Review of Literature
Fig-1: Skeletal basis of the nasal septum

Fig-2: Lateral wall of the nasal cavity with the mucous membrane intact

Fig-3: Lateral wall of the nasal cavity after cutting the conchae to reveal structures deep to them
Review of Literature

Nose is a hollow organ covered with skin, provided with muscles, supported by cartilage, bone and lined with a mucous membrane. The nose is subdivided into two nasal cavities by the cartilaginous nasal septum (Young et al, 2002). Each nasal cavity consists of a vestibule, respiratory and olfactory regions. (Fig – 1, 2, 3)

The anterior half to two third of the vestibular part of the lateral wall of the nose is lined by an extension of epidermis, provided with sebaceous and sweat glands and short hairs, the vibrissae. Posterior part of the vestibule is lined by non keratinized, stratified squamous epithelium which merges back with the respiratory epithelium. The movable nasal columella that separate nostrils – is covered with skin, which merges through a narrow, non keratinized zone of stratified squamous epithelium into the respiratory tract epithelium of septum (Friedmann and Osborn, 1976).

The mucosa of respiratory passage of the nose also known as schneiderian membrane is lined by pseudostratified columnar ciliated epithelium with numerous goblet cells; supported by richly vascular lamina propria containing serous and mucous glands (Young et al, 2002). (Fig – 4)

Paranasal sinuses are also lined by respiratory epithelium. Both nasal mucosa and that of sinuses often contains scattered lymphocytes, plasma cells and macrophages (Friedmann and Osborn, 1976).
Fig. 4: Nasal mucosa (H & E x720)
(E-Pseudostratified ciliated epithelium; M- Mucus glands; V-Venules; S- Serous glands)

Fig. 5: Olfactory mucosa (H & E x720)
(G- Bowman's gland; N- nerve fibres; B- terminal bar)
Olfactory area of the nose include upper one third of the mucosa of the septum, region opposite to the superior concha, the corresponding part of the very narrow roof of the nasal cavity and mucosa above, upper and medial aspects of superior concha itself (Friedmann and Osborn, 1976). (Fig – 5).

The olfactory mucosa is lined by ciliated, bipolar olfactory neural cells that connects to the central olfactory apparatus (Jerome B. Taxy, 1996). Nose arises during the 4th week of the gestation as a frontonasal elevation and by the end of the 6th week develop into definitive nasal cavities. (Jerome B Taxy, 1996).

Nose and paranasal sinuses are inflicted with inflammatory conditions – acute and chronic rhinitis, sinusitis including chronic hypertrophic rhinitis, chronic atrophic rhinitis and ozaena, fungal infections, tuberculosis & leprosy. Polypoidal masses in the form of nasal polyps is the fairly common condition involving nose and paranasal sinuses. Polyps were firstly described by Hippocrates (460-377 BC) and were known as “Nasarsah” in the ancient times.

Benign and malignant tumours of nasal cavity are not very common still constitute a important cause of nasal obstruction, morbidity and mortality. Benign tumour excluding polyps include papilloma, schwannoma, meningioma, hemangioma etc, where as malignant tumours are observed in the form of squamous cell carcinoma, adenocarcinoma, melanoma and various sarcomas. Sometimes paranasal sinus tumours present as a protruding nasal mass.
The various lesions of the nose include –

   a. Saddle nose
   b. Hump nose
   c. Deviated nose
   d. Stenosis and atresia of nose

2. Inflammatory and infectious conditions (Friedman & Osborn, 1976).
   a. Rhinitis and sinusitis
      i. Acute rhinitis
      ii. Chronic non specific rhinitis
      iii. Allergic rhinitis
      iv. Acute sinusitis
      v. Chronic sinusitis
   b. Nasal polyps
      i. Inflammatory
      ii. Allergic
   c. Chronic specific infective rhinitis
      i. Bacterial
         1. Tuberculosis
         2. Leprosy
         3. Syphilis
         4. Rhinoscleroma
         5. Lupus
ii. Fungal

