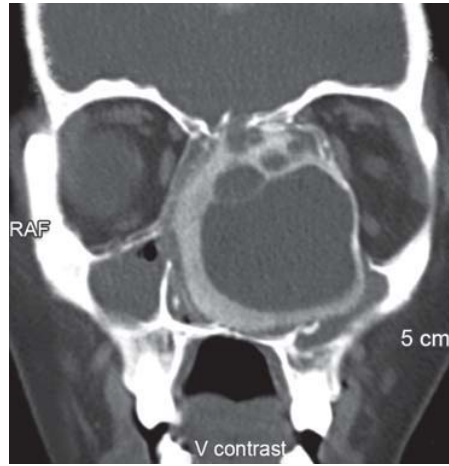


Fig. 2.18: Coronal CT of cementifying fibroma

The lesion is completely occluding both nasal cavities, left maxillary sinus as well as pushing the orbit and reaching up to the anterior cranial fossa.

Axial CT facilitates, the 3D reconstruction of lesion by surgeon for surgical planning (Fig. 2.19).

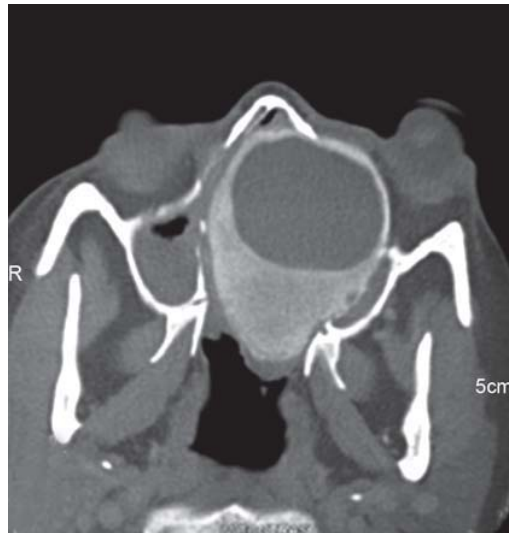


Fig. 2.19: Axial CT cementifying fibroma (Source: Photo by Dr Ashok Vaid)

MALIGNANT TUMORS OF PARANASAL SINUS (TABLE 2.4)

Table 2.4: Malignant tumors of paranasal sinus		
<i>Suprastructure</i>	<i>Infrastructure</i>	<i>Mesostructure</i>
Ethmoid	Floor of antrum	Maxillary antrum
Frontal	Floor of nose	Vestibule of nose
Sphenoid	Hard palate	Nasal septum
Olfactory area	Dental tumor	Lateral nasal wall

All types of benign and malignant tumors occur in paranasal sinuses. Malignant tumors are more common. They can be classified according to the site of origin in the paranasal sinuses. AJCCs. TNM classification is applied to paranasal sinus as well.



Fig. 2.20: Malignant tumor of right maxillary sinus

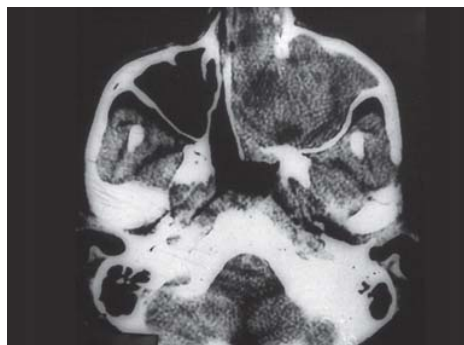


Fig. 2.21: Axial CT scan showing malignancy right nasal cavity and maxillary sinus. The CT scan of the paranasal sinus is invaluable in noting the extent, stage of the disease and in planning the treatment

Maxillary sinus is the most commonly affected site. The most common among the malignant tumors is a squamous cell carcinoma. Photograph shows a patient with malignancy of the maxillary sinus. The treatment for a very early lesion is curative radiotherapy or only surgical excision. A planned preoperative radiotherapy followed by total maxillectomy with or without an orbital exenteration is advised for advanced T3 or resectable T4 lesions.



Fig. 2.22: Blowout fracture of orbit with entrapment of inferior rectus
(Source: Photo by Dr Raj Anand, Oculoplastic surgeon, Delhi)



Fig. 2.23: Clinical photographs of same patient as in Figure 2.22. Top photo is preoperative showing restriction of upward gaze of left eye and lower photo is postoperative showing correction of the restriction after surgical release of inferior rectus and repair of the orbital floor with placement of orbital floor implant
(Source: Photos by Dr Raj Anand, Oculoplastic surgeon, Delhi)

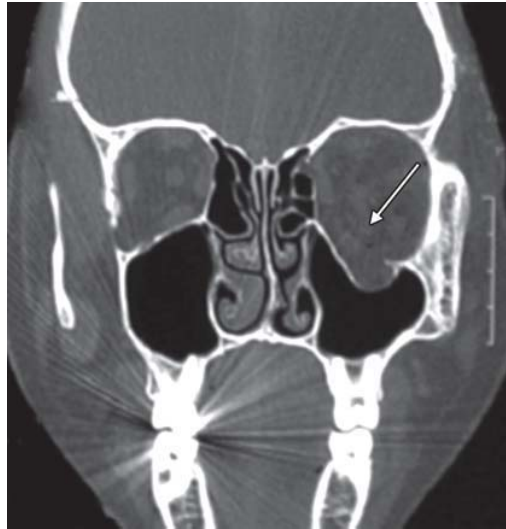


Fig. 2.24: CT: Blowout fracture orbit
(Source: Photo by Dr Raj Anand, Oculoplastic surgeon)

Blunt trauma to the eye is the most common cause of blowout fracture. Note the sagging of the inferior orbital wall (arrow) into the maxillary sinus. Also note the artifact caused by dental filling on right side.

Fracture of the facial bones may involve the nose and the paranasal sinuses. Patient shown had fracture of the frontal process of the maxilla and nasal bones.



Fig. 2.25: Maxillofacial fracture



Fig. 2.26: The management of facial injury should be in collaboration with the maxillofacial surgeon. Picture shows immobilization of the upper jaw with the help of a clamp

Facial pain has varied etiology. A detailed history about the site of pain, time of onset and factors aggravating or relieving the pain, is necessary to determine the cause. Table 2.5 depicts the various causes of facial pain.

Table 2.5: Causes of facial pain	
•	Sinusitis
•	Migrainous headache
•	Anterior ethmoidal neuralgia
•	Facial migraine
•	Trigeminal neuralgia
•	Mandibular malpositioning
•	Nasopharyngeal malignancy
•	Psychological
•	Multiple sclerosis
•	Brainstem lesions

Eye and the Paranasal Sinus

Recurrent epiphora because of dacryocystitis or nasolacrimal duct block is an indication for endoscopic dacryocystorhinostomy.



Fig. 2.27: Landmarks for lacrimal sac area in endoscopic DCR
(Source: Photo by Dr Nishi Gupta)

Landmarks for endoscopic localization of lacrimal sac are the agger nasi prominence, axilla of the middle turbinate, maxillary line (between conchal neck to top of inferior turbinate), ethmoidal bulla (0.5 cm behind it) and uncinat process.

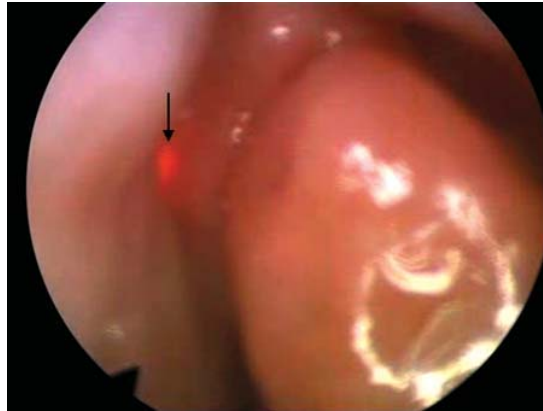


Fig. 2.28: Endoscopic picture of a patient taken up for DCR showing the illumination by light cable inserted through lower punctum (*Source:* Photo by Dr Nishi Gupta)

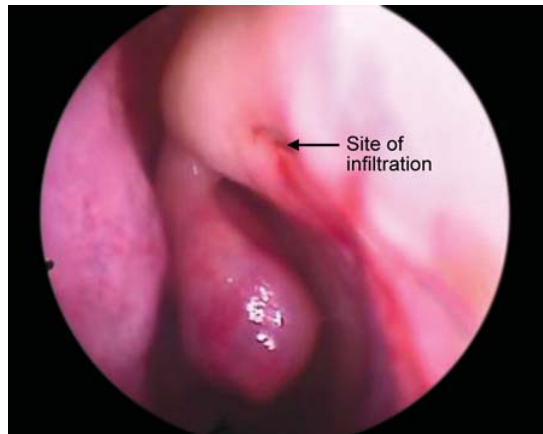
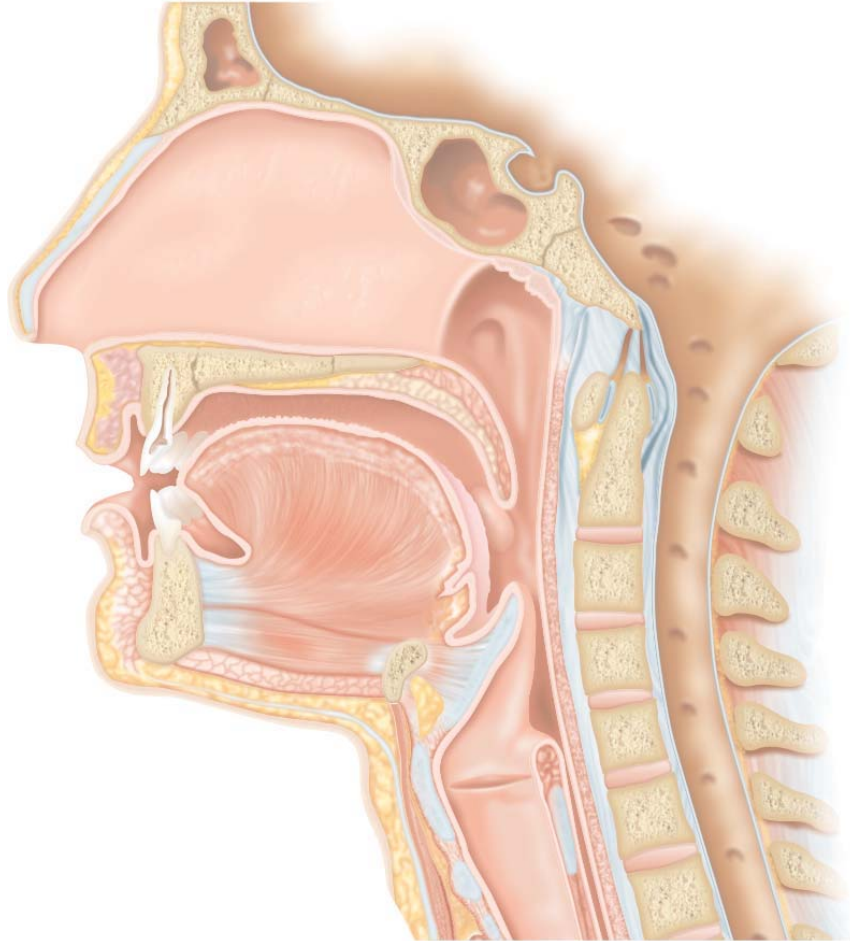


Fig. 2.29: Infiltration at the lacrimal sac area

Pharynx



Pharynx has three distinct parts: Behind the nasal cavity is nasopharynx; behind the oral cavity is oropharynx and below oropharynx is continuous with the laryngopharynx. These have distinct anatomical boundaries (Fig. 3.1).

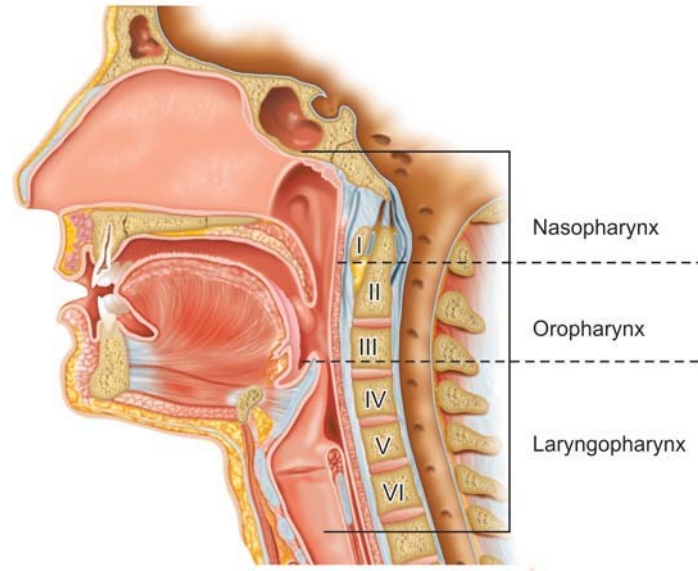


Fig. 3.1: Parts of pharynx

Pharynx is bound by superior, middle and inferior constrictor muscles (Fig. 3.2). It provides a channel for food and respiration. The posterior wall of the oropharynx is formed by 2nd to 4th cervical vertebrae. Superiorly and inferiorly it communicates with other parts of the pharynx. Anteriorly it opens in the oral cavity. Tonsil lies between the anterior and posterior pillars in the lateral wall (Fig. 3.4A). Soft palate closes the roof on swallowing.

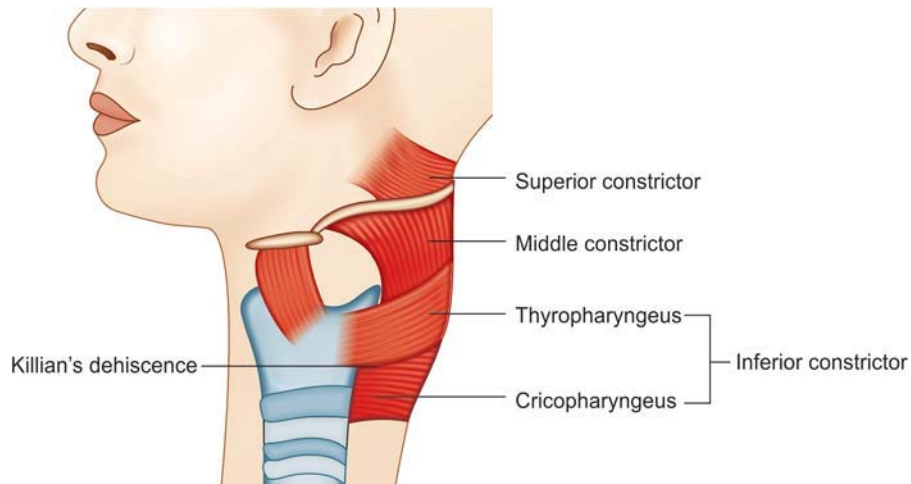


Fig. 3.2: Muscles of pharynx

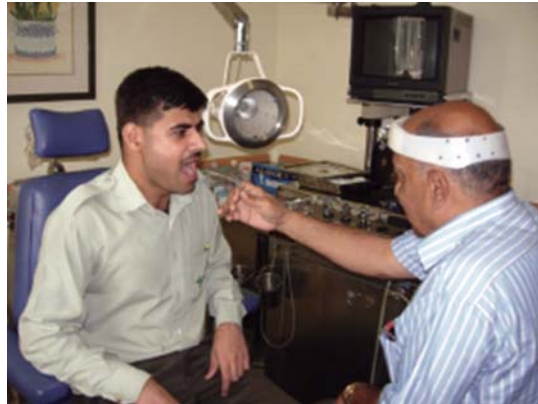
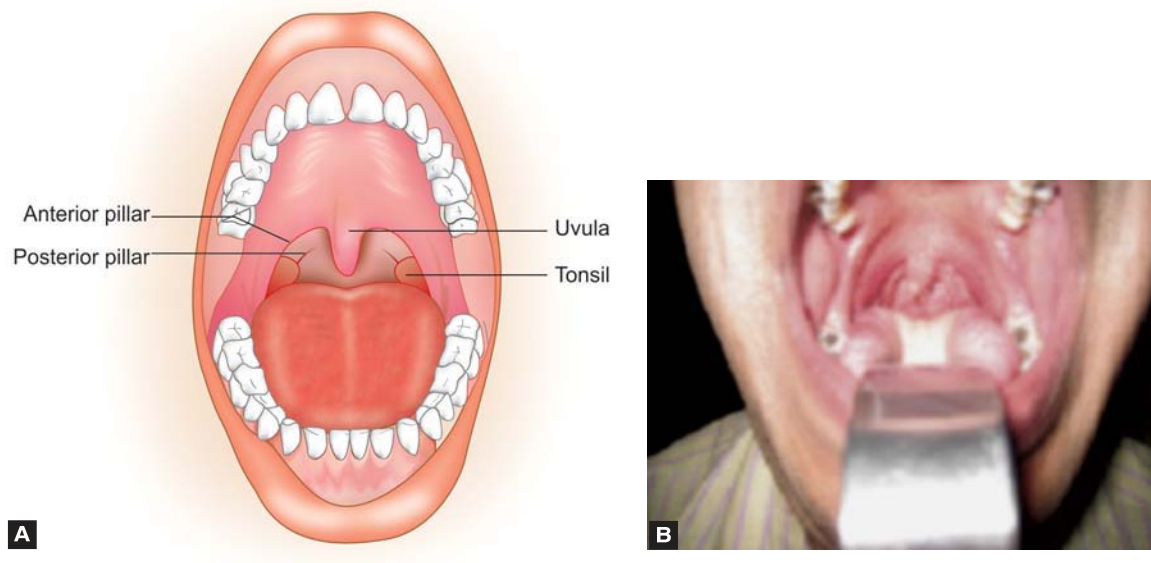


Fig. 3.3: Throat examination



Figs 3.4A and B: Oropharynx

The oropharynx examination needs a good source of light and a tongue depressor to reveal its full anatomy (Fig. 3.4B). The oral cavity, teeth, gingivolabial folds, tongue and its undersurface and hard palate, should be examined at same time.

Tongue depressor should be placed anteriorly to prevent gagging.

MALLAMPATI SCORE (TABLE 3.1)

Table 3.1: Mallampati score used by anesthetists to predict difficult airways is also used to assess snorers

Grade 1:	Faucial pillars, soft palate, and uvula could be visualized
Grade 2:	Faucial pillars and soft palate could be visualized, but uvula was masked by base of the tongue
Grade 3:	Only soft palate could be visualized
<i>Source:</i> Mallampati SR, Gatt SP, Gugine LD, et al. A clinical sign to predict difficult tracheal intubation: A prospective study. <i>Can Anest Soc J</i> 1985;32:429-34.	
<i>Samsoon and Young's Modified Mallampati Classification</i>	
Class 1:	Soft palate, fauces, uvula and pillars seen
Class 2:	Soft palate, fauces and uvula seen
Class 3:	Soft palate and uvula seen
Class 4:	Soft palate not visible at all
<i>Source:</i> Samsoon GL, Young JR. Difficult tracheal intubation: A retrospective study. <i>Anesthesia</i> 1987;42:487-90.	
Classification of oropharyngeal view was assessed with the patient in sitting position with the neck in neutral position and tongue fully protruded without phonation.	



Fig. 3.5: Position for examination of a child



Fig. 3.6: Ankyloglossia. *Note:* The congenital fixation of tongue causing feeding and speech problems

A child should be held by an assistant for examination (Fig. 3.5).



Fig. 3.7: Palpation of pharynx

A direct examination of nasopharynx is possible by a rigid or flexible fiberoptic nasopharyngoscope. A digital palpation of the nasopharynx can also provide information but causes inconvenience to the patient (Fig. 3.7). A lateral soft tissue X-ray of the nasopharynx reveals its outline (Fig. 3.10).



Fig. 3.8: Palpation of neck nodes

Palpation of the submandibular and cervical lymph nodes completes the pharyngeal examination (Fig. 3.8).



Fig. 3.9: Adenoids facies

A child with an open mouth, high arched palate, dull expression, overcrowded teeth and pinched nostrils is said to have an “adenoid facies” (Fig. 3.9). Various features caused by adenoid enlargement are listed (Tables 3.2 and 3.3).



Fig. 3.10: X-ray soft tissue neck lateral view showing adenoid hypertrophy with airway compromise

Table 3.2: Features of adenoid facies

• Mouth breather	• Spongy gums
• Noisy breathing	• Snoring
• Drooling saliva	• Flat chest
• Toneless voice	• Round shoulders
• Pinched nostril	• Deafness
• Prominent incisors	• Bad breath

Table 3.3: Sequelae of adenoid hypertrophy

• Nasal blockage	• Mental lethargy
• Recurrent otitis media	• Impaired speech
• Chronic maxillary sinusitis	• High arched palate.

**Fig. 3.11:** Large kissing tonsils

Tonsil Grading

- Grade 0: The tonsils are fully inside the pillars.
- Grade 1: Tonsils found to be enlarged and out of its pillars.
- Grade 2: Tonsillar enlargement extends just up to half the distance of the uvula.
- Grade 3: Tonsillar enlargement up to the level of the uvula.
- Grade 4: Tonsillar enlargement is so huge that they are virtually in contact with each other, i.e. kissing tonsil.

Chronic tonsillitis is produced as a result of repeated attacks of acute tonsillitis. The tonsils may be enlarged or small. The crypts may lodge debris. Photograph shows enlarged kissing tonsils in a child (Fig. 3.11).



Fig. 3.12: Visible and palpable jugulodigastric node secondary to chronic tonsillitis

Jugulodigastric glands become enlarged because of chronic infection in the tonsils and are seen behind the angle of the mandible.

Table 3.4: Complications of acute tonsillitis

<i>Local</i>	<i>Systemic</i>
• Chronic tonsillitis	• Rheumatic fever
• Suppurative cervical adenitis	• Acute glomerulonephritis
• Parapharyngeal abscess	• Chorea
• Quinsy	• Subacute bacterial endocarditis
• Recurrent otitis media	

Table 3.5: Indications of tonsillectomy

<i>Local</i>	<i>Focal</i>
• Recurrent acute tonsillitis	• Persistent cervical adenitis
• Peritonsillar abscess	• Recurrent attacks of acute rheumatism
• Streptococcal carrier	• Attack of glomerulonephritis.
• Tonsillar tumor	
• Stylalgia	<i>Systemic</i>
• Tonsillolith.	• Failure to thrive in children.

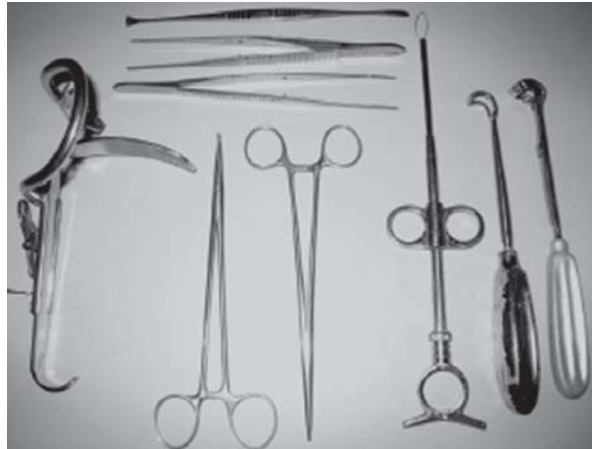


Fig. 3.13: Instruments for tonsillectomy. Tonsillectomy is done by dissection method with cold steel instruments. Newer techniques like coblation, radiofrequency are also becoming popular



Fig. 3.14: Adenotonsillectomy specimen (adenoids do not have a capsule)

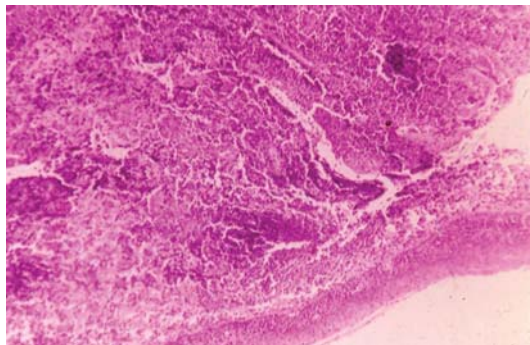
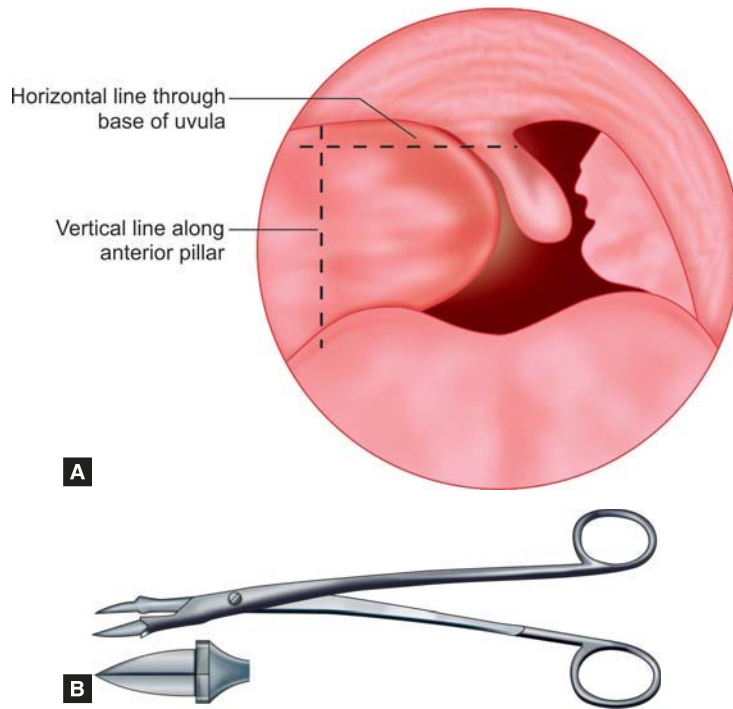


Fig. 3.15: HPE of tonsil specimen showing hyperplasia of lymphoid tissue



Figs 3.16A and B: Right peritonsillar abscess: site for incision is just lateral to the junction of horizontal and vertical lines. Quinsy forceps is shown



Fig. 3.17: Ludwig's angina is infection of submandibular space. Around 80% are due to dental infection



Fig. 3.18: X-ray soft tissue neck lateral view of a 3-year-old male with acute pyogenic retropharyngeal abscess. Treated with incision and drainage with injection metronidazole and clindamycin



Fig. 3.19: Nasopharyngeal angiofibroma. It has a predilection for young males. *Note:* The tumor and blood coming from right nostril. Biopsy is contraindicated. Preoperative embolization of feeding vessels followed by surgical excision is the accepted line of management in most cases



Fig. 3.20: Carotid angiogram showing the tumor blush. Embolization is done preoperatively to prevent excessive blood loss during surgery



Fig. 3.21: Specimen of nasopharyngeal angiofibroma

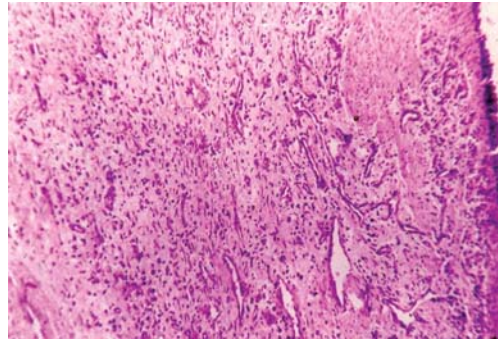


Fig. 3.22: Histopathology of nasopharyngeal angiofibroma

Microscopically the tumor consists of highly fibrous vascular tissue. Note the complete lack of muscular layer which accounts for the profuse bleeding tendency associated with this tumor.



