CHAPTER 67 ■ FACIAL PAIN

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INTRODUCTION

Facial pain syndromes are common in clinical practice. Many of these syndromes are also unique, given the complex anatomy and specialized sensory innervation of the head, face, and neck, and so can pose diagnostic challenges.

The common descriptive terms for facial pain complaints are frequently misleading. To avoid confusion, clinicians should be familiar with the International Headache Society’s Diagnostic Classification for Head, Face, and Neck Pain Disorders (Tables 67.1 through 67.6).1 Clinicians need to be able distinguish among painful conditions that arise from structural pathology, headache syndromes, oral and facial structures, temporomandibular joint disorders, myofacial pain disorders, and primary cranial neuralgias.

**TABLE 67.1**

INTERNATIONAL HEADACHE SOCIETY
INTERNATIONAL CLASSIFICATION OF HEADACHE DISORDERS II

<table>
<thead>
<tr>
<th>14 CATEGORIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>• The Primary Headaches: 1–4</td>
</tr>
<tr>
<td>• The Secondary Headaches: 5–12</td>
</tr>
<tr>
<td>• Cranial Neuralgias, central and primary facial pain and other headache disorders: 13–14</td>
</tr>
</tbody>
</table>


**TABLE 67.2**

THE PRIMARY HEADACHES (1–4)

1. Migraine
   * without aura
   * with aura
2. Tension-type headache
3. Cluster headache and other trigeminal autonomic cephalalgias
4. Other primary headaches

ORGANIZATION OF THE TRIGEMINAL NOCICEPTIVE SYSTEM

Although nociceptive transmission in the trigeminal and spinal systems is similar, the two systems have important differences. In the perioral region, the trigeminal divisions contain afferents that subserve the dermatomes, which include the lips, teeth, gingiva, and anterior two thirds of the tongue, upper pharynx, uvula, and soft palate. In addition to this cutaneous distribution, the trigeminal nerve contains afferents that provide sensory innervation to a variety of deep structures in the head, including the muscles of mastication and facial expression, the nasal and oral mucosa, the cornea, tongue, tooth pulp, temporomandibular joint, dura mater, intracranial vessels, external auditory meatus, and ear (partially, and with cranial nerves VII, IX, and X).

The trigeminal system carries somatosensory information from these cutaneous and deep afferent structures as well as from specialized organs that have principally nociceptive innervation.
Most nociceptive afferents relay through the trigeminal brainstem complex, with oral and perioral structures represented more rostrally than peripheral sites on the face. In addition, nociceptive afferents from the other cranial nerves and the upper cervical spinal segments (C2–C4) also are relayed through the trigeminal brainstem complex.

In the subnucleus caudalis, cells relaying nociceptive signals (nociceptive-specific cells and wide–dynamic-range cells) are primarily localized to analogous regions of lamina I and V in the spinal cord. Deep afferents also converge on cells that also receive cutaneous nociceptive input, providing a substrate for referred pain in the head, face, and neck through the trigeminal system. Finally, the trigeminal nociceptive relay cells are strongly modulated by central pathways (descending opioidergic, noradrenergic, and serotonergic) that may dynamically modulate nociception under a variety of environmental situations and behavioral states.

Although the trigeminal dermatomes do not generally overlap those supplied by the adjacent cervical spinal nerves and other cranial nerves, they overlap extensively in the spinal afferent system. Three adjacent spinal roots must be injured to render any one region anesthetic. In the trigeminal system, under normal conditions, a section of one trigeminal division renders almost the entire dermatome anesthetic. Because the peripheral sensory nerves overlap so little with the trigeminal system, nerve lesions may result in more pronounced central somatosensory changes than those evoked by similar lesions in spinal nerves. These changes may partly underlie trigeminal neuropathic pain disorders. Additionally, the trigeminal system may be developmentally and functionally distinct as a result of three hypothetical factors: (1) it innervates highly specialized tissues that are engaged in highly specialized functions; (2) it experiences two developmentally unique events: one programmed pain event and one programmed denervation event (eruption and exfoliation of teeth); and (3) it can be affected by dental surgery procedures performed with local anesthesia, which alters the afferent input into the system. These factors may also influence the development of chronic facial pain.

### DIAGNOSTIC EVALUATION

Pain in the mouth or face is one of the most common presenting symptoms in clinical practice. The majority of symptoms are related to dental disease and, in most cases, the cause can readily be established, the problem dealt with expeditiously, and the pain eliminated. However, in a few patients, pain may be persistent and defy attempts at treatment. Intractable oral and facial pains can be diagnostically challenging, given the many potential causes of pain, the anatomic complexity of the region, and the psychosocial importance of the face and mouth. A rigorous protocol for evaluating these patients includes a thorough history and an appropriate clinical examination (see Chapter 17).

A detailed history should always be obtained before examining the patient or ordering special tests or imaging studies because the history will establish a diagnosis in a majority of cases.

### Chief Complaint

The patient’s description of the pain may provide clues to its cause. Primary neuralgias are frequently described as sharp and lancinating, secondary neuralgias have a burning quality, vascular headaches are throbbing, and muscle pain is described as a deep and dull ache. The patient may not be able to give all these descriptions at the first interview, and corroborating information from relatives and friends may be needed to build a general picture of the pain as it affects the patient. Each pain complaint should be listed in order of severity.
History of Present Complaint

The intensity of the pain needs to be measured against the patient’s own experience of pain, need for medication, and effect on lifestyle. For example, does the pain interfere with work, sleep, or social activities? How severe is it on a 10-point scale? Does it fluctuate over time? The origin of the pain should be determined by asking the patient to indicate the site of the pain or the site of its maximum intensity. Its anatomic distribution should be accurately traced in terms of local anatomy.

The patient should be encouraged to remember the events surrounding the onset of the pain, even if it was several years ago. Any other instance of similar pain should be ascertained, even though the patient may not associate these with the present problem. The time relations of the pain should be clarified in terms of duration and frequency of attacks, as well as possible remissions.

Aggravating factors should be determined. Is the pain aggravated by the ingestion of specific foods or beverages, by lying down, during times of stress, talking, brushing the teeth, shaving, applying make-up, or by other identifiable factors? In addition, relieving factors (e.g., lying down, sleeping, heat, and cold) are important clues.

The effects of previous treatments need to be clarified. Which medications have helped? Has surgery altered the nature of the pain? Has endodontic treatment or extraction affected the pain? Finally, the presence or absence of associated factors (e.g., swelling of the face, flushing, tearing, nasal congestion, or facial weakness) needs to be ascertained.

Medical History

Take a detailed history of the patient and the reported pain. Especially, note any trauma to the head, face, and mouth. Identify current and past medications, relevant family history, and the use of over-the-counter medications, supplements, and alternative or complementary therapies. Identify any jaw habits, such as clenching, grinding, posture, or injury. The origin of the pain should be clarified.

Palpate the temporomandibular joint for tenderness. Clicks and pops and the position of the jaw should be noted. Determining whether or not these are eliminated by separating the teeth is important. The purpose of the physical examination is to discover any possible anatomic or physiologic basis for the pain; therefore, it is important to proceed systematically. Patients with facial pain should have a complete head and neck examination, not an examination directed by a presumed diagnosis.

Neurologic Function

The most important evaluations are of those of the cranial nerves (CN V trigeminal) and VII (facial) and the upper cervical nerves (C2–C4). The three divisions of the trigeminal nerve—supraorbital, infraorbital, and inferior alveolar nerves—supply the majority of sensation to the mouth and face. Examine the skin distribution of all three divisions, as well as the intraoral distribution of the second and third divisions. Directional sense, two-point discrimination, and sensory perception with von Frey hairs may help with the diagnosis. Heat, cold, and taste may need to be tested in certain situations. Pain to pressure over the six foramina may indicate trigeminal involvement. Corneal and gag reflexes should be assessed. The size and strength of the masticatory muscles reflect the motor division of CN V. Facial nerve function can be assessed by asking the patient to whistle, purse the lips, smile, close the eyes, and frown.

Upper cervical nerve sensation can be assessed on the scalp for C2 and at the angle of the jaw and upper neck for C3. Pressure over the midsuperior nuchal line directly affects the greater occipital nerve and may reproduce the headache in occipital neuralgia.

Because of the overlap of CNs V, VII, IX, and X and their convergence on the spinal trigeminal nucleus, a more detailed examination of these nerves may be necessary. CN IV and VI nerve palsies may indicate increased intracranial pressure.

Muscle Function

Pain in the masticatory muscles, face, posterior cervical spine, and upper back (the suprascapular and pectoral girdle) are common causes of head, face, and neck pain, so the neck, shoulder, and masticatory muscles should be thoroughly assessed. The size of the muscles can be assessed visually (e.g., temporal hollowing, masseteric hypertrophy). The muscles should be palpated, trigger points noted, and head and neck posture should be assessed. A more thorough evaluation of the masticatory muscles includes measuring the maximum intercinesal opening and lateral and protrusive excursions. Tremors and fasciculation should also be noted.

Temporomandibular Joint

Palpate the lateral pole of the mandibular condyle for tenderness with the mouth open and closed. Course and fine crepitation should be noted and joint noises auscultated. Clicks and pops and the position in the opening or closing cycle should be observed. Determining whether or not these are eliminated by separating the teeth with a tongue blade or by posturing the jaw forward will help focus on the functional importance of these joint noises.

Intraoral Examination

Note how the maxillary and mandibular teeth interdigitate when the mouth is closed (dental occlusion) as well as the state of the dentition and oral hygiene. Look for evidence of wear on the teeth, excessive toothbrush abrasion, or palatal erosion from repetitive vomiting. The health of the oropharyngeal mucosa should be recorded, as well as the moistness of the mucosa and pooling of saliva. The parotid and submandibular glands can be milked to evaluate the quality and quantity of saliva expressed. The tongue and soft palate should be centered midline and freely mobile. Excessive draping of the soft palate, as seen in sleep apnea, should be noted.

Diagnostic Imaging (see Chapter 19)

Periapical dental films and panoramic maxillofacial radiographs are inexpensive, readily available, do not expose patients to excessive radiation, and offer detailed information about the teeth and jaws. Computed tomography (CT) can provide more detailed images of the bony structures of the jaws, temporomandibular joints, and base of the skull. Three-dimensional imaging can be helpful in some instances. Magnetic resonance imaging (MRI) is best for evaluating the soft tissues and can be used for assessing the deep oro- and nasopharyngeal anatomy and the internal anatomy of the temporomandibular joints. In addition, the brain can be evaluated with MRI with and without gadolinium contrast. MRI studies can help determine whether the vasculature is impinging on the trigeminal ganglion, which can cause trigeminal neuralgia.

Bone scan with technetium-99m will highlight areas of metabolic activity within the bone and can help identify areas of infection, tumor extension and continued growth, or degenerative change in the temporomandibular joint.
DENTAL AND ORAL SURGICAL CONDITIONS

Routine blood tests include complete blood count and differential to exclude anemia and blood dyscrasias. Erythrocyte sedimentation rate may be elevated in temporal arteritis. Rheumatoid factor and Lyme titer may be helpful in evaluating temporomandibular joint disease.

FACIAL PAIN DISORDERS

Pain Attributed to Disorders of the Oral Cavity

Facial Pain of Dental Origin (Odontogenic)

Tooth pulp has a specialized and possibly exclusively nociceptive innervation. In contrast, periodontal tissues are innervated by a wide variety of sensory afferents. Pain of dental origin is extremely variable and can simulate nearly any pain syndrome. Dental pain may be spontaneous or induced in various ways, and it can be intermittent or continuous (Table 67.7). Because of the extreme variability of toothache, all pains about the mouth and the face should be considered to be of dental origin until proven otherwise. Dental pain is typically provoked by thermal or mechanical stimulation of the damaged tooth, but it can also be provoked by light touch, tooth-tooth contact (in eating and talking), and pressure. Dental pain is usually described as an aching sensation and sometimes as throbbing. When mild, it may be felt only as a tenderness or soreness. When severe, it may have a burning or electric- or shock-like quality. Patients often cannot localize pain arising solely from the dental pulp. They often cannot determine whether the offending tooth is mandibular or maxillary, much less which tooth is involved. The pain is felt diffusely in the teeth, jaws, face, and head. Clinical and radiographic findings of dental decay, fractured dental restoration, tooth fracture, or abscess drainage (fistula) may confirm dental pain as the source of the complaint (Tables 67.8 and 67.9).

Reversible and Irreversible Pulpitis. Dental pain occurs most commonly secondary to dental caries, which represent a loss of integrity of tooth enamel. When enamel integrity is sufficiently breached, sensitivity to cold or sweet stimulus may result. Carious progression occurs more rapidly with dentinal involvement. At this stage, the vital dental pulp is exposed to the oral environment, and inflammatory changes in the pulp tissue are evident histologically.

Table 67.7
DENTAL AND ORAL SURGICAL CONDITIONS

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dentoalveolar Pathology</td>
<td>&quot;pulpal&quot;</td>
</tr>
<tr>
<td>Odontogenic and Nonodontogenic Pathology</td>
<td>&quot;periodontal&quot;</td>
</tr>
<tr>
<td>Trigeminal Neuralgia and &quot;Equivalents&quot;</td>
<td></td>
</tr>
<tr>
<td>Headache and Neck pain</td>
<td></td>
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<tr>
<td>Temporomandibular Disorders</td>
<td></td>
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<tr>
<td>Oral Mucous Membrane Disease</td>
<td></td>
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<tr>
<td>Oral Manifestations of Systemic Disease</td>
<td></td>
</tr>
<tr>
<td>Neuropathic Pain (Persistent Idiopathic Facial Pain)</td>
<td>&quot; Burning Mouth/Tongue Syndrome&quot;</td>
</tr>
</tbody>
</table>

Table 67.8
FEATURES OF ODONTOGENIC PAIN

- Presence of etiologic factors for an odontogenic origin of pain
- Unilateral pain
- Localized pain (diagnosis-specific)
- Pain qualities (sharp, dull, aching, throbbing)
- Sensitivity to temperature
- Sensitivity to pressure, palpation, percussion
- Pain reduction by local anesthetic injection?

The pulpal inflammatory process is initially reversible. Reversible pulpitis is characterized by inflammation of the pulp that may recover or heal when the insult is removed. Continuous stimuli jeopardizes the pulp’s ability to respond and repair itself. Irreversible pulpitis can be distinguished from reversible pulpitis by the duration of symptoms. Both require a stimulus to initiate the pain; however, the duration of pain is measured in seconds in reversible pulpitis but in minutes or hours in irreversible pulpitis. Spontaneous odontogenic pain most frequently marks pulpal death or necrosis. Pain elicited with heat is most commonly associated with pulpal necrosis. The general clinical characteristics displayed by toothache of pulpal origin are described in Table 67.8.

Cracked Tooth. Incomplete fractures of a vital tooth may trigger intermittent pain when biting on the offending tooth. Risk factors include older age, extensive dental restoration, and parafunctional habits, such as teeth grinding. Unfortunately, the cracks are often difficult to find and do not appear on all radiographs. The pain is often confused with that of pulpsitis or trigeminal neuralgia, which may result in unnecessary treatment (see Table 67.9). Careful clinical examination, including staining or meticulous bite tests on each tooth cusp, may be useful.

