Chapter 3. Nose and Paranasal Sinuses

I. Introduction

A. Anatomy of the nose

1. Structure and support. The external nose has a bony and cartilaginous framework. The bony portion consists of the paired nasal bones, the ascending or frontal processes of the maxilla, and the bony septum. The cartilaginous framework consists of the alar (lower lateral) cartilages, the upper lateral cartilages, and the cartilaginous septum.

The inferior, middle, and superior turbinates are located on the lateral nasal wall. Below each turbinate is its corresponding meatus. The nasolacrimal duct opens into the inferior meatus. The frontal sinus, maxillary sinus, and anterior ethmoid cells open into the middle meatus. The posterior ethmoid cells open into the superior meatus, with the sphenoid sinus opening into the sphenoidal recess. The concept of the osteomeatal complex (OMC) is important anatomically, physiologically, and surgically. It incorporates the anterior ethmoid cells, maxillary sinus ostia, and anterior middle meatus. Mucosal swelling, infection, or anatomic variation can narrow this unit, adversely affecting ventilation and mucociliary clearance, the net result of which is recurrent acute or chronic sinusitis.

2. Blood supply. The nasal blood supply is provided by branches of both the internal and the external carotid arteries. The sphenopalatine, the descending palatine, and the superior labial arteries are branches of the external carotid artery. The anterior and posterior ethmoid arteries are derived from the internal carotid artery.

Anteriorly, the nasal septum is supplied by the sphenopalatine, greater palatine, superior labial, and anterior ethmoid arteries. The anastomoses of these vessels are called Kiesselbach's plexus. The posterior septum receives its blood supply from the posterior ethmoid and sphenopalatine arteries. The blood supply to the lateral nasal wall is from the anterior ethmoid, posterior ethmoid, and sphenopalatine arteries.

B. Examination of the nose and sinuses

1. Physical examination. A nasal speculum and focused light, such as a head mirror, should be used. The nasal speculum is held in the palm of one hand with the forefinger on the ala, and the blades are opened vertically. The other hand is used for positioning the patient's head.

a. Topical vasoconstrictors are useful adjuncts.

(1) Topical phenylephrine is used in 0.25-0.50% concentration. An atomizer can be used for application. Phenylephrine should be avoided in patients with arteriosclerotic cardiovascular disease or glaucoma.

(2) Topical epinephrine, 1:1000 concentration, can be applied using cotton pledgets, giving maximum vasoconstriction. Epinephrine is contraindicated in the presence of arteriosclerotic cardiovascular disease.
(3) **Topical cocaine**, a vasoconstrictor and topical anesthetic, is discussed under topical anesthesia (see b.2.).

b. **Topical intranasal anesthesia** can be obtained by application of lidocaine or cocaine. The anesthetic of choice is placed on cotton strips and inserted. All excess medication must be removed before placement to avert excessive absorption.

(1) **Lidocaine.** If 4% lidocaine is applied, it has a 15-minute duration of action. The total dose should not exceed 200 mg. The first signs of lidocaine toxicity are CNS hyperactivity followed by cardiovascular depression.

(2) **Cocaine.** Topical cocaine is used in a 4 or 5% solution. It has a 45-minute duration of action and also causes local vasoconstriction. The maximum topical dose should be approximately 200 mg.

**Toxicity.** Toxic effects have occurred with as little as 20 mg of cocaine applied topically. Early signs of cocaine toxicity are stimulation, garrulousness, restlessness, increased respiratory rate, increased blood pressure, tremors, convulsive movements, bradycardia, and then tachycardia. A patient can pass through this initial stage without its being recognized and progress to the depressive effects: hypotension, decreased respiratory rate, and convulsions leading to cardiorespiratory collapse.

Therapy of a toxic reaction includes cardiopulmonary support and short-acting barbiturate.

c. **Combined vasoconstrictors and topical anesthesia.** A combination of 1% phenylephrine and 4% xylocaine mixed in equal aliquots, administered via a nasal insufflator, is particularly efficacious for office nasal endoscopy.

2. **Diagnostic techniques**

a. **Transillumination** of the sinuses can be performed with a bright penlight in a darkened room. Maxillary sinus transillumination is performed by placing the light over each maxillary sinus and observing the transmission through the palate. Frontal sinus transillumination is performed by placing the light below the midportion of each eyebrow. In each instance, the light transmission through the sinuses is compared. If equal, usually opacification can be ruled out. It is of limited value today.

b. **Nasal culture.** The correlation between nasal cultures and sinus cultures taken at surgery is low. Although nasal cultures are of questionable reliability, they may have some validity when a purulent discharge is cultured at the sinus ostia. An antral aspirate is of greater value than nasal culture in cases when culture documentation is essential.

c. **Antral puncture.** Antral puncture is most often performed by placing a nasal antral trocar or 18-gauge spinal needle into the inferior meatus and by directing the trocar in a posterior and lateral direction through the thin bony plate separating the turbinate from the maxillary sinus. This procedure can be performed for drainage or irrigation of a maxillary sinus when an air-fluid level is seen on x-ray and a response has not been observed to a
conservative medical regimen (see sec. IV.B.3.). Aspiration can be used for culture documentation when specific identification of the pathogen is mandatory, as in immunosuppressed patients. The complications of antral puncture include hemorrhage, osteomyelitis, air embolism, injury to the globe, and injury to the optic nerve.

3. Radiology of the nose and paranasal sinuses. Radiography is often essential for the accurate diagnosis of sinus disease. Routine sinus x rays include the Caldwell, Waters, lateral, and submental vertex (base) views. The frontal and ethmoid sinuses are seen on the Caldwell view. This view best demonstrates air-fluid levels in the frontal sinus. The maxillary sinuses are best seen on the Waters' view. The lateral view demonstrates the sphenoid and frontal sinuses, along with the nasopharynx and sella turcica. The sphenoid sinuses, nasopharynx, nasal cavity, and the medial and lateral antral walls are seen on the submental vertex view.

Computed tomography (CT) scans add a new dimension. Polytomography is now less applicable; more reliable than plain films, it gives less definition than CT and is associated with more radiation exposure. CT can precisely pinpoint minor alterations in the osteomeatal complex (OMC). It is an invaluable adjunct for selecting surgical candidates for functional endoscopic sinus surgery (ESS), yet it cannot and should not be used as the principal determinant for surgery. Clinical correlation is essential. Both axial and coronal views should be performed, although information obtained by coronal views is far more applicable to surgical correlation.

II. Trauma to the nose and sinuses

A. Traumatic paranasal sinus fractures

1. Frontal sinus. A frontal sinus fracture usually results from direct force to the region, the area of impact being superior to the nasofrontal suture.

a. Examination usually reveals a depression of the anterior frontal sinus wall. The fracture may also involve the posterior wall and cribriform plate. The nasofrontal duct and dura are subject to injury as well. Dural tears may result from a fracture of the posterior frontal sinus wall, fracture of the roof of the ethmoid coexisting with a frontal sinus injury, or fracture of the cribriform plate following telescoping of the perpendicular plate of the ethmoid. Subsequent cerebrospinal fluid (CSF) rhinorrhea, as well as laceration of the anterior ethmoidal arteries, may be present. Usually there is only slight, if any, displacement of the nasal bones with forehead trauma.

b. Diagnosis. Fractures of the frontal sinus usually involve the anterior wall. These fractures can be diagnosed either by direct inspection and palpation or by radiography. In areas of question (eg, the posterior wall), polytomography or CT should be performed.

c. Treatment. If only the anterior wall is involved, the area can be explored through a laceration or brow incision. The fragments should be elevated and stabilized whenever possible. The sinus should be drained only if the integrity of the duct is questioned. In nondisplaced posterior wall fractures, no specific therapy is usually necessary. Displaced posterior wall fractures require a neurosurgical consultation. Similarly, displacement of the
posterior wall is an indication to explore the sinus and, in most instances, obliteration should be considered. Antibiotics may be necessary to prevent retrograde meningitis.