1. Candidiasis
2. Phycomycosis
3. Aspergillosis
4. Rhinosporidiosis
5. Leishmaniasis
6. Rhinophyma

iii. Non healing nasal granuloma of unknown cause

1. Wegener’s disease
2. Stewart granuloma

3. Tumours and tumour like condition of nasal cavity and paranasal sinuses (WHO 1978)
   a. Epithelial tumours
      i. Benign
         1. Squamous cell papilloma
         2. Transitional papilloma
            a. Exophytic
            b. Inverted
         3. Adenoma
         4. Oxyphilic adenoma (Oncocytoma)
         5. Pleomorphic adenoma (mixed tumour)
   ii. Malignant
      1. Squamous cell carcinoma
      2. Verrucous (squamous) carcinoma
3. Spindle cell (squamous) carcinoma
4. Transitional carcinoma
5. Adenocarcinoma
6. Mucinous adenocarcinoma
7. Adenoid cystic carcinoma
8. Mucoepidermoid carcinoma
9. Others
10. Undifferentiated carcinoma

b. Soft tissue tumours

i. Benign

1. Hemangioma
2. Hemangiopericytoma
3. Neurofibroma
4. Neurilemmoma
5. Myxoma
6. Fibroxanthoma
7. Others

ii. Malignant

1. Malignant hemangiopericytoma
2. Fibrosarcoma
3. Rhabdomyosarcoma
4. Neurogenic sarcoma
5. Malignant fibroxanthoma
6. Others
c. Tumours of Bone & Cartilage

i. Benign
   1. Chondroma
   2. Osteoma
   3. Ossifying fibroma
   4. Others

ii. Malignant
   1. Chondrosarcoma
   2. Osteosarcoma
   3. Others

d. Tumours of lymphoid and haematopoietic tissues

i. Malignant lymphomas
   1. Lymphosarcoma
   2. Burkitts tumour
   3. Reticulosarcoma
   4. Plasmacytoma
   5. Hodgkins disease

e. Miscellaneous tumours

i. Benign
   1. Teratoma
   2. Meningioma
   3. Odontogenic tumours
   4. Melanotic neuroectodermal tumour
   5. Others
ii. Malignant
   1. Malignant melanoma
   2. Olfactory neurogenic tumours
      (Esthesioneurogenic)
   3. Others
f. Secondary tumours
g. Unclassified tumours
h. Tumour like lesions
   i. Pseudoepitheliomatous hyperplasia
   ii. Oncocytic metaplasia and hyperplasia
   iii. Cysts
   iv. Mucocele
   v. Angiogranuloma (granuloma pyogenicum)
   vi. Nasal polyp
   vii. Fibromatosis
   viii. Fibrous dysplasia
   ix. Giant cell “reparative” granuloma
   x. Infective granuloma
   xi. Cholesterol granuloma
   xii. Lethal midline granuloma (Stewart’s granuloma)
   xiii. Wegener’s granulomatosis
   xiv. Nasal glial heterotopia (nasal “glioma”)
   xv. Meningocele /meningo-encephalocele
A. RHINITIS AND SINUSITIS

Rhinitis and sinusitis are the most frequently encountered of all pathologic conditions and are the common cause of sneezing, rhinorrhea and obstruction of airways.

The commonest form of acute rhinitis (acute coryza) primarily a viral infection and is the common cause of rhinorrhea, sneezing, fever. Recurrent attacks of acute rhinitis in the presence of predisposing factors (sinusitis, tonsillitis, adenoids, chronic irritation from dust, smoke, cigarette, snuff) leads to chronicity. Histologically the surface mucosa shows a spectrum of changes including thickening of mucous membrane and hyperplasia of mucous glands. The inflammatory infiltrate composed of eosinophils, neutrophils, lymphocytes, plasma cells and histiocytes (Friedmann and Osborn, 1976).

