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Orofacial Pain

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Orofacial pain (OFP) is the presenting symptom of a broad spectrum of diseases. As a symptom, it may be due to disease of the orofacial structures, generalized musculoskeletal or rheumatic disease, peripheral or central nervous system disease, or psychological abnormality; or the pain may be referred from other sources (eg, cervical muscles or intracranial pathology). OFP may also occur in the absence of detectable physical, imaging, or laboratory abnormalities. Some of these disorders are easily recognized and treated whereas others defy classification and are unresponsive to present treatment methods. The possible causes of orofacial pain are considerable and cross the boundaries of many medical and dental disciplines. An interdisciplinary approach is often required to establish a diagnosis and for treatment.

This chapter discusses new developments that have led to a better understanding of chronic pain and reviews the diagnosis and treatment of OFP disorders. Disorders of the musculoskeletal system that cause OFP are discussed in Chapter 10, "Temporomandibular Disorders".

▼ DEFINING PAIN

In this century, the concept of pain has evolved from that of a one-dimensional sensation to that of a multidimensional experience encompassing sensory-discriminative, cognitive, motivational, and affective qualities. The most recent definition of pain, produced by the Task Force on Taxonomy of the International Association for the Study of Pain (IASP) is, *"An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."*[1]

An accompanying explanatory note emphasizes the subjective and emotional nature of pain as well as the lack of correlation between pain and tissue damage.

> "Note: Pain is always subjective. Each individual learns the application of the word through experiences related to injury in early life. Biologists recognize that those stimuli which cause pain are liable to damage tissue. Accordingly, pain is that experience we associate with actual or potential tissue damage. It is unquestionably a sensation in a part or parts of the body, but it is also always unpleasant and therefore also an emotional experience. Experiences which resemble pain but are not unpleasant, eg, pricking, should not be called pain. Unpleasant abnormal experiences (dysesthesias) may also be pain but are not necessarily so because, subjectively, they may not have the usual sensory qualities of pain.
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> Many people report pain in the absence of tissue damage or any likely pathophysiological cause; usually this happens for psychological reasons. There is usually no way to distinguish their experience from that due to tissue damage if we take the subjective report. If they regard their experience as pain and if they report it in the same ways as pain caused by tissue damage, it should be accepted as pain. This definition avoids tying pain to the stimulus. Activity induced in the nociceptor and nociceptive pathways by a noxious stimulus is not pain, which is always a psychological state, even though we may well appreciate that pain most often has a proximate physical cause."[1]

Pain, in the medical model, is considered a symptom of disease, to be diagnosed and treated. Unfortunately, a cause and a diagnosis cannot always be established. Repeated attempts to identify a physical cause may result in unnecessary and sometimes harmful investigations and treatments. Establishing a precise diagnosis and providing effective treatment have become major challenges in medicine and dentistry. This has led to the development of a biobehavioral or biopsychosocial model to explain the phenomena observed in patients experiencing chronic pain. In this model, pain is not divided into physical versus psychological components. Instead, physical, psychological, and social factors are viewed as mutually influential forces with the potential to create an infinite number of unique pain experiences.[2] The biologic system deals with the anatomic, structural, and molecular substrates of disease. The psychological system deals with the effects of motivation and personality on the experience of illness and on reactions to illness. The social system deals with cultural, environmental, and familial influences on the expression and experience of illness. Each system affects and is affected by all of the others.[3]

The words "pain" and "suffering" have often been used synonymously, but the experience of suffering has been differentiated from pain. Suffering has been defined as including the experience of pain but as also including vulnerability, dehumanization, a lost sense of self, blocked coping efforts, lack of control over time and space, and an inability to find meaning or purpose in the painful experience.[2] The term "suffering" attempts to convey the experience of pain beyond sensory attributes.

Anatomic Considerations

Cranial nerve V (CN V), the trigeminal nerve, is the dominant nerve that relays sensory impulses from the orofacial area to the central nervous system. The facial (CN VII), glossopharyngeal (CN IX), and vagus (CN X) nerves and the upper cervical nerves (C2 and C3) also relay sensory information from the face and surrounding area (Table 11-1). (For a more detailed study of this topic, the reader is referred to the sources listed in the "Suggested Readings" section at the end of this chapter.)

Primary sensory neurons associated with pain (nociceptors) are characterized by small-diameter axons with slow conduction velocities (ie, finely myelinated A delta fibers and unmyelinated C fibers) (Figure 11-1). Nociceptors are activated by intense or noxious stimuli. Some are unimodal and respond only to thermal or mechanical stimuli; others are polymodal and respond to mechanical, thermal, and chemical stimuli. Nociceptors encode the intensity, duration, and quality of a noxious stimulus.

Information associated with pain is carried in the three divisions of the trigeminal nerve to the trigeminal sensory ganglion. The central processes of these neurons enter the pons, where they descend in the brainstem as the spinal trigeminal tract. Fibers from the spinal trigeminal tract synapse in the adjacent trigeminal nucleus that extends parallel to the tract in the brainstem. The spinal nucleus of CN V extends

TABLE 11-1 Cranial and Cervical Nerves That Provide Somatic and Visceral Sensation to the Orofacial Area

Nerve	General Area Served
V: Trigeminal	Skin of face, forehead and scalp as far as the top of the head; conjunctiva and bulb of the eye; oral and nasal mucosa; part of the external aspect of the tympanic membrane; teeth; anterior two-thirds of tongue; masticatory muscles; TMJ; meninges of anterior and middle cranial fossae
VII: Facial	Skin of the hollow of the auricle of the external ear; small area of skin behind the ear
IX: Glossopharyngeal	Mucosa of the pharynx; fauces; palatine tonsils; posterior one-third of the tongue; internal surface of the tympanic membrane; skin of the external ear
X: Vagus	Skin at the back of the ear; posterior wall and floor of external auditory meatus; tympanic membrane; meninges of posterior cranial fossa; pharynx; larynx
Cervical nerve 2	Back of the head extending to the vertex; behind and above the ear; submandibular, anterior neck
Cervical nerve 3	Lateral and posterior neck

TMJ = temporomandibular joint.

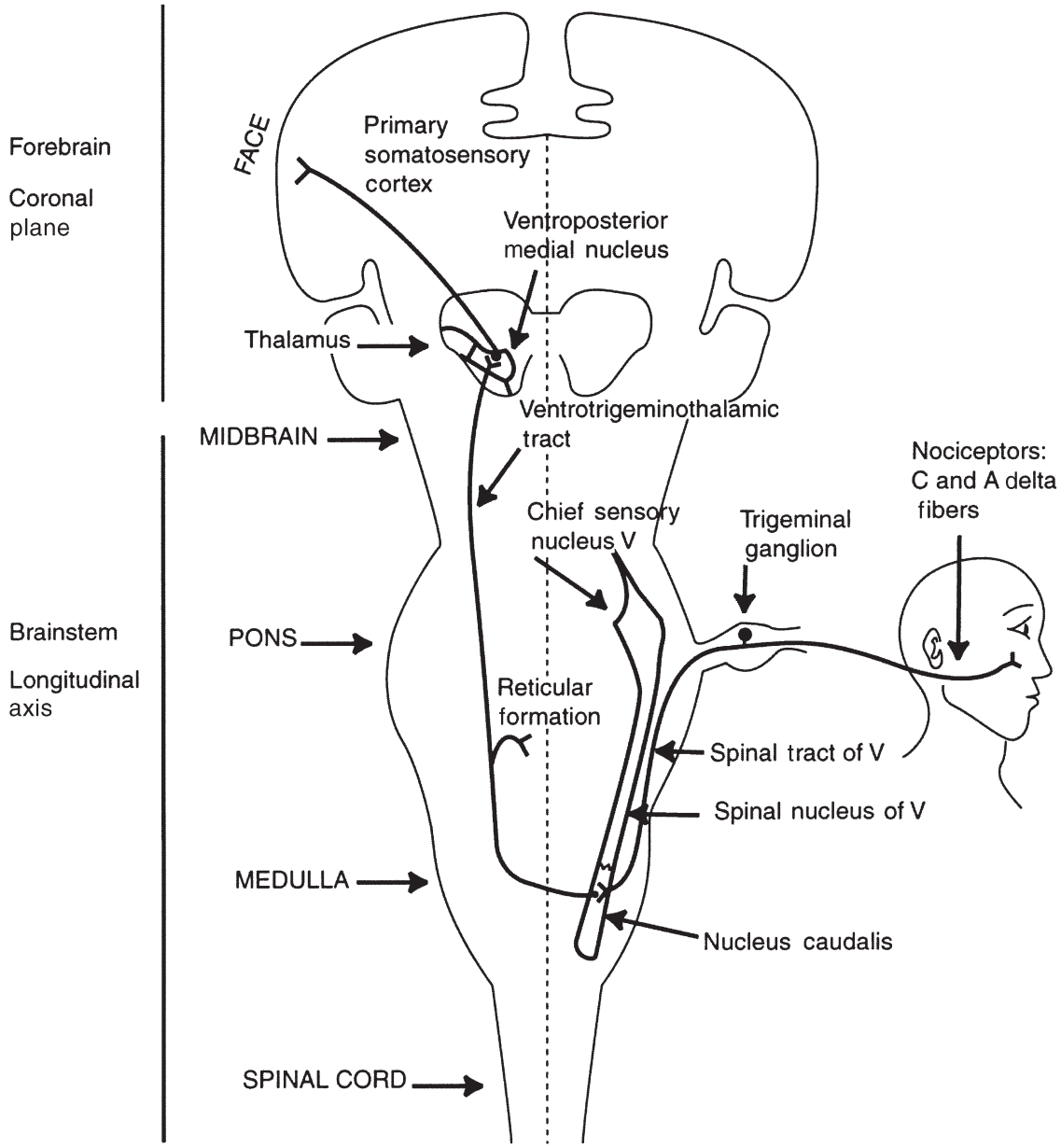


FIGURE 11-1 Nociceptive transmission associated with the trigeminal nerve.

from the chief sensory nucleus of CN V to the spinal cord, where it merges with the dorsal gray matter. The spinal nucleus is divided into three nuclei; the most caudal, the nucleus caudalis, is continuous with and resembles the dorsal horn of the cervical spinal cord.[4] Morphologic, clinical, and electrophysiologic observations indicate that the nucleus caudalis is the principal site in the brainstem for nociceptive information.[5–7] Axons from the spinal nucleus of CN V cross to the opposite side and ascend to the ventral posteromedial nucleus of the thalamus and also project to the reticular formation and the medial and intralaminar thalamic nuclei. From the thalamus, neurons course and end at the somatosensory cortex.

Measurement of Pain and Disability

There is no simple method of measuring pain. The intensity of an individual's pain is based on what is verbally or nonverbally communicated about the experience. Patients often express difficulty describing pain, and two people may have very different descriptions for pain that accompanies a similar injury. Within a specific diagnosis, great variability exists regarding the disabling effects of pain on an individual's life. Adding to this complexity is the lack of a direct correlation between the severity of a chronic pain disorder and the magnitude of the anatomic or pathologic change described by the clinical diagnosis.[8] In assessing OFP patients, pain intensity, emotional dis-

tress, and associated disability are important and cannot be captured with one scale or questionnaire. This has important implications for treatment because addressing the anatomic or pathologic abnormality alone may not eliminate pain and restore health. Individuals with cognitive impairment, infants, and children pose special challenges to the assessment of pain.

Pain intensity can be measured by using ratings such as a visual analog scale (VAS). A VAS consists of a 10 cm line on which 0 cm is "no pain" and 10 cm is "pain as bad as it could be." The patient marks the point along the line that best represents his or her pain, and the score is measured from the "no pain" end of the scale. Numeric scales (eg, 1 to 10) and descriptive rating scales (eg, no pain, mild, moderate, severe pain) are also used. Visual analog scales are sensitive to treatment effects,[9] can be incorporated into pain diaries, and can be used with children.[10]

The multidimensional aspects of pain are not well measured by scales that rate intensity. The McGill Pain Questionnaire (MPQ) (Figure 11-2) was created to measure the motivational-affective and the cognitive-evaluative qualities of pain, in addition to the sensory experience.[11] The questionnaire was designed to capture the multidimensional nature of pain and to provide quantitative measures of clinical pain that can be treated statistically. The questionnaire enables patients to choose from 78 adjectives (arranged in 20 groups) that describe pain. The form is designed to assess the sensory (groups 1 to 10), affective (groups 11 to 15), and evaluative (group 16) dimensions of pain and to produce a pain-rating index. There are also sections for the location and temporal characteristics of pain and a rating for present pain intensity.

The MPQ is used both by clinicians and researchers and has been helpful in pain research and treatment by providing a common language for assessing and comparing different pain experiences and treatment effects. Verbal descriptors have been shown to discriminate between reversible and irreversible damage of nerve fibers in a tooth[12] and between trigeminal neuralgia and atypical facial pain.[13] Toothache pain and pain from burning mouth syndrome were found to be equal in magnitude but significantly different in pain quality as assessed by the MPQ.[12]

Clinicians should include a rating or scale that can be used initially and during treatment to provide a reference for the course of the disorder and the treatment progress. Visual analog scales and numeric scales require no specific forms and are easily administered. The MPQ is available from the International Association for the Study of Pain (IASP) and is used in pain clinics and by clinicians focusing on pain management.

Patients experiencing pain may display a broad range of observable behaviors that communicate to others that they are experiencing pain (Table 11-2). These may be observable during the diagnostic interview or in response to physical examination procedures. An awareness of pain behaviors is valuable, but their presence or absence in any given situation is not necessarily diagnostic. These behaviors are often diminished or absent in patients with chronic pain and cannot be correlated with the presence or absence of pain or pain intensity.

TABLE 11-2 Observable Pain Behaviors

Behavior	Observations
Guarding	Abnormally slow, stiff, or interrupted movement
Bracing	Stiff, pain-avoidant posturing while in a static position
Rubbing	Touching, rubbing, or holding of the painful area
Sighing	Pronounced exhalation of air
Grimacing	Obvious facial expression of pain

Adapted from Keefe F et al.[14]

It is the patient's self-report that must be relied on for assessing the character and severity of pain. The scales and ratings described above are attempts to provide a rating that can be useful in diagnosis, treatment planning, and treatment progress and outcome assessment. Pain ratings also give the patient a method for keeping a pain diary to provide insight into what activities and events make the pain better or worse. Visual analog scales and numerical scales are relatively easy methods of charting pain intensity.

Assessments of disability related to a pain disorder and psychological status are important parts of any evaluation of chronic pain. Disability is defined as "a lack of the ability to function normally, physically or mentally."[15] The level of disability cannot be predicted on the basis of the anatomic diagnosis. One of the primary goals of chronic pain management (in addition to pain reduction) is the restoration of function. Since complete resolution of pain is often not possible, increasing function is an important measure of treatment success. There is no universally accepted method of assessing pain-related disability, but pain-related interference with activities and psychological impairments associated with pain are important aspects.