Late realignment of fracture deformities is difficult because of bony fixation. These defects are often repaired with implantable material.

d. Complications of frontal sinus fractures include osteomyelitis and secondary abscess formation. Intracranial complications can occur, including meningitis as well as epidural and subdural abscesses. Orbital cellulitis can develop from an extension of the infection to the contiguous orbit. Mucoceles and pyoceles within the sinus may evolve if the nasofrontal duct is injured and not recognized. A headache may be the only indication of an impending complication.

1. Injury to the nasofrontal duct with edema and hematoma formation may cause secondary obstruction. Pain can develop secondary to the vacuum within the sinus (aerosinusitis). The therapeutic goal is to ensure a physiologic conduit. Minor injury is treated expectantly with topical and systemic vasoconstrictors. With severe injury and ductal disruption, attempts can be made to reconstruct the duct. If unsuccessful, obliteration of the sinus may be necessary. Obliteration is mandatory when there is a persistent CSF leak via the posterior sinus wall.

2. CSF rhinorrhea. It is estimated that as many as 25% of frontoethmoid sinus fractures are associated with a CSF leak. Most leaks occur within 48 hours, with a late onset secondary to liquefaction of blood within the sinus. Usually, CSF rhinorrhea stops within several days after injury. The CSF leak is associated with dural tears as noted in 1.a. The site of origin can often be delineated by CT scan, with evidence of a fracture of the posterior frontal sinus wall, roof of the ethmoid, cribiform plate, or fluid within the sphenoid sinus. Air in the subdural space is often associated with a CSF leak. The leak can be confirmed by obtaining a glucose level on fluid collected. Clinistix and test tape measurements are often not accurate; however, they can be of some assistance early in the diagnostic spectrum. A definitive diagnosis can be made by injecting fluorescein, radioactive serum albumin, or indium into the subarachnoid space by lumbar puncture and subsequently testing for their presence on cottonoid pledgets appropriately inserted intranasally.

Routine packing is discouraged if a CSF leak is suspected. The head should be elevated. Antibiotics may be recommended in an effort to prevent meningitis. Surgical intervention is usually withheld for several weeks, as most often leaks subside spontaneously. When indicated, an osteoplastic flap approach to the frontal sinus can be used. The repair of a cribiform plate or roof of ethmoid fracture has been traditionally performed through an external ethmoidectomy approach. ESS is currently being evaluated for repair of these injuries.

2. Ethmoid sinus fractures often occur in association with other injuries, especially with injuries to the frontal sinus. These fractures result most often from an impact over the nasal bones with retrodisplacement of the interorbital structures. Often there are associated orbital rim fractures as well as telescoping of the ethmoidal plate, with lateral displacement of the medial orbital walls. Associated injuries include obliteration of the nasofrontal duct, laceration of the anterior ethmoidal artery, and disruption of the trochlea.
a. Signs and symptoms. Diagnostically, there is flattening of the midface and lateral displacement of the palpebral fissures. Pseudohypertelorism may occur secondary to tearing of the medial palpebral ligament. Extraocular muscle motion may be limited, especially of the medial rectus. The trochlea suspends the superior oblique muscle on the superior medial corner of the orbit, acting as a pulley. Displacement can be associated with diplopia on downward gaze. An associated CSF leak may be present.

b. Diagnosis. Radiographs are essential, and the complexity of the bony structures in this region necessitates axial and coronal CT scans.

c. Therapy. Antibiotics are almost always indicated. Nasal packing should be avoided because packing allows for an avenue of secondary infection. Depressed fractures of the nasal bones may require wiring with external stenting. External traction is rarely necessary. Septal reduction should precede nasal repair; however, excessive force during manipulation is contraindicated because movement of the perpendicular plate of the ethmoid, in association with septal mobility, may induce a CSF leak.

d. Complications. Epistaxis may occur and is usually secondary to a laceration of the anterior ethmoidal artery. Ligating the vessel via an external ethmoidectomy approach may be necessary. Other complications of ethmoid fractures include persistent visual disturbances, chronic frontal sinusitis secondary to injury of the nasofrontal duct, meningitis, and midface deformity.

3. Maxillary sinus fractures can occur with zygomaticomaxillary complex (ZMC) or orbital floor fractures.

a. Signs and symptoms. Occlusal changes are not infrequent with these fractures, and an open-bite deformity may occur. Often there is hypesthesia or anesthesia of the cheek secondary to edema or transection of the infraorbital nerve. Whenever the orbit is involved in a fracture, retinal artery occlusion may occur secondary to edema and/or hematoma. Vision must be assessed, and any changes merit ophthalmologic consultation.

b. Diagnosis. Orbital floor fractures can cause inferior rectus entrapment and upward gaze diplopia. A red glass cover test is the best means of evaluating diplopia. A red glass is placed in front of one eye, and the patient is asked to look at a light in all positions of gaze. If diplopia is present, the patient will see two separate, partially overlapping lights, and ophthalmologic consultation should be obtained.

Radiographically, posttraumatic clouding suggests a fracture, providing sinusitis was not present before the injury. CT scans are also valuable in assessing the degree of injury.

c. Therapy. The use of antibiotics is controversial in simple maxillary sinus fracture. If an open wound exists, antibiotics should be considered. Displacement requires reduction with fixation.

4. Sphenoid fracture. Sphenoid fracture usually occurs in association with massive head trauma. Vital signs should be checked regularly until stable. The head and neck are immobilized until a cervical spine fracture is ruled out radiographically. A rapid but thorough
neurologic and physical examination is performed, followed by repeat neurologic
examinations. The sphenoid sinus is evaluated radiographically by CT scans, and an air-fluid
level may be found. Frequently, CSF rhinorrhoea develops. Intracerebral injury may be
present; subdural and epidural hematoma can develop. The patient is also at risk for
developing cerebral edema. A neurosurgical consultation should be obtained if the patient has
signs of an intracranial injury.

5. Orbital complications of sinus fractures

a. The *trochlea* of the superior oblique muscle may be *lacerated*, causing diplopia on
downward gaze.

b. The *medial palpebral ligament* may be torn. This ligament anchors the tarsal plate
and passes anterior to the lacrimal canaliculi and sac. The therapy of medial palpebral
ligament tears is directed toward their reattachment to either the nasal bones or lacrimal
crests.

c. Injury to the *lacrimal apparatus* may be diagnosed by introducing fluorescein into
the conjunctival sac and documenting its presence or absence within the nose. Probing and
irrigation may open the ductal system. Often, patency can occur after many months, without
surgical intervention. An immediate dacryocystorhinostomy is rarely indicated. This operation
can be performed at a later date, after ophthalmologic consultation is obtained.

d. Entrapment of the medial rectus or inferior rectus muscles can occur with injuries
to the ethmoid complex or maxillary sinus. Entrapment can be diagnosed by demonstrating
diplopia on lateral gaze with medial rectus injuries, or on upward gaze with inferior rectus
injuries. The forced-duction test is valuable in assessing inferior rectus entrapment. In this
test, the eye is anesthetized, and the muscle in question is grasped with a forceps near the
globe. Passive attempts at movement are made, and limitation suggests entrapment.

e. Orbital cellulitis can occur with infection spreading from the contiguous sinus.
Characteristics of orbital cellulitis include lid edema, exophthalmos, and chemosis of the
conjunctiva, along with progressive immobility of the eye, which is associated with severe
pain. Hospitalization is mandatory, and intravenous antibiotics are essential. Frequent eye
exams with assessment of vision and eye motion must be performed. Incision and drainage
of an abscess in the infected region is necessary if resolution is not effected with medical
therapy. After initial drainage with quiescence, an external ethmoidectomy is often performed.

f. Retinal artery occlusion may also occur.