Fig. 3.23: Left tonsillar malignancy. *Note:* The gross asymmetry in size of both tonsils

MALIGNANCY OF THE TONGUE



Fig. 3.24: Malignant ulcer on tongue (arrow)



Fig. 3.25: Squamous cell carcinoma of lateral border of tongue



Fig. 3.26: Patient with nasopharyngeal malignancy

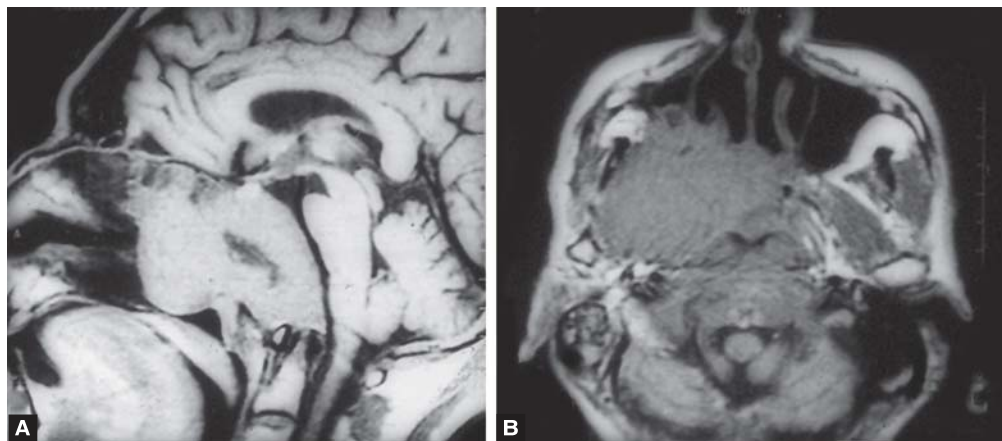
Table 3.6: Symptoms of nasopharyngeal cancer

- Lymphadenopathy (90%)
- Epistaxis
- Nasal blockage
- Ear blockage
- Proptosis

Trotter's triad

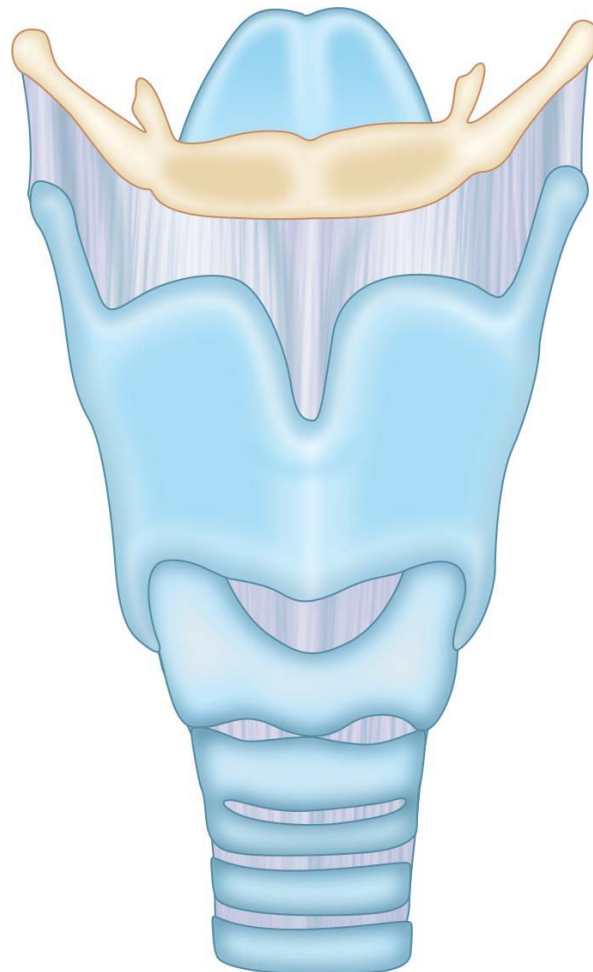
- Ipsilateral conductive deafness
- Ipsilateral temporoparietal neuralgia (Vth nerve)
- Ipsilateral palatal palsy

Malignant nasopharyngeal tumors produce variety of symptoms because of their physical mass and/or by infiltrating character. These often present as secondaries in the neck. In this age group, patients with non resolving/recurrent cases of unilateral otitis media with effusion should undergo nasal endoscopy to rule out nasopharyngeal malignancy.



Figs 3.27A and B: MRI nasopharyngeal malignancy: Sagittal and axial view showing the extent of tumor: Nasopharynx, pterygopalatine and infratemporal fossae

Larynx



Larynx is constituted by various cartilages connected with ligaments covered with muscles and mucosal lining. Single cartilages are epiglottis, thyroid and cricoid while arytenoid corniculate and cuneiform cartilages are paired.

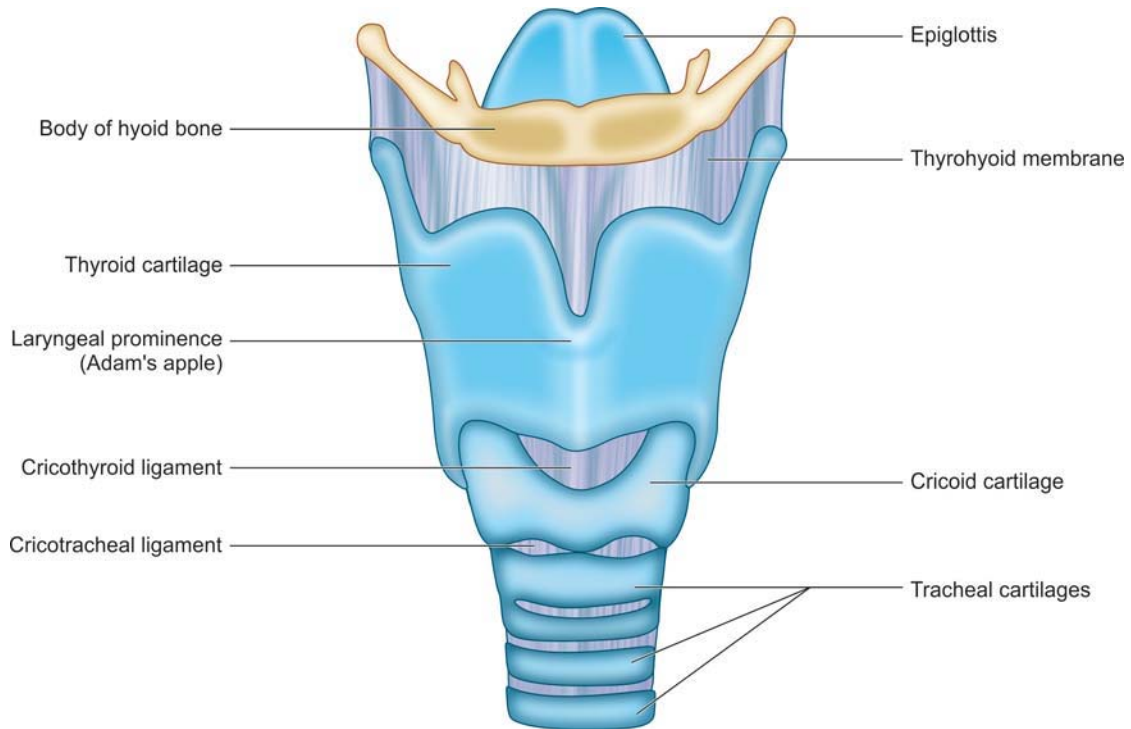


Fig. 4.1: Laryngeal cartilages, ligaments and membranes



Fig. 4.2: Check the laryngeal crepitus

An external examination of the larynx is done to check for widening of the thyroid cartilage and for laryngeal crepitus (movement of thyroid cartilage over the vertebral column). Laryngeal crepitus is absent in advanced post cricoid malignancy.

Table 4.1: Functions of larynx

- Respiratory air channel
- Sphincteric functions
 - Fixation of chest
 - Tussive
 - Protective closure in swallowing and vomiting
- Phonation and speech
- Receptive field for reflexes
 - Cough reflex
- Circulatory function.



Fig. 4.3: Procedure of indirect laryngoscopy

Indirect Laryngoscopy

- The mirror used is plane mirror with a long handle. It is held like a pen in the dominant hand with the mirror pointing downwards.
- The mirror is warmed and the temperature is tested on the back of the hand. This prevents fogging of the mirror.
- The patient is asked to protrude the tongue and tongue is held with gauze.
- The mirror is introduced into the mouth and gently slid under the uvula.
- The mirror is tilted to get good view of the larynx. The patient is asked to say eee. Vocal cord movements are seen on phonation.

The image of larynx visualized is reversed one, the near parts appear the farthest (anteroposterior inversion). The right and left structures are not actually reversed; the right hand structures are seen on the observers left.

The larynx can also be visualized in the outpatient department through a flexible endoscope.

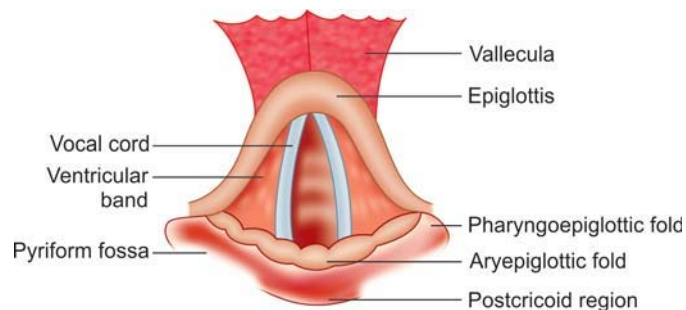


Fig. 4.4: View seen in indirect laryngoscopy



Fig. 4.5: X-ray soft tissue neck lateral view:
Reveals the silhouette of the larynx



Fig. 4.6: Direct examination of larynx in
operating room

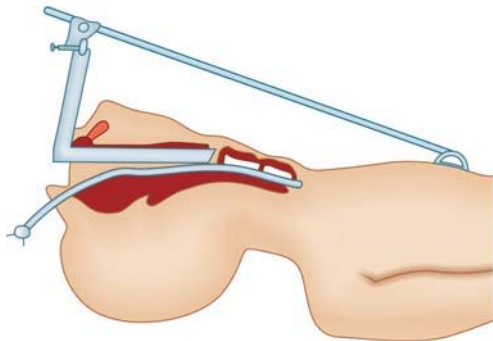


Fig. 4.7: Microlaryngoscope in position



Fig. 4.8: Instruments for microlaryngoscopy

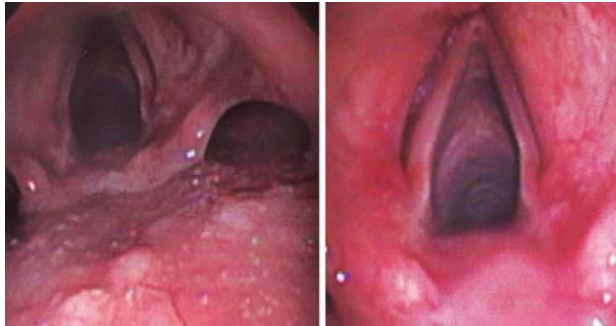


Fig. 4.9: Fiberoptic laryngoscopy view in deep inspiration

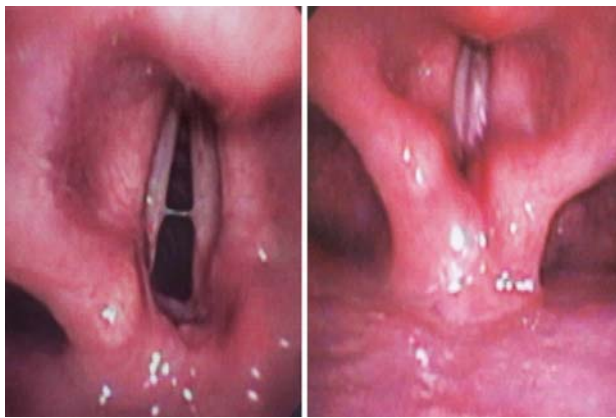


Fig. 4.10: Fiberoptic laryngoscopy view in phonation

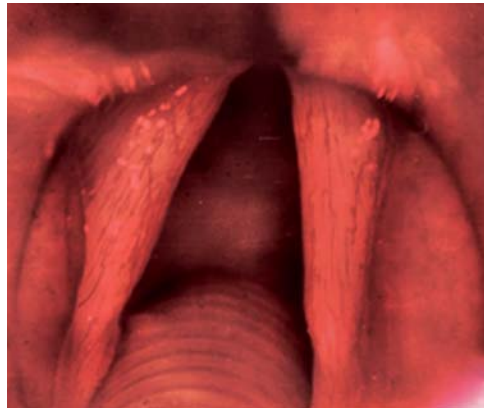


Fig. 4.11: Hyperaemic vocal cords

Acute laryngitis may occur as an independent entity or as a part of generalized upper respiratory infection. It produces hoarseness and examination shows acutely inflamed vocal cords. Complete voice rest, steam inhalation, antibiotics and analgesics provide relief to the patient.



Fig. 4.12: Leukoplakia right vocal cord

Leukoplakia of the larynx is a premalignant condition. Leukoplakia must be biopsied.

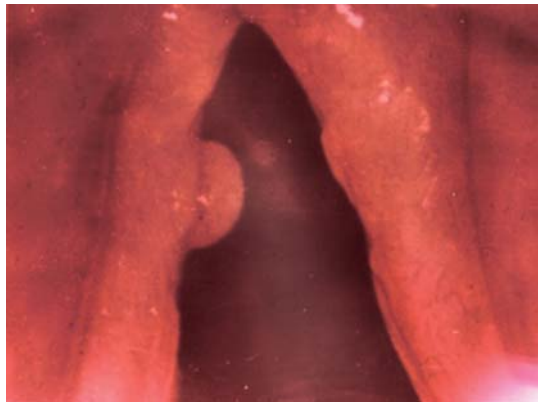


Fig. 4.13: Left vocal cord nodule. *Note:* The irregularity on the corresponding area of other cord

Vocal nodules are localized organization of the fibrous tissue on the edge of the vocal cords. They occur at the junction of the anterior and middle 1/3 of the vocal cords. They are mostly bilateral and need to be removed surgically. Speech training is helpful in long-term results.

Chronic laryngitis is chronic inflammation of the vocal cords. It is commonly seen in professional voice users and may be caused by excessive shouting or smoking. Examination of the larynx will show congestion of the vocal cords. In some cases Reinke's edema sets in for which microlaryngeal surgery is done by microflap technique.

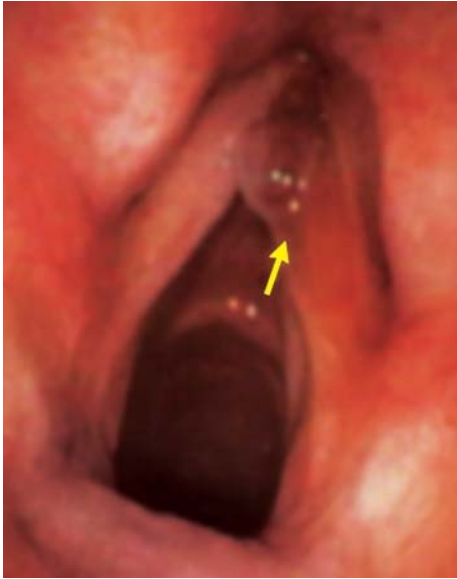


Fig. 4.14: Reinke's edema is caused by subepithelial collection of fluid and pseudo-myxomatous tissue within the lamina propria superficialis of the vocal cord (arrow)

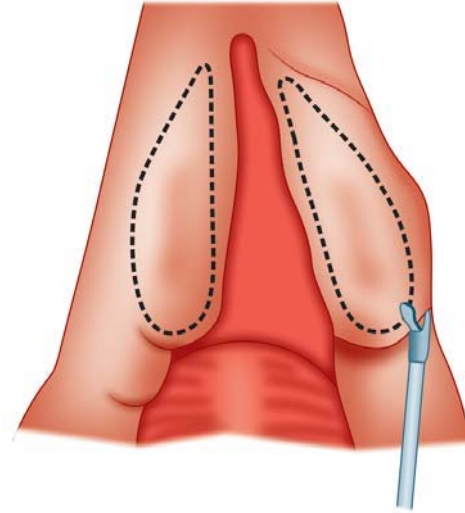


Fig. 4.15: Decortication for Reinke's edema done previously

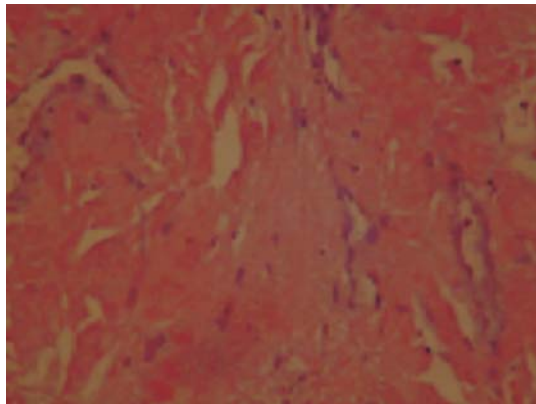


Fig. 4.16: Laryngeal amyloidosis: Congo red staining shows acellular eosinophilic glassy material in the connective tissue and in the walls of blood vessels (Source: Photo by Dr Nandini Vasdev, Pathologist)

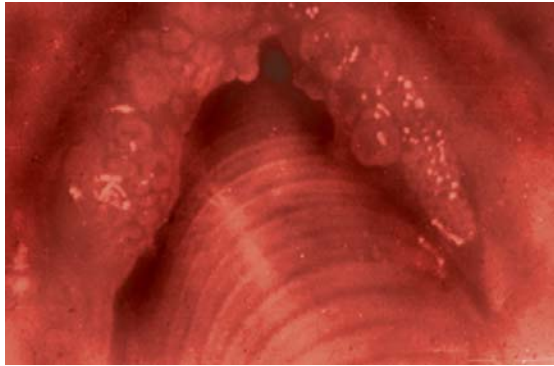


Fig. 4.17: Squamous cell carcinoma involving both vocal cords and anterior commissure

Cancer larynx is common in India. It affects males much more than females. Glottis is the most frequently involved site. The earliest symptom is hoarseness. Microlaryngoscopic view of glottic cancer is shown in the picture above.

Table 4.2: Symptoms and signs of cancer larynx

- Progressive continuous hoarseness
- Dyspnea and stridor
- Pain
- Dysphagia
- Swelling in the neck



Fig. 4.18: Axial CT scan showing laryngeal cancer involving right true and false cords, paraglottic space and involving right thyroid cartilage and sternothyroid muscle

Table 4.3: Classification of cancer larynx

<i>Classification of supraglottic tumors</i>	
T	Primary tumor
Tis	Carcinoma <i>in situ</i>
T1	Tumor limited to one subsite of supraglottis with normal vocal-cord mobility
T2	Tumor invades mucosa of more than one adjacent subsite of supraglottis or glottis or region outside the supraglottis (e.g. mucosa of base of tongue, vallecula and medial wall of piriform sinus) without fixation of the larynx
T3	Tumor limited to larynx with vocal-cord fixation and/or invades any of the following: postcricoid area, pre-epiglottic tissues, deep base of tongue
T4	Tumor invades through thyroid cartilage, and/or extends into soft tissues of the neck, thyroid and/or esophagus
<i>Classification of glottic tumors</i>	
Tis	Carcinoma <i>in situ</i>
T1	Tumor limited to vocal cord (may involve anterior or posterior commissures) with normal mobility
T1A	Tumor limited to one vocal cord
T1B	Tumor involves both vocal cords
T2	Tumor extends to supraglottis and/or subglottis and/or with impaired vocal cord mobility
T3	Tumor limited to the larynx with vocal cord fixation
T4	Tumor invades through thyroid cartilage and/or extends to either tissues beyond the larynx, e.g. to oropharynx, soft tissues of the neck
<i>Classification of subglottic tumors</i>	
Tis	Carcinoma <i>in situ</i>
T1	Tumor limited to the subglottis
T2	Tumor extends to vocal cord(s) with normal or impaired mobility
T3	Tumor limited to the larynx with vocal-cord fixation
T4	Tumor invades through the cricoid or thyroid cartilage and/or extends to other tissues beyond the larynx, e.g. to oropharynx, soft tissues of the neck.

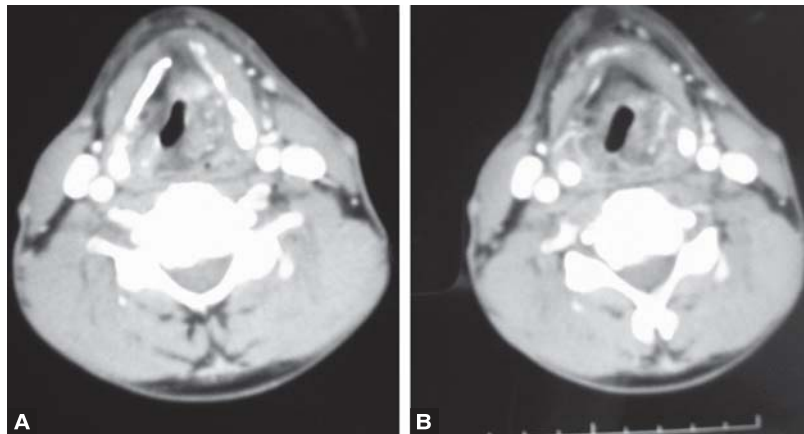
Table 4.4: Staging of cancer larynx

<i>Staging of carcinoma larynx: TNM classification</i>			
Stage 0	TIS	N0	M0
Stage I	T1	N0	M0
Stage II	T2	N0	M0
Stage III	T1	N1	M0
	T2	N1	M0
	T3	N0, N1	M0
Stage IV A	T4	N0, N1	M0
Stage IV B	Any T 1,2,3	N2	M0
Stage IV C	Any T	Any N	M1

Treatment of cancer larynx depends on the staging of the cancer.



Fig. 4.19: Early laryngeal cancer involving right vocal cord (*Source:* Photo by Dr Sowrabh K Arora)



Figs 4.20A and B: Carcinoma laryngopharynx involving right vocal cord, paraglottic space, supraglottis and pyriform fossa (*Source:* Photo by Dr Sowrabh K Arora)



Fig. 4.21: Total laryngectomy specimen

Surgical treatment for cancer of larynx is either total laryngectomy or near total or supraglottic laryngectomy. Resected specimen shows a supraglottic growth.

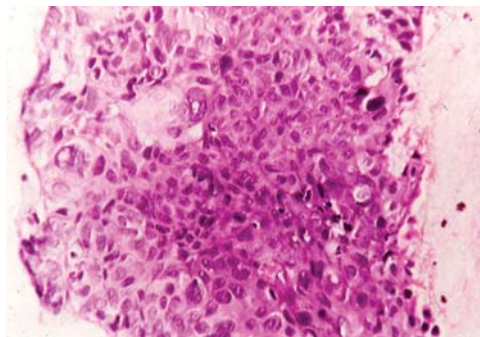


Fig. 4.22: Histopathology of glottic squamous cell carcinoma

Microscopically, the laryngeal tumor consists of a large anaplastic malignant cells.



Fig. 4.23: Laryngectomized patient with permanent tracheostome

Total laryngectomy patient can be rehabilitated by giving training for esophageal speech, electrolarynx or by creating a tracheoesophageal fistula and fitting voice prosthesis in it.

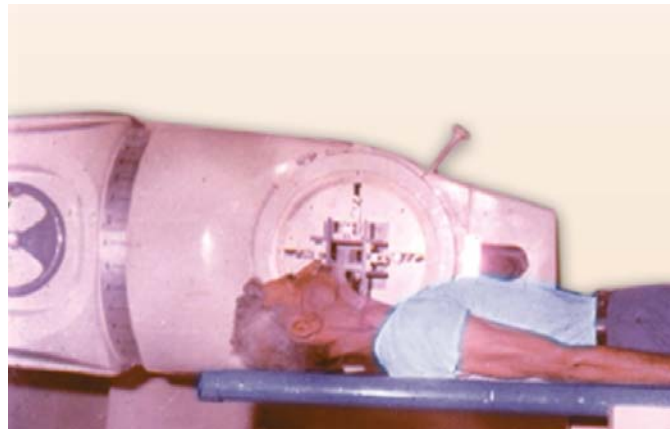


Fig. 4.24: Malignancy larynx being irradiated

Radiotherapy is used in cases with early glottic cancer or if the cancer is too advanced.

An opening made in the trachea to facilitate breathing is called tracheostomy. It is indicated for variety of reasons as shown in Table 4.5.



Fig. 4.25: Patient with tracheostomy tube in position

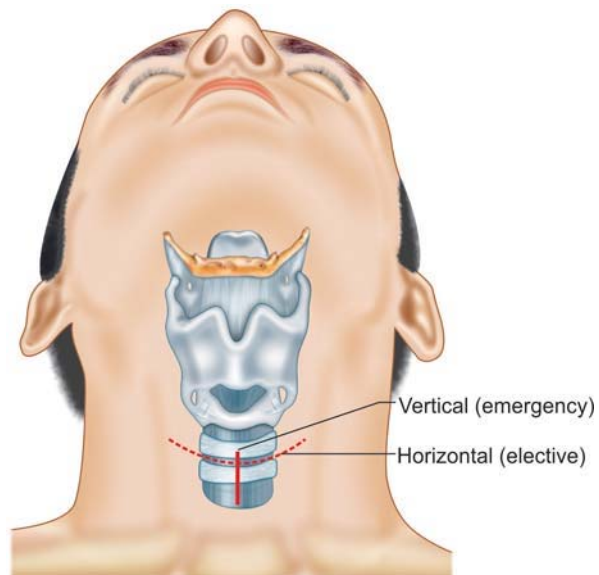


Fig. 4.26: Incisions for tracheostomy



Fig. 4.27: Instruments for tracheotomy

Tracheostomy is either performed as an emergency or a planned procedure with a set of tracheostomy instruments as shown in Figure 4.27.

Table 4.5: Indications for tracheostomy

<i>Relief of respiratory obstruction</i>	
Congenital	<ul style="list-style-type: none"> • Bilateral choanal atresia • Laryngeal web • Tracheal stenosis • Tracheoesophageal anomaly
Traumatic	<ul style="list-style-type: none"> • Blow on larynx • Open neck wounds • Inhalation of fumes • Swallowing of corrosives
Infections	<ul style="list-style-type: none"> • Acute epiglottitis • Laryngeal diphtheria
Tumors	<ul style="list-style-type: none"> • Tongue • Pharynx • Larynx • Thyroid
Bilateral Abductor Palsy	<ul style="list-style-type: none"> • Post-thyroidectomy • Bulbar paralysis • Vocal cord fixation • Rheumatoid arthritis • Gout
<i>Protection of tracheobronchial tree</i>	
	<ul style="list-style-type: none"> • Bulbar polio
	<ul style="list-style-type: none"> • Polyneuritis
	<ul style="list-style-type: none"> • Tetanus
	<ul style="list-style-type: none"> • Myasthenia gravis
	<ul style="list-style-type: none"> • Head injury
	<ul style="list-style-type: none"> • Barbiturate poisoning
	<ul style="list-style-type: none"> • Cardiac asthma
<i>Respiratory insufficiency</i>	
	<ul style="list-style-type: none"> • Chronic bronchitis
	<ul style="list-style-type: none"> • Emphysema
	<ul style="list-style-type: none"> • Postoperative pneumonia
	<ul style="list-style-type: none"> • Flail chest



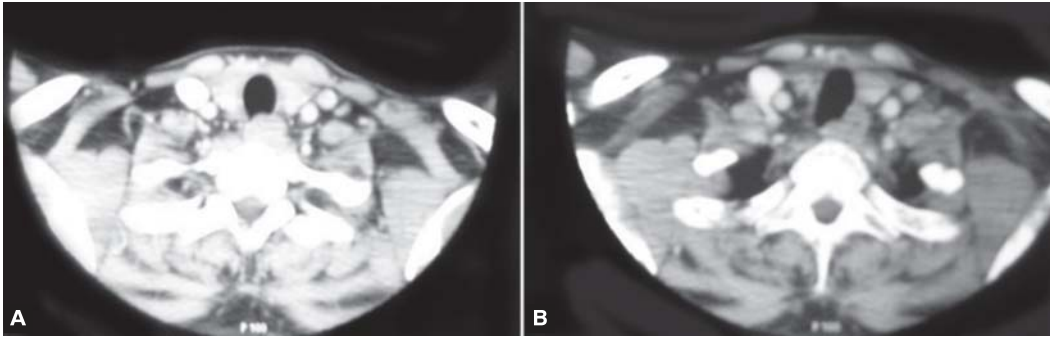
Fig. 4.28: Surgical emphysema following tracheostomy

Tracheostomy has some hazards. Sometimes the patient may get apneic or develop surgical emphysema as shown in the skiagram. Dry air produces crusting of the tracheal secretions which sometimes produces tracheal casts. It can be prevented by providing humidified air and repeated tracheal suction.



Fig. 4.29: Hypopharyngeal malignancy presenting as neck swelling (*Source:* Photo by Dr Sudhir Bahadur)

Progressive dysphagia, irritation in the throat and involvement of neck nodes are some features of hypopharyngeal malignancy. This can be diagnosed by X-ray lateral view of the soft tissues of the neck, barium swallow, CT neck, direct hypopharyngoscopy and biopsy.



