Paparella III: Section 2: Disorders of the Head and Neck

Part 1: Nose and Paranasal Sinuses

Chapter 4: Infectious Diseases of the Paranasal Sinuses

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Acute sinusitis occurs secondary to the extension of nasal and dental infections into the pathogen-free milieu of the paranasal sinuses. Inflammation secondary to nasal infection will often narrow or obstruct the natural sinus ostium, resulting in fluid stagnation and bacterial growth. Predisposing local factors include the common cold, mucosal hypertrophy from allergic or vasomotor rhinitis, septal deviation, polyps, obstructing adenoidal tissue and, less commonly, tumors, foreign bodies, or unilateral choanal atresia. Sinus infections of dental origin are due to periapical abscess, granuloma, or oroantral fistula following extraction. These infections arise in the floor of the maxillary sinus and at times may expand to include the ethmoidal and other sinuses. Generalized conditions associated with sinusitis include hypogammaglobulinemia, neutropenia, and immobile cilia syndrome.

Protection Against Infection

The nose has many features that aid in the prevention of upper and lower respiratory tract infection. The nares are guarded by vibrissae that trap large particles. The mucosal surface is designed to protect the airway and is composed of a respiratory ciliated mucous membrane covered by a sticky mucoid layer that is secreted by the goblet cells and the mucoid glands of the nose. Particles trapped here are carried posteriorly at a rate of approximately 6 to 7 mm/minute to be swallowed and destroyed in the gastrointestinal tract. The flow rate is adversely affected by low relative humidity, habitual smoking, and air pollution. Particles that penetrate to the mucosal layer are phagocytosed. The nose is an excellent trap; air entering it is passed as 1-mm sheets over a surface that is variegated and constantly changing because of the nasal cycle, thereby increasing the rate of particle capture. The airway makes a 90-degree bend at the nasopharynx, trapping more particles. Particles 5 to 10 microns are most efficiently trapped in the nose; those 2 to 4 microns may be carried through on air currents to the lung. Frequently, inspirated bacterial particles fit into the latter size range.

The highly vascular mucosal surface of the nose will respond abruptly to nasal irritants with increased tissue engorgement and nasal secretion (producing a flushing effect). Nasal secretions contain lysozymes, which are mucolytic enzymes that can cause swelling and lysis of some microorganisms. The importance of this system in controlling the common nasal pathogens is unclear. Nasal secretions also contain antibodies. Local immunity in the nose primarily results from concentrations of IgA and IgG, which are normally present in a ratio of 3:1 in nasal secretions, compared with concentrations of 1:5 in serum. The principal immunoglobulin in nasal secretions is IgA; however, the highest circulating antibody against respiratory viruses is IgG. IgA is formed locally as a 7S monomer in the plasma cells lining the respiratory tract and gut;

it is combined with a second IgA molecule and a small connecting protein, the secretory piece, to form an 11S dimer as it passes through the epithelium into the secretions. Most IgA is produced in response to local antigen stimulation. IgA antibody does not combine with complement and therefore is unable to lyse bacteria; it is, however, effective as a viral neutralizing substance. Failure to produce secretory IgA occurs in approximately 1 in 600 persons, most of whom live without ill effects. In contrast, patients with generalized hypogammaglobulinemia have frequent bacterial infections of the upper respiratory tract.

No single virus can be held responsible for the majority of common colds. Rhinovirus is responsible for about one third of cases; however, parainfluenza types 1 to 4 and influenza A and B are each responsible for 15 to 20 per cent of cases. Respiratory syncytial virus, enterovirus, adenovirus, and a group of other viruses are each responsible for 5 per cent or less of cases. These viruses are spread by droplet dissemination or by fomite transfer from the hands to the nose. There is no apparent relationship between susceptibility to colds and wet feet, dry air, or abrupt changes in temperature; colds are probably more common during the winter months because of increased crowding indoors.

During the first 24 hours of a viral cold, there is a significant increase in the IgA level in the nasal mucus; this is the result of a release of stored specific and non-specific IgA into nasal secretions. The degree of response depends on the type of infecting virus. During the secretory phase of viral rhinitis (days 2 to 5), there is necrosis and shedding of epithelial cells accompanied by marked transudation of serum albumin and IgA. At this time, virus particles stimulate the local synthesis of antigen-specific IgA and IgG, and as a result, a second rise in the IgA titer will be seen at approximately 1 week, peaking at 2 weeks. IgA does not speed clinical recovery, but renewed local specific IgA antibody stores provide protection against reinfection.

Hypogammaglobulinemia presents in the first year of life with recurrent respiratory infections and affects boys principally, as the disorder is X-linked. Many patients with congenital hypogammaglobulinemia survive to middle age and beyond and may be seen occasionally in adult practice. Transitory and acquired immunodeficiencies also occur. The initial workup would include quantitative immunoglobulin levels, B- and T-cell function testing, determination of complement levels, and analysis of phagocytic function. Sinusitis occurs intermittently among patients with acquired immune deficiency syndrome (AIDS). This disorder is caused by a profound defect in cell-mediated immunity following a selective infection of the helper (T4) subset of T-lymphocytes by the human immunodeficiency virus (HIV; HTLV III (human T-cell lymphoma virus)), which essentially aborts the initiation of the immune response.

The mucosa of the paranasal sinuses consists of a single layer of pseudostratified ciliated columnar epithelium; it has a few goblet cells and is thinner and smoother than nasal mucosa. The tunica propria is fine and fuses imperceptibly with the underlying periosteum. The cilia beat in a rapid, coordinated fashion toward the natural ostium and are necessary for the removal of the mucous blanket produced by the goblet cells and mucous glands.

Rarely, *immotile cilia syndrome* can be a cause of chronic sinusitis and must be kept in mind for puzzling cases, especially among children and young adults. A triad of otitis media, recurrent pneumonia or bronchiectasis, or both, and sinusitis will usually be manifested in childhood and may or may not be associated with situs inversus (50 per cent of cases) and, in later life, with male infertility. Diagnosis is made by electron microscopic examination of nasal and bronchial biopsies for aberrant cilia, especially a lack of outer dynein arms. There may be a family history for recurrent respiratory infection or even Kartagener's syndrome.

The differentiation of sinusitis into the categories of acute, subacute, and chronic is best done based on the underlying changes in the sinus mucosa. The acute infections are associated with the onset of a purulent air-fluid level or sinus opacification. The condition is considered subacute if the infection fails to resolve within a month and the mucosa has become increasingly thickened by the inflammatory process. Once these pathologic changes become irreversible, the patient is considered to have chronic sinus disease. Chronic sinusitis takes two forms: (1) hypertrophic sinusitis and (2) atrophic or sclerosing sinusitis with areas of squamous metaplasia. Both conditions are associated with thickened secretions, reduced blood flow, and low oxygen tension and pH, thus providing the atmosphere for anaerobic bacterial growth.

The normal bacterial flora of the anterior part of the nose and nasopharynx frequently include *Staphylococcus, Streptococcus pneumoniae, Haemophilus influenzae,* and beta-hemolytic streptococci. The presence of lack of rhinitis has little effect on the incidence of these bacteria. Cultures of the nose and nasopharynx must be interpreted with these facts in mind.

Acute Furunculosis

Acute furunculosis is secondary to *Staphylococcus aureus* infection involving the hair follicles or sebaceous glands. It must be regarded as a serious infection because occasionally it can be complicated by cavernous sinus thrombosis through venous spread. Treatment consists of surgical drainage of the abscess and the initiation of potassium penicillin G. If penicillinase-producing staphylococci are likely to be present, dicloxacillin should be used orally. For patients who have had an allergic response to penicillin, clindamycin or erythromycin may be used. The frequency with which *S. aureus* occurs in the nose makes isolation of asymptomatic carriers impractical even in a hospital, except in nurseries and in patients who are nearly immunologically suppressed.

Nasopharyngitis

Nasopharyngitis is a difficult disorder to define. The nasopharynx is host to many pathogenic organisms (*S. aureus* and *Streptococcus viridans, S. pyogenes*, and *S. pneumoniae*) even in seemingly healthy individuals. The diagnosis of nasopharyngitis is made on the basis of pain, redness, swelling, and discharge in this region. The cause and effect relationship to rhinitis, sinusitis, and otitis is complex. Treatment requires appropriate antibiotic therapy.