Acute dental pain typically responds to local treatments (e.g., ice packs and reduced mechanical stimulation), or systemic non-steroidal anti-inflammatory drugs (NSAIDs). Opioid analgesics (and combinations) are also indicated, depending on the extent of the pain. In many cases, treatment with antibiotics is appropriate and palliative until a definitive dental intervention is performed. Definitive dental procedures are generally curative.

Disorders of the Periodontium (Periodontal Disease)

Chronic periodontal disease is an immune-mediated inflammatory process initiated by pathogenic oral microorganisms, destroying either focal or generalized areas of tooth-supporting structures and the surrounding bone. Chronic periodontitis is generally not a chronically painful disorder. Typically, patients notice gingival sensitivity and tenderness or gingival enlargement secondary to inflammation and bleeding with brushing or probing examination. A tooth with lost bone support may have lost the gingival attachment surrounding the necks and soft tissue of the root, which may result in tooth sensitivity, tenderness, and mobility. In acute periodontal infection, tenderness to the touch, erythema, and bleeding may be evident. An acute periodontal abscess may cause swelling and purulence (see Table 67.9). When inflammation or infection (i.e., acute pericoronitis) occurs in the soft tissue or bone around an erupting or partially erupted tooth (particularly third molars, otherwise known as wisdom teeth), similar signs and symptoms may be seen, with pain as a primary complaint.

The pain of periodontal disorders also generally responds to NSAIDs, opioid analgesics, or combination analgesics. An acute abscess may also have to be locally incised and drained. Areas of generalized periodontitis may be treated with tooth scaling and...
ODONTIC PAIN

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Pulpitis</th>
<th>Peridontal</th>
<th>Cracked Tooth</th>
<th>Dentinal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnostic Features</td>
<td>Spontaneous and/or evoked deep/diffuse pain in compromised dental pulp. Pain may be sharp, throbbing, or dull.</td>
<td>Localized deep continuous pain in compromised periodontium (e.g., gingiva, periodontal ligament) exacerbated by biting or chewing.</td>
<td>Spontaneous or evoke brief sharp pain in a tooth with history of trauma or restorative work (e.g., crown, root canal).</td>
<td>Brief, sharp pain evoked by different kinds of stimulus to the dentin (e.g., hot or cold drinks).</td>
</tr>
<tr>
<td>Diagnostic Evaluation</td>
<td>Look for deep caries and recent or extensive dental work. Pain provoked/exacerbated by percussion, thermal, or electric stimulation of affected tooth. Dental radiographs helpful (periapical).</td>
<td>Tooth percussion over compromised periodontium provokes pain. Look for inflammation or abscess (e.g., periodontitis, apical dental radiographs helpful (periapical)).</td>
<td>Presence of tooth fracture may be detectable by radiograph. Percussion should elicit pain. Dental radiographs are helpful (periapical taken from different angles).</td>
<td>Exposed dentin or cementum due to recession of periodontium. Possible erosion of dentinal structure. Cold stimulation reproduces pain.</td>
</tr>
<tr>
<td>Treatment</td>
<td>Medication: NSAIDs, nonopiate analgesics. Dentistry: remove carious lesion, tooth restoration, endodontic treatment, or tooth extraction.</td>
<td>Medication: NSAIDs, nonopiate analgesics, antibiotics, mouthwashes. Dentistry: drainage and débridement of periodontal pocket, scaling and root planning, periodontal surgery, endodontic treatment, or tooth extraction.</td>
<td>Medication: NSAIDs, nonopiate analgesics. Dentistry: depends on level of the tooth fracture-restoration, treatment, or extraction of the tooth.</td>
<td>Medication: mouthwash (fluoride), desensitizing toothpaste. Dentistry: fluoride or potassium salts, tooth restoration, endodontic treatment. Patient education, diet, tooth brushing force and frequency, proper toothpaste.</td>
</tr>
</tbody>
</table>

Acute Necrotizing Ulcerative Gingivitis

Acute necrotizing ulcerative gingivitis (ANUG) is an aggressively destructive process. The diagnostic triad includes pain, ulcerated or “punched out” interdental papillae, and gingival bleeding. Secondary signs include fetid breath, pseudomembrane formation, “wooden teeth” feeling, foul metallic taste, tooth mobility, lymphadenopathy, fever, and malaise. The cause of ANUG is poorly understood. It appears to be an opportunistic infection in a host of lowered resistance. The most important predisposing factor is human immunodeficiency virus infection and the second, a history of necrotizing gingivitis. Other contributing factors include poor oral hygiene, unusual emotional stress, poor diet, inadequate sleep, recent illness, alcohol use, tobacco use, and various infections, such as malaria, measles, and intestinal parasites. Treatment consists primarily of bacterial control. Chlorhexidine oral rinses, professional débridement and scaling, and adjunctive antibiotic therapy with a soft diet rich in protein, vitamins, and fluids are important in treating and preventing the disease.10

Burning Mouth-Tongue Syndrome (Oral Burning)

Burning mouth-tongue syndrome (BMS) is an idiopathic pain condition of the oral mucous membranes akin to idiopathic neu-

Disorders of the Maxilla and Mandible

Numerous disorders of the bony substrate of the jaws may present with pain. These disorders are generally classified as being of odontogenic or nonodontogenic origin, cystic, cystic-like, or tumor, and benign or malignant (either primary or metastatic disease). Often, additional historical or examination findings warrant further evaluation (i.e., swelling, mass, discoloration, numbness, weakness, bleeding, drainage, tooth loss, or mobility). Pain can be treated symptomatically until a definitive diagnosis is established and definitive therapy is initiated (Table 67.11).

Salivary Gland Disorders

Disorders of the three major pairs of salivary glands (parotid, submandibular, and sublingual) and many hundreds of minor salivary glands in the mouth may also produce pain as a primary or associated symptom. These disorders are often accompanied by other signs and symptoms (including swelling, drainage, cervical adenopathy, or generalized signs of systemic infection), depending on the cause of the disorder. Disorders of the parotid gland can locally extend to produce otologic symptoms or CN (V, VII, or IX) involvement. Disorders of the submandibular gland may result in symptoms of impaired swallowing or impairment of CNs V, IX, XII (Table 67.12).
TABLE 67.10
COMMON PAINFUL MUCOSAL CONDITIONS

Infections
Herpetic stomatitis
Varicella zoster
Candidiasis
Acute necrotizing gingivostomatitis

Immune/Autoimmune
Allergic reactions (toothpaste, mouthwashes, topical medications)
Erosive lichen planus
Benign mucous membrane pemphigoid
Aphthous stomatitis and aphthous lesions
Erythema multiform
Graft versus host disease

Traumatic and Iatrogenic Injuries
Factitial, accidental (burns: chemical, solar, thermal)
Self-destructive (rituals, obsessive behaviors)
Iatrogenic (chemotherapy, radiation)

Neoplasia
Squamous cell carcinoma
Mucoepidermoid carcinoma
Adenocystic carcinoma
Brain tumors

Neurologic
Burning mouth syndrome and glossodynia
Neuralgias
Postviral neuralgias
Posttraumatic neuropathies
Dyskinesias and dystonias

Nutritional and Metabolic
Vitamin deficiencies (B12, folate)
Mineral deficiencies (iron)
Diabetic neuropathy
Malabsorption syndromes

Miscellaneous
Xerostomia, secondary to intrinsic or extrinsic conditions
Referred pain from esophageal or oropharyngeal malignancy
Mucositis secondary to esophageal reflux
Angioderma

TABLE 67.12
SALIVARY GLAND DISEASE

Inflammatory
Noninflammatory
Infectious
Obstructive
Immunologic (Sjogren’s Syndrome)
Tumors
Others (Red herrings)

syndrome-dry mouth, fungal infections) and systemic diseases (i.e., vitamin deficiencies, diabetes mellitus, immune connective tissue disorders, vasculitides). More recent evidence suggests that BMS is more likely a neuropathic pain disorder of either peripheral or central origin. Some recent taste-testing data and functional brain imaging studies support this hypothesis (Table 67.13).11–15 Current treatments for BMS focus on this hypothesis and use both topical (oral mucosa) and systemic antineuropathic pain medications (see neuropathic pain section below); however, there is little evidence that such treatments are effective.

Pain Attributed to Disorders of the Eye

Pain in and around the eye is a common presenting problem (Table 67.14). Most ophthalmologic conditions producing eye pain are associated with obvious ocular symptoms, signs, or histories that implicate the eye as the origin of pain. Several facial pain and headache syndromes present with “eye pain” as the chief symptom (Table 67.15). In addition, during the history and physical examination, several signs and symptoms warn of more serious eye disease and even of potential life-threatening problems (Table 67.16).

History and Ocular Examination

A complete ophthalmic history should include any prior visual loss, ophthalmic diseases (e.g., corneal infections, uveitis, and glaucoma), use of contact lenses, recent or remote ocular surgery, and ocular trauma. In addition to noting the specific features of pain when taking the history for eye pain, such as time of onset, severity, exacerbating and palliating factors, radiation, quality, duration, and frequency, ask about the specific location of pain; for example, intraocular, retrobulbar, periorbital, or frontal and associated symptoms, such as tearing, loss of vision, double vision, photophobia, and discharge.16

Simple instruments are required to perform the basic eye examination, in which the pain specialist can triage patients with eye pain and identify those who require formal ophthalmologic consultation. Such equipment includes a near vision card (Snellen card), a hand light, and a direct ophthalmoscope. The Snellen card is used to check the visual acuity (VA). The VA should be tested using the patient’s spectacle correction and each eye should also be tested individually. The pupil response to light, the regularity of the pupil, and relative afferent papillary defect should be evaluated using a hand light. Also, examine extraocular motility examination and the eyelids. Use a hand light to assess the conjunctiva for chemosis, injections, and foreign bodies and the cornea for keratitis, corneal foreign bodies, and lacerations. Evaluation of the optic nerve using the direct ophthalmoscope should be sufficient to exclude gross optic atrophy, fundoscopic abnormalities, and papilledema.

Ocular and Orbital Causes of Eye Pain

Although a large percentage of patients with headache attribute their pain to their refractive errors and present with many pairs...
TRIGEMINAL NEUROPATHIC PAIN DISORDERS

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Trigeminal Neuralgia</th>
<th>Deafferentation Pain</th>
<th>Acute and Postherpetic Neuralgia</th>
<th>Burning Mouth Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diagnostic Features</strong></td>
<td>Brief severe lancinating pain evoked by mechanical stimulation of trigger zone (pain free between attacks). Usually unilateral, affects the V2/V3 areas (rarely V1). Possible pain remission periods (for months/years).</td>
<td>Spontaneous or evoked pain with prolonged after-sensation after tactile stimulation. Trigger zone due to surgery (tooth extraction) or trauma. Positive and negative descriptors (e.g., burning, nagging, boring).</td>
<td>Pain associated with herpetic lesions, usually in the V1 dermatoma. Spontaneous pain (burning and tingling), but may present as dull and aching. Occasional lancinating evoked pain.</td>
<td>Constant burning pain of the mucous membranes of the tongue, mouth. Hard or soft palate or lips. Usually affects women age &gt;50 years.</td>
</tr>
<tr>
<td><strong>Diagnostic Evaluation</strong></td>
<td>MRI for evidence of tumor or vasoconstriction of the trigeminal tract or root (cerebropontine angle). Rule-out multiple sclerosis, especially in young adults.</td>
<td>Etiologic factors such as trauma or surgery in the painful area. Order MRI if the area is intact to rule-out peripheral or central lesions.</td>
<td>Rule-out salivary gland dysfunction (xerostomia) or tumor. Sjögren’s, candidiasis, geographic or fissured tongue, and chemical or mechanical irritations. Nutrition and menopause.</td>
<td></td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Medication: anticonvulsants (e.g., carbamazepine, gabapentin); antidepressants (e.g., amitriptyline, nortriptyline, desipramine); nonopiate analgesics, botulinum toxin. Combination of baclofen and anticonvulsants can produce good results. Surgery: microvascular decompression of trigeminal root, ablative surgeries (e.g., rhizotomy, gamma knife).</td>
<td>Medication: anticonvulsants (e.g., carbamazepine, gabapentin); antidepressants; nonopiate analgesics; topical agents (e.g., lidocaine 5% patches). Surgery: ablative surgeries (e.g., trigeminal rhizotomy, gamma knife).</td>
<td>Medication: acyclovir (acute phase) anticonvulsants, antidepressants; nonopiate analgesics; topical agents (e.g., lidocaine 5% patches). Surgery: ablative surgeries (e.g., rhizotomy, gamma knife).</td>
<td>Medication: anticonvulsants, benzodiazepines, antidepressants; nonopiate analgesics; topical agents (e.g., lidocaine, mouth washes). Cognitive-behavior: biofeedback, relaxation, coping skills.</td>
</tr>
</tbody>
</table>

Ocular Causes for Eye Pain with a White Eye ("Quite Eye"). Other causes of eye pain include acute angle-closure glaucoma, anterior or posterior uveitis, posterior scleritis, intraocular tumors, optic neuritis, and corneal disorders (Tables 67.17 and 67.18).

