from GABHS. To balance timeliness of diagnosis, cost, and accuracy, one strategy in children is to do a rapid strep screen in the office, treat if positive, and send a formal culture if negative. In adults, because other bacterial pathogens may be involved, throat culture for all bacterial pathogens is appropriate for those meeting clinical criteria described previously.

Testing for mononucleosis, gonorrhea, or HIV is done only when clinically suspected.

Treatment

Specific conditions are treated. Those with severe symptoms of tonsillopharyngitis may be started on a broad-spectrum antibiotic (eg, amoxicillin/clavulanate) pending culture results.

Symptomatic treatments such as warm salt-water gargles and topical anesthetics (eg, benzocaine, lidocaine, dyclonine) may help temporarily relieve pain in tonsillopharyngitis. Patients in severe pain (even from tonsillopharyngitis) may require short-term use of opioids.

Key Points

- Most sore throats are caused by viral tonsillopharyngitis.
- It is difficult to clinically distinguish viral from bacterial causes of tonsillopharyngitis.
- Abscess and epiglottitis are rare but serious causes.
- Severe sore throat in a patient with a normal-appearing pharynx should raise suspicion of epiglottitis.

SMELL AND TASTE ABNORMALITIES

Because distinct flavors depend on aromas to stimulate the olfactory chemoreceptors, smell and taste are physiologically interdependent. Dysfunction of one often disturbs the other. Disorders of smell and taste are rarely incapacitating or life threatening, so they often do not receive close medical attention, although their effect on quality of life can be severe.

Taste: Although abnormal taste sensations may be due to mental disorders, local causes should always be sought. Glossopharyngeal and facial nerve integrity can be determined by testing taste on both sides of the dorsum of the tongue with sugar, salt, vinegar (acid), and quinine (bitter).

Drying of the oral mucosa caused by heavy smoking, Sjögren's syndrome, radiation therapy of the head and neck, or desquamation of the tongue can impair taste, and various drugs (eg, those with anticholinergic properties and vincristine) alter taste. In all instances, the gustatory receptors are diffusely involved. When limited to one side of the tongue (eg, in Bell's palsy), ageusia (loss of the sense of taste) is rarely noticed.

Smell: The inability to detect certain odors, such as gas or smoke, may be dangerous, and several systemic and intracranial disorders should be excluded before dismissing symptoms as harmless. Whether brain stem disease (involvement of the nucleus solitarius) can cause disorders of smell and taste is uncertain, because other neurologic manifestations usually take precedence.

Anosmia (loss of the sense of smell) is probably the most common abnormality. Hyperosmia (increased sensitivity to odors) usually reflects a neurotic or histrionic personality but can occur intermittently with seizure disorders. Dysosmia (disagreeable or distorted sense of smell) may occur with infection of the nasal sinuses, partial damage to the olfactory bulbs, or mental depression. Some cases, accompanied by a disagreeable taste, result from poor dental hygiene. Uncinate epilepsy can produce brief, vivid, unpleasant olfactory hallucinations. Hyposmia (diminished sense of smell) and hypogeusia (diminished sense of taste) can follow acute influenza, usually temporarily.

Rarely, idiopathic dysgeusia (distorted sense of taste), hypogeusia, and dysosmia respond to zinc supplementation.

ANOSMIA

Anosmia is complete loss of smell. Hyposmia is partial loss of smell. Most patients with anosmia have normal perception of salty, sweet, sour, and bitter substances but lack flavor discrimination, which largely depends on olfaction. Therefore, they often complain of losing the sense of taste (ageusia) and of not enjoying food. If unilateral, anosmia is often unrecognized.

Etiology

Anosmia occurs when intranasal swelling or other obstruction prevents odors from gaining access to the olfactory area; when the olfactory neuroepithelium is destroyed; or when the olfactory nerve fila, bulbs, tracts, or central connections are destroyed (see Table 51–4).