Sinusitis

Acute Frontal Sinusitis

The acute form of frontal sinusitis presents with pain over the frontal sinus that is increased by tapping or bending forward. The sinus will fail to transilluminate, will have a fluid level, and will be opaque on radiographs. During the summer months, the condition may result from jumping or diving while swimming, thus forcing water into the sinus. All forms of sinusitis frequently follow viral upper respiratory tract infections and are particularly common in patients with septal deviations and nasal polyps.

If acute frontal sinusitis goes untreated, the patient may present with fever, swelling, and redness over the sinus associated with edema of the upper eyelid and diffuse headache. Pus may not be present in the nose, as the nasofrontal duct may be blocked by inflammation. Because of the proximity of the frontal sinus to the dura, meningitis and epidural, subdural, or brain abscesses are possible complications owing to extension of bacteria intracranially through phlebitic diploic veins. If allowed to persist, frontal sinusitis may result in osteomyelitis of the frontal bone. This condition causes a tender, doughy edema over the affected frontal sinus that has been named Pott's puffy tumor.

Acute frontal sinusitis frequently requires hospitalization for proper treatment. With few exceptions, these infections are caused by bacteria residing in the nose, primarily *S. pneumoniae* and *H. influenzae*. Intravenous antibiotics should be administered, along with topical nasal decongestant or 4 per cent cocaine packings, three times a day to induce drainage. The choice of antibiotic should be based on the presumed appropriate bacterial coverage and good central nervous system penetration. Older intravenous drug combinations, such as penicillin and chloramphenicol, are being replaced by second- and third-generation cephalosporins, such as cefuroxime or ceftriaxone. The cephalosporins are probably safe in patients who have had a rash-type allergic reaction to penicillin, but they should be avoided in patients who have had anaphylactic reactions to penicillin. If the sinus fails to drain and the patient's condition has not improved in 24 hours, surgical drainage followed by saline irrigation of the sinus should be undertaken.

Chronic Frontal Sinusitis

The chronic type of frontal sinusitis occurs in two basic forms. The first is a low-grade frontal sinusitis with thickened sinus lining and retained secretions; this is characterized by mild tenderness and chronic headache with associated intermittent drainage into the nose. Radiographs demonstrate an opacified sinus with sclerosis of the surrounding bone. These infections usually follow an acute sinusitis, and they result from an inadequately functioning nasofrontal duct system caused by allergic or hyperplastic mucosa, scarring, or traumatic ductal injury. The consequence is stasis of mucus, lowered partial pressure of oxygen, and lowered pH. Because of this milieu, most cultures are mixed, and the most prevalent pathogens are anaerobes that respond to high-dose penicillin therapy or certain of the cephalosporins, such as cefoxitin. For penicillin-

allergic individuals, clindamycin or chloramphenicol are choices, in combination with tobramycin. *H. influenzae* is the most important aerobe.

A *mucocele,* or *mucopyocele* when the mucocele becomes infected, is the second common type of frontal sinusitis. It is formed by an encysted portion of the respiratory lining of the frontal sinus that continues to secrete mucus, thereby gradually expanding within the sinus. These lesions are often painless but become evident as they gradually expand and erode the walls of the sinus; usually they break through the superior or medial wall of the orbit, resulting in swelling of the upper eyelid and at times exophthalmos and diplopia. They can erode through to the dura or down into the ethmoidal sinus. Radiographs may be deceiving because bony resorption gives the radiographic appearance of decreased density of the involved frontal sinus; however, the bony margins of the sinus have a characteristic appearance of having been erased.

Osteoplastic frontal fat obliteration of the sinus is the treatment of choice. Bacterial studies should include routine culture, anaerobic culture, and smear. Penicillin G or cefoxitin are good choices for postoperative antibiotic coverage until the cultures are returned.

Acute and Chronic Maxillary Sinusitis

Acute maxillary sinusitis may follow viral respiratory infections. It tends to occur if predisposing local factors are present, such as a deviated nasal septum, a scarred stenotic sinus ostium, or nasal polyps. Again, *H. influenzae, S. pneumoniae, Branhamella catarrhalis,* and *S. aureus* are the bacteria most frequently cultured. Anaerobes are also commonly cultured from a chronically infected sinus.

Acute maxillary sinusitis responds well to oral antibiotic management with commonly used and effective drugs such as amoxicillin, clavulanic acid (Augmentin), or cefaclor. For penicillin-allergic patients, erythromycin and trimethoprim and sulfamethoxazole together make a good combination. Doxycycline has the disadvantage that many strains of *S. aureus* and *H. influenzae* are resistant to it. Patients should be instructed in the use of vasoconstricting sprays followed by head-positioning maneuvers and moist air inhalations (breathing through a hot towel) during the acute phase in order to promote drainage of the affected sinus. Progress can be monitored by transillumination and a single upright Waters view x-ray film, on which evidence of a residual fluid level and mucosal thickening should be sought. An antral wash should not be attempted in untreated acute sinusitis but should be used to flush out retained secretions that fail to clear from the sinus after a week or more of antibiotic therapy. The antrum behaves like a poorly drained abscess in which infection and inflammation resolve slowly and tend to recur if not adequately treated. Therefore, antibiotic therapy for 3 weeks or more is often required to effect complete resolution of the condition.

A unilaterally opacified maxillary antrum on sinus x-ray film should be regarded as a potential sinus tumor until proved otherwise. Another principal cause of unilateral maxillary sinusitis is an apical root abscess draining into the sinus. The evaluation should include sinus x-ray films or computed tomography (CT) scan, if necessary, and dental bite wing views that

include the roots, or Panorex should be performed. If the diagnosis remains in doubt, the sinus should be explored. Four-smelling pus usually indicates a dental infection.

Osteomyelitis of the maxilla is unusual but rarely may result in fistula formation to the cheek, palate, or pterygoid fossa. Fungal infections are occasionally found.

Acute and Chronic Ethmoiditis

Acute viral ethmoiditis is commonly associated with viral rhinitis. Secondary bacterial infection can be recognized by a change from mucoid to mucopurulent nasal drainage. Bacterial infections generally respond quickly to antibiotics that are effective against gram-positive organisms. Because of the early development of the ethmoidal sinuses, ethmoidal sinusitis is the form of sinusitis most frequently seen among pediatric patients.

Chronic ethmoiditis is often seen in patients with allergic or hyperplastic sinusitis. Among the sinuses, the ethmoids are involved early, with polypoid mucosal changes and polyps that may become secondarily infected. Because of mucous stasis and poor vascularity of polypoid tissue, infection is often difficult to treat in this situation. Ethmoidal surgery may be required to control chronic infection.

Acute and Chronic Sphenoidal Sinusitis

Sphenoidal sinusitis occurs alone only occasionally; more often it is seen in pansinusitis. Isolated bacterial or, rarely, fungal infections of the sphenoid bone occur in debilitated elderly persons. Mucoceles of the sphenoid bone can occur. Physical findings are scant. Patients complain of a deep headache behind the eyes with pain referred to the vertex of the skull. Diagnosis requires a high index of suspicion - indeed, the condition often goes unrecognized. Sinus films demonstrate thickened mucosa and a fluid level. Chronically infected sinuses at times will demonstrate bony sclerosis on radiographs.

Uncomplicated acute sphenoidal sinusitis usually responds briskly to appropriate antibiotic treatment. In patients who fail to improve, sphenoidal puncture and irrigation might be tried by surgeons who have experience with this procedure. More commonly, surgical drainage of the sinus is accomplished by resection of the anterior sphenoidal wall by means of an external ethmoidectomy or transseptal approach. Surgical drainage is mandatory for survival if intracranial complications appear.

Complications of sphenoidal sinusitis often represent life-threatening intracranial disease. Among them are osteomyelitis of the sphenoid bone, cavernous sinus thrombosis, panhypopituitarism, and blindness.

Complications of Sinus Infection

Sinusitis is a relatively common disorder, yet major local, orbital, and central nervous system complications are relatively uncommon in this medical age. Undoubtedly this is because of the emergence and employment of effective antibiotic treatment. However, the use of antibiotics, although diminishing complications, has also in some cases masked their presentation, making recognition a matter of alert clinical judgment.

Local Complications

The most common complication of acute sinusitis is the development of *chronic mucosal inflammation* and thickening. This condition can result in partial or complete obstruction of the sinus ostia with intermittent recurrent infection often caused by anaerobic bacteria. The patient complains of a constant dull ache in the region of the sinus. There is intermittent thick yellow-green drainage.