Glaucoma. Glaucoma may cause acute or chronic eye pain. Glaucoma is a broad term for a large array of clinical disorders that are characterized by damage to the optic nerve with visual field defects generally associated with elevated intraocular pressure. Pain in glaucoma is entirely a function of the rate of rise of intraocular pressure, so only acute forms are likely to be painful. The aqueous humor is produced by the ciliary body in the posterior chamber; it flows through the pupillary aperture and exits the anterior chamber through the trabecular meshwork in the anterior chamber angle. Disorders of elevated intraocular pressure are of two types: open angle, in which the aqueous humor can flow through the trabecular meshwork, and angle-closure, in which the iris or some other structure is physically blocking access to the trabecular meshwork. These types are further subdivided into primary and secondary forms. Primary open-angle glaucoma is the most common type of glaucoma and is almost always entirely asymptomatic. Therefore, this form of glaucoma is rarely the cause of ocular pain in patients with a "quite eye." Interestingly, the miotic eye drops used to treat primary open-angle glaucoma are more likely to cause eye and brow ache. In contrast, acute angle-closure glaucoma is associated with severe, acute eye pain. Fortunately, it is far less common than primary open-angle glaucoma. Even though patients with angle-closure glaucoma can have a normal appearing eye, they typically present with a red eye, edematous cornea, blurred vision, a pupil that is often partially dilated, irregularly shaped, and poorly reactive to light, and intense eye pain. The pain may radiate widely and often is associated with nausea and vomiting. Teeth have been extracted to treat this disorder, as well as laparotomies for the accompanying gastrointestinal complaints.
### Table 67.14

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Paranasal Sinus Pain</th>
<th>Periocular Pain</th>
<th>Periauricular Pain</th>
<th>Head and Neck Cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diagnostic Features</strong></td>
<td>Bilateral or unilateral throbbing or pressure frontal area pain, exacerbated by leaning forward or palpitation over the sinus.</td>
<td>Pain or tenderness with or without eye movements, deep orbital pain, and referred pain.</td>
<td>Diffuse aching or sudden pain with or without aural discharge (e.g., otitis media).</td>
<td>Variety of symptoms. Pain may be due to tumor, nerve compression, secondary infection, secondary myofascial pain, deafferentation, radiotherapy, chemotherapy.</td>
</tr>
<tr>
<td><strong>Diagnostic Evaluation</strong></td>
<td>History of chronic allergies, frequent upper respiratory infections, sinusitis, headaches of various types, sinus surgery Refer to ear, nose, and throat specialist for endoscopic and/or CT study (e.g., sinus opacification).</td>
<td>Examine eyelids, lacrimal function, conjunctiva, and sclera. Ophthalmoscopy and ophthalmology referral. Rule-out primary headache, temporal arteritis, orbital pseudotumor.</td>
<td>The area is innervated by multiple cranial and cervical nerves so complete functional and structural exam necessary (e.g., inspect tympanic membrane, TMJ, and myofascial). CT and MRI invaluable for mastoiditis and cholesteatoma.</td>
<td>Complete evaluation by multidisciplinary team, CT, MRI, endoscopy, biopsy and surveillance. Treatment coordination by oncologist.</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Ear, nose, and throat specialist evaluation/treatment Medication: sinusitis-topical decongestants; systemic antibiotics. Chronic sinus pain-NSAIDs; nonopioid analgesics; topical agents (lidocaine spray); anticonvulsants, antidepressants; botulinum toxin. Surgery</td>
<td>Proper ophthalmologic evaluation and treatment. Medication: NSAIDs; nonopioid analgesics; systemic antibiotics, topical corticosteroids, botulinum toxin across forehead and glabellar areas in selected cases. Surgery</td>
<td>Proper ear, nose, and throat specialist evaluation and treatment. Medication: NSAIDs; nonopioid analgesics; systemic antibiotics, topical corticosteroids, botulinum toxin in selected cases. Surgery</td>
<td>Oncologist evaluation and treatment. Medication: anticonvulsants, antidepressants, opiate or nonopioid analgesics, topical agents, muscles relaxants. Surgery: ablative surgeries.</td>
</tr>
</tbody>
</table>

### Table 67.15

<table>
<thead>
<tr>
<th>HEADACHE AND FACIAL PAIN SYNDROMES WITH EYE PAIN</th>
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<tbody>
<tr>
<td>Cluster headache and cluster-tic syndrome</td>
</tr>
<tr>
<td>Paroxysmal hemicrania</td>
</tr>
<tr>
<td>SUNCT syndrome</td>
</tr>
<tr>
<td>Trigeminal neuralgia</td>
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<tr>
<td>Sphenopalatine neuralgia (Sluder’s neuralgia)</td>
</tr>
<tr>
<td>Ice-pick headache</td>
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<tr>
<td>Ice cream headache</td>
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<tr>
<td>Hypnic headache</td>
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<tr>
<td>Eye pain, headache and lung cancer</td>
</tr>
<tr>
<td>Nonorganic pain and headache (psychosomatic and psychiatric disorders)</td>
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</tbody>
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### Table 67.16

<table>
<thead>
<tr>
<th>RED FLAGS FOR A PATIENT WITH EYE PAIN</th>
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<tbody>
<tr>
<td>New visual acuity defect, color vision defect, or visual field loss</td>
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<tr>
<td>Relative afferent pupillary defect</td>
</tr>
<tr>
<td>Extraocular muscle abnormality, ocular misalignment, or diplopia</td>
</tr>
<tr>
<td>Proptosis</td>
</tr>
<tr>
<td>Lid retraction or ptosis</td>
</tr>
<tr>
<td>Conjunctival chemosis, injection, or redness</td>
</tr>
<tr>
<td>Corneal opacity</td>
</tr>
<tr>
<td>Hyphema or hypopyon</td>
</tr>
<tr>
<td>Iris irregularity</td>
</tr>
<tr>
<td>Nonreactive pupil</td>
</tr>
<tr>
<td>Fundus abnormality</td>
</tr>
<tr>
<td>Recent ocular surgery (&lt;3 months)</td>
</tr>
<tr>
<td>Recent ocular trauma</td>
</tr>
</tbody>
</table>
TABLE 67.17

PAIN IN OR AROUND THE EYE: “QUITE EYE” AND NORMAL EXAM

Cluster headache and cluster-tic syndrome
Paroxysmal hemicrania
SUNCT/SUNA syndrome
Migraine and tension-type headache
Ice-pick headache/Ice cream headache/Valsalvaa headache
Trigeminal neuralgia
Sinus disease (acute)
Teeth, jaws (TMD)
Carotid disease
Temporal arteritis
Eye pain, headache, and lung cancer

TABLE 67.18

PAIN IN OR AROUND THE EYE: “QUITE EYE” AND OPHTHALMOLOGIC FINDINGS

Ocular processes—glaucoma, corneal disease, uveitis, scleritis, intraocular tumors, ocular ischemia, hemorrhage

Intracranial processes—tumor, pseudotumor cerebri, infection, compressive or infiltrative optic neuropathy

Orbital processes—tumor, infection, inflammatory, vascular, posttraumatic

Cavernous sinus/retro-orbital processes—aneurysm, tumor, thrombosis, infection, inflammatory, C-C fistula, posttraumatic

Intracranial processes—tumor, pseudotumor cerebi, infection, inflammatory, vascular, intracranial pressure changes

Scleritis. Inflammation of the deeper layers of the eye wall, known as scleritis, usually presents with severe, intense, and boring pain. The pain is often localized to the eye but may radiate into the sinuses, jaw, or frontal region. The sclera may appear thin or have a bluish hue, and the globe is usually tender. About half the cases are idiopathic, with other causes including herpes simplex virus, herpes zoster, and collagen vascular disease.

Intraocular Tumors. Primary intraocular tumors typically do not cause pain, but orbital extension of tumors may produce trigeminal involvement (neuropathic pain). Intraocular tumors may also produce pain by secondary inflammatory reaction (uveitis) or elevated intraocular pressure induced by the tumor.

Conjunctivitis. Conjunctivitis, or “pink eye,” is the most common cause of a red, irritated eye. Because the conjunctiva has fewer pain fibers than the cornea, conjunctivitis is generally less painful than corneal epithelial defects, and visual acuity is usually only slightly reduced. The three most common types of conjunctivitis are viral, allergic, and bacterial. The viral type is often associated with an upper respiratory tract infection, cold, or sore throat, with adenovirus infection being the most common viral cause. The condition is characterized by a watery discharge, mild foreign-body sensation, and photophobia. Bacterial infection tends to produce more mucopurulent exudates. Allergic conjunctivitis is extremely common and is often mistaken for infectious conjunctivitis. Itching, redness, and epiphora are typical. The palpebral conjunctiva may become hypertrophic with giant excrences called cobblestone papillae. Irritation from contact lenses or any chronic foreign body can also induce formation of cobblestone papillae.

Keratoconjunctivitis Sicca. Also known as “dry eye,” keratoconjunctivitis sicca produces a burning, foreign-body sensation, as well as injection and photophobia. In mild cases, the eye appears surprisingly normal, but tear production, as measured by wetting of a filter paper (a Schirmer strip), is deficient. A variety of systemic drugs, including antihistamines, anticholinergic, and psychotropic medications, cause dry eye by reducing lacrimal secretion. Disorders that involve the lacrimal gland directly, such as sarcoidosis or Sjögren syndrome, also cause dry eye. Patients may develop dry eye after radiation therapy if the treatment field includes the orbit.

Blepharitis. Blepharitis refers to inflammation of the eyelids. The most common form occurs in association with acne rosacea or seborrheic dermatitis. The eyelid margins are usually colonized heavily by staphylococci. Upon close inspection, they appear greasy, ulcerated, and crusted with scaling debris that clings to the lashes. A chalazion is a painless, granulomatous inflammation of the meibomian gland that produces a pea-like nodule within the eyelid. Basal cell, squamous cell, or meibomian gland carcinoma should be suspected for any nonhealing, ulcerative lesion of the eyelids.

Dacryocystitis. Dacryocystitis is inflammation of the lacrimal drainage system and usually occurs after obstruction of the system. It can produce epiphora and ocular injection. Gentle pressure over the lacrimal sac evokes pain and reflux of mucous or pus from the tear puncta.

Herpes Simplex Infection. Primary ocular infection is generally caused by herpes simplex type 1, rather than type 2. It manifests as a unilateral follicular blepharoconjunctivitis and is easily confused with adenovirus conjunctivitis, unless vesicles appear on the periorificial skin or conjunctiva. A dendritic pattern of corneal epithelial ulceration revealed by fluorescein staining is pathognom-
monic for herpes infection but is seen in only a minority of primary infections. Recurrent ocular infection arises from reactivation of the latent herpes virus.

**Herpes Zoster Infection.** Herpes zoster recurrence from reactivation of latent varicella virus causes a dermatomal pattern of painful vesicular dermatitis, which is covered in detail elsewhere in this text.

**Disorders with Eye and Periorbital Pain as the Primary Presentation.** Several facial pain syndromes present with prominent ophthalmologic signs and symptoms (Table 67.19). The more common ones are discussed below.

**Primary Headache Disorders (see Chapter 61).** Many of the primary headache disorders can present with frontotemporal area head pain or sometimes with periorbital and eye pain (migraine, tension-type headache). Some of these disorders have a primary presenting symptom of pain in and around the eye (cluster headache, paroxysmal hemicrania, conjunctival injection and tearing [SUNCT], and short-lasting unilateral neuralgiform headache attacks with autonomic symptoms [SUNA]). In addition to the pain, other signs and symptoms consistent with the diagnosis of a headache disorder are often present (i.e., aura, autonomic phenomenon, etc.).

**Carotid Artery Disease.** Head, face, and neck pain are frequently reported by patients with subarachnoid hemorrhage, intracranial aneurysms, arteriovenous malformations, and carotid and vertebral artery dissections. Patients describe headache, eye pain, facial pain, and neck pain. Associated symptoms may also be present, depending on the vascular source of the pain. Subarachnoid hemorrhage can also present with meningismus, neck pain, nausea, altered consciousness, and seizure. The headache and facial pain is highly variable, ranging from mild pain to the “first and worst headache of my life.” Aneurysms can present with head and face pain with cranial nerve palsies, visual disturbances and sudden blindness, and retinal abnormalities. Imaging and lumbar puncture are often warranted for the diagnosis before definitive treatment. The primary symptom of carotid artery dissection is often headache, either alone or with other symptoms. Frequently, carotid artery dissection also presents with eye pain, facial pain, and neck pain. The headache can be focal or diffuse, whereas the facial pain is generally ipsilateral. Other findings may be pulsatile tinnitus, visual disturbance, central retinal artery occlusion, and Horner syndrome.

**Orbital Inflammatory Pseudotumor.** Orbital inflammatory pseudotumor is a pain syndrome thought to be caused by idiopathic orbital inflammation, which presents typically with eye pain and other orbital findings (proptosis, injection, chemosis, or ophthalmoplegia). The pain may be either unilateral or bilateral. Imaging (CT or MRI) typically shows evidence of idiopathic inflammation, enlargement, and contrast enhancement of orbital structures suggesting pathologic involvement. Orbital biopsy may be required to exclude other causes.

**Idiopathic Intracranial Hypertension (“Pseudotumor Cerebri”).** Idiopathic intracranial hypertension (“pseudotumor cerebri”) and facial pain occur primarily in young, obese women of childbearing age. The common presenting findings are daily headache, transient visual obscurations (seconds), pulsatile intracranial noises, and double vision. Typically, visual acuity and color representation are preserved, but many patients have optic nerve-related, visual-field defects (e.g., enlarged blind spots, generalized constriction, and inferior nasal field loss). Several predisposing factors have been identified, including the use of oral contraceptives, anabolic steroids, tetracycline, and vitamin A.

**Tolosa-Hunt Syndrome.** Tolosa-Hunt syndrome is an idiopathic inflammatory granulomatous process involving the cavernous sinus. Patients present with painful, steroid-responsive ophthalmoplegia and have episodic, unilateral orbital, or retro-orbital pain. The ophthalmoplegia (CN II, III, IV, VI) occurs simultaneously or within the first 2 weeks after the onset of pain. Facial sensation and visual acuity may be diminished. Other pathologic conditions should be excluded by physical examination or neuroimaging. Tolosa-Hunt syndrome is thought to be caused a nonspecific, granulomatous inflammatory infiltrative process with no obvious specific pathologic trigger in the region of the posterior superior orbital fissure, orbital apex, or cavernous sinus. The syndrome typically responds quickly (within 72 hours) to treatment with steroids.

**Raeder’s Paratrigeminal Syndrome (Paratrigeminal Oculo-Sympathetic Syndrome).** This uncommon facial pain syndrome presents with first division trigeminal neurogenic pain, sensory loss, or both, sympathetic dysfunction (miosis, ptosis, or both), but with normal forehead sweating (compared to Horner syndrome). The symptom cluster is localized to the middle cranial fossa medial to the trigeminal ganglion and lateral to the anterior clinoid process. Neuroimaging is necessary and important and, if negative, should be repeated over a period of time to avoid missing an underlying abnormality. This syndrome is not specific to any known pathologic condition.

**Herpes Zoster Ophthalmicus (Postherpetic Neuralgia).** Herpes zoster ophthalmicus is caused by reactivation of latent herpes zoster virus in the gasserian ganglion, which typically involves the ophthalmic division of the trigeminal nerve. Ocular symptoms can occur after zoster eruption in any branch of the trigeminal nerve but are particularly common when vesicles form on the nose, reflecting nasociliary nerve involvement (Hutchinson’s sign). Cranial neuropathies can also occur, usually weeks after the skin eruptions.

**Temporal Arteritis.** Temporal arteritis presents with periorbital or temporal headache, facial pain, and occasional neck pain. There may be “jaw claudication,” scalp tenderness, and visual loss. The headache is mild to severe and of acute or gradual onset; the patient is typically without a history of headache or of changes in headache pattern. Ocular motor disturbances, dizziness, vertigo, and hearing impairment and cervical myelopathy may be present, and very frequently, the superficial temporal artery is thickened, nodular, and pulseless.

Patients with temporal arteritis are generally male, smokers, and over age 50. The disorder may be associated with polymyalgia rheumatica, especially in elderly patients. If untreated or inad-
equate to treated, it may result in unilateral or bilateral blindness, brainstem strokes, and transient ischemic attacks. A careful history and physical examination gives a high index of suspicion for this diagnosis. Additionally, blood studies (elevated Erythrocyte sedimentation rate, C-reactive protein) and temporal artery biopsy may be necessary. However, biopsies can have false negatives as a result of skipped lesions; therefore, bilateral biopsies are often recommended. Treatment consists of immediate high-dose corticosteroids.

**Temporomandibular Disorders**

Temporomandibular disorder (TMD) typically presents with facial pain, limited and dysfunctional mandibular movements, temporomandibular joint noises (clicking, popping, crepitus), and a change in the way the teeth meet on mouth closure. In addition, reports of eye and periorbital pain, ear pain and stuffiness, headache, neck pain, dizziness, and limitation of neck movement are often present. For a more detailed description of evaluation and treatments for TMD, see the section on TMD in this chapter.

