

Chapter 14: Nose and Sinuses

Embryology of the Nose (see also Chap. 11)

Anatomy

A. External Nose

Bony framework of the external nose consists of:

1. Nasal bone
2. Frontal process of the maxilla
3. Nasal process of the frontal bone.

Cartilaginous framework of the external nose consists of:

1. Lower lateral (greater alar), right and left.
2. Quadrilateral cartilage of the septum.
3. Upper lateral (lateral nasal), right and left.
4. Lesser alar, right and left.
5. Sesamoid.

Muscles of the external nose are:

1. Nasalis (constrictor).
2. Depressor septi (constrictor).
3. Procerus (dilator).
4. Dilator naris (dilator).
5. Angular head of the quadratus labii superioris (dilator).
6. Depressor alaeque nasi (constrictor).

Arterial blood supply is from:

1. External carotid artery.

Facial artery:

- a. Lateral nasal.
- b. Angular.
- c. Alar.
- d. Septal.
- e. External nasal.

2. Internal carotid artery.

Ophthalmic artery:

- a. Dorsal nasal.

Lymphatics: Via anterior facial vein to submandibular nodes.

Sensory innervation is the trigeminal nerve through:

1. Ophthalmic division.

- a. Nasociliary.
- b. External nasal.
- c. Infratrochlear.

2. Maxillary division:

- a. Infraorbital.

B. Internal Nose

1. The roof of the nose is formed by the cribriform plate of ethmoid bone (most of the roof), the frontal bone anteriorly, and the sphenoid bone posteriorly.

2. The nasal septum consists of:

- a. Septal cartilage.
- b. Vomer.
- c. Perpendicular plate of the ethmoid.
- d. Maxillary crest.
- e. Premaxilla.

3. There are three turbinates arising from the lateral wall. The inferior turbinate is the largest. The mucous membrane covering this structure is thick and contains numerous venous plexus forming a cavernous erectile type of tissue. The bony portion connects the palatine bone, the ethmoid bone, the maxilla, and the lacrimal bone. The middle turbinate is the second largest and lies above the inferior turbinate. The mucous membrane is similar to that found in the inferior turbinate. It is actually a bony projection of the ethmoid bone. The superior turbinate is the smallest of three turbinates having a much thinner mucous membrane. This is also a projection of the ethmoid bone. The supreme turbinate is occasionally found and is extremely small.

4. There are three meatus in the lateral wall of the nasal cavity:

- a. Inferior meatus: Nasolacrimal duct opens here. Damage to the nasolacrimal duct can be minimized by avoiding damage to the attachment of the inferior turbinate. The orifice of

the duct is located on the lateral wall from 3-3.5 cm behind the posterior margin of the nostril.

b. Middle meatus: There are two prominent objects in view in the outer wall of the middle meatus, namely the convex surface of the bulla ethmoidalis and immediately beneath it the well-defined curved margin of the uncinate process of the ethmoid. Between these two structures there is a narrow interval, the semilunar opening or hiatus semilunaris; this opening serves as a communication between the middle meatus and the small channel or canal named the ethmoidal infundibulum.

The anterior ethmoid, maxillary, and frontal sinuses drain into the ethmoidal infundibulum. The frontal sinus and anterior ethmoid cells usually drain into the anterior upper portion, and the maxillary sinus drains posteriorly to the frontal sinus. Accessory ostia of the maxillary sinus are present in 30-40% of all sinuses.

c. Superior meatus: Posterior ethmoid sinus opens here.

d. Supreme meatus: Occasionally present.

e. Sphenoethmoidal recess: Sphenoid sinus drains into the sphenoethmoid recess above and behind the superior turbinate. The ostium is usually in the posterior wall of the recess. The large opening of the sinus in the base of the sphenoid is partially enveloped by a scroll-like bone, the sphenoidal concha or turbinate (bone of Bertin).

5. Arterial supply:

a. Internal carotid artery.

Ophthalmic artery:

1) Anterior ethmoid: Anterosuperior portion of septum and lateral wall. The anterior ethmoidal is the second largest vessel supplying the internal nose.

2) Posterior ethmoid: Septum and lateral wall superiorly.

b. External carotid artery.

Internal maxillary artery:

1) Sphenopalatine: Most of the posterior part of the nasal septum and most of the lateral wall of the nose, especially posteriorly.

a) Nasopalatine (posterior septal): Roof, septum, and floor.

2) Descending palatine: Lateral wall posteriorly.

3) Pharyngeal: Roof posteriorly.

The two terminal branches of the third part of the maxillary artery are the posterior nasal artery and the sphenopalatine artery (Nomina Anatomica, 1966). Others have used the term sphenopalatine to refer to the last centimeter of the maxillary artery, and described this as bifurcating into the lateral nasal and septal branches. (The septal branch also has been called the nasopalatine artery.)

Superior labial artery (from facial artery): Tip of the septum and ala nasi. Its anastomosis with a branch of sphenopalatine forms Kiesselbach's plexus in Little's area.

6. Venous drainage opens into:

- a. Sphenopalatine and anterior facial veins.
- b. Ophthalmic veins.
- c. Veins of the orbital surface of the frontal lobe of the brain (via foramina in the cribriform plate).
- d. Superior sagittal sinus (via foramen caecum).

7. Innervation:

a. Anterior ethmoid: From ophthalmic division of trigeminal.

1) Medial.

2) Lateral.

b. Branches of the sphenopalatine ganglion:

1) Lateral posterior superior (short sphenopalatine).

2) Medial posterior superior (septal).
Nasopalatine (long sphenopalatine).

3) Greater palatine.

c. Olfactory nerves: Roof, upper third of nasal mucous membrane.

8. Lymphatic drainage: Anterior part of cavity to submandibular nodes. Posterior part of cavity to upper deep cervical glands.

9. Mucous membrane:

a. Pseudostratified ciliated columnar epithelium, except in:

1) Vestibule and nares: Stratified squamous.

2) Olfactory areas: Nonciliated pseudostratified columnar epithelium with serous glands of Bowman and bipolar olfactory cells.

Nasal Physiology

The nose participates in three major functions: (1) respiration, (2) olfaction, and (3) phonation.

A. Respiration

1. Acts as rigid airway for inspiration and expiration of air.
2. Heat exchange: Inspired air is heated to about 36°C, while heat is withdrawn from expired air.
3. Humidification: Inspired air is humidified to 75-80%, while water is withdrawn from expired air to reduce the amount of insensible loss.
4. Filtration: The nose removes most particles 4.5 microm in size from inspired air, some as small as 1 nanom.
5. Mucociliary transport depends on ciliary activity and production of nasal secretions; over 1000 cc of secretions produced per day; acts to clear contaminants of inspired air from nose. Mucus contains muramidase, which breaks down bacterial cell walls, as well as IgA and IgE immunoglobulins.
6. Nasopulmonary reflex: Via hypothalamus, influences both nasal airway resistance and pulmonary compliance.

