Headache is one of the most common acute and chronic pain complaints reported in adolescents and adults. Estimates of the prevalence of headache differ somewhat, but most surveys report that over 80% of adults report periodic episodes of headache. The life-time risk for migraine has been reported to be over 35%. For unexplained reasons headaches are reported much less frequently in children, but it is not known whether the lower number represents an actual lower rate of headaches in children or an error in reporting since children often underreport other pains. Studies have shown that headache in general, and migraine in particular, become more prevalent in the adolescent years, after puberty, and increase in frequency into adulthood. Dentists are often asked to determine if orofacial pain or headache is caused by local pathology, such as infection or a temporomandibular disorder (TMD). The clinical manifestations of headache syndromes overlap with dental pathology and frequently lead to unnecessary dental treatment. This chapter addresses the clinical manifestations of headaches, methods for diagnosis, and treatment recommendations appropriate to primary health care providers in medicine and dentistry. The challenge for the dental provider is to be able to distinguish symptoms and signs of headache disorders from primary dental disease and TMD, and to assist other health care providers in determining effective treatment strategies.

Many attempts have been made to establish a clear classification system for headache, and several are in active use, including the classification systems reported...
in the International Classification of Disease, the International Headache Society (IHS) (Table 31–1), and the International Association for the Study of Pain.

All of these classification systems are similar, and the focus recently has been to develop highly specific classifications based on reliable signs and symptoms and stable inclusion and exclusion criteria. There are no specific diagnostic tests readily available that allow a guaranteed confirmation of a headache diagnosis. The final diagnosis of headache type is usually derived from a working diagnosis that is dependent upon the persistence and reliability of the patient’s report of symptoms and failure to detect other etiologic factors. Probably the most effective method of diagnosis is to combine a careful understanding of signs and symptoms common to the various headache groups and an objective analysis of responses to reversible treatments known to be specific for particular headache conditions (eg, sumatriptan success in migraine, etc.).

### Diagnostic and patient management considerations

The discussion of headache in this chapter is limited to the most common and important types of headaches, and those most commonly presenting in clinical practice that can easily be confused with local orofacial pathology and odontogenic disorders. The first and most important consideration when assessing patients with complaints of facial pain and headache is to rule out potentially progressive and damaging forms of disease that present with headache or facial pain as part of their symptom complex. Severe infections, whether intracranial or systemic, commonly produce headache, and, depending upon the location of inflammatory changes, can also cause facial pain. Intracranial tumors are also known to trigger gradually progressive headaches that become more frequent and severe as disease progresses. Intracranial and extracranial vascular lesions, such as aneurysms, and vascular occlusions can cause pain identical to headache or provoke pain referral into the face or neck. Extracranial tumors of the head, orofacial region, and neck can also cause pain that is localized or diffuse and sometimes similar to headache. A common systemic condition known to produce headache is uncontrolled hypertension which has been shown to produce hypertensive headaches in 15% of patients.

### Migraine

Migraine is considered to be one of the most painful of the headache groups. Patients usually rate episodes of migraine as moderately severe to very severe, and some rate the pain as the most severe that they have ever encountered. The life-time prevalence of migraine is approximately 35% in adults, and intractable migraine, which is a less common disorder, can be a completely disabling condition. Migraines occur in young children and become more prevalent after puberty. Genetic and familial risk is considerable, with more than 50% of those suffering migraine reporting that family members are also affected. Hormonal changes during and after puberty are thought to be an important contributor in migraine, and some women suffer severe premenstrual migraines as hormone levels change during their monthly cycle. Although migraines affect both genders, generally the ratio is 3:2, favoring females.

### Etiology and pathophysiology

The exact mechanisms responsible for migraine are just now becoming more clearly understood. The process involves vascular changes triggered via trigeminovascular innervation, causing vasoconstriction and dilatation of cerebral vessels innervated by branches of the trigeminal nerve. Many factors appear to contribute to the process, including physiologic and psychophysiologic stimulation of the central nervous system (CNS). It is thought that the trigeminal innervation of cerebral vessels provides a sensory role, protecting the brain from agents that cross through the circulation during pathologic insult and from agents produced within the brain. The trigeminal innervation of cerebral vessels also has a motor component that is activated upon threat of change in normal blood flow to the brain. Response to stimuli can trigger release of vasodilator peptides or vessel hemodynamic changes through central interaction with parasympathetic innervation of the seventh cranial nerve. It is thought that stimulation of trigeminovascular fibers by electrical, chemical, and mechanical stimu-
Migraine as classified by the IHS criteria is generally divided into two distinct subtypes: migraine with aura, and migraine without aura. The former presents with a variety of symptomatic and physical changes that predict the onset of the migraine episode. Generally these predictive changes occur 5 to 20 minutes before the onset of headache and include one or more of the following: visual changes, dysphasia, other unilateral sensory changes, feelings of hopelessness, altered sense of smell or vision, stiff neck, and feelings of paresthesia or other neurologic sensations around the head and neck on the side that will become painful. Some patients continue to have these same sensations during the headache attack, and others note a decrease in the aura as pain becomes the dominant sensation. Migraine pain rarely lasts less than 4 hours and normally is gone within 24 hours, but some episodes can last for a week or longer. Historically it was thought that migraine was a unilateral pain disorder, but recent epidemiologic studies suggest that up to 40% of migraine episodes are bilateral.