**Pain Referred to the Eye from Intracranial Disease**

Pain from intracranial diseases, especially those involving the dura and cavernous sinus, may be referred to the eye and orbit. The ophthalmic division of the trigeminal nerve serves the eye and the orbit. Interestingly, a tentorial-dural branch joins the ophthalmic division in the cavernous sinus, receiving sensory innervations from much of the intracranial dura, the arteries at the skull base, and the major venous structures. Inflammation, neoplasm, or ischemia involving intracranial structures may cause pain, which is often referred to as the ipsilateral eye. Therefore, eye pain that remains unexplained after a thorough ophthalmic evaluation may require neuroimaging to rule out an intracranial disorder.16,23,28

**Pain Attributed to Disorders of the Ear**

Otalgia is defined as pain localizing to the ear. Primary otalgia is pain that originates from the ear (see Table 67.14). Referred otalgia does not have a distinct otologic cause and is also called secondary, nonotogenic otalgia. Although earache is a frequent symptom, systematic population-based studies of the epidemiology of the different forms of pain associated with diseases of the ears have not been conducted. However, 97% of cases of otitis media present with earache,29 and otalgia was found to be referred in as many as 50% of adults in a general medicine population.24

An analysis of the symptoms of nasopharyngeal carcinomas revealed that deafness and earache, encountered in 85% of patients, were the most common symptoms beside swelling of the throat.30

The cranial and cervical nerves supply sensory innervation to the ear. The auriculotemporal branch of the mandibular division of the trigeminal nerve supplies sensation to the anterior aspect of the auricle helix (including the tragus), the anterior aspect of the external auditory canal, and the anterior aspect of the lateral tympanic membrane. The great auricular nerve derived from the cervical nerve plexus (C2 and C3) innervates the remaining parts of the lateral surface of the auricle and the medial surface as well. Afferents from facial, glossopharyngeal, and vagus nerves supply the posterior aspect of the external auditory canal, the posterior aspect of the lateral tympanic membrane, the postero-medial aspect of the auricle, and a patch of skin on the mastoid process. The middle ear receives sensory afferents primarily from the glossopharyngeal nerve as part of the tympanic plexus. The sensory afferents of the tympanic plexus are largely formed by Jacobson’s branch of the glossopharyngeal nerve. The vestibulocochlear nerve does not mediate pain afferents from the inner ear because it does not carry pain fibers. Therefore, marked inner ear pathology may develop without otalgia (see Table 67.14).30,31

In the ear’s embryologic development, the otic vesicles come to rest between branchial nerves 1, 2, 3, and 4. The sensory and motor nerves of these arches are CNs V, VII, IX, and X, respectively.32–34

Four distinct regions of afferent innervations of the head and neck sites refer pain to the ear: (1) the inferior gingiva, floor of the mouth, inferior buccal mucosa, and the anterior two thirds of the tongue, all of which are innervated by the third branch of CN V; (2) the tonsillar fossae, lateral bases of the tongue, and some of the inferior nasopharynx innervated by branches of CN IX; (3) the posterolateral oropharynx, hypopharynx, medial base of the tongue, and occasionally a small portion of the inferior nasopharynx innervated by mixed branches of CNs IX and X; and (4) the supraglottic larynx and lingual and laryngeal surfaces of the epiglottis innervated solely by branches of CN X.32–34

Otalgia may have otologic (primary) or nonotologic (secondary) causes. A systemic approach to diagnosis is necessary to prevent overlooking a serious condition and to establish the diagnosis and proper therapy.

**Primary Otalgia**

Primary otalgia is pain with a cause in the ear. Usually, it can be diagnosed by examination of the pinna, auditory canal, and tympanic membrane.

**Pinna**

Primary pinna pain may be caused by injuries or trauma, such as lacerations, burns, frostbite, or infections. Persistent minor lesions should be biopsied to rule out underlying malignancy.

**External Auditory Canal**

Otitis externa, sometimes referred to as “swimmer’s ear,” is an inflammation of the external auditory meatus with resulting edema, otorrhea, pruritus, and otalgia. The otalgia of otitis externa is mediated by sensory afferents of the auriculotemporal nerve, the complex of facial glossopharyngeal and vagus nerves, and the cervical nerves. External otitis arises from acute inflammation after an ear trauma, inadequate cleansing of the external auditory canal, or lengthy contact with liquids in bacterially contaminated water, especially in lakes or swimming pools. Ear wax buildup also may be responsible for earache and pressure in the ear.

The diagnosis is based on physical examination of the external auditory canal for edema, erythema, debris (desquamated epithelium), and otorrhea. Physical findings may be minimal, with only slight edema or hyperemia. In such cases, a history of recent water exposure or preceding ear instrumentation may be useful. Management includes suctioning of any debris or fluids from the external auditory canal, treatment with antibiotic and steroid otic drops, and dry ear precautions.29,35

Malignancy must be considered when evaluating a patient with otalgia and an apparent refractory otitis externa. A primary neoplasm of the external auditory canal will too often be misdiagnosed as an otitis externa, potentially resulting in a costly delay in treatment. Patients with malignant otitis externa may have severe otalgia, a severe form of otitis externa that has involved the bone and marrow of the skull base. It is usually found in diabetics or otherwise immunocompromised patients, so this diagnosis must be carefully considered in these patients with otalgia. Timely diagnosis and prompt referral of a patient with malignant otitis externa is crucial because progression from bone involvement to death is rapid.29,35

**Middle Ear**

Infection of the middle ear, otitis media, is likely the most common cause of primary otalgia.36 The pain from inflamed mucosa

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**Part IV: Pain Conditions**
in these patients is mediated by way of the glossopharyngeal nerve, which supplies sensation to the middle ear and medial aspect of the tympanic membrane. An acute infection of the mucous membrane of the middle ear usually stems from an infection of the upper air passages with dysfunction of the eustachian tube. Rhinitis and adenoid inflammation may also be causes of acute otitis media. The tympanic membrane will be red and swollen. Occasionally, a purulent discharge is present. Gradenigo syndrome, defined as the triad of otalgia, otorrhea, and abducens nerve palsy, results from bacterial infection of the petrous apex air cells. Such an infection causes dysfunction of the abducens nerve because it passes through a dural tunnel in proximity to the petrous apex. These complications must be considered in a patient with otalgia and a history of recent otitis media.

Other Primary Causes

Primary neoplasms arising from within the ear or skull base may cause primary otalgia. Such lesions may originate from the skin of the external ear or from glandular tissues of the external or middle ear. A careful otologic and CN examination will help make the diagnosis. The sternocleidomastoid muscle attaches to the mastoid process, and overuse or spasm may manifest as a dull, aching otalgia.

Secondary (Referred) Otalgia

In the absence of otologic factors, the pain is termed secondary, nonotologic, or referred otalgia. The classic definition of referred pain is pathology in one part of the body that gives rise to pain in another nonpathologic site. Referred ear pain may result from pathologic factors involving the sensory supply of the CN V, IX, and X and the spinal nerves C2 and C3. Irritation of the sensory branch of the facial nerve (CN VII) is not true reflex or referred pain. It is usually the initial symptom of Bell palsy or Ramsay Hunt syndrome, and the diagnostic finding of facial paralysis usually occurs within 24 to 48 hours after the onset of pain.33,34

Trigeminal Nerve Referred Pain. Pain referred to the ear from the second and third divisions of the trigeminal nerve is usually located anterior to the tragus and along the anterior wall of the external auditory canal, which is supplied by the auriculotemporal nerve. Any disease process involving the anterior two thirds of the tongue, the floor of the mouth, gingiva, mandible, anterior half of the palate, teeth, infratemporal fossa, paranasal sinuses, and the submandibular or parotid glands may result in trigeminal-nerve referred pain. The most common otalgia of fifth nerve origin are dental disorders.

Glossopharyngeal Nerve (CN IX) Referred Pain. Referred pain over the glossopharyngeal nerve may result from infections, ulcerations, and tumors of the palate tonsil, nasopharynx, eustachian tube, posterior half of the palate, and the posterior third of the tongue. This pain is usually felt deep in the ear, in contrast to the more superficially located pain mediated by the trigeminal nerve. Such ear pain is frequently the only symptom after tonsillectomy and adenoidectomy.

Vagus Nerve (CN X) Referred Pain. Ulcerative lesions due to malignancy or chronic infections in the larynx or hypopharynx may irritate the superior laryngeal branch of the vagus nerve, causing pain referred to the ear.

Spinal Nerves C2 and C3 Referred Pain. Pain in the mastoid area and over the posterior portion of the pinna is mediated by the great auricular nerve, which is derived from the spinal nerves C2 and C3. The most common cause of cervical pain is trauma to the cervical spine. Cervical arthritis, cervical disks, cervical tumors, and muscle traction headache should be considered in the differential diagnosis. The differential diagnosis for referred otalgia is extensive: the more common causes are described below.

Temporomandibular Disorders. In addition to otalgia, otologic manifestations of temporal mandibular joint (TMJ) disorders can include aural fullness, tinnitus, and vertigo.4,6,37 A study of approximately 430 patients with TMJ pain found that otalgia was the presenting symptom in 48%.34 In this study, the TMJ syndrome (and hence otalgia) was successfully managed with conservative therapies such as heat, massage, patient education, occlusal splints, and pain control.

Eagle Syndrome. Eagle syndrome is defined as otalgia, facial pain, sore throat, globus, or dysphagia secondary to elongation of the styloid process or ossification of the stylohyoid ligament. The abnormal styloid process may produce pain through different mechanisms, one of which is direct compression and irritation of the trigeminal, facial, glossopharyngeal, or vagus nerves. The styloid process is typically 20 to 30 mm long. However, 4% of the population has a styloid process longer than 30 mm and of these, only 4% are symptomatic. The degree to which an elongated styloid process causes pain is somewhat poorly defined and controversial.4,38

Gastroesophageal Reflux Disease. A large number of symptoms have been linked to extraesophageal reflux of gastric contents, including laryngitis, hoarseness, pharyngitis, bronchospasm, laryngospasm, and chronic cough.39 Gastroesophageal reflux disease (GERD) can cause otalgia by irritating the upper aerodigestive tract in the sensory distribution of the glossopharyngeal and vagus nerves. Because these nerves also innervate the ear, irritation and damage from acidic gastric secretions may be perceived as originating within the ear. The reflux of gastric secretions can also potentially extend superiorly to the eustachian tubes, irritating the ear directly. The diagnosis of reflux-related otalgia should be considered in all patients with otalgia, a normal otoscopic exam, and other symptoms of GERD. Consultation with a gastroenterologist may be beneficial in managing these patients.39,40

Neoplastic Process (see Table 67.14). Malignancies of the upper aerodigestive tract and tumors in various sites of the head and neck can cause otalgia. Tumors on the anterior aspect of the tongue can also manifest as otalgia if they affect the chorda tympani branch of the facial nerve. Nasal and sinus malignancies may present with otalgia secondary to eustachian tube dysfunction or direct neural involvement. In the latter case, the otalgia is mediated by the afferents from the posterior lateral nasal nerves by way of the sphenopalatine ganglion, which is associated with the second division of the trigeminal nerve. Lesions arising from the infratemporal fossa can cause otalgia by involvement of Arnold’s nerve (the auricular branch of vagus nerve) or Jacobson’s nerve (the tympanic branch of the glossopharyngeal nerve). Many of these patients face a costly delay in diagnosis if malignancy is not considered as a cause.

Treatment of referred otalgia must be directed specifically to the relevant local causes. Such causes may include pulpitis, periapical dental abscess, glossitis, sinusitis, benign or malignant growth in the mouth or sinuses, dental malocclusion, Ramsay Hunt syndrome, tonsils, hypopharynx, or larynx inflammation, or growths in the nasopharynx or eustachian tube.

Pain Attributed to Disorders of the Nose and Paranasal Sinuses (see Table 67.14)

These disorders are grouped together because the paranasal sinuses communicate with the nasal passages through small ostia. Most important among these disorders is sinusitis as it is com-
monly linked to headache by physicians and the general public alike. “Sinus trouble” as a cause of headache is a source of many controversies, including how to name them: “sinus headache,” “rhinosinusitis headache,” and “sinogenic facial pain” are used to refer to the same disorder.

Rhinosinusitis

Rhinosinusitis is the inflammation of the nasal passages (rhinitis) and one or more of the paranasal sinuses (maxillary, ethmoid, frontal, or sphenoid). The term is more accurate than “sinusitis” because rhinitis usually precedes sinusitis, the mucosa of the nose and sinuses are contiguous, both conditions may involve nasal obstruction and discharge.41,42

Sinusitis is overdiagnosed as a cause of headache and facial pain because of the belief that pain over the sinuses must be related to the sinus. Many of the 60% of patients with unrecognized migraine attribute their symptoms to sinusitis.43 Rhinosinusitis is an uncommon cause of facial pain: more than 80% of patients with purulent secretions visible on nasal endoscopy have no facial pain, most patients with nasal polyposis do not have pain, and facial pain persists in a large proportion of patients after endoscopic sinus surgery.44-47 Paradoxically, sinus disease also tends to be underestimated, and a potentially dangerous condition, sphenoid sinusitis, is frequently missed.48

Clinical Features. Diagnostic criteria for rhinosinusitis are defined in the ear, nose, and throat literature.49 In this diagnostic scheme, rhinosinusitis is subdivided into acute, recurrent acute, subacute, chronic, and acute exacerbations of chronic. Acute sinusitis lasts from 1 day to 4 weeks, subacute sinusitis from 4 to 12 weeks, chronic sinusitis from more than 12 weeks. Headache is considered a minor criterion for the diagnosis of acute rhinosinusitis, and headache in the absence of other diagnostic criteria is not considered to be diagnostic of sinuses.41,50,51

Key points in the history of pain secondary to rhinosinusitis are exacerbations of pain during an upper respiratory tract infection, an association with rhinological symptoms, pain that worsens when flying or skiing, and pain in response to medical treatment. Rhinosinusitis usually presents with facial tenderness and pain, nasal congestion, and purulent nasal discharge. Common signs and symptoms include anosmia or hyposmia, pain on mastication, and halitosis. Most cases of infectious rhinosinusitis that last less than 7 days are viral. Acute bacterial sinusitis in adults most often presents with 7 or more days of purulent anterior rhinorrhea, nasal congestion, postnasal drip, facial or dental pain or pressure, and cough, frequently at nighttime. Although approximately 50% of adults have fever and 60% of children have headache, facial pain, and fever often are of little value in diagnosing sinusitis. Williams et al.52 found that maxillary toothache was highly specific in making the diagnosis of rhinosinusitis: 93% of their patients with toothache had rhinosinusitis. However, only 11% of their patients had maxillary toothache.52,53

The headaches associated with rhinosinusitis are usually continuous. The location of the pain and the position that improves the headache varies on the sinus involved (Table 67.20).

