Maxillofacial Disorders

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KEY POINTS

- Temporomandibular joint dislocation is usually readily reduced in the emergency department after the patient has been pretreated with analgesic and antispasmodic agents.
- Epistaxis may be the initial complaint of a patient with a more serious systemic illness, such as a clotting disorder.
- When visible blood loss from the nasopharynx has been stopped, the clinician should examine the posterior oropharynx for ongoing occult blood loss.
- Posterior epistaxis accounts for about 10% of nasal hemorrhages and can result in large volumes of blood loss.
- Any abnormal neurologic or ocular physical findings in a patient with rhinosinusitis mandate further investigation to assess for central nervous system extension of the disease.

TEMPOROMANDIBULAR DISORDERS

EPIDEMIOLOGY

The temporomandibular articulations are unique within the body in that they are bilateral joints that are nearly continuously in use. Consequently, the temporomandibular joint (TMJ) is subject to both pain and dislocation. Discomfort of the TMJ was previously referred to as TMJ pain dysfunction syndrome. However, because it was realized that more than just the actual joint can be the source of a patient’s discomfort, the term has evolved to *temporomandibular disorder* (TMD). TMD is defined as craniofacial pain that involves the TMJ, masticatory muscles, and associated head and neck musculoskeletal structures. It is roughly estimated that more than 10 million people in the United States alone have symptomatic TMD. Most of those affected are women. TMJ dislocation is an uncommon disorder, with one case series reporting 37 occurrences in 700,000 patient visits.

PATHOPHYSIOLOGY

TMD is probably due to excessive strain on the muscles of mastication with resultant strain on the capsular ligaments of the TMJ. The result is that the mandibular condyle does not articulate properly in its joint. The patient feels pain and senses an occlusal disturbance.

Patients with TMJ dislocation are unable to close their mouth. With normal function, when the mandible is open, the mandibular condyle moves anteriorly and inferiorly. When the mandible closes, the condyle moves posteriorly and superiorly and returns to its original location (Fig. 30.1). TMJ dislocation results when the mandibular condyle moves anterior to the temporal eminence (the anterior portion of the mandibular fossa) (see Fig. 30.1). Once the dislocation occurs, the muscles of mastication spasm, which results in trismus and inability of the patient to return the mandibular condyle to its anatomic position. The dislocation usually results from excessive opening of the mouth, such as occurs with yawning or laughing. TMJ dislocation can also be the result of trauma, seizure, or dystonic drug reactions.

PRESENTING SIGNS AND SYMPTOMS

Unilateral pain in the region of the TMJ and clicking or crepitation that is exacerbated by chewing are the classic complaints of a patient with TMD (Box 30.1). The dull or throbbing pain is localized to the preauricular region or to the muscles of mastication and typically worsens with movement of the mandible, such as when eating or talking. Pain may be most severe in the morning if bruxism is an issue. If a click is present, the patient hears it when jaw opening is initiated. The pain may also radiate to the neck, ears, mandible, or temporal region.

Physical examination should include evaluation of the muscles of mastication by intraoral and external palpation. Palpation may reveal muscular spasm and tenderness. Palpation of the TMJ or muscles of mastication may reproduce the patient’s symptoms and trigger significant pain. The patient may have great pain with any attempt at jaw range of motion. Physical findings are also notable for reduced jaw opening and possible lateral deviation of the jaw.

An inability to close the mouth following extreme jaw opening, such as yawning, is the classic manifestation of TMJ dislocation. If the dislocation is unilateral, the mandible will deviate away from the side of the dislocation (Box 30.2).

DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING

When considering the diagnosis of TMD, the emergency practitioner must rule out odontogenic causes. If an intraoral cause remains possible after a carefully performed history and physical examination, the patient should be referred to a dentist.
SECTION III HEAD AND NECK DISORDERS

TREATMENT

TEMPOROMANDIBULAR DISORDERS
Management of TMD is primarily directed at improving the patient’s comfort, which is accomplished with analgesia, muscle relaxation, and behavioral modifications. In the absence of trauma, there is no indication for emergency imaging of the TMJ.

Pain should be addressed with antiinflammatory agents (e.g., ibuprofen, 600 mg by mouth every 6 hours, or naproxen, 500 mg by mouth every 12 hours) and narcotic pain medications (e.g., oxycodone, 5 to 10 mg by mouth every 6 hours as needed). Warm compresses should also be applied to the TMJ region for 15 minutes three times per day. Benzodiazepines are used to relieve masseter muscle spasm (e.g., diazepam, 5 mg by mouth every 8 hours as needed). Behavioral modifications include minimizing masseter muscle use through a soft diet and cessation of gum chewing. Reassurance is important because up to 40% of patients will experience resolution of their TMD symptoms with little or no treatment (Box 30.3). If bruxism is suspected, dental follow-up should be arranged, and a bite appliance can be considered. To date, experimentation with the use of botulinum toxin to reduce masseter muscle contractility and strength has yielded mixed results.