B. Nasal trauma

1. Nasal fractures (See Chap. 1, Nasal Fractures.)

a. Nasal trauma during delivery. During delivery, nasal trauma can result in external
nasal deformity, septal deviation, and obstruction. At birth the nasal supporting structure is
cartilaginous; once the stress is removed, minor aberrations gradually resume their
pretraumatic configuration. Major septal deformities are readily reduced in the nursery. In the
The rare instance of significant nasal vault trauma with an attendant obstruction, total rapid reduction of displaced segments is essential (see Chap. 1, Nasal Fractures).

2. Septal injuries. After trauma, a nasal septal deviation must be evaluated as to whether the deviation antedated or resulted from the most recent injury.

a. Signs and symptoms. The history can be helpful but may not be reliable. Most patients never carefully inspect their nose until a traumatic episode. On examination, mucosal lacerations, mucosal ecchymoses, and mobility usually signify an acute injury. The septum is deflected off the midline and obstructs the airway.

b. Diagnostic aids. X rays are not always essential since the diagnosis is primarily a clinical one. At times, x rays may assist in confirming a difficult diagnosis.

c. Therapy. Posttraumatic septal deviations should be corrected whenever possible within 5 days of injury. Asch forceps or a nasal elevator is used for the reduction. If an adequate reduction is not obtained, a septoplasty can be performed approximately 6 months after the acute injury.

d. Complications. Nasal septal hematoma is a complication of nasal trauma. Blood collects beneath the mucoperichondrium or mucoperiosteum. On intranasal examination, there is a markedly swollen, sometimes fluctuant area. This area is best palpated with a cotton-tipped applicator, and the diagnosis confirmed by aspiration with an 18-gauge needle. If blood is obtained, a vertical incision through the mucoperichondrium is made to provide adequate drainage. Then packing is usually necessary to oppose the mucosa to the underlying cartilage. Untreated, a nasal septal abscess is often a sequela. Hospital admission for this condition is warranted if systemic signs of infection (eg, fever) are present. Antibiotics must include therapy directed toward Staphylococcus as well as gram-negative organisms.

A nasal saddle deformity can result from an inadequately treated nasal septal hematoma or abscess.

III. Nasal obstruction

A. Infection: nasal furunculosis and cellulitis

1. Signs and symptoms. A nasal furuncle usually presents as an erythematous, indurated, raised, firm lesion of the nasal tip. It can have a surrounding area of cellulitis and is usually caused by a staphylococcal infection of the nasal vibrissae.

2. Therapy. The venous drainage of the nasal tip region is into the cavernous sinus. Thrombosis of the cavernous sinus infrequently results from staphylococcal infection.

The infection must be treated aggressively with an antibiotic that provides staphylococcal coverage, such as dicloxacillin, 500 mg PO qid. A topical antibiotic ointment (2% mupirocin (Bactroban) or neomycin-polymyxin-bacitracin) may be of value. Warm soaks are applied locally. Incision and drainage should not be performed until there is adequate antibiotic coverage, since this can result in bacterial seeding to the cavernous sinus.
patient is observed daily until the infection begins to resolve. If spreading cellulitis occurs or if systemic symptoms develop, hospitalization for intravenous therapy is appropriate.

**B. Nasal and sinus foreign bodies** result from penetrating trauma, childhood insertion, or iatrogenic causes.

1. **Signs and symptoms.** Adults present either with a chief complaint of a foreign body or with chronic or recurring unilateral sinus infection following facial injury. Children present with unilateral rhinorrhea or unilateral infection. Whenever a child presents with unilateral rhinorrhea, a foreign body should be suspected.

   During the extraction of a maxillary tooth, a portion of the root or tooth restoration can be forced into the antrum. This problem can go undetected until recurrent sinus infection occurs.

2. **Therapy.** In adults, nasal foreign body removal can be effected after application of topical cocaine 4%. The object can then be removed by suction or manipulation with appropriate instruments. Surgery is obviously required to remove a foreign body within the sinus. In children, general anesthesia is frequently necessary to remove the object. In a cooperative child, the foreign body can occasionally be retrieved as an office procedure.

3. **Sequelae.** Rhinoliths and maxillary sinus antroliths develop around a chronic foreign body nidus that has been in place for prolonged periods of time.

**C. Mucosal alterations**

1. **Common cold.** The common cold is the most frequent infectious process involving the nasal mucosa.

   a. **Signs and symptoms.** The well-known presenting symptoms include a sensation of nasal congestion, watery rhinorrhoea, and sneezing, often accompanied by malaise and myalgia. On physical examination, there is marked edema of the nasal mucosa. The edema can cause obstruction of the sinus ostia. Within a few days of the onset of symptoms, some patients develop a secondary bacterial sinusitis.

   b. **Therapy.** The common cold is treated symptomatically with analgesics, mucolytics, and vasoconstrictors. There is no known cure or proved method of consistently preventing this malady.

2. **Allergic rhinitis** can be seasonal or perennial. It presents with nasal obstruction, nasal pruritus, rhinorrhea, and sneezing. Patients often describe their symptoms as "sinus trouble" or a common cold.

   a. **Signs and symptoms.** The diagnosis of allergic rhinitis is based on a history of seasonal symptoms, including sneezing, pruritus, and rhinorrhea. On physical examination, swollen turbinates covered with edematous blue or pale mucosa are frequently observed. Nasal polyps are often found in association with allergic rhinitis, but can also be caused by chronic infectious sinusitis.
b. Diagnostic tests. An elevated immunoglobulin E (IgE) level, total eosinophil count, positive skin tests, or a radioallergosorbent test (RAST) confirms the history. The presence of eosinophils on nasal smear is also suggestive of allergic rhinitis.

c. Therapy. If possible, affected patients should avoid the responsible allergen. Symptomatic relief can be achieved with oral antihistamines and decongestants.

(1) Antihistamines. Chlorpheniramine, an alkylamine, can be given in an adult dose such as 4 mg qid. If patients do not respond, an antihistamine of a different class, such as tripeledamine, an ethylendiamine, should be given (see Chapter Appendix). The usual adult dose of tripeledamine is 25 mg qid. Often one class of antihistamine may be effective when another fails. The newer, "nonsedating" antihistamines are far more frequently prescribed - astemizole, terfenadine. Terfenadine has a relatively rapid onset of action (hours) and is administered bid. Astemizole has a slow onset (days) and a long half-life. Its use precludes skin testing for several weeks after cessation of the medication. In general, all antihistamines can make patients somnolent. Patients should be alerted of this possibility.

(2) Sympathomimetic medications with alpha-receptor activity can be given with or instead of an antihistamine. Pseudoephedrine is taken in 30- to 60-mg doses qid. This medication causes stimulation in some patients and somnolence in others. All patients must be advised of the potential side effects.