Pain in acute maxillary sinusitis is usually in the cheek, gums, and maxillary teeth on the affected side. Acute frontal sinusitis causes frontal headache with tenderness over the sinus and on the medial side of the orbital floor, under the supraorbital ridge, where the frontal sinus is thinnest. Frontal sinusitis can result in brain abscess, meningitis, subdural or epidural abscess, osteomyelitis, orbital edema, and orbital cellulitis. Acute ethmoid sinusitis typically produces pain in between the eyes. Coughing, straining, and lying supine can worsen the pain, whereas keeping the head upright lessens it. Complications of ethmoid sinusitis include meningitis, orbital cellulites, and cavernous sinus thrombosis.54

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<th>TABLE 67.20</th>
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<tr>
<td>INTERNATIONAL HEADACHE SOCIETY CRITERIA FOR ACUTE SINUSHEADACHE</td>
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<tr>
<td>• Diagnostic criteria: pain in one or more regions of the head, face, ears, or teeth.</td>
</tr>
<tr>
<td>• Clinical, laboratory, or imaging evidence of acute rhinosinusitis (e.g., purulence in the nasal cavity, nasal obstruction, fever, CT, MRI, or fiberoptic nasal endoscopy findings).</td>
</tr>
<tr>
<td>• Simultaneous onset of headache and rhinosinusitis.</td>
</tr>
<tr>
<td>• Headache lasts &lt;7 days after remission or successful treatment of acute rhinosinusitis.</td>
</tr>
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It is necessary, therefore, to differentiate headaches caused by rhinosinusitis from so-called “sinus headaches,” which are chronic headache attacks fulfilling the criteria for migraine without aura with prominent autonomic symptoms in the nose or migraine without aura triggered by nasal changes.

Acute sphenoid sinusitis, which accounts for only 3% of all cases of acute sinusitis, is frequently misdiagnosed.48 Although sphenoid sinusitis rarely causes headache, it can lead to marked morbidity and mortality and so must be identified early and managed aggressively.

As mentioned above, the cavernous sinus is lateral to the sphenoid sinus. The cavernous sinus contains the internal carotid arteries and the third, fourth, fifth, and seventh CNs. The maxillary division of the CN V may indent the wall of the sphenoid sinus. The sphenoid walls can be extremely thin, and sometimes the sinus cavity is separated from the adjacent structure by just a thin mucosal barrier. Because of this proximity, infection may spread to these structures and present as a central nervous system infection or neurologic catastrophe.48,54

Headache is always present in acute sphenoid sinusitis and it may be frontal, occipital, or temporal and most commonly is a combination of these locations. Periorbital pain is common and vertex pain is rare. Nasal pain and discharge are present in only 30% of cases, and fever occurs in more than 50% of patients. Also, pain or paresthesias in the facial distribution of the fifth nerve and photophobia or eye tearing suggest sphenoid sinusitis.48 The headache and associated symptoms may lead to a misdiagnosis of migraine, meningitis, trigeminal neuralgia, or brain tumor. A severe, intractable, new-onset headache that interferes with care and is not relieved by simple analgesics should raise the suspicion of sphenoid sinusitis. Neuroimaging is necessary for a definitive diagnosis. Complications include bacterial meningitis, cavernous sinus thrombosis, subdural abscess, ophthalmoplegia, and pituitary insufficiency.48,54

Acute sinus headache is defined by the International Headache Society (IHS) diagnostic criteria in the setting of an infectious process requiring verification through imaging and confirmation by response to appropriate antibiotics. The IHS established the following diagnostic criteria for acute sinus headache (rhinosinusitis headache) (see Table 67.20):48

A. Purulent discharge in the nasal passage, either spontaneous or by suction
B. Pathologic findings on radiographic examination, CT, or transillumination
C. Simultaneous onset of headache and sinusitis
D. Headache location (see Table 67.3 for location of pain with infection of corresponding paranasal sinus)
E. The disappearance of the headache after treatment for acute sinusitis

These criteria may not be valid for diagnosing sphenoid sinusitis because the purulent discharge is often missing and headache may precede sinus drainage. The IHS has not validated chronic
sinusitis as a cause of headache or facial pain unless it relapses into an acute stage.

Obviously, the term “acute sinus headache” or what is referred to as “rhinosinusitis headache” in this chapter does not address the primary headache disorder with secondary nasal symptomatology, commonly referred to as “sinus headache.”

Plain sinus radiographs can diagnose acute maxillary or frontal sinusitis but are often inadequate for ethmoid or sphenoid sinusitis. CT is the optimal imaging study to assess the paranasal sinuses. The mucosa of the normal, noninfected sinus approximates the bone so closely that it cannot be visualized on CT. Therefore, any soft tissue seen within a sinus is abnormal. CT may reveal mucosal thickening, sclerosis, clouding, or air-fluid levels. Scans of the sinuses without contrast in the coronal plain are highly sensitive for detecting nasal and paranasal sinus disease, including disease in the ethmoid and sphenoid sinuses. The prevalence of reversible sinus abnormalities visualized by CT in patients who have the common cold is high. This fact suggests that CT may not be specific for bacterial infections. Anterior ethmoid sinus infection is found in every patient who had frontal or maxillary sinusitis.

MRI is more sensitive than CT in detecting fungal infections. In MRI, T2-weighted images are highly sensitive for detecting retained fluid and inflamed tissue of the sinuses, a fact that may lead to exaggerating the importance of otherwise unremarkable sinus disease, such as mild inflammation, small polyps, and retention cysts. Transillumination and ultrasonography of the sinuses have low sensitivity and specificity for detecting similar findings. Diagnostic endoscopy with the flexible fiberoptic rhinoscope permits direct visualization of the nasal passages and sinus drainage areas.

Differential Diagnosis. Migraine and tension-type headache are often confused with true sinus headache because of their similar locations. Some patients, in addition to having all the features of migraine without aura, have head pain in the face, associated congestion of the nose, and headache triggered by weather changes. These patients do not have purulent nasal discharge or other diagnostic criteria of acute rhinosinusitis. The diagnostic criteria of rhinosinusitis and migraine are similar. Although facial pressure or pain, facial congestion, and nasal blockages are considered major criteria for rhinosinusitis, headache and fatigue are minor criteria, meaning that these symptoms have less diagnostic value but are not necessarily less frequent or less intense. In migraine, the semilunar emphasis for diagnosis is the severity, quality, and location of a headache associated with gastrointestinal or sensory symptoms. Nasal symptoms, including congestion, facial pressure or pain, and rhinorrhea, are commonly reported symptoms associated with migraines but are often ignored because they are not considered essential for the diagnosis of migraine. Therefore, it is important to differentiate headaches caused by rhinosinusitis from headaches fulfilling the criteria of migraine without aura with prominent autonomic symptoms in the nose or of migraine without aura triggered by nasal changes, the so-called “sinus headaches” ([Table 67.21]).

Management (see Table 67.21). Acute rhinosinusitis causes excruciating pain; therefore, analgesia is important. In one study, analgesia successfully treated nearly 80% of patients with maxillary sinusitis.

Emergency treatment goals are to facilitate drainage of the congested nasal sinuses and to eliminate the pathogenic bacteria. Steam and saline prevent crusting of secretions in the nasal cavity and facilitate mucociliary clearance. Locally active decongestants provide symptomatic relief by shrinking inflamed and swollen nasal mucosa. Oral decongestants should be used if prolonged treatment (>3 days) is necessary. These agents are α-adrenergic agonists that reduce nasal blood flow without the risk of rebound vasoconstriction. Mucoevacuant (guaifenesin) and intranasal steroids may improve the symptoms, but antihistamines are not helpful. Most patients with rhinosinusitis respond to treatment with antibiotics. Amoxicillin is the first choice, unless the patient has been treated within the previous month or lives in an area that has a high prevalence of β-lactamase-resistant H influenza. There is no clear evidence that culturing purulent secretions contribute to managing acute rhinosinusitis, but obtaining a culture and defining its antibiotic sensitivity may help, particularly if there are orbital or intracranial complications.

Acute frontal and sphenoid sinusitis require immediate referral to an otolaryngologist for treatment to avoid intracranial complications. Consultation should also be considered when the symptoms are not relieved with at least two consecutive 2-week courses of antibiotics.

Sometimes rhinosinusitis does not respond to medical treatment and surgical intervention is necessary to relieve worsening and excruciating pain. The goal of surgery is to improve sinus drainage by enlarging the orifices, removing obstructive anatomic structures, or both. Endoscopic sinus surgery alleviates the facial pain in 75% to 83% of cases, producing greater improvement in headache than in facial pain.

### TABLE 67.21

**AN OTOLARYNGOLOGY, NEUROLOGY, ALLERGY, AND PRIMARY CARE CONSENSUS ON DIAGNOSIS AND TREATMENT OF SINUS HEADACHE**

**Diagnostic Recommendations:**
- Stable pattern of recurrent, self-limiting headache associated with rhinogenic symptoms are most likely migraine
- Prominent rhinogenic symptoms associated with fever, purulent discharge with headache as one of several complaints (pain) is likely rhinogenic in origin
- MRI or CT as appropriate based on headache history, patterns, changes, and physical signs
- Referral to headache specialist for new onset, frequent headache, headache associated with neurological symptoms or signs, or headache that does not respond to appropriate therapy (migraine or rhinogenic)

**Therapeutic Recommendations:**
- Migraine with no evidence of infection should be given a trial of migraine-specific medication and scheduled for follow-up evaluation
- Noninfectious rhinogenic symptoms with headache as a minor complaint should be provided with a trial of nasal steroids and/or selective antihistamines and/or oral decongestants


**Isolate Rhinogenic Disorders Causing Headache**

Rhinogenic headache and facial pain can be caused by septal impaction or contact, rhinitis (allergic or vasomotor) and nasal polyps, trauma, intranasal tumors, and septal hematoma. In patients without evidence of acute or chronic sinusitis, nasal polyps, or a tumor, Chow found that pain was caused by a septal spur in 12 of 18 patients who had rhinologic sources for their primary symptom of facial pain or headache.

**Deviated Nasal Septum.** A deviated nasal septum can produce symptoms similar to those of nasal obstruction. Symptoms may be marked if the deviation is in the region of the nasal valve. However, deflection of the nasal septum is not important as a possible cause of headache.
Inflammatory Rhinitis. Inflammatory rhinitis is accompanied by rhinorrhea, fever, pain affecting the middle part of the face and the distribution of the first and second trigeminal branches, and symptoms of an infection of the upper respiratory tract. Inflammatory rhinitis differs from allergic rhinitis in having more neutrophils on a nasal swab, whereas allergic rhinitis will show an increase in eosinophilic leucocytes.

Allergic Rhinitis. As a rule, allergic rhinitis does not cause primary pain, but it may give rise to acute sinusitis in which facial pain is a secondary development.

Vasomotor Rhinitis. The symptoms of vasomotor rhinitis are similar to those of allergic rhinitis but with less sneezing, and the patient does not test positive for allergies. The pathophysiology involves an imbalance between the parasympathetic and sympathetic autonomic nerve supply of the nasal mucosa. The former predominates, the increased vascularity causing nasal obstruction.

Pain Attributed to Temporomandibular Disorders

TMD are defined as a subgroup of craniocfacial pain disorders that involve the TMJ, the masticatory muscle system, and the associated head, face, and neck musculoskeletal complex (muscles, ligaments, and joints). Patients with TMD most frequently present with pain, limited or asymmetric mandibular motion, and TMJ sounds. The pain or discomfort is located around the jaw, TMJ, and the muscles of mastication. Commonly associated symptoms include ear pain and stuffiness, tinnitus, dizziness, eye pain, neck pain, arm and shoulder pain, and dysfunction and headache. In some cases, the onset is acute and symptoms are mild and self-limiting. Other patients experience chronic TMD with persistent pain in association with a combination of physical, behavioral, psychologic, and psychosocial symptoms similar to those of other chronic pain syndromes. An estimated 75% of the U.S. adult population has experienced one or more of the signs and symptoms of TMD. Epidemiologic studies indicate a prevalence of 40% to 75% of adults having at least one sign of joint dysfunction and approximately 33% of persons having at least one symptom of TMD. Some signs appear to be relatively common in the general population; TMJ sounds and deviation on opening occur in approximately 50% of healthy people. Other signs are relatively rare; limited mouth opening and occlusal changes occur in fewer than 5% of the population. These disorders are disorders of middle-aged adults (ages 20 to 50), with women seeking care more than men (female:male ratio ranges from 3:1 to 9:1). Despite large numbers of people experiencing signs and symptoms of TMD over their lifetime, only 5% to 10% of these individuals are believed to actually need treatment.

Temporomandibular Disorders: A Triad of Dysfunctions

At least three distinct and separate dysfunctions create or affect the symptoms described by the TMD patient:

I. Muscle disorders (myofascial pain dysfunction) are related to muscle dysfunction, often leading to muscle spasms, pain, and dysfunction. This type of dysfunction can occur in any skeletal muscle. The triggering area lies in the fascial coverings and attachment zones of the muscles, thus the term myofascial. This syndrome is sometimes incorrectly referred to as myofascial pain dysfunction.

II. Temporomandibular joint articular disorder (TMJD) is related to specific problems in the TMJs. These problems may range from joint sounds to locking, pain, and degenerative changes of the joints themselves. Invariably, muscle dysfunction is a secondary effect of true TMJD.

III. Cervical spinal dysfunction is related to the spinal column, vertebrae, and the associated ligaments and muscles. The majority of symptoms not directly related to the jaw muscles are triggered or affected by this syndrome.

TMDs are classified in the eleventh major category of the IHS’s Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias, and Facial Pain as headache or facial pain associated with disorders of the cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cranial structures (see Table 67.3). The American Academy of Orofacial Pain adopted a part of this classification and broadened it to include a more focused classification of TMD (Tables 67.22 and 67.23). In this classification, TMDs are broadly divided into two major groups: the masticatory muscle disorders and the articular disorders. The masticatory muscle disorders include myofascial pain, myositis, myospasm, local myalgia, myofibrotic contracture, and neoplasia. The articular disorders include congenital and developmental disorders, disc derangement disorders, temporomandibular dislocation, inflammatory disorders, osteoarthritis, ankylosis, and fracture. The most common muscle disorders are localized myalgia and myofascial pain syndrome. The most common articular disorders are disc derangement disorders and osteoarthritis.

Treatment of TMD has included, among other therapies, oral orthopedic jaw appliances, occlusal adjustment, reconstructive orthodontic correction, biofeedback, biobehavioral stress management, psychotherapy, nutritional guidance, physical therapy, acupuncture, pharmacotherapy, and surgical management. Each of these techniques is supported by some degree of success and therefore merits notice. However, the key to successful treatment is an accurate diagnosis, which relies on knowing the symptoms and their probable causes on an interdisciplinary approach (Table 67.24).

<table>
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<tr>
<td>TEMPOROMANDIBULAR JOINT ARTICULAR DISORDERS</td>
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<tr>
<td><strong>Congenital or developmental</strong></td>
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<tr>
<td>Aplasia</td>
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<td>Hypoplasia</td>
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<td>Hyperplasia</td>
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<td>Neoplasia</td>
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<td><strong>Disc derangement disorders</strong></td>
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<td>Disc displacement with reduction</td>
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<td>Disc displacement without reduction</td>
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<tr>
<td><strong>Temporomandibular joint dislocation</strong></td>
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<tr>
<td><strong>Inflammatory disorders</strong></td>
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<tr>
<td>Capsulitis/synovitis</td>
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<tr>
<td>Polyarthritis</td>
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<td><strong>Osteoarthritis (noninflammatory)</strong></td>
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<tr>
<td>Primary osteoarthritis</td>
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<td>Secondary osteoarthritis</td>
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<td><strong>Ankylosis</strong></td>
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<td><strong>Fracture</strong></td>
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Clinical Presentation

The most common symptom of TMD is muscle pain. It is usually accompanied by restricted movement. Patients often also present with reduced jaw opening, as well as impaired range of motion in the cervical vertebrae, shoulders, and arms. Other commonly associated symptoms of TMD are listed below.