Migraine without aura presents as a headache without warning. Both forms of migraine are usually rapid in onset and can move from preheadache to headache status within a few minutes. Although most attacks of migraine are unilateral and can repeat on the same side and location or move from side to side with each episode, the location of pain can encompass the entire side of the head or localize to a much smaller area. It can even be felt in one tissue or organ (eye, tooth, sinus, temporomandibular joint [TMJ], etc.). Once pain begins, there are few differences in the manifestation of migraine with or without aura. Most patients report that neurologic stimulation of any type increases pain and nausea if present. Neurologic stimulation that aggravates migraine includes light, sound, motion, smells, exercise, and touch. As a result, most migraineurs seek a dark quiet space during attacks and generally do not like to be disturbed. In contrast patients with tension type headaches (TTHA) often find that exercise decreases symptoms as do other activities that distract them. Most migraines do not occur more than 2 to 4 times a month, although occasionally, patients encounter migraine recurrences up to 6 to 8 times per month. Factors that provoke recurrent episodes of migraine are not clearly understood, and for some patients no specific triggering mechanism can be detected. Certain smells trigger episodes in those with olfactory sensitive migraines. The most common olfactory triggers are perfumes and cosmetic smells. Other patients report migraine triggered by alcohol, preserves in foods, artificial sweeteners, and foods. The most commonly reported food triggers are chocolate, dairy products, citrus fruits, and wine or other alcoholic drinks. Hunger and hypoglycemia have also been reported to trigger attacks, as have changed sleep patterns and sleep deprivation. Stressful life events have been reported by about 50% of migraineurs to trigger headache episodes. In some patients, exhaustive analysis of triggering factors fails to reveal any obvious patterns.

**Facial migraine**

Facial migraine is a clinical variant of migraine in which the major location of the migraine is within the orofacial complex, including the jaws, TMJ, dentition, salivary glands, and maxillary sinus. The pattern is usually similar to that seen in other forms of migraine and an aura may be present or the pain onset can be without warning. Facial migraine is frequently incorrectly diagnosed as dental pathology by both physicians and dentists because the severity of pain and the precise location of symptoms easily resemble odontogenic infection, sinusitis, or acute TMJ dysfunction. During attacks, the
teeth in the field of pain can be hypersensitive to per-
cussion and temperature, leading to an incorrect diag-
nosis of pulpitis or maxillary sinusitis. Tactile stimula-
tion of the dentition and periodontal tissues can also
trigger increased symptoms, mimicking acute periodon-
tal infection. If the pain is located around the TMJ and
ear, patients often provide a history of repeated visits to
their primary care doctor to rule out ear infections. A
key report by most patients with facial migraine is a
report that the pain stops after sleeping. Complete reso-
lution of symptoms after sleeping is uncommon in den-
tal infection, TMJ dysfunction, or sinusitis. Another key
clinical report that should cause the clinician to consider
migraine is the report that light and sound increase
symptoms, or that nausea occurs with pain episodes.
Patients with facial migraine may report recurrent pain
thought to be odontogenic in origin. Frequently, they
report repeated endodontic treatments and even extrac-
tions. Those with less clearly localized facial migraine
commonly report prior unsuccessful treatment of sinus
disease. One or more of these reports should cause the
clinician to consider facial migraine and pursue that
diagnosis prior to engaging in additional irreversible
treatments. A surprising number of patients with
migraine and facial migraine report attempts at treat-
ment through chiropractic therapy. This usually occurs
in those with some level of cervical or occipital symp-
toms associated with migraine attacks. Although not
commonly known, migraine can be preceded by neck
stiffness, and during the headache episode pain can
radiate throughout one side of the neck and into the
head. The nature of the migraine episode with complete
resolution of symptoms within 24 hours often causes
the patient or treating doctor to assume that their treat-
ment was responsible for resolution of symptoms. This
error leads to repeated use of the same therapy with
each recurrence.

Other migraine variants

Other symptoms and changes reported in migraine and
facial migraine include weakness, indigestion, diarrhea,
and visual changes, including partial blindness and tun-
nel vision. Ophthalmoplegic migraines are accompanied
with paresis of ocular cranial nerves, and retinal
migraines typically produce blindness or scotoma of one
eye. The full range of generalized and systemic symp-
toms reported by some migraine patients is not appreci-
ated by many clinicians. Prodromal features can extend
to variation in temperature regulation; fatigue; gastro-
intestinal symptoms, including either constipation or
diarrhea; aphasia; hypersensitivity to touch; tinnitus;
phonophobia; nasal congestion; hypertension; vertigo;
and even loss of consciousness. Basilar migraine often
presents with stupor and emotional changes, including
aggressive behavior and swearing. Hemiplegic migraine
causes unilateral changes in sensory and motor control.
An interesting migraine variant seen in the orofacial
region is ice pick migraine, which manifests as a severe
stabbing sensation in a tissue or region. It often triggers
stabbing pains through the eye, temple, TMJ, or teeth.
The symptom lasts only a second or two and can recur
from once or twice a day to many times. It is often conf-
fused with a cracked tooth syndrome, pulpitis, or
momentary catching of the meniscus of the TMJ. The
difference is that all of the other conditions require some
form of jaw movement or activity to illicit the symptom,
but ice pick migraine occurs at times when the mouth
and jaws are completely inactive. A variety of exertional
migraines have also been reported. Most common
among these migraine variants are those triggered by
exercise and sexual activity. They are usually of much
shorter duration (20–60 min) than other migraines. Last
among the migraine variants is persistent or intractable
migraine, which continues for days or weeks. It is the
most impairing of all the migraine variants and carries a
risk of vascular accident and crisis. It can require emer-
gency medical treatment to interrupt the episode.