B. Olfaction: Odoriferous particles must be in solution. They stimulate bipolar neurons which converge to form the olfactory nerve, which leaves the nose via the cribriform plate.

C. Phonation: Adds resonance to the voice.

Nasal Pathology - External Nose

A. Furunculosis

1. Superficial abscess, occurring most frequently around the nasal vestibule in the region of hair follicles, causing erythema, swelling, and marked pain.
2. Staphylococcus aureus usual infecting agent.
3. Untreated, can cause serious complications, i.e. cavernous sinus thrombosis.
4. Treatment with penicillinase resistant antibiotics, heat, incision and drainage (I and D) if pointing.

B. Impetigo

1. Superficial skin infection presenting with a vesicopustule formation which forms yellow crusts.
2. Caused by both hemolytic staphylococci and group A hemolytic streptococci.
3. Treatment with appropriate antibiotic.

C. Erysipelas

1. Acute streptococcal infection of the skin.
2. Initial symptoms include feverishness, headache, and malaise.
3. Lesion has advancing border which is raised from surrounding skin. Skin is tense and dark dull red, hot, and tender.
4. Treated with systemic penicillin.

D. Lupus vulgaris

1. Form of tuberculosis involving external nose and adjacent facial areas.
2. Produces brownish, gelatinous nodules which may coalesce.
3. Therapy is identical to that for tuberculosis of other anatomic areas.

E. Lupus erythematosus

1. Disease of unknown cause, featuring altered immune reactivity, which can effect any organ system.
2. Pleomorphic, erythematous, maculopapular eruptions occur commonly on face, sometimes presenting as a "butterfly" rash.
3. Diagnosis is difficult and no curative treatment is known.

F. Syphilis

1. Venereal spirochetosis caused by the organism Treponema pallidum.
2. Primary syphilis of the nose is rare, usually occurring 3-4 weeks after contact as indurated, painless ulceration at the mucocutaneous junction or adjacent septum, accompanied by disproportionate adenopathy.
3. Widespread secondary lesions occur as the primary lesion is disappearing. They appear as erythematous papulosquamous eruptions, annular lesions, mucous patches, and

condyloma. The disease resembles an acute rhinitis in the nose accompanied by vestibular fissures.

4. Tertiary syphilis, developing in one-third of untreated patients, presents as nasal gumma, smooth circumscribed swellings covered by inflamed mucosa, with a predilection for the septum. Septal perforations causing "saddle deformity" may occur.

5. Penicillin is the drug of choice.

G. Senile Keratosis

1. Precancerous skin lesion frequently caused by sun exposure.

2. Raised, yellow brown to black pigmented lesion covered with crusts, overlying smooth, vascular surface.

3. Treatment is removal or destruction.

H. Rhinophyma

1. Disease of the skin of the nose characterized by chronic inflammation and hypertrophy of the skin, producing a large, red, violaceous nasal tip and comedones.

2. Typically affects Caucasian males between ages 40 and 60 years and involves the lower one-half of the nose.

3. Histology shows sebaceous gland hypertrophy, scarring, acanthosis, and frequently telangiectasia. These features also are seen in acne rosacea. Excessive intake of alcohol is not a direct cause.

4. Treatment is strictly surgical, via full thickness excision or decortication. Skin grafting is rarely necessary.

I. Dermatofibrosarcoma Protuberans

1. Involves the dermal layer of skin, presenting as a multinodular, firm, painless mass. Has potential of local growth and rare metastases.

2. Composed of small, uniform, fibrocystic-appearing cells arranged in a cartwheel pattern.

3. Treatment is wide excision.

J. Relapsing Polychondritis

1. Rare connective tissue disorder characterized by episodic recurring inflammation of cartilaginous structures.

2. Cartilage of ear, joints, and nose most commonly affected, producing pain and swelling. May produce saddle nose.
3. Nonspecific laboratory findings of anemia and elevated ESR.
4. Diagnosis based on history and biopsy.
5. Treatment is symptomatic, with anti-inflammatory agents (salicylates and corticosteroids) and occasionally cytotoxic drugs.

Nasal Pathology - Traumatic

A. Nasal Fractures

1. Most commonly fractures bone in body.
2. Symptoms include nasal deformity, swelling, pain, tenderness, mobility of nasal bones, and nasal obstruction.
3. Treatment includes repair of nasal lacerations, examination for septal hematomas with drainage if found, and elevation of nasal bones under satisfactory local anesthesia. Intranasal packing usually is not necessary. External protection with splint.

B. Septal Deviation

1. Deviations and dislocations of the septum may be classified in two groups: traumatic and developmental.
2. Septal deformities after trauma are separated in three groups.
 - a. Lateral type: Results from lateral fracture of the nose with displacement of the septum from the vomerian groove and maxillary crest.
 - b. Depressed type: Following a crushing frontal fracture and resulting in bizarre septal configurations of buckling, twisting, reduplication, and fibrosis.
 - c. Laterofrontal type: Combines the above two.
3. In the developmental type of septal deformity, the cartilaginous septum is dislocated and deflected and there is similar deviation of the underlying bony septum.
4. Major symptom is airway obstruction and may contribute to rhinitis.
5. Correction is surgical.
6. Correction of deviated septum is frequently necessary to correct external nasal deformities.

C. Septal Hematoma

1. Collection of blood beneath the mucoperichondrium and mucoperiosteum of the septum.
2. Cause is usually traumatic.
3. Symptoms include nasal obstruction. Palpation will reveal fluctuation of septum.
4. If untreated, may result in septal abscess, cartilaginous necrosis, and saddle nose.
5. Treatment includes incision and drainage, nasal packing to prevent reaccumulation, and antibiotics if infected.

D. Septal perforation

1. Caused by trauma both iatrogenic (septal surgery) and self-induced (nasal picking), septal abscess, granulomatous disease, i.e. Wegener's granulomatosis, and drug induced, i.e. cocaine.
2. Symptoms include crusting, epistaxis, discharge, and whistling on inspiration.
3. Treatment includes local measures (saline douches, application of ointments to perforation edges to diminish crusting), surgical closure (frequently fails), and insertion of septal buttons.