Acute inflammation of nasal sinuses is almost always the result of extension of infections from the nose itself, rarely due to swimming and diving, trauma and dental infections. Acute infection lasting for months or years is called chronic sinusitis. Histologically surface mucosa exhibits basal cell hyperplasia, squamous metaplasia, atypia, ulceration and regeneration. The inflammatory infiltrate is composed of eosinophils, neutrophils, lymphocytes, histiocytes and plasma cells (Fu and Perzin, 1997).
B. NASAL POLYPS

Nasal polypi is the most frequently encountered of all pathologic conditions, both in the clinic and laboratory and most commonly present as nasal mass. Friedmann et al (1976) subgroups polypi into allergic and non allergic while Wilkes (1978) into inflammatory and allergic. Allergic polyps most frequently occur in the turbinate and ethmoid regions while choanal polyps found in the posterior nasal cavity. Histologically the surface ciliated respiratory epithelium shows several abnormalities including basal cell hyperplasia, squamous metaplasia, atrophy and ulceration. Connective tissue stroma is edematous infiltrated by infiltrate including neutrophils, eosinophils, lymphocytes and plasma cells.

Dysplastic epithelial changes in nasal polyps reported by Bustill (1978) and Granulomas in nasal polyp studied by Bustill (1975).


Norlander et al (1993) studied formation of mucosal polyps in the nasal and maxillary sinus cavities by infection.


C. RHINOSPORIDIOSIS

Rhinosporidiosis is a chronic infection caused by rhinosporidium seeberi, causing polypoidal lesion in the nasal cavity characterized by the presence of thick walled sporangia measuring 50-350 microns in diameter and containing numerous spores associated with a heavy chronic
inflammatory reaction with occasional foci of suppuration and foreign body giant cells (Shanmugaratnam, 1978).

This condition has been reported by various authors like – Minchin et al (1905), Tirumurthi (1914), Sengupta SK (1957), Purandare and Deoras (1953), Satyanarayan (1960), Dube and Veliath (1964), Sammaddar and Sen (1990), Mohan et al (1995), Makannavar and Chavan (2001).

According to Kunnam Kutty (1963) disease is commonly seen in the age group of 20-40 years of age.

Disease is transmitted through droplet infection, by close contact with animals and bathing and diving in contaminated water (Makannavar and Chavan, 2001).

D. RHINOSCLEROMA

Rhinoscleroma is caused by gram negative coccobacillus, Klebsiella rhinoscleromatis. They are characterized by heavy infiltration by plasma cells, lymphocytes and large macrophages with abundant clear or vacuolated cytoplasm (Mikulicz cells). There are also numerous russel bodies.

Rhinoscleroma has been reported by various authors such as Ardoin et al (1957), Barilyak (1962), Fisher (1964), Khiga (1965), Derapa (1965) and Yasin et al (1966).

Fisher et al (1964) mentioned that electron microscopy clearly reveals bacillary form within large vacuolated cells corresponding to mikulicz cells.
Yassin et al 1966 reported a squamous cell carcinoma change in a case of rhinoscleroma.

E. TUBERCULOSIS

It is very rare in the nose and present in the form of polypoidal or a tumour like mass. Microscopic picture shows typical tubercles as elsewhere in the body.

Ahmed Hakeem (1958) reported a case of tuberculosis in 20 years old pregnant lady.

F. LEPROSY

Leprosy (usually the lepromatous form) may involve the mucous membrane of nasal cavity and nasopharynx. Histologically the tissues are infiltrated by chronic inflammatory cells including variable numbers of large somewhat foamy macrophages (Lepra cells) with the acid fast stain mycobacterium lepra can usually be demonstrated in lepra cells cytoplasm.

BENIGN LESIONS / TUMOURS OF NOSE (Friedmann & Osborn, 1976)

Ratio of benign to malignant tumours in the nasal cavities is about six to one while in the nasal sinuses the ratio has been almost exactly the reverse of these figures (Friedmann & Osborn, 1976). Honso (1965) found 1 benign for every 3 malignant tumours in the nasal cavity. Most of the benign tumour presented with blocked nose and episodes of epistaxis.