For a full dental evaluation. This evaluation can be done concurrently with the start of TMD treatment. Less common mimics, including jaw tumors and trigeminal neuralgia, should also be considered.

Central nervous system (CNS) disorders must be ruled out in the evaluation of a patient with symptoms suggestive of TMD. The clinician must be especially careful to address this possibility when the patient’s complaints include CNS-related symptoms such as headache and vertigo. A detailed neurologic examination must be performed. Except for possible hyperesthesia immediately around the TMJ, patients with TMD should have no abnormal neurologic findings.

When evaluating a patient for a possible TMJ dislocation, the emergency practitioner must rule out a dystonic medication reaction or a mandibular fracture, both of which can be manifested in similar fashion. In patients with a history of trauma, panoramic radiographic evaluation of the mandible, a TMJ radiographic series, or computed tomography (CT) of the mandible should be performed to assess for a fracture before attempting reduction.

TEMPOROMANDIBULAR DISLOCATION
TMJ reduction is performed in the emergency department (ED) and is usually readily accomplished. Controlling the
patient’s pain and masseter spasm facilitates the procedure. Analgesics (e.g., morphine, 5 mg intravenously as needed) and antispasmodics (e.g., diazepam, 5 to 10 mg intravenously titrated to the patient response) should be administered before reduction is attempted. Atraumatic TMJ dislocations do not require imaging.

Once the patient is comfortable, the clinician faces the patient and grasps the mandible inferiorly with the fingers of both hands. The clinician’s thumbs should be heavily wrapped in gauze for protection and then placed on the occlusive surfaces of the mandibular molars. Downward pressure is applied to move the mandibular condyle inferior to the temporal eminence. The mandible is then pushed posteriorly (Fig. 30.2).

Once the condyle is posterior to the temporal eminence and pressure is released, the condyle returns to its anatomic position in the mandibular fossa. At the time of reduction, the masseter muscles may contract forcefully and cause the patient to inadvertently clench the jaw. The clinician must be aware of this possibility and ensure that the thumbs are guarded during the procedure and remove the thumbs from the occlusive surface of the molars as quickly as possible. If this method does not work, both thumbs may be placed simultaneously on the dislocated side and the reduction reattempted.

In an effort to minimize risk to the practitioner, an extraoral approach to TMJ reduction was proposed by Chen et al. in 2007. The physician faces the patient and places a thumb on the palpable coronoid process that is displaced anteriorly. The fingers of that hand are placed on the mastoid process for stability. On the nondislocated side, the thumb is placed on the zygoma and the fingers hold the mandible angle. The nondisplaced side of the mandible is pulled anteriorly while concomitant pressure is applied posteriorly to the displaced coronoid process. Although this approach is less successful than the traditional approach, there is no risk of injury to the practitioner.

When reduction of the TMJ has been accomplished, the patient needs to avoid excessive mouth opening to prevent recurrent dislocation and may be discharged home. Postreduction pain can be treated with analgesics and antispasmodics. Advising a soft diet for 2 weeks will also minimize the patient’s discomfort. The patient should be evaluated by an oromaxillofacial surgeon within 2 weeks.

**EPISTAXIS**

**EPIDEMIOLOGY**

The incidence of epistaxis is unknown, but it is estimated to occur in up to 60% of all individuals. The vast majority of these episodes are self-limited and only 6% require medical attention. Epistaxis affects both adults and children, with a higher incidence in children younger than 10 years and adults older than 35 years.

The anterior nasal septum is the source of bleeding in 90% of patients with epistaxis; the posterior nasal septum and lateral nasal wall account for 10% of cases of nasal bleeding. Posterior nasal hemorrhage is more common in the elderly population.

**PATHOPHYSIOLOGY**

The nasal mucosa is a highly vascular area, and any disruption of the mucosa can result in bleeding. Although epistaxis can be caused by trauma, this is not the most common cause. Bleeding more commonly results from upper respiratory infections (URIs), a dry environment, nasal foreign bodies, allergic rhinitis, nasal mucormycosis, topical nasal medications (including antihistamines and corticosteroids), and drugs taken intranasally such as cocaine (Box 30.4). Additionally, epistaxis may be the initial symptom of a primary or secondary systemic bleeding disorder. One study found that 45% of patients with bleeding severe enough to warrant hospitalization had an associated systemic disorder that may have contributed to the epistaxis.

The relationship of hypertension to epistaxis is controversial. It is unclear whether elevated blood pressure is the cause or the effect of epistaxis; therefore, hypertension alone is not known to be an independent risk factor for nasal hemorrhage.