Migraine management

The management of migraine can be divided into three
specific strategies: prevention, abortive therapy, and
symptomatic or palliative therapy (Table 31–2). For
those with infrequent recurrences of less than once or
twice a month the best therapy may be abortive or pal-
liative, since neither requires continuous use of medica-
tion to prevent onset of symptoms. Patients who expe-
rience more frequent or severe episodes may desire
therapy aimed at aborting an attack in the initial stage
or treatment that prevents recurrence. Response to any
of the three management approaches can be highly var-
ied among individuals, and even in the same patient
over time. Factors that cause widespread variation in
response to interventions are not well understood.

Preventive protocols for migraine commonly employ
continuous use of either a beta blocker, such as propran-
olol, or one of the tricyclic antidepressants, such as
amitriptyline or nortriptyline. Recently, some success
has been reported with use of the newer generation of
selective serotonin reuptake inhibitor (SSRI) antidepres-
sants, such as fluoxetine or sertraline, although both
have also been associated with increased headaches.
Membrane stabilizers, such as carbamazepine, valproic
acid, and gabapentin, have also shown value in pre-
venting migraine in some patients that have not
responded to other migraine protocols. Thermal
biofeedback, which trains the patient to raise the tem-
perature of the hands and feet, has been shown to reduce headache frequency and abort headaches in some migraine patients. The mainstay of abortive therapy primarily continues to be three approaches: use of ergotamine derivatives, sumatriptan and other triptan medications, and Midrin.

**Tension-type headache**

The most common type of headache is tension-type headache (TTHA). Over 80% of adults experience TTHA periodically. These headaches are also common in children and adolescents. The condition is usually divided into two main categories: episodic TTHA, and chronic TTHA. Symptoms can range from mild to very severe, with the pattern of episodes varying depending on factors related to headache triggers. Most patients who suffer TTHA do not seek specific medical treatment for the condition and choose to use over-the-counter (OTC) medications to combat symptoms.

**Etiology and pathophysiology**

Tension-type headache has been widely studied relative to issues of both etiology and pathophysiology. It is generally considered to be triggered by psychophysiologic changes related to stress, worry, and depression. It is more common during episodes of stress and unhappiness and often becomes infrequent during times of relaxation and low stress. Some clinical scientists divide TTHA into two clinical subtypes: without tender pericranial muscles, and with tender pericranial muscles. The two conditions are distinguished by a patient response to palpation of pericranial muscles during a headache attack. The muscles involved include the frontalis, temporalis, suboccipitalis, masseter, other paravertebral and cervical muscles, and even the muscles of facial expression. The finding of muscle tenderness in some patients with TTHA leads to the assumption that TTHA and myofascial pain are related and specifically so in TTHA with tender pericranial muscles. Studies have not confirmed that patients with TTHA with tender muscles have significantly higher readings on electromyography (EMG). The pathophysiology of TTHA without tender muscles remains unresolved but generally is thought to include actions within the CNS that lead to peripheral pain without evidence of peripheral pathology.

**Clinical presentation and diagnosis**

Tension-type headache presents differently than migraine, yet many patients with moderate to severe TTHA believe that they have migraine. Usually, TTHA is bilateral during attacks and often includes a band-like sensation of tightening around the head. The regions of the temporalis and frontal muscles are often painful, and as the headache progresses, the entire head and upper cervical region may become painful. Some patients, however, report that their symptoms are primarily bitemporal with pounding sensations that are nonpulsating in character. Tension-type headache often begins in the late morning and persists throughout the day. Nausea and vomiting are not normally a constituent of TTHA except when pain becomes severe, which initiates sensations of nausea in a minority of patients. Most patients report that they are more likely to develop symptoms during times of stress and worry or depression. Sleep loss also increases frequency of episodes of TTHA. If pericranial muscles are tender to palpation, the diagnosis of TTHA with pericranial muscle tenderness is established. Since EMG levels in such patients are normal, it has been suggested that the process of tenderness is neurogenic and centrally controlled.

The condition most commonly confused with TTHA is myofascial pain dysfunction (MPD) of the muscles of