A. PAPILLOMAS

These tumour are generally polypoid. They may be further classified into inverted and exophytic papillomas.
The fungiform or exophytic type typically occurs on nasal septum has a papillary or exophytic growth with fibrovascular cores and affects younger patients than inverted papillomas. The proliferating cells are similar to those of inverted papillomas, including mature squamous cells. Inverted papillomas presented with a variety of nonspecific symptoms (nasal stuffiness, fullness, rhinorrhea and epistaxis) and found most often on lateral nasal wall. Histologically, inverted papillomas show a variable proliferation of basal cells, thickened epithelium, squamous metaplasia and koilocytic changes.


Disease is more common in men past the age of 40 years. Osborn (1956), Verner et al (1959).

Osborn DA (1956) mentioned that condition was almost confined to one side only.

Histological evidence of malignancy was observed by various authors, Hellman (1897), Ringertz (1938), Austin J Smith (1963) and Lampertico (1963).

**B. HEMANGIOMA**

Hemangiomas are found most often in the anterior nasal septum, followed by turbinates and vestibule. Capillary hemangiomas are seen most frequently while cavernous hemangiomas are identified less often.

In nasal regions angioma has its peak incidence in 5th and 6th decades (Friedmann & Osborn, 1976).
Microscopically well formed capillaries are arranged in a lobular pattern, the intervening connective tissue is of varying density but often rather odematous. Lesions in which there is heavy inflammatory infiltrate are known as “pyogenic granuloma”.

Osborn (1959) studied 51 cases and held that majority of the cases occur in 5th decade and there is no significant sex difference.

C. ANGIOFIBROMA (JUVENILE ANGIOFIBROMA)

It is a disease pre-eminently of boys. It is rare before the age of 10 years. The neoplasm occurs almost exclusively in the nasopharynx of young males in the 10-25 years of age group. Clinically angiofibromas produce nasal obstruction, epistaxis and massive haemorrhage.

Microscopically, these tumour are composed of a characteristic fibrous stroma in which are found numerous blood vessels of various sizes and shapes (Fu and Perzin, 1997).

D. NEURILEMMOMA (SCHWANNOMA)

Neurilemmoma rarely involve the nasal cavity and paranasal sinuses. Benign tumour of schwann cells is usually well demarcated or encapsulated.

Microscopically, characteristic Antoni type A pattern with regimentation of the nuclei in twisted rows or palisades (verocay bodies) and an Antoni type B pattern with loosely arranged cells with in a wide meshed micocystic fibrillar stroma may be recognized (Shanmugaratnam and Sobin, 1978).
Cancer of nasal cavity or paranasal sinuses is a relatively rare problem with a yearly risk factor estimated at approximately one case for every 100,000 people. These cancers occur more often in men (2 to 1) and usually after the age of 40 except for tumours of minor salivary gland origin and esthesio neuroblastomas, which may appear before the age of 20 (Devita et al, 2000).

Mac comb and Martin's (1942) concluded that mean age for malignant nasal tumours is 55 years and is more common in males than females.

Sino-nasal tumours are infrequent but include the entire gamut of both epithelial and mesenchymal benign and malignant neoplasms (Vaideeswar et al, 1999).

Wille found that only 1.62% of the cancer patients suffered form malignant tumours of the nose and accessory sinuses.

Edgar L Frazell et al (1963) mentioned that cancer of the nasal cavity and sinuses comprised approximately 3% of all cases of malignant tumours in the upper alimentary and respiratory tracts. Out of these 3% cases, 27.1% occurred in the nasal cavity alone.

There is evidence that occupational exposure to certain inhaled irritants – chromium, nickel, arsenic, wood dust, sawmill work, leather, and carpentry is a causative factor in some cases (Willis, 1967).

Newman (1890) reported a large adenocarcinoma of the nares of a man aged 47 years who had worked for 20 years in a chromate plant.
Stephens (1933), Bridge (1939), Morgan (1958) and others referred to cases of nasal cancer in nickel workers.

Keen et al (1955) reported a high incidence of nasal carcinomas among South African Bantu who habitually take a snuff made from burnt plant stems, which contains benzpyrene.