Major causes include

- Head trauma (young adults)
- Viral infections and Alzheimer's disease (older adults)

Table 51–4. SOME CAUSES OF ANOSMIA

CAUSE	SUGGESTIVE FINDINGS	DIAGNOSTIC APPROACH
Intranasal obstruction		
Allergic rhinitis	History of chronic allergic symptoms (eg, congestion, clear rhinorrhea), no pain	Clinical evaluation
Nasal polyps	Polyps usually visible on examination	Clinical evaluation
Destruction of olfactory neuroepithelium		
Atrophic rhinitis	Chronic rhinitis with atrophic and sclerotic mucous membranes, patency of nasal passages, crust formation, foul odor	Clinical evaluation Sometimes biopsy, which shows the normal ciliated columnar epithelium converted to stratified squamous and the lamina propria reduced in amount and vascularity
Chronic sinusitis	Chronic mucopurulent drainage, documented infections	Clinical evaluation CT Panograph, which shows apices of maxillary teeth to rule out tooth abscess
Some viral URIs	Onset after clinical infection	Clinical evaluation
Tumors (rare cause)	Possibly visual difficulty or only anosmia	CT MRI
Drugs (eg, amphetamines, enalapril, estrogen, naphazoline, phenothiazines, reserpine; prolonged use of decongestants)	Usually, an apparent history of exposure	Clinical evaluation
Toxins (eg, cadmium, manganese)	Usually, an apparent history of exposure	Clinical evaluation
Destruction of central pathways		
Alzheimer's disease	Progressive confusion and loss of recent memory	MRI Sequential memory tests
Head trauma	Apparent by history	CT
Intracranial surgery, infection, tumor	Surgery and CNS infection apparent by history Tumors with or without other neurologic symptoms	CT or MRI

Prior URI, especially influenza infection, is implicated in 14 to 26% of all presenting cases of hyposmia or anosmia.

Drugs can contribute to anosmia in susceptible patients. Other causes include prior head and neck radiation, recent nasal or sinus surgery, nasal and brain tumors, and toxins. The role of tobacco is uncertain.

Evaluation

History: History of present illness should assess the time course of symptoms and their relation to any URI or head injury. Important associated symptoms are nasal congestion, rhinorrhea, or both. The nature of rhinorrhea should be assessed (eg, watery, mucoid, purulent, bloody).

Review of systems should assess neurologic symptoms, particularly those involving mental status (eg, difficulty with recent memory) and cranial nerves (eg, diplopia, difficulty speaking or swallowing, tinnitus, vertigo).

Past medical history should include history of sinus disorders, cranial trauma or surgery, allergies, drugs used, and exposure to chemicals or fumes.

Physical examination: The nasal passages should be inspected for swelling, inflammation, discharge, and polyps. Having the patient breathe through each nostril sequentially (while the other is manually occluded) may help identify obstruction.

A complete neurologic examination, particularly of mental status and cranial nerves, is done.

Red flags: The following findings are of particular concern:

- Previous head injury
- Neurologic symptoms or signs
- Sudden onset

Interpretation of findings: Sudden onset after significant head trauma or toxin exposure strongly implicates that event as the cause.

A history of chronic rhinosinusitis is suggestive, particularly when significant congestion, polyps, or both are visible on examination. However, because these findings are common in the population, the physician should be wary of missing another disorder. Progressive confusion and recent memory loss in an elderly patient suggest Alzheimer's disease as a cause. Waxing and waning neurologic symptoms affecting multiple areas suggest a neurodegenerative disease such as multiple sclerosis. Slowly progressive anosmia in an elderly patient with no other symptoms or findings suggests normal aging as the cause.

Testing: An in-office test of olfaction can help confirm olfactory dysfunction. Commonly, one nostril is pressed shut, and a pungent odor such as from a vial containing coffee, cinnamon, or tobacco is placed under the open nostril; if the patient can identify the substance, olfaction is presumed intact. The test is repeated on the other nostril to determine whether the response is bilateral. Unfortunately, the test is crude and unreliable.

If anosmia is present and no cause is readily apparent on clinical evaluation (see Table 51–4), patients should have CT of the head (including sinuses) with contrast to rule out a tumor or unsuspected fracture of the floor of the anterior cranial fossa. MRI is also used to evaluate intracranial disease and may be needed as well, particularly in those patients with no nasal or sinus pathology on CT.

A psychophysical assessment of odor and taste identification and threshold detection is done as well. This assessment commonly involves use of one or several commercially available testing kits. One kit uses a scratch-and-sniff battery of odors, whereas another kit involves sequential dilutions of an odorous chemical.

Treatment

Specific causes are treated, although smell does not always recover even after successful treatment of sinusitis.