Chronic maxillary sinusitis is treated with long courses of antibiotics and local intranasal measures to promote drainage by way of the natural bony passages. For patients who fail to respond to these measures, a Caldwell-Luc procedure with creation of a large nasoantral window to provide proper drainage is used. In a few patients, some pain persists despite this surgery. Chronic frontal sinusitis is best treated by osteoplastic frontal exposure with fat obliteration.

A *mucocele* or *mucopyocele* arises most commonly in the frontal sinus, less commonly in the ethmoidal sinus, and rarely in the sphenoidal sinus. These benign cysts gradually fill and at times expand beyond the sinus by pressure and bony resorption. A mucocele of the frontal sinus can present in the superior medial aspect of the orbit as a painless soft mass that may displace the eye inferiorly and laterally. Also, the posterior sinus wall may be eroded, posing a hazard for central nervous system infection. The diagnosis can be made by radiographic studies. Treatment for a frontal mucocele consists of complete surgical removal. Again, osteoplastic frontal exposure with fat obliteration is most effective.

Mucoceles of the ethmoidal and sphenoidal sinuses are most likely to expand into the orbit or, occasionally, centrally. Orbital examination may demonstrate unilateral exophthalmos, diplopia, and signs of optic nerve pressure. Polytomography, CT scans, and orbital ultrasonographic studies are extremely useful methods of analyzing these problem cases. Arteriograms are rarely necessary. Thyroid ophthalmopathy is the most common cause of unilateral exophthalmos. However, when exophthalmos is caused by a mucocele, the mucocele should be surgically removed by external ethmoidectomy.

Osteomyelitis secondary to sinusitis is quite unusual. It occurs most commonly following trauma, radiation therapy, or debilitating disease. In the maxillary sinus, osteomyelitis can occur subsequent to a dental root abscess or dental extraction. Rarely, fistulization occurs and may involve the palate, cheek, or pterygomaxillary space.

The frontal sinus is the most common site of this type of osteomyelitis, which occurs secondary to periostitis and causes edema over the sinus (Pott's puffy tumor). There is a red, tender swelling of the forehead skin with associated fever. Pre- and postoperative antibiotics and surgical drainage (trephination) followed by surgical excision of sequestrated bone and fat obliteration of the sinus compose the usual treatment. Further reconstruction often is not necessary.

Orbital Complications

A *pneumocele* of the orbit may result from a small bony defect between the orbit and maxillary or ethmoidal sinuses following forceful blowing of the nose. The patient should be started on appropriate antibiotics and observed for signs of orbital cellulitis.

Orbital cellulitis is a frequent complication of acute ethmoiditis in children (less so in adults) secondary to spread of infection either directly through the lamina papyracea or via phlebitic veins. The former results in a subperiosteal inflammation and the latter results in a rapidly progressing cellulitis of the periorbital fat and soft tissues. Both conditions are characterized by considerable lid swelling, chemosis, and proptosis. Pain is variable but may be severe. Frequently there is mild to markedly restricted eye motion. In uncomplicated cases, vision remains good and pupillary reflexes are normal; however, these two parameters must be carefully followed in order to ensure the safety of the eye. Treatment at this point should consist of intensive antibiotic therapy.

Surgical drainage should be considered early if the cellulitis fails to respond rapidly to antibiotics. The surgeon should not wait for the formation of an obvious abscess but should be guided by increasing inflammation, particularly if there is onset of sluggish light and accommodation pupillary reflexes (an early indication of optic nerve swelling) and impending loss of vision.

Drainage is accomplished by making a 2- to 3-cm curved incision at the upper medial aspect of the orbit, carrying it medially along the periosteum. A collection of pus may be found within the orbit or subperiosteally. The wound is drained and left to granulate, with little permanent scarring. Alternately, drainage is performed through an external ethmoidectomy approach.

Dacryocystitis is another complication of acute bacterial rhinosinusitis. It is manifested by localized, painful, red swelling below the medial canthus over the lacrimal sac. Gentle pressure over the sac will at times express pus from the punctum. This complication occurs more often in elderly patients and generally responds well to antibiotics. Surgical drainage is required only occasionally.

The *superior orbital fissure syndrome* is a rare complication of sphenoidal sinusitis. It occurs when the sphenoidal sinus infection affects the structures of the superior orbital fissure by direct extension through a thin bony plate or by sphenoidal osteomyelitis in this region. The

symptoms consist of deep orbital and unilateral frontal headache pain in association with progressive sixth, third, and fourth cranial nerve palsies, usually in that order.

In the past, chronic sinusitis was thought to play an important role in the development of optic neuritis. However, a causal relationship between these two entities has not been established except in rare cases.

Central Nervous System Complications

Meningitis may develop secondary to infection in the frontal, sphenoidal, or ethmoidal sinuses and is characterized by headache, meningismus, an unexplained high temperature, and a change in mental alertness. Previous antibiotic treatment for sinusitis complicates recognition of the disease; therefore, a spinal tap should be done when this disorder is suspected, glucose and protein concentrations should be determined, and a complete cell count should be performed. The cerebrospinal fluid should be Gram-stained for bacteria and cultured; fungal and tuberculosis cultures should be included if there is reason to suspect their presence. The most common bacteria found in sinusitis related to meningitis are *Streptococcus pyogenes, S. pneumoniae, Staphylococcus aureus,* and *H. influenzae.* Treatment consists of immediate initiation of intensive antibiotic therapy for 2 weeks or longer in addition to thorough surgical drainage of the involved sinus.

Localized empyema should be suspected in all cases of meningitis in patients with sinusitis. Subdural abscesses are associated with the general symptoms of meningitis, whereas an epidural abscess is more difficult to diagnose. In this disorder, there may be no signs of meningeal irritation. Patients complain of headache and fever and may become obtunded. The spinal tap, however, will show only a few cells, probably lymphocytes. Electroencephalograms (EEGs), CT scans, and magnetic resonance imaging (MRI) are important tools in establishing this diagnosis.

A *brain abscess* in the frontal area can be large and still fail to present localizing neurologic signs. The spinal tap may or may not show white blood cells, depending on whether there is a communication with the subarachnoid space. One clue is a high cerebrospinal fluid protein concentration. EEGs will be abnormal, demonstrating slowing over the area of the abscess. Again, CT scans and MRI have greatly improved the recognition of this disorder.

Posterior ethmoiditis and sphenoidal sinusitis may be responsible for *cavernous sinus thrombosis.* This infection is characterized by high spiking fevers in a patient with high toxicity. There is a rapid onset of oculomotor involvement, including almost simultaneous involvement of the third, fourth, and sixth cranial nerves, resulting in a painful panophthalmoplegia, or fixed eye. Pupillary responses are usually lacking, and a large pupil is common. Optic nerve involvement is manifested by congestion of the optic disc, field cuts, or complete loss of vision. In a responsive patient, sensation involving the first division of the fifth nerve may be diminished or lacking. Continued extension of the thrombosis to the remaining cavernous sinus or other venous sinuses is a grave prognostic sign. Treatment consists of intensive antibiotic therapy,

drainage of the contiguous infected ethmoidal and sphenoidal sinuses, and anticoagulation.

Nosocomial Sinus Infections

Nosocomial sinus infection in patients with chronic polypoid rhinosinusitis is often not recognized as such. Initially, symptoms may be relatively minor in patients who have been released from the hospital; however, the combination of hospital-acquired bacteria and the altered immunity of a postoperative diseased nose and sinuses can result in a persistent, troublesome infection, especially if colonized with *Pseudomonas* or a penicillin-resistant strain of *S. aureus.* This is a particularly important consideration for patients with cystic fibrosis. Therefore, whenever possible, surgery for polypoid hyperplastic sinus disease should be done as outpatient surgery. Broad-based prophylactic antibiotic coverage, for example, with cefazolin, should be begun preoperatively and continued postoperatively until all packing has been removed. It is unusual that all packing cannot be removed in 24 to 48 hours from surgery. Long-term packing when antibiotics are being taken invites *Pseudomonas* or fungal infection.

Sinus infections in the critically ill patient or in the patient in the intensive care unit (ICU) are precipitated by foreign objects placed through the nose, namely, nasogastric tubes, feeding tubes, nasotracheal tubes, nasopharyngeal airway tubes, and packing. *Pseudomonas aeruginosa, Klebsiella pneumoniae, Enterobacter, Proteus mirabilis, Escherichia coli, S. aureus,* beta-hemolytic streptococci, and *Bacteroides* are among the bacteria found, often as multibacterial infections. Treatment consists of removal of nasal tubing, administration of intravenous antibiotics, and at times, especially in life-threatening situations, sinus drainage. It is likely that sinusitis among patients in the ICU occurs more commonly than recognized and is the result of a broader spectrum of hospital-acquired bacteria.