Sympathomimetics are contraindicated in patients with any form of arteriosclerotic cardiovascular disease. As is discussed under rhinitis medicamentosa (see 7.), nasal drops or sprays containing sympathomimetic medications should not be used over prolonged periods of time.

(3) Steroids. Aerosolized dexamethasone spray can be used for acute exacerbations. Patients must be cautioned not to overuse this medication, as adrenal suppression can result. Beclomethasone can be used intranasally with a decreased potential for adrenal suppression. Patients who are skin test-positive for specific allergens often benefit from a course of desensitization therapy.

For patients with edematous inferior turbinates occluding the nasal airway, electrocautery of the turbinates or site steroid injection may also afford relief.

3. Nasal polyps represent localized areas of swelling within the nasal or sinus mucosa, usually arising in the ethmoid sinuses. The polyps can progressively enlarge, obstructing the nasal airway. They can be associated with either allergic rhinitis or infectious rhinosinusitis. Treatment is necessary when there is (1) symptomatic nasal obstruction, (2) recurrent or chronic infection, or (3) a consideration of malignant change.

Medical therapy can be initiated with Beclomethasone spray. For allergic-type polyps, the usual dosage is 2 sprays bid or tid. A short course of systemic steroids can also be tried in acute situations, if no associated infection is present. Intranasal chromolyn spray is an adjunctive preparation. Antibiotics are necessary with associated infection.
Patients not responding to medical therapy frequently benefit from a nasal polypectomy. Choanal polyps appearing in the nasopharynx may originate in the maxillary sinus, making surgical antrostomy either by ESS or open (Caldwell-Luc) technique a consideration.

4. Cystic fibrosis. Patients with cystic fibrosis have a thick, viscid mucus, their main symptoms being nasal obstruction and rhinorrhea. Often, secondary nasal and sinus infections occur. On examination, tenacious mucus and polyps are observed in 10-20% of the cases.

Therapy is directed toward reducing secondary infection and establishing a nasal airway. Nasal cultures should be taken and the patient appropriately treated. A bedside humidifier and nasal irrigations with normal saline can prove beneficial. Polypectomy may be needed repeatedly to maintain a patent nasal airway. Maintaining a patent airway is not a simple task, often requiring extensive resections in the form of endoscopic intranasal ethmoidal surgery.

5. Atrophic rhinitis (ozena) is associated with an offensive nasal odor, epistaxis, anosmia, nasal obstruction, and purulent nasal crusting. On examination, there is nasal crusting, atrophy of the turbinates, and secondary enlargement of the nasal cavity. The etiology of the primary condition is unknown, but it can be a sequela of excess fibrosis following nasal surgery. Histologically, there is fibrosis of the submucosa, without inflammatory infiltration.

Medical therapy consists of nasal irrigations with isotonic saline and correction of underlying medical problems when feasible. For those not responding to medical therapy, there are two surgical procedures currently used. One procedure is an endonasal microplasty in which the internal nasal dimensions are markedly decreased. The other is staged complete closure of the nostrils. Neither procedure is common.

6. Vasomotor rhinitis presents with symptoms of nasal obstruction or rhinorrhea or both. On physical examination, these patients may not have swollen, edematous turbinates or excessive nasal mucus. An allergy evaluation is negative. Some benefit may be obtained from aerosolized steroids. Symptomatic relief at times is obtained from antihistamines and decongestants (see 2.c.).

Patients with edematous turbinates not responding to medical therapy often benefit from electrocautery of the inferior turbinate or from submucosal injection of long-acting corticosteroids, eg, prednisolone tebutate (Hydeltra-TBA). Intranasal cryosurgery to the postganglionic parasympathetics in the medial aspect of the pterygomaxillary fossa provides relief for many patients.

7. Rhinitis medicamentosa develops after prolonged use of topical vasoconstrictors. Symptomatically, the effective duration of the medication decreases, and rebound develops. Subsequently, marked erythema with edema of the nasal mucosa evolves.

The patient's understanding of the condition is the cornerstone of management. Vasoconstrictor usage is curtailed with the assistance of aerosolized intranasal beclomethasone or injections of prednisolone tebutate. Normal saline spray intranasally may give some
symptomatic relief during the period of withdrawal. Systemic decongestants, such as pseudoephedrine, are also of some value. A brief course of oral steroids (less than 1 week - methylprednisolone) may rapidly improve symptoms of withdrawal while topical therapy begins to take effect.

8. Intranasal neoplasms can cause nasal obstruction as a primary symptom, in addition to epistaxis, external nasal swelling, and sinusitis secondary to obstruction of the sinus ostia. Symptoms are usually unilateral. Pain is frequently present with malignancy.

Intranasal neoplasms include inverted papilloma, hemangiopericytoma, melanoma, esthesioneuroblastoma, squamous cell carcinoma, plasmacytoma, lymphoma, and rhabdomyosarcoma.

D. Nasopharyngeal obstruction

1. Adenoid hypertrophy. Enlarged adenoids often obstruct the posterior nasal choanae in childhood. Snoring, mouth breathing, slow eating, nasal discharge, drooling, and adenoid facies are all potential sequelae. A narrow, high-arched palate with associated malocclusion may occur. The degree of facial alteration can be measured on cephalometric radiographs. Rarely, a child will develop cor pulmonale from upper airway obstruction secondary to adenoid or tonsillar hypertrophy or both. This condition is an absolute indication for adenoidectomy and tonsillectomy.

Other indications for adenoidectomy due to upper airway obstruction are not clearly defined, and the decision to perform surgery must be individualized. The relationship of adenoid hypertrophy to otitis media is still being investigated. Recent data suggest some efficacy in removing the adenoids in selected cases.

2. Choanal polyps. Choanal polyps originate in the maxillary sinus and extend through the sinus ostia to the nasal cavity and posteriorly into the nasopharynx. These polyps can cause nasopharyngeal obstruction. The treatment is surgical; a maxillary antrostomy is necessary to ensure complete removal. Endoscopic techniques are becoming more commonplace in the management of the condition.

3. Angiofibromas of the nasopharynx are rare lesions, occurring primarily in adolescent males.

a. Signs and symptoms. Epistaxis and nasal obstruction are the primary symptoms. Less frequent are serous otitis, anosmia, hyponasal speech, and sinusitis. With skull base involvement, the second, third, fourth, and sixth cranial nerves can be affected.

b. Physical. Mucus secondary to obstruction may preclude visualization on anterior rhinoscopy. Nasopharyngeal examination frequently demonstrates a purplish, lobulated mass.
c. Studies

(1) Lateral skull films in most instances demonstrate a soft tissue mass.

(2) Extension is seen on computed tomography (CT) scans of the region. Bone destruction is evident in approximately 50% of cases.

(3) Arteriography is essential to delineate the major contributing vessels.

d. Pathology. The ratio of vascular to connective tissue varies significantly from tumor to tumor.

e. Treatment

(1) Surgery

(a) Blood loss is markedly reduced by preoperative embolization.

(b) Estrogen therapy has been reported to decrease vascularity. It is not indicated if embolization is used.

(c) Cryosurgery was used prior to embolization. It now has merit for accessible recurrences.

(d) Surgery is the treatment of choice. Biopsy is not indicated, as bleeding can be profuse. Wide exposure is essential to minimize recurrence.

(2) Radiotherapy. Most feel radiotherapy should be reserved for lesions not amenable to resection; however, long-term control of primary lesions has been reported.

f. Prognosis. A 50% incidence of recurrence was not uncommon in the past. Better imaging, decreasing blood loss, and more aggressive surgery should decrease the recurrence rate.