<table>
<thead>
<tr>
<th>Preventive</th>
<th>Abortive</th>
<th>Palliative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beta blockers (propranolol)</td>
<td>Ergotamines (oral)</td>
<td>Narcotic analgesics (oral)</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>Midrin (oral)</td>
<td>Meperidine with promethazine (injectable)</td>
</tr>
<tr>
<td>Antidepressants: amitriptyline, nortriptyline, doxepin, fluoxetine, sertraline</td>
<td>Sumatriptan (oral, injectable, nasal)</td>
<td>DHE (dihydroergotamine mesylate) for persistent migraine</td>
</tr>
<tr>
<td>Ergotamine</td>
<td>Zolmitriptan (oral)</td>
<td>Ice packs</td>
</tr>
<tr>
<td>Anticonvulsants: valproate, carbamazepine, gabapentin</td>
<td>Acetaminophen (oral)</td>
<td>Sleep</td>
</tr>
<tr>
<td>Baclofen</td>
<td>Lidocaine (intranasal)</td>
<td>Dark room and sensory deprivation</td>
</tr>
<tr>
<td>Lithium</td>
<td>Thermal biofeedback</td>
<td></td>
</tr>
<tr>
<td>Sodium caffeine benzoate</td>
<td>Imagery and relaxation therapy</td>
<td></td>
</tr>
<tr>
<td>Thermal biofeedback</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress management, cognitive behavioral therapy</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
mastication. Both can present as facial pain and headache, and both have chronic durations of several days or cyclic patterns like chronic daily headache. Both exhibit pain in the region of the temporalis and other pericranial muscles and both seem to be triggered or aggravated by the same exogenous and endogenous factors, including stress and other behavioral dysfunctions. Both TTHA and MPD are common conditions, with prevalence rates that result in a significant likelihood of both occurring in the same patient. Tension-type headache with tender pericranial muscles has the potential for causing referred pain to the maxilla, mandible, TMJ, and odontogenic structures. Both conditions are associated with a tendency for increased somatic focus (expression of behavioral stressors as physical symptoms).

Subtypes of tension-type headache

Tension-type headache can have a highly varied duration, with episodes lasting longer than a week or less than 30 minutes. Current IHS criteria for TTHA diagnosis require that to establish a diagnosis of episodic TTHA requires 10 prior episodes of the same set of headache symptoms, pain that is mild to moderate, bilateral pain, lack of symptoms commonly associated with migraine (photophobia, nausea, etc.), and pain that last more than a few minutes. Episodic and other forms of TTHA also have in common a lack of symptom increase from walking or stair climbing.

A diagnosis of chronic TTHA requires that the other criteria for TTHA are met plus the presence of the headache 15 days per month over a period of 6 or more months. Such headaches are usually more difficult to manage and result in risk of rebound headache, because of chronic use of analgesics, including both non-narcotic agents, acetaminophen, ibuprofen, aspirin, and narcotic analgesics. The chronic use of any of these medications on a daily basis leads to rebound headache when medications are discontinued. As with episodic TTHA, chronic stress and other behavioral or psychological factors are thought to play a major role in the pathophysiology of chronic TTHA. Treatment is often difficult, since the triggering factors may not be evident. Some patients can experience transformation of vascular headaches, such as migraine, to chronic daily headaches, and others report symptoms of both TTHA and migraine, leading to the suggestion that they suffer from a mixed headache syndrome made up of both migraine and TTHA.

Patients may also report TTHA that arises in the posterior of the neck and sometimes spreads to encompass most of the pericranial regions. These cervicogenic headaches can be a part of TTHA or represent manifestations of myofascial pain involving the cervical muscles. Cervical dysfunction representing pain caused by cervical nerve irritation can also provoke pain in the head, neck, and shoulders, but it usually follows neurologic distributions and generates symptoms down the arm and into the hand. Cervical nerve compression headache usually is accompanied with other neurologic symptoms, including paresthesia involving the neck or arm, tingling sensations that radiate down the arm, and a significant increase in pain with neck movements or compression of the cervical vertebra. It is always wise to seek consultation in patients reporting pain in the neck with referral into the cranial or facial region, since cervical nerve compression can arise from degenerative arthritis of the cervical spine, tumors, and vascular lesions and as the result of injury to the cervical region. In cases of recent trauma, referral is particularly wise, since stress on the cervical vertebrae could cause additional neurologic symptoms and, in some cases, damage the spinal cord. Pain radiating down the arm could also suggest referred ischemic cardiovascular pain and warrants medical consultation.

Management of tension-type headache

Management of TTHA depends upon the frequency and severity of episodes. It is generally recommended that simple TTHA occurring less than twice a week be treated symptomatically with analgesics, including common OTC medications (Table 31–3). Most are not dependency-producing, but some have the potential for habituation or triggering rebound headaches if used excessively. Since TTHA appears to be associated with behavioral factors, attention should be directed toward altering psychological and behavioral issues. This may necessitate referral to a psychologist or other mental health professional for assessment and treatment. An important component of treatment should be working with the patient to identify conditions that increase headache episodes. Noting the pattern of headache recurrences and associating them with exposure to stressful social or personal events can lead to preventive interventions. Late-life tension headache is seen in depressed elderly individuals and requires exploration of endogenous sources of depression more than exploration of exogenous factors, such as worry about health issues, financial status, or loneliness. Organic degenerative brain syndromes in the elderly can cause headaches and depression.

The management of tension headache using a preventive process is usually reserved for those patients who experience more than two headaches per week or have particularly severe TTHA that persists for more than a day. Preventive protocols are also worth consideration in patients who cannot comply with the requirements of symptomatic treatment (excess use of narcotic analgesics,
Cluster headache

Cluster headache is significantly less prevalent than migraine but is confused with migraine because of its extreme pain. Cluster is more common in males and is infrequent in adolescents and young adults, although cases have occurred in individuals less than 25 years of age. Cluster, like migraine, is unilateral and is among the most painful of the headache disorders. It, in addition to pain, is frequently accompanied by a variety of autonomic changes caused by contribution of cross-signaling of trigeminal and autonomic components of the orofacial region. Cluster occurs more frequently in smokers and is sometimes related to a variant of trigeminal neuralgia.