Turk and Rudy[8,16] have developed the Multiaxial Assessment of Pain (MAP) classification and have tested it on several pain populations, including a group of patients with temporomandibular disorders.[17] Their assessment included a 61-item questionnaire, the West Haven-Yale Multidimensional Pain Inventory (WHYMPI),[18] which measures adjustment to pain from a cognitive-behavioral perspective. The following three distinct profiles emerged: (1) "dysfunctional, characterized by patients who perceived the severity of their pain to be high, reported that pain interfered with much of their lives, reported a higher degree of affective distress, and maintained low levels of activity; (2) interpersonally distressed, characterized by a common perception that 'significant others' were not very understanding or supportive of the patient's problems; and (3) adaptive copers, patients with high levels of social support, relatively low levels of pain, perceived interference, affective distress, and higher levels of activity and perceived control".[19] Turk and Rudy found that when they used the MAP profiles, psychosocial and behavioral response patterns to pain were similar despite different medical and dental diagnoses. An assessment in this domain can be combined with any classification scheme related to OFP disorders, to provide a more comprehensive profile of the presenting problem. Establishing

MEASUREMENT OF PAIN

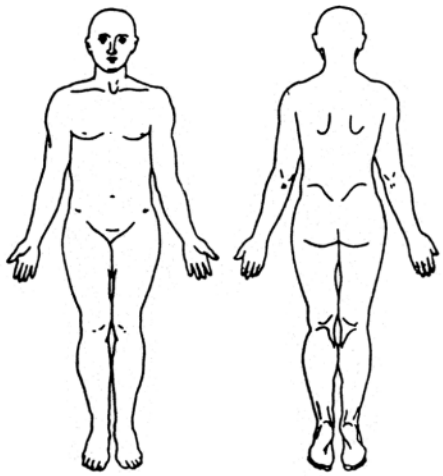
McGill Pain Questionnaire

Patient's Name _____ Date _____ Time _____ am/pm

PRI: S _____ (1-10) A _____ (11-15) E _____ (16) M _____ (17-20) PRI(T) _____ (1-20) PPI _____

1 FLICKERING QUIVERING PULSING THROBBING BEATING POUNDING	11 TIRING EXHAUSTING	
2 JUMPING FLASHING SHOOTING	12 SICKENING SUFFOCATING	
3 PRICKING BORING DRILLING STABBING LANCINATING	13 FEARFUL FRIGHTFUL TERRIFYING	
4 SHARP CUTTING LACERATING	14 PUNISHING GRUELLING CRUEL VICIOUS KILLING	
5 PINCHING PRESSING GNAWING CRAMPING CRUSHING	15 WRETCHED BLINDING	
6 TUGGING PULLING WRENCHING	16 ANNOYING TROUBLESOME MISERABLE INTENSE UNBEARABLE	
7 HOT BURNING SCALDING SEARING	17 SPREADING RADIATING PENETRATING PIERCING	
8 TINGLING ITCHY SMARTING STINGING	18 TIGHT NUMB DRAWING SQUEEZING TEARING	
9 DULL SORE HURTING ACHING HEAVY	19 COOL COLD FREEZING	
10 TENDER TAUT RASPING SPLITTING	20 NAGGING NAUSEATING AGONIZING DREADFUL TORTURING	
	PPI 0 NO PAIN 1 MILD 2 DISCOMFORTING 3 DISTRESSING 4 HORRIBLE 5 EXCRUCIATING	

BRIEF MOMENTARY TRANSIENT	RHYTHMIC PERIODIC INTERMITTENT	CONTINUOUS STEADY CONSTANT



E = EXTERNAL
I = INTERNAL

COMMENTS:

FIGURE 11-2 The McGill Pain Questionnaire.

a specific OFP diagnosis and assessing the psychosocial and behavioral issues is critical in the treatment and prognosis of chronic pain.

Dworkin, LeResche, and colleagues have developed a method for assessing dysfunctional chronic pain as part of a classification system, the Research Diagnostic Criteria.[20] They used the Graded Chronic Pain Severity scale,[21] the depression and vegetative-symptom scales from the symptom checklist-90-revised (SCL-90-R),[22] and a "jaw disability checklist." All three of these scales are based on questionnaires that are completed by the patient. The criteria were developed to advance the research in temporomandibular disorders. The criteria require validation, but the design of the classification makes it applicable to clinical practice. The Graded Chronic Pain Severity scale has four grades of disability and pain intensity based on seven questions, of which three are related to pain intensity and four are related to disability. The SCL-90-R depression scales are used to identify patients who may be experiencing significant depression, a problem commonly associated with chronic pain. These issues are discussed further in the section on assessment in this chapter.

▼CHRONIC PAIN

Although a precise definition of chronic pain has not been established, a distinction between acute and chronic pain has emerged. The somatosensory system serves the valuable function of warning the individual of actual or potential tissue damage. Nociceptors, specialized recptors that signal tissue damage, terminate in a highly ordered manner in the dorsal horn of the spinal cord and its homologous subnucleus caudalis in the spinal trigeminal nucleus. Information is transferred directly or through relay to the ventrobasal thalamus and then to the cortex. In the spinal cord, other pathways from the dorsal horn pass to the ventral horn and activate flexor motor neurons, generating the withdrawal flexion reflex.

This model draws attention to the protective aspect of the sensation of pain and is consistent with the qualities of acute pain. In other circumstances following peripheral tissue or nerve injury, a pathologic state may develop, resulting in persistent pain long after the injured tissue has healed. In this state, pain no longer represents a warning signal of potential or actual tissue damage; pain itself becomes the disorder.

Chronic pain is now recognized as a complex disorder that is influenced by biologic factors and by a range of psychosocial factors, including emotion, psychological distress, family and work environment, cultural background, the meaning of the pain, and appraisals of the controllability of the pain. Chronic pain has been defined as pain that persists past the normal time of healing,[23] but this may not be an easy determination. Alternatively, chronic pain has been related to duration (ie, pain that lasts longer than 6 months). Recently, pain lasting longer than 3 months has been used to define chronic pain. In the IASP publication on classification, Merskey describes chronic pain as "a persistent pain that is not amenable, as a rule, to treatments based on specific remedies, or to the routine methods of pain control such as non-narcotic analgesics."[1]

As pain persists, psychosocial issues (including depression, maladaptive beliefs about pain, medication abuse, strained interpersonal relationships, and ineffective coping strategies) become prominent aspects of the disorder.[24,25] The term "chronic pain syndrome" has been used to describe a condition that may have started because of an organic cause but is now compounded by psychological and social problems. The term has been criticized since it may obscure more-accurate physical and psychiatric diagnoses. It has sometimes been used pejoratively and has been interpreted by some to suggest a pain disorder that is psychological.[1] Originally, this label was used in an attempt to characterize a disorder that (regardless of its original cause) had evolved into a condition in which psychological and social problems were contributing to the persistence or exacerbation of the illness and in which significant disability was present.

In situations in which no ongoing peripheral injury was present to explain the pain, it was assumed that the pain was psychological. Patients need to be educated about the psychological distress and depression that can be a consequence of chronic pain. This is an important issue for clinicians and patients because of the demoralization and doubt patients develop about the condition and about their mental health.

Pathophysiology

The gate-control theory, introduced by Melzak and Wall in 1965,[26] articulated a model that explained the pain experience as a multidimensional process with many modulating influences. The proposed explanation for the persistence of pain after healing relates to changes (neuroplasticity) in the central nervous system.[27] Neurons are thought to be capable of altering their structure and function in response to stimuli, resulting in new stimulus-response relationships. This sensitization does not require ongoing peripheral input but is a consequence of changes in the sensitivity of neurons in the spinal cord.[28] These changes include the following:

1. A reduction of the stimulation threshold, with the result that the neurons no longer require a noxious stimulus in order to be activated
2. An alteration in the temporal pattern of the response, so that a transient stimulus evokes a sustained burst of activity
3. An increase in the general responsiveness of the motor neurons, so that a noxious stimulus produces a greater effect
4. The expansion of receptive fields, with the result that responses are evoked over a much wider area.

The clinical manifestations of these changes include hyperalgesia (an increased response to a stimulus that is normally painful); allodynia (pain due to a stimulus that does not normally provoke pain); and spontaneous, radiating, and referred pain.

The interaction between the sympathetic and somatosensory nervous systems has been associated with chronic pain and thought to be the cause of many but not all cases of com-

plex regional pain syndrome (CRPS). The relationship may be a coupling mediated by the neurotransmitter noradrenaline, which is released from sympathetic nerve endings acting on α-adrenoreceptors in the membrane of afferent neurons, causing depolarization. The mechanism is thought to be more likely a sensitivity of the somatosensory system than a hyperactivity of the sympathetic efferent system.[29]

Behavioral Issues

The observation that some individuals with high levels of pain continue to work while others are completely disabled led to the exploration of behavioral assessment and theories as a possible explanation.[30] Behavioral theories suggest that pain behaviors influence and are influenced by the patient's social environment.[30,31] The behavioral model views the pain behavior and associated disability as being as important as the underlying pathophysiology. A major goal in therapy is to modify pain behavior, thus improving function even when pain itself cannot be treated directly. Behavior therapy focuses on eliminating or reducing maladaptive behavior without theorizing about inner conflicts. It is based on principles of learning theory, particularly operant and classic conditioning.[3]

Pain itself can be viewed as a stress. The consequences of chronic pain (eg, loss of income, marital difficulties) are also significant stressors. Emotional distress is a component of pain, but it is also a consequence of pain, a cause of pain, or a concurrent problem with independent sources. These distinctions have not always been made clear, and there has been debate and confusion concerning whether emotional processes should be conceptualized as causes or consequences of pain.[32] The belief that chronic pain is a psychological disorder arose from two unproven assumptions: (1) chronic pain patients are a homogeneous group whose pain can be explained in terms of a more or less consistent constellation of personality characteristics, and (2) psychosocial disturbances (such as anxiety, depression, and social isolation) in pain patients reflect life events before the pain and are thus significant in explaining its onset.

The prevalence of depression is substantially higher in chronic pain patients compared to individuals without pain, but the majority of chronic pain patients are not depressed.[33] An association between chronic pain and depression exists, but no one hypothesis has emerged or has been proven to explain the relationship. Theories proposed include the following:

1. Depression causes hypersensitivity to pain.
2. Pain is a "masked" form of depression.
3. Depression is caused by the stress of chronic pain.

Depression, anxiety, and anger frequently coexist with chronic illness, but these reactions are not necessarily "psychopathological."[34] The literature suggests that in general, pain is more likely to cause emotional disturbances than to be precipitated by them.[35] The fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) uses the classification "mood disorder due to a general medical condition" to describe this. This classification is not considered to be a mental disorder.

A substantial group of chronic pain patients can be characterized as "dysfunctional" because of a consistent pattern of high levels of pain severity, affective distress, life interference, and lower-than-average levels of life control and activity.[36] The loss of customary roles at work, in the family, and in social settings, accompanied by the realization that neither one's own best efforts nor the interventions of highly respected health care professionals have been effective, is a major stressor. Challenges to the legitimacy of the complaints also represent a major source of stress. Excess use of medical services, hospitalizations for surgery, and abuse of medications are part of the profile of patients with dysfunctional chronic pain.

Treatment

Treatment that is specific to a particular pain disorder is discussed in a later section. The following is a discussion of general principles of treatment.

Even though it may occur in different locations, chronic pain tends to have certain characteristics regardless of the anatomic diagnosis or site. This tendency has led to the development of treatments to address the effects of chronic pain and to restore activity. These therapies are applied in multidisciplinary pain clinics (MPCs) regardless of whether pain is arising from the jaw, neck, back, or other anatomic site. MPCs have been organized in response to the recognition that pain is a complex physiologic, psychological, and sociologic experience beyond the expertise of any individual or discipline. Interdisciplinary therapy includes education, counseling, medication, pain management techniques (eg, electrical nerve stimulation techniques, nerve-blocking procedures, and acupuncture), psychological therapy (eg, cognitive, behavioral), relaxation training (eg, biofeedback, mental imagery, yoga, and meditation), hypnosis, occupational therapy, physical therapy modalities (eg, thermal and ultrasonic therapies, postural training), and stretching, strengthening, and conditioning programs. Treatment goals usually focus on managing medication misuse or abuse, increasing function, reducing the use of medical resources, decreasing pain intensity, and managing associated depression and anxiety. Behavioral therapy has been shown to be effective at reducing pain and improving function at work and at home.[37]

Pain reduction is a primary goal, but it is not always achieved. Published studies of pain reduction after treatment in pain clinics report pain reduction ranges of from 14[38] to 60%,[39] with an average pain reduction of between 20 and 30%.[40] Other treatment outcome criteria include reductions in addictive medication, reductions in the use of health care services, increased activity (including return to work), and closures of disability claims.[41] Providing effective treatment of chronic pain is challenging, and many of the treatments that are effective for acute pain fail to relieve chronic pain. Individuals suffering from chronic pain often seek care from many different practitioners and may be willing to submit to treatments that may complicate the problem or be harmful. This can result in more suffering and disability.

Chronic pain management is often seen as a low priority among health care providers; it is perceived as complicated, time-intensive, and often ineffective.[2] Ineffective medications are often overprescribed, repetitive examinations are conducted in an attempt to find a simple anatomic problem that is causing the pain, and comorbidities are ignored.[36] The failure to understand that chronic pain is a relevant clinical entity with physiologic and psychological consequences has been a barrier to improved care. This is complicated by a reluctance of patients to learn pain management or coping techniques, because their energy and attention is usually focused on finding a cure.

COGNITIVE THERAPY

Cognitive therapy is based on the theory that an individual's affect and behavior are largely determined by the manner in which she or he structures the world. A person's structuring of the world is based on ideas and assumptions (developed from previous experiences). For example, "I am stronger if I don't need medicine" is a cognition that contributes to poor adherence to prescribed medication.[3]

In chronic OFP practice, it is not unusual to encounter patients who express ideas that are based on faulty assumptions. Examples include the following:

1. A firm belief that an allergy or an undiscovered or low-grade infection is the cause of pain. Diagnostic testing that fails to find evidence of infection or allergy is not always sufficient to re-direct a patient's energy and attention to the pursuit of other factors or treatment. Infection and allergies are possible causes of pain, but when clinical findings are not supportive, the patient's persistent beliefs or attitudes may become a barrier to effective treatment.
2. Acceptance of the possibility that the pain is not a signal of ongoing or increasing tissue damage or life-threatening disease. This often prompts the patient to seek several consultations and to submit to invasive tests or procedures in an attempt to find a cause and (ultimately) a cure.
3. Anxiety about the possibility of further injury when pain increases with activity, resulting in deconditioning, inactivity, and increased emotional distress and limiting the potential for restored function and activity.
4. A focus on an orofacial structure or on a deviation from the ideal in respect to teeth and jaws, even though it is not responsible for the pain. This may complicate the situation and make it more difficult for the patient to accept a more multidisciplinary approach that includes behavioral management.

These are examples of maladaptive thoughts that lead to behaviors that contribute to the disability. Cognitive therapy is an effective method of exploring these thoughts and addressing them as part of the management. Cognitive-behavioral therapy attempts to alter patterns of negative thoughts and dysfunctional attitudes in order to foster more healthy and adaptive thoughts, emotions, and actions.[44] The cognitive-behavioral model suggests that patients develop negative and distorted convictions regarding their functional capacities, diagnoses, prognoses, and futures. These convictions about illness affect behavior and are reinforced when activity or reconditioning proves to be painful. Cognitive therapy interventions share four basic components: education, skill acquisition, cognitive and behavioral rehearsal, and generalization and maintenance.[42] Treatment is intended to identify and reframe negative cognitions while increasing the patient's range of activity.[34]

RELAXATION THERAPY

Relaxation techniques are used for nondirected calming rather than for achieving a specific therapeutic goal. They do not always reduce pain intensity and are recommended as an adjunctive treatment. The results of relaxation therapy may be more significant in reducing the distress associated with pain. Other benefits may include improved sleep, reduced skeletal-muscle tension, and decreased fatigue. Guided imagery, sometimes considered a relaxation technique, involves the recall of a pleasant or peaceful experience. Patients should be reassured that they are receiving this therapy not because "the pain is imaginary and they just need to relax," but because the therapy addresses an important area of distress that arises from having chronic pain.

Relaxation techniques share two basic components:[42] (1) a repetitive focus on a word, sound, prayer, phrase, body sensation, or muscular activity, and (2) the adoption of a passive attitude toward intruding thoughts and a return to focus.

Relaxation training produces physiologic affects that are opposite to those of anxiety (ie, slower heart rate, increased peripheral blood flow, and decreased muscle tension or activity). Relaxing muscle groups in a fixed order (progressive relaxation), imagining oneself in a place associated with pleasant relaxed memories (guided imagery), and doing yoga are examples. The reader is referred to the references listed in "Suggested Readings" at the end of this chapter for a more detailed discussion and for specific exercises that can be applied in management.