Headache. Symptoms of TMD-related bilateral head and face pain involve multiple postural muscles, the muscles of mastication, or both. The pain is typically of moderate intensity, dull and aching in quality, and described as deep and constant.\(^71\)–\(^75\) Pain is often exacerbated by use of the affected muscles. Morning headaches may be related to nocturnal bruxism or sleep disorders,\(^76\) whereas increasing pain during the day may be related to masticatory muscle use or head posture.\(^77\)

Front of Head. Patients reporting pain in the front of the head often refer to it as "sinus headache." The pain is usually accompanied by pressure along the upper anterior teeth, bridge of the nose, and behind the eyes. Chronic front pain generally indicates a primary headache disorder, not chronic sinus disease. These symptoms can also be caused by a reduced posterior occlusal dimension, causing heavy incisal contact, resulting in pain of pressure in the anterior vortex of the face. A band-like feeling of the front of the head can also be brought about by posterior neck muscle contractions or muscle tension of the frontalis muscle.

Side of the Head. Temporal headaches are mainly related to muscle contraction and fiber spasm of the temporalis muscle. The temporalis muscle has three groups of muscle fibers: anterior, middle, and posterior. The anterior fibers bring the lower jaw up and forward, and the middle and posterior fibers swing the jaw to full closure and retract the mandible.

Clenching, grinding, or biting on objects while the jaw is displaced anteriorly (edge-to-edge) generally creates pain in the anterior temporal group (i.e., the patient has pain in the "temple" area). Individuals who work at desk jobs with their heads forward and down tend to clench and grind in this position because gravity

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<tr>
<td><strong>TEMPOROMANDIBULAR DISORDERS</strong></td>
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<tr>
<td><strong>Diagnosis</strong></td>
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has a greater affect on the mandible. This condition is further aggravated by habits such as pencil or pen biting, pipe smoking, or gum chewing.

Clenching or grinding during sleep or clenching with the jaw in a posterior position tends to tire the middle and posterior group of fibers, and the pain is more posteriorly located. Generally, the temporalis is affected in any dysfunction of the lower jaw.

**Back of Head.** Deep, dull pain, constant and aggravating in the back of the head, is usually a result of spasms of the trapezius and sternocleidomastoid muscles. These muscles are long and strong, and under tension, they pull on their bony attachments to the skull—the occiput and mastoid areas. This pulling leads to soreness in the bone and to deep dull pain radiating up the back of the head and down the neck. The muscle tension may be independent of, or secondarily related to, displacement of the cervical and upper thoracic vertebrae.

**Face Pain.** Pain in the sides of the face or pain described by the patient as "sinus" pain in the zygomatic or orbital area may also have a musculoskeletal origin.69 Clenching, acute or chronic stress, and reduction of dental height related to the loss of posterior teeth combined with daytime tooth clenching and acute or chronic stress can create muscle trigger points or muscle fatigue. Patients are often aware of this condition after meals and report "a heavy and tired feeling" in the jaw muscles. Face pain related to sinuses and other pathologies are discussed separately in this chapter.

**Eye Pain.** Orbital pain symptoms are often described as unilaterally, constant, and "boring." Orbital pain is frequently seen in patients with TMD that includes pain involving the eye and periocular region.69,78–80 Patients with a history of trauma, chronic upper cervical vertebral subluxations, or nerve root impingements related to the occiput and the atlantoaxial region may present with orbital symptoms. In addition, entrapment of the greater occipital nerve at the occiput level can also produce this type of pain, which is often diagnosed as occipital neuralgia. Orbital pain often responds to physical medicine, along with changes in head posture and mandibular position through the use of dental bite appliances.

**Ear Symptoms.** Pain, stuffiness, and tinnitus may have a musculoskeletal cause.81,82 Mandibular posture in relationship to the maxilla can affect eustachian tube function and can precipitate middle ear infections in children, as well as ear stuffiness with changes in pressure in the ear in adults. Maxillary and mandibular dysfunctions aid in the development and maintenance of such symptoms.

Tinnitus and other types of ear sounds may also have a peripheral musculoskeletal cause. Specifically, cervical and mandibular postural factors are found in patients with tinnitus. A combination of physical medicine and dental jaw appliance therapy has been effective in some cases where trauma or aberrations in childhood growth and development have affected the proper expansion of the maxilla.

"Sharp, jabbing ear pain upon movement of the mandible is often seen in patients who have an internal derangement of the TMJ. Usually this derangement presents unilaterally and ipsilateral to the joint in question.

Ear pain and symptoms such as stuffiness in the absence of positive otologic findings are among the most common reasons to evaluate the patient for dental and maxillomandibular imbalance. Treatment often alleviates or reduces the impact of the symptoms on the patient.83–88

**Temporomandibular Joint Symptoms.** Pain and sounds related to the TMJs are very common.89,90 Patients commonly report clicking or grating noises in the "jaw joints." Generally, clicking is not accompanied by pain. Grating noises are usually unilateral and accompanied by pain radiating to the ear on movement. These noises may be related to trauma or bruxism in the presence of missing posterior teeth, resulting in injury or anterior disc displacement without reduction "locking" of the temporomandibular joint.

**Clicking.** Much has been written about clicking of the TMJs. Clicks can be classified as immediate-opening clicking, midopening clicking, late-opening clicking, and reciprocal clicking.

The reason for the click, however, remains the same. When the mouth opens, the condyle hits the back of, and "clicks under," the articulator disc. This situation can happen at any point during movement, depending on the relative position of the condyle and disc.

Typically, the articular disc is attached to the head of the condyle on the medial and lateral poles. The superior head of the lateral pterygoid is inserted into the anterior portion of the disc and soft, elastic tissue borders the disc posteriorly.

This arrangement holds the articular disc in position with the condyle in various jaw movements. If the joint is anatomically healthy, there cannot be a click. Tearing or stretching of the articulator attachments are a prerequisite for clicking.

Clicking can be caused by acute trauma, such as an automobile accident, whiplash, or a blow to the face. It can also be caused by chronic microtrauma, such as loss of posterior vertical dimension of occlusion or loss of teeth.

Dental interventions can also create this problem by retreating the condyle posteriorly during reconstruction or orthodontic retraction. Difficult extractions can also affect the joint-disc assembly.

**Grating Sounds.** Grating sounds generally occur in a later stage of TMJ articular dysfunction, along with articular cartilage degeneration. The disc is either torn and shredded or missing completely. The grating is caused by bone-to-bone friction and usually indicates osteoarthritic degenerative changes in the joint.

**Locking of the Joint.** The jaw lock mainly occurs after clicking has been evident for a while. The articular disc, which is biconcave under normal circumstances, loses its shape and becomes spherical or ball-like if anteriorly displaced for a period of time. In this case, the condyle cannot travel normally along the anterior wall of the glenoid fossa. The patient perceives this restriction as an inability to open the mouth fully. Sometimes the cartilage may fold on itself with the same clinical effect.

**Hypermobility.** Increased movement in the jaw comes when the TMJ ligaments are stretched or torn. This loss of integrity allows excessive movement, sometimes to the point of anterior open dislocation in a wide opening movement, such as yawning. Treatment is often a combination of dental therapies, jaw appliance therapy, and physical medicine and can be supplemented with treatment for bruxism and stress management through biofeedback relaxation and sleep medicine.

**Neck Pain.** Neck stiffness and pain are commonly part of the TMD complex.91–93 Trauma, poor posture, and musculoskeletal tension can have chronic effects on the cervical spine, creating pain, stiffness, and trigger point flare up in the muscles of the head and neck. The trapezial and cervical nerves help maintain head, neck, and jaw posture.94 Mastication and jaw function rely on all the anterior and posterior cervical muscles. In addition,
mandibular and head postures interact to maintain the airway space during function and sleep.

Studies of maxillomandibular position and the cervical spine have shown that reductions of the vertical dimension of the teeth and a deep bite can adversely affect cervical muscle function, leading to chronic stiffness, pain, and reduced range of motion. It is therefore important to assess the dental factors in patients with chronic neck pain.

Arm and Back Symptoms. Patients presenting with TMD may also commonly present with shoulder pain: pain radiating down the arm that may or may not be accompanied by tingling and or numbness. Physical examination often reveals thoracic outlet syndrome, costoclavicular syndrome, vertebral subluxations or nerve impingement of the brachial plexus, and even previously undiagnosed rotator cuff injuries.

Temporomandibular Joint Articular Disorders (see Tables 67.22 to 67.24)

The TMJ is a synovial diarthrodial joint that allows the temporal bone to articulate with the condylar head of the mandible. The joint allows for sliding as well as hinge movement of the mandible during functional mastication. The condyles are not perfectly round but are wider medio–laterally than anterior–posteriorly. Individual variations follow functional loads and depend on the thickness of connective tissue layers covering the articulating surface.

The condyles travel within their respective mandibular fossae. Each mandibular fossa or glenoid fossa forms the temporal component of the TMJ. This component is a concave area on the inferior border of the squamous part of the temporal bone and is also referred to as the articular fossa.

Between the bones that form the TMJ are interposing discs or articular cartilages. Each articular disc is formed of dense fibrous connective tissue and divides the joint cavity into two separate compartments: the upper discotemporal space and the lower discomandibular space. Both compartments are lubricated by synovial fluid. The inferior surface of the disc is concave to match the articular surface of the condyle, whereas the superior surface of the disc is convex to follow the concave surface of the articular fossa. The articular disc is firmly attached to the medial and lateral poles of the condyle. The lateral ligamentous attachments are relatively thin and weak compared to the medial pole attachments and tend to tear more frequently than the medial ligamentous attachments. This weakness is the reason for more anterior medial than lateral disc displacements.

Viewed sagittally, the articular disc is divided into three parts: a thicker anterior section called the anterior band, a middle thinner intermediary zone, and a broader posterior band that is the thickest of the three. The thinner intermediary zone, along with the two broader anterior and posterior zones, gives the articular cartilage its classic bowtie appearance on MRI.

In the adult, the central part of the articular disc is avascular and lacks innervations, which allows for changes in the central thin part of the disc to occur without pain. The articular disc has a relatively random arrangement of type I collagen fibers, elastic fibers, and glycosaminoglycans comprised of chondroitin sulfate, dermatan sulfate, and hyaluronic acid. The discs allow for rotation in the upper joint compartment and translation in the lower joint compartment. Thus, rotation is approximately the first 22.5 mm of mouth opening, and translation from that point to full mouth opening ranges from 45 to 53 mm between the front teeth.

In each joint, the anterior part of the articular cartilage attaches to the superior head of the lateral pterygoid muscle, and the inferior head of the lateral pterygoid muscle attaches into the fovea of the condyle. In humans, this attachment of the superior head of the lateral pterygoid is variably inserted into 40% to 60% of the articular disc. The posterior part of the articular cartilage blends into loose retrodiscal tissue consisting of blood vessels, loose connective tissue, and nerves.

On mouth closure, the retrodiscal tissue is squeezed like a sponge and allows the condyle to be fully seated in its fossa. As the mouth opens and the condyles move forward in their respective fossa, the blood vessels in the retrodiscal tissues expand to fill the void left by the translating condyles and their interposing articular cartilages. This act of a sponge being squeezed and then being filled is repeated during jaw function. This region can be injured if the mouth suddenly opens and closes as a result of a blow or injury to the mandible. This injury can lead to bleeding in the joint space, followed by pain and limitation of movement.

Congenital or Developmental Disorders. Congenital or developmental disorders, such as aplasia, hypoplasia, hyperplasia, and neoplasia, can be odontogenic or nonodontogenic and primarily present as esthetic and functional problems. Neoplastic lesions, such as osteomas and osteoblastomas of the bone, produce pain in more advanced stages as do other primary tumors, such as chondroblastoma and benign giant cell tumors. The most common metastatic tumors are squamous cell carcinoma, nasopharyngeal tumors, and parotid gland tumors, such as adenoid cystic carcinomas.

FIGURE 67.1 A,B. Gross anatomy of the TMJ as seen in sagittal sections. bz, bilaminar zone of disc; c, mandibular condyle; d, intra-articular disc; e, eminence; eac, external auditory canal; f, glenoid fossa; lfp, lower head of the lateral pterygoid muscle.
Disc derangement disorders or articular disc displacements, by far the most common TMJ articular disorders, are characterized by an abnormal position of the articular disc relative to the head of the condyle or temporal fossa. Disc displacement is usually marked by a “clicking or popping” sound in the TMJ when the mouth is opened and closed. Pain is initially not part of the presenting symptoms, as long as there is full function. Disc displacements suggest torn or stretched collateral discal ligaments that bind the disc to the condyle.

Disc displacements are usually anterior or anteromedial, although posterior and lateral displacements have been described. Anterior or anteromedial displacement may be related in part to the fact that the thinnest discal attachments are on the lateral pole of the condyle, as well as the medial direction of pull by the lateral pterygoid and the inward condylar movement during mouth opening.

Disc Displacement With Reduction. A clicking sound on mouth opening and closing is classified as disc displacement with reduction. The term reduction describes the process of the misaligned disc temporarily coming back (or slipping back) to its proper interposition between the condyle and fossa during full mouth opening (see Fig. 67.2). On closing the mouth, the disc again displaces as the teeth come closer together. This repetitive, ongoing displacement on opening and closing produces a reciprocal noise (clicks) and is hence termed “reciprocal clicking.” Given the very common nature of its occurrence, this displacement may actually represent a stage of physiologic accommodation that need not be treated. Where displacement progresses, the incidence of intermittent “locking” may increase as a result of the momentary impedance of the disc as it follows the path of the condyle. This stage generally occurs as a sequel of a chronic clicking condition in patients who tend to clench and grind their teeth at night (nocturnal parafunction) and who have missing posterior teeth with subsequent overclosure of the bite. The teeth act as the stops for the TMJs and support the ultimate position of the TMJ on full closure. Good dental vertical dimension without shift on closure is essential in reducing the risk factors for progression. Bite appliance therapy is effective in treating muscular as well as disc displacement problems.

Disc Displacement Without Reduction. Progression to the next stage is sometimes referred to as a “closed lock.” In this stage, the disc has been permanently displaced, and its shape has been deformed so that it prevents the condyle of the mandible from translating to a full open position (Fig. 67.3). Jaw opening is
usually limited to 22 to 25 mm, or about the length of the tips of two fingers inserted between the upper and lower incisors. Pain may reduce chewing capacity, the mandible deviates to the side of the lock, the joint becomes inflamed, and normal occlusion or “bite” may be disrupted. MRI is the standard for assessing the soft tissue of the articular cartilage and its displacement, whereas CT is generally done to assess hard tissue for chronic osteoarthritic or bony changes.\textsuperscript{110,111}

**Temporomandibular Joint Dislocation.** In TMJ dislocation, or open lock or condylar subluxation, the condyle translates beyond the anterior eminence of the articular fossa and becomes trapped in this open-mouth position. Chronic hypertranslation can usually be managed by having the patient physically manipulate the jaw back into position. The patient learns to relax the jaw-closing muscles and to slip the condyle back into position. The most common subluxation occurs during yawning or opening the mouth widely when eating.