E. Foreign Bodies

1. Usually introduced voluntarily by patient, most often in children.
2. Symptoms include unilateral nasal obstruction and persistent unilateral purulent discharge.
3. Rhinoliths or nasal concretions usually have a foreign body nucleus. They may be bacteria, blood, crusts, or self-introduced foreign body, and are composed largely of calcium and magnesium salts. They have rarely been reported bilaterally.
4. Treatment of foreign bodies and rhinoliths is removal. This may require general anesthesia.

F. CSF Rhinorrhea

1. Flow of cerebrospinal fluid from the nose. May originate from the cribriform plate, frontal, sphenoid, or ethmoid sinuses, or middle ear via the eustachian tube.
2. Trauma is the most common cause, i.e. motor vehicle accidents. Can occur postoperatively (i.e. hypophysectomies), secondary to tumors (i.e. chromophobe adenoma), or may be idiopathic.

3. Diagnosis is based on history (clear, watery discharge, intermittent or constant, which increases with change in head position) as well as radiologic methods (polytomography). Localization of leaks is accomplished by intrathecal injections of various stains (usually fluorescein) followed by examination of appropriately placed cotton pledgets (middle meatus, cribriform plate, sphenoid recess).

4. Test for glucose in secretions is unreliable, since substantial amounts of glucose can occur both in nasal discharge and tears.

5. Treatment may be conservative initially; prophylactic antibiotics are probably of little value. Surgical approach to closure usually is via external ethmoidectomy. Various flaps or muscle and fascia plugs are used for closure.

G. Epistaxis

1. Etiology

a. Local disorders including traumas, crusting and ulceration, infection, and foreign body reactions.

b. Neoplastic disorders including malignant neoplasms, and juvenile nasopharyngeal angiofibromas.

c. Systemic disorders: Hypertension.

d. Hematologic disorders: Leukemia, anemia, purpura, polycythemia vera, hemophilia and other coagulopathies, lymphoma, anticoagulation (including ASA).

e. Other disorders: Osler-Wever-Rendu disease (familial hemorrhagic telangiectasia), hepatic disease, chronic nephritis, sudden pressure changes.

f. Ninety percent of epistaxis comes from Kiesselbach's plexus on the nasal septum. Posterior inferior bleeding usually comes from the sphenopalatine vessels. Superior bleeding usually comes from the anterior and posterior ethmoidal arteries.

2. Medical management

a. Locate bleeding site.

b. Remove clots from nose.

c. Shrinkage (decongestants, cocaine).

d. Adequate anesthesia important: cocaine, lidocaine (Xylocaine), tetracaine (Pontocaine).

e. Pressure while medications are working.

f. Cautery: Either chemical (silver nitrate) or electrical.

g. Carefully placed petroleum gauze pack will stop most bleeding. May need to place bilaterally for adequate measure.

h. Severe posterior bleeding may require posterior pack, either conventional or Foley-type. (Beware of pressure necrosis to the nasal vestibule.)

i. Begin prophylactic antibiotics when due to blockage of sinus ostia.

j. Posterior packs may produce hypoxia. Check blood gases, and provide supplemental O₂ as indicated.

k. Beware of excessive sedation.

l. Transfuse as necessary.

m. Workup and treat causes of bleeding (include sinus x-rays).

3. Surgical management

a. For uncontrolled or recurrent epistaxis.

b. Ligation of anterior and posterior ethmoidal arteries. For superior epistaxis, this is done through an external ethmoidectomy approach.

c. Ligation of branches of the internal maxillary artery. Through a Caldwell-Luc approach the posterior antral wall is exposed. This is removed under microscopic visualization to expose the pterygomaxillary fossa. Self-locking neurosurgical clips are placed on the branches of the internal maxillary artery as close to their entrance to the nose as possible.

d. Ligation of the external carotid artery: Easy to approach, but less successful because of its distal relationship to the actual bleeding vessels.

e. If vessel ligation fails to control epistaxis, angiography with Gelfoam embolization of the offending vessel may be of some aid.

f. Septal dermaplasty should be considered in patients with recurrent epistaxis from familial hemorrhagic telangiectasia.

g. Injection of the pterygomaxillary fossa via the greater palatine foramen may temporarily control posterior epistaxis.

Pathology - Congenital

- A. Cysts: See Benign Tumors Nose and Sinuses.
- B. Glioma: See Benign Tumors Nose and Sinuses.
- C. Encephalocele: See Benign Tumors Nose and Sinuses.
- D. Teratomas: See Benign Tumors Nose and Sinuses.
- E. Choanal Atresia

1. A result of persistence of the nasobuccal membrane.
2. Eighty to ninety percent are bony or membrano-osseous, the remainder being membranous. Familial tendency.
3. One-third are bilateral, two-thirds are unilateral.
4. When bilateral the condition causes airway obstruction in neonates, who are obligate nasal breathers for up to several weeks. The apneic state is broken if the infant cries. It can cause death if not recognized.
5. Unilateral atresia causes persistent nasal drainage and excoriation of the nasal vestibule.
6. Diagnosis is made by passing a flexible catheter through the anterior nares. It should pass more than 32 mm.
7. Bilateral atresia is treated initially with McGovern's nipple.
8. Surgical correction is accomplished by a transnasal, transseptal, or transpalatal approach.

Pathology - Rhinitis

Rhinitis is hyperfunction of the nose due to various stimuli, producing rhinorrhea and nasal obstruction. Classification as to etiology difficult.

A. Vasomotor Rhinitis

1. Nonspecific diagnosis of unexplained hypersensitivity rhinitis.
2. Increase in acetylcholine in nasal mucosa (possibly due to deficient acetylcholinesterase) characterized by increased parasympathetic tone.
3. Nasal smear may show mastocytosis, with sparsity of eosinophils.
4. Allergic workup negative.
5. Treatment.

- a. Medical: Steroids, topical, submucosal, or systemic.
- b. Surgical: Directed to inferior turbinate or vidian nerve.

B. Allergic Rhinitis

1. IgE-mediated response causing release of vasoactive substances from mast cells.
2. Nasal smear shows eosinophilia.
3. Other allergic manifestations usually present.
4. Treatment.
 - a. Avoidance.
 - b. Medications: Include antihistamines, topical steroids (beclomethasone, flunisolide), decongestants.
 - c. Desensitization: About 70% effective.

C. Atrophic Rhinitis

1. Etiology unknown. Theories include hereditary, developmental, and endocrine factors, bacterial infections, and nutritional deficiencies. Can be iatrogenic (i.e. excessive turbinate resection).
2. Symptoms include nasal obstruction, epistaxis, anosmia, and offensive odor.
3. Examination reveals excessive crusting and turbinate atrophy.
4. No cure available. Nasal irrigation helpful. Surgical approaches are designed to narrow internal dimensions of nose.