Fungal Sinus Infections

Nonopportunistic Infections

Fungal infections of the paranasal sinuses in the immunocompetent patient are being recognized with increasing frequency. Infections are of two very different orders of magnitude based on whether they are noninvasive or invasive in character, that is, if fungal forms are found within tissue. *Aspergillus fumigatus* is the most common causative agent. However, mucormycosis has been reported as well. Immunocompetent patients, who have recurring chronic and subacute maxillary sinusitis and have failed repeated antibiotic treatment may exhibit a noninvasive fungal sinusitis. In this instance, the fungus lives saprophytically as a small mycetoma on the mucosa of the sinus floor, inciting recurrent bacterial infection. Sometimes on x-ray film there is the appearance of a calcified foreign body in the sinus. The treatment is the removal of the fungus and improved sinus ventilation. This disease often begins as a dental infection or follows an oroantral fistula.

Allergic *Aspergillus* sinusitis has been described recently and is believed to be the paranasal sinus equivalent of allergic bronchopulmonary aspergillosis. The disease, which often

affects young adults, is characterized by recurrent polypoid rhinosinusitis, a history of asthma, and pansinusitis documented by x-ray studies. The diagnosis is made histologically by examination of mucinous material for eosinophils, septate hyphae, and Charcot-Leyden crystals, and by immunologic testing for an IgG-mediated positive skin test or by antigen-specific serum IgE elevations. There is no tissue invasion by the fungi. Treatment consists of surgical extirpation and aeration and long-term oral steroid therapy. The prevalence of this disorder is not yet known among allergic polypoid rhinosinusitis patients.

Opportunistic Infections

Invasive fungal infections of the head and neck, in general, occur under special circumstances, namely, diabetic ketoacidosis, immune alteration secondary to antibiotic and steroid therapy, and profound granulocytopenia. In this era, many patients who are at risk are undergoing treatment with cancer chemotherapy or, in some centers, undergoing immunosuppression for organ transplantation, especially for bone marrow transplantation.

The earliest clinical presentation of a fungal sinusitis is an unexplained fever, a slight cloudy rhinorrhea, and facial tenderness. Sinus x-ray studies are misleading, as the patient's impaired inflammatory response does not produce signs of sinusitis on x-ray films until the disease is advanced. On nasal examination, gray nonsensate areas may represent early tissue invasion and infarction. Friable, bloodless biopsy specimens should cause concern and must be processed immediately for light microscopy and culture. Surgical excision should be performed as quickly as possible, as these infections advance rapidly. Black necrotic tissue is a sign of advanced fungal infection. Nonseptate hyphae found within tissue are evidence of mucormycosis, and septate hyphae with branching at 45 degrees are typical of aspergillosis. Other opportunistic infections include those from *Candida*, herpes simplex, and *Pseudomonas*. Patients with a low granulocyte count (less than 1000 cu mm) can have a progression of disease despite treatment with amphotericin B. The best hope for survival is an improved granulocyte count.

Mucormycosis

Mucormycosis is a fulminant opportunistic infection usually caused by *Rhizopus oryzae*, although at times it is caused by other members of the order Mucorales. The infection, accompanied by fever and increased obtundation, usually arises in the nose and ethmoidal sinuses; however, it can arise in the lungs or bowel also. If uncontrolled, it is fatal in a period of days to weeks.

The patient complains to the otolaryngologist of headache, nasal blockage, and serosanguinous nasal discharge. The infection spreads by way of the blood vessels, invading and penetrating the walls and causing thrombosis and necrosis; these conditions can progress rapidly to the orbit, resulting in panophthalmoplegia and proptosis, or they may extend intracranially, in which case they are followed by seizures, coma, and death.

The prognosis is grave. Diagnosis by biopsy demonstrates nonseptate, branching hyphae. Amphotericin B should be initiated as soon as possibly intravenously and at times intrathecally. Thorough debridement of infected tissues should be carried out promptly if the disease is recognized in time.

Aspergillosis

Although aspergillosis occurs most commonly as a chronic pulmonary disease, it may also be a chronic inflammatory granulomatous infection of the middle ear, external auditory canal, nose, and paranasal sinuses. It has a characteristic green-brown discharge. The fungus may be part of the normal oropharyngeal flora. However, in debilitated or immunosuppressed patients, acute aspergillosis may become a very aggressive nasal and sinus infection. This results in a relentlessly progressive vasculitis and thrombosis resembling mucormycosis. Extension from the nose and paranasal sinuses can quickly involve the orbit and central nervous system.

Proper diagnosis is made by biopsy, culture, and examination of nasal secretions for mycelial forms. In the chronic form, aspergillosis is not life-threatening and should be treated by debridement and local therapy. In acute life-threatening disease, prompt debridement is required. Systemic amphotericin B therapy is occasionally effective.

Granulomas of the Upper Respiratory Tract

The list of potential causes of nasal granulomatous disorders is long. It includes syphilis, tuberculosis, sarcoidosis, leprosy, brucellosis, rhinoscleroma, yaws, leishmaniasis, rhinosporidiosis, glands, and anthrax. Granuloma can also result from actinomycosis, blastomycosis, moniliasis, aspergillosis, histoplasmosis, coccidioidomycosis, and torulosis. Only some of these disorders will be discussed here.

Because of the relatively infrequent appearance of granulomatous lesions in North America, it is necessary for the physician to keep their many possible causes in mind when evaluating such lesions. Most causes can be eliminated by the history and geographic factors. Evaluation should include a thorough head and neck examination, searching for associated mucosal lesions in the nasopharynx or larynx, lymphadenopathy, and cutaneous signs. Chest and sinus x-ray studies should be obtained, as should a biopsy of the lesions and cultures of drainage material and the tissue obtained by biopsy. Special bacterial and fungal smears and cultures should be ordered as indicated by the history. A change in a purified protein derivative (PPD) skin test for tuberculosis is sometimes a helpful clue, and serologic tests for increased antibody titers are required for histoplasmosis and coccidioidomycosis. Reagin tests and fluorescent treponemal antibody absorption (FTA-ABS) titers help in the diagnosis of syphilis and yaws.

Tuberculosis

The incidence of tuberculosis has fallen dramatically in the USA and with it the incidence of associated ear, nose, and throat findings. However, although ear, nose, and throat

manifestations of tuberculosis are uncommon, they are not rare.

Approximately 2 per cent of patients with active pulmonary tuberculosis show evidence of upper respiratory tract involvement, usually of the larynx; however, the nasopharynx and nose can also be involved. The condition can present in the nose as a beefy, red edematous area with associated exudate and ulceration or as a granulomatous growth or polyp, most commonly on the septum or anterior inferior turbinate. When an abnormal chest x-ray film is lacking, nasal tuberculosis is very unlikely; however, further evaluation would include smears and cultures of sputum and nasal drainage followed by biopsy for evidence of a tuberculous granulomatous reaction and acid-fast bacilli. Currently, an 18- to 24-month course of isoniazid (INH) and ethambutol is a common choice of therapy. INH-resistant disease should be considered if the patient has had previous treatment or acquired the infection in an area of known high INH resistance, such as Asia, Africa, or South America. The incidence of tuberculosis in nontreated contacts is 1 to 2 per cent. Improvement of the nasal lesions coincides with that of the pulmonary disease.

Sarcoidosis

Sarcoidosis is a disease of worldwide distribution; however, increased incidence rates have been noted in certain rural regions and in populations such as American blacks and Scandinavians. The disorder affects young adults of both sexes about equally. The cause is unknown. It is a generalized disease and, as such, presents in a variety of ways. Patients often complain of malaise, low-grade fever, and weight loss; on chest films there may be pulmonary involvement, with hilar adenopathy and peripheral lymphadenopathy as early signs. However, the disease is characterized by disseminated granulomatous lesions that can involve any tissue in the body; in the later, more progressive stage of the disease, there are increased pulmonary, liver, spleen, skin, eye, and renal symptoms, among other findings.

These patients may present to the otolaryngologist with complaints of nasal congestion or obstruction and occasionally recurrent rhinitis. The nose is engorged and slightly inflamed, and 1- to 3-mm nodular lesions may be seen at times on the septum or inferior turbinates, particularly after the use of a vasoconstricting spray. A biopsy of these lesions demonstrates noncaseating granulomas that are characteristic but not diagnostic of sarcoidosis. At times, nasal symptoms may precede the recognition of generalized symptoms. In the end stages, atrophic changes such as erosion and crusting of the mucous membrane and fibrosis occur.