Etiology and pathophysiology

The etiology of cluster is not understood, and it is considered to be a member of the vascular headache group, with some characteristics related to neuralgias and migraine. Abnormal function within the trigeminovascular system has also been suggested as part of the pathophysiology. Oxygen desaturation appears to play a role and trigger serotoninergic reactions. Attacks are often seen during periods of reduced oxygen, including high altitude and rapid eye movement (REM) sleep, and attacks have been aborted with exposure to 100% oxygen. It has been suggested that chemoreceptors in the carotid body become hypersensitive in cluster. Neurogenic inflammation is also a component of cluster, with CGRP levels elevated during attacks. Stimulation of the sphenopalatine ganglion occurs in cluster, including parasympathetic autonomic innervation in the face and sinuses. Exact mechanisms responsible for the trigeminal, trigeminovascular, and autonomic innervation that results in the pain and physical characteristics of cluster have not been resolved. Attacks can trigger pain within cranial nerves V, VII, IX, and X and cervical nerves 1, 2, and 3. Irritation of the sphenopalatine region by tobacco smoke or other irritants plays a role in recurrent episodes of cluster in some patients but not others. Blocking the trigeminal, sphenopalatine, and occipital nerves have all resulted in relief of cluster symptoms. It is evident that active cluster involves vasodilation of extracranial vessels but

use of multiple medications, obtaining medications from more than one source, etc.). When narcotic analgesics are used for TTHA or any chronic pain syndrome, it is wise to establish a medication compliance contract with the patient that outlines the clinician’s rules for use of the medication. Most narcotic analgesic patient contracts usually include a series of statements that the patient must agree to or the medication should not be prescribed or renewed. The list of requirements varies but generally includes the items listed in Table 31–4.

### Cluster headache

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**Table 31–3** Management of Tension-Type Headaches (TTHA)

<table>
<thead>
<tr>
<th>Symptomatic Treatments</th>
<th>Preventive</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Episodic TTHA</strong></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>Behavioral management protocols relaxation training, stress management, meditation, cognitive behavioral therapy biofeedback</td>
</tr>
<tr>
<td>Caffeine (40–50 mg)</td>
<td>Sleep hygiene</td>
</tr>
<tr>
<td>Acetaminophen</td>
<td>Exercise</td>
</tr>
<tr>
<td>NSAIDs (ibuprofen, naproxen, meclofenamate)</td>
<td>Antidepressants (amitriptyline)</td>
</tr>
<tr>
<td>Codeine, hydrocodone, oxycodone</td>
<td></td>
</tr>
<tr>
<td>Butalbital (50 mg)</td>
<td></td>
</tr>
<tr>
<td>Propoxyphene (50, 65, 100 mg)</td>
<td></td>
</tr>
<tr>
<td><strong>Chronic TTHA</strong></td>
<td>Migraine medications: antidepressants, beta blockers, calcium channel blockers, anticonvulsants</td>
</tr>
<tr>
<td>Same as for episodic TTHA with attention to possible rebound reactions to analgesics</td>
<td>Cognitive behavioral therapy</td>
</tr>
</tbody>
</table>

**Table 31–4** Component of a Medication Contract

- Medications will only be received from one provider.
- The patient agrees to use the medication only as prescribed.
- The patient will protect the medication from loss or theft, and it will not be replaced early under those circumstances.
- The medication will not be used with other agents, such as alcohol or street drugs.
- The patient will not call early and request that the medication be renewed prior to the appropriate time.
- The medication will be used on a time-contingent basis and not saved to be used in greater concentrations when pain is high.
- Medications from others will not be used to augment the prescribed medication.
- Violation of the agreement will result in the prescribed medication being discontinued.
Cluster is an interesting and perplexing member of the orofacial pain group. Episodes are usually unilateral and last less than 60 minutes, but can persist for up to 3 hours. It is common for episodes to start and stop within a 5-minute window of activity. During the episodes patients often report pain rated as 10 on a 0 to 10 pain scale. Unlike migraine, the pain is so aversive that it makes sufferers walk the floor, pound their head, or do other physical activities to distract themselves from the pain. Between episodes they usually report no symptoms and feel completely normal. Attacks start rapidly, without warning, and stop just as suddenly. It is often impossible to function normally during attacks. Autonomic changes include facial flushing, sensations of facial swelling, rhinorrhea, lacrimation, injection of the mucosal of the eye, salivation, and even facial edema. During episodes, the structures in the region are often hyperpathic, and the dental occlusion can feel altered. Teeth can become hypersensitive to cold and percussion during pain episodes, with rapid resolution of symptoms after the pain has passed. The location of pain can vary and involve small regions of the head, neck, face, dentition, and mouth, or expand to create pain in several tissues adjacent to each other. Pain can be localized to the area around, in, or above one tooth or in the TMJ. Episodes recur from once a day up to 4 to 5 times in 24 hours. Pain can occur while awake or asleep, and it is common for patients to report that they wake during the night in extreme pain. Patients with cluster often undergo unnecessary dental and medical therapy when their symptoms are located in the region of the sinuses or dentition. Symptoms around the teeth and jaws can mimic the severe pain of pulpitis or odontogenic infection. Cluster often follows seasonal patterns or can go into remission and recur months or years later.