If the problem is related to trauma or a sudden acute translation, the subluxation is considered to be an acute dislocation. Acute dislocation requires medical intervention in which the muscles are relaxed by anesthesia, analgesics, or injected into the muscles and joint, followed by manipulating the joint downward and backward to let it slip past the anterior eminence of the glenoid fossa. Follow-up with anti-inflammatory medication, ice, and rest or a dental appliance may be necessary until the acute stage passes.\textsuperscript{112}

**Inflammatory Disorders.** Capsulitis and synovitis are relatively common in the TMJ secondary to macro- or microtrauma, irritation, or infections. These insults are accompanied by pain on movement and inflammation with extreme tenderness of the TMJ or on distraction of the joints. MRI may show effusions in the T2-weighted signal, the teeth may not be brought together completely, and pain may occur in the ear.

Joint inflammation may also be a result of systemic polyarthritis. Symptoms are similar to those in other joints of the body and are secondary to connective tissue diseases that affect the same population of patients.

**Osteoarthritis (Noninflammatory).** Primary osteoarthritis is a degenerative condition of the joints characterized by hard-tissue abrasion and degradation of the articular surface of the condyle related to overload. The condition is frequently seen in patients with a long history of missing and unplaced teeth or in patients with dentures due to remodeling effects. Remodeling is usually slow and generally painless in the early stages. Slow progression may remain relatively benign over the life span of the patient.

Primary osteoarthritis is usually identified by radiography, such as a dental panoramic image, tomography, or a dental CT scan and grating (crepitus) noises in the joint during movement.\textsuperscript{115} Secondary osteoarthritis is usually associated with a single prior event, such as trauma or infection, or by rheumatoid arthritis. An idiopathic degenerative condition primarily affecting adolescent girls is termed condylysis. It is seen as a sudden lysis of the condyle, which can create a shift of the jaw to the affected side and an anterior open bite. The cause of condylysis is not clear but the condition is associated with young women with rheumatoid arthritis.\textsuperscript{113}

**Ankylosis.** Ankylosis is usually related to joint trauma, with subsequent bleeding and restricted mandibular movement. Ankylosis maybe fibrous or bony in nature. Fibrous ankylosis usually is seen in the upper joint compartment as a result of adhesions forming after a joint bleed and prolonged immobility. The jaw still opens slightly, usually enough to accommodate 1 or 2 fingers placed horizontally between the central incisors. Bony ankylosis has no movement associated with it. Both conditions may require surgical release and postsurgical mobilization.\textsuperscript{114}

**Fracture.** Trauma to the chin, mandible, or any part of the face may result in bony fractures of the condylar neck, condylar head, bodies of the mandible and maxilla, and temporal fossa. Untreated, these fractures will generally result in reduced range of jaw motion, pain, and fibrosis or bony ankylosis. If the fracture is uncomplicated and results in a nondisplaced fracture fragment, immediate treatment may not be necessary, as long as function is not compromised (Fig. 67.5).\textsuperscript{115}

**Muscular Disorders**

Muscular pain and dysfunction are the most common symptoms of a patient with TMD. The muscles of the masticatory system are affected in the same way as other striated skeletal muscles of the body. Ligaments, nerves, and muscles all function as a complex system to stabilize the head on the shoulders, maintain a functional airway space, and allow three-dimensional movement of the mandible. This system is called the craniofacial-mandibular or the stomatognathic system. Breakdown in this complex and finely tuned system ultimately affects the musculoskeletal system, most commonly in the form of muscular disorders leading to pain in the head, face, and neck.

**Myalgia due to Trauma.** Masticatory muscles can be injured by acute muscle strain or by direct trauma. It is difficult to completely eliminate movement in the masticatory muscles because of the need for speech, swallowing, and chewing. If this is further compounded by dental parafunctional activities, the healing process takes longer.

Soft tissue injury results in bleeding, inflammation, and swelling, causing the muscle to respond with myalgia, muscle spasm, muscle splitting, or myositis.\textsuperscript{116} Myofascial trigger points occur in various combinations in the muscle and are considered by Travell\textsuperscript{117} to be the primary source of muscular pain.

Injury results in a deep, sharp ache on contraction of the
muscle. Depending on the area of injury, the pain may emanate from the tendon attachments (tendonitis), the fascial component (myofascitis), or the body of the muscle (myositis). The temporalis tendon attachment to the coronoid process is the most frequent site of masticatory tendonitis.

In acute or chronic internal derangement of the TMJ complex, the muscles that support and move the joints can be secondarily affected. Protective muscle splinting helps prevent further injury to the joint. The injured joint is often immobilized by anterior disc displacement without reduction (closed lock). Splinting of the masticatory elevator muscles is maintained until the joint is healed.

If the internal derangement is not adequately treated, the muscles remain chronically shortened and may eventually undergo contracture. The combination of acute trauma, loss of posterior teeth, and moderate to severe parafunction may bring about an anterior disc displacement with intermittent locking of the TMJ. Patients will often report histories of trauma that are followed by a variable period of clicking, progressively increasing in frequency and culminating in an abrupt disappearance and an inability to open the mouth. Differential diagnosis of a patient who has limited mouth opening must include internal derangement as well as muscle trismus.

Myalgia secondary to injury of the cervical spine can cause headaches, facial pain, and masticatory muscle pain. Affected masticatory muscles then affect mandibular position. Mandibular dysfunction results in tightening the muscles of the cervical spine, thereby perpetuating the cycle. This interaction is the reason that a substantial proportion of TMD patients also present with a history of cervical injury. This craniofacial and cervical syndrome requires multidisciplinary treatment of both the jaw and the neck.

Disc herniations can affect the cervical muscles through protective splinting, which can eventually lead to chronic postural changes. Nerve impingement and nerve root injuries can also affect muscle function. Cervical problems are frequently comorbidities in TMD patients. A reduction in the space between the posterior spine of the atlas and the base of the occiput as reported by Rocabado may cause pain by compression of the suboccipital tissues. The pain will be perceived as a headache starting from the back of the head.

Acute or chronic trauma can shift the occiput-atlas relationship and may lead to chronic tension in the suboccipital muscles with resulting fixation and irritation of nerves C1 and C2. The pain will be referred from the back of the head to the eye, along the side of the head, along the skin over the TMJ, and down along the angle of the mandible, radiating into the neck. Rotation of the atlas is commonly seen in patients with TMDs and may be linked to changes in occlusal contact patterns and instability of mandibular position. Osteoarthritic degeneration and ligament and muscle injury also occur at this level in acceleration-deceleration injuries.

Hypermobility caused by a disruption of the C1-C2 articulation also may result in excessive stretching or kinking of the vertebral artery and may lead to temporary vertebrobasilar syndrome with symptoms of vertigo, nausea, tinnitus, and visual disturbances.

The patient with a combined craniofacial-cervical syndrome will have a history of direct or indirect injury to the head and neck. The injury is usually not a direct trauma to the part, but rather a low-grade impact to the body that suddenly twists, flexes, or extends the neck. Symptoms may range from headache, nausea, visual disturbances, neck weakness, and pain, to stiffness accompanied by noises on rotation, flexion, and extension of the head. Depending on the level of the initial injury, branches of the cervical and brachial plexus and the areas they supply can also be affected.
Secondary muscles affected can cause superimposed acute or chronic pain, requiring a specific cervical evaluation.

Depending on how the cervical problem affects the masticatory system or vice versa, occlusal appliances may reduce muscle tension.

Myalgia Secondary to Parafunction. Oral parafunction includes bruxism, clenching, lip biting, thumb sucking, and any other oral habit not associated with chewing, swallowing, or speaking. Bruxism and clenching are the most common activities, with a prevalence of up to 90% in the general population.

In most patients, parafunction is mild and intermittent and does not require treatment. Moderate or severe bruxism and clenching can damage oral structures, causing wear of the teeth, breakdown of the periodontium in the presence of inflammation, and internal derangement and muscular dysfunction.

Bruxism and clenching can create excessive force for extended periods, whereas normal tooth contact during chewing and swallowing over a 24-hour period is about 20 minutes. Parafunc-
tional forces exceed normal masticatory forces, and the resultant force vector is primarily horizontal. Under such conditions, the teeth and periodontium are likely to be damaged. Ironically, most treatments are designed to protect the occlusion in function rather than in parafunction.

If the teeth, periodontium, TMJ, and muscles are considered to be “links in a chain” working together for proper function, the parafunction usually disrupts the weakest of these structures. The other structures remain relatively healthy or become second-
arily affected. For example, parafunctional wear of the canines may shift force to the other teeth. If these teeth are strong enough to withstand the excessive force, the pathology may shift to the TMJ. The patient can therefore present with both tooth and joint pathology.

Patients may present with pain in the cervical muscles as a result of chronic bruxism and clenching. Cervical muscle activity is related to occlusal contact. The patient may report restless sleep, waking up with limited mandibular range of motion, head-
ache, facial pain, and neck pain. The pain and stiffness usually improve as the day progresses.

If a patient reports that stiffness and pain increase as the day progresses, diurnal activity should be suspected. The patient may report marked stress and depression. Palpable muscle soreness will primarily affect the elevators and the lateral pterygoid. Clenching on the wear facets by moving the mandible laterally—a provocation test—will increase the pain.

A testing device known as a Bruxcore can quantify nocturnal activity. A portable electromyographic biofeedback instrument has also been used to monitor bruxism.

Repeated monitoring on different nights at a sleep laboratory is the most accurate assessment of parafunction; however, it is rarely necessary. Electromyographic analysis may show a higher resting tension level than normal, but tension depends on the specific type of muscle disorder and the specific muscle being analyzed.

Myalgia Secondary to Postural Hypertonicity. As stated above, the stability and function of the cervical region affects the position of the mandible relative to the maxilla and depends on the position of the head on the shoulders. This position is affected by gravity and the functional adaptation of the individual.

A healthy craniofacial complex stabilizes head position through a series of learned and complex antagonistic muscle interactions. Forward head posture (FHP) leads to shortening and greater tension of the posterior cervical muscles. The trapezius, sternocleidomastoid, and deeper muscles contract to prevent the head from tipping forward, leading to hyperactivity and chronic tension.

Chronic hyperactivity, such as working on a computer or in an office, can cause the shortened muscles to develop trigger points and the accompanying symptoms. The cervical spine is forced to adapt to the forces applied by strong cervical muscles, which may affect normal cervical lordosis.

The body adapts to FHP by rounding the shoulders, leading to chronic shortening of the pectoral muscles, which further maintains FHP. Pectoral muscle tension along with FHP leads to upper thoracic breathing and tighter intercostal muscles. The anterior and middle scalenes may entrap the brachial plexus at the thoracic outlet, or the first rib can be pulled up to the clavicle, resulting in costoclavicular entrapment.

Mehta and Forgione have discussed the effect of chronic FHP and the relative position of the occiput, atlas, and axis with respect to each other and the craniofacial complex. FHP and cervical muscle tension can lead to changes in occlusal contacts. Analyzing occlusal contact in maximum intercuspation with the patient in a supine position does not allow an accurate evaluation of occlusion in function. Likewise, measures to improve posture and cervical stability should be considered before definitive occlusal therapy is instituted.

Proper positioning during sleep is important for resting the postural muscles. A patient who habitually sleeps prone with the neck twisted at 90 degrees experiences the same effects as some-
one whose head is turned to one side all day long. In people who sleep on their sides with the lower arm outstretched under the pillow and head, the brachial plexus tends to become entrapped at the costoclavicular level. This side position can be stressful to the cervical muscles and can result in acute torticollis of the sternocleidomastoid muscle. Neck stiffness and trigger points are observed in patients with sleep habits that involve strained head positions.

Standing posture may be affected by leg-length discrepancies, hip rotation, and flat feet. Individuals who lean over machinery are likely have cervical and low-back symptoms. Shoes that are unevenly worn or that have extremely high heels affect balance and tend to cause a secondary protective adjustment of the pos-
tural muscles. This adjustment may result in chronic muscular shortening, trigger points, and spasm. Shifts in body posture and compensatory cervical changes affect mandibular position and tooth contact patterns.
Radiographs CT scans or MRI scans may be needed to evaluate spinal curvature and to rule out other pathologies.

Short-term exercise can increase range of motion and improve posture, muscle re-education can strengthen the muscles in the therapeutic postural position, and home exercise programs can maintain therapeutic postural position.115

Myofascial Pain and Trigger Points. In 1952, Travell and Rinzler116 introduced the concept of myofascial pain and trigger points. They defined a myofascial trigger point as a hyperirritable locus within a taut band of skeletal muscle located in a muscle or in its associated fascia or tendon. The spot is painful on compression and can evoke characteristic referred pain and autonomic phenomena.116

Trigger points may be active or latent. Active trigger points may cause pain spontaneously or during movement. Latent trigger points afflict nearly half of the population by early adulthood.140 Latent trigger points are usually not painful, but create weakness and restrict movement. Trigger points can be activated by a sudden overloading contraction, viral infection, cold temperatures, fatigue, and increased emotional stress.

The complex nature of myofascial trigger points and their common presence in acute and chronic muscle dysfunction require an understanding of their clinical features. According to Travell and Simons,140 there are seven such features:

- local tenderness over the trigger point
- referred pain, tenderness, and autonomic phenomena
- a palpable taut band associated with the trigger points
- a local twitch response of a trigger point in a palpable taut band
- perpetuation of trigger points
- a therapeutic effect when stretching the muscle containing the trigger points
- weakness and fatigability of muscles afflicted with trigger points relative to unafflicted muscles

Myofascial pain often refers to pain at the head and neck and is considered by some to constitute tension-type headaches. Myofascial pain and trigger points of the masticatory muscles can send pain to the eyes, ears, TMJ, and teeth, depending on the specific muscles. The pain is usually a dull or intense ache that varies daily and is strongly related to posture and muscle activity. The pain can usually be localized by the patient and can be indicated on a diagram of the body. Trigger points commonly affect the muscles of posture and mastication, and pain may occur in the same dermatome, myotome, or sclerotome. Satellite trigger points may occur within the pain reference zone. Clinically, movement is restricted, passive stretching is painful, and strong contractions markedly increase the pain. Resistive testing reveals weakness from protective splinting.

Trigger points are palpated by rubbing the fingertip lightly along the long axis of the muscle. If present, a taut band will be located first, and then the more sensitive trigger point. Applying pressure on a trigger point elicits a grimace or an involuntary sound from the patient called the “jump sign.” A snapping palpation of the taut band will produce a latent trigger response confirming the presence of the trigger point. Final confirmation comes on reproducing the patient’s pain by digital pressure on the point.139

Pressure algometers quantify the amount of pressure applied to the trigger point, which allows the clinician to document the severity of the trigger point. It may also be used to objectively record the efficacy of treatment.

Treating myofascial trigger points includes spray-and-stretch techniques with ethyl chloride or fluoromethane for its cooling effects, followed by stretching, hot compresses, and range-of-motion exercises. Trigger point injections of procaine (0.5% solution in saline) or lidocaine (2% without epinephrine) have also been the standard in pain management programs (Fig. 67.9). More recently, botulinum toxin (Botox) has been added to this arsenal.140–144

Other techniques involve ischemic compression for 30 to 60 seconds, acupressure, and pharmacologic therapy, including analgesics, muscle relaxants, antidepressants, and NSAIDs. Physical therapy modalities such as myofascial release and craniosacral techniques, including postural correction and exercise, have also

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FIGURE 67.8 A,B. Oral orthosis appliance for the jaws.