The effect of thorotrast, containing radioactive metal thorium has been well studied and there is association between thorotrast and maxillary sinus carcinoma (Devita et al, 2000).

Little is known about the pathogenesis of carcinomas of nasal cavity and nasal sinuses. Lewis and Castro (1972) reported that previous inflammatory lesions may predispose to the development of carcinoma. Hasegawa (1988) reported malignant transformation of nasal polyp. Parrott LH (1994) reported extramedullary plasma cytoma in a benign appearing nasal polyp in 52 year old white male.

Carcinomas – squamous carcinomas, carcinomas of transitional type, anaplastic carcinomas and adenocarcinomas – account about 50 percent of malignant tumours of nasal cavities. In the nasal sinuses they account for about 80%.

Clinically, malignant tumours involving the upper respiratory tract produce nonspecific symptoms including nasal obstruction, epistaxis, nasal discharge and pain. They readily spread by direct extension into adjacent structures – sinuses, cranial cavity and orbital cavity.
A. SQUAMOUS CELL CARCINOMA

In the nasal cavity about 75% of carcinomas are squamous and arise anteriorly. Because of their situation these tumours are diagnosed earlier and have better prognosis than those arising in nasal sinuses. Carcinomas that arise farther back in the nasal cavities are equally divided between transitional and anaplastic growth with only an occasional adenocarcinoma (Friedmann & Osbon, 1976).

The involvement of human papillomavirus is suggested by the histologic observation of koilocytotic change within the squamous mucosa associated with nasal squamous carcinoma and by studies using the technique of modern molecular biology (Jerome B Taxy, 1996).


Histologically, these tumours are divided into Keratinizing and nonkeratinizing variants. The presence of intercellular bridges and keratinization are obvious in well differentiated neoplasm. These features are barely evident in the poorly differentiated carcinomas (Fu and Perzin, 1997).

The most commonly used system for histological grading is a modification of the original Broder’s system, consisting of three grades based on the amount of keratin, the degree of nuclear atypia and the mitotic activity. On the basis of these features squamous cell carcinoma is divided into –

- Well differentiated
- Moderately differentiated
- Poorly differentiated

**B. UNDIFFERENTIATED CARCINOMA**

A highly anaplastic and aggressive carcinoma, most patients have bony, cranial and orbital involvement. The tumour cells appear undifferentiated and without keratinization.

**C. ADENOCARCINOMA**

Adenocarcinoma originate from salivary glands or from mucoid cells. Histologically most salivary gland tumors are adenoid cystic carcinomas or adenocarcinomas of no specific histologic type. The tumour exhibit no sex difference.

An increased incidence of adenocarcinoma of nasal cavity and paranasal sinuses have been found in wood and furniture workers (Fu and Perzin, 1997).


Keenam and Elliott (1991) reported, adenocarcinoma with unusual presentation.

**D. VERRUCOUS CARCINOMA**

A warty variant of squamous cell carcinoma, characterized by a pronounced overgrowth of well differentiated keratinizing epithelium thrown up into regular vertical folds. The verrucous carcinoma is distinguished form well differentiated squamous cell carcinoma by
minimal atypia, its growth pattern and absence of metastasis
Shanmugaratnam and Sobin (1978).

**E. HEMANGIOPERICYTOMA**

Occurs usually in older adults. Histologically tumour is characterized by
the proliferations of round, oval or spindle shaped cells of rather uniform
size surrounded by reticulin fibres and arranged about vascular spaces
lined by a single layer of endothelial cells.

Mangwana et al (1996) reported hemangiopericytoma in a 28 year old
female.

**F. CHONDROSARCOMA**

A malignant tumour characterized by the formation of cartilage, but not
of bone, by the tumour cells.

Sudhanshu M Sethy (1999) reported 3 cases of chondrosarcoma nose and
paranasal sinuses.

**G. TERATOCARCINOSARCOMA**

Are rare malignant tumours which display combined features of an
immature malignant teratoma and a carcinosarcoma.