Diagnosis of cluster is made through clinical assessment, after ruling out serious head and neck pathology. There are no specific commonly employed laboratory or other diagnostic tests, except for administration of 100% oxygen during an attack. If administration of oxygen results in resolution of the pain more rapidly than would normally occur, the diagnosis of cluster is confirmed. Application of 4% lidocaine intranasally has been reported to abort cluster attacks. Beyond these medication trials, diagnosis is clinical and based upon the classic nature of symptoms and signs along with response to trials of medications known to be effective in cluster.

Cluster-trigeminal neuralgia

Classic cluster cannot be provoked by tactile stimulation of the tissues in the head and neck, but a small percentage of patients with cluster also have symptoms of trigeminal neuralgia which is triggered by touch of the trigger zone within trigeminal innervation. The combined occurrence of these conditions causes spontaneous episodes of severe unilateral headache that cannot be provoked with tactile stimulation, along with pain triggered by light mechanical stimulation in the distribution of the trigeminal nerve. The combination of these two symptom complexes often results in confused diagnoses and inappropriate therapy. Treatment often requires joint therapy for trigeminal neuralgia and cluster. Patients with the combined condition can experience significant symptoms when the dentition is stimulated (thermal, percussion), leading to a false diagnosis of pulp pathology.

Management of cluster

Treatment of cluster usually targets prevention of recurrences, since each episode is so brief that the pain is gone by the time the medication has taken effect. There are exceptions to that rule, and abortive medications are useful in confirming the diagnosis of cluster. Abortive therapies include administration of 100% oxygen at the outset of an episode, which results in a rapid resolution of symptoms in over 70% of cases, or administration of 4% lidocaine solution sprayed intranasally, which also terminates the pain episode in approximately 40 to 50% of patients. Other abortive treatments are less effective. Preventive therapies include systemic prednisone, methysergide, and cyproheptadine. Refractory cases sometimes respond to migraine medications, such as sumatriptan, ergotamine, dihydroergotamine (DHE), lithium, or valproate (Table 31–5). Since cluster occurs as a series of episodes of pain and usually goes into remission, it is common for patients to experience months or years free of symptoms only to have them return. After several months of preventive therapy it is usually advisable to discontinue the medication to see if remission has occurred.

Chronic paroxysmal hemicrania

Chronic paroxysmal hemicrania (CPH) is another of the vascular headache syndromes that are commonly confused with odontogenic, regional pathology of the head and neck, or TMD. It has features common to migraine and may have some of the same etiologic and pathophysiologic components.
Clinical presentation and diagnosis

As with cluster, CPH onsets rapidly and provokes severe pain that lasts from a few minutes up to 45 minutes. It is consistently unilateral and occurs on the same side of the head and face. The temporal area, orbital region, face, or structures of the jaws can be the sites of pain. Attacks are often more frequent than experienced in cluster, with more than five episodes occurring per day during peak periods of recurrence. Associated findings include conjunctival injection, sinus stuffiness or rhinorrhea, lacrimation, and edema of the region of pain. No specific diagnostic test is available and diagnosis is via clinical decision based on symptoms and findings of autonomic changes during attacks. In contrast to cluster, CPH is almost always responsive to indomethacin in dosages of 25 mg per day to 150 mg per day. Cluster is often unresponsive to indomethacin.

Management

Chronic paroxysmal headache usually responds well to indomethacin, but because it can have significant impact on the stomach and gastrointestinal system the lowest possible therapeutically effect dosage should be used. When patients cannot tolerate indomethacin medications, therapies effective for cluster can be administered with a positive effect.

Headaches associated with head trauma

Acute post-traumatic headache

Acute post-traumatic headaches (APTH) occur secondary to actual physical injury, edema, and irritation of extracranial and intracranial tissues. In its mild form, APTH represents the result of benign trauma and soreness in the head and neck. Symptoms are most commonly reported to be in the temporal, frontal, and occipital area, with symptoms in the face and jaw less often reported. Of primary concern in patients presenting with APTH is the potential that they are experiencing significant cranial edema or even an intracranial hematoma. In such cases, emergency assessment and management is necessary to ensure that brain damage caused by intracranial hemorrhage or edema is prevented. With that consideration, it is recommended that any patient seen in the primary care medical or dental setting who reports significant head pain following trauma be evaluated carefully, and if any evidence of diminished cognitive, reflex, or other neurologic function is detected, the patient must be referred for complete neurologic assessment and cranial imaging. Use of any medication that increases intracranial pressure, masks symptoms of progressive cranial involvement, or increases the risk of intracranial hemorrhage is contraindicated in patients with acute head trauma.

Chronic post-traumatic headache

Chronic post-traumatic headache (CPTH) is caused by organic and neurochemical alterations in the CNS following blunt trauma to the head. The onset of CPTH does not require serious injury to the brain, such as concussion, subdural hematoma, or skull fracture; CPTH can occur without direct trauma to the skull and has been reported to develop after whiplash or other acceleration-deceleration accidents. Approximately 60% of patients with documented closed head injuries develop CPTH. Symptoms vary considerably depending upon the type of trauma but often include chronic generalized headaches, neck pain, or shoulder symptoms. Many patients develop cognitive and reasoning deficits, memory changes, and significant alterations in personality and mood. It is not uncommon for family or friends to report that the patient has become argumentative, short tempered, and hostile. Vertigo is another common feature, as are migraine-like throbbing headaches that can be localized or widespread throughout the head and neck. The structures of the face and jaw can be involved in the regions of pain, although cranial and temporal symptoms are more common. Cluster-like severe episodic headaches also occur in some patients, and generalized cervical pain can occur in the absence of cervical spine or muscular damage. Postural changes are also known to aggravate headache symptoms. Most CPTH patients complain of headaches, mood changes, and memory and other cognitive dysfunctions. They have difficulty in concentrating, experience disordered thoughts, and feel depressed without reason. Some report pseudo-seizures or seizure-like episodes related to, or independent of pain. Headaches are frequent and chronic,