FIGURE 67.9 Trigger point injection of the masseter muscle.
been effective in managing myofascial trigger points. Stress and nutritional and hormonal factors should also be addressed.117

In case of the masticatory muscles, occlusal appliances may be used.118 Dental appliances are effective in reducing muscle symptoms and trigger points in mandibular elevators. These appliances are usually flat plane and fully cover either the upper or lower teeth, depending on when they are to be worn.

**Muscle Splinting.** Muscle splinting is a reflex by which skeletal muscles stabilize an injured area to protect it from further injury. The involved muscles become hypertonic and painful. The associated feeling of weakness, although alarming to the patient, is a normal protective reaction to discourage moving the affected part.

Muscle splinting is often a sequel to muscle injury and follows myositis. If splinting is protracted, muscle spasm may follow with or without trigger points, leading to a chronic cycle of myofascial pain and dysfunction.140

Initially, injections are not indicated; ice, rest, and relaxation are the basis of acute therapy. Stretching, ultrasound, or light massage can be used in the initial stages. Once the muscles have started to heal, splinting decreases, at which point therapy to regain mobility can begin. Mobilization techniques, gentle stretching, and range-of-motion exercises are required to prevent ongoing myospasm, contracture, and atrophy.

**Muscle Spasm (Sustained).** A muscle spasm is the painful contraction of a striated muscle caused by trauma, tension, or disease.113,123 The spasm manifests as pain and interference in function. The involved muscles become hypertonic and painful. The associated feeling of weakness, although alarming to the patient, is a normal protective reaction to discourage moving the affected part.

Muscle spasm is thought to be caused by the ischemia induced in a skeletal muscle by its continued contraction. The muscle becomes tender and lactic acid builds up, leading to the release of bradykinin, causing pain.

In the masticatory musculature, spasm of the masseter or temporalis muscle limits range of motion, which in turn causes the jaw to deflect to the ipsilateral side on mouth opening. If a spasm is isometric, the muscle will be rigid and resistant to stretch. Condylar position can be affected in true spasm of the elevator muscles and may predispose a patient to internal derangement. As with all chronic pain, the severe pain and inability to function can bring about psychosocial problems.113

Spastic muscles must be differentiated by palpation from: the painful soft muscle of myositis; relatively normal, albeit painful, muscle splinting; and localized taut bands and areas of myofascial trigger points. In contrast, a muscle in spasm has a stiff hard surface that is painfully resistant to stretch.

Electromyographic recordings show high standing tension in the affected muscle but lower electromyographic activity relative to the unaffected side. Pressure threshold meters and tissue compliance measurements may help identify the muscles in spasm. Thermography is also being investigated for routine diagnostic use.145–146

Initial treatment should be directed to eliminating the cycling spasm. The patient should restrict movement to within painless limits, but some function is necessary to regain a normal stretch reflex, which helps relax the muscle. In spasm of a masticatory muscle, the teeth can be disengaged with a stabilization (flat plane) appliance if the mouth can open enough to insert a temporary emergency splint. Splints are thought to work by shutting off proprioceptive input from the teeth that may help maintain spastic activity.

Muscle relaxants administered judiciously can help reduce dysfunction and spasm and can be adjuncts to other therapy, such as injection of spastic muscles. Spray-and-stretch techniques and massage and acupressure techniques may also be effective.

As the muscle starts to respond to treatment, stretching and range-of-motion exercises will help prevent contractures and bring the muscle back to full function. Ongoing passive jaw motion exercises will help maintain range of motion. In the masticatory system, structural factors, such as the bite, should be corrected only after the muscle is pain-free and fully functional.118

**Myositis.** A direct blow to a muscle can trigger a localized inflammatory response accompanied by swelling, pain, and immobilization.118 The main presenting symptoms will be localized soreness, swelling, and pain, along with weakness and immobilization of the affected structure. Pain is generally dull, deep, boring, and constant. Episodes of sharp pain related to movement of the affected structure may also be reported.

Injury to the head and neck muscles will result in an accompanying reduction in neck movement and changes in shoulder height and head posture as a means of protecting against further trauma. The pain may be perceived by the patient as a headache. An accompanying feeling of weakness in the neck may be experienced.

Patients with myositis of the masticatory muscles may report pain in the face or jaw accompanied by a change in the bite with the ability to chew, swallow, and speak comfortably. Locally, the area may appear swollen and discolored because of extravasations of inflammatory products. An inflamed muscle will present as a soft, painful mass on palpation. Pain will be associated with active as well as passive movement of the affected structure. The skin may be warmer to the touch than the surrounding area. The patient may present with low-grade fever if secondary infection is involved. Pressure threshold measurements with a threshold meter will indicate that discomfort occurs at a lower threshold than before the injury. Proximal to the injury, there may be a cold spot caused by vasoconstriction, which may affect healing.

Initial therapy of the acute symptoms of myositis includes immediately applying ice to the affected structure to reduce swelling, limiting movement to within painless boundaries, and resting the affected part. In injured mandibular muscles, restricting mouth opening during function and using a dental intraoral bite guard to control closure should be supported by NSADIs. In a severe case, immediately applying a methylprednisolone dose pack can control the amount of the immediate swelling. Antibiotics can be used if secondary infection is possible.

After the acute symptoms have subsided, treatment should include increased heat and mobilization of the affected structure, then an exercise program to regain full range of motion, and finally a muscle strengthening program.

**Fibrosis and Contracture.** Myotactic contracture occurs in muscles that are not allowed to function within their full range of motion. The muscle will lose its stretch reflex capabilities and will gradually shorten. Prolonged muscle spasm is thought to be caused by the ischemia induced in a skeletal muscle by its continued contraction. The muscle becomes tender and lactic acid builds up, leading to the release of bradykinin, causing pain.

Injury to the head and neck muscles will result in an accompanying reduction in neck movement and changes in shoulder height and head posture as a means of protecting against further trauma. The pain may be perceived by the patient as a headache. An accompanying feeling of weakness in the neck may be experienced.

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After the acute symptoms have subsided, treatment should include increased heat and mobilization of the affected structure, then an exercise program to regain full range of motion, and finally a muscle strengthening program.
on opening but not on protrusion. Lateral movement will be normal. Pain will not be present without sudden and forceful stretching or biting.

The treatment for myotatic contracture is to gradually stretch the involved muscle. Ultrasound with 5% to 10% hydrocortisone cream can be used as adjunct therapy. Massage and myofascial release along with daily stretching and exercise will bring the muscle slowly back to function. Myofibrotic contracture is irreversible and requires surgical intervention for a patient whose function is severely impaired.

**Muscle Disorders Secondary to Internal Derangement.** In the presence of acute or chronic internal derangement, the muscles that support and move the joints can be secondarily affected. Splinting helps prevent further injury to the joint. In cases of anterior disc displacement without reduction (closed lock), the joint will often be immobile. The masticatory elevator muscles should be splinted until the joint is healed.

The clinical finding of elevator spasm often causes the physician or dentist unfamiliar with TMD to prescribe muscle relaxants. These medications override the body’s defense mechanisms and so may do more harm than good. If the internal derangement is not adequately treated, the muscles remain chronically shortened and may eventually undergo contracture. A patient with acute closed lock often presents with a history of joint clicking. The patient may or may not be able to pinpoint an eliciting event. Sometimes the patient wakes up with the jaw locked. Other times, it locks during chewing. The patient will often have loss of posterior support through tooth wear, tooth breakdown, or missing or poorly restored posterior teeth.

Closed lock may be accompanied by a change in occlusion resulting from disc displacement, accompanying spasm of the lateral pterygoid, or both. The occlusion may shift to the contralateral side with a corresponding posterior open bite developing on the ipsilateral side. The patient may attempt to position the posterior teeth into contact but is hampered by joint pain and the lateral pterygoid, which pulls the mandible in the opposite side.

After acute trauma, the occlusion settles back to its preinjured state once the TMJ inflammation has subsided. However, if the patient has a parafunctional habit or has lost vertical dimension, the joint will continue to be unevenly loaded, and healing will be delayed. In such instances, the joint may become chronically inflamed and the muscles of mastication may continue to be in a state of protective splinting, spasm, or both.

If a patient has a combination of acute trauma, loss of posterior teeth, and moderate-to-severe parafunction, anterior disc displacement with intermittent locking of the TMJ is likely. Patients often report histories of trauma that are followed by a variable period of clicking, progressively increasing in frequency and culminating in an abrupt disappearance and an inability to open the mouth. Differential diagnosis of a patient who has limited mouth opening must include internal derangement, as well as muscle trauma.

**Muscle Disorders Secondary to Cervical Spinal Dysfunction.** Muscle disorders may occur secondary to rotations, fixations, fusions, or injury or locking of the facets of the cervical, thoracic, lumbar, and sacral vertebrae. The history, physical examination, and radiographic evaluation, often done in conjunction with a physiatrist or orthopedist, will reflect the acuteness and severity of the vertebral problem.

Disc herniations can secondarily affect the cervical muscles through protective splinting, which can eventually lead to chronic postural changes. Nerve impingement and nerve root injuries can also affect muscle function. Commonly seen cervical problems in relation to TMDs occur at the following cervical levels.

**Occiput-Atlas.** A reduction in the space between the posterior spine of the atlas and the base of the occiput, as reported by Rocabado, may cause pain by compressing the suboccipital tissues. The pain will be perceived as a headache starting from the back of the head.

Acute or long-standing trauma can shift the occiput-atlas relationship and may lead to chronic tension in the suboccipital muscles with resulting fixation and irritation of the C1 and C2 nerves. The pain will be referred from the back of the head to the eye, along the side of the head, along the skin over the TMJ, and down along the angle of the mandible, radiating into the neck.

Rotation of the atlas is commonly seen in patients with TMDs and may be linked to changes in occlusal patterns and instability of mandibular position. Osteoarthritic degeneration and ligament and muscle injury also occur at this level in acceleration-deceleration injuries.

**Atlas/Axis Level.** Trauma to this level may disrupt the transverse ligament holding the odontoid process of the axis against the anterior arch of the atlas, allowing forward subluxation or dislocation of the atlas on the axis. A disruption of the C1-C2 articulation may also result in excessive stretching or kinking of the vertebral artery secondary to hypermobility. This may lead to temporal vertebralbasilar syndrome with symptoms of vertigo, nausea, tinnitus, and visual disturbances.

Cervical rotation will be reduced because 40% to 50% of rotation occurs at the atlas-axis articulation. Pain also limits movement. Fractures are always to be considered in trauma to this region.

**C4, C5, and C6.** The level of greatest instability against acceleration-deceleration forces appears to be in the C4 to C6 region, with C4-C5 being primarily affected in hyperextension and C5-C6 in hyperflexion. Trauma may be to the ligaments, discs, and vertebral bodies, depending on the direction and magnitude of the force.

The cervical curve can be affected, and the patient will often have a compensatory forward head posture, further perpetuating the problem.

**Cranial Neuralgias**

**Trigeminal Neuralgia**

The cranial neuralgias, particularly trigeminal neuralgia, affect the face and have specific diagnostic criteria and treatment modalities. These disorders are covered in Chapter 66.

**Neuropathic Facial Pain (see Table 67.13)**

The International Association for the Study of Pain (IASP) defines neuropathic pain as “Pain initiated or caused by a primary lesion or dysfunction in the nervous system.” Thus, neuropathic pain results from pathology in the peripheral or central nervous system. These disorders are particularly common in the head and neck, probably as a result of the dense and specialized sensory innervation of this region. Unfortunately, these disorders greatly affect the patient’s life by interfering with important functions, such as feeding and speech.

In the past, any facial pain disorder without a definable cause was considered to be an idiopathic, “atypical” facial pain syndrome. Over the years, many inappropriate “descriptive” diagnoses were given to these disorders (atypical trigeminal neuralgia, atypical facial pain, atypical odontalgia, phantom tooth syndrome, and so on.) Under the current IHS classification (see Tables 67.5 and 67.6), these disorders are considered “central” causes for headache and facial pain and are classified as “persistent idiopathic facial pain” (category 13.18).
Complex regional pain syndrome (CRPS), a form of neuropathic pain can occur in the face.\textsuperscript{149} CRPS is covered in detail in Chapter 25. Likewise, postherpetic neuralgia commonly affects the face and is covered in detail in Chapter 27.

**PSYCHOSOCIAL CONSIDERATIONS**

**Assessment**

As with other chronic pain conditions, psychosocial factors explain much of the variance in the outcome of persistent facial pain disorders. Affective and anxiety symptoms, especially emotional trauma, have been implicated in precipitating and maintaining chronic orofacial pain.\textsuperscript{150} Marked somatic overconcern or somatization disorder can also compromise treatment in these disorders. Similarly, chronic disability behavior further compromises the patient's status.

It has become a minimum standard of care to address critical psychosocial factors within the diagnostic interview, as underscored by the IASP Curricula on Pain for Dental Schools.\textsuperscript{151,152} Consistent with this attention, the Research Diagnostic Criteria includes a 31-item questionnaire addressing psychosocial and physical domains of chronic TMD. Other validated self-report facial pain scales also address psychosocial issues, and their use within multidisciplinary facial pain facilities is common.\textsuperscript{153–155}

**Treatment**

As in other areas of chronic pain management, behavioral interventions have been a mainstay of treatment for persistent facial pain. Time-limited, structured relaxation training is as effective as conventional occlusal splint therapy for temporal joint dysfunction and related chronic myofascial facial pain disorders. Combined treatment is even more effective and provides longer lasting effects.\textsuperscript{156} Similarly, improving cognitive coping skills reduces pain and improves function.\textsuperscript{157} Behavioral interventions target reducing anxiety and improving perceived control over pain. Biofeedback-assisted relaxation can also be effective, perhaps more so in limited subpopulations.\textsuperscript{158–160} More intensive, interdisciplinary treatment is often indicated for patients with a constellation of severe psychosocial and disability behaviors.

Cost-effective behavioral group programs have also been used in the early stages of facial pain syndromes and have reduced pain and improved coping skills. Most recently, Stowell et al.\textsuperscript{161} compared a brief behavioral treatment program emphasizing early intervention to a standard approach in patients with "acute TMD" (pain of less than 6 months duration). One-year follow-up data revealed clinical improvement and substantially fewer days of headache.\textsuperscript{161} Treatment is frequently indicated for evaluating difficult or refractory cases. Similarly, chronic disability behavior further compromises the patient's status.

**Conclusion**

Craniofacial pain is a common clinical problem and often a diagnostic challenge. Given the complex anatomy of the region and the numerous discrete syndromes, a multidisciplinary approach is frequently indicated for evaluating difficult or refractory cases.

**References**


132. Mehta NR, Roeber FW, Haddad AW, et al. Stresses created by occlusal pre-
AUTHOR QUERIES

AQ1–Please confirm cross reference.

AQ2–There was a comma here instead of a period. That suggests to me that the last part of this sentence may be missing. Please complete the sentence.