The Kiesselbach plexus is located at the anterior portion of the nasal septum and is the source of bleeding in patients with anterior epistaxis. The plexus is supplied by branches of the
BOX 30.4 Risk Factors for Epistaxis

| Alcoholism | Nasal polyps |
| Allergic rhinitis | Platelet inhibitor use |
| Anticoagulant use | Pregnancy |
| Barotrauma | Recent otorhinolaryngologic surgery |
| Blood dyscrasia | Septal deviation or perforation |
| Diabetes mellitus | Sinusitis |
| Endometriosis | Trauma, including nose picking |
| Intranasal drug use | Upper respiratory tract infection |
| Intranasal medication use | |
| Intranasal neoplasm | |
| Low-humidity environment | |
| Nasal foreign body | |

internal and external carotid arteries via the sphenopalatine, ethmoidal, and superior labial arteries. Another name for the Kiesselbach plexus is the Little Area. The posterior nasal septum and lateral nasal wall are supplied by the sphenopalatine artery, which is the source of bleeding for posterior epistaxis.

Functionally, epistaxis is considered anterior when the site of bleeding can be visualized in the anterior portion of the nasopharynx. Posterior nasal hemorrhage cannot be directly visualized and occurs in the posterior or lateral parts of the nose.

PRESENTING SIGNS AND SYMPTOMS

Epistaxis may be manifested as minor bleeding with small quantities of blood dripping from the nares or as major bleeding with the patient vomiting blood. Approximately 90% of cases of epistaxis are anterior and the bleeding source is unilateral. Many patients, however, have blood flowing from both nares because blood from the unilateral bleeding source travels around the septum posteriorly and exits on the other side.

Patients with epistaxis may arrive at the ED with various home treatments in progress. All foreign bodies (cotton, tissues, tampons) should be removed from the nose after the patient arrives to better assess the location and quantity of bleeding.

Important historical elements include the duration of the bleeding, laterality, estimated blood loss, recent trauma, and other associated symptoms.

Obtaining a detailed history is often the key to determining the cause of the patient’s epistaxis. The clinician must know whether the patient has recurrent epistaxis, easy bruising, or other sources of bleeding, such as when shaving or brushing the teeth, or is taking a platelet inhibitor or anticoagulant medication. The past medical history is important in a patient who has hepatic disease, atherosclerosis, Osler-Weber-Rendu disease (hemorrhagic telangiectasia), diabetes mellitus, or cancer with ongoing chemotherapy treatment because each of these conditions is a risk factor for epistaxis.16 Women are more prone to epistaxis during pregnancy.

Trauma, nose picking, recent otorhinolaryngologic surgery, nasal foreign body, URI, nasal polyps, and exposure to a low-humidity environment all predispose to epistaxis and should be addressed in the patient’s history. The family medical history may reveal recurrent epistaxis in multiple family members. In the absence of a hereditary bleeding disorder, such a history suggests familial idiopathic epistaxis. A social history must also be obtained to identify alcoholism, intranasal drug use, or domestic violence.

Physical examination of a patient with epistaxis must focus on the nasopharynx and oropharynx; however, a complete examination is helpful. A skin examination may reveal ecchymoses or petechiae (suggestive of a systemic bleeding disorder), spider angiomas or caput medusa (suggestive of hepatic disease), or signs of trauma. Cardiac examination may demonstrate an irregular heartbeat and should prompt the clinician to inquire further about anticoagulant medications.

Concurrent with examining the nasal mucosa, the posterior oropharynx must also be evaluated. The nares may be occluded with nasal packing or clotted blood but continue to have significant blood loss into the oropharynx. The patient swallows this blood, and without a careful examination, the ongoing loss may not be evident until hematemesis or hemodynamic instability develops.

Alternatively, a patient may have a chief complaint of hemoptysis. If a patient has low-volume nasal bleeding with blood flowing posteriorly into the oropharynx, coughing up gross blood may be the initial symptom of epistaxis.

DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING

Mechanical causes of epistaxis, such as trauma, nose picking, nasal foreign bodies, and nasogastric or nasotracheal intubation, directly disrupt the nasal mucosa. Blood dyscrasias, hepatic disease, platelet inhibitors, and anticoagulant medications result in decreased blood clotting and predispose the host to bleeding. Infectious causes include sinusitis, rhinitis, mucormycosis, and URIs that result in nasal congestion and vasodilation.16 In women, both endometriosis and pregnancy must be considered as causes of epistaxis. Environmental factors also play a role in the incidence of epistaxis. Visits to the ED for the treatment of epistaxis are more common in the winter months.11 This has been proposed to be due to lower ambient humidity and subsequent drying of the nasal membranes during the winter season. Barotrauma can also incite epistaxis.

Recurrent, refractory unilateral epistaxis merits investigation for a possible neoplastic etiology, especially with abnormal findings on neurologic examination.

In patients with larger-volume hemorrhage, hematemesis may be the initial complaint. Once the emesis has been controlled, examination of the oropharynx shows blood entering superiorly from the nasal cavity. The clinician can then work to localize the source of bleeding in the nose without being misled into investigating a gastrointestinal source.

TREATMENT

In the prehospital setting or if the patient has to wait before being seen in the ED, the patient can apply direct pressure to the area by squeezing the soft tissue portion of the nose with the fingers. The nares are successfully compressed when a change in the patient’s voice is heard.