Further evaluation and management of this disease required consultation with an internist and an ophthalmologist. Additional testing might include delayed hypersensitivity skin testing with *Candida*, streptokinase-spreptodornase, or PPD antigens to check for the anergic test response found in this disease. Also, serum immunoglobulins, particularly IgG, might be elevated. The Kveim test, which produces positive results 80 per cent of the time, is unavailable except in research centers.

Nasal symptoms associated with sarcoidosis generally respond to steroids but not to inhaled topical steroid preparations. Treatment should be instituted after consultation with the other physicians involved in the patient's care.

Syphilis

It is important to realize that nasal disease secondary to infection with *Treponema pallidum* can occur in every age group from the neonate to the elderly patient. The physician who practices in this era of liberal antibiotic usage must be aware of fleeting or atypical signs of this disease.

The lesion of primary syphilis can appear on the external nose or inside the vestibule. It presents as a hard, nonpainful, ulcerated papule that is often associated with an enlarged, rubbery nontender node approximately 3 weeks after exposure. Serum obtained from this lesion may be darkfield stain-positive for *T. pallidum;* however, serologic tests, both reagin and specific *Treponema* antibody tests, generally produce negative results until 8 to 12 weeks have elapsed.

The secondary stage of syphilis is the most infectious and is characterized by a macular or darkfield-positive maculopapular rash. Occasionally, manifestations include pale gray mucinous patches on the nasal mucous membrane; however, these highly infectious lesions are more commonly noted on the oral mucosa. During secondary syphilis, reagin tests such as the Venereal Disease Research Laboratory (VDRL) test produce uniformly positive results, and a specific treponemal antibody test, such as FTP-ABS test, is likely to be reactive.

Early congenital syphilis, the onset of which may occur from birth to 2 years of age, is a pediatric disorder of greater severity; it is roughly analogous to secondary acquired syphilis. It is characterized by marked rhinitis, variable rashes, anemia, and irritability. The rhinitis is nicknamed the "snuffles" because of nasal obstruction from mucosal inflammation and a mucopurulent discharge that is often bloody secondary to mucosal ulceration. If not recognized and treated, the rhinitis can lead to chondritis and osteitis and a nasal saddle deformity. Again, the lesions are highly infectious and darkfield-positive. Reagin test results are always positive. As a general rule, adequate, prompt treatment of these early forms of syphilis, as already discussed, will greatly reduce or eliminate positive reagion titers. Usually, FTA-ABS levels remain stable despite treatment, unless reinfection raises them further.

In rare instances, late syphilis can present in the nose as a gummatous reaction of the bony septum or palate as much as 30 to 40 years after the initial infection. These foul-smelling, draining granulomatous lesions may fistulize through the hard palate. Although the reagin test result may be negative in late syphilis, even without treatment over many years, gummatous lesions are usually associated with high reagin test titers as well as positive FTA-ABS test results. Rapid healing of the granulations after the institution of penicillin therapy serves to confirm the diagnosis.

Late congenital syphilis can also present with gummatous lesions of the bony septum or palate as well as of other bones, skin, or viscera from the age of approximately 8 years and up. Other stigmata of the disease include interstitial keratitis, sensorineural deafness, Hutchinson's incisiors (Hutchinson's triad), rhagades, mulberry molars, and saber shin. With treatment of these late forms of the disease, as discussed previously, reagin test titers decrease only slowly and FTA-ABS levels are unaffected.

One satisfactory treatment for adults with nasal syphilis is penicillin G benzathine (Bicillin), 2.4 million units. For patients with penicillin sensitivity, erythromycin, 250 mg four times a day for 2 weeks, is sufficient. Neurosyphilis may require more intensive treatment.

Wegener's Granulomatosis

Wegener's granulomatosis is an unusual but not rare disease that is potentially fatal and is commonly seen at about 40 years of age. It is rare among blacks. The disease is characterized by a focal necrotizing vasculitis that affects the upper and lower respiratory tracts, skin, joints, and kidneys. The cause is unknown, but it may represent an unusual form of autoimmune response. The disease involves adults primarily, but it is occasionally seen in children. Although the onset can be fulminant, involving multiple organ systems, more often it has a gradual beginning with the development of a granulomatous condition in the upper respiratory tract followed by bronchitis, pneumonia, and glomerulonephritis. Death from uremia may occur within 6 months if treatment fails.

The disease may present to the otolaryngologist as a painful ulcer in the nose or granular draining otitis media and hearing loss that is unrelentingly progressive and unresponsive to treatment. These lesions are often secondarily infected by *S. aureus* and *P. aeruginosa*. Lesions of Wegener's granulomatosis may occur anywhere in the respiratory mucosa. No single laboratory test is diagnostic, although an elevated erythrocyte sedimentation rate, hypergammaglobulinemia, and anergy are usually present. Histologic diagnosis is made by a nasal or lung biopsy that demonstrates granulomas with giant cells and widespread areas of vasculitis; these lesions result in focal ischemia or hemorrhagic necrosis, which resembles that of polyarteritis nodosa. Renal biopsy may demonstrate a glomerulitis and vasculitis with immune complex deposits in vascular walls. Treatment with steroids and radiation have failed to alter the course of the disease in the past. Cytotoxic drugs, such as cyclophosphamide, or alternatively azathioprine, have revolutionized therapy because they have the ability to produce clinical remission. These drugs reduce gammaglobulin production and produce a lymphocytopenia followed by a return of renal function and abatement of the respiratory system.

Midline Granuloma

Midline (lethal) granuloma is a rare disease. It is characterized by ulceration and induration in the tissues of the nose or nasopharynx, and extends into adjacent structures and results in the gradual destruction of the tissues of the midface. Initially, the disorder remains localized, but as it progresses, lesions are observed in the skin, lungs, and lymph nodes. Death

may occur in a matter of months or after many years and is usually secondary to meningitis, pneumonia, debilitation, or uncontrolled hemorrhage. This condition occurs in a younger age group than does Wegener's granulomatosis; men are more often affected than are women by a ratio of 3:1.

The patient will present with an ulceration, associated discharge, and pain. The diagnosis is made by biopsy. It is important to secure a deep biopsy from tissue other than the exposed ulcerated area. Biopsies should be repeated until the diagnosis can be established with assurance.

The histopathologic alterations include a dense polymorphic infiltration of the tissues by atypical lymphoid and reticulum cells with granulation tissue and neutrophils in the areas of active ulceration. In contrast to Wegener's granulomatosis, there is no giant cell reaction or vasculitis or renal involvement, but areas of focal necrosis occur secondary to thrombosis of small vessels.

When midline granuloma is localized, high doses of radiation (4000 to 6000 rad) may produce long resmissions or cures. Corticosteroids and cytotoxic agents are of no value, nor is surgical removal helpful; however, reconstructive surgery may begin, when necessary, after 1 year.

Nasal Diseases Uncommon in the USA

Atrophic Rhinitis

Atrophic rhinitis is a condition that has become uncommon in the USA. In most cases, it represents an end stage of prolonged infection and, as such, does not have a pathognomonic histopathologic appearance. No specific bacterium has been identified as the universal pathogen; there are probably a variety of antecedent infections. It is characterized by progressive chronic inflammation, atrophy, and fibrosis; these conditions affect the respiratory mucosa, submucosa, seromucinous glands, nasal vasculature and, ultimately, the periosteum and underlying bone. The result is an excessively patent nasal passage and a crusting, atrophic respiratory passage with islands of squamous metaplasia. The mucus is scant and thick and forms large crusts that at times harden into casts, obstructing the nose. The most prominent characteristic is a fetid odor from the foul mucus that the patient fails to appreciate fully, as the sense of smell is attenuated or lost.

Examination demonstrates a very patent nasal airway secondary to atrophy of the turbinates so that there is a clear view of the nasopharyngeal mucosa. The nasal breath is putrid. Mucosa is pale, shiny pink and is covered with crusts that often must be softened before they can be removed; bleeding may occur upon removal.

No entirely satisfactory treatment has been developed. Frequent nasal irrigations and continuous care are only partially helpful. Implant operations designed to reduce the nasal lumen work initially, but the implants are extruded in 80 per cent of the cases. Probably the best surgical procedure is bilateral closure of the nostrils in two stages, one side at a time; however,

experience with this procedure is limited to small numbers of patients (Young, 1967). The result is disappearance of the ozena and nasal discharge, but the patient must suffer the discomfort of being a mouth breather. During a 3-year period of closure, the condition apparently resolves, and normal mucous membrane is found open reopening the nostrils. The improvement is probably permanent. Partial, rather than total, nostril closure is advocated by others who claim an excellent cure rate if the naris is reduced to a diameter of 3 mm or less.

Rhinoscleroma

Although rhinoscleroma is a rare disease in the USA, it is endemic to subtropical countries in Asia, eastern Europe, Africa, South America, and Central America. The incidence is probably related to poverty, poor hygiene, and prolonged contact with infected individuals. The causative agent is *Klebsiella rhinoscleromatis.* Frequently, the infection begins in the anterior part of the nose as a firm submucosal plaque; it gradually expands into hard insensitive nodules that over many years fill and obstruct the nose. If left untreated, there is a tumorlike expansion into the skin of the nose and upper lip. Ulceration, fibrosis, and scarring follow. There is no discharge.

The nose is the most common site of the disease; however, the nasopharynx, paranasal sinuses, oropharynx, and lacrimal ducts can be involved as well. The disease is not fatal except when stenosing laryngeal or tracheal lesions occur.

The diagnosis is made by biopsy and culture. Microscopic examination demonstrates granuloma and fibrosis. Mikulicz's cells with a clear or vacuolated cytoplasm and a small dark nucleus are characteristically found. With special staining, the rod-shaped bacteria can be seen within the cytoplasm. Cultures are routinely positive only if they are taken from fresh biopsy material. The current suggestion for treatment is ampicillin 2 gm/day over a 6-week period.

Rhinosporidiosis

Rhinosporidiosis is a rare granulomatous disease of the nose, pharynx, and conjunctiva that apparently results from repeated exposure to the fungus *Rhinosporidium seeberi.* Usually, it is found in young men and is not contagious. The majority of cases occur in Sri Lanka, India, and parts of Africa, where the disease is endemic among low-income groups.

The otolaryngologist should suspect this diseae in recent immigrants from India who present with an irregular dull pink to red polyp or mass that bleeds on touch. Associated symptoms of obstruction, epistaxis, and purulent discharge are usually present. Treatment consists of complete surgical excision, during which brisk bleeding is often encountered. No medical treatment has been found effective.

Pathologic specimens are characteristic and demonstrate a chronic inflammatory reaction with interspersed spores; they range in size and appearance from small round cells with clear cytoplasm to large (300 micron) cysts filled with thousands of small, newly developed spores.

Leprosy

Leprosy is a chronic granulomatous disease that is endemic to warm, medically deprived countries. It is also endemic to the souther USA, principally Louisiana, Texas, California, and Hawaii, where occasional cases occur. The causative agent is *Mycobacterium leprae,* which is acquired by susceptible individuals only after a long, intimate exposure. The bacterium is attracted to subcutaneous nerves and secondarily to the skin and mucosa of the upper respiratory tract. The disease takes two principal forms that are named for the histologic appearance of the nodules present in each.

Tuberculoid leprosy is a relatively benign and often self-limited form of the disease that occurs in individuals who are relatively resistant to the organisms, as shown by lepromin test, a measure of cellular immunity against *M. leprae.* Patients with tuberculoid leprosy demonstrate a positive lepromin skin test result, for example, a strong inflammatory reaction to an intradermal injection of sterilized lepromatous granulation tissue containing killed *M. leprae.* This inflammatory reaction is thought to prevent the generalized spread of the bacteria, and in such cases the disease is often limited to one or more anesthetic macular plaque, which present as an area of skin innervated by an involved nerve that is often enlarged and palpable. Biopsy of the nerve demonstrates a noncaseating granuloma, or tuberculoid granuloma. The nose is less commonly involved in this form of leprosy.

Lepromatous leprosy is a widely disseminated form of the disease that occurs in patients with low resistance to the *M. leprae* bacillus; this is demonstrated by a lack of inflammatory response to the lepromin test. The bacteria can be found throughout the tissues of the entire body, but they have tendency to form lepromatous nodules in the skin of the cooler regions of the body. They appear as multiple, small, smooth-surfaced macules or papules and coalesce as they enlarge to form thickened plaques or nodules, which are commonly found on the alae of the nose, the ear lobe, the skin of the forehead, and the lips, causing the characteristic leonine facies. Biopsy of these nodules demonstrates the lepromatous reaction, which is characterized by macrophages filled with *M. leprae,* many giant cells containing lipid (lepra cells), and a lack of lymph cell reaction.

Patients with chronic lepromatous disease, and occasionally those with tuberculoid leprosy, may complain of nasal symptoms; however, lesions also occur in the mouth, pharynx, and larynx. Early nasal lesions commonly appear as red, granular ulcerations of the mucous membrane of the cartilaginous septum. As the granulations appear on both sides of the septum and enlarge, a perforation may appear. This is followed by frequent epistaxis from the margins of the perforation, as well as crusting and a purulent discharge that may contain numerous *M. leprae* bacilli. The end result is total destruction of the septum followed by atrophic rhinitis.

The diagnosis of early nasal leprosy is made by the identification of *M. leprae* found in scrapings of granulomatous lesions using Ziehl-Neelsen stains or by identification of the bacteria in biopsy material.

Local treatment includes good nasal hygiene and frequent use of oily nasal sprays to protect the dry, crusting mucosa. Generalized treatment consists of long-term administration of dapsone, a folate antagonist. Resistance to dapsone is increasing in patients undergoing long-term treatment. Rifampin is effective, but the high cost is a limiting factor and Federal Drug Administration (FDA) approval is pending. Clofazimine and ethionamide are among the drugs under investigation.

Surgery for Infection of the Maxillary Sinus

Antral Irrigation

The procedure of antral irrigation is used primarily in the physician's office to clear infected purulent material from the maxillary sinus after the patient has been taking antibiotics for at least 24 hours. Sinuses with thickened membranes are not appropriate for irrigation. The nose is anesthetized with a 4 per cent cocaine solution (or the equivalent) and a cannula is introduced through the actual ostium or better still is pushed through the medial wall of the sinus midway back under the inferior turbinate. Alternatively, the trocar and cannula can be inserted through the gingivobuccal sulcus and canine fossa into the sinus. Sterile saline is gently injected into the sinus, irrigating out purulent drainage contained therein. There should be no pain during the irrigation, and care should be taken not to inject air.

Creation of Nasoantral Window

This procedure is performed through the inferior meatus after the inferior turbinate has been in-fractured for exposure. It should be made large enough (± 2 cm) to preclude subsequent closure.

The Caldwell-Luc Procedure for Sinus Disease

This operation may be performed under local anesthesia with sedation or general anesthesia. The buccogingival sulcus in the region of the canine fossa is injected with lidocaine (Xylocaine), 1 per cent, with a 1:100.000 epinephrine solution to provide anesthesia and a dry field. A 1.5- to 2-cm incision is made either with a knife or the cutting Bovie, and the dissection is carried down to the periosteum, which is incised with a sharp blade. The elevation of periosteum is best accomplished with a Kitner forceps and is carried superiorly only enough to allow satisfactory access to the sinus. Identification of the inferior margin of the forament of the infraorbital nerve serves no purpose and can lead to symptoms secondary to stretching the nerve. Also, cheek retractors should be placed medially or laterally to the position of the infraorbital nerve, not over it. The bony opening is best made in the midanterior wall of the sinus and, if possible, should be limited to 1 cm in diameter, a size just large enough to inspect the entire sinus and removed diseased tissue. This technique reduces the incidence of injury to the dental roots. A rotating bur is the most atraumatic method of fenestration, although a 4-mm chisel and Kerrison forceps may be used as well. Material within the sinus should be Gram-stained and cultured for aerobic, anaerobic, tuberculous, and fungal pathogens, when appropriate. Normal-

appearing mucosa should be left behind to regenerate into the areas from which the inalterably diseased mucosa has been removed. The sinus ostium should be inspected from within the sinus.