Figs 4.30A and B: Carcinoma hypopharynx involving postcricoid area and left pyriform fossa
(Source: Photo by Dr Sowrabh K Arora)

Table 4.6: Hypopharyngeal malignancy	
<i>Signs and symptoms</i>	
•	Neck swelling
•	Swallowing difficulty
•	Ear blockage/ache on same side as swelling
<i>Investigations</i>	
•	CT scan with contrast
•	MRI
<i>Treatment</i>	
•	Laryngopharyngectomy
•	Radiation

The treatment of the malignancy of the hypopharynx depends on the extent of the disease and is possible through laryngopharyngectomy, radiation or a combination therapy.

Vocal cord paralysis may be because of paralysis of adductor or abductor muscles. It may be unilateral or bilateral. Semons' law postulates that in paralysis of organic origin abductors are affected before adductors.

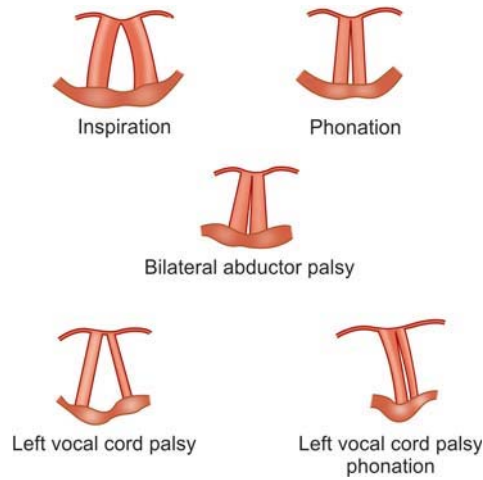


Fig. 4.31: Various positions of vocal cord

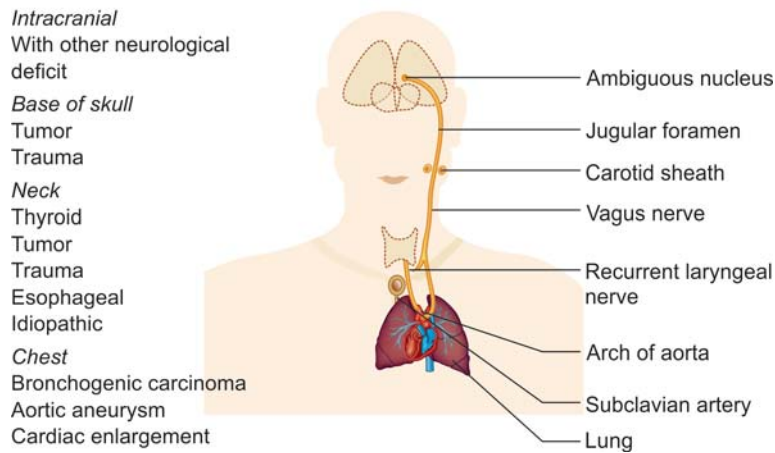


Fig. 4.32: Left recurrent laryngeal nerve: Causes of paralysis and sites

A patient presenting with aphonia with a normal sounding cough is functional. Pure adductor paralysis is functional. Causes of vocal cord palsy are listed in the Figure 4.32.

5

PS Saharia, Deepti Sinha

Ear



The ear is divided into external ear, middle ear and internal ear. The external ear consists of the auricle or pinna, external auditory canal and tympanic membrane.

Outer one-third of the canal is cartilaginous and inner two-thirds is bony. External auditory canal (EAC) measures 2.4 cm in adults and is tortuous, while it is short and wide in children.



Fig. 5.1: Examination of ear

In adults pinna is pulled outwards and downwards and in children outwards and upwards to straighten the canal for a clear view of the tympanic membrane.



Fig. 5.2: External ear examination with ear speculum

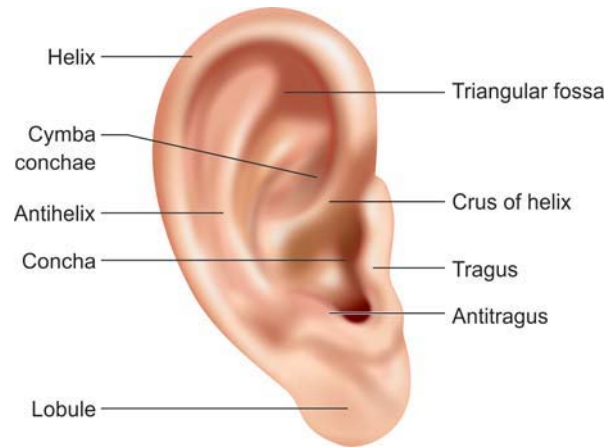


Fig. 5.3: Auricle

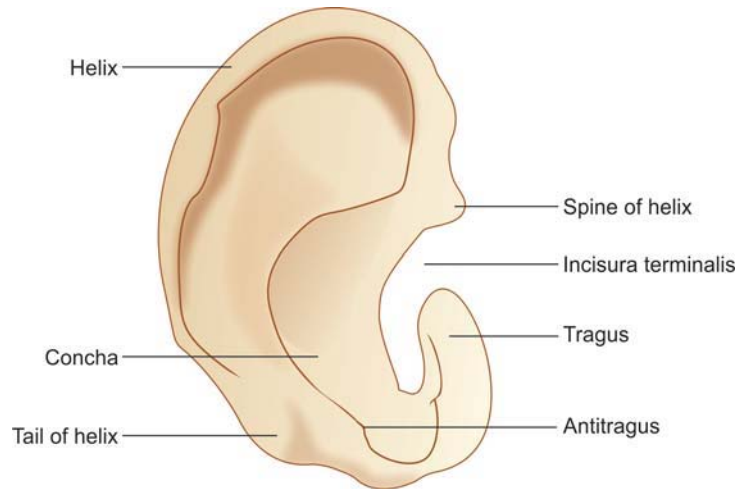
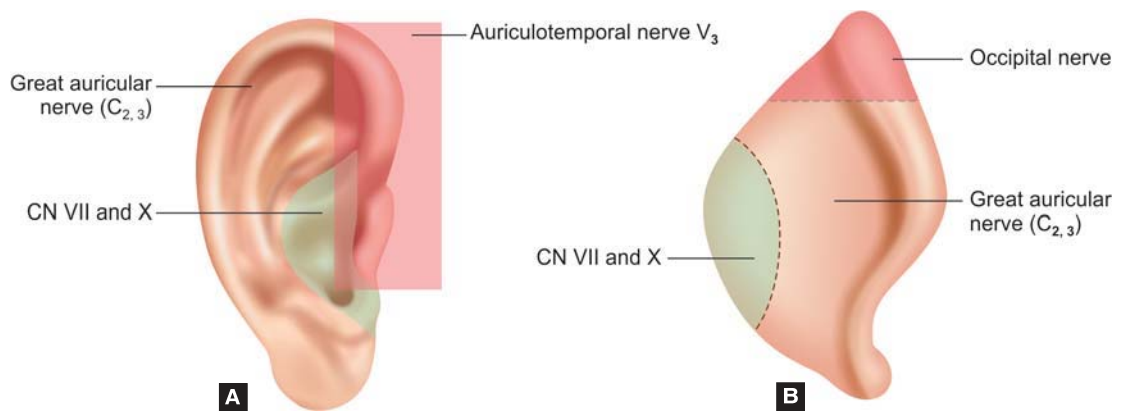


Fig. 5.4: Auricular cartilage



Figs 5.5A and B: Nerve supply of lateral surface and medial surface of auricle



Fig. 5.6: Press tragus to elicit any tenderness in case of otitis externa. Acute Otitis externa is treated with oral and topical antibiotics, analgesics and aural toilet

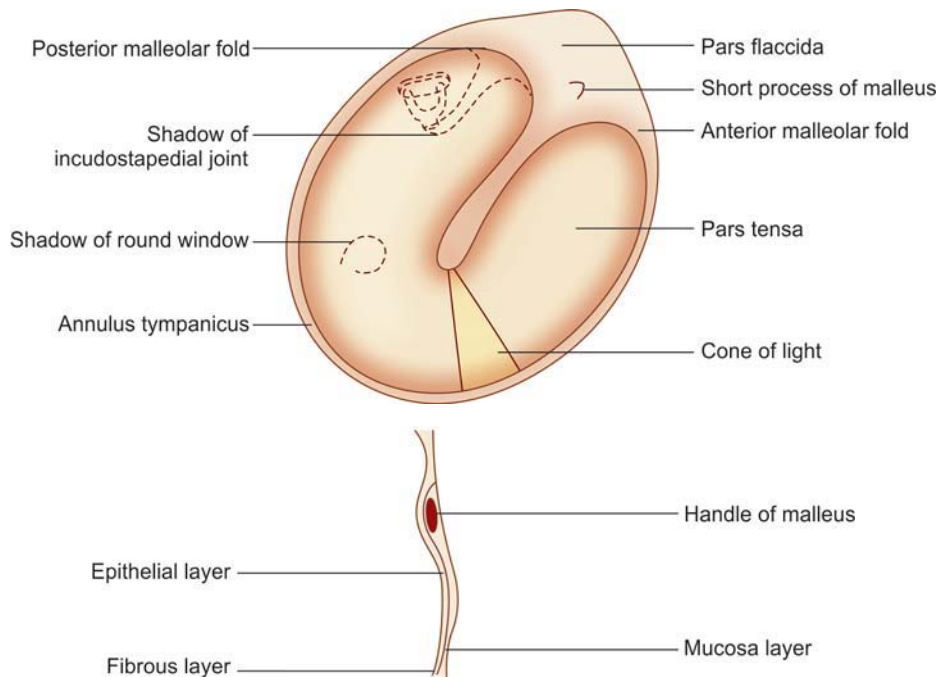


Fig. 5.7: Tympanic membrane is 9–10 mm long, 8–9 mm wide and 1 mm thick

Tympanic membrane (TM) consists of three layers: outer epithelial, middle fibrous and inner layer of mucosa. The drum lies in the annulus tympanicus. Tympanic membrane is divided into pars tensa and pars flaccida. The handle of malleus, short process of malleus, anterior and posterior malleolar folds, umbo and cone of light are the normal landmarks.



Fig. 5.8: Normal tympanic membrane (Left side). Note: The cone of light anteroinferiorly and shadow of ossicular chain posterosuperiorly.

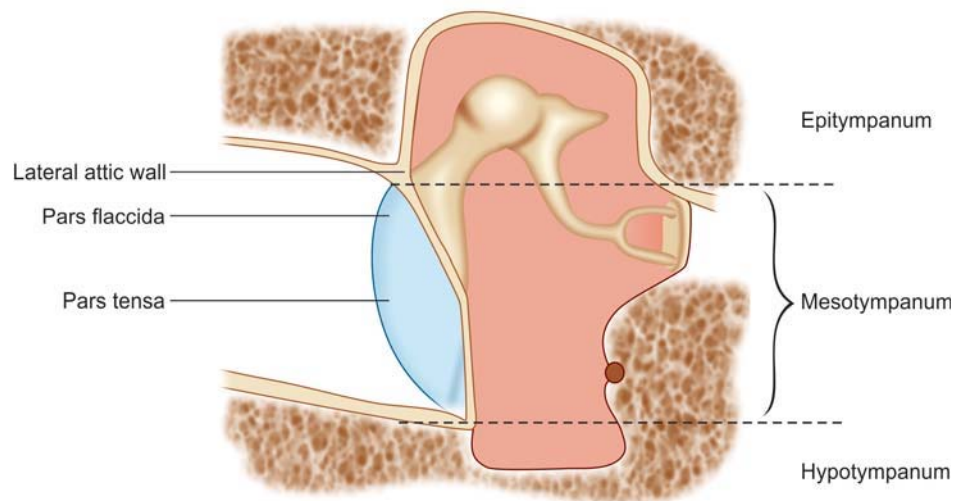


Fig. 5.9: Middle ear cavity can be divided into epitympanum, mesotympanum and hypotympanum

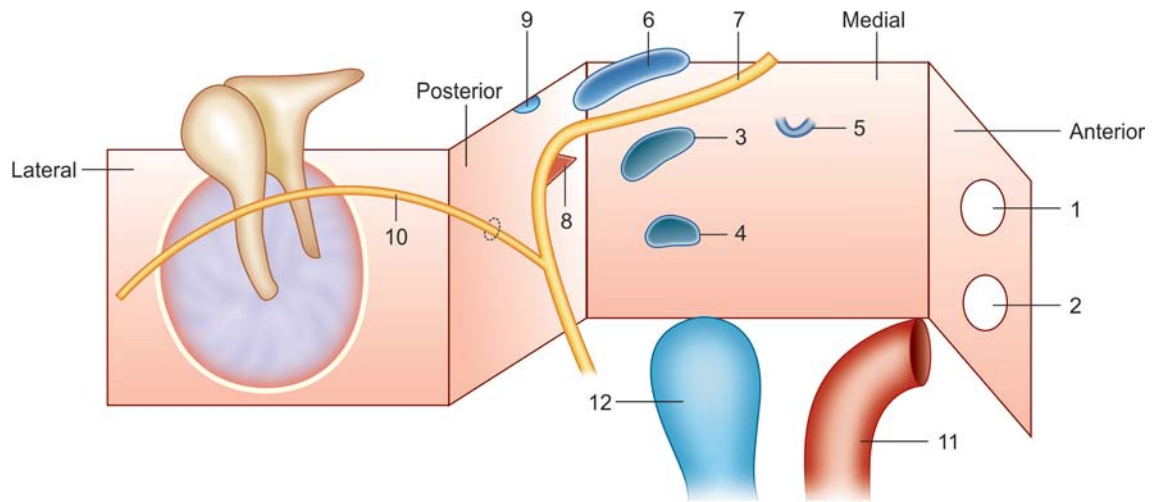


Fig. 5.10: Relations of middle ear

1. Canal for tensor tympani; 2. Opening of Eustachian tube; 3. Oval window; 4. Round window; 5. Processus cochleariformis; 6. Lateral semicircular canal; 7. Facial nerve; 8. Pyramid; 9. Aditus; 10. Chorda tympani; 11. Carotid artery; 12. Jugular bulb

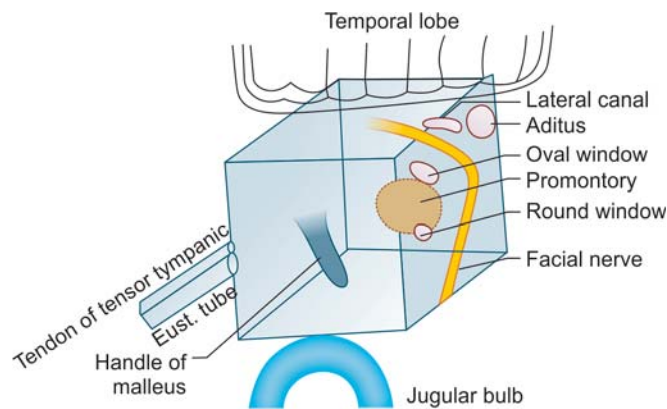


Fig. 5.11: Middle ear cleft

Diagram shows left middle ear, its contents and anatomical relations.

Internal ear comprises of a bony labyrinth embedded in the petrous temporal bone and within it lies the membranous labyrinth. Membranous labyrinth is filled with endolymph. Space between bony and membranous labyrinth is filled with perilymph.

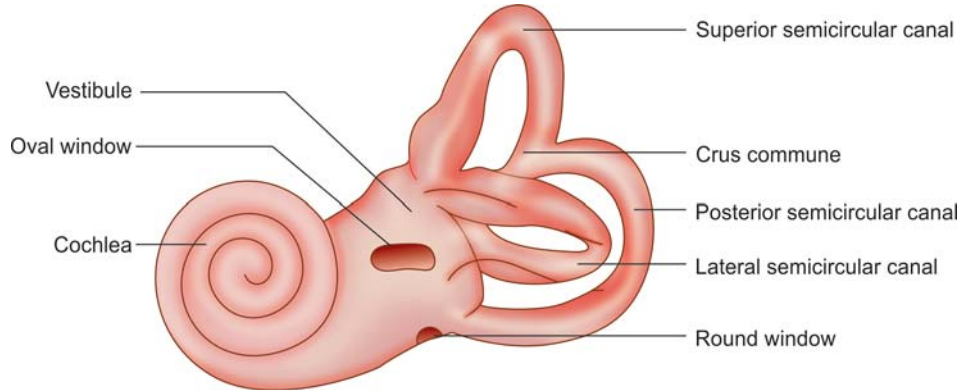


Fig. 5.12: Left bony labyrinth, its consists of vestibule, semicircular canals and cochlea

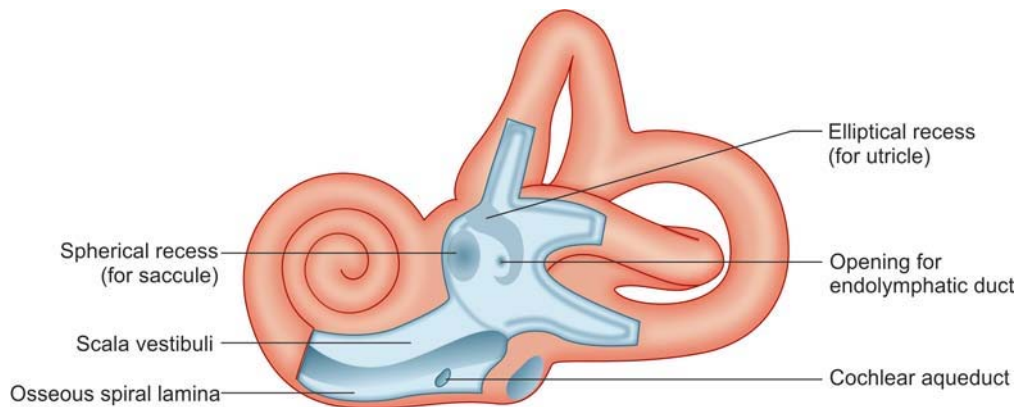


Fig. 5.13: Cut section of bony labyrinth

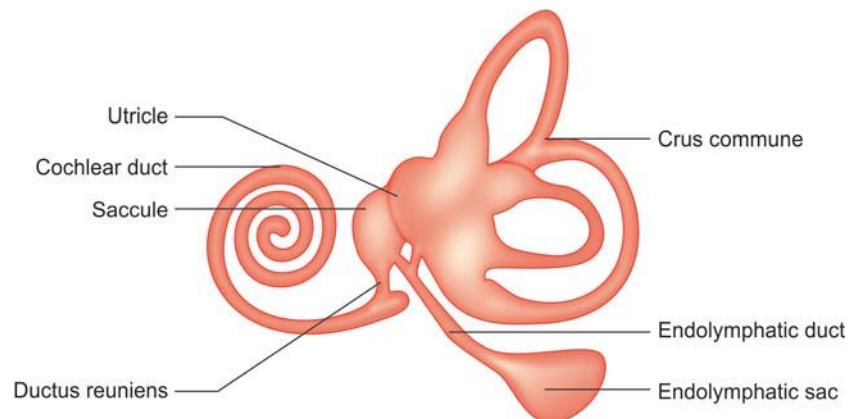


Fig. 5.14: Membranous labyrinth

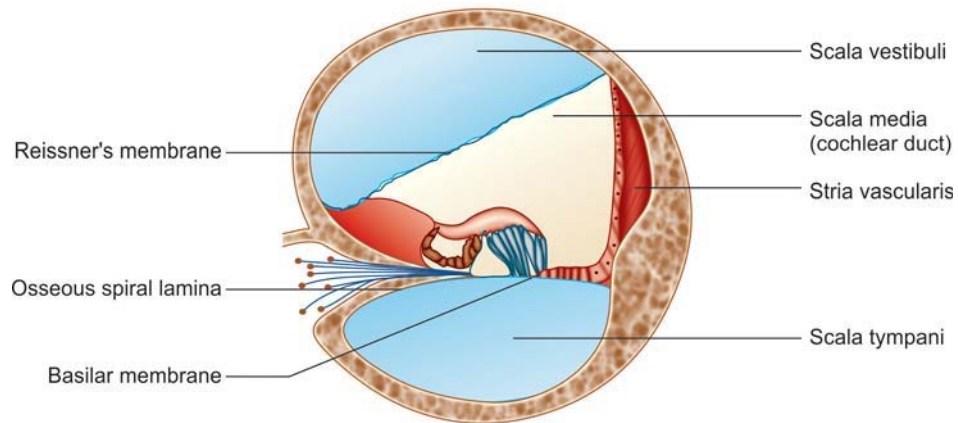


Fig. 5.15: Cross section of cochlea showing the organ of Corti

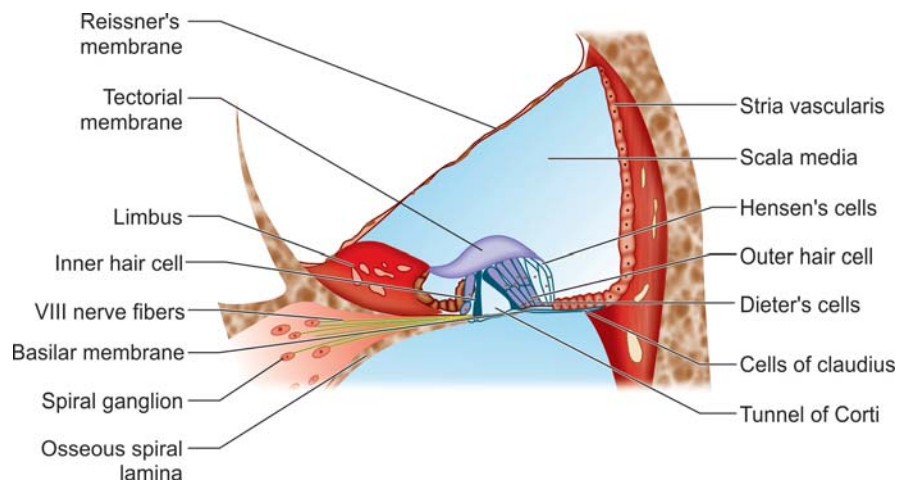


Fig. 5.16: Organ of Corti

Organ of Corti is the end organ for hearing and lies in scala media.

PHYSIOLOGY OF HEARING

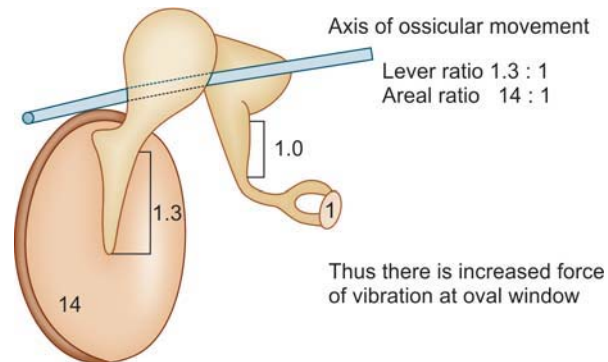


Fig. 5.17: Transformer action of the middle ear. Total transformer ratio $14 \times 1.3 = 18.2 : 1 = 18.1$

Sound waves traverse the external auditory canal and strike the tympanic membrane causing it to vibrate. These vibrations set the ossicular chain in motion and convey it to the stapes footplate which seals the oval window. The difference in the lengths of the malleus handle and long process of incus (Lever Ratio) as well as the difference in the vibratory surface of the tympanic membrane and stapes footplate (Areal ratio) amplifies the sound.

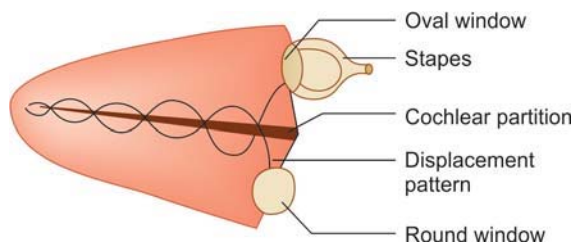


Fig. 5.18: Physiology of hearing

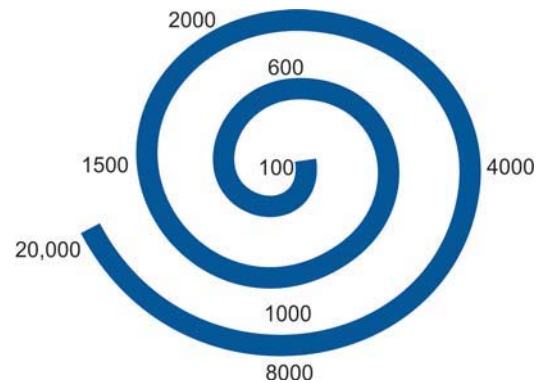


Fig. 5.19: Frequency specific areas of cochlea

The footplate of the stapes which rests on the oval window conducts the vibrations into the internal ear. The travelling wave on the basilar membrane produces electrical potentials which are conducted via the VIII nerve to the auditory area of the cerebral cortex. It is the auditory area which interprets the electric signals as speech.



Fig. 5.20: Eustachian tube patency test—Valsalva's maneuver



Fig. 5.21: Eustachian tube testing—Poltizerization

Eustachian tube connects the nasopharynx with the tympanic cavity. The objective of Valsalva's maneuver and politzerization is to build positive pressure in the nasopharynx so that air enters the Eustachian tube.

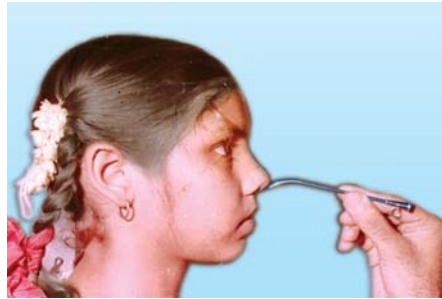


Fig. 5.22: Eustachian catheterization

Table 5.1: Functional examination of the ear

Hearing

- Voice and whisper tests
- Tuning fork tests
- Pure tone audiometry
- Tympanometry

Balance

- Observe for nystagmus
- Fistula test
- Positional test
- Cold caloric test
- Differential caloric test
- Rotation test
- Electronystagmography

HEARING ASSESSMENT



Fig. 5.23: Tuning fork testing—Rinne test: Air conduction

Rinne test compares the air and bone conductions. Normally, the air conduction is better than bone conduction and their ratio is taken as positive ratio and hence Rinne positive. When the ratio is reversed, the test is said to be negative. Similarly, both air and bone conduction can be heard. Always ask the patient whether air conduction is heard. In patients with dead ear, air conduction is not heard whereas bone conduction is heard through the other side and should be recorded as 'False negative Rinne'.



Fig. 5.24: Rinne test: Bone conduction

A negative Rinne for 256, 512, 1024 Hz tuning fork indicates at least an air-bone gap of 15, 30 and 45 dB respectively.



Fig. 5.25: Weber's test

Weber's test compares the degree of deafness in both ears. In unilateral conductive loss the Weber's test is lateralized to the affected ear while in perceptive deafness, it is lateralized to the better hearing ear. Lateralization occurs at a difference of 5 dB between the ears.



Fig. 5.26: Tuning fork test—absolute bone conduction test

The absolute bone conduction (ABC) is a comparison of hearing duration of the patient with that of a normal hearing person. It is either normal or shortened in comparison to examiner. Examiners' hearing should be normal for a valid comparison.



Fig. 5.27: Masking with Barany noise box

Masking is done to prevent the non test ear from participating in the test. Masking the normal ear with white sound is done by a Barany noise box. It may be necessary to mask the normal ear in cases with unilateral hearing loss.

Hearing can be measured and recorded by a pure tone audiometer in a sound treated room. Various frequencies are presented at different intensities and the patient responds at the minimal hearing level and an audiogram is charted.