DRUG THERAPY

Drug therapy continues to be a significant part of chronic pain management. Analgesics are generally divided into three groups: non-opioids, opioids, and adjuvants. (Adjuvants are drugs that have been approved for use for conditions other than pain; anticonvulsants are an example.) The drugs in these groups have different pharmacologic actions although their analgesic actions are often not well understood. With the exception of clonazepam, benzodiazepines are not thought to be analgesic and are not recommended for long-term chronic pain management although they may be helpful for relief of muscle pain due to tension or spasm. Drug therapy for chronic pain often involves the simultaneous use of more than one drug. This takes advantage of the different mechanisms of action of different drugs. It may also allow the use of smaller

doses and may reduce adverse effects or risks. The most common example of this in dentistry is the combination of an opioid (such as codeine) with aspirin or acetaminophen, in which each drug acts at different sites.

Choosing the analgesic group (or groups) and the specific drugs is the first step in management. Drug therapy requires the individualizing of regimens for the greatest effect. In chronic pain management, a drug should also be selected to deal with "breakthrough" pain, an episode of increased pain that the regular regimen is not able to control. This drug is usually a μ-receptor agonist (e.g. oxycodone) with a relatively rapid rate of onset for a brief period. The oral route is preferred for compliance and convenience, and the drug dose requires titration to establish the appropriate regimen. An analgesic is most likely to be effective when given before pain increases and is usually best prescribed with a fixed dose schedule that does not require an increase in pain to signal the need for analgesia.

Non-opioid Analgesics. This group consists primarily of acetaminophen and the large group of nonsteroidal anti-inflammatory drugs (NSAIDs). Acetaminophen is dispensed over the counter and is also available in controlled formulations in combination with codeine and other opioids. Acetaminophen generally has fewer adverse effects when compared to NSAIDs. It does not affect platelet function, rarely causes gastrointestinal (GI) disturbances, and can be given to patients who are allergic to aspirin or other NSAIDs. Caffeine has been shown to enhance the effectiveness of non-opioid drugs and is often added to the analgesic.[43] The mechanism of action of acetaminophen is different from that of the NSAIDs but remains unknown; there is some evidence that suggests a central action.[44] Acetaminophen is generally used for mild pain of all types and is also combined with opioids for an additive analgesic effect or to reduce the amount of opioid required. Due to its potential to cause liver damage, it may pose a danger to patients with liver disease, patients who regularly consume moderate to large amounts of alcohol, and patients who are fasting.[45] Acetaminophen has an analgesic "ceiling," and the recommended maximum dose in a 24-hour period is 4 grams.[46]

NSAIDs are thought to work primarily at the site of injury by inhibiting the enzyme cyclo-oxygenase (COX), which is required for the synthesis of prostaglandins, substances that sensitize peripheral sensory nerves and contribute to the experience of pain. Users of NSAIDs do not exhibit tolerance or physical dependence, but these drugs do have an analgesic ceiling. Patients may vary in their response to NSAIDs, and if appropriate dosage adjustment does not produce an analgesic effect after several days to 1 week, it is appropriate to switch to a different NSAID. It is inadvisable to prescribe two different NSAIDs at the same time; rather, one NSAID should be used and its dose and timing adjusted for maximum analgesic effect. Combinations of NSAIDs increase the risk of side effects. Several NSAIDs (aspirin, ibuprofen, ketoprofen, and naproxen) are available in nonprescription formulations. The usual starting dose for these drugs is the dose recommended by the manufacturer. Titrating doses by starting at a lower dose and assessing incremental effects every 5 to 7 days has been recommended to achieve the greatest effect with the lowest dose.[47]

Prostaglandins (PGs) perform other functions in the body, and this is responsible for many of the side effects. PGs maintain the protective layer of gastric mucosa, and the loss of this layer makes the mucosa more vulnerable to erosion. The longer NSAIDs are administered, the greater the risk of GI bleeding. This effect is a systemic one and is not avoided by administering the drug by other routes (eg, rectal suppository). NSAIDs should be taken with food or at least with a full glass of water. Coadministration of misoprostol (a PG analogue) has resulted in a reduced risk of GI bleeding.[48] Risk factors that are indications for using misoprostol include age of more than 60 years, concurrent steroid therapy, high doses of the NSAID, and a history of ulcer disease. While all NSAIDs pose a risk of GI bleeding, ibuprofen and diclofenac are considered to pose a lower risk, and ketoprofen and piroxicam are considered to pose a higher risk.[49] Nabumatone is also considered to be less likely to cause GI effects.[50] With any NSAID, the risk increases when high doses are prescribed.

NSAIDs are available that selectively inhibit only one of the isoforms of COX, namely, COX-2. The inhibition of COX-2 seems to be related to the anti-inflammatory and analgesic effects whereas the inhibition of COX-1 is thought to be responsible for many of the side effects. The COX-2 inhibitors celecoxib and rofecoxib pose less risk of GI bleeding and do not inhibit platelet aggregation.

Opioids. The largest group of opioids that are commonly used for analgesia consists of the morphine-like agonists. Their most important effects are on the central nervous system and GI system. These drugs bind to μ opioid receptors, resulting in actions that lead to the analgesic effects. Opioids exert a number of effects after binding to receptor sites. Effects at the membrane level include opening potassium channels and inhibiting voltage-gated calcium channels, leading to a decrease in neuronal excitability. Opioids increase activity in some neuronal pathways (such as the descending inhibitory pathways) but may do so by suppressing the firing of inhibitory interneurons. At the spinal level, morphine inhibits the transmission of nociceptive impulses through the dorsal horn.[51] All μ agonists relieve pain by the same mechanism, but patients may vary in their responsiveness to the analgesic and to the adverse effects of specific agents. The use of opioid therapy in moderate to severe acute pain and cancer pain is well established. There has been an increased interest in the use of opioid analgesics for chronic nonmalignant pain. The practice remains controversial, and concern about addiction and behavior is the argument presented against opioid use. The concern relates to the risk of additional disability and antisocial behavior with long-term opioid use. The anecdotal literature suggests that in certain circumstances, opioids are an effective part of management and do not cause the predicted problems of addiction and antisocial behavior.[52] An

agreement between the patient and doctor along with close monitoring minimizes potential misuse.

Agonist-antagonist drugs such as buprenorphine, butorphanol, and pentazocine are used to treat moderate to severe acute pain. As a group, they have a more limited role than the μ agonists. Agonist-antagonist drugs may cause withdrawal symptoms in patients who are taking μ agonists, and they are more likely to cause psychotomimetic effects such as agitation, dysphoria, and confusion. Butorphanol nasal spray (Stadol [Bristol-Myers Squibb, New York, N.Y.]) is used for the treatment of migraine headache.

Adjuvant Drugs. This group of drugs has been approved for use in conditions other than pain. Alone or in combination with other analgesics and adjuvants, they have been found to be of value in pain management. Sequential trials are often necessary due to the variability of side effects and treatment responses; this may mean trying different drugs in the same group and in different groups. In controlled clinical trials, carbamazepine (an anticonvulsant) has proven to be effective for the treatment of trigeminal neuralgia.[53]

Amitriptyline (a tricyclic antidepressant), the antidepressant that has been most frequently studied in clinical trials, has been proven to be effective in chronic OFP treatment.[54,55] A patient with chronic pain who is receiving an antidepressant is considered to be better off than 74% of chronic pain patients who are receiving a placebo.[56] The magnitude of the analgesic effect was not different (1) for pain having an organic or psychogenic basis, (2) in the presence or absence of depression (masked or manifest), (3) in the presence or absence of an antidepressant effect, (4) in normal doses and in doses smaller than those that are usually effective in depression, and (5) for sedating and nonsedating drugs.

Information relating to pain from the periphery crosses a common synaptic pathway in the dorsal horn of the spinal cord and its homologue, the spinal trigeminal nucleus in the brainstem. The neurotransmitters serotonin and norepinephrine are thought to play a role in the descending inhibitory transmissions from the brain to the dorsal horn, modulating nociceptive impulses. Tricyclic antidepressants (TCAs) block the reuptake of serotonin and norepinephrine (NE), and this is thought to enhance the central inhibitory system in pain processing. These effects occur at doses that are lower than those required for an antidepressant effect, but further evidence is still required to explain the mechanism. TCAs are usually introduced at low doses and are gradually increased in an attempt to reduce the adverse effects, which can be intolerable even at low doses. Side effects such as dry mouth, increased appetite and weight gain, cardiac effects, sedation, and dysphoria may prevent the use of these drugs.

Anticonvulsant drugs are effective in the treatment of trigeminal neuralgia and diabetic neuropathy and for migraine prophylaxis.[57] There have been no clinical trials for the treatment of other OFP disorders with anticonvulsants. These drugs frequently produce side effects (including sedation, dizziness, ataxia, and mood changes) that can limit their usefulness. Newer anticonvulsants (specifically felbamate,[58] lamotrigine,[59] and gabapentin[60,61]) are receiving attention as possible therapies for pain. Gabapentin has become commonly used in pain management partly because of its relatively few side effects.[62] Movement disorders have been reported with gabapentin. The disorders resolve after administration of the drug is stopped.[63]

A variety of other drugs are used in the treatment of chronic pain although there is little research involving chronic OFP. These drugs include mexiletine,[64] clonidine,[65] clonazepam,[66] and alprazolam.[67]

Topical Medications. Topical analgesic therapy on the skin or oral mucosa has the advantage of reduced systemic absorption and thus a reduced risk of side effects. Capsaicin used as a topical cream has been the most researched drug in this field. It is effective in treating postherpetic neuralgia. Capsaicin is a natural product (extracted from the pungent red chili pepper) that has been used topically to treat a variety of pain conditions.[68] In a single application, neurogenic inflammation occurs and causes a burning sensation, followed by hyperalgesia. After multiple applications, the burning and hyperalgesia resolve. Topical application blocks C-fiber conduction, inactivates the release of neuropeptides from peripheral nerve endings,[69] and subsequently depletes the stores of substance P from sensory neurons.[70] The therapeutic effect is thought to be due to the depletion of substance P in C fibers,[68] decreasing their input to the central nervous system (CNS) neurons. Topical NSAIDs have been demonstrated to be effective for musculoskeletal pain.[71] Doxepin, clonidine, ketamine, cyclobenzaprine, and carbamazepine have been used topically in a variety of vehicles but have not been subjected to controlled trials.

Drug therapy for chronic pain is complex and often involves multiple drugs with different routes of administration. Patients often express anxiety about dependence on medication and may sometimes feel that drug therapy is used or recommended in place of "getting to the real cause" of the pain. When using drug therapy to treat the pain as the disorder, patients need information and education about the potential value of drug therapy as part of the comprehensive management of chronic pain.

▼CLASSIFICATION OF OROFACIAL PAIN

Classification is more than an academic exercise as it provides researchers and practitioners with a way of communicating and understanding groups of individuals who share a set of relevant characteristics. An understanding of the mechanisms of a disorder, the prescription of treatment, and the prognosis are important clinical issues that can be addressed in an effective classification system. Most of the present classifications are based on a consensus of existing knowledge and on unstructured examination findings or assumptions about the consistency of signs and symptoms. This weakness was illustrated in

a study of 35 patients who were diagnosed with atypical facial pain and whose findings were compared to the criteria established by the International Headache Society (IHS). Bilateral pain, pain-free periods, and paroxysms of pain were common in the patient group but were inconsistent with the criteria.[72]

Current Classification Schemes

Chronic pain classifications that address the physical, psychological, and social aspects of chronic pain provide a more comprehensive view of the disorder. Turk and Rudy proposed the Multiaxial Assessment of Pain (MAP), which integrates physical, psychosocial, and behavioral data.[8] They also developed a classification of chronic pain patients that is based on psychosocial and behavioral data alone.[16] They hypothesized that certain patterns exist in chronic pain patients regardless of the medical diagnosis. Three different response patterns emerged: dysfunctional patients, interpersonally distressed patients, and adaptive copers. The study indicated that despite differences in medical/dental diagnoses, patients had similar psychosocial and behavioral responses. A classification such as the MAP may be useful when combined with a classification that focuses on biomedical or physical conditions. The TMJ Scale,[73] the computer-based assessment system for psychosocial and behavioral issues (IMPATH),[74] and the Research Diagnostic Criteria (RDC)[75] are assessment systems for OFP that include psychosocial parameters.

The IHS, the American Academy of Orofacial Pain (AAOP), and the IASP have all produced classification schemes that include OFP. The IASP classification, originally published in 1986 and revised in 1994, is composed of five axes[1] (Table 11-3). The IASP has categorized OFP within the section termed "Relatively Localized Syndromes of the Head and Neck" (Table 11-4); listed within this section are 67 different disorders. The IASP publication includes a comparison between the IASP and IHS diagnostic categories that shows that there are significant differences between these two systems.

Two of thirteen categories in the IHS classification[76] specifically relate to OFP disorders: category 11, "headache or facial pain associated with disorders of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cranial structures," and category 12, "cranial neuralgias, nerve trunk pain, and de-afferentation pain." Category 11 includes temporomandibular joint disease and disorders of teeth, jaws, and related structures. Disorders in category 12 are listed in Table 11-5. The AAOP has used the IHS classification as the basis for a classification on OFP disorders.[77] A separate axis (not included in the publication) is recommended for defining psychosocial factors and diagnosing mental disorders. OFP disorders in this classification are listed in Table 11-6.

TABLE 11-3 Scheme for Coding Chronic Pain Diagnoses*

Axis	Definition
1	Regions (eg, head, face, and mouth)
2	Systems (eg, nervous system)
3	Temporal characteristics of pain (eg, continuous, recurring irregularly, paroxysmal)
4	Patient's statement of intensity: time since onset of pain (eg, mild, medium, severe; 1 month or less; more than 6 months)
5	Etiology (eg, genetic, infective, psychological)

Adapted from Merskey H, Bogduk N.[1]

*International Association for the Study of Pain (IASP) classification.

Classification of Idiopathic Facial Pain

ATYPICAL FACIAL PAIN

The use of the term "atypical facial pain" as a diagnostic classification has been recently discouraged.[1,77] Originally, the term was used to describe patients whose response to neurosurgical procedures was not "typical."[78] The term has been applied to various facial pain problems and has been considered to represent a psychological disorder although no specific diagnostic criteria have ever been established.

Atypical facial pain (AFP) is defined more by what it is not than by what it is. Feinmann characterized AFP as a nonmuscular or joint pain that has no a detectable neurologic cause.[54] Truelove and colleagues described AFP as a condition characterized by the absence of other diagnoses and causing continuous, variable-intensity, migrating, nagging, deep, and diffuse pain.[79] In the TMD classification of the AAOP, AFP is listed in the glossary of terms and is defined as "a continuous unilateral deep aching pain sometimes with a burning component." AFP was not included as a diagnostic category.[80] The IHS classification (IHS 12.8) uses the term, "facial pain not fulfilling other criteria" for AFP[81] (Table 11-7). Recent advances in the understanding of chronic pain suggest that at least a portion of patients who have been diagnosed with AFP may be experiencing neuropathic pain.

ATYPICAL ODONTALGIA

Atypical odontalgia (AO), described as a chronic pain disorder characterized by pain localized to teeth or gingiva,[1] has been considered to be a variant of AFP. The condition has also been called "phantom tooth pain" and defined as persistent pain in endodontically treated teeth or edentate areas for which there is no explanation to be found by physical or radiographic

TABLE 11-4 Classification of Localized Syndromes of the Head and Neck*

Neuralgias of the head and face
Craniofacial pain of musculoskeletal origin
Lesions of the ear, nose, and oral cavity
Primary headache syndromes, vascular disorders, and cerebrospinal fluid syndromes
Pain of psychological origin in the head, face, and neck
Suboccipital and cervical musculoskeletal disorders
Visceral pain in the neck

Adapted from Merskey H, Bogduk N.[1]

*International Association for the Study of Pain (IASP) classficiation.