Standard supplies for the treatment of epistaxis include suction with a Frazier tip, nasal speculum, bayonet forceps, cotton-tipped applicators, silver nitrate cautery sticks, packing
material, nasal tampons or balloons, gelatin foam (Gelfoam) or oxidized cellulose (Surgicel), a posterior nasal balloon with syringe, and a good light source. Universal precautions should be followed at all times. The clinician must also wear eye protection because of the risk for ocular exposure during management of the epistaxis.

In the absence of massive epistaxis, the initial step is to evacuate intranasal clots and apply a topical vasoconstrictor and analgesic to the mucosa (Box 30.5). This can be achieved by soaking cotton pledgets in a combined vasoconstrictor and topical analgesic solution. If cotton is not available, rolled 2- × 2-inch gauze can be used. The analgesic is added to improve patient comfort during any necessary interventions once vasoconstriction has occurred. These medications are left in contact with the mucosa for 5 to 10 minutes.

After removal of the pledgets, the nasal cavity and oropharynx are inspected. If the source of bleeding is identified in the nose and the oropharynx is dry, anterior epistaxis has been confirmed. For anterior epistaxis in which vasoconstriction has mostly controlled the bleeding, chemical cautery with silver nitrate is an excellent first choice. The silver nitrate cautery stick is moistened and applied directly to the area of bleeding from peripheral to central and from superior to inferior. This method minimizes the quantity of blood that comes between the cautery stick and the nasal mucosa. Septal damage from silver nitrate can occur. For this reason, the cautery stick should not be in contact with the nasal mucosa for more than 15 seconds, and bilateral septal coagulation should be avoided. Bilateral coagulation can lead to septal ischemia, necrosis, and perforation.

Another approach to anterior epistaxis is to apply a topical hemostatic packing agent, such as gelatin foam or oxidized cellulose, directly to the bleeding area. These products will be absorbed or degrade and do not require removal.

If neither of the preceding measures is successful, direct compression of the mucosa through packing the anterior nasal cavity is necessary. This can be accomplished with nonadherent ribbon gauze packing or a nasal tampon or balloon. Preformed nasal tampons and nasal balloons are now widely available and are easily inserted. The tampon or balloon is lubricated with a water-based lubricant and then directed posteriorly into the nasal cavity. The tampon should be placed gently but firmly and quickly into the nasal cavity. If it is inserted too slowly, the first part may expand from contact with blood before insertion is complete, which makes further insertion more difficult for both the patient and clinician. If after insertion the tampon is not fully expanded, saline or a vasoconstrictive agent (see Box 30.5) can be dripped into the nasal cavity until expansion is achieved. If a balloon is used, it will need to be inflated with saline or air, depending on the model used. If nonadherent ribbon gauze is to be used instead, the technique is to insert the packing in an accordion pattern from posterior to anterior and inferior to superior. If the septum bow to the contralateral side after packing, packing the other side may be necessary. Pain medication may be needed to alleviate the discomfort from nasal packing. The packing should remain in place for 1 to 3 days.

Bleeding from posterior epistaxis is more challenging to control. By definition, the bleeding site is not readily visualized, thus making compression and treatment more difficult. The most rapid method for controlling posterior epistaxis is insertion of a posterior balloon device. Typically, this device has a double-balloon design, one balloon to tamponade the posterior nasal cavity and the other for the anterior nasal cavity. After the device is lubricated, it is inserted into the nasal cavity to its hub. The posterior balloon is inflated and the hub is then drawn out away from the nose until resistance is met. Resistance indicates that the posterior balloon has set in the posterior nasal cavity and is not in the pharynx. Next, the anterior balloon is inflated. The quantity of saline required to fill each balloon varies by device but is typically 7 to 10 mL for the posterior balloon and 15 to 30 mL for the anterior balloon.

If a posterior balloon device is not available, a Foley catheter can be used in its place. A 12- to 14-French catheter with a 30-mL balloon is used. The catheter is inserted through the nose until the noninflated balloon can be seen in the posterior oropharynx. The balloon is then inflated with 10 mL of saline, and the catheter is slowly withdrawn from the nose until resistance is met. While traction is maintained, the anterior nasal cavity is packed with ribbon gauze. The catheter is secured by placement of a padded umbilical clamp or other equivalent device around the catheter at the point where it exits the nostril. Securing the catheter properly is extremely important. If the catheter is allowed to migrate posteriorly, the inflated balloon descends into the oropharynx and possibly the trachea and obstructs the airway. Using a Foley catheter for control of posterior epistaxis is only a temporary technique, and the catheter should be exchanged for a safer double-balloon device as soon as possible.