Fig. 5.28: Pure tone audiometry. Hearing loss can be conductive, sensorineural or mixed (both conductive and sensorineural). Pure tone audiometry is done to assess hearing across the frequency range (250 Hz-6000 K Hz) at different loudness levels

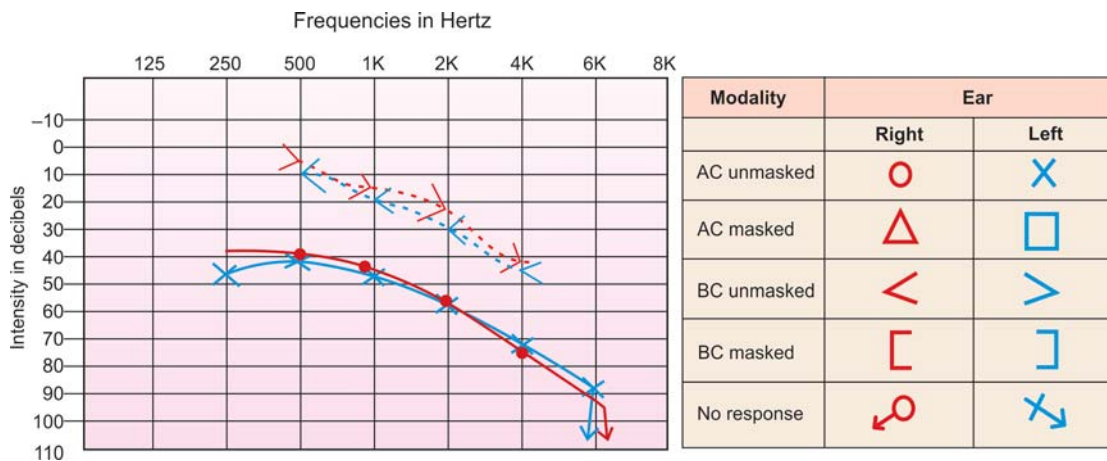
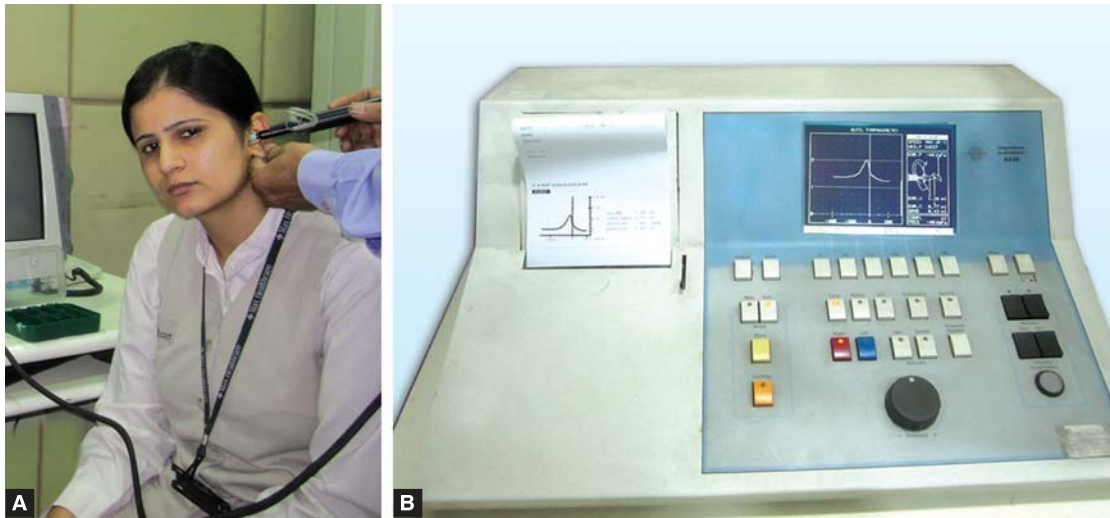
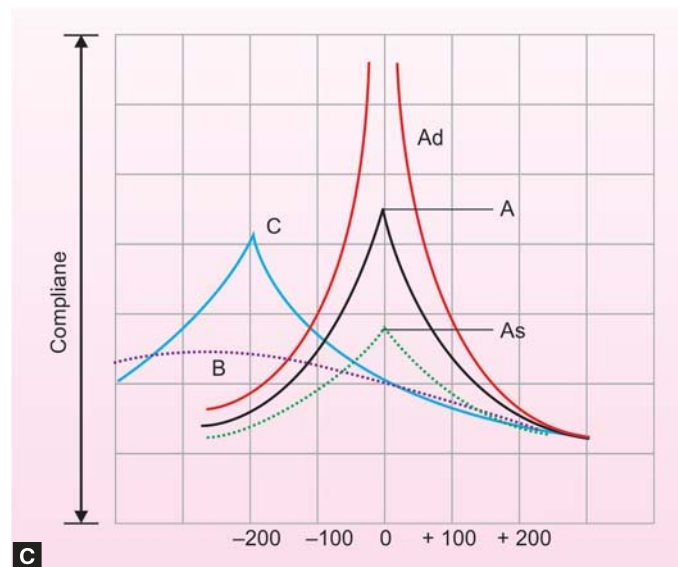


Fig. 5.29: Audiogram showing bilateral mixed deafness



Figs 5.30A and B: (A) Tympanometry; (B) Tympanometer



Figs 5.30C: Types of tympanograms—A: Normal; As: Reduced compliance at ambient pressure (otosclerosis); Ad: Increased compliance at ambient pressure (ossicular discontinuity); B: Flat or dome-shaped (fluid in middle ear); C: Maximum compliance at pressures more than -100 mm H_2O (negative pressure in middle ear)

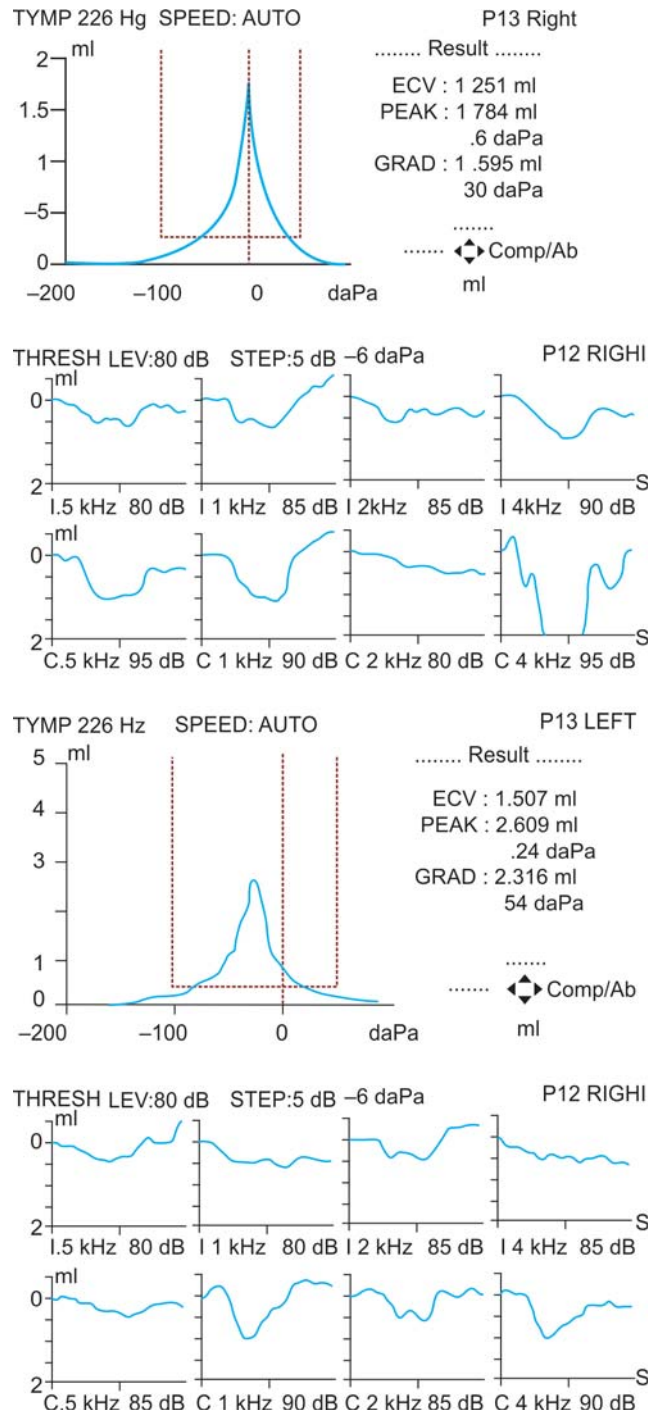
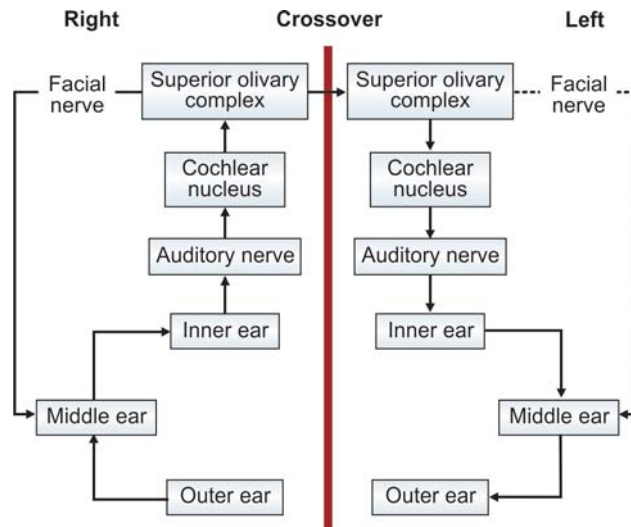


Fig. 5.31: Tympanogram

Tympanometry is recording of the pressure changes in the middle ear cleft.

Flow chart 5.1: Acoustic reflex pathway**Table 5.2:** Acoustic reflex

- Normal: Present at 70 to 100 dB HL
- Reveals the integrity of auditory and facial nerves
- Cochlear implant (CI) candidates: Usually absent because of the amount of hearing loss.

Acoustic reflex is based on the fact that a loud sound (70–100 dB suprathreshold) will cause bilateral contraction of stapedius muscles which can be detected by tympanometry. It is an objective hearing test for infants and young children. It can also be used to identify malingerers feigning total deafness.

For children different tests are used to estimate hearing.

Hearing Test for Children

Table 5.3: Behavioral observation audiometry

Unconditioned Response Procedures

- Children of age range: 8–12 months old
- Observe the child's response to sound

Advantages

- It can be used with children who cannot be conditioned

Disadvantages

- Difficult to eliminate tester bias
- Responses of infants and young children are quick to reach extinction without reinforcement
- Wide variance of responses
- Only sensitive to patients with severe to profound hearing losses
- Not sensitive to unilateral hearing loss

Table 5.4: Visual reinforcement audiometry

Conditioned Response Procedures

- Conditioning: Head turn
- Reinforcement: Lighted/animated toy
- Age range: 6–30 months old

Advantages

- It can present stimuli through speakers, earphones or bone oscillator
- Less intersubjective and intrasubjective variability
- It can obtain minimal response to level close to threshold
- Sensitive to even mild hearing losses

Disadvantages

- Some infants cannot be conditioned until about 12 months
- Many infants will not tolerate earphones or will not turn their heads with earphones

Conditioned Response Procedures

Table 5.5: Conditioned play audiometry

Conditioning

- Play activity
- Reinforcement: Play activity and social. It may also use visual reinforcement
- Age range: 30 months–4 years

Advantages

- Accurate thresholds can be reliably obtained
- It can be accomplished with traditional equipment
- Stimuli can be presented through speakers, earphones, or the bone oscillator

Disadvantages

- They may have to change activities many times to keep child's interest
- Child may need to be re-conditioned when activities change

Objective Measurements

Table 5.6: Objective measurements in evaluation of deaf child

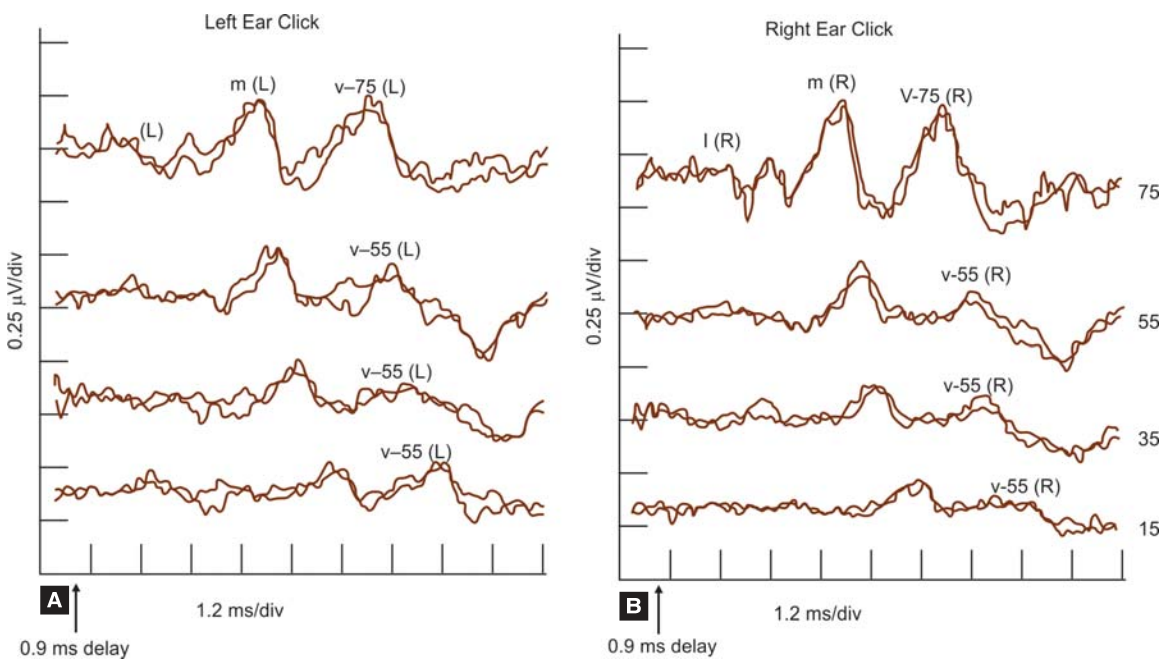
- Tympanometry and acoustic reflex
- Brainstem evoked response audiometry (BERA)
- Auditory steady state response (ASSR)
- Otoacoustic emissions (OAEs)

Table 5.7: Otoacoustic emissions: Clinical applications

- Newborn screening
- Pediatric audiometry
- Functional hearing loss
- Differentiation of cochlear vs retrocochlear dysfunction
- Monitoring hearing changes for ototoxicity and/or noise exposure.

Table 5.8: Auditory brainstem responses (ABR)

• Wave I	Cochlear nerve
• Wave II	Cochlear nucleus
• Wave III	Superior olivary complex
• Wave IV	Lateral lemniscus
• Wave V	Inferior colliculus



Figs 5.32A and B: ABR waveforms

VESTIBULAR FUNCTION TESTS



Fig. 5.33: Fistula test

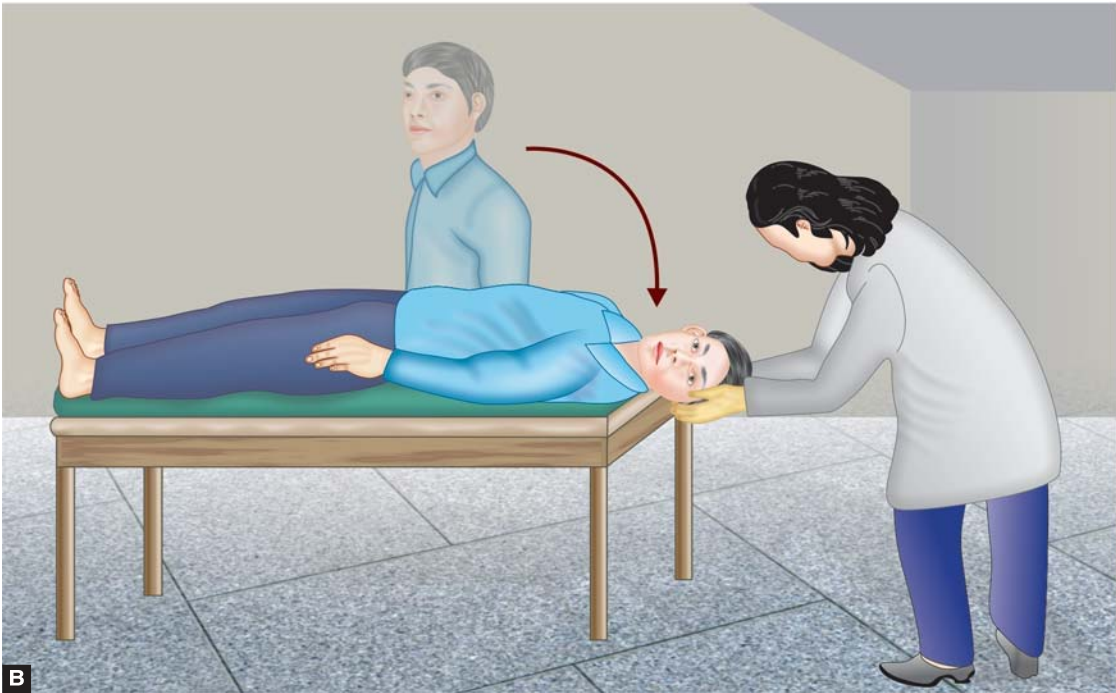
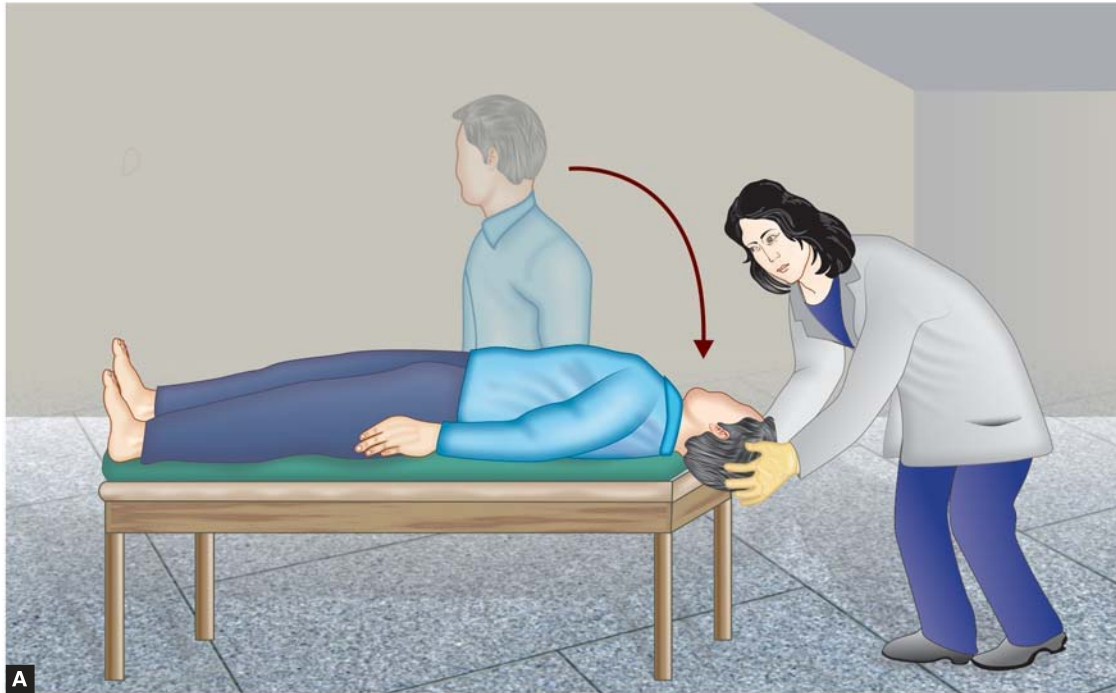
Fistula test is carried out to detect the presence of fistula in the lateral semicircular canal. It is positive when patient on intermittent pressing and releasing the tragus, complains of vertigo and develops nystagmus. Negative test signifies either a normal labyrinth or a completely dead labyrinth (False negative fistula test).



Fig. 5.34: Walking on straight line test



Fig. 5.35: Head rotation test



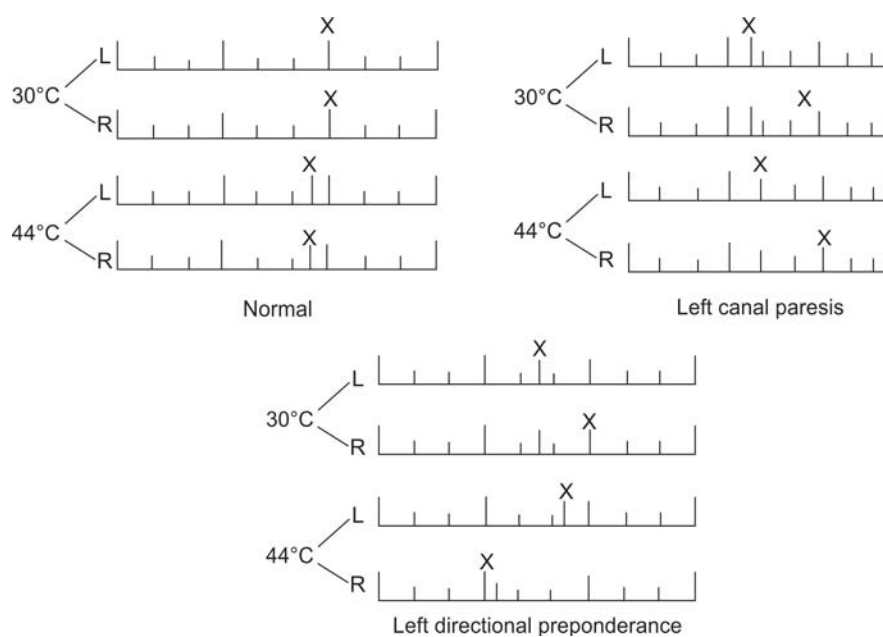
Figs 5.36A and B: Dix Hallpike test for testing positional vertigo

Table 5.9: Caloric test

Kobrak cold caloric test is done with ice cold water
Differential caloric test is done with water at 30° and 44°C
 responses are as follows:

<i>Symptoms</i>	<i>Sign</i>
Vertigo	Nystagmus
Nausea	
Vomiting	

(Pneumonic for nystagmus: COWS: Cold water to Opposite side,
 Warm water to Same side)

**Fig. 5.37: Directional preponderance**

In caloric test only the lateral semicircular canal is tested.

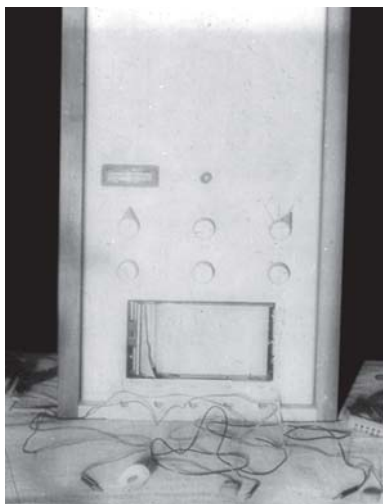


Fig. 5.38: Electronystagmography machine

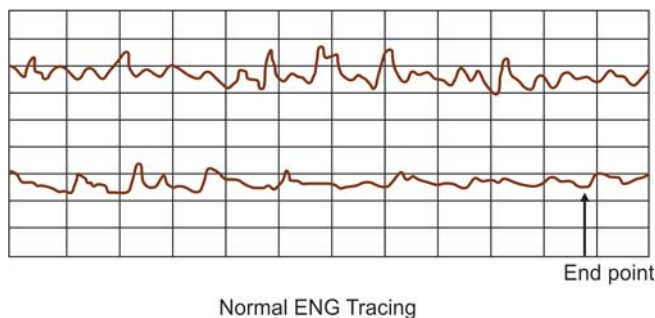
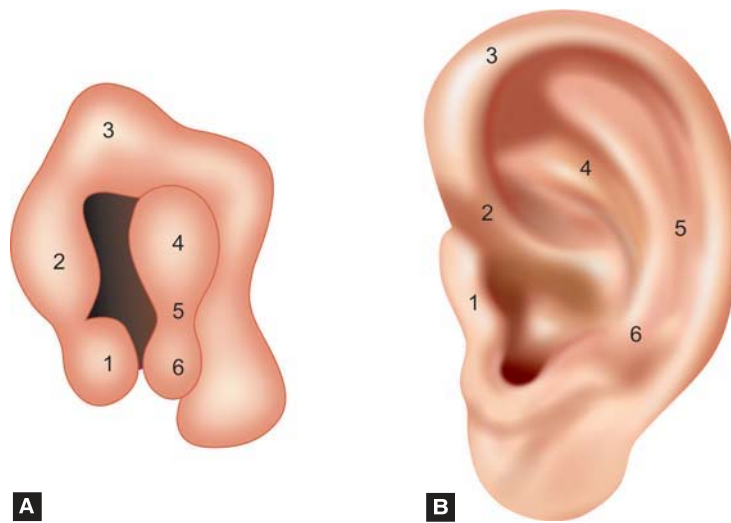


Fig. 5.39: Electronystagmography: Tracing



Fig. 5.40: X-ray mastoid, Law's lateral view

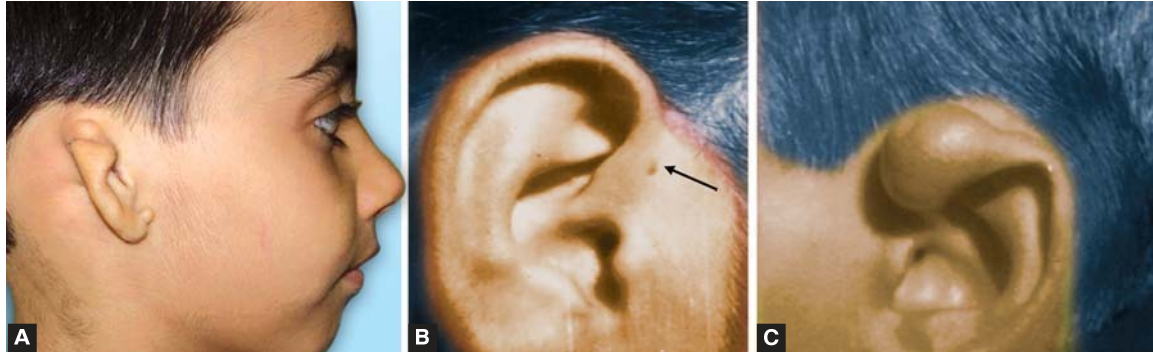
Conventional radiological examination of the mastoid in a Law's lateral view and Towne's view provides information about the type of the mastoid air system. Periorbital view of the petrous bone reveals the internal auditory meatus and information about its expansion in case of acoustic neuroma.



Figs 5.41A and B: Development of auricle

Ear develops from first and second branchial arches. Abnormality during development may affect either of the two arches and may result in various abnormalities.

A patient with atresia of external canal may have conductive or perceptive hearing loss.



Figs 5.42A to C: Congenital anomalies of external ear: (A) Microtia; (B) Preauricular sinus (arrow); (C) Dermoid

A preauricular sinus may go completely unnoticed until infected. It will then need surgical excision under antibiotic cover. Dermoids need surgical excision.