TABLE 11-5 Classification of Cranial Neuralgias, Nerve Trunk Pain, and De-afferentation Pain*

IHS Category	Specific Disorders or Definition
12.1 Persistent (in contrast to ticlike) pain of cranial origin	Compression or distortion of cranial nerves and 2nd or 3rd cervical roots Demyelination of cranial nerves (optic neuritis) Infarction of cranial nerves (diabetic neuritis) Inflammation of cranial nerves (herpes zoster and postherpetic neuralgia) Tolosa-Hunt syndrome Neck-tongue syndrome
12.2 Trigeminal neuralgia (TN)	Idiopathic TN Symptomatic TN (caused by demonstrable structural lesion)
12.3 Glossopharyngeal neuralgia (GN)	Idiopathic GN Symptomatic GN (caused by demonstrable structural lesion)
12.4 Nervus intermedius neuralgia	Rare disorder characterized by brief paroxysms of pain felt deeply in the auditory canal
12.5 Superior laryngeal neuralgia	Rare disorder characterized by severe pain in the lateral aspect of the throat, submandibular region, and underneath the ear, precipitated by swallowing, shouting, or turning the head
12.6 Occipital neuralgia	Paroxysmal jabbing pain in the distribution of the greater or lesser occipital nerves, accompanied by diminished sensation or dysesthesia in the affected area; commonly associated with tenderness over the nerve concerned
12.7 Central causes of head and facial pain other than tic douloureux	Anesthesia dolorosa: painful anesthesia or dysesthesia, often related to surgical trauma of the trigeminal ganglion, evoked most frequently after rhizotomy or thermocoagulation for treatment of idiopathic TN Thalamic pain: unilateral facial pain and dysesthesia, attributed to a lesion of the quintothalamic pathway or thalamus
12.8 Facial pain not fulfilling criteria in groups 11 and 12 (previously used terms: atypical facial pain, atypical odontalgia)	Persistent facial pain that does not have the characteristics of the cranial neuralgias classified above and is not associated with physical signs or a demonstrable organic cause

Reproduced with permission from Olesen J.[76]

*International Headache Society (IHS) classification.

examination.[82] In the AAOP classification, AO is listed within the category "facial pain not fulfilling other criteria" and is considered to be a de-afferentation pain.[77] AO appears in the IASP classification under "lesions of the ear, nose, and oral cavity" and is defined as a severe throbbing pain in the teeth in the absence of a major pathology.

In an attempt to identify chronic OFP due to neuropathic injury, Lynch and Elgeneidy suggested additional categories to the IASP taxonomy.[83] They also recommended replacing AFP with the term "not otherwise specified." This is the terminology used in the DSM-IV for a condition that does not conform to criteria in another category.[84] While the term "atypical facial pain" has a long history and has been associated with a number of different etiologies, it still is used by clinicians to identify an OFP disorder that does not meet other diagnostic criteria and that is characterized by its chronicity and lack of response to most treatments. The term may fade away as new knowl-

TABLE 11-6 Differential Diagnosis of Orofacial Pain*

Intracranial pain disorders	Neoplasm, aneurysm, abscess, hemorrhage, hematoma, edema
Primary headache disorders (neurovascular disorders)	Migraine, migraine variants, cluster headache, paroxysmal hemicrania, cranial arteritis, carotodynia, tension-type headache
Neurogenic pain disorders	Paroxysmal neuralgias (trigeminal, glossopharyngeal, nervus intermedius, superior laryngeal) Continuous pain disorders (de-afferentation, neuritis, postherpetic neuralgia, post-traumatic and postsurgical neuralgia) Sympathetically maintained pain
Intraoral pain disorders	Dental pulp, periodontium, mucogingival tissues, tongue
Temporomandibular disorders	Masticatory muscle, temporomandibular joint, associated structures
Associated structures	Ears, eyes, nose, paranasal sinuses, throat, lymph nodes, salivary glands, neck

Reproduced with permission from Okeson J.[77]

*American Academy of Orofacial Pain classification.

TABLE 11-7 Classification of Idiopathic Orofacial Pain*

Daily pain that is deep and poorly localized, persisting for most or all of the day
Pain at onset confined to a limited area on one side of the face and that may spread to the upper and lower jaws or a wider area of the face or neck
Pain not associated with sensory loss or other physical signs, and laboratory investigations (including radiography of face and jaws) do not demonstrate relevant abnormality.

Reproduced with permission from Committee on Headache Classification, International Headache Society.[81]

*International Headache Society (IHS) classification 12.8: Facial Pain Not Fulfilling Other Criteria.

edge identifies causes for these disorders and allows for better classifications and treatment.

NEURALGIA-INDUCING CAVITATIONAL OSTEONECROSIS

Ischemic osteonecrosis of the jaws has been presented as a cause of idiopathic facial pain. The term given to describe this disorder is "neuralgia-inducing cavitational osteonecrosis" (NICO). The pain is described as slowly progressive over time and spreading. It may be intermittent and may vary in extent, location, and character. This disorder has been described as occurring at a wide range of ages but is more frequent in the fourth and fifth decades of life. It is thought to occur most frequently in the mandibular molar area. Most NICO sites are thought to involve edentulous areas or areas associated with radiographically successful endodontic procedures. No specific imaging criteria are diagnostic.[85] There continues to be significant debate about NICO as a cause of facial pain.[86] Treatment by surgically entering and curretting these cavities raises a concern about the possibility of exacerbating the disorder rather than controlling it. Procedures that risk nerve injury are generally not recommended for patients with persistent neuropathic pain. The lack of clearly defined criteria for the diagnosis of these conditions raises the risk of additional injury and aggravation of the symptoms.

▼ EXAMINATION AND ASSESSMENT OF THE PATIENT WITH CHRONIC OROFACIAL PAIN

The examination and assessment of patients with chronic OFP is challenging for all clinicians. In most disorders, no specific biologic marker, validated diagnostic criteria, or "gold standard" exists. Biologic markers, including tyramine,[87] oxygen free radicals,[88] and metabolites of neurotransmitters in lumbar cerebrospinal fluid,[89] have been studied in a limited manner in regard to OFP and are not applicable in diagnosis. Even test procedures that are considered objective, such as local anesthetic nerve blocking[90] and the testing of sensation after nerve damage,[91] have yielded inconsistent results. A systematic approach for collecting diagnostic information is needed to minimize the risk of missing critical information. A formal and systematic approach increases the probability of identifying disease that occurs from time to time and is life threatening.[92]

History, physical examination, and behavioral assessment usually serve as the basis for diagnosis. Frequent re-evaluation, including assessment of the effects of treatment, is an important part of this process. In circumstances in which treatment is ineffective or only partially successful, patients are at risk of seeking additional and alternative treatments that may be inappropriate and potentially dangerous.[93] Even when a diagnosis is uncertain or when previous treatment has failed, the clinician can make a valuable contribution by coordinating the further use of medical and dental services and by being available to advise the patient about possible treatments. Validating patients' feelings and symptoms in these times is critically important and serves to reduce suffering.

History

Evaluation of OFP symptoms must include all of the standard components of a medical interview: chief complaint, history of present illness, past medical history, medications, review of systems, and family and social history. A diagnosis can sometimes be made on the basis of the history, or the possibilities can be significantly narrowed. Since there are a number of OFP disorders that do not produce physical abnormalities, the history and description of pain may serve as the basis for the diagnosis.

HISTORY OF THE PRESENT ILLNESS

A history of the present illness should include a detailed description of the pain and its location (Table 11-8). The VAS or numeric scale described above can be used to assess intensity, and a questionnaire such as the MPQ can capture the multidimensional experience of the pain. Details of previous injuries, surgeries, and radiation therapy should be obtained. Questions about habits such as gum chewing and tooth clenching or grinding may reveal important contributing factors of which the patient is unaware. The effects of eating, opening the mouth wide, rest, exercise, and heat and cold on pain should be explored. Referred pain to the orofacial region is an important clinical consideration. The location of pain,

TABLE 11-8 Pain Characteristics

Intensity
Quality
Location
Onset
Associated events at onset
Duration and timing of pain
Course of symptoms since onset
Activities or experiences that increase pain
Activities or experiences that decrease pain
Associated symptoms (eg, altered sensation, swelling)
Previous treatments and their effects

therefore, will not always correspond to its source. A mechanism that has been proposed to explain referred pain is convergence,[94–96] in which primary afferent fibers from different sites converge on the same second-order neuron in the brainstem nucleus. Patients should mark the location and extent of pain on a diagram.

PAST MEDICAL HISTORY AND REVIEW OF SYSTEMS

The past medical history and review of systems should help provide an insight into the general health of the patient and may provide clues regarding the present pain complaint. Pain may be a presenting feature or an ongoing complaint in systemic disease (eg, connective-tissue disease, demyelinating disease of the CNS, metastatic disease).

The patient's use of medication (including over-the-counter preparations, naturopathic and homeopathic remedies, and prescription drugs) should be recorded. Prescription medication is often used incorrectly; therefore, the directions as well as the actual usage should be determined. The medication list may uncover a medical condition that the patient failed to mention in other questioning. Drug effects such as fatigue, dizziness, anxiety, insomnia, or depression may affect the patient's pain complaints. The use of tobacco, alcohol, caffeine, or illicit drugs should be explored.

FAMILY, SOCIAL, AND OCCUPATIONAL HISTORY

Chronic pain can have disastrous effects on one's ability to maintain daily activities and fulfill responsibilities. Pain has profound and often negative effects on family and social relationships, and it is important to assess the level of dysfunction that may have occurred. Traumatic events or emotional distress prior to the onset of pain, a history of other close family members with chronic illness or pain, and changes in work and or marital status should be explored because these can be significant stressors.

Physical Examination

The physical examination may identify an abnormality that explains the cause of pain. It can also help eliminate from diagnosis the presence of serious disease related to the pain. The examination should include the following:

1. Inspection of the head and neck, skin, topographic anatomy, and swelling or other orofacial asymmetry
2. Palpation of masticatory muscles, tests for strength and provocation
3. Assessment and measurement of the range of mandibular movement
4. Palpation of soft tissue (including lymph nodes)
5. Palpation of the temporomandibular joint
6. Palpation of cervical muscles and assessment of cervical range of motion
7. Cranial nerve examination (Table 11-9)
8. General inspection of the ears, nose, and oropharyngeal areas
9. Examination and palpation of intraoral soft tissue
10. Examination of the teeth and periodontium (including occlusion)

MUSCLE EXAMINATION

Pain that is reproduced or increases as a result of muscle palpation may point to the source of the pain and to a diagnosis. The degree of finger pressure will influence the result of the palpation examination, and patients' responses to palpation may vary with time. Pressure algometers have been used in research to help standardize examination procedures[97] but are not commonly used in clinical practice. Muscle palpation has been shown to yield reliable scores among examiners, but the diagnostic validity and reliability of muscle palpation has not been established. The masseter and temporalis muscles can be palpated bilaterally to identify differences in size or firmness. The suprahyoid muscles, mylohyoid, and anterior belly of the digastric should be included in the palpation examination. Intraoral techniques have been described for palpating the medial and lateral pterygoids. The ability to perform a meaningful palpation examination of the lateral pterygoid has been questioned.[98] Palpation techniques have been described, but it may be difficult to distinguish tenderness associated with the procedure from an actual muscle abnormality.[99] The temporalis tendon, where it inserts onto the coronoid process, can also be reached intraorally for palpation. Testing muscles against resistance in a static position and having the patient clench on separators to prevent the teeth from coming together may help identify the source of pain.[100]

Palpation of cervical muscles and a general assessment of the cervical range of motion may indicate an abnormality contributing to the pain complaint. Pain localized to the orofacial region can be referred from neck muscles.[94] The cervical muscles to be palpated include the trapezii and the sternocleidomastoid and the muscles that lie deeper between them, including the capitus and scalene muscles and the levator scapulae.

RANGE OF MOTION ASSESSMENT

Mandibular and cervical ranges of motion should be assessed. Movements with and without pain should be noted. Mandibular movements with comfort, with pain, and with examiner assistance should be measured and recorded. Cervical motion can be examined during active, passive, and resisted motions. When restrictions in movement are thought to be caused by muscle guarding, the application of a vapocoolant spray such as Fluori-Methane Spray (Gebauer Co., Cleveland, Ohio) followed by stretching may significantly increase range, confirming a muscle cause. Alternatively, injection of a local anesthetic into muscle may block pain and thus identify the source of the pain and the restricted movement.

INTRAORAL EXAMINATION

A systematic intraoral inspection looking for changes in form, symmetry, color, and surface texture should be carried out. The examination should include manipulation of the tongue and mandible to clearly visualize all areas. Pooling of saliva on the floor of the mouth should be observed. The palate and tongue should be examined at rest and during function to detect underlying masses that might displace or alter the normal structures. The examiner's finger should palpate the alveolar processes, lateral and posterior parts of the tongue, floor

TABLE 11-9 Summary of Cranial Nerve Examination

Cranial Nerve	Function	Usual Complaint	Test of Function	Physical Findings
I (Olfactory)	Smell	None or loss of "taste" if bilateral	Sense of smell with each nostril	No response to olfactory stimuli
II (Optic)	Vision	Loss of vision	Visual acuity Visual fields of each eye	Decreased visual acuity or loss of visual field
III (Oculomotor)	Eye movement Pupillary constriction	Double vision	Pupil and eye movement	Failure to move eye in field of motion of muscle Pupillary abnormalities
IV (Trochlear)	Eye movement	Double vision, especially on down and medial gaze	Ability to move eye down and in	May be difficult to detect anything if 3rd nerve intact
V (Trigeminal)	Facial, nasal, and oral sensation Jaw movement	Numbness Paresthesia	Light touch and pinprick sensation on face Corneal reflex Masseter contraction	Decreased pin and absent corneal reflex Weakness of masticatory muscles
VI (Abducens)	Eye movement	Double vision on lateral gaze	Move eyes laterally	Failure of eye to abduct
VII (Facial)	Facial movement	Lack of facial movement, eye closure Dysarthria	Facial contraction Smiling	Asymmetry of facial contraction
VII (Auditory and vestibular)	Hearing Balance	Hearing loss Tinnitus Vertigo	Hearing test Nystagmus Balance	Decreased hearing Nystagmus Ataxia
IX (Glossopharyngeal)	Palatal movement	Trouble with swallowing	Elevation of palate	Asymmetric palate
X (Vagus)	Vocal cords	Trouble swallowing	Vocal cords	Brassy voice
XI (Spinal accessory)	Turns neck	None	Contraction of sternocleidomastoid and trapezius	Paralysis of sternocleidomastoid muscle
XII (Hypoglossal)	Moves tongue	Dysarthria	Protrusion of tongue	Wasting and fasciculation or deviation of tongue

of the mouth, buccal mucosa, and hard and soft palate to identify abnormalities that may not be readily observed. The finger should not meet significant resistance as it moves across a normally lubricated mucosa. The openings of the submandibular and parotid salivary gland ducts should be isolated and dried with cotton; the glands should then be "milked" to verify a clear flow of saliva.

The dentition should be examined for wear, damaged teeth, and evidence of caries. This inspection should be followed by probing, palpation for tooth mobility, and percussion of teeth. If a pulpal problem is suspected, thermal and vitality tests should be included. Applying differential pressure on the teeth by having the patient bite down on cotton rolls, wooden bite sticks, or one of the commercially available instruments designed to apply concentrated pressure on cusps may identify pain associated with a vertical crown or root fracture. Periodontal structures should be examined for color changes suggestive of inflammation, altered gingival architecture that occurs with chronic disease, swelling, or other surface changes. Periodontal probing should be performed to identify bleeding points and pocket depths. Tooth contacts in the maximum intercuspal position, in centric relation, and during excursive movements should be identified. Heavy contacts or interferences in association with tooth mobility or tooth sensitivity may indicate conditions contributing to occlusal trauma.