Pai et al (1997) reported 3 cases of sino-nasal teratocarcinosarcoma. The
patients (2 male, 1 female) were 33, 60 and 69 years old respectively.

Kailash and Panicker (1999) reported a case of teratocarcinosarcoma in a
46 yr old male.

**H. PLASMACYTOMA**

The upper respiratory tract is the most frequent site of extramedullary
plasmacytoma, it arises most frequently in the mucous membrane of the
nasal cavity itself. Histologically, these neoplasms are composed of a pure population of plasma cells, usually growing in solid sheets. Most of the cells show atypical features.


1. LYMPHOMAS

Any of the types of malignant lymphoma may arise as a primary tumour in the upper respiratory tract.

They account for 10 percent of all malignant tumours of the nasal region. The disease shows male predominance and occurred over a wide age range, with a median of 52 years. Most nasal / nasopharyngeal lymphomas are peripheral T-cell neoplasms (Chan et al, 1987).

PATTERN OF SPREAD OF MALIGNANT NASAL TUMOURS

(Mendenhall et al, 2000)

1. Malignant lesions of nasal vestibule (squamous cell carcinoma, basal cell carcinoma & adenexal carcinoma) - invade alar and septal cartilages, skin of nose, upper lip, nasal cavity. Lymph node spread is usually to a solitary ipsilateral submaxillary node but may be bilateral.

2. Lesions of nasal cavity- those arising on the lateral wall invade axillary sinus, ethmoids and orbits.
Those arising in olfactory region invade ethmoids, orbits, anterior cranial fossa, frontal lobes. These lesions also destroy septum and may invade through nasal bone to skin.

In advanced lesions nasopharynx and sphenoid sinuses are involved.

Esthesio-neuroblastoma and minor salivary gland tumours have greater propensity for perineural spread.

3. Maxillary sinus tumours spread to the oral cavity, infratemporal fossa, nasal cavity, ethmoid and frontal sinuses, lacrimal apparatus and medial inferior orbit.

4. Ethmoid sinuses lesions spread to nasal cavity, medial orbit, maxillary antrum, nasopharynx, sphenoid sinus and anterior cranial fossa.

5. Sphenoidal sinus tumours spread to nasopharynx and nasal cavity.

TUMOUR STAGING (Ackerman’s Pathology, 9th Ed, 2004)

The AJCC staging system for tumours of the nasal vestibule, nasal cavity and paranasal sinuses is as follows:

PRIMARY TUMOUR (T)

Nasal vestibule: The staging system for skin cancer is used.

MAXILLARY SINUS

Tx Primary tumour cannot be assessed.

T0 No evidence of primary tumour.

Tis Carcinoma in situ
T1  Tumour limited to the maxillary sinus mucosa with no erosion or destruction of bone.

T2  Tumour causing bone erosion or destruction including extension into the hard palate and/or middle nasal meatus, except extension to posterior wall of maxillary sinus and pterygoid plates.

T3  Tumour invades any of the following: bone of the posterior wall of maxillary sinus, subcutaneous tissues, floor of medial wall of orbit, pterygoid fossa, ethmoid sinuses.

T4a Tumour invades anterior orbital contents, skin of cheek, pterygoid plates, infratemporal fossa, cribiform plate, sphenoid or frontal sinuses.

T4b Tumour invades any of the following: orbital apex, dura, brain, middle cranial fossa, cranial nerves other than maxillary division of trigeminal nerve (vz), nasopharynx or clivus.

**NASAL CAVITY AND ETHMOID SINUS**

Tx  Primary tumour cannot be assessed.

T0  No evidence of primary tumour

Tis Carcinoma in situ

T1  Tumour restricted to any one subsite, with or without bony invasion.

T2  Tumour invading two subsites in a single region or extending to involve an adjacent region within the nasoethmoidal complex, with or without bony invasion.
T3 Tumour extends to invade the medial wall or floor of the orbit, maxillary sinus, palate, or cribiform plate.

T4a Tumour invades any of the following: anterior orbital contents, skin of nose or cheek, minimal extension to anterior cranial fossa, pterygoid plates, sphenoid or frontal sinuses.