Fig. 5.43: Congenital aural atresia



Fig. 5.44: Retract pinna to look for canal atresia. Non canalization of EAC seen on pulling pinna



Fig. 5.45: Accessory auricle on the other side

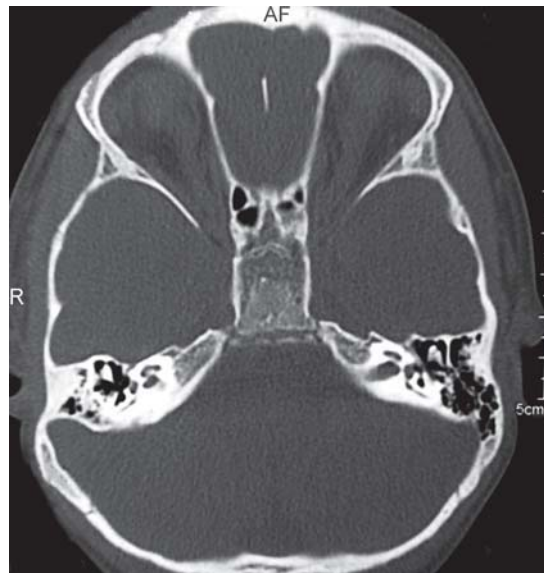
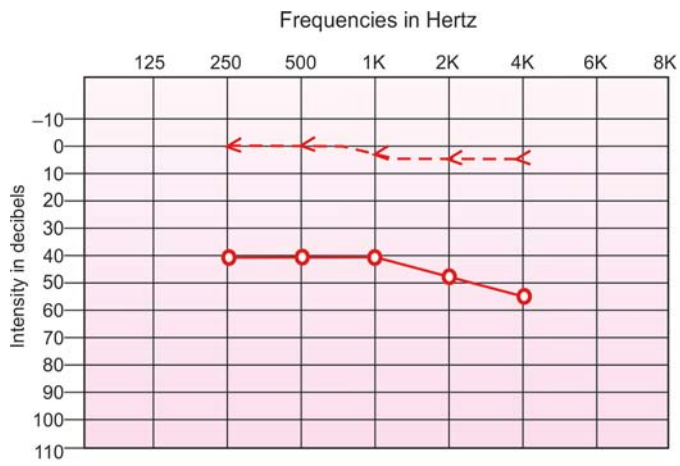
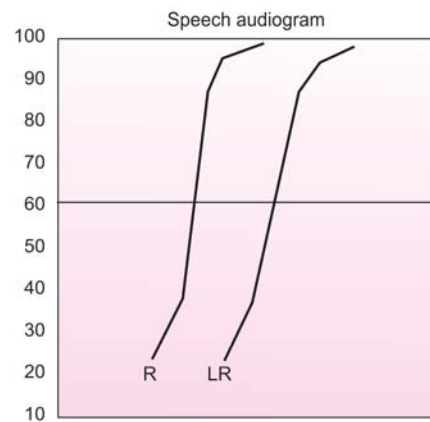
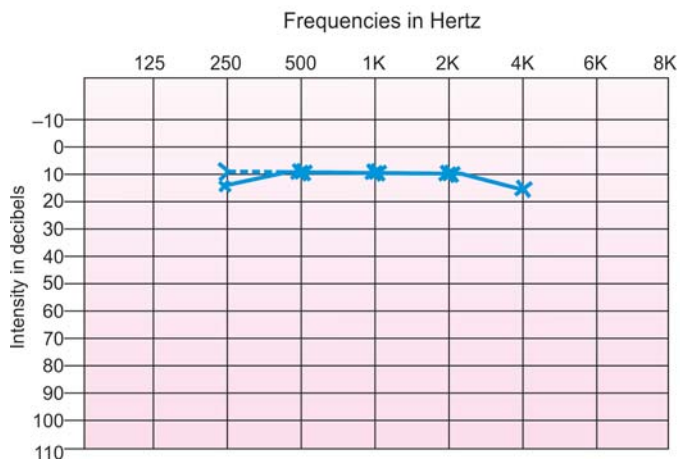


Fig. 5.46: CT scan of same patient as in Figures 5.43 to 5.47 showing right canal atresia with poorly developed mastoid air cell system and malformed ossicles. Compare this with a well pneumatized mastoid with 'ice cream cone' appearance of the ossicles on left



Modality	Ear	
	Right	Left
AC unmasked	○	×
AC masked	△	□
BC unmasked	<	>
BC masked	[]
No response	↻	↻



Figs 5.47A and B: Pure tone audiometry of same patient (Figs 5.43 to 5.46) showing right conductive hearing loss

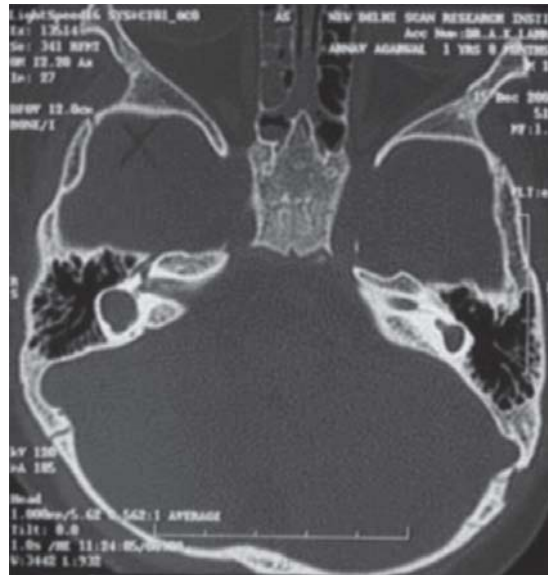


Fig. 5.48: Single cavity cochlea
(Source: Photo by Dr AK Lahiri)



Figs 5.49A to C: Affections of the pinna: (A) Hematoma of the auricle;
(B) Traumatic atresia; (C) Erysipelas



Fig. 5.50: Wrestlers cauliflower ear is caused by repeated trauma resulting in cosmetic deformity

Table 5.10: Classification of otitis externa

- Acute
 - Diffuse
 - Localized: furuncle
- Chronic
- Bacterial/viral/fungal
- Seborrheic otitis externa
- Eczematous otitis externa
- Malignant otitis externa

Otitis externa is inflammation of the external auditory canal can be because of variety of reasons. It can be localized or diffuse in character and needs appropriate local and systemic treatment.



Fig. 5.51: Wax can fill the whole ear canal



Fig. 5.52: Ear syringing is done to remove wax from the external auditory canal

A stream of water should be directed along the floor of canal so that it goes behind the wax and pushes it out. The direction of water should not be posterior (to avoid vagal stimulation) or straight onto the tympanic membrane (to avoid perforation). Water should be at body temperature to avoid giddiness.



Fig. 5.53: Basal cell carcinoma external ear

Common malignant tumors of the pinna and external canal are rodent ulcer and squamous cell carcinoma.



Fig. 5.54: Melanoma external ear

Other malignant tumors of the skin like melanoma can also occur. Complete excision of the tumor mass with neck dissection is the line of treatment.

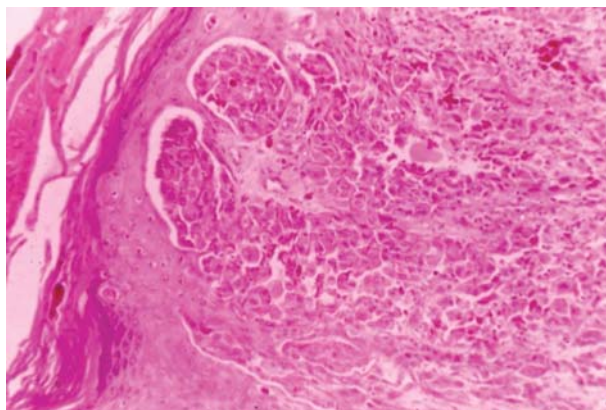


Fig. 5.55: Histopathological examination of melanoma external ear

Microscopically, melanoma consists of group of malignant cells which contain melanin pigment in their cytoplasm.

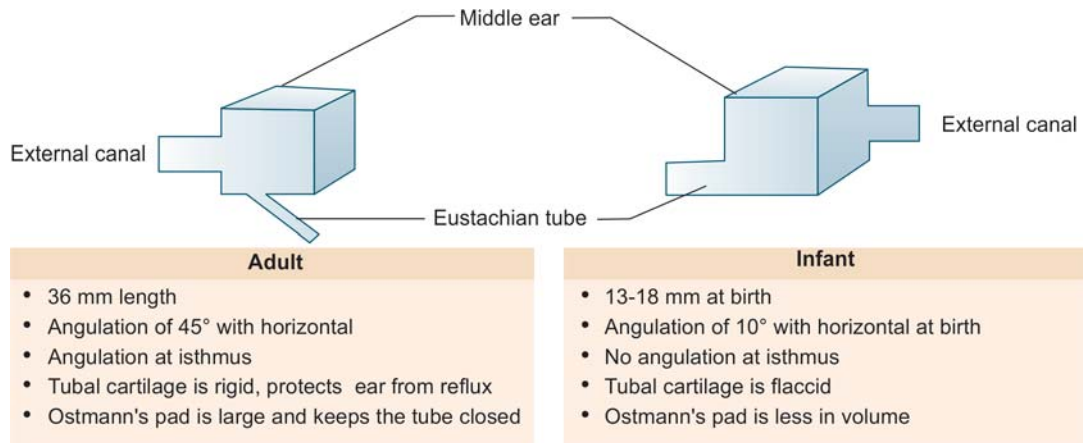
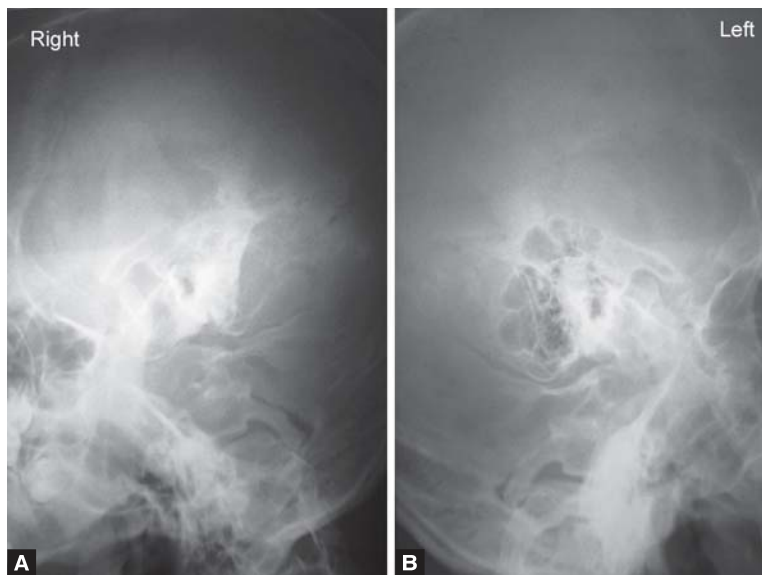
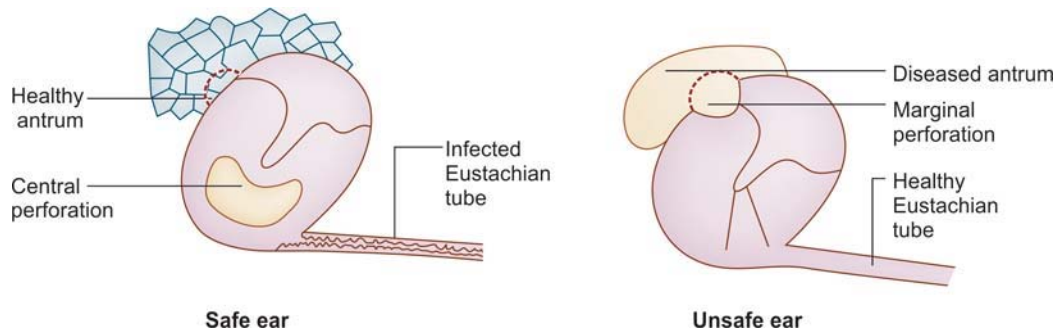


Fig. 5.56: Eustachian tube in child and adult

Eustachian or pharyngotympanic tube helps to maintain atmospheric pressure in the middle ear cleft. There is marked difference in Eustachian tube of an adult and children. It is shorter, wider and straighter in children making them vulnerable to ascending infections. Mothers should be advised to nurse infants in propped up position to prevent ascending infections.



Figs 5.57A and B: X-ray mastoid lateral oblique view: Sclerotic mastoid (Right) and well pneumatized mastoid (Left)



Figs 5.58A and B: CSOM safe and unsafe

The tympanic membrane is likely to get perforated either by trauma or by vascular necrosis as frequently seen in cases of chronic otitis media. The attic-antral disease may cause tympanic membrane to retract and result in retraction pocket which ultimately turns into a perforation. In safe Chronic suppurative otitis media (CSOM), a central perforation occurs, while in unsafe CSOM, a marginal or an attic perforation results.

Secretory otitis media is collection of nonpurulent fluid in the tympanum and may present as a hair line or air bubbles behind the intact tympanic membrane.

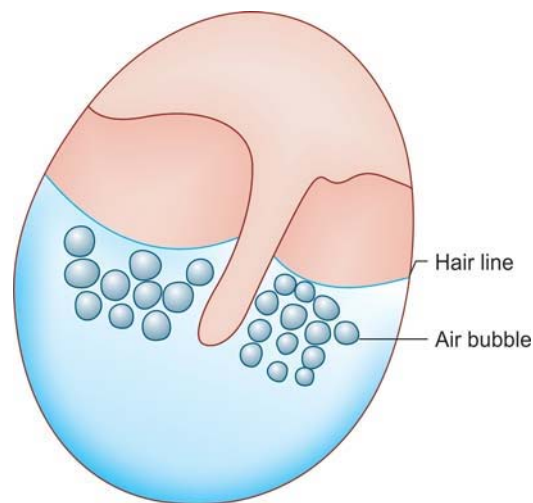


Fig. 5.59: Secretory otitis media

Secretory otitis media is usually bilateral. In a case of unilateral secretory otitis media in an adult, nasopharynx must be examined to rule out any growth or tumor.

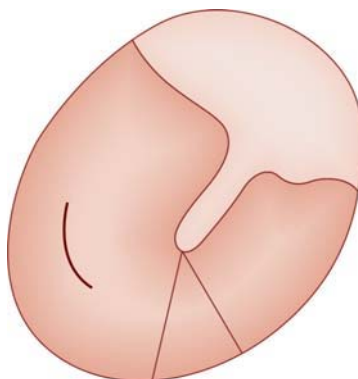


Fig. 5.60: Myringotomy–incision

Myringotomy is done in secretory otitis media when medical treatment fails. Myringotomy is necessary to relieve the pus from the middle ear in case of acute otitis media with complications like facial weakness. The incision and instruments needed for myringotomy as shown in Figures 5.60 to 5.62.

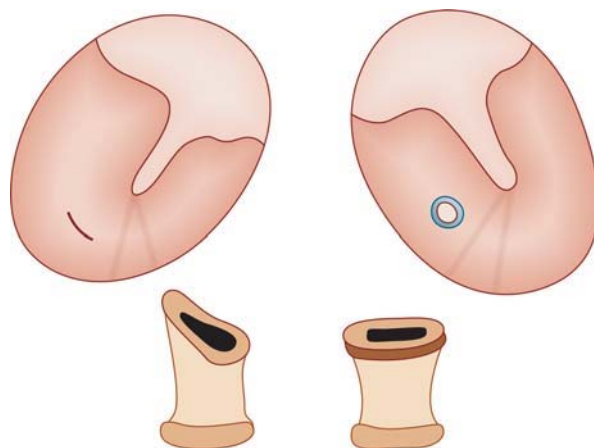


Fig. 5.61: Ventilation tube

Secretory otitis media is treated by myringotomy and insertion of ventilation tubes (grommet) when medical treatment fails.

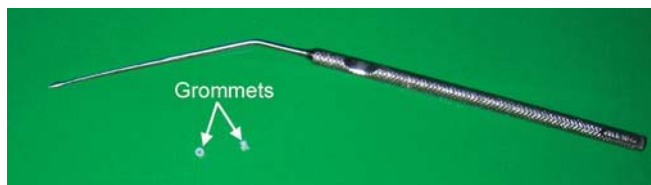


Fig. 5.62: Myringotomy knife and grommet

Table 5.11: Classification of chronic otitis media	
<i>Nonsuppurative</i>	
•	Serous otitis media
•	Glue ear or secretory otitis media
<i>Suppurative</i>	
•	Tubotympanic
•	Atticoantral
<i>Tuberculous</i>	

Chronic otitis media is infection of the middle ear of long duration. Classification of chronic otitis media is shown in the chart.

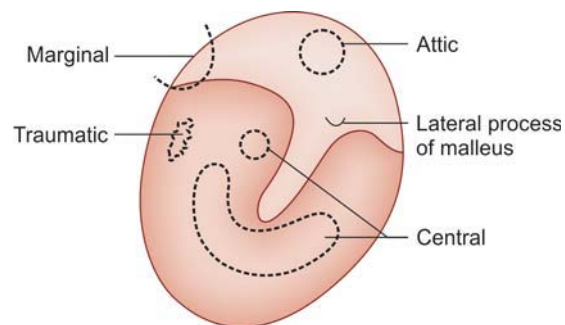


Fig. 5.63: Various perforations of tympanic membrane

Chronic suppurative otitis media due to the tubotympanic pathology produces central perforation and due to atticoantral pathology it causes posterosuperior or an attic perforation and is associated with cholesteatoma.

AURAL POLYP

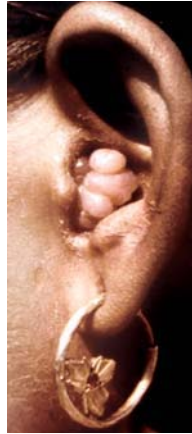


Fig. 5.64: Aural polyp

The atticoantral disease produces osteitis. This initiates formation of granulation tissue which may present as ear polyp.

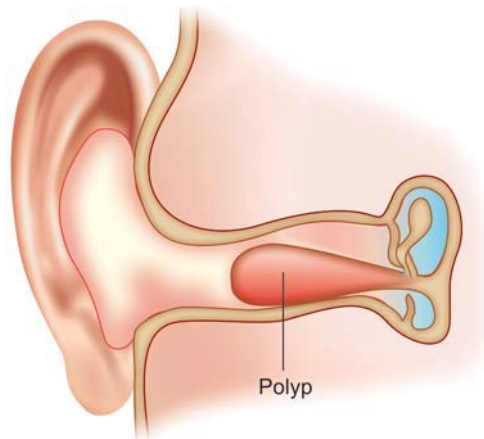


Fig. 5.65: Diagrammatic representation aural polyp

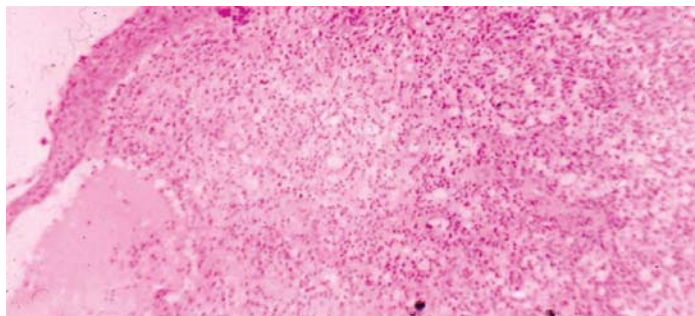


Fig. 5.66: Histopathological examination of aural polyp

Photomicrograph of the ear polyp shows marked inflammation of the connective tissue covered with mucosal lining.

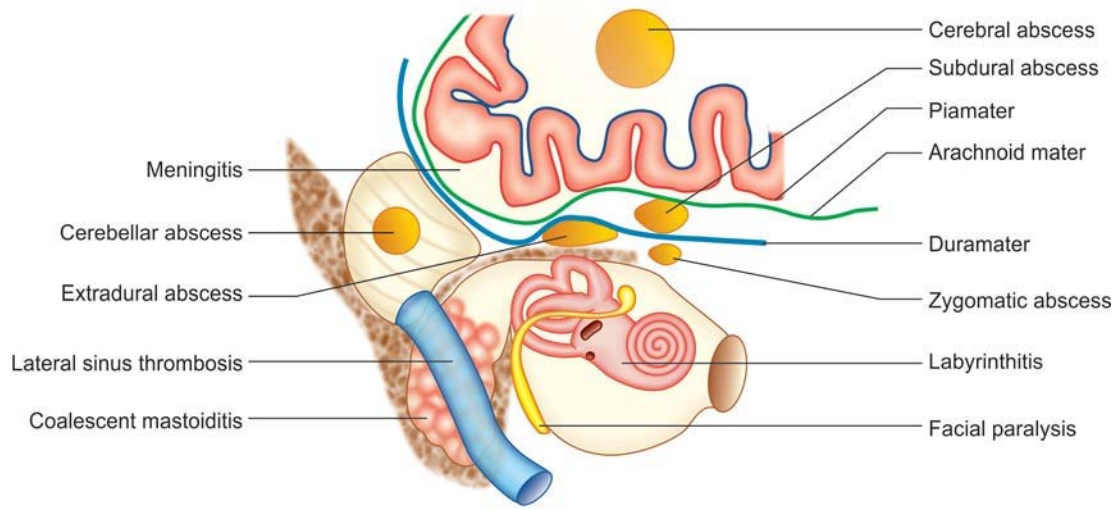


Fig. 5.67: Complications of CSOM

Chronic suppurative otitis media associated with cholesteatoma results in several complications through thrombophlebitis and osteitis. Figures 5.68 and 5.69 showing the various complications of otitis media is shown.



Fig. 5.68: Zygomatic abscess



Fig. 5.69: Postaural abscess

A zygomatic abscess (Fig. 5.68) or a postaural abscess (Fig. 5.69) can form as a complication of acute otitis media or acute exacerbation of the chronic otitis media.

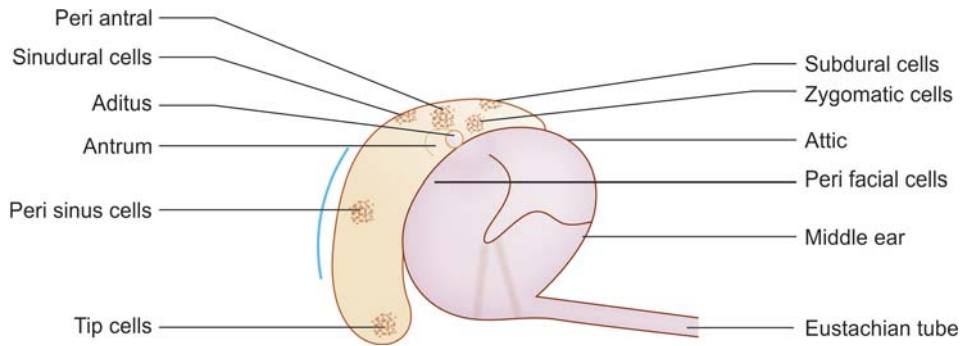
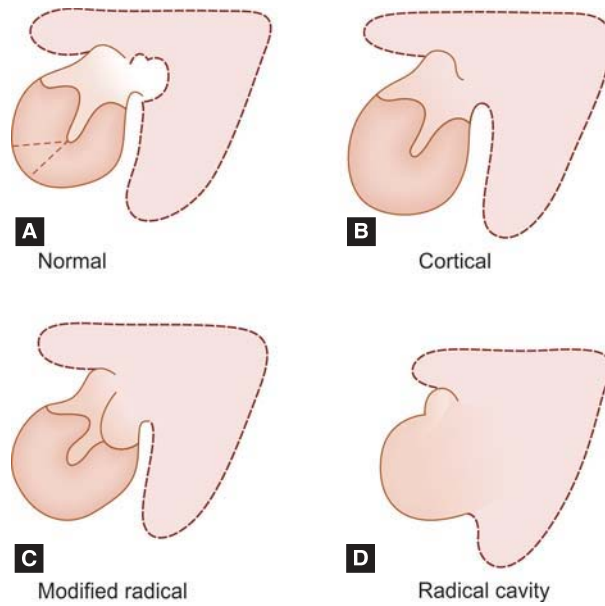


Fig. 5.70: Middle ear cleft and mastoid air cells



Figs 5.71A to D: Mastoidectomy

Mastoidectomy is an operation to provide relief in case of mastoiditis. Surgery is classified into three categories, simple or cortical, radical and modified radical mastoidectomy according to the extent of the surgical removal.



Fig. 5.72: Mastoidectomy instruments

The instruments required for the operation of mastoidectomy are shown. A bone drill makes it precise as well as minimizes the time for surgery.

Table 5.12: Prerequisites for tympanoplasty

- Good cochlear reserve
- Good Eustachian tube function
- Dry ear
- Healthy middle ear mucosa
- Sound transmission to oval window and sound protection to round window.

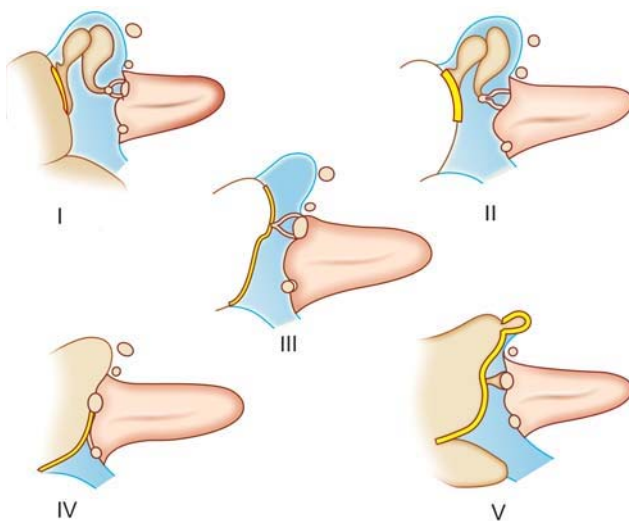


Fig. 5.73: Types of tympanoplasty

Various types of tympanoplasty with repair of the hearing mechanism in the ear are shown.

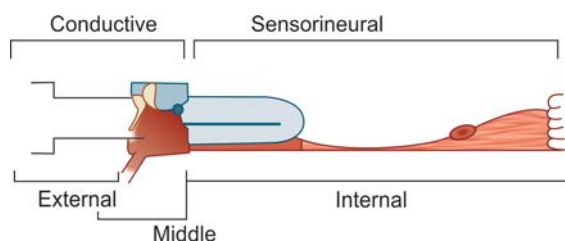


Fig. 5.74: Causes of deafness

Deafness is due to defective transmission or abnormal perception of the sound waves. The cause may be in the external, middle or inner ear mechanism.

Table 5.13: Deafness: External, middle and inner ear causes

<i>External ear</i>	<i>Middle ear</i>	<i>Internal ear</i>
Atresia	Congenital	Congenital
Wax	Otitis media	Viral
Foreign body	Otosclerosis	Presbycusis
Otitis externa	Trauma	Ménière's syndrome
Trauma	Tumor	Noise trauma
Tumor		Ototoxic drugs
		Head injury
		Labyrinthitis
		Vascular
		Idiopathic

Table 5.14: Management of deafness

<i>Medical</i>
• Antibiotics
• Antihistaminics and decongestants
• Suction, aural toilet, topical antibiotics
<i>Surgical</i>
• Adenoidectomy
• Myringotomy and grommet insertion
• Tympanoplasty
• Stapedotomy
• Bone anchored hearing aid (single sided deafness, congenital deafness, deafness with cavity problems)
• Cochlear implants
• Brainstem implants after acoustic neuroma excision in NF2
<i>Rehabilitation</i>
• Hearing aid
• Lip reading

In majority of cases deafness can be treated. Complete deafness before the speech development (prelingual) needs an approach different from cases where deafness is moderate and speech has developed (postlingual).

SUDDEN SENSORINEURAL HEARING LOSS: AN OTOLOGIC EMERGENCY

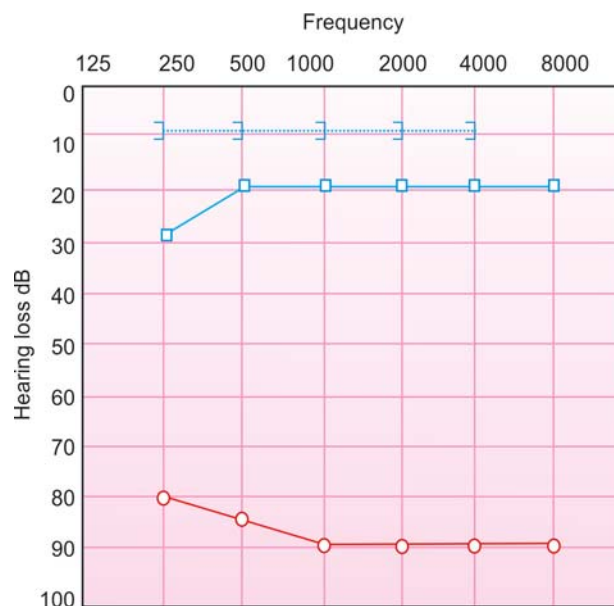


Fig. 5.75: Sudden sensorineural hearing loss in a telecaller

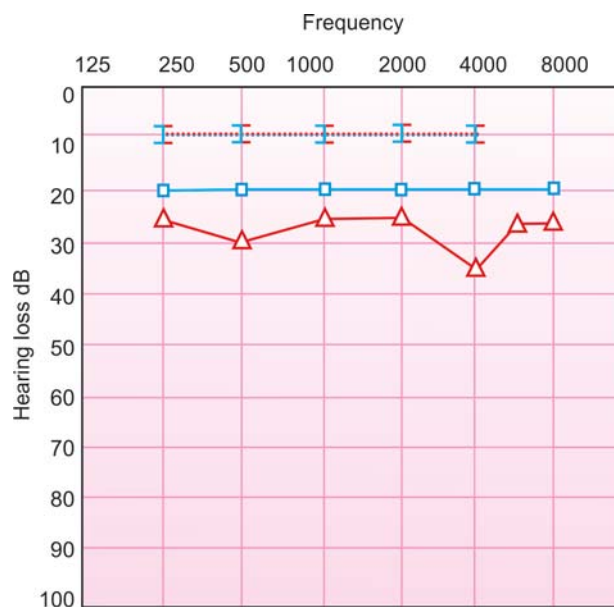


Fig. 5.76: Complete recovery of same patient as in 5.75 after 3 weeks [treated with oral steroids (prednisolone, 1 mg/kg/day), acyclovir, cinnarizine, Vitamin B12, low salt diet]

COCHLEAR IMPLANT

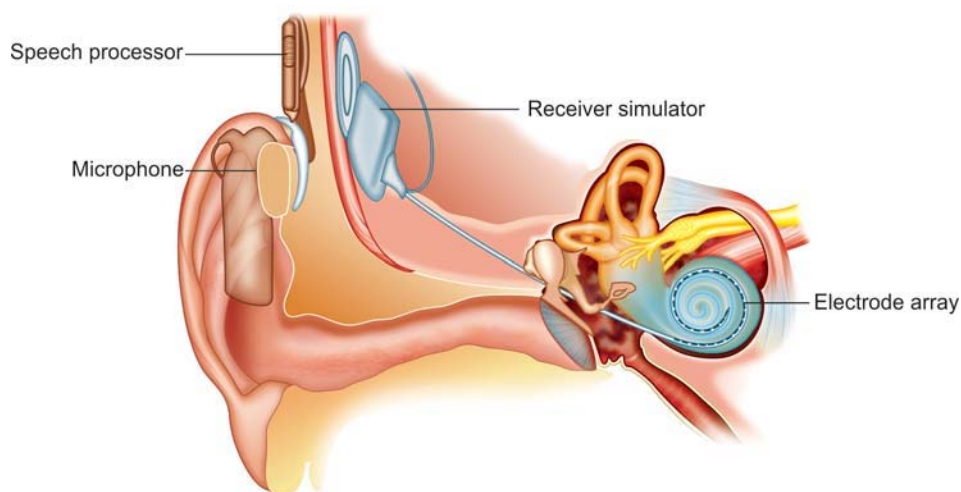


Fig. 5.77: Cochlear implant

Table 5.15: How does a cochlear implant work?