4. Squamous cell carcinoma. Squamous cell carcinoma of the nasopharynx may present with cervical lymphadenopathy but more frequently will present with nasal obstruction, frank epistaxis, or bloody nasal discharge. This entity must be ruled out whenever an adult presents with a unilateral serous otitis. Squamous cell carcinoma is less frequent in children.

5. Stenosis of the nasopharynx. Nasopharyngeal stenosis can be congenital or acquired. Congenital stenosis may be incomplete choanal atresia (see Chap. 1) or a hypoplastic nasopharynx. The latter is usually associated with generalized skull deformities.

Acquired stenosis may be posttraumatic, iatrogenic, or secondary to granulomatous infection such as tuberculosis, syphilis, or diphtheria. Acquired stenosis usually has a gradual onset and can be asymptomatic until the obstruction is nearly complete. Nasal obstruction, discharge, and secondary bacterial rhinitis herald the diagnosis. On anterior rhinoscopy there
may be contraction of the posterior intranasal space. The diagnosis can be established by indirect mirror examination, transnasal fiberoptic study (giving a dynamic functional assessment), cinefluoroscopy with contrast media and CT scan. Many surgical procedures have been described for restoring patency; however, adequate functional dimensions are frequently difficult to achieve.

IV. Nasal discharge

A. Purulent rhinitis rarely occurs as a primary problem. It is associated with acute problems such as sinusitis, nasopharyngitis, nasopharyngeal obstruction, intranasal foreign body, or as a secondary bacterial infection associated with a viral URI. Resolution depends on treatment or recovery from the primary disorder. Chronic disease states (eg, cystic fibrosis) are noted for frequent exacerbations and usually are slow to respond to medical measures. (See sec. II.E.4.)

B. Acute sinusitis presents with pain, malaise, nasal obstruction, and purulent rhinorrhea. Often the onset follows a viral URI or an exacerbation of allergic rhinitis. Patients with nasal polyposis, septal deviation, allergic rhinitis, sinus ostial obstruction, or cystic fibrosis are more commonly affected. Streptococcus pneumoniae and non-typable Haemophilus influenzae remain the most common pathogens isolated. Anaerobic bacteria are found in up to 10% of cases. Staphylococcus aureus is rare, and Moraxella catarrhalis is more common in children; however, S. pneumoniae and H. influenzae predominate. Carefully performed cultures yield a viral etiology in up to 20% of cases of acute adult sinusitis.

1. Acute frontal sinusitis present with a unilateral or bilateral frontal headache. Concomitant nasal obstruction and discharge are not constant features. The sinus is tender to palpation and percussion. A Caldwell view of the skull demonstrates either an air-fluid level or opacification of the affected frontal sinus.

   a. The primary treatment of frontal sinusitis includes the administration of oral antibiotics. Although S. aureus is an uncommon sinus pathogen, should this organism be etiologic, the frontal sinus is the most likely to be affected. Amoxicillin-clavulanate 500 mg q8h is a good initial drug. A vasoconstrictor mucolytic preparation such as pseudoephedrine and guaifenesin provides symptomatic relief in conjunction with steam inhalation, warm compresses, and humidification. A short-duration of a topical nasal spray (oxymetazoline hydrochloride - 2 sprays tid for 3 days) encourages ostial patency.

   Adults allergic to the penicillin group should be started on trimethoprim-sulfamethoxazole (160/800 mg) PO bid. Those with significant arteriosclerotic cardiovascular disease are best not given pseudoephedrine.

   b. Complications. Complications of acute frontal sinusitis include osteomyelitis, meningitis, epidural abscess, subdural abscess, and brain abscess. When frontal sinusitis is associated with severe pain, with or without swelling, frontal bossing, and edema of the upper lids, hospitalization and intravenous antibiotics are necessary. In this situation, purulent material in the middle meatus should be cultured and a Gram stain made. The reliability of intranasal cultures in frontal sinus infection has not been established; however, when choosing
an antibiotic, any predominant organism on Gram stain and culture should be covered. Blood cultures should also be obtained.

Hospitalized patients should be started on ceftriaxone. If a patient is allergic to penicillin, a careful history of the reaction must be obtained. Patients who have had only a rash can be placed on cephalosporins; however, patients with a history of significant urticaria, anaphylaxis, or upper airway or laryngeal edema must not be given a cephalosporin because of the possibility of cross sensitivity. Alternatively, these patients should be placed on clindamycin.

If a positive response, evidenced by decreasing pain, temperature, and percussion tenderness, is not noted within 36-48 hours, trephination of the affected sinus should be performed.

c. Chronic frontal sinusitis is a low-grade infection. It presents with headache, intermittent rhinorrhea, and sinus tenderness. Antibiotic management should be predicated on nasal culture whenever possible. An increasing number of anaerobes are being identified with chronic sinus disease. Often, the infection resolves only after a surgical procedure is performed to establish drainage and eradicate the chronically infected mucosae.

When a mucopyocele is present, surgery is necessary. Exploration can be accomplished by an osteoplastic frontal procedure with obliteration of the sinus, by a frontoethmoidectomy with reconstruction of the nasofrontal duct or, in selected cases, by ESS.

2. Ethmoiditis usually presents with a persistent dull medial orbital sensation of pressure. There may be an associated diffuse headache, lacrimation, or lid edema. Lateral displacement of the globe suggests that the process has extended through the lamina papyracea. Sinusitis in children frequently involves the ethmoid sinuses, given that these sinuses are partially developed at birth.

On examination, pus can be seen emanating from the middle or superior meatus. Radiographs show opacification of the affected sinus. Primary therapy is similar to that outlined for acute frontal sinusitis (see 1.a.). However, amoxicillin is the initial antibiotic to consider.

a. Complications of ethmoiditis include orbital cellulitis, orbital abscess, and intracranial extension.

(1) Orbital cellulitis is suspect when purulent rhinorrhea antedates the rapid onset of periorbital erythema, conjunctival engorgement, and associated pain and toxicity, including an elevated temperature. When present or suspect, cultures must be taken and IV antibiotic therapy initiated as part of a hospital management regimen. Chloramphenicol is often used in combination with ampicillin, in appropriate dosage, as the initial form of medical management until culture specific antibiotics can be started. Ceftriaxone is an excellent alternative.

Both oral and topical decongestants should be used in a cooperative adult patient. The nose is packed with cotton pledgets, saturated with a 4% cocaine solution, providing a
measure of pain relief in addition to vasoconstriction. Ophthalmologic consultation should be considered immediately, to assess the presence or absence of spontaneous venous pulsations. Visual acuity must be assessed and frequently monitored for signs of deterioration.

(2) Orbital abscess presents much the same clinical picture as orbital cellulitis, but exophthalmos and orbital fixation are more common.

(3) Surgical management. Evidence of deteriorating vision or a failure of a favorable response to IV antibiotic therapy within 48 hours mandate surgical drainage. The documentation of an abscess with CT scan mandates drainage acutely via an external ethmoidectomy approach.

b. Chronic ethmoiditis occurs alone, in association with nasal polyps, or with isolated allergic diathesis. If there is no response to antibiotic therapy, ethmoidectomy is required. Endoscopic sinus surgery has become the procedure of choice because of the precise nature of this technique, which encourages normal tissue preservation. A trial of antibiotic therapy following appropriate cultures is warranted prior to surgical intervention.