### Table 31–5  Cluster Treatments

<table>
<thead>
<tr>
<th>Abortive Medications</th>
<th>Preventive Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen: 100% at 7 L/min</td>
<td>Prednisone: oral, 20–40 mg/d</td>
</tr>
<tr>
<td>Lidocaine: 4% nasal spray</td>
<td>Cyproheptadine: oral, 4 mg every 8 hr</td>
</tr>
<tr>
<td>Sumatriptan: (oral, injectable, nasal)</td>
<td>Methysergide: oral, 2–4 mg every 12 hr</td>
</tr>
<tr>
<td></td>
<td>Lithium: oral, 300 mg every 8 hr</td>
</tr>
<tr>
<td></td>
<td>Valproate: oral, 250–500 mg every 12 hr</td>
</tr>
<tr>
<td></td>
<td>Carbamazepine: oral, 200 mg–400 mg every 8 hr</td>
</tr>
</tbody>
</table>
with durations that extend for days or weeks. Anhedonia and loss of sexual interest is also a common feature of CPTH. Treatment is similar to that of other vascular headache syndromes, but patients often respond better to tricyclic antidepressants, including amitriptyline. Recently, gabapentin has also been reported to be effective. Headaches and other symptoms usually slowly resolve over a period of several months to 2 years, but medications may need to be continued longer. If significant cognitive and neuro-motor changes have occurred, occupational therapy and psychotherapy may be required to improve productivity and a return to pretrauma levels of function.

### Headaches associated with vascular disorders

A wide range of headaches and cephalalgias occur as the result of vascular changes and disease within the CNS. Most are less common than the conditions presented thus far, but some discussion of these vascular conditions is important, since they have significant morbidity associated with their progression. Diagnosis usually requires vascular imaging along with neurologic assessment. Although headache is a common component, it is possible for these conditions to develop without headache, and the first manifestation can be other neurologic deficits including memory loss, decreased motor control, sensory changes, or pain in other regions of the body or within the cranium or head and neck. Several of the conditions require urgent care to avoid permanent brain damage or death. Since disruption of vascular supply to regions of the brain can occur, early discovery is important. Specific conditions that represent intracranial and nearby vascular pathology resulting in headache are included in Table 31–6.

#### Subarachnoid hemorrhage

Most of the conditions listed in Table 31–6 require immediate medical or neurosurgical intervention. Subarachnoid hemorrhage (SAH) results in paralysis, and most of the other conditions also cause serious, if not life-threatening brain damage if not managed early and effectively. Onset of head and neck pain with SAH is usually sudden and described as the most severe headache of the patient’s life; it is accompanied with nausea, vomiting, and mental and emotional changes. More than half of patients with SAH experience a warning leak with headache that precedes the full hemorrhagic episode. Detection during the leak period can result in saving life, however, mortality overall is almost 50%. Cranial imaging is necessary for detection, but not all patients with SAH are detected during such examinations. Lumbar puncture is essential as part of the diagnostic approach in suspected SAH and other intracranial sources of suspected pathology.

#### Carotid lesions

Of all vascular disorders, the most commonly detected in dental settings are carotid lesions, since they can often be identified by direct palpation of the carotid artery, which provokes the symptom complaint of the patient. Symptoms are similar to those of giant cell arteritis with palpation-triggered pain that radiates upward into the head and face. Both are seen predominantly in the elderly and are extremely uncommon before 60 years of age. Segmental enlargement of the carotid suggests that a dissecting aneurysm or carotid body tumor is present. Final diagnosis requires magnetic resonance angiography or an angiogram.

#### Giant cell arteritis

Giant cell arteritis (GCA) or temporal arteritis is an inflammatory disorder of the arterial vessels of the head and neck. Coronary vessels can also be involved, but most commonly the vessels affected include the ophthalmic artery, the temporal artery, and the carotid. Symptoms include diffuse headache that is unilateral, which can also include facial, neck, and jaw pain. Symptoms are initially low-grade and gradually become more severe and persistent until they reach a constant state. Pain increases at night and when the patient reclines. Fatigue is a common feature, and patients report that they feel ill. Pain in the maxilla and maxillary teeth is often reported, and symptoms also radiate into the TMJ and ear. In later stages, the patient takes on a pale complexion and distention of the involved vessels is evident. Since the vascular system is compromised and blood flow to the region is decreased, muscle activity can result in claudication of the jaw. This manifests as jaw fatigue with eating. Long-term risks associ-

### Table 31–6  Vascular Disorders Associated with Headache

- Subarachnoid hemorrhage
- Giant cell arteritis and other forms of arteritis
- Venous thrombosis
- Arterial hypertension
- Intracranial hematoma
- Unruptured vascular malformations
- Carotid and other vertebral artery disorders
- Acute ischemic cerebral vascular disease
- Intracranial aneurysm
ated with GCA include blindness caused by occlusion of vessels of the eye, stroke, and myocardial infarction, if coronary vessels are involved. Diagnosis is accomplished by ordering an erythrocyte sedimentation rate (ESR), which is almost always elevated, and arterial biopsy, to confirm the presence of giant cells. Treatment requires high-dose prednisone for up to 1 year. Prompt treatment is essential to avoid ocular damage or other CNS complications.