For patients with massive epistaxis, use of nasal tampons and balloons is the fastest way to minimize or stop the bleeding. Bilateral anterior tampons or balloons are placed and then the oropharynx examined. If active bleeding continues into the oropharynx, one tampon or balloon is removed and replaced with a posterior balloon device. If significant bleeding continues, the second tampon or balloon is removed and a second posterior balloon inserted. Immediate

**BOX 30.5 Treatment of Epistaxis**

<table>
<thead>
<tr>
<th>Vasoconstrictors</th>
<th>Analgesics</th>
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<tbody>
<tr>
<td>4% cocaine</td>
<td>4% cocaine</td>
</tr>
<tr>
<td>Epinephrine (1:1000)</td>
<td>4% lidocaine</td>
</tr>
<tr>
<td>Oxymetazoline</td>
<td></td>
</tr>
<tr>
<td>Phenylephrine</td>
<td></td>
</tr>
</tbody>
</table>

**RED FLAGS**

**Epistaxis**

Epistaxis may be the initial manifestation of a primary or secondary blood dyscrasia. Occluded nares may mask ongoing bleeding. Hematemesis may be the initial complaint in patients with large-volume bleeding. An antihypertensive agent (i.e., beta-blocker) can mask the early stages of hemorrhagic shock by limiting tachycardia.

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otolaryngologic consultation is then necessary. Bilateral posterior balloons should be in place for the minimal amount of time possible. Fluid resuscitation is then begun for any patient with significant blood loss or signs of shock.

Laboratory tests have a limited role in patients with epistaxis. Patients with low- or moderate-volume anterior bleeding who have no known blood dyscrasia and who are not taking anticoagulant medications do not require any laboratory studies.12

If, however, the patient’s history or physical examination raises concern for a systemic cause of the epistaxis, studies should be ordered as appropriate for the clinician’s specific suspicions. Such studies may include a prothrombin time, activated partial thromboplastin time, coagulation factor levels, bleeding time, vitamin K level, and liver function tests.16 In patients exhibiting signs of shock, the hematocrit should be checked to obtain a starting value with which to compare serial measurements. Additionally, blood typing and screening may be necessary for a possible transfusion.

Otolaryngologic consultation is not routinely necessary and should be reserved for refractory epistaxis, for which endoscopy for direct visualization and cautery, surgical sary and should be reserved for refractory epistaxis, for which endoscopy for direct visualization and cautery, surgical

### Epistaxis

- Avoid nose blowing, bending over, and straining.
- Be sure to open your mouth when you sneeze.
- Avoid any activity that puts you at risk for nasal injury.
- Use humidifiers and saline nasal spray to help keep the interior of your nose moist.
- Take pain medications as needed.
- Do not take aspirin or aspirin products.
- Follow up with an otolaryngologist in 24 to 72 hours.
- Take your antibiotics as prescribed. It is important that you continue to take antibiotics as long as your nasal packing or balloon is in place.
- Do not put anything into your nose.
- If bleeding recurs, compress your nose by squeezing the bottom half of it with your thumb and index finger. Hold this compression for 10 minutes. If bleeding continues beyond this time, see your doctor or return to the emergency department.
- See your doctor or return to the emergency department if a fever or rash develops.

### PRIORITY ACTIONS

**Epistaxis**

- Apply a topical vasoconstrictive agent to the nasal mucosa.
- Examine the nasopharynx and the posterior oropharynx to localize the source of bleeding.
- Chemical cautery should be used with caution and bilateral septal cautery should be avoided to minimize the risk for septal injury.
- Ensure that a posterior nasal balloon is properly secured to avoid airway occlusion.

### FOLLOW-UP AND NEXT STEPS IN CARE

A hemodynamically stable patient with anterior epistaxis in whom hemostasis has been achieved and maintained should be discharged home. The patient is instructed to avoid nose blowing, bending over, straining, closed-mouth sneezing, and any activity that raises the risk for nasal trauma. In dry environments, humidifiers and saline nasal spray are recommended to help keep the nasal mucosa moist. Pain medications are prescribed as needed for patient comfort; however, aspirin products should be avoided. Any patient with anterior packing or recurrent epistaxis must be evaluated by an otolaryngologist in 24 to 72 hours.

Patients with nasal packing or balloons should be treated with antistaphylococcal antibiotics to minimize the risk for sinusitis and toxic shock syndrome.17 Drug choices include amoxicillin–clavulanate potassium, 875 mg two times per day, and cephalexin, 500 mg four times per day. The packing should be left in place for 1 to 3 days and antibiotics continued until the packing is removed. Nasal packing containing antibiotics is also available; these products have been shown to inhibit the growth of nasal flora and may supplant the need for additional systemic antibiotics.

Patients with posterior epistaxis require admission to the hospital. If a posterior balloon were to become dislodged and migrate posteriorly, airway compromise could occur. Additionally, such a patient may have a drop in PaO₂ and a rise in PaCO₂ after placement of posterior packing. Bradycardia and other cardiac dysrhythmias have also been reported. The mechanism of these events is unclear.