D. Rhinitis Medicamentosa

1. Caused by excessive use of topical nasal decongestants, both sympathetic amines and the imidazoles.
2. Treatment includes avoidance of topical deongestants, along with the use of systemic decongestants and topical steroids.

E. Metabolic Rhinitis

1. Seen with hypothyroidism.

F. Hormonal Rhinitis

1. Frequent side effect of pregnancy. Increased estrogen levels inhibit acetylcholinesterase thus increasing parasympathetic tone.
2. Also seen at puberty and with the use of oral contraceptives.

G. Infectious Rhinitis

1. Both viral ("common cold") and bacterial (purulent).

Necrotizing Lesions of the Nose

These lesions are evidence of an underlying systemic disease. Identification of causative organism, appearance of characteristic morphology, and presentation of a careful history will clarify pathogenesis. Final diagnosis rests with biopsy.

A. Vasculitis

1. Wegener's granulomatosis.
 - a. Necrotizing granulomas with vasculitis involving the respiratory tract and kidney (necrotizing glomerulitis).
 - b. Nasal obstruction, followed by mucosal destruction, crusting, and septal perforation.
 - c. Systemic symptoms (also night sweats, malaise, arthralgias).
 - d. Biopsy of nose reveals inflammation and necrosis.
 - e. Treated with cytotoxic agents and steroids.
2. Systemic lupus erythematosus (see Pathology - External Nose).
 - a. Ulcerations of nasal mucosa may occur during severe exacerbations of SLE.
3. Scleroderma
 - a. Chronic disease of unknown cause characterized by diffuse sclerosis of the connective tissue of the integument and other organs.
 - b. Obliterative vascular lesions may result in ulceration of nasal mucous membranes.
4. Sjögren-Sicca Syndrome
 - a. Syndrome complex including keratoconjunctivitis, xerostomia, enlargement of the lacrimal and salivary glands, and rheumatoid arthritis.

b. Nasal mucosa may be dry, atrophic, and ulcerated.

B. Infectious

1. Tuberculosis.

a. Uncommon in the nasal cavity.

b. Symptoms include discharge, crusting, pain, and nasal obstruction.

c. May involve the anterior septum or inferior turbinate with gross appearance ranging from superficial ulceration to a papillomatous mass. Septal perforations may occur.

d. Diagnosis is made by a positive smear, culture, and biopsy.

e. Treatment is the same as for pulmonary tuberculosis.

2. Syphilis: see Nasal Pathology - External Nose.

3. Leprosy.

a. Chronic granulomatous infection which attacks superficial tissues, especially skin, peripheral nerves, and nasal mucosa.

b. Caused by Mycobacterium leprae (Hansen's bacillus).

c. Nasal symptoms include epistaxis and obstruction.

d. Necrosis of nasal structures results in septal perforation and saddle nose.

4. Glanders.

a. Infection primarily of equine animals which can be transmitted to man, due to Pseudomonas mallei.

b. Produces extensive ulcerating granulomatous lesions of the nasal mucosa causing mucopurulent discharge.

5. Rhinoscleroma.

a. A granulomatous disease caused by Klebsiella rhinoscleromatis.

b. Foul-smelling rhinorrhea followed by atrophy and crusting, and finally granulomatous nodules causing fibrosis and stenosis.

c. Culture from infected tissue is diagnostic.

d. "Mickulicz cell" (foamy histiocyte) characteristic.

- e. Tetracycline is the drug of choice in treating disease.
- 6. Rhinosporidiosis.
 - a. Caused by Rhinosporidium seeberi, a funguslike organism.
 - b. Presents with exophytic, branching, granular, deep red, pedunculated or sessile polypoid growths in the nose.
 - c. Symptoms include nasal obstruction and epistaxis.
 - d. Treatment is by surgical removal or lesions followed by dapsone.
- 7. Mucormycosis: see Fungal Sinusitis.
- 8. Aspergillosis: see Fungal Sinusitis.
- 9. Candidiasis: see Fungal Sinusitis.
- 10. Actinomycosis.
 - a. Caused by Actinomyces israeli, and A. bovis, a bacterial form.
 - b. Can cause a purulent abscess of the nose and antrum.
 - c. Distinguishing feature is the presence of sulfur granules.
 - d. Treatment is with penicillin.
- 11. Histoplasmosis.
 - a. A granulomatous fungal disease caused by Histoplasma capsulatum.
 - b. Usually involves lungs, larynx, and tongue.
 - c. Nodules and ulcers composed of masses of organisms are found.
 - d. Epithelioid or histiocytic granuloma is seen with organisms within the granuloma.
 - e. Treatment is with amphotericin B.
- 12. Cryptococcosis.
 - a. Caused by the fungus Cryptococcus neoformans.
 - b. Usually affects the pulmonary system, central nervous system, or general body, but may be localized to the nose or sinuses.

- c. Produces granulomatous lesions typically lacking a prominent inflammatory reaction.
- d. Treatment is with amphotericin B.

13. Blastomycosis.

a. Chronic granulomatous and suppurative disease, involving the respiratory tract and spreads to other sites, including the face, external nose, and nasal cavity.

b. Caused by Blastomyces dermatitidis.

c. Produces ulcerated or verrucous granuloma of the nasal mucosa with a serpiginous advancing, elevated border.

d. Treatment is with amphotericin B.

C. Neoplastic Disease

1. Histiocytosis.

a. Includes three separate entities: (1) Eosinophilic granuloma, (2) Hand-Schüller-Christian disease, and (3) Letterer-Siwe disease.

b. Development of infiltration of differentiated histiocytes, accompanied by a variable mixture of eosinophils, giant cells, neutrophils, foamy cells, and areas of fibrosis.

c. Etiology of disease is unknown.

d. May present with nasal mass - diagnosis by biopsy.

e. Treatment includes surgery, radiotherapy, steroids, and cytotoxic therapy.

2. Lymphoma.

a. Lymphocytic lymphoma most frequently is encountered in the sinonasal tract.

b. Symptoms suggest an inflammatory disease which initiates a more disfiguring and destructive phase.

c. Diagnosis is based on biopsy.

d. Treatment is with radiotherapy and chemotherapy.

3. Mycosis fungoides.

a. Uncommon neoplastic disease of the lymphoreticular system first manifested in the skin.

- b. May cause shallow ulcerations of the nasal mucosa, although tumors may develop.
- c. Diagnosis is made on biopsy.

D. Treated with topical and systemic chemotherapy, topical and local steroids, and radiation.

4. Lymphoepithelioma.

- a. More frequently found in the nasopharynx.
- b. Diagnosis is by biopsy.
- c. Highly radiosensitive.