- Sound is received by microphone
- Sound is sent from microphone to speech processor
- Speech processor selects and codes useful sounds
- Code is sent to transmitting coil
- Transmitting coil sends code across skin to receiver/stimulator
- Receiver/stimulator converts code to electrical signals
- Electrical signals are sent to electrodes to stimulate VIII nerve fibers
- Signals are recognized as sounds by the brain, producing a hearing sensation

Table 5.16: Cochlear implant rehabilitation

Postsurgery

- Switch on—first tune up and program the implant
- Regular mapping over next few weeks

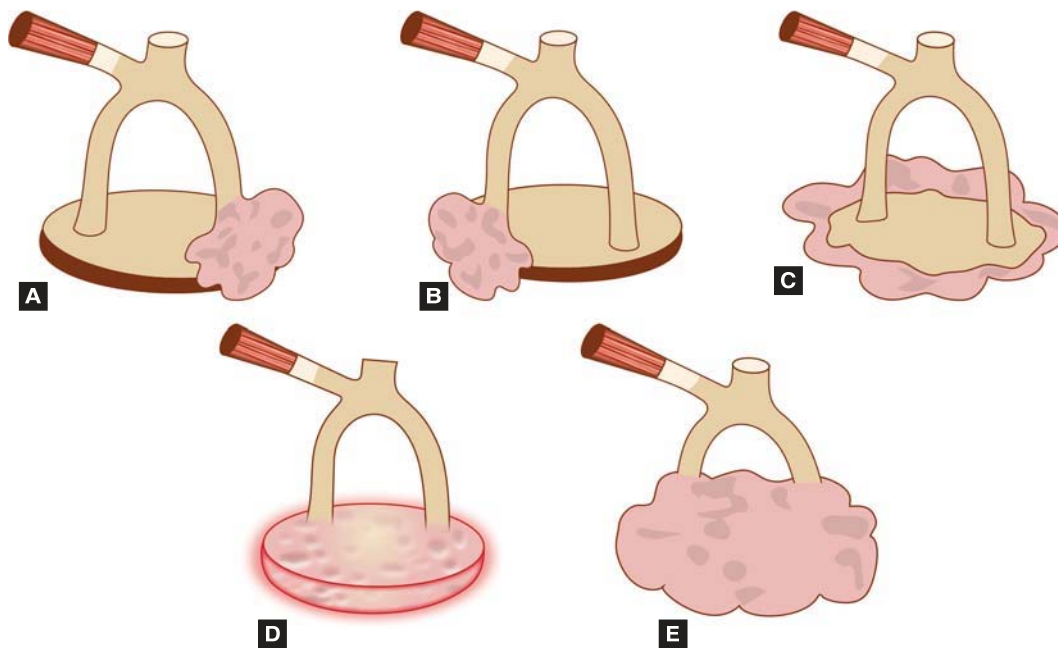
Listening training

- An individual program is worked out for each recipient
- Focus is on listening
- For children: Listening training follows normal child speech and language developmental milestones (hearing age vs chronological age)
- Family must be involved with children. The parents are trained to carry over listening to home in a natural environment

OTOSCLEROSIS

Table 5.17: Otosclerosis

<i>Clinical features</i>	
•	Progressive conductive deafness
•	Age 20–40 years
•	Females more than males (2:1)
•	Familial tendency present (50%)
•	Commonly bilateral (70%)
•	Paracusis Wilsii (patient hears better in noisy environment)
•	Normal tympanic membrane
•	Advanced case may have sensorineural deafness (Due to cochlear involvement)
•	Flamingo red sign in active otosclerosis (Schwartz sign)
<i>Pathology</i>	
•	Replacement of normal lamellar bone by spongy vascular bone of increased cellularity and density which fixes the footplate of stapes
<i>Treatment</i>	
•	Stapedotomy
•	Hearing aid



Figs 5.78A to C: Types of otosclerotic focus (A) Anterior; (B) Posterior; (C) Annular; (D) Biscuit; (E) Obliterative

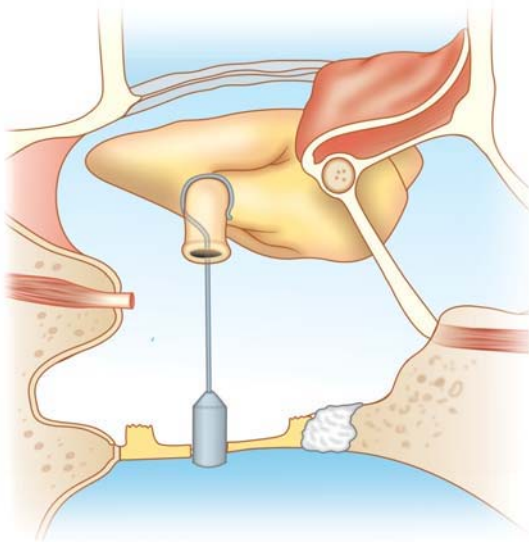


Fig. 5.79: Stapes piston

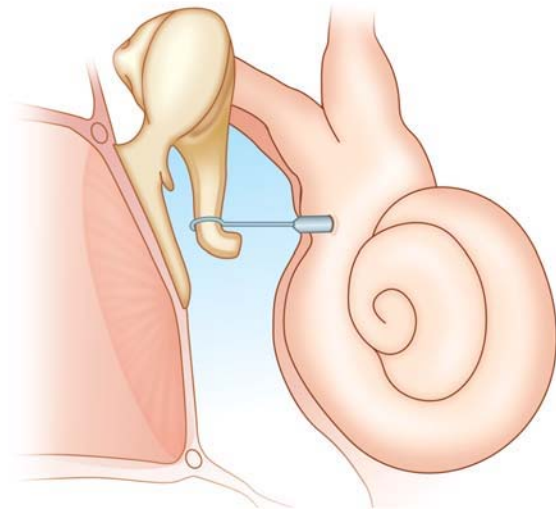


Fig. 5.80: Stapes piston *in situ* in coronal view

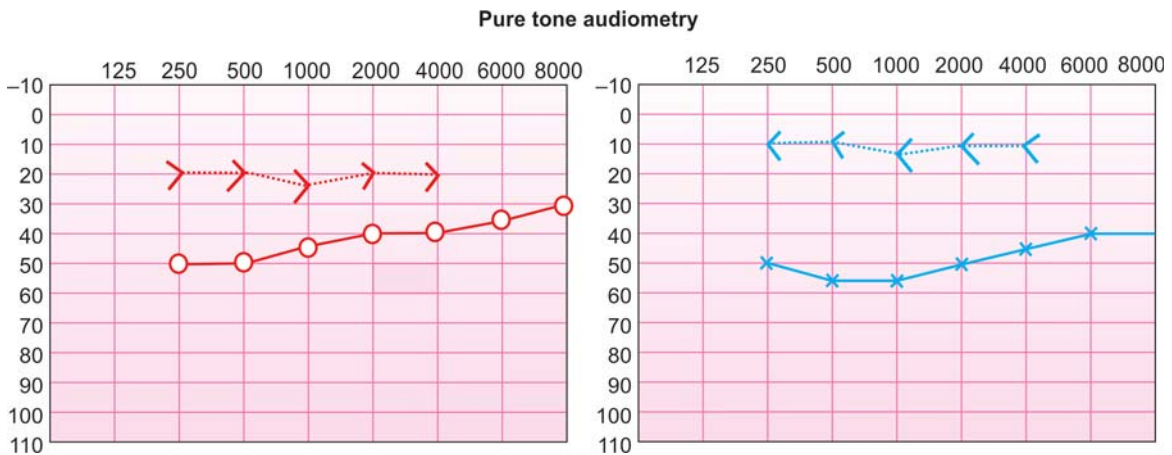


Fig. 5.81: Audiogram of a patient with otosclerosis showing bilateral conductive hearing loss

A bilateral progressive conductive deafness in a young adult female who has history of familial tendency is generally due to otosclerosis. The deafness aggravates during pregnancy.

Tympanometry usually shows type As curve with reduced compliance. Stapedial reflex is absent in late stages of the disease.

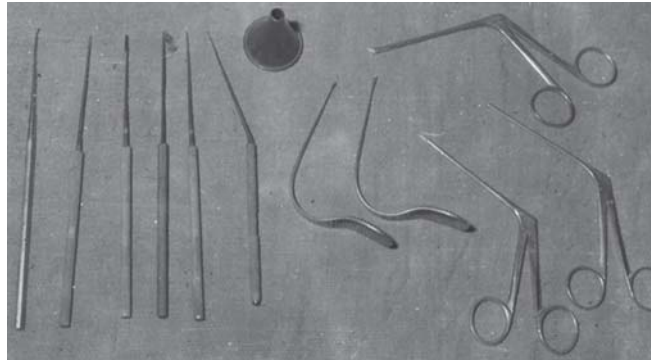


Fig. 5.82: Instruments for stapedotomy

In otosclerosis stapes gets fixed by new bone formation, stapedotomy is the recommended treatment. A set of instrument used for stapedotomy is shown. One may be benefited by using a hearing aid if the operation is not undertaken.

Table 5.18: Causes of vertigo

<i>Ear</i>	<i>Central</i>	<i>Vascular</i>	<i>Miscellaneous</i>
Labyrinthitis	Migraine	Cervical vertigo	Orthostatic hypertension
Vestibular neuronitis	Epilepsy	Vertebrobasilar insufficiency	Hypoglycemia
Cupulolithiasis	Head injury	Vascular accidents	Hyperventilation syndrome
Perilymphatic fistula	Multiple sclerosis		Hypothyroidism
Ménière's disease	Drugs		Hormonal imbalance
Syphilitic vertigo			
Acoustic neuroma			
Ototoxic drugs			
Herpes oticus			
Eustachian tube dysfunction			
Wax			

Maintenance of satisfactory equilibrium depends on the coordination between labyrinth, muscles, tendons, joint sense, skin receptors and visual centers. Any interference with coordination of these components leads to a sense of vertigo. A detailed history and thorough examination of the ear is of paramount importance to find out the nature and thereby the causes of vertigo. Causes of vertigo are listed in the Table 5.18. Treatment of the detected cause and suppression of the vertigo by antivertigo drugs cinnarizine, betahistine or prochlorperazine followed by rehabilitation is the line of management.

MÉNIÈRE'S DISEASE

Table 5.19: Ménière's disease
Cardinal symptoms
• Episodic vertigo
• Fluctuating deafness
• Tinnitus.
Other symptoms
• Aural fullness
• Headache
• Nystagmus
• Anxiety state.
Pathology
• Endolymphatic hydrops.

Ménière's disease is characterized by acute vestibular and cochlear symptoms. Its attacks come in bouts.

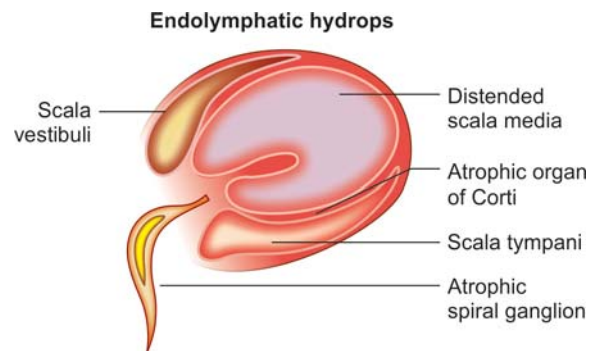


Fig. 5.83: Pathogenesis of Ménière's disease

Figure 5.83 shows the endolymphatic hydrops. This is a characteristic finding of Ménière's disease.

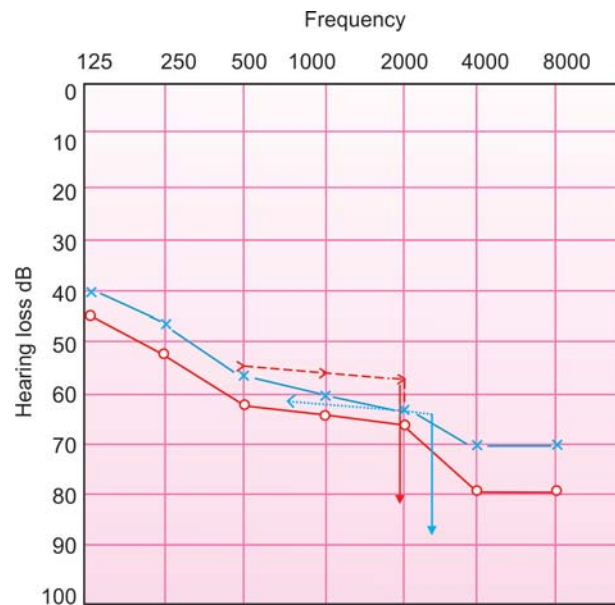


Fig. 5.84: Audiogram of patient with Ménière's disease

Audiogram reveals perceptible deafness as seen in Ménière's disease. Initially only lower frequencies are affected. As disease progresses, all frequencies get involved.

Table 5.20: Ménière's disease treatment

<i>Acute phase</i>	
•	Reassurance
•	Complete bed rest
•	Sedation
•	Stellate ganglion block.
<i>Remission phase</i>	
<i>Medical</i>	
•	Salt restricted diet
•	Diuretics (Thiazide)
•	Antihistamines (Cinnarizine)
<i>Surgical</i>	
•	Sac decompression
•	Sac shunt
•	Vestibular neurectomy
•	Labyrinthectomy (Chemical/Surgical) when hearing is poor

Treatment for Ménière's disease depends on whether it is in acute phase or in remission phase.



Fig. 5.85: Malignancy ear: Fibrosarcoma. Note ipsilateral facial weakness

Tumors of the ear are uncommon. However, squamous cell carcinoma is the most common malignancy of the middle ear. Sarcomas are more common in children. A child with fibrosarcoma involving the ear and producing facial palsy is shown in the photograph.



Fig. 5.86: X-ray skull

Radiograph shows complete destruction of the petrous temporal bone by malignancy of the ear. CT scanning of the temporal bone has brought in revolutionary changes in the diagnosis and treatment planning for these cases.

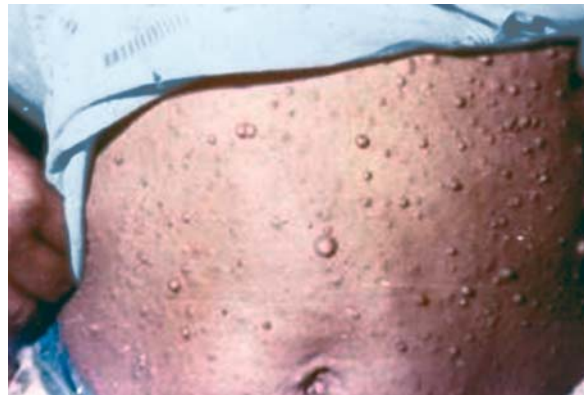
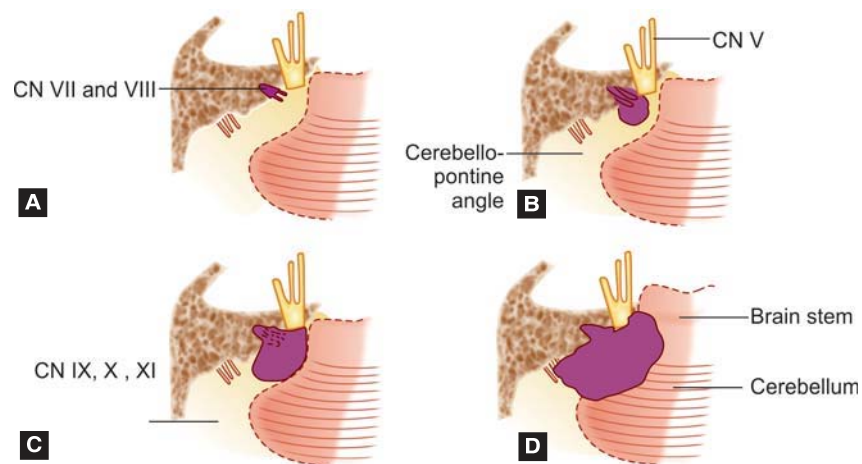


Fig. 5.87: Neurofibromatosis (NF2) can also present as acoustic neuroma

Acoustic neuroma is a tumor of the eighth cranial nerve. It may occur as a solitary tumor or be a part of generalized neurofibromatosis. Patients with unilateral sensorineural deafness, tinnitus or deafness and/or absence of corneal reflex should be fully investigated.



Figs 5.88A to D: Acoustic neuroma and its expansion: (A) Intracanalicular; (B) Tumor extending into cerebellopontine angle; (C) Tumor pressing on CN V; (D) Very large tumor pressing on CN V, IX X, XI, and brainstem and cerebellum

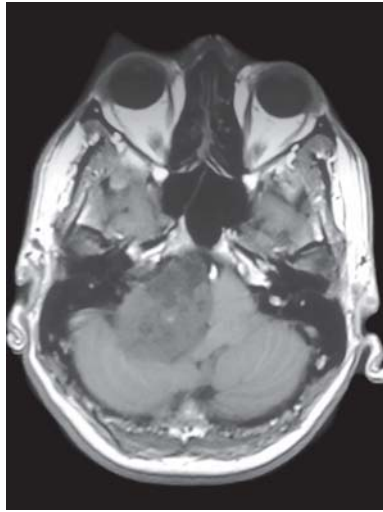


Fig. 5.89: T1 weighted axial MRI showing giant tumor arising from vestibulocochlear nerve spreading into cerebellopontine angle. *Note:* The relation of the tumor to the temporal bone (*Source:* Photo by Dr Bhavin Parikh-Vadodara)



Fig. 5.90: Gadolinium enhanced T1 weighted axial MRI showing brightly enhancing acoustic neuroma (*Source:* Photo by Dr Bhavin Parikh)

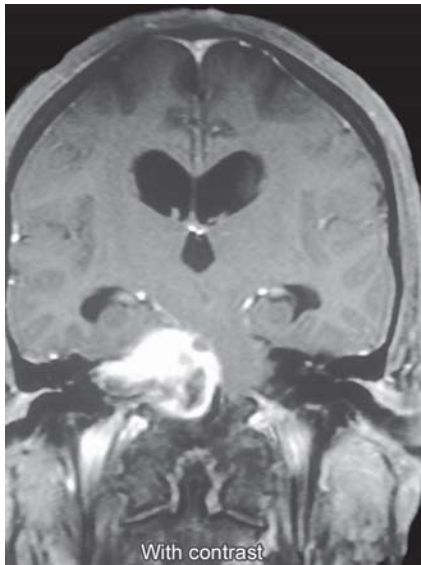


Fig. 5.91: Gadolinium enhanced T1 weighted coronal MRI showing brightly enhancing acoustic neuroma tumor causing marked deviation of the brainstem to opposite side with hydrocephalus. *Note:* The extension of the tumor in internal auditory meatus (*Source:* Photo by Dr Bhavin Parikh)

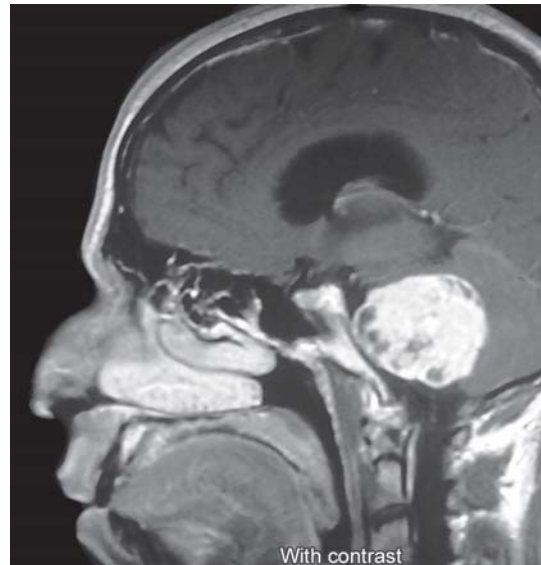


Fig. 5.92: Gadolinium enhanced T1 weighted sagittal MRI showing brightly enhancing acoustic neuroma tumor with hydrocephalus causing compression of the cerebellum with tonsillar herniation into foramen magnum with hydrocephalus (*Source:* Photo by Dr Bhavin Parikh)

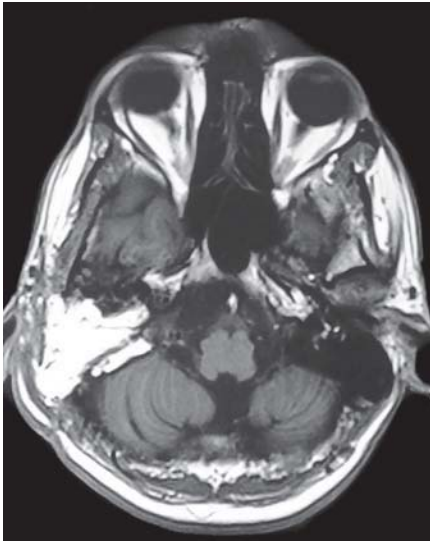


Fig. 5.93: Postoperative T2 weighted axial MRI picture after 10 days of removal of tumor by translabyrinthine approach. *Note:* The abdominal fat used for filling the defect in mastoid cavity and cerebellopontine angle to prevent CSF leak (*Source:* Photo by Dr Bhavin Parikh)

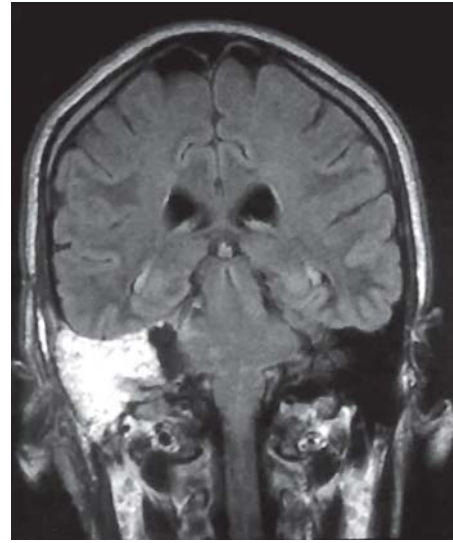


Fig. 5.94: Postoperative T1 weighted coronal MRI picture after 10 days of removal of tumor by translabyrinthine approach. *Note:* The hydrocephalus, compression of cerebellum and deviation of brainstem has resolved (*Source:* Photo by Dr Bhavin Parikh)



Fig. 5.95: Glomus jugulare

Glomus jugulare (nonchromaffin paraganglioma) is the most common benign tumor of the ear. It produces deafness, tinnitus or presents as a bleeding ear polyp. It may also involve other related structures; patient shown is having a right glomus jugulare tumor which had extended intracranially.

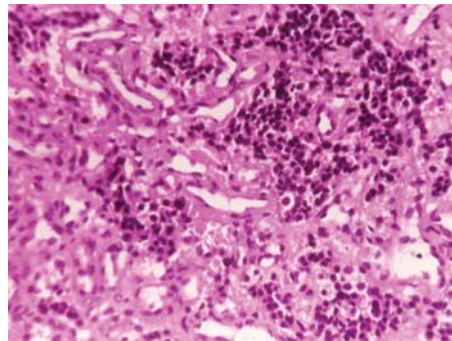


Fig. 5.96: Histopathological examination of glomus jugulare

Microscopically, the tumor consists of monomorphic cells arranged in groups which are separated by vascular connective tissue septae.

FACIAL NERVE

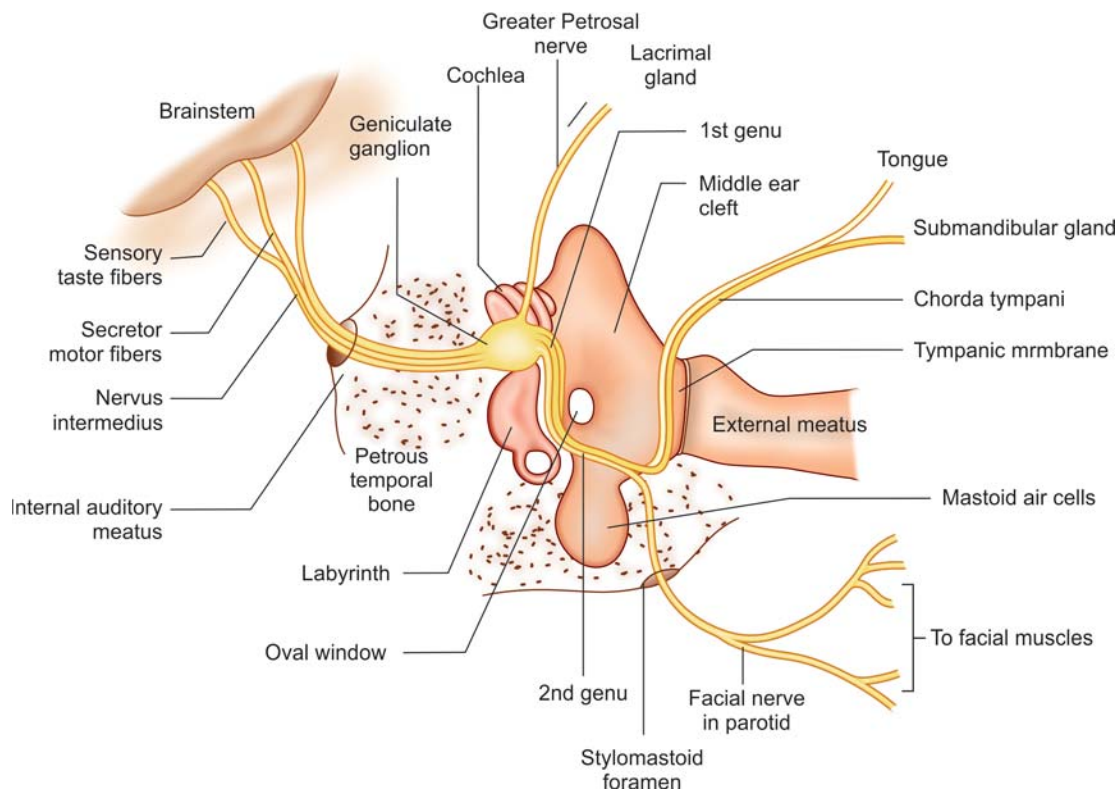


Fig. 5.97: Facial nerve

Facial paralysis can be caused damage to the facial nerve of different levels as shown.