The nasoantral window may be created by enlarging the natural ostium, or it may be placed through the nasoantral party wall under the inferior turbinate. An antrostomy through the natural ostium is effective only when the sinus mucosa has suffered minimal mucosal damage, as good ciliary function is required for drainage. Disease involving the anterior ethmoidal cells must be removed as part of this procedure, as must any bony or mucosal obstruction of the ostium. An ostium 5 mm or more can be created in this site. An antrostomy through the inferior meatus is more reliable for control of chronic infection, especially when extensive amounts of mucosa have been removed. One method of creating a nasoantral window is to press a pointed rasp through the party wall. The opening is enlarged and bone and mucosal fragments are removed with Takahashi or Kerrison forceps. In general, these openings will close to some degree, so they should be made large enough to preclude this possibility.

Bacitracin- or chlortetracycline (Aureomycin) saturated 1-inch iodoform gauze packing is used to pack the sinus and is brough out through the nasoantral window. The purpose is to prevent the sinus from filling with blood during the first postoperative hours, and in general the packing can be removed after 24 hours. Long-term postoperative packing invites reinfection.

The incision is closed with 2-0 chromic sutures placed as simple or interrupted mattress sutures. An ice pack for the cheek during the first 24 hours is helpful. The use of postoperative antibiotics should be based on the surgical findings and should be continued for at least as long as packing remains in place.

Surgery of the Frontal Sinus

Acute Frontal Sinus Disease

Trephination of the Frontal Sinus

Indications. Acute infection of the frontal sinus is associated with marked frontal pain, fever, and swelling of the overlying periosteum and soft tissues. Hospitalization is required for intensive antibiotic therapy, principally because the central extension of the infection through the diploic veins of the posterior sinus wall is a hazard. Spontaneous drainage through the nasofrontal duct can be encouraged by packing the region of the middle turbinate with cotton strips soaked in a vasoconstrictor, for example, 4 per cent cocaine four times a day, or by the use of topical vasoconstrictor sprays. If free drainage is quickly established and symptoms begin to resolve with antibiotics, continued observation should be sufficient; however, if no drainage is obtained, the sinus should be trephined. Attempts to probe or irrigate the nasofrontal duct are never indicated.

Procedure. After establishing adequate local or general anesthesia and performing a temporary tarsorrhaphy if required, a 1.5-cm skin incision should be made in the superomedial aspect of the orbit immediately below the brow. Blunt dissection should be directed toward the

inferior edge of the supraorbital rim and from there in a subperiosteal direction, exposing a small area of the floor of the frontal sinus. Occasionally, patients have a temporary or permanent numbness of the forehead caused by trauma to the supratrochlear or supraorbital nerves, and they should be forewarned of this possibility. Using a bur or curette, the thin floor of the bone is opened, and the pus is cultured and evacuated. A 1 inch-long polyethylene tube large enough to permit irrigation of the sinus is placed in the sinus and brought out through the medial edge of the incision. An additional procedure for patients with unilateral acute frontal sinusitis and large sinuses with a delicate interfrontal septum is the removal of a portion of the septum through a slightly enlarged trephination of the sinus floor. Mastoid curettes and small Kerrison foceps may be introduced to create a fenestra between the right and left sinuses in order to provide drainage through the noninvolved nasofrontal duct. The brow incision is closed in two layers, and the drain is sutured in place. On occasion, trephination must be done bilaterally; however, bilateral acute frontal sinusitis is unusual.

Postoperatively, irrigation of indwelling tubing with warmed sterile saline solution is performed four times a day until the return is clear and passes freely through the nasofrontal duct. Scarring from this procedure is usually minimal.

An office procedure for trephination, culture, and irrigation of the frontal sinus has been described using a Turkel bone biopsy needle. As experience is gained with this technique, it may replace the procedures already described in some clinical situations.

Chronic Frontal Sinusitis and Other Disorders

Indications. Long-standing, noncomplicated chronic frontal sinusitis alone is not a criterion for frontal sinus surgery even when there is radiographic evidence of thickened lining, sinus opacification, and bony sclerosis. Small, nonexpansile and nonobstructing mucoceles, polyps, and osteomas also may be safely left to observation. In general, the purpose of this surgery is to relieve intractable frontal sinus pain; to remove large expanding mucoceles, pyoceles, or benign or malignant tumors; and to prevent or control further central, orbital, or external extension of infection.

Procedures. In most instances, the procedure of choice consists of fat obliteration of the frontal sinus using an osteoplastic flap to gain access to the sinus. However, the Lynch operation is preferred for malignant disease because it does not bury diseased tissue behind an obliterative procedure and provides a drainage route for the necrotic debris that may accompany radiotherapy.

A number of frontal sinus procedures have been employed in the past and will be reviewed here, as they are occasionally useful. However, they are all complicated by problems of frequent recurrence of mucocele formation and chronic drainage.

In the *Riedel operation*, the frontal sinus is obliterated by removal of the anterior wall and floor and at times posterior walls (Mosher modification) of the sinus. It is cosmetically unacceptable except in cases of malignancy. The *Killian operation* is similar except that there

is less postoperative deformity because the supraorbital rim and a 10-mm portion of the anterior wall are left behind to suport the brow. The *Lynch operation* combines an ethmoidectomy and removal of the floor of the frontal sinus and its contents with the placement of a plastic tube stent from the sinus into the nose for several months to form a widely patent nasofrontal drainage tract. This operation is popular because it has been relatively successful; however, it tends to create some deformity and requires a great deal of postoperative care. Furthermore, recurrent mucoceles may form owing to incomplete removal of mucous membranes or stenosis of the reconstructed nasofrontal duct.

The Osteoplastic Frontal Sinus Operation

Cultures of the nose are taken 1 week prior to surgery, and the selection of postoperative antibiotics is based on the sensitivities of any pathogen grown. A template of the frontal sinuses is made from a radiograph (Caldwell view) and sterilized prior to surgery. Discussion of the advantages and disadvantages of brow and coronal incisions should be held with the patient preoperatively. Brow incisions usually leave a noticeable scar and are associated with greater loss of sensation over the distribution of the supratrochlear and supraorbital nerves than are coronal incisions. The disadvantage of the coronal incision is that it is necessary to shave a portion of the scal 1.5 inches back from the hairline and, as the hairline recedes, this incision may become visible in time.

Procedure. Following the induction of general anesthesia, the patient's face and abdomen are prepped and draped. Bilateral temporary tarsorrhaphies are performed, using 5-0 polyethylene suture material. Incision lines are infiltrated with 1 per cent lidocaine with epinephrine. Brow incisions run along the entire upper margin of the brow and are connected in the natural skin crease across the root of the nose when bilateral procedures are necessary. When making the coronal incision, hemostatic clips are required for control of bleeding. Elevation of the tissues is easily accomplished in the plane between the frontalis muscle and the frontal periosteum.

The template is placed over the frontal bone, and the periosteum is incised and elevated 0.5 cm from the anterior sinus wall. Next, a beveled bone cut is made through the outer table of the sinus with a small chisel or an oscillating sagittal saw, making sure to cut along the entire superior margin and including the superior orbital rims on both sides. Using a chisel, the front wall is carefully pried up; this will hinge on the periosteum inferiorly after fracturing along the floor of the sinus. Cultures are now taken, and the diseased tissue is removed. All remnants of mucosa are removed by carefully cleaning the entire sinus cavity with a cutting bur. Subcutaneous adipose tissue, obtained through a horizontal incision in the left lower quadrant of the abdomen, is used to fill the sinus and obstruct the nasofrontal ducts from above. The osteoplastic flap is replaced by suturing it to the periosteum with 3-0 polyglycolic acid (Dexon). If the flap is unstable, it can be fixed in position with fine-gauge stainless steel wire placed through two sets of opposing drill holes in the outer table. Postoperatively it is very important that the had be elevated and that a pressure dressing be placed over the forehead and eyes for at least 48 hours. Otherwise, inordinate swelling and hematoma formation will occur. Antibiotics are administered for 7 days postoperatively.

Repair of Frontal Sinus Fractures

Patients with suspected frontal sinus fractures should undergo careful radiologic evaluation, including sinus films, polytomography, or CT scan. Uncomplicated nondisplaced anterior wall fractures to not require exploration. Surgical exploration is required with comminuted or depressed fractures of the anterior or posterior walls, evidence of cerebrospinal fluid leakage, or delayed infection or mucocele resulting from a fracture.

At times, small depressed fractures of the anterior wall can be "popped" back into place following trephination of the floor of the sinus and elevation of bone fragments through this opening with a blunt hook or the tip of a curved hemostat. Occasionally the sinus can be explored and repaired through a laceration overlying the sinus cavity. Fractures resulting in the displacement of the supraorbital rim require an inferior brow incision and wiring of fragments with fine-gauge stainless steel wire. Badly comminuted anterior wall fractures that fail to hold their position after reduction can be held in place by fat obliteration of the sinus after complete removal of all mucous membranes.