T4b Tumour invades any of the following: orbital apex, dura, brain, middle cranial fossa, cranial nerves other than V2, nasopharynx or clivus.

REGIONAL LYMPH NODES (N)

Nx Regional lymph nodes cannot be assessed.

N0 No regional lymph node metastasis.

N1 Metastasis in a single ipsilateral lymph node, 3cm or less in greatest dimension.

N2 Metastasis in a single ipsilateral lymph node, more than 3cm but not more than 6cm in greatest dimension or in multiple ipsilateral lymph nodes, none more than 6cm in greatest dimension, or in bilateral or contralateral lymph nodes, none more than 6cm in greatest dimension.

N2a Metastasis in a single ipsilateral lymph node, more than 3cm but not more than 6cm in greatest dimension.

N2b Metastasis in multiple ipsilateral lymph nodes, none more than 6cm in greatest dimension.

N2c Metastasis in bilateral or contralateral lymph nodes, none more than 6cm in greatest dimension.
Metastasis in a lymph node, more than 6cm in greatest dimension.

**DISTANT METASTASIS (M)**

Mx Distant metastasis cannot be assessed.

M0 No distant metastasis.

M1 Distant metastasis.

**DIAGNOSTIC METHODS FOR STUDY OF NASAL LESIONS AND TUMOURS**

1. **ANTERIOR RHINOSCOPY**

This procedure is carried out using a head mirror and a light source. Examination of nasal vestibule by raising the tip of nose for redness and swelling (eg. furunculosis).

An assessment of nasal airway is done by keeping a cold glass slide or a metallic tongue depressor just in front of nostrils. The difference on the two sides is an indication of nasal obstruction.

**A) EXAMINATION WITH A NASAL SPECULUM**

A Thudicum’s speculum or a St. clair. Thompson’s speculum with a handle are commonly used for examination. The nasal cavities (floor, lateralwall, septum and posterior portions) are properly examined. Colour and congestion of nasal mucosa if any variation from normal are observed. Septal deviation, spurs, prominent vessels or crusting in little’s area, perforation of septum are noted.

The anterior ends of inferior and middle turbinates are visible on anterior rhinoscopy. Any atrophy (e.g. atrophic rhinitis) or hypertrophy (Chronic rhinitis, vasomotor rhinitis or allergic rhinitis) of turbinates is noted.
The meati are mostly covered by the turbinates and hardly visible on anterior rhinoscopy. The meati are noted for discharge, local edema or redness.

**B) POSTURAL TEST**

Is done to check the group of sinus involvements (anterior or posterior).

**C) EXAMINATION OF ORAL CAVITY AND OROPHARYNX**

On examination of the oral cavity in relation to nasal and paranasal sinus disease, it is important to note following –

- Gingivobuccal sulcus for any fullness or discharge
- Carious teeth, loose teeth
- Widening of alveolus
- Oroantral fistula
- Bulging of soft palate because of mass in nasopharynx

**2) POSTERIOR RHINOSCOPY**

This procedure permits the examination of the posterior aspects of nose and nasopharynx. The posterior end of septum is seen as a vertical edge. On each side of posterior end of septum are seen posterior choane. The posterior edges of the inferior, middle and superior turbinates are seen on the lateral side of nasal cavity.

Discharge from the maxillary sinus, posterior ethmoidal sinuses and sphenoid sinuses can be seen by posterior rhinoscopy.

Antrachooanal polyp, nasopharyngeal angiofibroma or nasopharyngeal cancer if present can also be noted by posterior rhinoscopy.
3. RHINOMANOMETRY

Is done for measurement of nasal air flow in studying nasal obstruction.

4. NASOPHARYNGOSCOPY

Is useful for evaluating cases of suspected cancer. The nasopharyngoscope is passed along the inferior turbinate and examination of nasopharynx done through its window.