Pain-Related Disability and Behavioral Assessment

An interview most often serves as the basis for a behavioral assessment. Self-report questionnaires and instruments that include methods of scoring are also in use to assess disability and psychological factors. The assessment should explore the following:[101]

1. Events that precede and follow exacerbation of pain
2. The patient's daily activities
   - — How time is spent during the day and in the evening
   - — Activities that have been performed more often or less often since the onset of pain
   - — Activities that have been modified or eliminated since the onset of pain
3. Relatives or friends that suffer chronic pain or disabilities of a similar nature
4. The degree of affective disturbance
   - — Change in mood or outlook on life
   - — Satisfaction level with friends and family relationships

— Vegetative signs of depression (sleep disturbance, change in food intake, decreased sexual desire)

While psychosocial factors are of great importance in pain disorders, studies indicate that physicians and dentists do not always adequately recognize psychological problems.[102–104] One of the problems dentists face is the lack of formal training in psychological assessment. A great deal of study has been focused on the use of questionnaires to assess psychosocial status. Inventories that are completed by the patient, such as the Minnesota Multiphasic Personality Inventory (MMPI), the Beck Depression Inventory,[105] the Zung Self-Rating Depression Scale, the Personality Diagnostic Questionnaire,[106] and the General Health Questionnaire,[107] are examples of self-report questionnaires used for psychological assessment. The TMJ Scale,[73] IMPATH,[74] and (more recently) the RDC[20] are instruments designed for evaluating OFP, and they include behavioral assessments. No one method has gained widespread acceptance for evaluating OFP patients. One strategy for addressing the lack of psychological assessment skills among physicians and dentists is to provide a method of screening that identifies OFP patients who might benefit from a more thorough behavioral assessment. Gale and Dixon[108] found that the following two questions correlated with lengthier questionnaires:

1. How depressed are you?
2. Do you consider yourself more tense than calm or more calm than tense?

Oakley et al used a five-item questionnaire that allows patients to rate levels of depression, anxiety, and recent life stresses showed moderate to strong association with results from extensive psychological testing.[109] Two of the questions were similar to those used by Gale and Dixon.

Being asked open-ended questions about common areas of life experience provides the patient with an opportunity to express concerns or problems that may not otherwise be communicated, such as what the patient feels may be the cause of pain; activities or problems in the common areas of life (work, love, and play); and complaints of current or previously diagnosed or undiagnosed pain elsewhere in the body. Responses to these questions may be helpful in identifying abnormal thought patterns, external stressors, or other symptoms that are suggestive of a more generalized pain disorder. Recent research indicates that the prevalence of a history of physical and sexual abuse in patients with chronic pain is higher than expected, but how to identify patients who should be referred to experienced therapists remains a challenge.[101]

Self-report instruments are used for clinical and research purposes to assess psychological variables associated with pain. They provide standardized assessments and are sensitive to treatment-related changes. Instruments such as the Minnesota Multiphasic Personality Inventory (MMPI) and the revised MMPI (MMPI-2) have been used to evaluate psychological distress in chronic pain patients. The use of the MMPI or MMPI-2[110] with chronic pain patients has been questioned because the subjects who were used to standardize the inventories were not chronic pain patients.[111–113] In chronic pain patients, elevations on the hypochondriasis, depression, and hysteria scales have been associated with severe pain, affective disturbance, and disability.[114,115] MMPI profiles have been unable to predict treatment outcomes.[101] Other shorter and simpler instruments such as the Beck Depression Inventory are used in place of the MMPI.[116] The shorter inventories are likely to get better patient compliance as well. Questionnaires such as the SCL-90-R,[22] the Millon Behavioral Health Inventory,[118] and the Illness Behavior Questionnaire[117] are examples of shorter inventories that take less time to complete. A universally accepted assessment instrument for chronic pain patients does not exist.

Dworkin and colleagues[20] have published (as part of the RDC) a classification for assessing pain-related disability, identifying depression, and characterizing limitations related to mandibular functioning. The RDC were produced to increase the standardization of assessment and classification applied to clinical research on TMD. While this assessment/classification requires further validation, it may be of value to clinicians. The pain-related disability assessment is based on the "Graded Chronic Pain Status," a seven-item questionnaire, and specific scoring.[21] An explanation of the scoring and the scale can be found in Von Korff's article.[21] The assessment method, scoring, and discussion of the pain-related disability status have been published by Dworkin, LeResche, and colleagues.[20]

From the discussion above, it should be apparent that there is no universal standard that can be relied on to provide a screening assessment of behavioral and pain-related disability. Table 11-10 lists questions discussed in this section that may be valuable as part of this assessment. These questions may be posed during the interview to explore possible behavioral, psychological, or other systemic problems that may have an impact on the diagnosis and management of an OFP disorder. This is not a scale or instrument with scoring but questions that may provide an opportunity for the patient to communicate issues

TABLE 11-10 Questions to Consider for Screening Assessment

What events precede and follow increased episodes of pain?
How is time spent during the day and the evening?
What activities are performed more often or less often since the onset of pain?
What activities have been modified or eliminated since the onset of pain?
Do relatives or friends suffer chronic pain or disabilities of a similar nature?
Do you characterize yourself as depressed?
Do you have changes in sleep pattern, food habits, sexual desire (vegetative signs of depression)?
Have there been changes in your relationships with friends, family, co-workers?
Do you characterize yourself as being anxious or tense?
Do you think you have experienced a lot of stressful situations over the past year?
What do you think is causing the pain?
Do you presently have any diagnosed or undiagnosed pain complaints elsewhere in the body?

that may be important to the complaint. The threshold for deciding when the information obtained indicates a more thorough investigation is a clinical judgment. There are no well-defined rules to govern this decision.

When psychosocial issues are thought to be significant and to require assessment and possible management by a psychologist or psychiatrist, the patient should be so advised. This should be done in a conversation that allows the patient to respond and that asks for feedback since the patient may have some insight into the issue. The interviewer's opinion may help to validate the patient's own assessment, and the possibility of successfully addressing these issues may be increased. Communicating with the patient's general physician or referring the patient to his or her general physician to explore this further may be an effective method of managing the situation.

When a psychiatric disorder is suspected, a direct referral to a psychiatrist or psychologist may be indicated.[119] The patient may resist this referral for the following reasons:

1. Perception of the referral as a judgment that the problem is only psychological or as a personal rejection
2. Beliefs about the legitimacy of psychiatric therapy and about the kind of people who consult psychologists or psychiatrists
3. Beliefs about the condition that do not include the possibility of a psychological or emotional component

A patient is most likely to accept a recommendation if a trusting relationship is present. The following are suggestions that may facilitate the referral:

1. Make the referral a part of the evaluation. Inform the patient that the consultation is part of your complete evaluation and that it will be part of the other clinical findings for determining the diagnosis and management.
2. Arrange the appointment at the same time that the patient is in the office if the patient agrees. This will facilitate the process.
3. Provide the patient with information about what the consultation will involve.
4. Schedule a follow-up appointment to review the findings and discuss treatment.

Diagnostic Imaging

Imaging can be used to confirm a suspected abnormality, to screen or rule out possible abnormalities that are not detectable by other methods, or to establish the extent of an identified disorder. It is the best method for evaluating a suspected tumor, infection, or ongoing inflammation in sites that are not easily accessible. Many OFP disorders do not produce abnormalities demonstrable with imaging, and its greatest value may be to rule out serious life-threatening disease.

Diagnostic Nerve Blocks

Nerve blocks interrupt the transmission of nociceptive impulses through specific pathways. If pain relief occurs, it is presumed to be due to the interruption of the nerves via the pathways suspected of being involved. Conversely, the absence of pain after a successful block suggests the possibility of a central process.[120] False-positive results may occur due to systemic effects of local anesthetics, blockade of afferent pathways other than those intended, and placebo effects. Conversely, lack of pain relief may be due to technical or anatomic factors.[121] Diagnostic nerve blocks are a valuable part of an assessment, but the results can be equivocal and do not always contribute to an accurate diagnosis. There is a high frequency of placebo response to local anesthetic blocking, even among patients diagnosed with neuropathic pain.[90]

Nerve blocks to diagnose sympathetically maintained pain include local anesthetic block of the sympathetic chain (eg, stellate ganglion), regional guanethedine block (intravenous injection into an arm or leg), and intravenous phentolamine to block the α-adrenoreceptor, preventing the excitation of afferent nociceptors by noradrenaline. The interpretation of these tests has been challenged because of the lack of placebo-controlled procedures and because of a high placebo response,[122,123] but the weight of evidence supports the hypothesis that the sympathetic nervous system contributes to chronic pain in some circumstances.

Local anesthetic blocking should be considered in the context of all of the clinical findings. Topical, intraligament, infiltration, and regional block anesthesia may identify a peripheral site that is responsible for pain. A complete resolution of pain after local anesthetic application or injection should prompt an investigation for a local cause. The injection of local anesthesia may produce ambivalent results when patients report a change in symptoms but not necessarily resolution of pain. In these circumstances, one should consider a more central cause of pain.

Laboratory Tests

Laboratory tests have limited value except in special circumstances. Most OFP disorders do not cause abnormalities that can be identified in laboratory specimens. Exceptions include temporal arteritis, in which the erythrocyte sedimentation rate is elevated and temporal artery biopsy is abnormal and collagen vascular diseases that cause detectable immunologic abnormalities.

Consultation and Referral

Referral and consultation are recommended for a number of reasons, and there are few rigid rules. For a complex pain problem, it may be necessary to include examinations by other dental specialists, otolaryngologists, neurologists, psychologists or psychiatrists, and internists. Referrals may be of value when (1) the referral is for confirming or establishing a suspected or unknown diagnosis, (2) the referral is for the purpose of treatment after a diagnosis has been made, and (3) the referral is for obtaining a second opinion to review an established diagnosis or treatment recommendation.

Suggesting referral to a patient may be met with ambivalence and anxiety. Concerns about the seriousness of the problem, financial issues, time commitments, and having to estab-

lish a new relationship with another health care provider may be sources of resistance. The practitioner may feel pressure to do something before a diagnosis is established, and this may lead to ineffective and inappropriate treatment.

Special Circumstances in Assessment of Orofacial Pain Patients

OFP DISORDERS POSSIBLY CONFUSED WITH TOOTHACHE

Patients who choose to consult a dentist regarding a pain complaint do so because they believe it may be a tooth-related problem. Several OFP disorders have characteristics that may be confused with those of a toothache (Table 11-11). This confusion may occur because of (1) the location of the pain, (2) the quality of pain that suggests an inflammatory process, or (3) increased pain associated with stimulation of the teeth or surrounding tissues.

TABLE 11-11 Orofacial Disorders That May Be Confused with Toothache

Trigeminal neuralgia
Trigeminal neuropathy (due to trauma or tumor invasion of nerves)
Atypical facial pain and atypical odontalgia
Cluster headache
Acute and chronic maxillary sinusitis
Myofascial pain of masticatory muscles

OFP SYMPTOMS INDICATING SERIOUS DISEASE

Presenting signs or symptoms may suggest the possibility of a serious or life-threatening disorder and indicate an urgent need to establish a diagnosis. These conditions may warrant referral as part of a thorough and timely evaluation (Table 11-12).[124]

HEADACHE AND OFP SYMPTOMS ASSOCIATED WITH SYSTEMIC DISEASE

For most of the systemic diseases that manifest in the oral cavity, there is little information on the frequency with which signs and symptoms identified in the oral cavity lead to the recognition and diagnosis of systemic disease. Table 11-13 lists systemic diseases that have been associated with headache and OFP.[125–133] The literature in this area is primarily case reporting and is a poor guide to the likelihood of finding evidence that implicates a previously undiagnosed systemic disease process as the cause of a patient's unexplained facial OFP. Hyperparathyroidism and metastatic disease will eventually produce radiologic findings that lead to a diagnosis. In other situations, physical signs or laboratory evidence will direct the diagnostic process, but in the early stage of disease, pain (with or without altered sensation) may be the first indication of the disorder.

Diseases such as diabetes mellitus (which occurs with some frequency in the population) will often be found, but evidence associating the systemic disease and the oral symptoms may be harder to find. Clinical investigation of the majority of patients referred after initial evaluation by dentists and physicians for an unsolved oral complaint only rarely detects undiagnosed systemic disease. More often, abnormal blood values such as glucose or iron levels have been noted at earlier examinations. Treating the abnormality, does not always eliminate the oral symptoms. Alternatively, both patient and physician are aware of the presence of the systemic disease, but the methods used to control it have been inadequate. Referral consultations for unexplained oral complaints may thus result in recommendations for additional treatment of systemic disease noted at the time of consultation. In many cases, however, these conditions are not specifically related to the oral complaint.

Despite the time and money invested in extensive searches for systemic disease that only rarely find a possible cause of unexplained oral symptoms, such searches are sometimes justified. Unexplained chronic oral symptoms generate considerable anxiety in addition to the discomfort experienced by the patient, and a "leave no stone unturned" approach often seems necessary to allay these anxieties. Patients with problems sometimes demand a continued bat-

TABLE 11-12 Orofacial Pain Symptoms That Indicate Serious Disease

Orofacial Pain Symptom	Disease Indicated
Pain at the angle of the mandible, brought on by exertion, relieved by rest	Cardiac ischemia
New onset; localized progressive headache; superficial temporal artery swelling, tenderness, and lack of pulse; severe throbbing temporal pain; transient visual abnormalities; systemic symptoms of fever, weight loss, anorexia, malaise, myalgia, chills, sweating	Temporal arteritis
New onset of headache in adult life; increasing severe headache, nausea, and vomiting not explained by systemic illness or migraine; nocturnal occurrence; precipitated or increased by changes in posture; confusion, seizures, or weakness; any abnormal neurologic sign	Intracranial tumor
Earache, trismus, altered sensation in the mandibular branch distribution	Carcinoma of the infratemporal fossa
Trigeminal neuralgia in a person less than 50 years of age	Multiple sclerosis
Pain associated with altered sensation confirmed by physical examination	Neurogenic disorder; tumor invasion of nerve

TABLE 11-13 Systemic Diseases Associated with Headache and Orofacial Pain

Paget's disease
Metastatic disease
Hyperthyroidism
Multiple myeloma
Hyperparathyroidism
Vitamin B deficiencies
Systemic lupus erythematosus
Vincristine therapy for cancer
Folic acid and iron deficiency anemias

tery of sophisticated studies. In these circumstances, the clinician's judgment is needed to prevent the unnecessary repetition of tests and to advise the patient on the likelihood of a particular procedure providing additional useful diagnostic information.

ABSENCE OF A CONVINCING PHYSICAL EXPLANATION FOR SYMPTOMS

Patients who have no convincing physical explanation for their symptoms are the most difficult patients for the practitioner. The resultant problems are not restricted to oral medicine, and all who practice medicine and dentistry usually become aware of them early in their careers. Such patients are seen with greater frequency in specialty practices, simply because unresolved problems commonly lead to a referral for further diagnostic testing. For residents in specialty training, it is often a discovery that a considerable number of patients will not be concerned with clearly defined pathologic states that are amenable to treatment. Patients with unexplained oral sensory abnormalities still require management and some form of treatment even when a thorough diagnostic search fails to find an explanation.

RESPONSES TO UNEXPLAINED SYMPTOMS

The assumption underlying all diagnostic procedures is that an explanation will be found for the patient's complaint of pain. When extensive and reasonably adequate diagnostic investigations fail to find such an explanation, the initial response by the patient and doctor is that further testing to probe for more unusual conditions is needed. When this approach fails, the doctor may begin to assume that the symptoms are not real and that they represent exaggeration for some secondary gain or represent a psychiatric abnormality. Alternatively, the doctor may judge that a borderline abnormality found by palpation or by diagnostic imaging might be more serious than was first considered and might represent evidence of a lesion. Both of these responses on the doctor's part may be exaggerated, and they represent two pitfalls that may complicate the diagnostic and treatment process. First, concluding that symptoms are evidence of a psychiatric abnormality may deny the patient the opportunity for further diagnostic testing that may provide an explanation and solution to the unusual symptoms. Second, performing surgical treatment (even when there are only minimal physical findings) risks complications from the surgical procedure. While all clinicians are vulnerable to these errors, awareness of these pitfalls does help prevent such extremes of response on the doctor's part.