5. Malignant melanoma.

- a. Poorest prognosis of all primary neoplasms of the sinonasal tract.
- b. Patients complain of nasal obstruction, epistaxis, and occasionally black discharge.
- c. Pigmented necrotic hemorrhagic mass causing obvious destruction is seen within the nose.
- d. Surgery remains the most effective treatment.

D. Unknown Etiology

1. Sarcoidosis.

- a. Systemic disease, usually with abnormal chest roentgenograms.
- b. May involve the external nose (papular lesion), septum, or turbinates with classic granulomas. Patient may complain of nasal obstruction, plus clear watery rhinorrhea.
- c. Biopsy reveals noncaseating epithelioid granulomas in the lymphoreticular tissue.
- d. May require systemic steroids and surgery.

2. Necrotizing sialometaplasia.

- a. Benign lesion that represents nonspecific reaction of salivary and mucous glands to ischemic injury.
- b. May produce a deep excavating crater in the nose, causing severe epistaxis.
- c. Treatment is with surgical excision.

3. Pyogenic granuloma.

a. Localized, specific, often ulcerated polypoid lesion composed of newly formed capillaries in an edematous matrix.

b. Present as bright red to reddish brown vascular tumors which tend to form crusts and undergo necrosis.

c. May spontaneously involute, or may need excision.

4. Idiopathic midline granuloma.

a. Destructive localized or diffuse lesions, with characteristic extension through the palate and facial soft tissues.

b. Localized to airway and upper aerodigestive tract.

c. May remain histologically benign or evolve into polymorphic reticulosis.

d. Inflammatory reaction is nonspecific. Granulomas and giant cells are infrequent.

e. Impairment of delayed hypersensitivity often is seen.

f. Treatment is irradiation.

5. Polymorphic reticulosis.

a. Unlike idiopathic midline granuloma, systemic complaints are often out of proportion to nasal symptoms.

b. Destruction of facial soft tissues similar to idiopathic midline granulomas.

c. No vasculitis or glomerulonephritis. May not have airway lesions.

d. May involve into lymphoma (extranodal or disseminated).

e. Characteristic atypical and polymorphic lymphoreticular cellular infiltrate, angiocentric growth patterns. May simulate vasculitis, but fibrinoid necrosis absent in vessel walls.

f. Treatment is radiation.

Disturbance in Olfaction

A. Hyperosmia

1. Oversensitive sense of smell.
2. Occurs with hunger, cystic fibrosis, and Addison's disease.

B. Parosmia

1. Perverted sense of smell.
2. May occur with injury to uncus of temporal lobe.
3. Occurs secondary to some drugs (i.e. streptomycin).
4. May occur in postinfluenzal patients.

5. Must be differentiated from olfactory hallucinations as seen in schizophrenia, and from uncinata seizures secondary to lesions of the uncinata gyrus, amygdaloid nucleus, or hippocampus.

C. Hyposmia

1. Impaired sense of smell.
2. Seen with increasing age, postmenopausal women, and in tobacco smokers.

D. Anosmia: Loss of sense of smell.

1. Intranasal conditions that block the flow of air to the olfactory mucosa.
 - a. Seen in rhinitises, sinusitis, polyps, intranasal or nasopharyngeal tumors, choanal atresia, and vitamin A deficiency.
2. Trauma
 - a. Contrecoup frontal damage from occipital head injury.
 - b. Fracture of the cribriform plate (LeFort's II or III, frontoethmoidal fractures).
 - c. Injury to olfactory nerves or bulb.
 - d. One-third of patients with traumatic anosmia.
3. Infection.
 - a. Usually postinfluenza.

b. Vitamin A has been advocated as treatment, though proof of efficacy is lacking.

4. Tumor.

a. Sphenoidal ridge meningiomas produce ipsilateral anosmia, unilateral optic atrophy or papilledema, and exophthalmos.

b. Olfactory groove or cribriform plate meningiomas present with unilateral anosmia, retrobulbar neuritis, or optic atrophy.

c. Frontal lobe gliomas present with unilateral anosmia and optic atrophy.

d. Foster-Kennedy syndrome, produced by frontal lobe tumors, consists of ipsilateral anosmia, and optic atrophy, with contralateral papilledema.

e. Parasellar or pituitary tumors may present with bilateral anosmia.

5. Congenital.

a. May be complete, or selective.

b. May occur sporadically, or may be either autosomal or sex-linked.

c. Complete anosmia is seen in familial dysautonomia and chromatin-negative gonadal dysgenesis.

6. Hysterical anosmia

a. Hysterical anosmiacs will deny smelling ammonia which is sensed by the trigeminal nerve, not the olfactory nerve.

Benign Tumors of the Nose and Paranasal Sinuses

A. Nasal Polyp

1. Most common tumor of the nasal cavity. Equal incidence in sexes and socioeconomic groups.

2. Multiple causes exist.

a. Twenty-five percent of an allergic group had polyps.

b. Fifty-four percent of patients with polyps had allergies.

c. Also seen with infection, trauma, metabolic disease, cystic fibrosis, aspirin intolerance, asthma.

3. Nasal obstruction, rhinorrhea, and anosmia are usual symptoms.

4. Translucent, pear-shaped polyps (single or multiple) seen unilaterally or bilaterally. Unilateral "polyps" may represent choanal polyp, encephalocele, inverted papilloma, angiofibroma, carcinoma, or sarcoma.

5. Increased eosinophilia commonly are seen on nasal smear.

6. Medical therapy includes diet, desensitization, decongestants, antihistamines, antibiotics (when indicated), and steroids.

7. Surgical therapy includes polypectomy, with or without ethmoidectomy, and Caldwell-Luc operation if indicated.

B. Inverted Papilloma

1. Usually unilateral, male preponderance.

2. May resemble nasal polyps but far less translucent.

3. Thickened surface epithelium with invagination into supporting stroma.

4. About 10% develop squamous cell carcinoma.

5. Major symptoms include nasal obstruction, sensation of mass in nose, epistaxis, and rhinorrhea.

6. Complete excision is important because of frequent recurrence.

a. Calcaterra suggests lateral rhinotomy, medial maxillectomy, and ethmoidectomy.

C. Squamous Papilloma

1. Similar to squamous papilloma of other areas of the skin (commonly called a wart).

2. Usually found in the area of the ala or nasal vestibule.

3. If further posterior in nasal passage may be more mucoid, softer, and vascular.

4. May involve the sinuses.

5. May cause nasal obstruction or hemorrhage.

6. Usually require surgical excision.

D. Nasal Glioma

1. Herniation of brain tissue through the floor of the anterior cranial fossa. If it communicates with ventricular system it is an encephalocele.