5. PLAIN X-RAYS

It is difficult to examine the nasal cavity and all paranasal sinuses on one projection, so the examination of individual sinus requires many views. The few standard views that are taken are as follows –

a) Occipitomental view (waters view) – Demonstrate mainly maxillary sinuses, nasal cavity, septum, frontal sinuses.

b) Occipitofrontal view (Caldwell view) – Visualizes frontal sinuses, maxillary antrum and nasal cavity.

c) Submentovertical view – This view is useful for demonstrating sphenoid sinuses, ethmoids, nasopharynx, petrous apex, post wall of maxillary sinus.

d) Lateral view – The maxillary, ethmoidal and frontal sinuses superimpose each other but this film is useful for demonstrating the adenoid mass or tumour in the nasopharynx.

6) COMPUTED ANGIOGRAPHY (CT)

CT is currently the modality of choice in the evaluation of paranasal sinuses and adjacent structures. Its ability to optimally display bone, soft tissue and air facilitates accurate depiction of anatomy and extent of
disease in and around paranasal sinuses. In contrast to standard radiography, CT can clearly show the fine bony anatomy of the osteomeatal channels.

7) MAGNETIC RESONANCE

Although magnetic resonance imaging better visualizes soft tissue than CT it does not visualize cortical bone.

8) BIOPSY

The lesions present at surface that is easily approachable, biopsy is performed directly under local anaesthesia and infiltration by epinephrine.

Lesions present in sinuses should be biopsied transnasally. The material obtained were subjected for histological and immunohistochemical study.

9) ASPIRATION CYTOLOGY


10) ANGIOGRAPHY

The external carotid angiography may be helpful in nasopharyngeal angiofibroma and other vascular lesions of the nose and paranasal sinuses.


Leprini et al (1993) reported immunohistochemical study of nasal mucosa of patients with fibrous polyps.
Coste et al (1996) studied nasal polyposis pathogenesis by immunohistochemical study of epithelial cell proliferation.

**TREATMENT**

*Nasal vestibule* : Both surgery and radiation therapy produce a high degree of success. Radiation therapy is usually the preferred treatment because of the deformity produced by excision. Excision is preferred for very small lesions, the removal of which will not produce cosmetic deformity or require reconstruction.

**NASAL CAVITY**

*Selection of treatment modality* – The histology, extent and location of the malignant tumour in the nasal cavity are all considered when treatment decisions are made.

Inverting papilloma is treated initially by surgical excision.

Irradiation is recommended for lesions that are incompletely resected, for patients with multiple recurrences and for those in which carcinoma is found in the specimen.

Squamous cell carcinoma and adenocarcinoma of the nasal cavity can be treated with surgery, irradiation or both.

*Surgical treatment* – Lateral rhinotomy provides the best access for resection of lesions of the nasal cavity. The lateral wall of the nose may be removed by this approach for resection of inverting papilloma and other localized neoplasms. More advanced lesions require removal of involved sinuses and orbit. A craniofacial procedure may be required.
Irradiation technique – The external-beam irradiation technique emphasizes an anterior portal with one or two lateral portals. Contiguous structures such as maxillary sinus, ethmoid sinus, medial orbit, nasopharynx, base of the skull, and sphenoid sinus are generally included in the initial treatment volume as required. The treatment volume is reduced after 50 Gy to include the original gross disease with a margin. Advanced lesions may require inclusion of an entire orbit.

Combined treatment policies – If combined treatment is planned, perform the operation first to avoid obscuring the extent of tumor. Irradiation is started 4 to 6 weeks after ward. The dose is usually 60 Gy in 6 weeks to 65 Gy in 7 weeks for clear margins.

MAXILLARY SINUS

Surgical resection gives the best results. Early infrastructure lesions may be excised and cured by surgery alone, but for most other cases, irradiation is given postoperatively even if margin are negative. Extension of cancer to the base of the skull, nasopharynx or sphenoid sinus contraindicates surgical excision.

ETHMOID SINUS

Ethmoid sinus lesions are usually extensive when first diagnosed. Radiation therapy alone produces better results than surgery alone and is the preferred single treatment.

SPHENOID SINUS

The treatment of sphenoid sinus lesions is with radiation therapy.