Patients may respond to the lack of an adequate explanation and treatment by requesting further tests or consultation or by independently seeking further consultation. Considering the wide variety of training and traditions that exist in the health professions, it is not difficult to appreciate that a patient will find a practitioner who will provide some treatment that the patient feels might alleviate the symptoms. Multiple consultation and heavy use of surgical services are characteristic features of patients with chronic disorders, especially among those whose symptoms remain unexplained.[134–138] In three separate studies, OFP patients averaged 5, 6, and 7.5 physician/dentist consultations.[72,139,140]

ORAL SYMPTOMS OUT OF PROPORTION TO RECOGNIZED ORAL LESIONS

Patients with unexplained oral symptoms do not always present completely free of dental, periodontal, and mucosal lesions that might be considered possible causes for the unusual symptoms. The evaluation of these patients commonly involves decisions as to whether a degenerating pulp, a coarsely fissured tongue, or muscle tension, for example, may explain complaints of chronic pain or a burning and painful tongue. When possible, treatment of the abnormality by root canal therapy, increased oral and tongue hygiene, or administration of muscle relaxants (in the situations just described) may resolve the question. However, when symptoms persist in the face of apparently adequate treatment, the clinician must decide whether the patient's symptoms possibly arise from another cause or whether they represent an exaggerated response to the particular oral abnormality that has been found and presumably adequately treated.

Among patients with unexplained oral symptoms, there is a group of patients whose salient features are the atypical or exaggerated response to the pain focus and (perhaps) the length of time their symptoms have persisted. It is important to identify the patient whose problem appears to be an inability to cope with minor oral sensory abnormality and who reacts to chronic low-grade pain in the same manner as he or she reacts to pain of greater intensity. Although this identification must be made cautiously and must be reviewed from time to time as treatment progresses, it does help focus treatment on the behavioral component of the patient's pain problem and help reduce continued and unnecessary diagnostic searches.

PSYCHOLOGICAL AND EMOTIONAL FACTORS

Clues that a patient may be reacting in an unusual fashion to abnormal sensory stimuli of low intensity can come from a variety of inquiries during the diagnostic interview. Patients may reveal evidence of a thought disorder during the inter-

view. Patients may reveal the inability to provide clear and consistent statements about symptoms or events that can be checked with reasonable certainty. Confusion between symptoms and an emotionally charged event or personal relationship; the use of bizarre, mechanical, or animalistic explanations for oral symptoms; and the patient's inability to separate him- or herself from real or imaginary objects or people indicate a need for further psychological evaluation. The dentist also will recognize those who express marked paranoia (eg, the pain that is due to an object purposely left behind by the surgeon, who is acting as the agent of God or any enemy of the patient).

None of these phenomena alone substantiates a diagnosis of mental disease. Specific diagnoses (such as schizophrenia, paranoia, and depression) made by the dentist on this basis are unjustified, but the dentist who becomes aware of compromised mental ability in his or her patient should consider the likelihood that abnormal psychological factors may be complicating the diagnostic situation. Such mental confusion may involve organic or functional mental disease that will require further consultation and assessment.

Mental disease, mental retardation, and the inability to conform to society do not produce oral symptoms, but they may affect the individual's ability to handle sensory abnormality. Conversely, pain and other abnormal oral sensations also are experienced by mentally ill persons in response to physical causes, and the clinician must always be on guard against discounting oral symptoms in mentally ill individuals in favor of a psychological explanation without thorough examination of the patient. Table 11-14 lists the IASP classification categories of "pain of psychological origin in the head, face, and neck."[1]

The DSM-IV[84] includes the classification entitled "pain disorder" within a larger category of "somatoform disorders." Somatoform disorders are characterized by the presence of physical symptoms that suggest a general medical condition but that are not fully explained by the medical condition, the direct effects of a substance, or another mental disorder. A pain disorder is characterized by "pain as the predominant focus of clinical attention where psychological factors are judged to have an important role in its onset, severity, exacerbation, or maintenance".

The majority of patients for whom emotional factors obviously complicate their oral symptoms do not have diagnosable mental disease although they may often be referred to as "crazy." Periods of increased emotional stress, whether brought about by interpersonal conflicts, external pressure from work or family, or an individual's own physical and personal drives, are normal for everyone, but such episodes also frequently reduce pain tolerance and the ability to handle chronic low-grade sensory abnormality. To the observer, the influence of the patient's emotions on the oral symptoms may at times be quite evident; for the patient, the interaction of emotional distress and physical disease may be impossible to manage, and he or she may be unable to control either aspect without assistance from the clinician. The following factors are clues that may provide insight into complicating emotional factors:

1. *The setting of the story.* The time of onset of the symptoms may have occurred in a period of increased personal, family, or work stress.
2. *A history of extensive medical/dental treatment.* Unusually extensive and (perhaps) multiple surgical procedures and the use of many medications despite minimal signs of "disease" that others tolerate as part of life indicate "increased help-seeking behavior" that may be maladaptive.[141]
3. *The "naive" or medically inexperienced patient.* Patients who have been free of oral disease until adulthood and who then need dental procedures may respond with excessive anxiety.[142] Paradoxically, those who have suffered painful traumatic and surgical episodes in childhood and have learned excessively apprehensive or other maladaptive responses within their families[143] may also become intolerant of the discomfort associated with dental procedures.
4. *The presence of a psychiatric illness or personality disorder.* An association exists between chronic pain and psychiatric illness.[144,145] However, this does not confirm an etiologic relationship; rather, it is important to

TABLE 11-14 Classification of Pain of Psychological Origin*

Pain of Psychological Origin Classification	Definition
Muscle Tension	Virtually continuous pain in any part of the body due to sustained muscle contraction and provoked by persistent overuse of particular muscles
Delusional or hallucinatory	Pain of psychological origin and attributed by the patient to a specific delusional cause
Hysterical, conversion, or hypochondriacal	Pain specifically attributable to the thought process, emotional state, or personality of the patient in the absence of an organic or delusional cause or tension mechanism
Associated with depression	Pain occurring in the course of a depressive illness usually not preceding the depression and not attributable to any other cause. (Note: not to be confused with depression that commonly occurs with chronic pain arising from physical reasons)

Reproduced with permission from Merskey H, Bogduk N.[1]

*International Association for the Study of Pain (IASP) classification.

appreciate that psychiatric illness requires concomitant treatment to effectively treat the OFP.

5. *Normal oral structures mistakenly identified as physical disease.* Under the stress of the death of a friend or family member or the discovery of life-threatening disease in a close relative or friend, normal structures or sensations may be thought of as potential signs of disease.
6. *Disrupted oral functions.* The mouth serves as a means to obtain food, a modulator for producing speech, and a part of facial expression in interpersonal communication; it also features prominently in sexual encounters. It is not surprising that a limitation of oral function due to oral sensory abnormality can lead to a strong emotional reaction.
7. *Imagined or symbolic functions traditionally assigned to the mouth that may be threatened.* Unsupported by facts of physiology and anatomy, these functions of the mouth feature prominently in our language and thoughts and may be perceived by the patient as being threatened.

The extent to which these traditional images exist in the thoughts of patients with oral disease is largely undocumented and could probably be revealed only by psychoanalysis. However, comments patients make in regard to their oral symptoms during regular diagnostic interviews suggest that such symbolism is a common accompaniment of oral disease. It is important that the clinician recognize these psychological interactions because it may allow him or her to distinguish complaints that are essentially psychological in nature from those that are more directly related to altered physiologic states; the treatment of one is quite different from the treatment of the other, and simultaneous treatment of both problems may be needed. It is an error to consider the patient who uses symbolic images in relating oral problems to be necessarily psychologically abnormal even when the images appear to be somewhat bizarre and overly graphic.[146] It is likely that oral symbolism is normally well developed in most minds, and concern about oral pain and discomfort simply allows patients to be somewhat less reserved about expressing their thoughts than they might usually be. The following metaphors are examples: the "mouthpiece of the mind" (a source of pleasant, virtuous, complimentary, and encouraging statements as well as smiles, laughs, and blessings, versus an invective tight-lipped mouth); an "organ of perception" (the ability to distinguish pleasurable from noxious foods and by extension, pleasurable from unhappy aspects of life); and a "source of pleasure" (the mouth can provide kisses or caresses or can mark an aggressive hostile personality).

If the clinician suspects a psychological cause for OFP, it is important to keep the following in mind:

1. However sophisticated the diagnostic procedures used, no diagnosis is final, and time may often reveal a previously unrecognized organic problem underlying the patient's symptoms.
2. The diagnostic procedures used should be as exhaustive as possible, even in the presence of major psychological dysfunction.
3. Psychological and psychogenic pains cannot be clearly distinguished from pain that has an obvious organic cause; psychological factors are a component of all painful experiences.
4. Pain associated with overwhelming psychological dysfunction (psychogenic pain) is as real to the patient as is pain from an obvious organic cause and cannot be dismissed as something that is "just in the patient's head."
5. A diagnosis of psychological pain should be confirmed by psychiatric evaluation of the patient.

Importance of Follow-up and Repeated Examination and Testing: Of prime importance in the management of patients with unexplained oral symptoms is the recognition that an identification of the cause of the symptoms may come only with time. Several studies of chronic oral sensory complaints have shown that with time, as many as one-half of patients with unexplained OFP were found to have specific pathologic diagnoses that explained their symptoms (provided that repeated examinations and diagnostic tests were continued beyond the initial period of consultation).[136,137]

The success of referral clinics in managing problems of this type derives partly from a program of continued surveillance of the patient by a coordinated group of consultants[137,147] and partly from the availability of sophisticated diagnostic equipment. With time, small lesions such as tumors in the nasopharynx, parotid gland, infratemporal fossa, and cranium that can impinge on oral sensory and motor nerves increase in size and become apparent through the development of other abnormalities. Systemic neurologic diseases such as multiple sclerosis[148] develop from a prodromal stage, in which only unusual oral symptoms are present, to a stage in which a variety of tests will reveal the presence of disease and explain the patient's oral symptoms. The literature contains numerous references to patients whose oral symptoms remained unexplained for varied periods of time until further growth of a tumor revealed the focus of the patient's symptoms.[125,149–159] Included among these reports are many descriptions of tumors of the parotid gland, infratemporal space, and cranial cavity that initially mimicked the symptoms of a TMD. Such reports emphasize the need for continuous awareness of such possibilities.[158] Newer imaging techniques may reveal such lesions and are important tools in the management of undiagnosed chronic OFP.[152,160–162]

▼ DIAGNOSIS AND MANAGEMENT OF SPECIFIC OROFACIAL PAIN DISORDERS

Facial Neuralgias

The classic neuralgias that affect the craniofacial region are a unique group of neurologic disorders involving the cranial

nerves and are characterized by (a) brief episodes of shooting, often electric shock–like pain along the course of the affected nerve branch; (b) trigger zones on the skin or mucosa that precipitate painful attacks when touched; and (c) pain-free periods between attacks and refractory periods immediately after an attack, during which a new episode cannot be triggered. These clinical characteristics differ from neuropathic pain, which tends to be constant and has a burning quality without the presence of trigger zones. Neuropathic pain most often results from disorders that involve the spinal nerves whereas involvement of the cranial nerves may result in either chronic neuropathic pain or the classic brief episodes of shooting pain. Whether a lesion involving a cranial nerve causes constant neuropathic pain or brief episodes of shooting pain depends on both the nature of the underlying disorder and the position of the lesion along the course of the nerve. For example, tumors involving the trigeminal nerve between the pontine angle in the posterior cranial fossa and the ganglion in the middle cranial fossa will usually result in the lacinating pain of trigeminal neuralgia whereas more peripheral lesions will usually result in neuropathic pain. The major craniofacial neuralgias include trigeminal neuralgia, glossopharyngeal neuralgia, and occipital neuralgia. Geniculate neuralgia involving the sensory portion of CN VII is a similar but rare disorder. Postherpetic neuralgia and post-traumatic neuralgia are common causes of neuropathic pain.

TRIGEMINAL NEURALGIA

Trigeminal neuralgia (TN), also called tic douloureux, is the most common of the cranial neuralgias and chiefly affects individuals older than 50 years of age. When younger individuals are involved, suspicion of a detectable underlying lesion such as a tumor, an aneurysm, or multiple sclerosis must be increased.

Etiology and Pathogenesis. The cause of the majority of cases of TN remains controversial, but approximately 10% of cases have detectable underlying pathology such as a tumor of the cerebellar pontine angle, a demyelinating plaque of multiple sclerosis, or a vascular malformation. The most frequent tumor is a meningioma of the posterior cranial fossa. The remainder of cases of TN are classified as idiopathic. Several theories exist regarding the etiology of TN.

The most widely accepted theory is that a majority of cases of TN are caused by an atherosclerotic blood vessel (usually the superior cerebellar artery) pressing on and grooving the root of the trigeminal nerve. This pressure results in focal demyelinization and hyperexcitability of nerve fibers, which will then fire in response to light touch, resulting in brief episodes of intense pain.[163]

Evidence for this theory includes the observation that neurosurgery that removes the pressure of the vessel from the nerve root by use of a microvascular decompression procedure eliminates the pain in a majority of cases. In a recent study of 1,185 patients who had microvascular decompression surgery for TN that did not respond to drug therapy, 70% of the patients were pain free 10 years after the surgery.[164] Additional evidence for this theory was obtained from a study using tomographic magnetic resonance imaging (MRI), which showed that contact between a blood vessel and the trigeminal nerve root was much greater on the affected side.[165]

Evidence against this theory includes the finding by neurosurgeons that manipulation of the area of the nerve root may eliminate the painful episodes even when an atherosclerotic vessel is not pressing on the nerve root. Other investigators believe that a major factor in the etiology of TN is a degeneration of the ganglion rather than the nerve root.[166]

Clinical Features. The majority of patients with TN present with characteristic clinical features, which include episodes of intense shooting stabbing pain that lasts for a few seconds and then completely disappears. The pain characteristically has an electric shock–like quality and is unilateral except in a small percentage of cases. The maxillary branch is the branch that is most commonly affected, followed by the mandibular branch and (rarely) the ophthalmic branch. Involvement of more than one branch occurs in some cases.

Pain in TN is precipitated by light touch on a "trigger zone" present on the skin or mucosa within the distribution of the involved nerve branch. Common sites for trigger zones include the nasolabial fold and the corner of the lip. Shaving, showering, eating, speaking, or even exposure to wind can trigger a painful episode, and patients often protect the trigger zone with their hand or an article of clothing. Intraoral trigger zones can confuse the diagnosis by suggesting a dental disorder, and TN patients often first consult a dentist for evaluation. The stabbing pain can mimic the pain of a cracked tooth, but the two disorders can be distinguished by determining whether placing food in the mouth without chewing or whether gently touching the soft tissue around the trigger zone will precipitate pain. TN pain will be triggered by touching the soft tissue whereas pressure on the tooth is required to cause pain from a cracked tooth. Just after an attack, there is a refractory period when touching the trigger zone will not precipitate pain. The number of attacks may vary from one or two per day to several per minute. Patients with severe TN may be severely disabled by attacks that are triggered by speaking or other mouth movements.

Diagnosis. The diagnosis of TN is usually based on the history of shooting pain along a branch of the trigeminal nerve, precipitated by touching a trigger zone, and possibly examination that demonstrates the shooting pain. A routine cranial nerve examination will be normal in patients with idiopathic TN, but sensory and/or motor changes may be evident in patients with underlying tumors or other CNS pathology. Local anesthetic blocks, which temporarily eliminate the trigger zone, may also be helpful in diagnosis. Since approximately 10% of TN cases are caused by detectable underlying pathology, enhanced MRI of the brain is indicated to rule out tumors, multiple sclerosis, and vascular malformations.