### SINUSITIS

**EPIDEMIOLOGY**

Sinusitis is an inflammatory disease of the paranasal sinuses, and rhinitis is an inflammatory disease of the membranes lining the nose. Because sinusitis without associated rhinitis is rare, the term *rhinosinusitis* is now the accepted nomenclature for this disease complex.18-22 In much of the current literature, however, the terms *sinusitis* and *rhinosinusitis* are used interchangeably.

Rhinosinusitis is estimated to affect one in seven adults in the United States and has a significant impact on missed workdays and health care costs.23-24 It is a complication of URI in 5% to 10% of children and 0.5% to 2% of adults. The actual number of people affected by rhinosinusitis may be significantly higher than reported because of the multitude of over-the-counter sinus medications available.

Over the past decade, five major expert organizations have generated guidelines on the definitions and treatments of rhinosinusitis and its subtypes.16-22 There is variation among many of the guidelines’ definitions and recommendations. The information presented in this chapter reflects the areas of greatest consensus among the expert groups.

*Rhinosinusitis* is the parent term for several subtypes of disease. In acute rhinosinusitis (ARS), symptoms last 4 weeks or less; recurrent ARS is defined as three or more episodes of ARS within 1 year, with resolution of symptoms between
differentiate early rhinosinusitis from URI because viral URIs are the most common event precipitating rhinosinusitis and the two may be present concurrently.

The signs and symptoms of chronic rhinosinusitis include those of ARS plus a decreased sense of smell. The symptoms are often more vague and less severe and, by definition, must be present for 12 weeks or longer.

Physical examination will reveal nasal mucosal erythema and edema leading to nasal obstruction. Purulent secretions may also be seen in the nose or the posterior oropharynx. Purulent secretions have the highest positive predictive value for rhinosinusitis of any physical finding. The nasal cavities must also be thoroughly inspected for foreign bodies, especially in children.

Sinus tenderness to percussion may be present on examination, although this finding is neither sensitive nor specific. Transillumination of the frontal and maxillary sinuses is also neither sensitive nor specific, and it is not possible to differentiate a fluid-filled sinus from a congenitally small sinus after a single evaluation. The clinician must examine the oral cavity for evidence of a dental infection, which can be the source of maxillary sinusitis. Periorbital edema may also be found.

DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING

The diagnosis of ARS is clinical, and in the absence of specific concerns raised by the history and physical examination, no imaging or laboratory tests are warranted. Nasal cultures may be considered during outpatient care in the event of treatment failure. Evaluation for systemic disorders predisposing to rhinosinusitis, such as cystic fibrosis or immunodeficiency, can be done on a nonemergency basis.

The infectious organisms causing rhinosinusitis are most commonly viral, then bacterial, then fungal. The most common viruses are rhinovirus, parainfluenza virus, and influenza virus. The most common bacterial pathogens of ARS and recurrent ARS in an immunocompetent host are Streptococcus pneumoniae, Haemophilus influenzae, and Moraxella catarrhalis. Rhinosinusitis pathogens more commonly found in immunocompromised hosts are Pseudomonas aeruginosa, Rhizopus, Aspergillus, Candida, Histoplasma, Blastomyces, Coccidioides, and Cryptococcus. P. aeruginosa is also a common pathogen in patients with cystic fibrosis.

The pathogens of chronic rhinosinusitis include those of acute bacterial rhinosinusitis plus group A streptococci, Staphylococcus aureus, P. aeruginosa, and fungi.

Noninfectious causes include congenital diseases that inhibit ciliary function, such as cystic fibrosis and Kartagener syndrome; autoimmune diseases, such as Wegener granulomatosis and sarcoidosis; anatomic obstruction, such as from nasal polyps, nasal tumors, or foreign bodies; and facial trauma that directly disrupts sinus drainage.

Any abnormality found on neurologic or ocular examination in a patient with rhinosinusitis must raise concern for CNS extension of the disease and warrants imaging. CT or magnetic resonance imaging should include the brain, orbits, or sinus (or any combination of these structures), depending on clinical suspicion.

Acute or chronic frontal rhinosinusitis can lead to erosion through the frontal bone and a resultant subperiosteal abscess.

### Table 30.1 Clinical Forms of Rhinosinusitis Based on Duration of Symptoms

<table>
<thead>
<tr>
<th>TYPE</th>
<th>DEFINITION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute</td>
<td>Symptoms present &lt; 4 wk</td>
</tr>
<tr>
<td>Chronic</td>
<td>Symptoms present &gt; 12 wk</td>
</tr>
<tr>
<td>Recurrent</td>
<td>&gt;3 acute episodes within 1 yr</td>
</tr>
</tbody>
</table>

### Box 30.6 Diagnostic Criteria for Acute Rhinosinusitis

<table>
<thead>
<tr>
<th>Primary Diagnostic Indicators</th>
<th>Other Suggestive Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasal congestion, obstruction, or blockage</td>
<td>Hyposmia or anosmia</td>
</tr>
<tr>
<td>Purulent anterior or posterior rhinorrhea</td>
<td>Cough</td>
</tr>
<tr>
<td>Facial pain or pressure</td>
<td>Dental pain</td>
</tr>
<tr>
<td></td>
<td>Ear pain or pressure</td>
</tr>
<tr>
<td></td>
<td>Fatigue</td>
</tr>
<tr>
<td></td>
<td>Halitosis</td>
</tr>
<tr>
<td></td>
<td>Headache</td>
</tr>
</tbody>
</table>

Episodes; and chronic rhinosinusitis requires symptoms for 12 weeks or longer (Table 30.1).