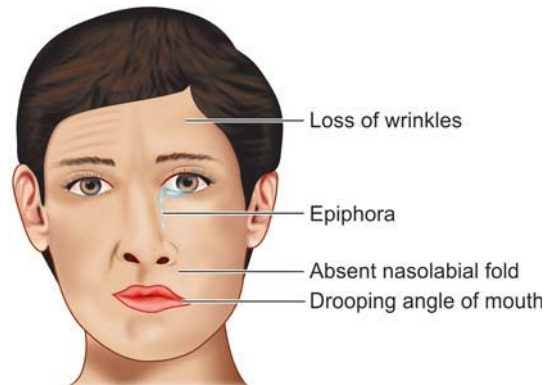


Fig. 5.98: Bell's palsy

Bell's palsy is a unilateral, idiopathic, rapid onset facial palsy. It may be partial or complete and is usually self limiting. It is the commonest cause of facial paralysis. It is treated with antivirals, steroids and physiotherapy.

Table 5.21: Causes of facial paralysis

<i>Intracranial</i>
Brainstem lesions <ul style="list-style-type: none"> • Vascular accidents • Poliomyelitis • Multiple sclerosis • Tumors
CP angle lesions <ul style="list-style-type: none"> • Acoustic neuroma • Primary cholesteatoma • Arachnoid cysts • Meningitis
Intratemporal <ul style="list-style-type: none"> • Otitis • Trauma • Tumors • Ramsay Hunt syndrome • Bell's palsy
Infratemporal <ul style="list-style-type: none"> • Parotid tumors • Trauma
Miscellaneous <ul style="list-style-type: none"> • Sarcoidosis • Polyneuritis • Melkersson syndrome • Leukemia

TEMPORAL BONE TRAUMA



Fig. 5.99: CSF otorrhea with Battles sign in a patient with temporal bone fracture following road traffic accident

HRCT of patient in Figure 5.99 showed longitudinal fracture of temporal bone. Facial canal and labyrinth had escaped. Otorrhea ceased after conservative management.

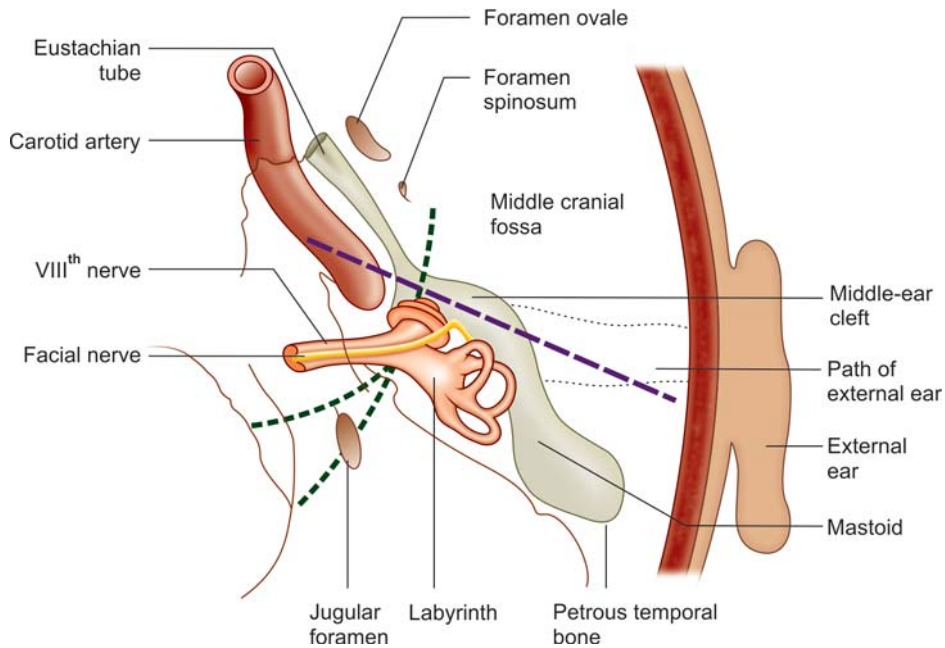


Fig. 5.100: Temporal bone fractures: Longitudinal (big dash) and transverse (small dot)

Transverse temporal bone fractures are more likely to involve the facial canal or labyrinth. Incidence of longitudinal temporal bone fractures is more common as compared to transverse fractures.

RAMSAY HUNT SYNDROME



Fig. 5.101: Herpes zoster oticus

Herpes zoster oticus presents with herpetic eruptions of the external ear and produces infranuclear facial paralysis. It is treated with antivirals (systemic and topical Acyclovir) steroids and analgesics.

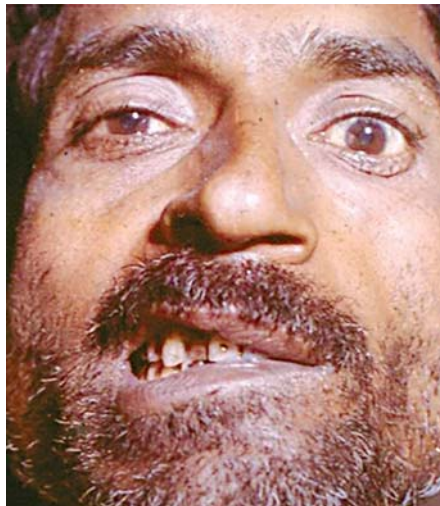


Fig. 5.102: Left facial paralysis in herpes zoster oticus

Recovery rates of facial paralysis in herpes zoster oticus are poorer than Bell's Palsy.

ESSENTIAL BLEPHAROSPASM



Fig. 5.103: Hyperkinetic disorder of facial nerve: Essential blepharospasm—patient unable to open eyes
(*Source:* Photo by Dr Raj Anand, Oculoplastic Surgeon, Delhi)



Fig. 5.104: Essential blepharospasm relieved by Botox (*Source:* Photo by Dr Raj Anand)

Differential Diagnosis of Referred Otalgia

Table 5.22: Referred otalgia

<i>Via V cranial nerve</i>	<i>Via 9th and 10th cranial nerve</i>
Sinusitis	Acute tonsillitis
Nasopharyngeal malignancy	Retropharyngeal abscess
Nasopharyngitis	Post-tonsillectomy
Cariou tooth	Laryngopharyngeal lesions
Costen syndrome	Glossopharyngeal neuralgia
Deviated nasal septum	Styloid process neuralgia
<i>Via 2nd and 3rd cervical nerve</i>	<i>Via 7th nerve</i>
Cervical disc lesions	Herpes zoster oticus
Arthritis	
Temporomandibular	
Fibrositis of sternomastoid muscle	
Herpetic lesions	

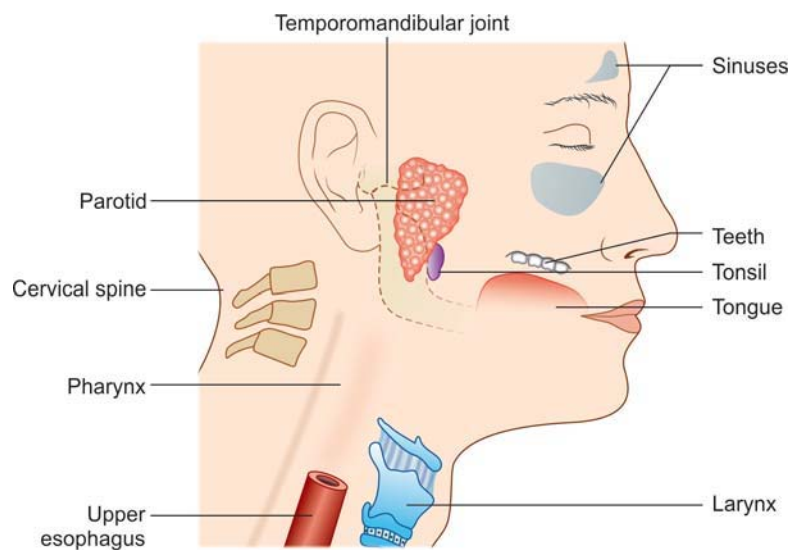


Fig. 5.105: Referred otalgia



Fig. 5.106: OPG of patient with styalgia (Eagle's syndrome)
Elongated styloid process marked by arrows on right side

Palpate the tonsillar fossa in patients with unexplained otalgia.

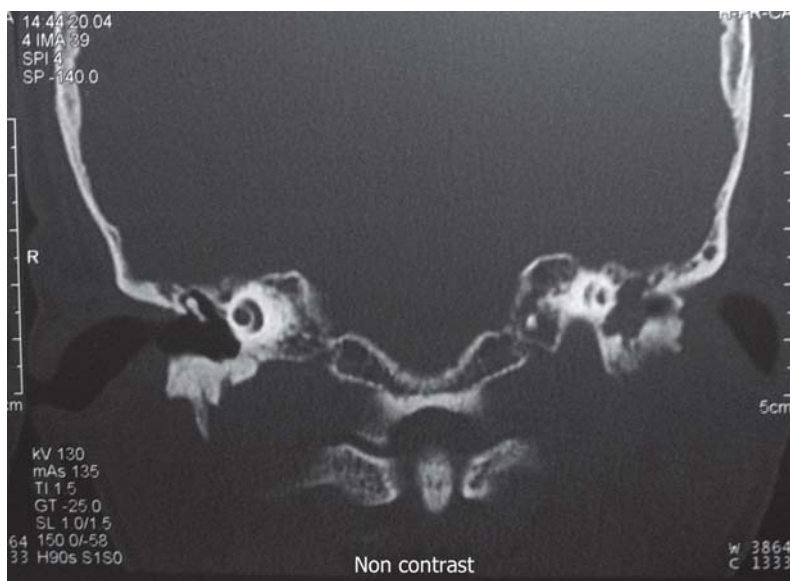


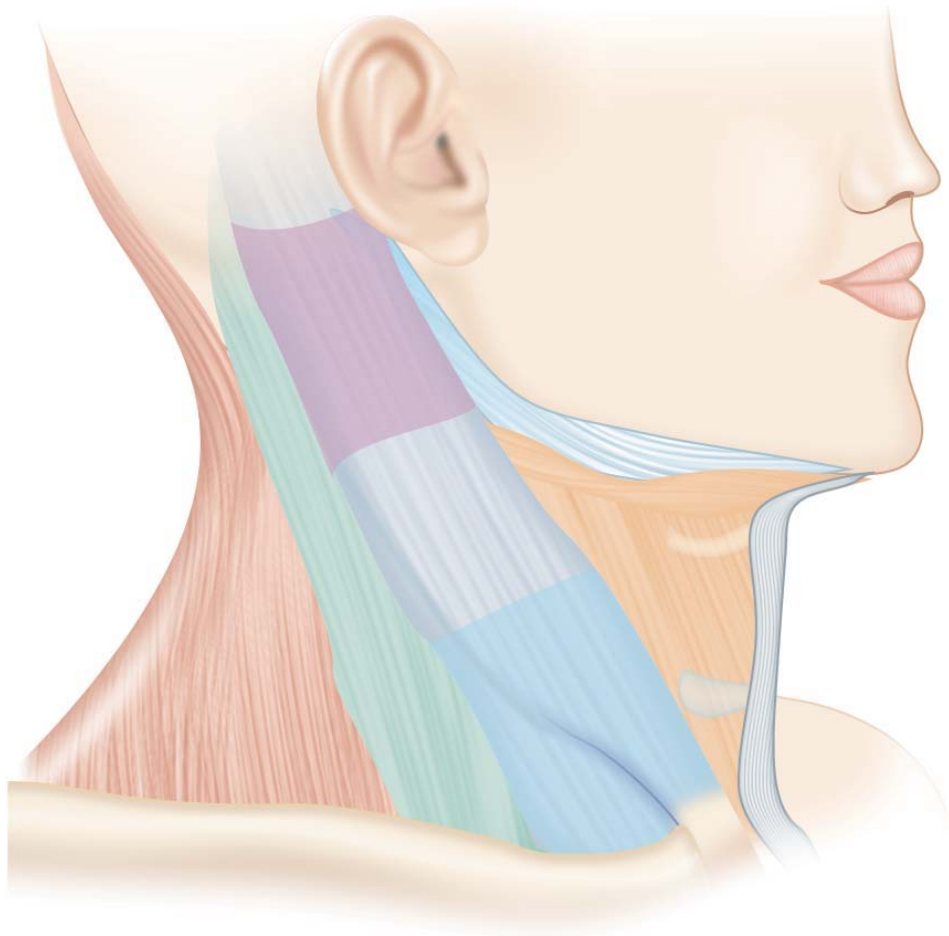
Fig. 5.107: HRCT temporal bone of patient with left malignant otitis externa

'Malignant' otitis externa is an aggressive disease termed 'malignant' because of poor treatment outcome and mortality. It occurs typically in diabetic or immunocompromised individuals. It is usually caused by *Pseudomonas aeruginosa*. It begins as a non healing external otitis which progresses into an osteomyelitis of the temporal bone and spreads along the skull base.

6

PS Saharia, Deepti Sinha

Neck



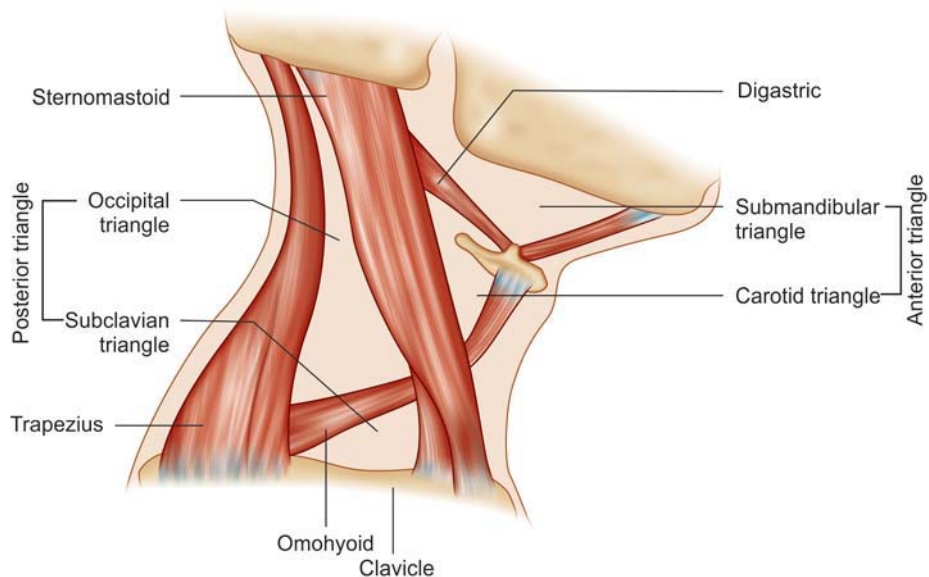


Fig. 6.1: Triangles of the neck



Fig. 6.2: Palpation of neck nodes

Flex the patient's neck slightly and stand behind the patient while palpating the neck.

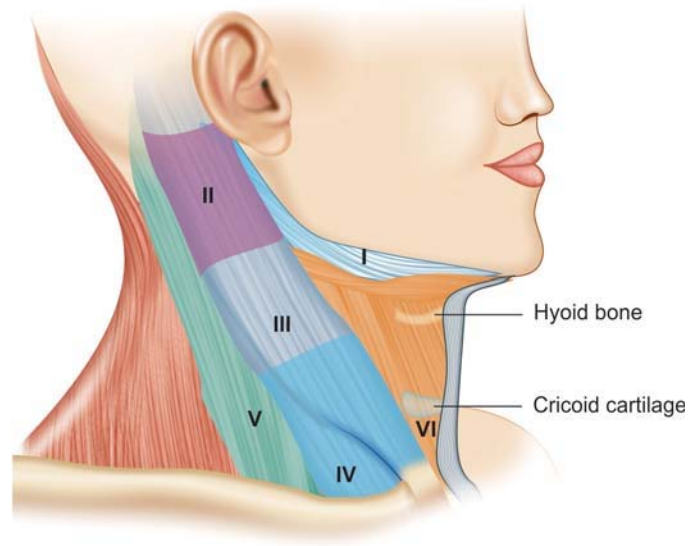


Fig. 6.3: Levels of neck nodes

Table 6.1: Levels of lymph nodes in neck

Level I A	:	Submental nodes
Level I B	:	Submandibular nodes
Level II, III and IV	:	Upper, mid and lower jugulodigastric nodes
Level V	:	Posterior triangle
Level VI	:	Below the hyoid in the anterior neck
Level VII	:	Anterior mediastinum

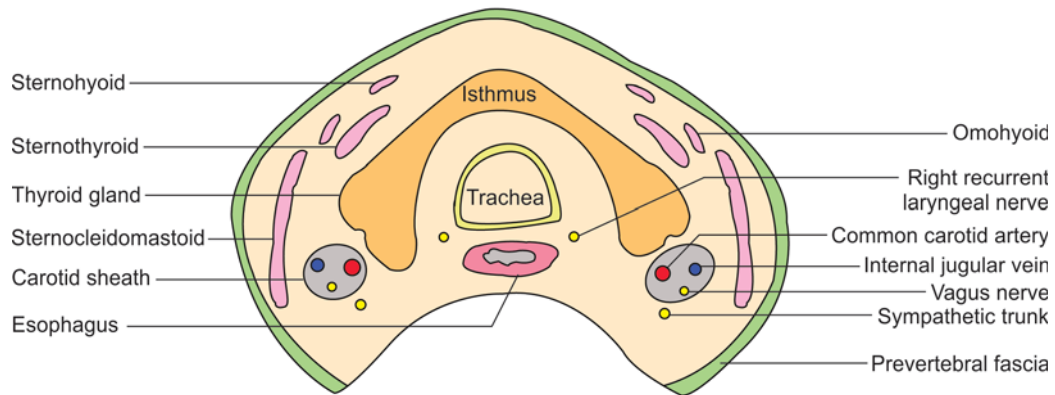


Fig. 6.4: Axial section of neck at level of thyroid isthmus

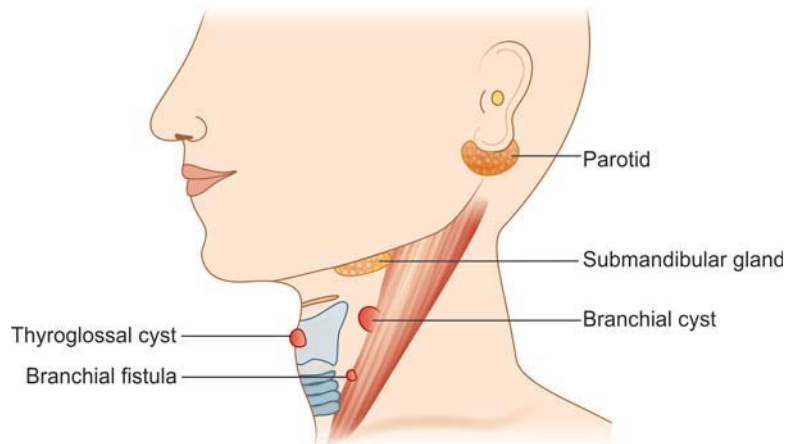


Fig. 6.5: Sites of various cysts, fistulae and tumors in neck

Neck swellings may be midline or lateral. Thyroglossal cyst is commonly encountered as a midline cyst. Thyroglossal cyst is the only midline neck swelling which moves up on protrusion of tongue.

TUBERCULAR LYMPHADENITIS

Tubercular lymphadenitis commonly affects upper deep jugular nodes in the neck. Initial stage is of enlargement of multiple nodes followed by matting due to periadenitis. This may further progress to caseation followed by rupture and cold abscess formation. Cold abscesses are termed cold because the swelling is without heat, redness, pain or fever. All patients with tubercular lymphadenitis are screened for pulmonary tuberculosis. WHO Multidrug regimen for TB is followed (2 months treatment with isoniazid, rifampicin, pyrizinamide, ethambutol + 4 months continuation therapy with isoniazid and rifampicin). Nonresponders or MDR cases are referred to dedicated TB centers for treatment.



Fig. 6.6: Tubercular cold abscess

Microscopically, tuberculous lymph node is characterized by granulomas consisting of lymphocytes, epithelioid cells and Langhans giant cells as shown in low and high power view.

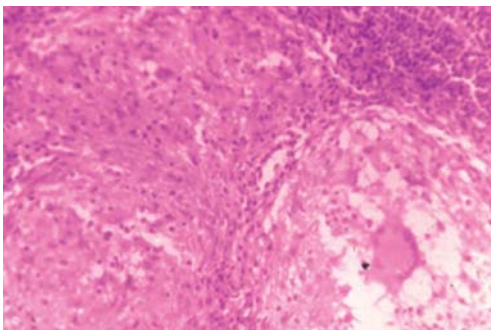


Fig. 6.7: Langhans cells

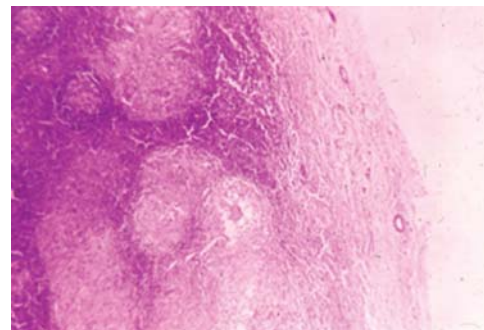


Fig. 6.8: Histopathology of tubercular lymphadenitis

METASTATIC NECK NODE



Fig. 6.9: Hard and fixed cervical lymph node

The cervical lymph nodes may be involved by malignant lesions in the tongue, pharynx or nasopharynx. Large fixed nodes as in Figure 6.9 mostly represent advanced malignant disease and are often unresectable. After establishing diagnosis appropriate curative or palliative treatment is offered.

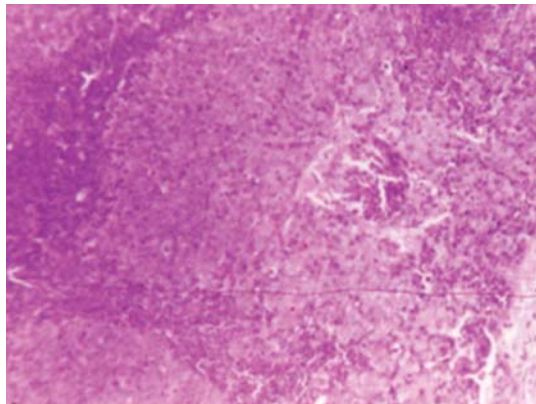
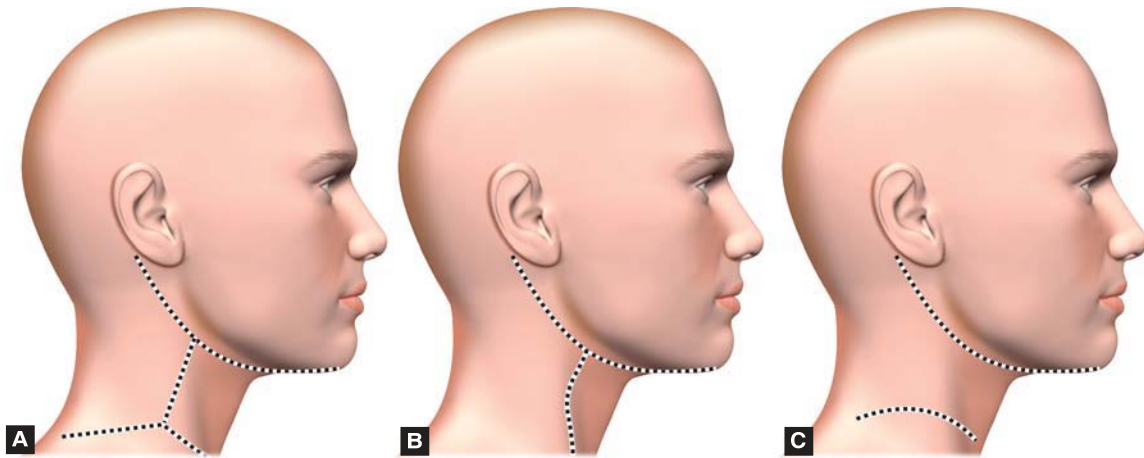


Fig. 6.10: Histopathological examination of metastatic cervical node



Figs 6.11A to C: Incisions for block dissection: Double Y,Y, Mc Fee

Block dissection of the cervical lymph node is indicated for the metastatic cancer. A double Y incision for the block dissection is shown. With the development of the newer diagnostic facilities, better pre- and postoperative management and improved tissue transfer technique, surgical procedures dealing with a lesion in the base of the skull have evolved in coordination with neurosurgical colleagues.

PAROTID GLAND

Parotid glands are the largest salivary glands. The parotid duct exits the gland medially, crosses the superficial border of the masseter, pierces the buccinator, and enters the oral cavity through the buccal mucosa opposite the second maxillary molar.

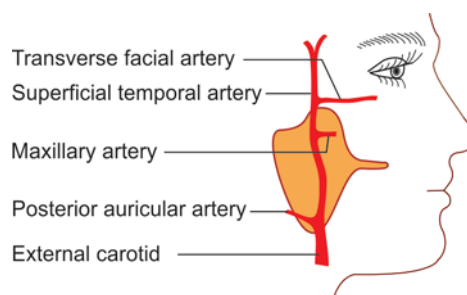


Fig. 6.12: Arterial supply of parotid gland

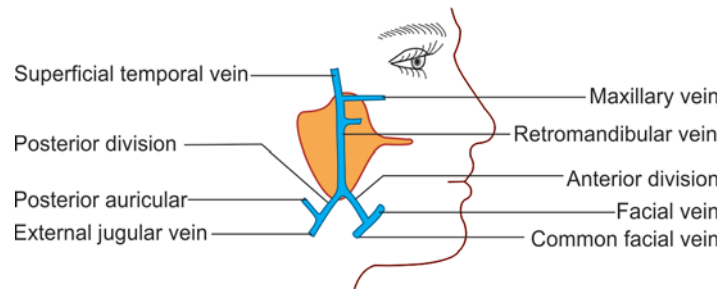


Fig. 6.13: Venous drainage of parotid gland

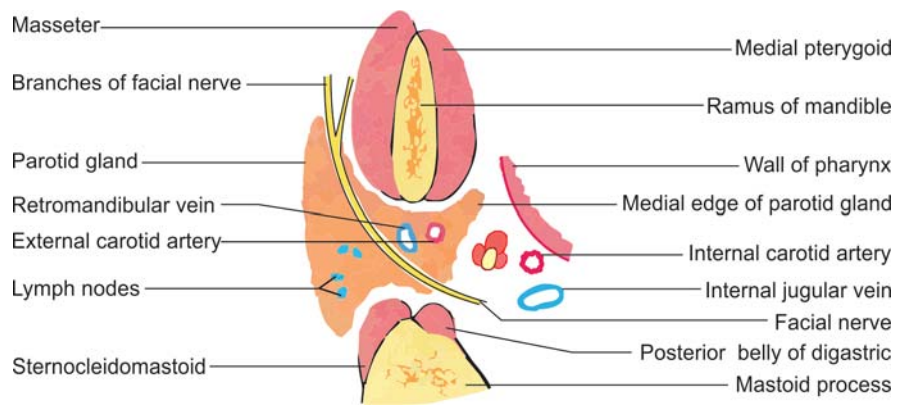


Fig. 6.14: Relations of parotid gland: coronal section

Parotid gland is divided into a superficial and deep portion by the facial nerve, which passes through the gland.



Fig. 6.15: Pleomorphic adenoma

Pleomorphic adenoma of the parotid gland is a common tumor. It is slow growing and can attain a big size if neglected. Treatment is superficial parotidectomy with preservation of the facial nerve. Recurrence may occur if removal is not complete.

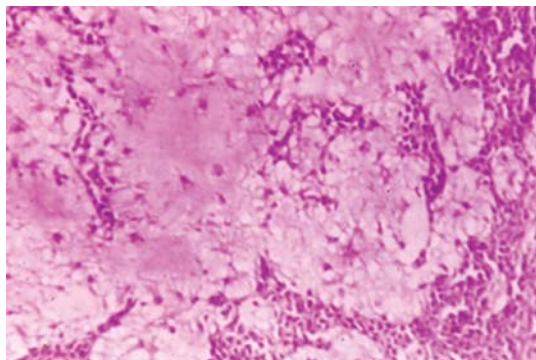


Fig. 6.16: Histopathological examination of pleomorphic adenoma

Photomicrograph of pleomorphic adenoma showing epithelial component merging imperceptibly with mesenchymal tissue. Pseudocartilaginous change is seen in the mesenchyme.