Management. Initial therapy for TN should consist of trials of drugs that are effective in eliminating the painful attacks. Anticonvulsant drugs are most frequently used and are most effective. Carbamazepine is the most commonly used drug and is an effective therapy for greater than 85% of newly diagnosed cases of TN. The drug is administered in slowly increasing doses until pain relief has been achieved. Skin reactions, including generalized erythema multiforme, are serious side effects. Patients receiving carbamazepine must have periodic hematologic laboratory evaluations because serious life-threatening blood dyscrasias occur in rare cases. Monitoring of hepatic and renal function is also recommended. Patients who do not respond to carbamazepine alone may obtain relief from baclofen or by combining carbamazepine with baclofen.[167] Gabapentin, a newer anticonvulsant that has fewer serious side effects than carbamazepine, is effective in some patients but does not appear to be as reliable as carbamazepine. Other drugs that are effective for some patients include phenytoin, lamotrigine, and pimozide.[168] Since TN may have temporary or permanent spontaneous remissions, drug therapy should be slowly withdrawn if a patient remains pain free for 3 months.

Clinicians treating TN must be aware that drug therapy often becomes less effective over time and that progressively higher doses may be required for pain control. In cases in which drug therapy is ineffective or in which the patient is unable to tolerate the side effects of drugs after trials of several agents, surgical therapy is indicated. A number of surgical procedures that result in temporary or permanent remission of the painful attacks have been described. These include procedures performed on the peripheral portion of the nerve, where it exits the jaw; at the gasserian ganglion; and on the brainstem, at the posterior cranial fossa. Peripheral surgery includes cryosurgery on the trigeminal nerve branch that triggers the painful attacks. This procedure is most frequently performed at the mental nerve for cases involving the third division and at the infraorbital nerve for cases involving the second division. The potential effectiveness of this procedure can be evaluated prior to surgery by determining whether a long-acting local anesthetic eliminates the pain during the duration of anesthesia. This procedure is usually effective for 12 to 18 months, at which time it must be repeated or another form of therapy must be instituted.

The most commonly performed procedure at the level of the gasserian ganglion is percutaneous radiofrequency thermocoagulation[169] although some clinicians continue to advocate glycerol block at the ganglion[170] or compression of the ganglion by balloon microcompression.[171] An infrequent but severe surgical complication is anesthesia dolorosa, which is numbness combined with severe intractable pain. The most extensively studied surgical procedure is microvascular decompression of the nerve root at the brainstem. In a report of 1,185 patients who were observed for 1 to 6 years, 70% of the patients experienced long-term relief of symptoms.[172] It should be noted that 30% of the patients experienced a recurrence of symptoms and required a second procedure or alternative therapy. Complications were rare but included stroke, facial numbness, and facial weakness.

In summary, therapy for TN presently includes a variety of both medical and surgical approaches, each of which is effective for some patients. Drug therapy including trials of several drugs or combinations of drugs should be attempted before surgery is recommended. When surgery is necessary, the patient should be carefully counseled regarding the advantages and disadvantages of the available surgical procedures. Clinicians should also remember that since spontaneous remissions are a feature of TN, procedures resulting in temporary relief might be all that is necessary for some patients.

GLOSSOPHARYNGEAL NEURALGIA

Glossopharyngeal neuralgia is a rare condition that is associated with paroxysmal pain that is similar to, though less intense than, the pain of TN. The location of the trigger zone and pain sensation follows the distribution of the glossopharyngeal nerve, namely, the pharynx, posterior tongue, ear, and infra-auricular retromandibular area. Pain is triggered by stimulating the pharyngeal mucosa during chewing, talking, and swallowing. The pain can be easily confused with that of geniculate neuralgia (because of the common ear symptoms) or with that of TMDs (because of pain following jaw movement).

Glossopharyngeal neuralgia may occur with TN, and when this occurs, a search for a common central lesion is essential. Glossopharyngeal neuralgia also may be associated with vagal symptoms, such as syncope and arrhythmia, owing to the close anatomic proximity of the two nerves. The application of a topical anesthetic to the pharyngeal mucosa eliminates glossopharyngeal nerve pain and can aid in distinguishing it from the pain of other neuralgias. The most common causes of glossopharyngeal neuralgia are intracranial or extracranial tumors and vascular abnormalities that compress CN IX. Treatment is similar to that for TN, with a good response to carbamazepine and baclofen. Refractory cases are treated surgically by intracranial or extracranial section of CN IX, microvascular decompression in the posterior cranial fossa, or (more recently) by percutaneous radiofrequency thermocoagulation of the nerve at the jugular foramen.[173]

NERVOUS INTERMEDIUS (GENICULATE) NEURALGIA

Nervous intermedius (geniculate) neuralgia is an uncommon paroxysmal neuralgia of CN VII, characterized by pain in the ear and (less frequently) the anterior tongue or soft palate. The location of pain matches the sensory distribution of this nerve (ie, the external auditory canal and a small area on the soft palate and the posterior auricular region). Pain may be provoked by the stimulation of trigger zones within the ipsilateral distribution of the nerve. The pain is not as sharp or intense as in TN, and there is often some degree of facial paralysis, indicating the simultaneous involvement of the motor root. Geniculate neuralgia commonly results from herpes zoster of the geniculate ganglion and nervus intermedius of CN VII,[174] a condition referred to as Ramsay Hunt syndrome.[175] Viral vesicles may be observed in the ear canal or on

the tympanic membrane. The symptoms result from inflammatory neural degeneration, and a short course (2 to 3 weeks) of high-dose steroid therapy is beneficial.[174] Acyclovir significantly reduces the duration of the pain. Patients with geniculate neuralgia are also treated with carbamazepine and antidepressants. Patients who do not respond to these medications may undergo surgery to section the nervus intermedius.

OCCIPITAL NEURALGIA

Occipital neuralgia is a rare neuralgia in the distribution of the sensory branches of the cervical plexus (most commonly unilateral in the neck and occipital region). The most common causes (in descending order of frequency) are trauma, neoplasms, infections, and aneurysms involving the affected nerve(s). Palpation below the superior nuchal line may reveal an exquisitely tender spot. Treatment has included corticosteroids, neurolysis, avulsion, and blocking the nerve with a local anesthetic.[176]

POSTHERPETIC NEURALGIA

Etiology and Pathogenesis. Herpes zoster (shingles), described in detail in Chapter 2 is caused by the reactivation of latent varicella-zoster virus infection that results in both pain and vesicular lesions along the course of the affected nerve. Approximately 15 to 20% of cases of herpes zoster involve the trigeminal nerve although the majority of these cases affect the ophthalmic division of the fifth nerve, resulting in pain and lesions in the region of the eyes and forehead. Herpes zoster of the maxillary and mandibular divisions is a cause of facial and oral pain as well as of lesions. In a majority of cases, the pain of herpes zoster resolves within a month after the lesions heal. Pain that persists longer than a month is classified as postherpetic neuralgia (PHN) although some authors do not make the diagnosis of PHN until the pain has persisted for longer than 3 or even 6 months.[177] PHN may occur at any age, but the major risk factor is increasing age. Few individuals younger than 30 years of age experience PHN whereas more than 25% of individuals older than 55 years of age and two-thirds of patients older than over 70 years of age will suffer from PHN after an episode of herpes zoster.[178] Elderly patients also have an increased risk of experiencing severe pain for an extended period of time.[179] The pain and numbness of PHN results from a combination of both central and peripheral mechanisms. The varicella-zoster virus injures the peripheral nerve by demyelination, wallerian degeneration, and sclerosis,[180] but changes in the CNS, including atrophy of dorsal horn cells in the spinal cord, have also been associated with PHN.[181] This combination of peripheral and central injury results in the spontaneous discharge of neurons and an exaggerated painful response to nonpainful stimuli.[180]

Clinical Manifestations. Patients with PHN experience persistent pain, paresthesia, hyperesthesia, and allodynia months to years after the zoster lesions have healed. The pain is often accompanied by a sensory deficit, and there is a correlation between the degree of sensory deficit and the severity of pain.[182]

Management. Many treatment options are available for the management of PHN, and the method chosen should depend on the severity of the symptoms as well as the general medical status of the patient. Treatment includes topical and systemic, drug therapy and surgery.

Topical therapy includes the use of topical anesthetic agents, such as lidocaine, or analgesics, particularly capsaicin. Lidocaine used either topically or injected gives short-term relief from severe pain.[183] Combinations of topical anesthetics such as EMLA Cream (AstraZeneca) have also been reported as helpful.[184] Capsaicin, an extract of hot chili peppers that depletes the neurotransmitter substance P when used topically, has been shown to be helpful in reducing the pain of PHN, but the side effect of a burning sensation at the site of application limits its usefulness for many patients.

The use of tricyclic antidepressants such as amitriptyline, nortriptyline, doxepin, and desiprimine is a well-established method of reducing the chronic burning pain that is characteristic of PHN.[185–188] Because a significant number of elderly patients cannot tolerate the sedative or cardiovascular side effects associated with tricyclic antidepressants, the use of other drugs, particularly gabapentin, has been advocated. In one controlled clinical trial, the use of gabapentin reduced pain by more than 30% and also improved sleep and overall quality of life.[189] Patients who undergo episodes of shooting pain may experience relief through the use of anticonvulsant drugs, such as carbamazepine or phenytoin.[188]

When medical therapy has been ineffective in managing intractable pain, nerve blocks or surgery at the level of the peripheral nerve or dorsal root have been effective for some patients. The best therapy for PHN is prevention. There is evidence that the use of antiviral drugs, particularly famciclovir, along with a short course of systemic corticosteroids during the acute phase of the disease may decrease the incidence and severity of PHN.[190] Although investigators agree that the use of antivirals and corticosteroids decreases acute pain and accelerates the healing of lesions, further controlled trials are necessary before the long-term benefits of using antivirals and corticosteroids are known.[191] The use of tricyclic antidepressants during the acute phase of herpes zoster has been advocated as an effective method of decreasing PHN.[192]

POST-TRAUMATIC NEUROPATHIC PAIN

Etiology and Pathogenesis. Trigeminal nerve injuries may result from facial trauma or from surgical procedures, such as the removal of impacted third molars, the placement of dental implants, the removal of cysts or tumors of the jaws, genioplasties, or osteotomies. In some individuals, nerve injury results only in numbness whereas others experience pain that may be either spontaneous or triggered by a stimulus. The pain associated with nerve injury often has a burning quality due to spontaneous activity in nociceptor C fibers.[193] Minor nerve injuries (classified as neurapraxia) do not result in axonal

degeneration but may cause temporary symptoms of parasthesia for a few hours or days. More serious nerve damage (classified as axonotmesis) results in the degeneration of neural fibers although the nerve trunk remains intact. These injuries cause symptoms for several months but have a good prognosis for recovery after axonal regeneration is complete. Total nerve section (neurotmesis) frequently causes permanent nerve damage, resulting in anesthesia and/or dysesthesia.[194] Central sensitization probably plays a role in the symptoms of neuropathy.

Clinical Manifestations. The pain associated with peripheral nerve injury may be persistent or may occur only in response to a stimulus such as light touch. Patients with nerve damage may experience anesthesia (loss in sensation), paresthesia (a feeling of "pins and needles"), allodynia (pain caused by a stimulus that is normally not painful), or hyperalgesia (an exaggerated response to a mildly painful stimulus).

Management. Treatment of neuropathic pain may be surgical, nonsurgical, or a combination of both, depending on the nature of the injury and the severity of the pain. Systemic corticosteroids are considered helpful in decreasing the incidence and severity of traumatic neuropathies when administered within the first week after a nerve injury. Steroids used after this initial period are of little value. The most frequently used medications for the management of neuropathic pain include tricyclic antidepressants (TCAs) and gabapentin.

TCAs such as amitriptyline, doxepin, and nortriptyline have been extensively studied and widely used to treat neuropathic pain, including traumatic neuropathies of the trigeminal nerve.[195] The TCAs can be used alone; in severe intractable cases, they potentiate the effect of narcotic analgesics. The clinician prescribing TCAs must be aware of potential serious side effects in patients with cardiac arrhythmias or glaucoma and must be able to help the patient manage common side effects that include drowsiness, weight gain from increased appetite, and dry mouth.

Gabapentin, an anticonvulsant drug approved for the treatment of epilepsy, has been used with increasing frequency to treat a variety of neuropathic pain syndromes, including diabetic neuropathy and PHN. The low incidence of serious side effects has encouraged widespread use of this drug. A controlled clinical trial that compared the effectiveness of gabapentin with that of the TCA amitriptyline demonstrated that both were equally effective in controlling neuropathic pain associated with diabetic neuropathy.[196,197]

Topical capsaicin may also be effective in controlling pain and is especially useful for patients who are unable to tolerate the side effects of systemic therapy.

Complex Regional Pain Syndrome 1 (Reflex Sympathetic Dystrophy)

The terms "complex regional pain syndrome type 1" (CRPS-1) and "reflex sympathetic dystrophy" (RSD) are used to describe a poorly understood syndrome that consists of localized pain, motor and sweat abnormalities, and trophic changes in the soft tissues of the muscles and skin.

ETIOLOGY AND PATHOGENESIS

The constellation of signs and symptoms associated with CRPS is believed to result from changes after trauma that couples sensory nerve fibers to sympathetic stimuli. Evidence for the existence of CRPS includes studies that show that surgical or drug-induced blockades of the sympathetic nervous system relieve the symptoms. In a new taxonomy included in the classification of chronic pain, CRPS-1 is used in place of RSD, and CRPS-2 replaces causalgia, which is a pain syndrome resulting from a major nerve injury. RSD has rarely been described as involving the trigeminal nerve distribution, and the role of the sympathetic nervous system in chronic facial pain is unknown. One study of chronic facial pain patients who also had evidence of autonomic dysfunction described a subset of patients who improved after a stellate ganglion block, suggesting a possible role for the sympathetic nervous system.[198] There are also case reports of facial pain resolving after sympathectomy.[199]

CLINICAL MANIFESTATIONS

The most constant symptom of CRPS is spontaneous chronic burning pain and tenderness, frequently accompanied by motor dysfunction, sweating, and cutaneous atrophy. The involved skin may also be edematous and erythematous as a result of changes in blood flow, and the underlying bone is commonly demineralized. Allodynia and hyperesthesia are common symptoms, and movement exacerbates the pain. This syndrome most commonly involves the extremities distal to an injury. The existence of this disorder in the head and facial region is controversial.

TREATMENT

The recommended therapy for CRPS involves a multidisciplinary approach that includes physical therapy, nerve blocks, and drug therapy. Blockades of regional sympathetic ganglia or regional intravenous blockades with guanethidine, reserpine, or phenoxybenzamine combined with a local anesthetic have been reported as successful[200] and are used in anesthesia pain clinics. Bisphosphonates such as alendronate or pamidronate have decreased pain in some RSD patients when used intravenously. It is unclear whether these drugs are helpful because of their effect on bone or because of anti-inflammatory properties.[201]

Atypical Odontalgia (Atypical Facial Pain)

A classification that includes the diagnoses of atypical odontalgia (AO) and atypical facial pain (AFP) is controversial, and many workers in the field of facial pain believe that these terms should be discarded because they are often used either as catchalls to denote patients who have not been adequately evaluated or because they imply that the pain is purely psychological in origin. Some classification systems, including the IHS system, use the term "facial pain not fulfilling other criteria" to describe patients in this category. The disputed terms are still commonly used in clinical practice, however, since there exists a group of individuals who (1) have a chronic facial pain

syndrome with characteristic clinical features, (2) have been thoroughly investigated, and (3) do not fall into any other diagnostic categories. The term "atypical odontalgia" is used in this context when the pain is confined to the teeth or gingivae whereas the term "atypical facial pain" is used when other parts of the face are involved.

ETIOLOGY AND PATHOGENESIS

There are several theories regarding the etiology of AO and AFP. One theory considers AO and AFP to be a form of de-afferentation or phantom tooth pain. This theory is supported by the high percentage of patients with these disorders who report that the symptoms began after a dental procedure such as endodontic therapy or an extraction. Others have theorized that AO is a form of vascular, neuropathic, or sympathetically maintained pain. Other studies support the concept that at least some of the patients in this category have a strong psychogenic component to their symptoms and that depressive, somatization, and conversion disorders have been described as major factors in some patients. It is frequently difficult to accurately study the psychological aspects of a chronic pain syndrome since anxiety and depression are part of the clinical picture of all patients with chronic pain.