### PATHOPHYSIOLOGY

There are four pairs of paranasal sinuses: frontal, maxillary, sphenoid, and ethmoid. Maxillary sinusitis is the most common, followed by ethmoid, frontal, and then sphenoid sinusitis.

Each sinus drains through an ostium, which communicates with the nose. The frontal, maxillary, and anterior ethmoid sinuses empty into the middle meatus, between the middle and inferior turbinates. This region is termed the ostiomeatal complex. Obstruction of this complex is a critical step in the development of rhinosinusitis.

Each sinus is lined with ciliated epithelium and mucous goblet cells. Healthy sinuses have relatively few mucus-producing goblet cells, and the cilia beat the mucus toward the ostium of the sinus. The patent ostium allows the free flow of mucus and air from the sinus to the nose. When an ostium is occluded, air and mucus no longer flow freely, new mucous-producing cells develop, and mucostasis results.

### PRESENTING SIGNS AND SYMPTOMS

The diagnosis of ARS is based on three signs or symptoms: nasal congestion, obstruction, or blockage; anterior or posterior purulent rhinorrhea; and facial pain or pressure. Other suggestive symptoms support the diagnosis but serve only as adjuncts (Box 30.6).

A detailed history to determine the duration, severity, and course of the symptoms is the only readily available tool to differentiate acute viral rhinosinusitis from acute bacterial rhinosinusitis (Fig. 30.2). It can be challenging to
known as Pott puffy tumor. In addition to signs and symptoms of rhinosinusitis, patients with this disorder have a severe localized headache, swelling of the forehead, and possibly orbital abnormalities. These infections are most commonly polymicrobial. Frontal sinusitis can also lead to osteomyelitis. Complications of frontal sinusitis are most common during the second and third decades of life.

Intracranial complications of rhinosinusitis, such as an intracranial abscess, meningitis, epidural abscess, subdural empyema, and cavernous sinus thrombosis, are serious sequelae of the disease. Disease can progress intracranially through direct extension or via thrombophlebitis of the dilated veins. Any focal neurologic deficits found on examination must raise concern for intracranial extension of rhinosinusitis. Patients with subdural empyema exhibit systemic toxicity, nuchal rigidity, and photophobia, as well as cranial nerve deficits. The emergency practitioner must maintain a high index of suspicion. A review of patients found that headache and fever without other neurologic abnormalities were the most common manifestations of early intracranial complications of rhinosinusitis.

Pansinusitis leads to orbital sequelae in 60% to 80% of patients. Preseptal cellulitis is the most common complication. Clinical findings suggestive of preseptal cellulitis are periorbital edema without any associated change in vision or limitation of ocular mobility. Orbital cellulitis, orbital subperiosteal abscesses, and orbital abscesses are other complications of pansinusitis; they are manifested as periorbital edema, proptosis, orbital pain, and limitations in ocular mobility.

**TREATMENT**

Treatment of ARS is primarily based on reducing the obstruction of the ostiomeatal complex and thereby relieving the patient's discomfort.

Intranasal corticosteroids are known to decrease nasal inflammation and may therefore improve ostial patency. These agents have very few identified side effects and are effective in reducing symptoms even as monotherapy.\(^{29}\) Mometasone nasal spray, 200 mcg in each nostril twice daily, may be used.\(^{29,30}\)

Topical and oral \(\alpha\)-adrenergic decongestants are often prescribed for patients with rhinosinusitis to induce vasoconstriction and reduce nasal mucosal swelling, thereby improving ostial patency and sinus drainage. No controlled clinical trials, however, have examined the efficacy of these agents,\(^{28}\) and the recommendations by the five expert panels are widely disparate.\(^{18,20,21}\) There is no evidence that decongestants are harmful in patients with rhinosinusitis, and therefore the decision regarding their use is left to the clinician. Topical sprays, such as oxymetazoline, two sprays in each nostril every 12 hours, or oral decongestants, such as pseudoephedrine, 60 mg every 6 hours, should be considered on the basis of the risk-to-benefit profile of the individual patient. Decongestant nasal sprays should not be used for longer than 5 days because of the risk for rebound nasal congestion (rhinitis medicamentosa).