3. Maxillary sinusitis

a. Signs and symptoms. The maxillary antra are the most frequently infected sinuses in the adult. Infection often presents with dental pain, pressure over the maxilla, nasal obstruction, and purulent rhinorrhea. The affected sinus is tender to palpation, and purulent discharge may emanate from the middle meatus. The infection can occur as a sequela of a periapical infection of an upper tooth. Affected teeth are usually tender to percussion.

b. Diagnosis. In most instances the diagnosis can be made on the clinical information available, and therapy is instituted. X rays in this setting need not be ordered before initiating therapy. Three weeks after the onset of treatment, a follow up film will provide a base line for future use and a test of the completeness of resolution. In questionable cases, x rays should be ordered when the symptoms first present. A Waters' projection is the most informative plain view, often demonstrating a thickened lining, an air-fluid level, or total opacification.

c. Therapy

(1) Acute. Assuming patient compliance, proper medical management almost always effects resolution of the uncomplicated acute process.

(a) Antibiotics. The proper choice of antibiotics reflects a knowledge of the most common organisms, as noted above (IV.B.). Amoxillin or TMP-SMZ are two excellent choices. A minimum of 14 days of treatment is essential, and frequently 21 days are necessary. Close patient observation is mandatory to ensure complete resolution.

(b) Rest. For the first 48 hours rest is important.
(c) **Vasoconstrictors.** Early drainage speeds resolution. A topical nasal vasoconstrictor spray used tid for a maximum of 5 days should be prescribed. Vasoconstrictor preparations may be beneficial, and their use appears to be warranted for patients readily tolerating them.

(d) **Heat.** Moist heat also seems beneficial when applied over the infected sinus.

(2) **Ancillary measures**

(a) **Culture.** A nasal culture does not necessarily reflect the organisms present in the sinus. It is reserved for cases not responding to initial therapy and should be combined with culture data from the involved antra. Aspiration of the sinus contents provides material for staining and culture.

(b) **Antral puncture.** Often, gentle irritation with saline can speed resolution in selected cases. Aspiration and irrigation are procedures best performed by an experienced physician. Although exceedingly rare in competent hands, injury to the globe, air embolus, bleeding, and osteomyelitis are potential complications of antral lavage.

**d. Chronic maxillary sinusitis** develops after prior acute episodes of infection with subsequent irreversible mucosal change, or OMC obstruction. Chronic rhinorrhea, halitosis, and occasionally mild pharyngitis, secondary to the persistent posterior nasal discharge, are the most consistent diagnostic features. Cultures of the infected sinus frequently grow anaerobes, and antibiotic therapy must cover these organisms. With chronic disease the antibiotic response is limited, and judicious surgery is often necessary.

4. **Sphenoiditis** rarely occurs as an isolated sinus infection. Acute sphenoiditis can present with severe retroorbital pain, and frequently the pain is also referred to the vertex and basiocciput. The diagnosis is confirmed radiographically when clouding or opacification of the sinus is evidenced on lateral and submental vertex skull films.

**Therapy.** All patients with obstructive sphenoid sinusitis should be hospitalized and placed on antibiotics as for complications of frontal sinusitis (see 1.b.). Any purulent drainage in the sphenoid recess should be Gram stained and cultured. Blood cultures should be obtained, and 4% cocaine-impregnated strips should be placed in the nose for 10 minutes q8h until drainage is achieved. Oral decongestants such as pseudoephedrine are indicated (60 mg q6-8h). A bedside humidifier should be considered. If there is no response to the medical regimen within 36 hours, a sphenoidotomy is performed. Impending complications mandate drainage.

5. **Sinus ostial closure** can occur from secondary scarring induced by infection or iatrogenically from repeated cannulation of the ostium. Stenosis decreases effective drainage as well as aeration, predisposing the sinus to infection. Surgical intervention should be directed whenever possible toward establishing drainage. Frontal ablation is reserved for the most refractory cases or those with major associated complications.

6. **Barosinusitis** refers to paranasal sinus symptomatology due to a pressure differential between the sinus cavity and the environment. This condition most frequently
Symptoms range from a sensation of fullness over the affected sinus to local excruciating pain. The ideal therapy is to return the patient to the initial altitude, slowly repeating the descent. Oral decongestant preparations and topical vasoconstrictors are indicated. Only with associated infection are antibiotics warranted.

7. Immunosuppression in patients with AIDS or receiving chemotherapy may predispose to infections from a number of uncommon bacterial or fungal pathogens. Sinus aspiration must be performed early in the course of the disease so that appropriate therapy is instituted. Surgical drainage may be required as initial treatment, because antimicrobial agents previously discussed may be ineffective. Evidence is emerging that MRI is useful in identifying mycotic infections of the paranasal sinuses.

C. CSF rhinorrhea may occur spontaneously or after an episode of trauma. Frontoethmoid fractures can be complicated by a CSF leak. Watery or blood-tinged nasal discharge should be analyzed for glucose. A level of 40-100 mg/dL is diagnostic for a CSF leak. Dipstick and tablet-type analysis of glucose levels are not reliable.

Unless the rhinorrhea is profuse, the initial management is conservative. Hospitalization and the consideration of IV antibiotics are warranted. A semisitting position is beneficial, and the patient is instructed to abstain from nasal blowing and straining. Most traumatic CSF leaks resolve without intervention.

1. Complications. Undiagnosed CSF rhinorrhea can lead to recurrent meningitis.

2. The site of the leak must be located. Topical vasoconstriction in the office setting and subsequent endoscopic evaluation can disclose the site of leakage. For cases in which a diagnosis cannot be made by this method, fluorescein and radioactive indium are adjunctive diagnostic measures.

Technique. Three cotton pledgets are placed in each nasal chamber - one in the sphenoethmoid recess, a second in the cribiform region, and a third in the middle meatus. Each pledget is appropriately labeled prior to placement. A spinal tap is performed, with the removal of 10 mL of CSF. Next, 0.5 mL of 5% fluorescein is mixed with 10 mL of CSF, and the mixture is slowly injected intrathecally. It is important to use fluorescein approved for intravenous injection and not topical fluorescein. In addition, 500 microCi of indium is injected intrathecally. Between 2 and 4 hours postinjection the patient is scanned. Frequently, the site of the leak can be identified on this scan. The cotton pledgets are removed and each examined under a Wood's lamp for fluorescence. Subsequently, the pledgets are evaluated for their radioactivity. An increased pledget count is considered a positive test result, and the location of the pledget is compared to the scan. Preoperative identification of the leak helps to define the appropriate corrective surgical procedure.

V. Olfaction. The olfactory cells of the first cranial (olfactory) nerve pass into the superior nasal fossa through the cribiform plate. Synapsing, second-order neurons enter the rhinencephalon where the olfactory stimulus is processed. All patients complaining of
olfactory dysfunction should have a complete otorhinolaryngologic and neurologic examination. In patients complaining of dysosmia (alteration in smell), it is important to rule out sources such as bronchiectasis, caries, or sinusitis.

The initial evaluation can be performed using coffee, chocolate, lemon oil, tobacco, ammonia, menthol, and acetone. The last three are strong trigeminal stimulants. Malingering should be suspected whenever a patient cannot "smell" these. Commercial tests for olfaction are also available and may be more reliable.

CT scans are appropriate if a symptom or sign other than anosmia can be found.

A. Etiology. Olfactory aberrations can be related to nasal obstruction, congenital dysfunction, trauma, infection, neoplasms, or environmental pollutants. Hyposmia is seen as part of the aging process. As many as 80% of individuals over the age of 60 years will note some decrease in the sense of smell awareness. Investigations have linked the early onset of anosmia with the subsequent development of Alzheimer's disease.