There is often strong disagreement between facial pain experts who stress the biologic basis of AO and AFP and others who stress the emotional basis, but the etiology remains unknown at this time. It is likely that there are subgroups of patients who fall into the category of AO and AFP, some of whom have a strong component of de-afferentation pain while others have a psychological basis for similar symptoms. It is also possible that a combination of both neuropathic and psychological mechanisms are important in the etiology of this presently poorly understood facial pain syndrome.

CLINICAL MANIFESTATIONS

The major manifestation of AO and AFP is a constant dull aching pain without an apparent cause that can be detected by examination or laboratory studies. AO occurs most frequently in women in the fourth and fifth decades of life, and most studies report that women make up more than 80% of the patients. The pain is described as a constant dull ache, instead of the brief and severe attacks of pain that are characteristic of TN. There are no trigger zones, and lancinating pains are rare. The patient frequently reports that the onset of pain coincided with a dental procedure such as oral surgery or an endodontic or restorative procedure. Patients also report seeking multiple dental procedures to treat the pain; these procedures may result in temporary relief, but the pain characteristically returns in days or weeks. Other patients will give a history of sinus procedures or of receiving trials of multiple medications, including antibiotics, corticosteroids, decongestants, or anticonvulsant drugs. The pain may remain in one area or may migrate, either spontaneously or after a surgical procedure. Symptoms may remain unilateral, cross the midline in some cases, or involve both the maxilla and mandible.

A thorough history and examination including evaluation of the cranial nerves, oropharynx, and teeth must be performed to rule out dental, neurologic, or nasopharyngeal disease. Examination of the masticatory muscles should also be performed to eliminate pain secondary to undetected muscle dysfunction. Laboratory tests should be carried out when indicated by the history and examination. Patients with AO or AFP have completely normal radiographic and clinical laboratory studies.

MANAGEMENT

Once the diagnosis has been made and other pathologies have been eliminated, it is important that the symptoms are taken seriously and are not dismissed as imaginary. Patients should be counseled regarding the nature of AO and reassured that they do not have an undetected life-threatening disease and that they can be helped without invasive procedures. When indicated, consultation with other specialists such as otolaryngologists, neurologists, or psychiatrists may be helpful. TCAs such as amitriptyline, nortriptyline, and doxepin, given in low to moderate doses, are often effective in reducing or (in some cases) eliminating the pain. Other recommended drugs include gabapentin and clonazepam. Some clinicians report benefit from topical desensitization with capsaicin, topical anesthetics, or topical doxepin.

Burning Mouth Syndrome (Glossodynia)

Burning sensations accompany many inflammatory or ulcerative diseases of the oral mucosa, but the term "burning mouth syndrome" (BMS) is reserved for describing oral burning that has no detectable cause. The burning symptoms in patients with BMS do not follow anatomic pathways, there are no mucosal lesions or known neurologic disorders to explain the symptoms, and there are no characteristic laboratory abnormalities.

ETIOLOGY AND PATHOGENESIS

The cause of BMS remains unknown, but a number of factors have been suspected, including hormonal and allergic disorders, salivary gland hypofunction, chronic low-grade trauma, and psychiatric abnormalities. The increased incidence of BMS in women after menopause has led investigators to suspect an association with hormonal changes, but there is little evidence that women with BMS have more hormonal abnormalities than matched controls who do not have BMS. Studies of estrogen replacement therapy used to treat BMS have yielded mixed results, and few investigators recommend hormone replacement as a primary therapy for BMS in patients who do not require it for other reasons.

Allergic reactions have also been suspected, but there is no evidence to support the hypothesis that BMS is the result of allergic reactions to food, oral hygiene products, or dental materials. A contact allergy can affect the oral mucosa and result in burning sensations, but inflammatory, lichenoid, or ulcerative lesions are present in cases of contact allergy and absent in BMS patients. It was theorized that BMS is related to decreased salivary gland function, but most studies have

shown no clear-cut association between BMS and decreased salivary flow rates.[202] Changes in taste have been reported in over 60% of patients with BMS, and BMS patients have been shown to have different thresholds of taste perception than matched controls.[203] Dysgeusia (particularly an abnormally bitter taste) has been reported by 60% of BMS patients.[204] This association has led to a concept that BMS may be a defect in sensory peripheral neural mechanisms.[202]

BMS has been associated with psychological disorders in many studies. Depression is frequently associated with BMS, and in some studies, close to one-third of BMS patients have significant depression scores although, as with any chronic pain disorder, it is unclear if depression is the cause or the effect of the symptoms.[205–207] It is likely that some cases of BMS have a strong psychological component, but other factors, such as chronic low-grade trauma resulting from parafunctional oral habits (eg, rubbing the tongue across the teeth or pressing it on the palate), are also likely to play a role.

CLINICAL MANIFESTATIONS

Women experience symptoms of BMS seven times more frequently than men.[208] When questioned, 10 to 15% of postmenopausal women are found to have a history of oral burning sensations, and these symptoms are most prevalent 3 to 12 years after menopause.[209] The tongue is the most common site of involvement, but the lips and palate are also frequently involved. The burning can be either intermittent or constant, but eating, drinking, or placing candy or chewing gum in the mouth characteristically relieves the symptoms. This contrasts with the increased oral burning noted during eating that occurs in patients with lesions or neuralgias affecting the oral mucosa. Patients presenting with BMS are often apprehensive and admit to being generally anxious or "high-strung." They may also have symptoms that suggest depression, such as decreased appetite, insomnia, and a loss of interest in daily activities.

Other causes of burning symptoms of the oral mucosa must be eliminated by examination and laboratory studies before the diagnosis of BMS can be made. Patients with unilateral symptoms should have a thorough evaluation of the trigeminal and other cranial nerves to eliminate a neurologic source of pain. A careful clinical examination for oral lesions resulting from candidiasis, lichen planus, or other mucosal diseases should be performed. Patients complaining of a combination of xerostomia and burning should be evaluated for the possibility of a salivary gland disorder, particularly if the mucosa appears to be dry and the patient has difficulty swallowing dry foods without sipping liquids. When indicated, laboratory tests should be carried out to detect undiagnosed diabetic neuropathy, anemia, or deficiencies of iron, folate, or vitamin B_{12}.

TREATMENT

Once the diagnosis of BMS has been made by eliminating the possibility of detectable lesions or underlying medical disorders, the patient should be reassured of the benign nature of the symptoms. Counseling the patient in regard to the nature of BMS is helpful in management, particularly because many patients will have had multiple clinical evaluations without an explanation for the symptoms. Counseling and reassurance may be adequate management for individuals with mild burning sensations, but patients with symptoms that are more severe often require drug therapy. The drug therapies that have been found to be the most helpful are low doses of TCAs, such as amitriptyline and doxepin, or clonazepam (a benzodiazepine derivative). It should be stressed to the patient that these drugs are being used not to manage psychiatric illness, but for their well-documented analgesic effect. Clinicians prescribing these drugs should be familiar with potential serious and annoying side effects.

Burning of the tongue that results from parafunctional oral habits may be relieved with the use of a splint covering the teeth and/or the palate.

Vascular Pain

Pain originating from vascular structures may cause facial pain that can be misdiagnosed and mistaken for other oral disorders, including toothache or TMD. This section discusses the major pain disorders of vascular etiology that have prominent orofacial signs and symptoms.

CRANIAL ARTERITIS

Cranial arteritis (temporal arteritis, giant cell arteritis) is an inflammatory disorder involving the medium-sized branches of the carotid arteries. The temporal artery is the most commonly involved branch. The blood vessel abnormality may be localized to the head and face or may be part of the generalized disease, polymyalgia rheumatica.

Etiology and Pathogenesis. Both cranial arteritis and polymyalgia rheumatica are caused by immune abnormalities that affect cytokines and T lymphocytes, resulting in inflammatory infiltrates in the walls of arteries. This infiltrate is characterized by the formation of multinucleated giant cells. The underlying trigger of the inflammatory response is unknown.

Clinical Manifestations. Cranial arteritis most frequently affects adults above the age of 50 years. Patients have a throbbing headache accompanied by generalized symptoms including fever, malaise, and loss of appetite. Patients with polymyalgia rheumatica will have accompanying joint and muscle pain. Examination of the involved temporal artery reveals a thickened pulsating vessel. Since the mandibular and lingual arteries may be involved, a throbbing pain in the jaw or tongue may be an early sign or even a presenting sign. A serious complication in untreated patients is ischemia of the eye, which may lead to progressive loss of vision or sudden blindness. These visual manifestations may be prevented by early diagnosis and prompt therapy.

Laboratory abnormalities include an elevated erythrocyte sedimentation rate (ESR) and anemia. Abnormal C-reactive protein may also be an important early finding. The most definitive diagnostic test is a biopsy specimen (from the

involved temporal artery) that demonstrates the characteristic inflammatory infiltrate. Since the entire vessel is not involved, an adequate specimen must be taken to detect the changes. A negative biopsy result does not rule out temporal arteritis, and the diagnosis should continue to be considered in patients over 50 years of age who have chronic pounding head or orofacial pain and an elevated ESR.[210]

Treatment. Individuals with cranial arteritis should be treated with systemic corticosteroids as soon as the diagnosis is made. The initial dose ranges between 40 to 60 mg of prednisone per day, and the steroid is tapered once the signs of the disease are controlled. The ESR may be used to help monitor disease status. Patients are maintained on systemic steroids for 1 to 2 years after symptoms resolve. Steroids may be supplemented by adjuvant therapy with immunosuppressive drugs, such as cyclophosphamide, to reduce the complications of long-term corticosteroid therapy. Immediate steroid therapy should be initiated if visual symptoms are present.

CLUSTER HEADACHE

Cluster headache (CH) is a distinct pain syndrome characterized by episodes of severe unilateral head pain occuring chiefly around the eye and accompanied by a number of autonomic signs. The term "cluster" is used because individuals who are susceptible to CH experience multiple headaches per day for 4 to 6 weeks and then may be without pain for months or even years.

Etiology and Pathogenesis. There are several theories regarding the etiology of CH and its characteristic combination of both severe localized pain and autonomic symptoms. Some investigators postulate that a CH attack originates in the hypothalamus, which stimulates both the trigeminal and vascular systems in the brain.[211] Others believe that the pain originates peripherally in the cavernous sinus since sympathetic, parasympathetic, and sensory fibers from the first division of the trigeminal nerve are present and because organic lesions of the cavernous sinus can result in symptoms that resemble CH.[212]

Clinical Manifestations. Eighty percent of patients with CH are men. The attacks are sudden, unilateral, and stabbing, causing patients to pace, cry out, or even strike objects. Some patients exhibit violent behavior during attacks. This contrasts with the behavior of migraine patients, who lie down in a dark room and try to sleep. Individuals with CH frequently describe the pain as a hot metal rod in or around the eye. The symptoms most commonly affect the area supplied by the first division of the trigeminal nerve, but second-division symptoms may also occur, causing patients to consult a dentist to rule out an odontogenic etiology. Unnecessary extractions of maxillary teeth are often performed before a correct diagnosis is made. The severe painful episodes begin without an aura and become excruciating within a few minutes. Each attack lasts from 15 minutes to 2 hours and recurs several times daily. A majority of the painful episodes occur at night, often waking the patient from sleep. The pain is associated with autonomic symptoms, particularly nasal congestion and tearing. Sweating of the face, ptosis, increased salivation, and edema of the eyelid are also common signs. During a cluster period, ingestion of alcohol or use of nitroglycerin will provoke an attack.

Treatment. An acute attack of CH can be aborted by breathing 100% oxygen, and CH patients may keep an oxygen canister at bedside to use at the first sign of an attack. Injection of sumatriptan or sublingual or inhaled ergotamine may also be effective therapy. Several drug protocols are recommended for preventing CH during active periods. Lithium is effective therapy for those who can tolerate the side effects, and patients who are using long-term lithium must be monitored for renal toxicity. Other drugs that are useful for preventing attacks include ergotamine, prophylactic prednisone, and calcium channel blockers. Methysergide is also effective therapy, but pulmonary or cardiac fibrosis are potential side effects, particularly during prolonged use.

CHRONIC PAROXYSMAL HEMICRANIA

Chronic paroxysmal hemicrania (CPH) is believed to be a form of CH that occurs predominantly in women between the ages of 30 to 40 years. The episodes of pain tend be shorter, but attacks of 5 to 20 minutes' duration can occur up to 30 times daily. Initially, episodes of CPH occur with a periodicity similar to that of CH; however, CPH symptoms tend to become chronic over time. CPH responds dramatically to therapy with indomethacin, which stops the attacks within 1 to 2 days. CPH will recur if indomethacin is discontinued.

MIGRAINE

Until recently, headaches were believed to be either vascular or muscular in origin, but studies performed in the past decade have indicated that many patients with frequent or chronic headaches have a mixture of both vascular and muscular pain and that headaches are frequently somewhere on a continuum between being purely vascular and purely muscular. Migraine is the most common of the vascular headaches, which may occasionally also cause pain of the face and jaws. It may be triggered by foods such as nuts, chocolate, and red wine; stress; sleep deprivation; or hunger. Migraine is more common in women.

Etiology and Pathogenesis. The classic theory is that migraine is caused by vasoconstriction of intracranial vessels (which causes the neurologic symptoms), followed by vasodilation (which results in pounding headache). Newer research techniques suggest a series of factors, including the triggering of neurons in the midbrain that activate the trigeminal nerve system in the medulla, resulting in the release of neuropeptides such as substance P. These neurotransmitters activate receptors on the cerebral vessel walls, causing vasodilation and vasoconstriction. There are several major types of migraine: classic, common, basilar, and facial migraine (also referred to as carotidynia).

Clinical Manifestations. Classic migraine starts with a prodromal aura that is usually visual but that may also be sensory or motor. The visual aura that commonly precedes classic migraine includes flashing lights or a localized area of depressed vision (scotoma). Sensitivity to light, hemianesthesia, aphasia, or other neurologic symptoms may also be part of the aura, which commonly lasts from 20 to 30 minutes. The aura is followed by an increasingly severe unilateral throbbing headache that is frequently accompanied by nausea and vomiting. The patient characteristically lies down in a dark room and tries to fall asleep. Headaches characteristically last for hours up to 2 or 3 days.

Common migraine is not preceded by an aura, but patients may experience irritability or other mood changes. The pain of common migraine resembles the pain of classic migraine and is usually unilateral, pounding, and associated with sensitivity to light and noise. Nausea and vomiting are also common.

Basilar migraine is most common in young women. The symptoms are primarily neurologic and include aphasia, temporary blindness, vertigo, confusion, and ataxia. These symptoms may be accompanied by an occipital headache. Facial migraine (carotidynia) causes a throbbing and/or sticking pain in the neck or jaw. The pain is associated with involvement of branches of the carotid artery rather than the cerebral vessels.[213] The symptoms of facial migraine usually begin in individuals who are 30 to 50 years of age. Patients often seek dental consultation, but unlike the pain of a toothache, facial migraine pain is not continuous but lasts minutes to hours and recurs several times per week. Examination of the neck will reveal tenderness of the carotid artery. Face and jaw pain may be the only manifestation of migraine, or it may be an occasional pain in patients who usually experience classic or common migraine.[214]

Treatment. Patients with migraine should be carefully assessed to determine common food triggers. Attempts to minimize reactions to the stress of everyday living by using relaxation techniques may also be helpful to some patients. Drug therapy may be used either prophylactically to prevent attacks in patients who experience frequent headaches or acutely at the first sign of an attack. Drugs that are useful in aborting migraine include ergotamine and sumatriptan, which can be given orally, nasally, rectally or parenterally. These drugs must be used cautiously since they may cause hypertension and other cardiovascular complications. Drugs that are used to prevent migraine include propranolol, verapimil, and TCAs.[215] Methysergide or monoamine oxidase inhibitors such as phenelzine can be used to manage difficult cases that do not respond to safer drugs.

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