**Headaches associated with nonvascular intracranial disorders**

Many other intracranial disorders besides vascular changes can trigger pain and headache. Some of these can be life-threatening and, as with vascular disorders, prompt diagnosis is critical. Most of these conditions can be associated with headache or pain within the head and neck, but the onset of pain is highly variable, and depending upon the location of the lesion, pain can develop either early or late in the course of the disorder. Most of these conditions cause other neurologic and cognitive changes in addition to sensory and pain symptoms. Table 31–7 list several of the more common disorders.

Tumors are most commonly diagnosed via brain imaging, either magnetic resonance imaging (MRI) or computed tomography (CT). The location of pain, when present (about 50% of tumor patients report no headache), is often persistent and gradually radiates out over larger areas. Headache associated with tumors is worse in the morning and can cause nausea and vomiting. Symptoms increase when the patient bends over. Aggressive tumors can lead to rapid escalation of headache and other cranial symptoms, including motor dysfunction.

Intracranial infections normally are accompanied with high fever, hematologic evidence of systemic infection, and other neurologic changes. Viral infections are the most common type of intracranial infection seen in the United States. In most cases, intracranial infections are easily identified, except in cases of chronic low-grade infections, such as seen in Lyme disease, which, by nature is usually low-grade. Mood alterations are common in chronic intracranial infections, as is chronic fatigue. Diagnosis depends on the type of infection and whether serologic tests are available. Culture of spinal fluid can be valuable in establishing the final diagnosis.

**Low cerebrospinal fluid pressure headache**

Low cerebrospinal fluid (CSF) headache occurs after spinal anesthesia and after some cases of cranial trauma resulting in skull fracture, with gradual loss of fluid into the ear or nasal cavity. Occasionally CSF hypotension occurs in the absence of actual fluid loss from trauma. Symptoms include onset of headache when standing, with reduction of pain when the patient reclines. The pain is often located in the frontal region and accompanied with nausea, vomiting, dizziness, and stiffness of the cervical region. Diagnosis is established by lumbar puncture and detection of very low CSF pressure (less than 30 mm).

**High cerebrospinal fluid pressure headache**

High CSF fluid headaches (idiopathic intracranial hypertension or pseudotumor cerebri) are idiopathic, and the etiology is unclear. They occur in young adult obese females and are persistent. Symptoms often include generalized head pain with the retrobulbar location being the most common site. Eye movement and head movement cause symptoms to increase. The high level of hypertension within the brain causes significant pain, visual changes, such as diplopia, stiffness in the neck, and dysfunction of the sixth cranial nerve. Tetracycline use has been associated with the condition. Excessive pressure causes papilledema and threatens vision. Lumbar puncture reveals excessive fluid pressure. In some patients, compression of the jugular results in decreased pulsatile tinnitus, which is common in the disorder. Treatment includes removal of spinal fluid to reduce pressure, prednisone at or above 40 mg per day, and diuretics. If pressures continue to be high, surgical implantation of a shunt may be in order.

**Headaches caused by substances or withdrawal of substances**

A number of medications, illicit drugs, and other agents trigger the onset of headache or facial pain when discontinued. The most commonly known example is rebound headache, which occurs when patients have continually taken headache medications over an extended period of time. Rebound can occur with almost
any pain medication and is seen in headache patients after discontinuing acetaminophen, ibuprofen, and other prescription or OTC medications. The rebound process encourages patients to continue to use medications that may not be necessary or indicated, except that rebound headache occurs without the medication or agent. Coffee is another example of a potential rebound agent.

Other agents reported to trigger headaches include artificial sweeteners. Tobacco and alcohol withdrawal have been associated with onset of headaches in patients who are chronic users of narcotics and other psychoactive medications. Selective serotonin reuptake inhibitor antidepressants are known to commonly trigger headaches. Determination of which agent or medication is responsible for the headache requires removal of the medication, if possible. Rebound can occur from medications that have just been withdrawn and incorrectly suggest to the examiner that the medication should be restarted because it is providing a therapeutic effect. Obtaining a complete list of all OTC and prescribed medications is an important component in exploring headaches and their management.

Headaches caused by foods also represent a significant source of head pain. Common triggers are alcohol, pickled foods, chocolate, dairy products, nitrites, bananas, oranges, and raisins. Onions, beans, and nuts are also known to provoke headaches. An elimination diet may be required to discover the specific agent in food that triggers the headaches.

Metabolic disorders-induced headaches

A number of metabolic disorders can cause headaches that are generally diffuse and low-grade. Onset occurs when metabolic changes are significant enough to alter metabolic processes. Hypoglycemia is a common source of headache and occurs in diabetics and patients that have low blood sugar. Hyperglycemia can also trigger headaches, as can other metabolic disorders, such as hyperthyroidism. Conditions that decrease oxygen supply can provoke headaches, including respiratory disease, sleep apnea, and other functional causes of oxygen deprivation. Autoimmune diseases, such as lupus, are also known to trigger chronic severe headaches.

Suggested reading