2. Clinically, smooth mass which may be polypoid and swell with crying.
3. Beware of removal without neurosurgical evaluation and therapy.

E. Neural Tumors

1. Uncommon in nose.
2. Neurofibroma and neurilemoma are most often seen.

F. Hemangioma

1. Capillary hemangioma: Loosely arranged tissue containing thin-walled blood vessels.

- a. Occurs most frequently on the nasal septum.
- b. Treated by excision and electrocautery.

2. Cavernous hemangioma: Thin-walled growth with afferent and efferent vessels.

- a. Usually seen in lateral wall of the nose.
- b. Infiltrates, destroys tissue and can cause severe hemorrhage.
- c. May require surgery, cauterization, freezing, and occasionally requires ligation of the external carotid artery.

G. Osteoma

1. Most commonly found at frontoethmoid suture.
2. May occur in frontal, ethmoid, or maxillary sinuses, or from nasal septum or turbinate.
3. Slow growing and produce symptoms (usually headache in frontal sinus) when large.
4. May extend into orbit or cranial cavity.
5. Diagnosed radiographically.
6. Surgical removal is symptomatic or expanding.

H. Cysts

1. Radicular cyst, seen in maxilla, develops from apical abscess of an erupted tooth.
2. Globulomaxillary cyst results from entrapment of epithelium at the junction of the premaxilla and maxillary process. May distort teeth.
3. Nasoalveolar cyst: A developmental cyst located in the soft tissue at the junction of the premaxilla and alveolar process.
4. Rathke's pouch tumor: Cyst lined with squamous cells, filled with keratin, and located in the nasopharynx.
 - a. Due to failure of Rathke's pouch to obliterate.
 - b. Pouch formed by invagination of oral ectoderm in formation of anterior of the pituitary gland.
- I. Dermoid cyst: Usually present at birth and seen at suture line between nasal bones and nasal process frontal bones.

1. May extend into nasal cavity, frontal, ethmoid, or maxillary sinuses, or intracranially.

J. Thornwald's cyst: Derived from pharyngeal notochord remnant and found in the nasopharynx, in the midline superior to adenoidal pad.

1. Can become infected and cause drainage and discomfort.
2. Requires surgical excision or marsupialization.

K. Chordoma: Arises from remnant of notochord in nasopharynx.

1. Causes nasal obstruction or cranial nerve involvement.
2. Histologically benign but cervical metastases have been reported.
3. Surgical excision is therapy but complete excision is difficult.

L. Plasmacytoma (extramedullary): May be isolated or part of multiple myeloma.

1. Most found in nose, nasopharynx, and sinuses.
2. Therapy and prognosis uncertain.

M. Teratoma: Derived from all germ layers and often associated with other cranial abnormalities. Seen in midline.

N. Fibrous Dysplasia

1. Mixture of fibrous tissue and cancellous bone seen in maxilla.
2. Slow growing tumor causes cosmetic defect.
3. Requires surgical excision and may undergo malignant degeneration.

Anatomy of the Paranasal Sinuses

a. Maxillary Sinuses (Antrum of Highmore)

1. The largest of the paranasal sinuses is located in the body of the maxilla and has a volume of 15 mL.
2. It is pyramidal cavity with its apex extending laterally and its base directly toward the nasal cavity.
3. The floor of the antrum, formed by the alveolar process of the maxilla, is usually 3-5 mm below the level of the floor of the nasal cavity, except in children where the floor is at or above the level of the floor of the nasal cavity.
4. The thin medial wall is the lateral wall of the nasal cavity. It is through this wall that the sinus drains into the nose, into the middle meatus behind a projection made by the uncinat process of the ethmoid. Often an accessory opening is seen posterior to the primary opening (30-40%). These openings are above the floor of the sinus and require active transportation of mucus and purulent matter to empty the sinus. Improved drainage is gained by creating a "nasoantral window" (nasal antrostomy) from the inferior meatus to the lower portion of the sinus.
5. The roof is formed by the orbital surface of the maxilla and also serves as the floor of the orbit. The infraorbital nerve is contained in a ridge located in the center of the roof.
6. The cheek overlies its anterior wall. The surgical approach to the sinus is through the canine fossa over the bicuspid and molars and through the anterior sinus wall. (Caldwell-Luc operation)
7. The posterior wall overlies the pterygoid space. Opening this wall allows access to the internal maxillary artery and the vidian nerve as well as tumors which might spread from the nasopharynx to this area (juvenile angiofibroma, rhabdomyosarcoma).
8. The sinus is best appreciated roentgenologically in a Waters' view. Serial Waters' views can be used to follow the progress of acute sinusitis. Polytomography and computerized axial tomography (CAT scan) better define spread in lesions and bony destruction.

B. Ethmoid Sinuses

1. Located between the middle turbinate and the medial orbital wall, the thin walled ethmoid sinuses number 7-15 per side and have a volume of 14 mL.

a. The anterior ethmoid sinuses (usually small and numerous) are found below the attachment of the middle turbinate, drain into the middle meatus, and form the bulla ethmoidalis in the nose.

b. The posterior ethmoid sinuses (larger and fewer) are superior and posterior to the attachment of the middle turbinate, and drain into the superior meatus.

c. The ethmoid cells may invade any of the surrounding bones, including frontal, sphenoid, and maxillary bones. The anterior cells extend into the agger nasi and uncinat process.

2. The middle turbinate is an osseous shelf approximately 3.5-4 cm in length and is sometimes pneumatized with an ethmoidal cell (4-12%). Its anterior attachment is to the cribriform plate. This plate measures only 2 cm long by 5 mm wide and is 2 mm thick. These small dimensions leave little room for error while performing intranasal surgery. Any instrumentation medial to the attachment of the medial turbinate should be done with extreme caution.

3. The lateral wall of the ethmoid sinuses (the medial wall of the orbit) is known as the lamina papyracea (paper-thin plate). Indeed, so thin is the bone that occasionally natural dehiscences occur in its surfaces, permitting the development of orbital cellulitis from an ethmoiditis.

a. Where the lamina papyracea articulates with the frontal bone lie the anterior and posterior ethmoidal foramina, each containing an artery and neural twig. These arteries supply blood to the superior aspect of the nose and occasionally are ligated for severe epistaxis. The optic nerve lies 3-8 mm deeper than the posterior artery.

b. The anterior and posterior foramina form a line parallel to, and just inferior to, the anterior cranial fossa (cribriform plate) and serves as a valuable landmark.

4. The posterior ethmoid cells are never far from the optic nerve, the closeness being dependent on the pneumatization of the sphenoid bone. The sphenoid sinus may surround the nerve or if the sinus does not pneumatize, the posterior ethmoidal cells may invade the bone and come into intimate relationship in the nerve. This relationship makes external ethmoid surgery safer than intranasal or transantral ethmoid surgery especially where there is poor access to the sinus.