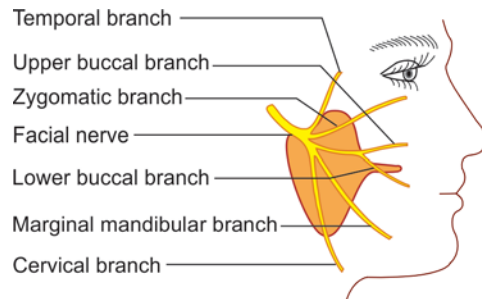


Fig. 6.17: Facial nerve branches in the parotid

The facial nerve emerges from the stylomastoid foramen and courses through the substance of the parotid dividing it into superficial lobe (superficial or lateral to the facial nerve) and deep lobe (deep or medial to the facial nerve). The facial nerve branching pattern within the parotid, can be highly variable. The main trunk bifurcates into the zygomaticotemporal branch and the cervicofacial branch at the pes anserinus and further into the temporal, zygomatic, buccal, marginal, and cervical branches. Pes is located 1.3 cm from the stylomastoid foramen.

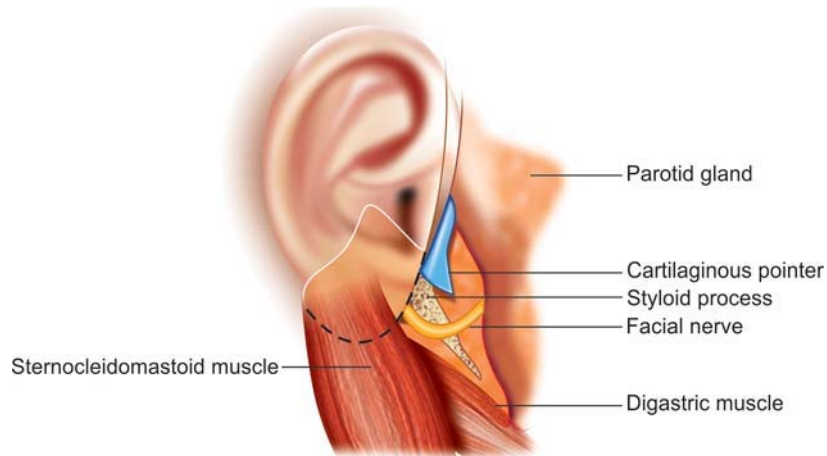
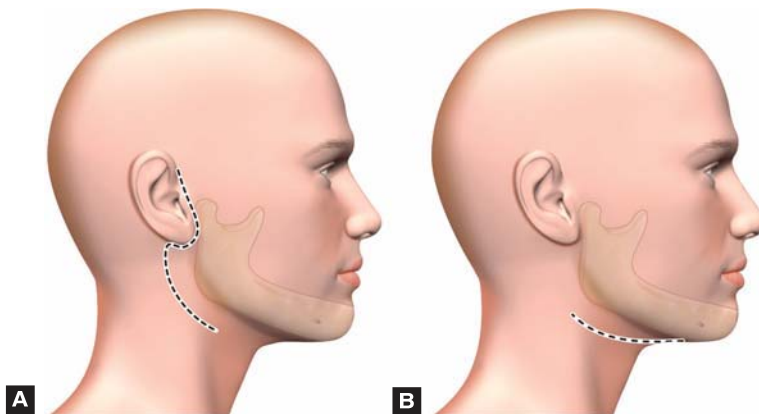


Fig. 6.18: Surgical landmarks of facial nerve in parotid surgery



Figs 6.19A and B: Incisions for parotid and submandibular gland surgery respectively

SUBMANDIBULAR GLAND

Submandibular gland lies in the submandibular triangle. It consists of a larger superficial and smaller and more posterior deep lobe which connect around the posterior border of mylohyoid muscle.



Fig. 6.20: Submandibular duct stones on X-ray

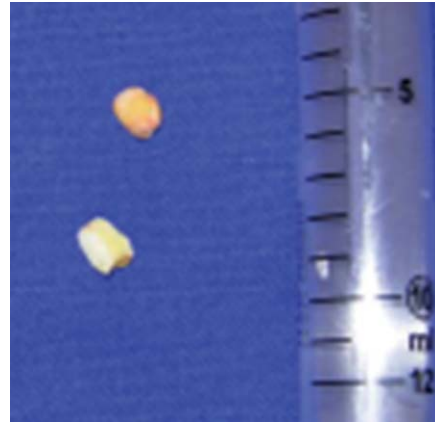


Fig. 6.21: Submandibular duct stones specimen

Ninety percent of salivary calculi are found in submandibular salivary glands due to the nature of secretions and antigravity drainage.

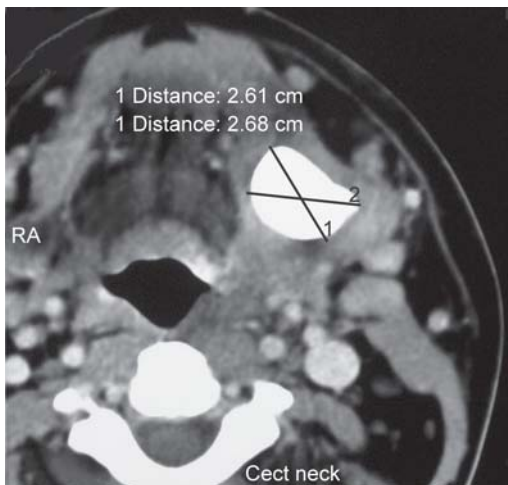


Fig. 6.22: Large submandibular gland stone on CT



Fig. 6.23: Large submandibular gland stone on X-ray

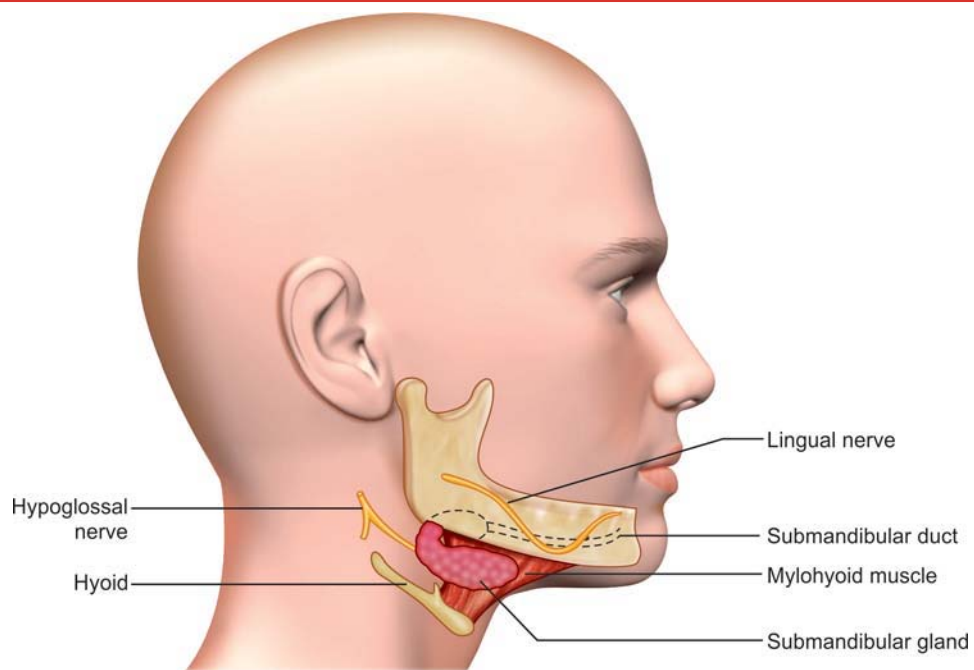


Fig. 6.24: Relations of submandibular duct during gland surgery

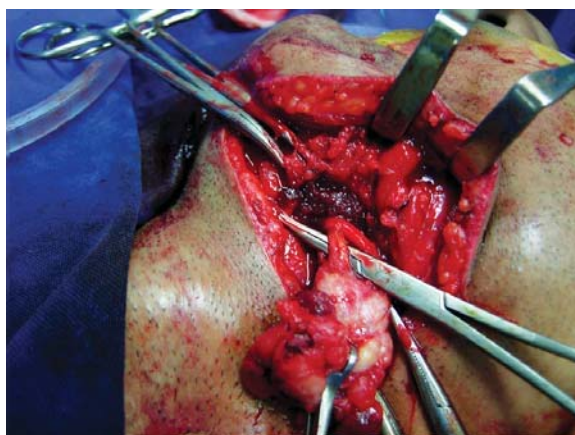


Fig. 6.25: Intraoperative photograph of submandibular gland excision



Fig. 6.26: Submandibular gland specimen with stone

The salivary duct prior to being cut. Note the lingual nerve crossing over it (Fig. 6.25).

THYROID

Thyroid gland occupies the center of neck, in front of and close to the trachea just above the thoracic inlet. It has two lobes which are connected across midline by the isthmus. Because of fascial attachments the gland moves upwards with swallowing and slides under the examining fingers.



Fig. 6.27: Thyroglossal cyst (moves with deglutition and is the only midline neck swelling which moves up on tongue protrusion)

A thyroid scan should be done prior to excision of thyroglossal cyst to confirm that it is not the only thyroid tissue. Since the thyroglossal tract is closely related to the hyoid bone, the central part of the hyoid must be excised together with the cyst; to prevent recurrence. Also the tract is traced upwards to a foramen caecum, where it is excised along with a core of lingual muscle.



Fig. 6.28: Goiter

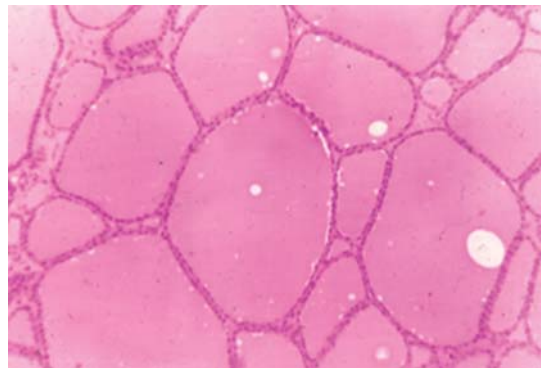


Fig. 6.29: Histopathological examination of colloid goiter

The term goiter is used to describe generalized enlargement of thyroid gland. A discrete swelling in one lobe with no palpable abnormality elsewhere is termed as solitary thyroid nodule. Photomicrograph of goiter shows dilated thyroid acini filled with pink colloid material.

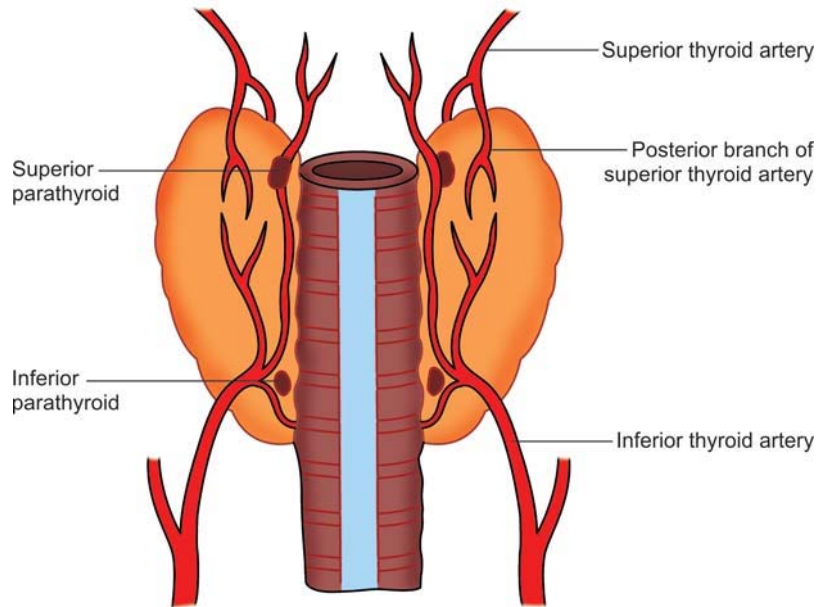


Fig. 6.30: Arterial supply of thyroid gland

Each thyroid lobe is supplied by a superior and an inferior thyroid artery and drained by three veins.

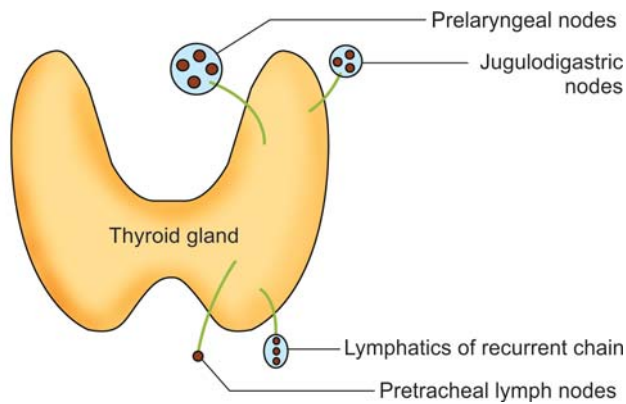
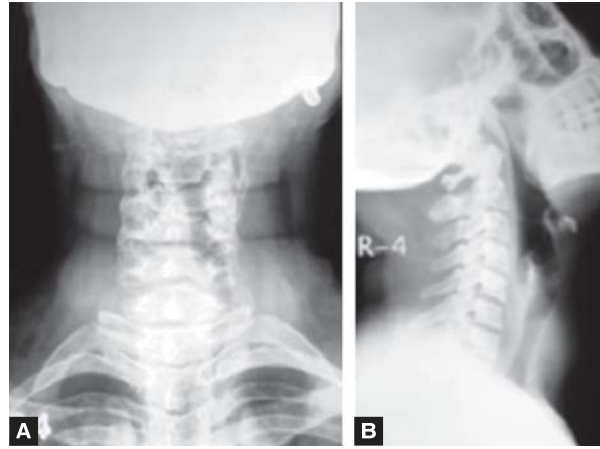


Fig. 6.31: Lymphatic drainage of thyroid gland



Fig. 6.32: Right thyroid lump



Figs 6.33A and B: X-ray showing tracheal shift and compression by thyroid lump



Fig. 6.34: Thyroid scan showing cold nodule on the right side
(Same patient as in Figures 6.32 and 6.33)

Cold nodules are more likely to be malignant as compared to warm nodules.

CAROTID BODY TUMOR



Fig. 6.35: CT with 3D reconstruction: Carotid body tumor (*Source: Carotid Paraganglioma AK. Monga, Deepti Sinha; Indian Journal of Otolaryngology and Head and Neck Surgery. Special issue 2005;217-20*)

A 50-year-old male had presented with a pulsatile swelling in the left carotid triangle. Carotid body tumors (CBT) are treated by surgery or radiotherapy. The patient in figure 6.35 underwent successful surgery.



Fig. 6.36: Carotid body tumor in Axial CT scan (*Carotid Paraganglioma. AK Monga, Deepti Sinha. Indian Journal of Otolaryngology and Head and Neck Surgery. Special issue 2005;217-20*)

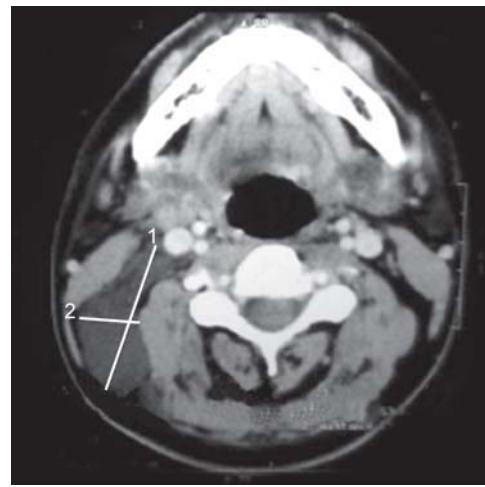


Fig. 6.37: CT: Cervical lymphangioma (*Source: Photo by Dr Mukul Gupta*)

7

PS Saharia, Deepti Sinha

Endoscopy



DYSPHAGIA

Common complaints of swallowing are difficulty in swallowing (Dysphagia), pain on swallowing (Odynophagia), lump or sticking in throat (globus). Dysphagia may affect the oral, pharyngeal or esophageal phases of swallowing. Thorough history and physical examination are important in the diagnosis and treatment of dysphagia.

Table 7.1: Dysphagia

- A detailed history often suggests the cause
- Look for cranial nerve palsies
- Barium swallow is diagnostic in most cases
- Remember that dysphagia and aspiration often occur together
- Aspiration is best assessed with videofluoroscopy
- Endoscopy is mandatory if malignancy is suspected



Fig. 7.1: Drink test

Make the patient drink water to see at what stage of swallowing the patient has dysphagia. Direct observation of lip closure, jaw closure, chewing and mastication, tongue mobility and strength, palatal and laryngeal elevation, salivation, and oral sensitivity is necessary. Cranial nerves V, VII-XII should be examined. Gag reflex should be tested.

Table 7.2: Causes of dysphagia

Oral cavity			
<i>Pharynx and larynx</i>			
	<i>Lumen</i>	<i>Wall</i>	<i>Outside</i>
Stomatitis	Impacted FB	Pharyngitis	Retropharyngeal abscess
Quinsy		Laryngitis	Malignancy
Tonsillitis		Growth	
Palatal palsy		Hysterical	
		Plummer Vinson syndrome	
		Bulbar palsy	
Esophagus			
	<i>Lumen</i>	<i>Wall</i>	<i>Outside</i>
Foreign body		Stricture	Retrosternal goiter
		Cardiospasm	Pharyngeal diverticulum
		Bulbar poliomyelitis	Aneurysm of aorta
		Malignancy	Mediastinal mass
			Dysphagia lusoria

**Fig. 7.2:** Patient with Patterson Brown-Kelly syndrome

Angular stomatitis (white arrow in figure above), glossitis, koilonychia and hypochromic microcytic anemia constitute Patterson Brown-Kelly syndrome. The patient has increasing dysphagia. It is a precancerous condition, commonly seen in female adults.

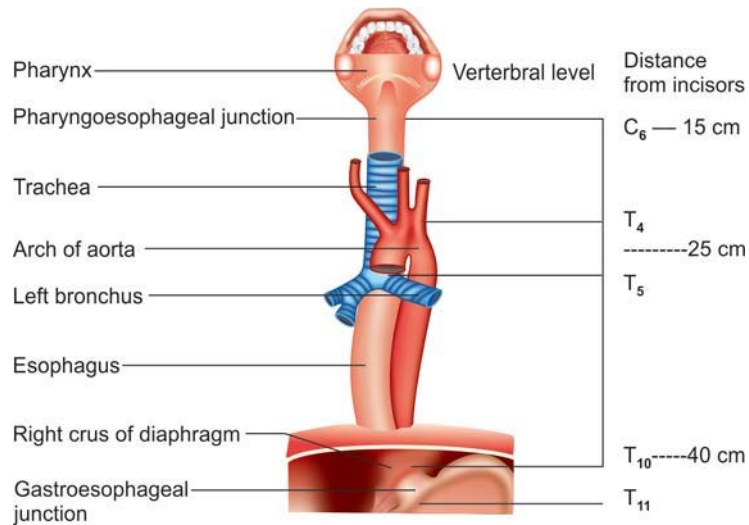


Fig. 7.3: Levels of constrictions of esophagus with distance from incisors

Table 7.3: Key points in management of esophageal foreign bodies

Esophageal foreign bodies

- A carefully taken history is important
- Lateral soft-tissue X-ray of the neck is often helpful. Coins, bones, metallic part of dentures are often seen. Air in the upper esophagus is also a sign of an impacted foreign body. Sharp foreign bodies should be removed at the earliest opportunity
- Ruling out esophageal perforation is important
- Soft foreign bodies may be treated conservatively for a short period
- Esophageal foreign bodies can perforate the esophagus

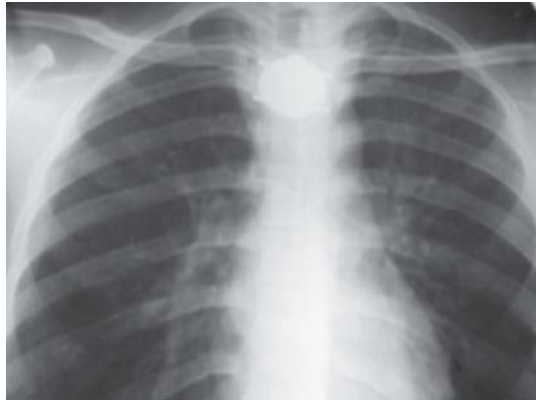


Fig. 7.4: Wrist watch dial in esophagus (*Source:* An amazing case of working wrist watch in the esophagus. Nitin Aggarwal, Deepti Sinha. Indian Journal of Otolaryngology and Head and Neck Surgery 2006;58(1);105-106)



Fig. 7.5: Wrist watch removed (*Source:* An amazing case of working wrist watch in the esophagus. Nitin Aggarwal, Deepti Sinha. Indian Journal of Otolaryngology and Head and Neck Surgery 2006;58(1);105-106)



Fig. 7.6: Foreign body (coin) at level of cricopharynx



Fig. 7.7: Ring in esophagus
(Source: Photo by Dr AK Khurana)

A foreign body in the food passages and bronchi is more common in children than in adults. The size of the foreign body is important to produce the relevant symptoms. Coin is a frequent foreign body to get stuck at the cricopharynx of a child. A denture with or without hooks is common in adults. A sharp foreign body may injure the soft tissue and produce complications.

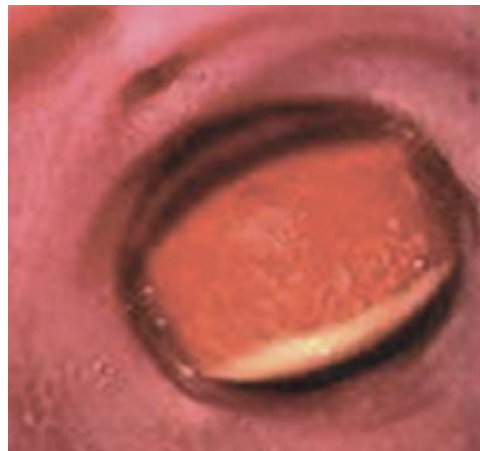


Fig. 7.8: Foreign body esophagus (Source: Photo by Dr AK Khurana)



Fig. 7.9: Foreign body seen in esophagus (*Source:* Photo by Dr AK Khurana)

Mucosal injury may be caused by drugs like potassium chloride, nonsteroidal anti-inflammatory drugs (NSAIDs), or antibiotics (doxycycline, azithromycin, clindamycin, trimethoprim-sulfamethoxazole). Xerostomia may be caused by antihistamines, anticholinergics, alpha-adrenergic blockers or angiotensin-converting enzyme (ACE) inhibitors.

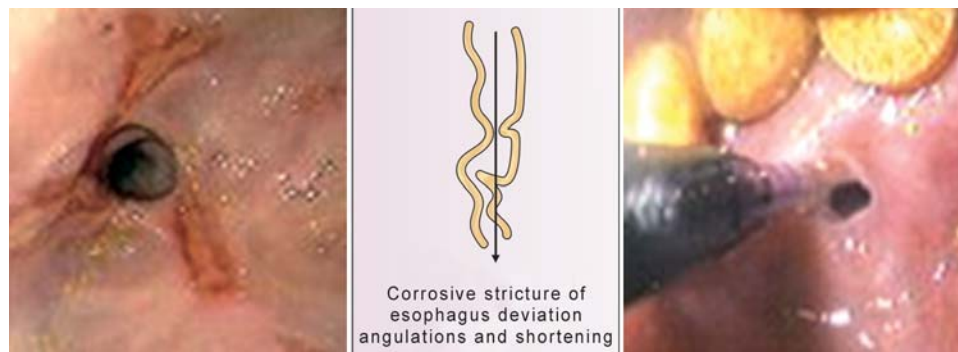


Fig. 7.10: Stricture of esophagus (*Source:* Photo by Dr AK Khurana)

Stricture of the food passage is invariably because of corrosive poisoning. Stricture may be partial or complete. Stricture may distort the shape and the size of the esophagus. A passable stricture is gradually dilated. Retrograde dilation is better.



Fig. 7.11: Pharyngeal diverticulum (*Source:* Photo by Dr AK Khurana)

A pharyngeal diverticulum may be central or lateral. It is the herniation of the pharyngeal mucosa through Killian's dehiscence. Regurgitation of food and dysphagia are the common symptoms. A visible swelling may be felt when the pouch is big. Barium swallow helps in the diagnosis. Diathermy or surgical excision provides the best treatment for the symptomatic cases.

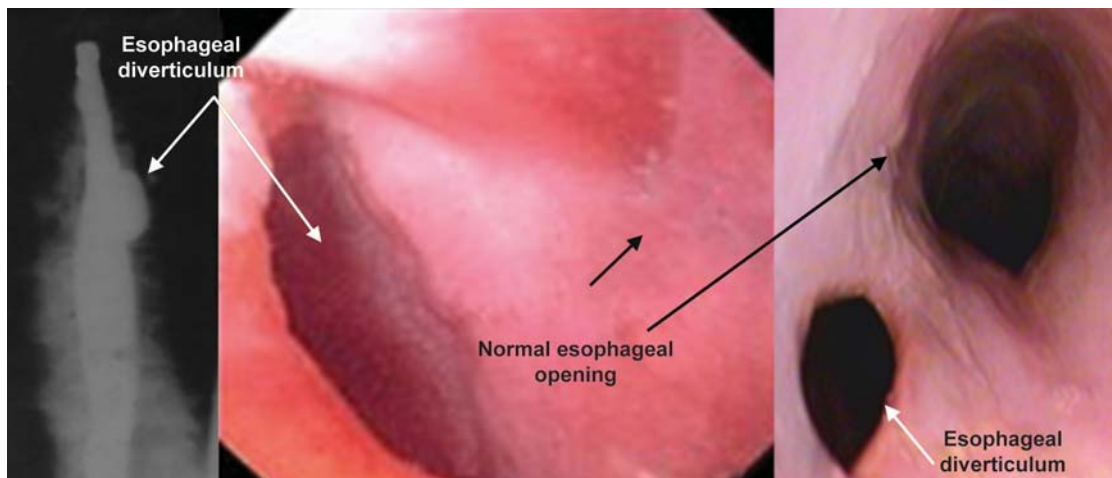


Fig. 7.12: Esophageal diverticulum (*Source:* Photo by Dr AK Khurana)

Diverticulas in the esophagus are rare and usually asymptomatic and are accidentally discovered by barium swallow done for some other reasons.



Fig. 7.13: Barium swallow

Malignancy of the esophagus is not uncommon. The diagnosis is often delayed. A barium swallow showing rat tailing appearance of the esophagus.



Fig. 7.14: Endoscopic view of esophagus showing the obstruction to lumen



Fig. 7.15: Stenting—a stent in the esophagus



Fig. 7.16: Achalasia cardia

Achalasia cardia is failure of the cardiac end of the esophagus to open to allow the food to go into the stomach. Subsequently, esophagus gets enlarged. Patient develops progressive dysphagia and regurgitation of food. Repeated esophageal dilatation is effective. Heller's operation is indicated when dilatation falls.

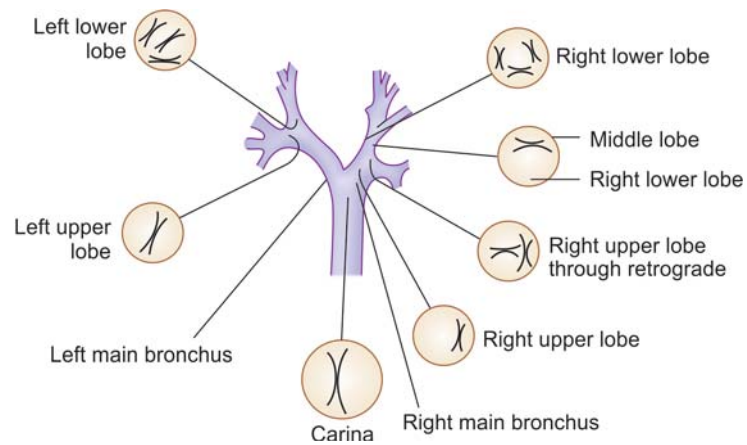


Fig. 7.17: Bronchial tree

Direct examination of the bronchial tree is termed as bronchoscopy. It is performed for diagnostic and therapeutic purposes such as biopsy or foreign body removal in India. Peanuts and other nuts are the most common airway foreign bodies in children.

Table 7.4: Size of bronchoscope

<i>Age group</i>	<i>Lumen (mm)</i>		<i>Length (cm)</i>
Infant	4	X	30
Small child	5	X	30
Large child	6	X	35
Adolescent	7	X	40
Small adult	8	X	40
Large adult	9	X	40

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