Patients with radiographic evidence of a posterior wall fracture require surgical exploration. Evidence of a dural tear on radiography may be shown by a fluid level in the sinus or possibly air in the cranial cavity. The patient will note a clear, salty fluid running from one side of the nose in drops or in "gushes" with changes of position. At surgery, a laceration of the dura should be exposed with Kerrison bone forceps and sutured with interrupted stitches of 5-0 silk on a tapered needle. The suture line can be reinforced by placing temporalis fascia over the repaired dura and tucking it under the bony edge of the posterior frontal craniotomy. After thoroughly removing all frontal sinus mucosa, the sinus is packed with abdominal fat and is closed.

Surgery of the Ethmoidal Sinuses

Intranasal Ethmoidectomy

The most frequent indication for intranasal ethmoidectomy is recurrent obstructive nasal polyposis in which a patent nasal airway cannot be maintained by polypectomy in the physician's office. A second indication is chronic ethmoiditis that results from blocked nasal drainage. Occasionally, frontal sinusitis, sphenoiditis, or maxillary sinusitis will respond to removal of a nidus of infection located in infected polypoid ethmoidal cells. Preoperative x-ray studies will demonstrate opacification of the ethmoidal sinuses and upper nasal airways and decalcification of the bony ethmoidal septa.

The surgical landmarks described in textbooks are useful when present; however, because of the destructive nature of polypoid sinus disease and the frequency of previous polypectomies, the middle turbinate, bulla ethmoidalis, and infundibulum may be lacking or greatly distorted. Therefore, very often the surgeon must depend on his or her knowledge of more general anatomic relationships and resect diseased tissue until recognizable regional limits are reached.

The patient should be positioned flat on the operating table with his or her head straight. A slightly reversed Trendelenburg position will help reduce venous bleeding. Submucous resection is often done initially to straighten any septal deflection that may impede the surgeon's view. The level of the pupils marks the level of the cribiform plate. The roof of the ethmoid bone lies between the cribriform plate (roof of the olfactory slit) and the orbital plate of the frontal bone. The ethmoidal cell tract is usually narrower anteriorly (0.5 to 1 cm) and widens over about 4 to 5 cm to about 1 to 1.5 cm posteriorly. The anterior wall of the sphenoid bone is another constant landmark, as it indicates the posterior limit of the ethmoidal cells.

The dissection begins with resection of polyps in the nose. Next, the anterior half of the middle turbinate is resected with turbinate scissors or snare. This maneuver exposes the anterior ethmoidal cells, which are removed with a Coakley curette and Takahashi forceps. The dissection is carried superiorly, gradually resecting the bony attachment of the middle turbinate up to the cribriform plate. Bleeding is controlled by cautery using an insulated Pilling suction and by packing the resection area with strips of gauze soaked in a 1:100.000 epinephrine solution. Because of the need for frequent packing to control bleeding, the surgery progresses gradually by shifting from side to side to gain a dry field. Using the sphenoidal rostrum as a guide, the anterior face of the sphenoidal sinus is identified, and the remainder of the middle turbinate is resected. The dissection carefully progresses posteriorly with the cribriform plate and anterior face of the sphenoid bone in view. Diseased tissue is easily removed with a gentle tug. Any structure that does not come easily should not be removed until it has been positively identified.

The ethmoidal cavity is packed with a 36-inch strip of 1-inch gauze impregnated with chlortetracycline ointment. Inferiorly, finger cot packs are placed between the inferior turbinate and the septum. The packing is removed in 24 to 48 hours. Ideally, patients should be seen twice weekly for 2 weeks postoperatively for removal of large fibrin clots and disruption of synechiae as healing progresses.

External Ethmoidectomy

The external ethmoidectomy is used for treatment of extensive polypod sinus and nasal disease, and chronic sinus infection; as an approach to tumors of the frontal, ethmoidal, and sphenoidal sinuses; in searching for and repairing cerebrospinal fluid leaks in the cribriform, ethmoidal, and sphenoidal regions; and as an extracranial approach in hypophysectomy. It is also part of orbital decompression procedures. The operation can be and often is done in combination with other procedures such as the Lynch frontal sinus operation, sphenoidectomy, and the Caldwell-Luc procedure. External ethmoidectomy is frequently done bilaterally. However, when ethmoidectomy is carried out as part of a combined procedure, it is our opinion that the sinus surgery should be staged: each side should be operated on separately, with 1 or 2 months between, as bilateral procedures of this magnitude are associated with large blood losses and high morbidity.

The patient's head is positioned flat on the operating table and held with a donut. Bilateral temporary tarsorrhaphy is done routinely. The incision sites are infiltrated with 1 per cent

lidocaine with a 1:200.000 epinephrine solution, and the nose is packed with cotton strips soaked in 4 per cent cocaine to induce vasoconstriction. A superficial 2- to 3-cm incision is made through the skin in a vertical direction halfway between the inner canthus and the dorsum of the nose. The subcutaneous tissues are dissected cautiously down to the periosteum so that the angular vein and artery can be identified and cauterized. Ligatures in this region can result in postoperative discomfort for patients who wear glasses. The periosteum is elevated off the ascending process of the maxilla, then posteriorly off the anterior lacrimal crest out of the lacrimal fossa, thus lifting out the lacrimal sac, and off the posterior crest.

At this point, three retraction sutures are placed through the periosteum and skin on each side of the wound. After two 4 inch-square moist gauze pads are placed as protection over each eye, heavy hemostats are hung from the sutures to provide sufficient traction to hold the wound open.

The periosteal elevation is continued posterosuperiorly, lifting off the middle canthal ligament and following the lamina papyracea up to the frontoethmoid suture line, which is located about three quarters of the way up the medial wall of the orbit. This is an important landmark, as it marks the uppermost extension of the posterior ethmoidal cells. The anterior and posterior ethmoidal arteries are found in this suture line and should be catuerized using a bipolar cautery tip. The posterior ethmoidal artery marks the posterior limit of the periosteal elevation.

After identifying the lacrimal duct to avoid injury to it, a mastoid cavity curette is used to break through the thin bone of the lacrimal fossa. A Kerrison rongeur is then used to remove the entire lacrimal bone and the adjacent portion of the ascending process of the maxilla, thus exposing the agger nasi cells located anterior to the tip of the middle turbinate. These are removed with Brownie or Takahashi forceps or curettes. The Brownie forceps is blunter and broader than the Takahashi and is therefore safer to use for the major portion of the ethmoidal resection. The narrow Takahashi forceps is used in small recesses and crevices. Upon removal of the agger nasi cells, the surgeon confronts the undersurface of the mucosa of the lateral wall of the anterior portion of the nose; this is best opened with a posteriorly based U-shaped incision exposing the nasal packing, which is removed. Thus oriented, the operation continues by combined intranasal and external dissection. The middle turbinate or what remains of it is identified and removed with turbinate scissors. Using Brownie forceps, the dissection is carried superiorly to the cribriform plate and posteriorly to the frontal face of the sphenoid bone. Through the external incision, the posterior ethmoidal cells and usually the anterior half of the lamina papyracea are removed. Exposure for this work is gained with a curved orbital retractor that also serves to protect the orbital periosteum from injury. The surgeon must be well aware of the position and proximity of the optic nerve when removing the posterior ethmoidal cells.

Bleeding is controlled by cautery using an insulated Pilling suction and frequent packing with strips of gauze soaked in a 1:100.000 epinephrine solution. The front wall of the sphenoid bone is routinely removed. The expected blood loss may amount to 1 to 2 units, and transfusion of packed red cells is sometimes required. One-inch gauze impregnated with chlortetracycline is used to pack the surgical defect. Finger cots, used to pack the nose, support the ethmoid packing.

Retraction and tarsorrhaphy sutures are removed, and a compressed absorbable gelatin sponge (Gelfoam) is placed over the packing visualized through the wound. The periosteum is closed with 4-0 chromic gut sutures, and the skin is sutured with 5-0 polyethylene.

The patient's head should remain elevated postoperatively. A moderate pressure dressing composed of eye pads and fluffs held in place by elastic tape from the forehead to the cheek is used for 24 to 48 hours to prevent postoperative edema and ecchymosis. Packs are removed in 1 to 2 days. At this time, fibrin clots and mucus are removed, and frequently thereafter, until the sinus cavity is healed to prevent infection and synechiae formation.