5. The ethmoid sinuses are best seen on Caldwell's (AP) and lateral roentgenograms. CAT scans have greatly improved our ability to evaluate these sinuses.

C. Frontal Sinuses

1. The frontal sinuses are located in the frontal bone above and deep to the superior orbital ridge. The two sinuses, usually unequal in size, form an irregular pyramid with the apex directed upward. The volume is usually 6-7 mL. About 15% of adult skulls have only one sinus and 5% have none.

2. The sinuses are separated by a thin septum of bone which is not always present and only occasionally in the midline. The depth of the two sinuses often vary causing confusion on AP roentgenograms. Lateral films are necessary for full appreciation of frontal sinus opacification.

3. The sinus drains into the nose through the nasofrontal duct located on the anteromedial aspect of the sinus floor. It runs through the ethmoidal labyrinth and enters the ethmoidal groove at the anterior end of the middle meatus. Drainage also may occur into the frontal recess anterior to the infundibulum (55%), above the infundibulum (30%), into the infundibulum (15%), and above the bulla (1%).

4. For acute infections not responding to medical therapy a frontal trephination through the floor of the sinus might be necessary. A drain for irrigation usually is left in place.

5. Chronic frontal sinusitis usually is treated by an osteoplastic flap procedure with fat obliteration of the sinus cavity. This gives good exposure of the sinus and is not deforming, as were many of the earlier procedures.

D. Sphenoid Sinuses

1. The sphenoidal sinuses are located in the sphenoid bone behind the upper part of the nasal cavity. They vary greatly in size and shape probably because they represent an ingrowth from the nose. The usual volume is 7.5 mL.

a. They are commonly very deep in their anteroposterior dimension.

b. Laterally they may invade the greater and lesser wings, pterygoid process, and lateral pterygoid plates of the sphenoid.

c. The sinuses usually are separated by a septum which usually deviates to one side but is occasionally absent.

2. The openings of the sinuses are medial and superior in the rostrum of the sphenoid and the sinuses drain into the superior meatus through the sphenoid recess.

3. The cavernous sinus and optic nerve are located laterally to the sphenoid sinuses. The carotid artery and vidian nerves commonly cause a ridge on the lateral wall if the sinus pneumatically expands laterally for any distance.

4. The pituitary gland is located posteriorly and superiorly and commonly bulges into the superior wall. This relationship enables one to perform a transsphenoid hypophysectomy.

5. Respect to the lateral and superior walls must be shown. The inferior wall is the safest.

6. Three types of pneumatization are recognized:

a. Postphenoid pneumatization (about 60%): Pneumatization extends posteriorly below the pituitary fossa so that the sella turcica projects its anterior wall into the sinus.

b. Presphenoid pneumatization (about 40%): The sinus is pneumatized as far as the anterior bony wall of the pituitary fossa.

c. Conchal pneumatization (about 1%): The sinus is rudimentary, having little depth. A contraindication to transsphenoid hypophysectomy.

7. The type of pneumatization is well shown on the lateral roentgenographic view of the sinuses. The sinus also is seen on the basal view.

Diseases of the Sinus

A. Acute Sinusitis

1. Etiology: Usually follows an upper respiratory infection, but may follow trauma, excessive drying, edema (allergic), or nasal obstruction. Gram-positive organisms (usually pneumococci, Haemophilus influenzae, group A beta streptococci, or Staphylococcus aureus.)

2. Symptoms: Heaviness in the sinus, pain, nasal obstruction, and blowing of a malodorous yellow or green mucopurulent discharge.

3. Findings: Pus in the nose, edema and redness of nasal mucosa, tender over sinuses, opaque sinus, sometimes with an air-fluid level.

4. Therapy: Antibiotics (usually ampicillin/amoxicillin), nose drops, (oral decongestants), and analgesics. Occasionally need to drain frontal sinus if therapy does not work.

B. Chronic Sinusitis

1. Etiology: May follow poorly treated acute sinusitis, may be associated with allergy.

2. Symptoms: Chronic purulent discharge and nasal obstruction may be the only symptoms. Pain and pressure on occasion.

3. Findings: This purulent discharge and nasal obstruction. Occasional polypoid changes.

4. Therapy: May respond to antibiotics, but also may require surgical intervention:

a. Maxillary sinus: Repeated antral lavage with saline might cure. Otherwise, Caldwell-Luc and nasal antrostomy.

b. Ethmoid sinus: External ethmoidectomy is the safest approach. Intranasal ethmoid surgery usually reserved for polyps.

c. Frontal sinus: Osteoplastic flap procedure with obliteration of the sinus cavity with abdominal fat is the operation of choice. Cannulization of the nasofrontal duct should be avoided.

C. Fungal Sinusitis (rare)

1. Mucormycosis (rhinocerebral phycomycosis): A severe acute illness in debilitated persons (50% with untreated diabetes; also immunosuppressive agents and prolonged corticosteroid usage). Spreads rapidly to involve the eye and central nervous system. Black crusting in the nose, with perforated septum, cranial nerves III, IV, VI involvement and loss of vision. X-ray: Nodular thickening of soft tissues lining sinuses. Hyphae seen on biopsy; high mortality. Therapy is prolonged use of amphotericin B.

2. Aspergillosis: May follow other infection or prolonged antibiotics. Similar signs and symptoms of chronic sinusitis. May require Caldwell-Luc. Excellent prognosis.

3. Candidiasis: Similar to above. Use nystatin irrigation.

Complications of Sinus Disease

A. Orbital Pain

1. Common in acute infections of all sinuses.

a. Pain from sphenoid is usually retro-orbital.

2. Less common with chronic infections or tumors, but when disease spreads to orbit, pain is increased.

B. Lid Swelling

1. Seen with acute maxillary, frontal, and ethmoid sinusitis.

2. Location of sinus determines which lid is swollen.

C. Exophthalmos

1. Not usually of sinus origin.
2. Present if sinus infections spread into the orbital spaces or by extension of tumor from sinus into orbit.

D. Orbital Cellulitis

1. Etiology: Usually from ethmoid sinusitis, but may come from any of the sinuses.
2. Symptoms:
 - a. Lid edema, followed by exophthalmos, chemosis, and progressive immobility of eye.
 - b. Patient is usually very ill with a high fever and severe pain in the eye.
 - c. Roentgenograms identify the origin of infection.
3. Therapy:
 - a. Vigorous intravenous antibiotic therapy.
 - b. Exploration of orbit with I and D and usually external ethmoidectomy.
 - c. Drain abscess for at least 4 days.