1. Nasal airway obstruction due to any of the factors previously discussed can influence olfactory function. Opinions differ as to whether septal deviation can affect the sense of smell. Relief of anosmia, secondary either to nasal polyposis or to allergic rhinitis, has been reported with steroids.

2. Complete congenital anosmia is rare and has been reported as an isolated familial disorder. It occurs in Kallmann's syndrome (agenesis of olfactory lobes and secondary hypogonadism due to lack of gonadotropins). Many patients have a specific anosmia for a limited number of odors; however, this is usually asymptomatic.

3. Trauma. Anosmia can occur after head trauma. Most commonly, it is related to occipital or frontal blows. Unilateral anosmia can be present in conjunction with CSF rhinorrhea following head trauma.

4. Infection. Viral upper respiratory tract infections can alter the sense of smell directly by affecting the olfactory cell or secondarily by effecting nasal airway obstruction. Anosmia is especially frequent following influenza. Postinfluenza patients may continue to have anosmia, and others have transient parosmia as olfaction returns.

5. Neoplasia. Neoplasms known to cause altered olfaction include meningiomas of the dura of the cribriform plate, tumors of the third ventricle, frontal lobe glioma, sphenoidal ridge meningioma, temporal lobe tumors, and suprasellar meningioma.

6. Pollutants. Industrial exposure to benzene, ethyl acetate, formaldehyde, menthol, pain solvents, oil of peppermint, butyl acetate, carbon disulfide, and trichloroethylene has been reported to cause anosmia or hyposmia.

7. Olfactory disturbances can occur with psychiatric disorders such as schizophrenia, hysteria, confusional states, and depression.
B. Therapy is directed toward removing the causative agent or toward correcting the underlying disturbance. Therapy for postviral or idiopathic smell disorders is limited. The use of zinc sulfate has been occasionally successful. Vitamin A as a therapy is currently being investigated.

VI. Facial pain has multiple etiologies, including tumors, neuralgia, inflammation of the paranasal sinuses, or vascular occlusion. All patients with facial pain must have a complete otolaryngologic and neurologic evaluation.

A. Sinus pain. Maxillary sinus pain usually presents with a sensation of pressure over the sinus. As the pain intensifies, it involves all areas abutting the sinus walls, including the ipsilateral orbit. Ethmoid sinusitis produces orbital pain and pain between the eyes, which can extend to the temporal region. Frontal sinus pain is usually localized over the affected sinus. Sphenoid sinusitis causes severe pain behind the eyes, which can radiate to the parietal or occipital regions (see sec IV.B.4.).

B. Neuralgia is typified by paroxysms of lancinating pain of a few seconds' to a few minutes' duration. The paroxysm is usually initiated by any stimulus to a trigger point or region.

1. Trigeminal neuralgia (tic douloureux). The brief, lancinating, paroxysmal pain of trigeminal neuralgia is confined to the branches of the fifth cranial nerve. The maxillary division is most frequently affected, followed in frequency by the mandibular. On sensory testing the nerve is found to be intact. Facial trigger points are frequently found, with symptoms brought on by chewing, yawning, swallowing, shaving, etc. Most patients are over the age of 40 years, with women affected more often than men. Rarely, further investigation of trigeminal neuralgia with associated hypesthesia in the distribution of the facial nerve, other cranial neuropathies, and with onset before age 40 years may disclose multiple sclerosis or a posterior fossa tumor.

   Therapy. Oral carbamazepine and phenytoin have each been used with limited success. The usual adult dose of phenytoin is 100 mg tid. Phenytoin can have detrimental effects on the endocrine, nervous, integumentary, gastrointestinal, cardiac, and hemopoietic systems. It frequently causes gingival hyperplasia.

   Carbamazepine should be prescribed only by a physician who will be following the patient closely. The initial adult dose is 200 mg PO qid. Among the more significant side effects are aplastic anemia, agranulocytosis, jaundice, Stevens-Johnson syndrome, congestive heart failure, and cardiovascular collapse. Baclofen may be used alone or in conjunction with phenytoin or carbamazepine. The initial dosage is 5-10 mg tid, with gradual increase to 20 mg qid as needed.

2. Glossopharyngeal neuralgia begins with severe pain, starting in the lower pharynx, tonsil, or base of the tongue. It can radiate to the ipsilateral ear, mandible, and teeth. The pain is unilateral and so intense that patients will not speak during an episode. The trigger point is often in the base of the tongue or tonsillar region. In some individuals, the pain is initiated by deglutition. The diagnosis is made if local anesthesia to the trigger point provides temporary relief of the pain. Once recognized, nerve section can provide relief.
3. **Sphenopalatine neuralgia** presents with lower facial pain radiating to the orbit, temple, forehead, and upper cervical region. It is accompanied by ipsilateral lacrimation, conjunctivitis, rhinorrhea, and nasal congestion. If local anesthesia to the sphenopalatine ganglion provides symptomatic relief, the diagnosis is confirmed. Local anesthesia can be accomplished by topical placement of a cocaine-soaked cotton pledget in the posterior nasal chamber.

4. **Atypical facial neuralgia** is a unilateral aching pain of hours’ or days’ duration that is not limited to a specific nerve distribution. It often spreads over the cervical root distribution and can involve the nose, eye, cheek, ear, neck, and shoulder. Attacks have been precipitated by fatigue, tooth extractions, tension, and anxiety. Associated autonomic symptoms can include pallor, diaphoresis, lacrimation, and rhinitis. This pain is refractory to section of the trigeminal nerve, local anesthesia to the sphenopalatine ganglion, and resection of the superior cervical sympathetic ganglion. The etiology of atypical facial neuralgia is unknown, and multiple causation is likely. Patients presenting with these symptoms should be evaluated to rule out local pathology as well as any depressive symptoms. Treatment includes phenytoin and carbamazepine.

C. **Carotidynia** is pain that comes from the carotid artery and radiates to the ipsilateral eye, ear, and malar region. The episodes are periodic, and there are no associated visual disturbances. On examination, the common carotid is tender to palpation. The pain usually resolves within 2 weeks. Symptomatic relief is often provided by aspirin, or nonsteroidal anti-inflammatory agents. Occasionally, ergotamine can diminish the pain.

D. **Migraine headaches** may start in childhood, adolescence, or early adulthood. An aura precedes the pain, and the prodromes include scintillating scotomas, flashing lights, hemianopsia, paresthesias, and hemiparesis. The pain is localized to one part of the head, usually unilateral, and can vary in intensity from mild discomfort to severe throbbing pain. There is often associated photophobia, nausea, emesis, altered taste, or smell. Migraine headaches can last from a few hours to 3 days. Females are more frequently affected. Basilar artery migraine can produce occipital head pain and is often accompanied by episodes of vertigo that recur with the headache.