There are no treatments for anosmia. Patients who retain some sense of smell may find adding concentrated flavoring agents to food improves their enjoyment of eating. Smoke alarms, important in all homes, are even more essential for patients with anosmia. Patients should be cautioned about consumption of stored food and use of natural gas for cooking or heating, because they have difficulty detecting food spoilage or gas leaks.

Geriatrics Essentials

There is a significant loss of olfactory receptor neurons with normal aging, leading to a marked diminution of the sense of smell. Changes are usually noticeable by age 60 and can be marked after age 70.

Key Points

- Anosmia may be part of normal aging.
- Common causes include URI, sinusitis, and head trauma.
- Cranial imaging is typically required unless the cause is obvious.

52 Oral and Pharyngeal Disorders

Oral and pharyngeal disorders include adenoid disorders, epiglottitis, parapharyngeal abscess, peritonsillar abscess and cellulitis, retropharyngeal abscess, salivary stones, sialadenitis, submandibular space infection, tonsillopharyngitis, Tornwaldt's cyst, and velopharyngeal insufficiency. Oral, pharyngeal, and salivary gland tumors are discussed in Ch. 55.

SIALADENITIS

Sialadenitis is bacterial infection of a salivary gland, usually due to an obstructing stone or gland hyposecretion. Symptoms are swelling, pain, redness, and tenderness. Diagnosis is clinical. CT, ultrasound, and MRI may help identify the cause. Treatment is with antibiotics.

Etiology

Sialadenitis usually occurs after hyposecretion or duct obstruction but may develop without an obvious cause. The major salivary glands are the parotid, submandibular, and sublingual glands. Sialadenitis is most common in the parotid gland and typically occurs in patients in their 50s and 60s, in chronically ill patients with xerostomia, in those with Sjögren's syndrome, and in those who have had radiation therapy to the oral cavity. Teenagers and young adults with anorexia are also prone to this disorder. The most common causative organism is *Staphylococcus aureus*; others include streptococci, coliforms, and various anaerobic bacteria.

Symptoms and Signs

Fever, chills, and unilateral pain and swelling develop. The gland is firm and diffusely tender, with erythema and edema of the overlying skin. Pus can often be expressed from the duct by compressing the affected gland and should be cultured. Focal enlargement may indicate an abscess.

Diagnosis

CT, ultrasound, and MRI can confirm sialadenitis or abscess that is not obvious clinically, although MRI may miss an obstructing stone.

Treatment

- Antistaphylococcal antibiotics
- Local measures (eg, sialagogues, warm compresses)

Initial treatment is with antibiotics active against *S. aureus* (eg, dicloxacillin, 250 mg po qid, a 1st-generation cephalosporin, or clindamycin), modified according to culture results. With the increasing prevalence of methicillin-resistant *S. aureus*, especially among the elderly living in extended-care nursing facilities, vancomycin is often required. Hydration, sialagogues (eg, lemon juice, hard candy, or some other substance that triggers saliva flow), warm compresses, gland massage, and good oral hygiene are also important. Abscesses require drainage. Occasionally, a superficial parotidectomy or submandibular gland excision is indicated for patients with chronic or relapsing sialadenitis.

OTHER SALIVARY GLAND INFECTIONS

Mumps often cause parotid swelling (see Table 155–1 on p. 1462). Patients with HIV infection often have parotid enlargement secondary to one or more lymphoepithelial cysts. Cat-scratch disease caused by *Bartonella* infection often invades periparotid lymph nodes and may infect the parotid glands by contiguous spread. Although cat-scratch disease is self-limited, antibiotic therapy is often provided, and incision and drainage are necessary if an abscess develops.

Atypical mycobacterial infections in the tonsils or teeth may spread contiguously to the major salivary glands. The PPD may be negative, and the diagnosis may require biopsy and tissue culture for acid-fast bacteria. Treatment recommendations are controversial. Options include surgical debridement with curettage, complete excision of the infected tissue, and use of anti-TB drug therapy (rarely necessary).

SALIVARY STONES

(Sialolithiasis)

Stones composed of Ca salts often obstruct salivary glands, causing pain, swelling, and sometimes infection. Diagnosis is made clinically or with CT, ultrasonography, or sialography. Treatment involves stone expression with saliva stimulants, manual manipulation, a probe, or surgery.