E. Mass in the Orbit

1. Location of mass identifies sinus of origin.
 - a. Supraorbital: Frontal sinus.
 - b. Medial: Ethmoid sinus.
 - c. Infraorbital: Maxillary sinus.
2. Most common lesions producing masses are carcinomas, mucocele, and osteomas.

F. Retrobulbar Neuritis

1. Etiology.
 - a. Fifteen percent of the cases are caused by sinus disease by direct extension or phlebitis.
 - b. Similar symptoms also can be from tumors of the sinuses and pituitary.

2. Symptoms: Loss of vision, sudden or gradual.
3. Therapy: Antibiotics and sinus surgery.

G. Superior Orbital Fissure Syndrome

1. Etiology: Acute or chronic infection, cyst, or neoplasm extending through the lateral wall of sphenoid sinus.

2. Symptoms: Involvement of contents of superior orbital fissure:
 - a. First cranial nerve VI, followed by III, IV, and V (with pain in the eye).
 - b. Later exophthalmos and ophthalmoplegia.
3. Diagnosis: Polytomography and CT scans important in establishing diagnosis.
4. Therapy: Immediate exploration of the sphenoid.

H. Cavernous Sinus Thrombosis (see intracranial complications).

Osteomyelitis

A. Frontal Bone

1. Etiology: Most patients under 30.
 - a. Hematogenous spread in children. Trauma during acute frontal sinusitis in adults.
 - b. Females more common than males.
 - c. May follow swimming, acute exacerbation, chronic sinusitis, or frontal sinus surgery.
 - d. Staphylococcus aureus most common.
2. Symptoms:
 - a. Acute course: Headache, edema upper eyelids, doughy swelling of skin over frontal sinus (Pott's puffy tumour), pericranial abscess, may spread intracranially.
 - b. Chronic course (no perforation inner table): Insidious onset, low-grade fever, local pain, doughy swelling forehead, malaise, and chills. May form fistula.
3. Diagnosis: Physical findings plus x-rays which may not show positive changes (necrosis) for 7-10 days after onset of swelling.

4. Therapy:

- a. Early onset, high-dose intravenous antibiotics (based on culture).
- b. Drainage of localized abscess.
- c. Trephine for inspection and culture.
- d. Local heat, nasal spray.
- e. When in doubt as to involvement of the posterior wall or continuous sinusitis, osteoplastic frontal adipose obliteration surgery.

B. Superior Maxilla

1. Etiology:

- a. Usually of dental origin.
- b. In infants may be from buccal infection.
- c. Sinusitis may produce osteitis and fistulous tract with extension to:
 - 1) Facial surface with abscess formation.
 - 2) Palatine and alveolae process with fistula to roof of mouth.
 - 3) Zygomatic process with extension to pterygoid process.

2. Symptoms:

- a. Signs and symptoms of sinusitis, plus:
- b. Swelling and chemosis cheek.
- c. Exophthalmos and limitation of eye movement.

3. Therapy:

- a. Specific high-dose antibiotics.
- b. Surgical drainage.
- c. Heat.

C. Sphenoid bone (rare)

1. Etiology:

a. Associated with osteomyelitis at base of skull or infection of the petrous portion of the temporal bone.

b. Hemolytic streptococcus and Staphylococcus aureus.

2. Symptoms:

a. Profuse postnasal drainage and deep seated headache.

b. May spread laterally to cause superior orbital fissure syndrome.

c. Later spread can cause cavernous sinus thrombosis, brain abscess, encephalitis, and intracranial hemorrhage.

3. Diagnosis:

a. Frequently not diagnosed until complications develop.

b. Polytomography and CT scan help.

4. Therapy:

a. Specific high-dose antibiotics.

b. Surgical drainage.

Frontal Sinus Pneumocele

1. A collection of air under pressure in the tissues which air escapes through a defect in the bony wall of the sinus.

2. May follow trauma (fracture, operation), congenital cleft, dehiscence, or necrosis (secondary to syphilis, osteomyelitis, sinusitis).

3. May be internal (posterior to sinus), external (forehead), or pneumosinus dilatans (excessive dilatation of sinus) which is associated with acromegaly, localized osteitis, or fracture in the region of the sinus.

Intracranial Complications of Sinus Disease

A. Etiology

1. Pathways for intracranial involvement:
 - a. Trauma.
 - b. Through congenital dehiscences.
 - c. Direct pathway through sinus wall.
 - d. Along sheath of olfactory nerve.
 - e. By way of the communicating veins.
 - f. By means of septic thrombi along diploic veins.
 - g. By way of ethmoid or angular veins to cavernous sinus.
 - h. By way of the orbit.

B. Meningitis

1. Most common intracranial complication (70%).
2. Sphenoid sinus most common site of origin (followed by ethmoid, frontal, and maxillary sinus).
3. May follow nasal or sinus surgery.
4. Organisms include hemolytic streptococcus, pneumococcus, and H. influenzae.
5. Therapy includes antibiotics and drainage of infected sinuses when defined.

C. Epidural Abscess

1. Purulent collection between internal surface of the cranium and the dura.
2. Usually from frontal sinus, with usual sinusitis organisms.
3. CT scan helpful in making diagnosis.
4. Similar therapy to that for osteomyelitis.

D. Intracranial Mucocele

1. If expansion of a mucous retention cyst from the sphenoid or frontal sinus.
2. If infected it is a pyocele with the same organisms as osteomyelitis.
3. May be seen on polytomograms.
4. Therapy requires surgery on the sinus.

E. Subdural Abscess (Empyema)

1. Purulence between dura and pia mater.
2. Usually from the frontal sinus, secondary to thrombophlebitis.
3. Intensive headaches and rapid change in consciousness.
4. Therapy requires drainage of subdural space and elimination of sinus infection.

F. Cavernous Sinus Thrombosis

1. Cavernous sinus is reached through nonvalved veins from sphenoid and ethmoid sinuses most frequently.
2. Exophthalmos, chemosis, and eyelid edema followed by ocular muscle paralysis as well as systemic signs.
3. Coagulase-positive S. aureus is usual cause.
4. Therapy is with antibiotics and possibly anticoagulants.

G. Brain Abscess

1. High mortality despite antibiotic therapy.
2. Frontal lobe abscess, usually from frontal sinus but occasionally from sphenoid or maxillary.
3. Temporal lobe abscess, usually otogenic in origin but may result from maxillary or sphenoid sinusitis.
4. Staphylococcal and streptococcal infections are the usual cause.
5. Develops slowly with change in temperament.
6. Shown well by CT scan.

7. Requires neurosurgical removal.