1. **Therapy**

   a. For an **acute attack**, a combination of ergotamine tartrate and caffeine often provides relief. This medication can be prescribed as a tablet or suppository. Initial adult PO dosage is 2 mg ergotamine tartrate and 200 mg of caffeine taken during the onset of the attack. If necessary, an additional 1 mg ergotamine and 100 mg caffeine can be taken PO one-half hour after the initial dose for further relief of symptoms. This medication is contraindicated with arteriosclerotic cardiovascular disease, hypertension, impaired hepatic function, impaired renal function, or pregnancy.

   b. **Prophylaxis.** A 50% reduction in symptoms should be considered successful management. Prophylaxis should not be continued beyond 1 year. Multiple medications have shown efficacy for prevention, but methysergide and propranolol are time-tested and remain appropriate.
(1) **Methysergide** has been used for long-term prophylaxis of migraine; it is not effective for acute attacks. The standard dosage is 2 mg, 2 to 4 times daily. Methysergide should be prescribed only by a physician who will be following the patient closely, since it has serious adverse effects including retroperitoneal fibrosis and fibrosis of cardiac valves. Contraindications include pregnancy, any form of arteriosclerotic cardiovascular disease, pulmonary disease, impaired hepatic or renal function, valvular heart disease, serious infection, and phlebitis or cellulitis of the lower limbs.

(2) **Propranolol** can be used for prevention of the migraine attack. The initial dose is 20 mg PO qid. This dose can be gradually increased to 40-60 mg qid. The patient's blood pressure and pulse must be monitored while adjusting the propranolol dose. It is contraindicated in patients with bronchial asthma, congestive heart failure, and sinus bradycardia. Abrupt withdrawal of propranolol in patients with angina pectoris has resulted in myocardial infarction.

E. Cluster headache (Horton's cephalgia)

1. **Signs and symptoms.** Cluster headache is idiopathic, excruciating, knifelike pain that involves the ipsilateral temple, forehead, face, head, and neck but spares the lips and tongue. There is no preceding aura; it lasts from 10 minutes to several hours. The attacks can recur several times within 24 hours; they often repeat daily for several weeks, followed by a prolonged symptom-free interval. Males are affected 4-6 times as often as females. The first attack usually occurs between the ages of 20 and 35.

2. **Therapy**

   a. **Acute.** Ergotamine tartrate and caffeine can be used for relief of the pain. The total maximum weekly dose, however, is 10 mg of ergotamine. Therefore, patients should take only 1-2 mg of ergotamine initially. Some patients require injection of ergotamine tartrate, since they do not benefit from the oral medication. These patients require 0.25 mg IV qid. The contraindications for this medication have been discussed (see 1.a.).

   b. **Prophylaxis.** Methysergide calcium channel blocking agents are both effective (see 1.b.). Prophylactic lithium has been given when the attacks begin, with doses necessary to obtain a blood level of 0.6-1.2 mEq/L should be used. Methysergide is contraindicated in pregnancy, renal disease, cardiovascular disease, and in patients on diuretics. There are several serious reactions, including an encephalopathic syndrome, hypothyroidism, seizures, peripheral circulatory collapse, arrhythmias, stupor, and coma. Serum levels must be monitored and should be drawn 8-12 hours after the oral dose is taken.

F. **Tension headaches** commonly start in the occipital region and spread in a band distribution around the head. Patients often describe a viselike sensation over the entire head. These headaches can be intermittent or can persist throughout a 24-hour period with varying intensity. Muscle relaxants are occasionally of value, along with an analgesic.

G. **Glaucoma** can present with an acute, unilateral, deep penetrating pain or with a chronic, dull pain. All patients presenting with a headache should be evaluated to rule out glaucoma.
H. Temporal arteritis is an illness of patients over 50 years of age and is characterized by a severe inflammatory reaction around the vessels and with multinucleated giant cells in the media. It is similar to periarteritis nodosa.

1. Signs and symptoms include pain in the distribution of the temporal artery, along with systemic symptoms of lethargy, low-grade fever, and weight loss. Visual loss can occur when the central retinal artery is involved.

2. Diagnosis is made with an elevated erythrocyte sedimentation rate (ESR) of 60-120 mm/hour. Biopsy of the artery confirms the diagnosis; however, angiography may be needed in some cases.

3. Management is with corticosteroids at high doses (prednisone 45 mg/day) particularly if vision is affected. Steroids frequently can be discontinued after 6 months, because temporal arteritis is a self-limiting disease.

I. Other causes of facial pain include temporomandibular joint syndrome and muscular spasm.
Appendix: Antihistamine Usage

Class
- Generic examples
- Trade name
- Oral dosage - pediatric
- Oral dosage - adult
- Characteristics

Ethylendiamines
* - Pyrilamine maleate
  - Triaminic preparations
  - Infant 16 mg qid / 2 lbs
  - 75-200 mg/24 hr in divided doses
  - Low sedative and anticholinergic effects; gastrointestinal complaints common
  (reduced by giving drug with meals).

Ethanolamines
* - Clemastine fumarate
  - Tavist preparations
  - 6-12 yr, 1-2 tsp bid
  - 1.34 mg bid - 2.68 mg (max 8.04 mg/24 hr)
  - Drowsiness most frequent complaint but no marked sedation; anticholinergic effects
  weak and gastrointestinal complains uncommon.
* - Diphenhydramine hydrochloride
  - Benadryl
  6-12 yr, 5 mg/kg/24 hr, in 4 divided doses
  - 25-100 mg q 6-8h
  - Sedative effects high; anticholinergic effects moderate; gastrointestinal complaints
  uncommon.

Alkylamines
* - Brompheniramine maleate
  - Dimetane; Dimetapp
  - 0.5 mg/kg/24 hr in 4-6 divided doses (max 6 mg/24 hr for ages 2-6 yr; 12 mg/24 hr
  for ages 6-12 yr)
  - 4 mg q4-6h (max 24 mg/24 hr), or 8-12 mg q8-12h using time-released form
  - Low sedative, anticholinergic and gastrointestinal effects; approximately 75% of
  over-the-counter preparations contain an alkylamine antihistamine.
* - Chlorpheniramine maleate
  - Chlor-Trimeton; Novahistine; Ornade; Numerous over-the-counter preparations
  - 0.35 mg/kg/24 hr in 4 divided doses. Over 7 yr, 8 mg q12h (time-released)
  - 2-4 mg q6-8h, as time-released form, 8-12 mg q 12h
  - As above.
Piperazines

* - Hydroxyzine hydrochloride
  - Atarax
  - 2-5 mg/kg/24 hr in 3 divided doses
  - 25-100 mg q 6-8h
  - Drowsiness and dry mouth common; used to treat pruritus.

* - Meclizine hydrochloride
  - Antivert
  - Not recommended < 12 yr
  - 25-100 mg/24 hr in 3-4 divided doses
  - As above; used primarily for motion sickness and vertigo.

Phenothiazines

* - Promethazine hydrochloride
  - Phenergan
  - 0.5 mg/kg/dose q6-8h
  - 25 mg at bedtime or 12.5 mg qid
  - Marked sedative effect; used primarily as sedative and antiemetic.

Piperidines

* - Azatadine maleate
  - Trinaline
  - Dosage not established < 12 yr
  - 1-2 mg bid
  - Drowsiness most common side effect; chemically similar to cyproheptadine.

* - Cyproheptadine hydrochloride
  - Periactin
  - 2-6 yr, 2 mg q 8-12h (max 12 mg/24 hr)
  - 4-20 mg/day in divided doses (max 0.5 mg/kg/24 hr)
  - Drowsiness most common side effect; weight gain common; useful for pruritus and especially for cold urticaria; do not use in newborn or premature infants.

Nonsedating anti-histamines

* - Terfenadine
  - Seldane
  - Not recommended < 12 yr
  - 60 mg bid
  - Well accepted; rapid onset nonsedating; negligible side effects.

* - Astemizole
  - Hismanal
  - Not recommended < 12 yr
  - 10 mg qid
  - Well accepted; half life 1 day; diminishes response to skin testing for up to two weeks.