Antihistamines are a useful adjunct for patients whose rhinosinusitis has an allergic cause. Diphenhydramine, 25 to 50 mg every 6 hours, loratadine, 10 mg daily, or fexofenadine, 180 mg daily, may all be used. No studies have indicated that antihistamines play a role in nonallergic rhinosinusitis.\(^{20}\)

Saline nose drops prevent crusting of nasal secretions and facilitate elimination of these secretions. Physiologic saline and hypertonic saline sprays both increase mucociliary clearance and increase nasal airway patency.\(^{11,32}\) Saline nose drops, normal or hypertonic, may be a useful adjunct to aid in relieving the symptoms of rhinosinusitis.

Guaifenesin is an expectorant that decreases sputum viscosity. It has been shown to improve the ease of sputum expectoration in patients with respiratory infections but has not been demonstrated to aid in the management of rhinosinusitis (Box 30.7).

The vast majority of cases of ARS are caused by viruses, with only 0.5% to 2.0% estimated to have a bacterial cause.\(^{33}\) For this reason, antibiotic treatment should be initiated only in patients for whom the clinician has high suspicion of acute bacterial rhinosinusitis (Table 30.2).

First-line antibiotic therapy is amoxicillin. If the incidence of \(\beta\)-lactamase–positive *S. pneumoniae*, *H. influenzae*, or *M. catarrhalis* infection is high in the area of patient care, adults should be treated with amoxicillin–clavulanate potassium. Alternative first-line agents are certain second- and third-generation cephalosporins. Third- or fourth-generation quinolone antibiotics are also appropriate agents for adult acute bacterial rhinosinusitis. Azithromycin or clarithromycin are additional treatment choices, but the local resistance patterns of *S. pneumoniae* and *H. influenzae* must first be assessed. The clinician should tailor the choice of antibiotic to the specific resistance patterns of the practice environment. See Box 30.8 for doses and duration of treatment.

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**Box 30.7  Treatment of Acute Rhinosinusitis**

<table>
<thead>
<tr>
<th><strong>Intranasal corticosteroids</strong></th>
<th><strong>Decongestants</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antihistamines if an allergic cause is suspected</strong></td>
<td><strong>Saline nasal spray</strong></td>
</tr>
<tr>
<td><strong>Antibiotics only if the signs and symptoms are consistent with acute bacterial rhinosinusitis</strong></td>
<td></td>
</tr>
</tbody>
</table>

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**Table 30.2  Differentiating Acute Viral from Acute Bacterial Rhinosinusitis**

<table>
<thead>
<tr>
<th>VIRAL</th>
<th>BACTERIAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptom duration &lt; 10 days</td>
<td>Symptom duration ≥ 10 days</td>
</tr>
<tr>
<td>Worst symptoms at days 2-3</td>
<td>Increasing symptoms 5 days after onset</td>
</tr>
<tr>
<td>Improving symptoms after day 3</td>
<td>Worsening symptoms after initial improvement</td>
</tr>
<tr>
<td>Severe symptoms (high fever, unilateral facial or tooth pain, periorbital swelling, orbital cellulitis)</td>
<td></td>
</tr>
</tbody>
</table>
Previously, sulfamethoxazole-trimethoprim was a popular antimicrobial agent for the treatment of rhinosinusitis; however, as resistance to it has increased, its clinical utility has become limited.20

Acute bacterial rhinosinusitis should be treated with antibiotics for 10 to 14 days (except azithromycin, as noted previously). The patient should be reassessed 3 to 5 days after antibiotic treatment has begun. If no improvement is seen, concern for resistant organisms is raised, and a change in antibiotics should be considered. Chronic rhinosinusitis is treated with a 21-day course of antibiotics, although data supporting the optimum duration of treatment are minimal.22,23

ARS for is treated for 10 days, unless otherwise noted. Chronic rhinosinusitis is treated for a minimum of 21 days.

Any patient with evidence of ocular or intracranial extension of sinus disease requires immediate otorhinolaryngologic, ophthalmologic, or neurosurgical consultation (or any combination of the three). Broad-spectrum intravenous antibiotic therapy, such as with a third-generation cephalosporin and vancomycin, must be started.24 However, because many of these complications require surgical intervention, the choice of antibiotic should be made in conjunction with the surgical service. A patient with this complication must be admitted to the hospital.

**Box 30.8 Antibiotic Choices for Acute and Chronic Rhinosinusitis**

- Amoxicillin, 45 mg/kg per dose PO every 12 hr; adult dose, 500 mg twice daily*
- Amoxicillin-clavulanate potassium, 875 mg PO twice daily
- Cefuroxime, 500 mg PO twice daily
- Cefpodoxime, 400 mg PO twice daily
- Levofloxacin, 500 mg PO daily
- Gatifloxacin, 400 mg PO daily
- Azithromycin, 500 mg once then 250 mg daily for 4 additional days
- Clarithromycin, 500 mg PO twice daily
- If protracted or severe course, consider anaerobic coverage:
  - Clindamycin, 450 mg every 8 hr for 14 days
  - Metronidazole, 500 mg every 8 hr

*Avoid in areas with high β-lactam resistance.

PO, By Mouth.

**Suggested Reading**


**References**

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES