

Orofacial Pain

A Guide to Medications and Management

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Chapter 1

The 30 most prevalent chronic painful diseases, disorders, and dysfunctions that occur in the orofacial region

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1.1 Introduction and definitions

Although there are many more than 30 orofacial pain conditions, this chapter focuses on the ones most commonly seen in clinical practice. The distinction between a disease, a disorder, and a dysfunction is somewhat arbitrary: The terms “disorder” and “dysfunction” are used more or less interchangeably to mean an ailment or impaired functioning of a bodily system. The term “disease” implies a pathological condition of a part, organ, or system of an organism, resulting from various causes, such as infection, genetic defect, or environmental stress, and characterized by an identifiable group of signs or symptoms.¹ Regardless of how they are classified, these 30 conditions can be logically clustered into 7 subgroups. A clinician who can learn about these subgroups and distinguish between these 30 conditions will be a long way toward having the expertise required to properly manage patients with chronic orofacial pain. Toward this goal, the chapter begins with several tables that summarize information about the characteristics, appropriate diagnostic tests, age predilection, and known prevalence of these 30 conditions. These tables are accompanied by discussion of the process necessary to render a differential diagnosis for a patient with chronic orofacial pain complaints. Table 1.1 briefly describes the clinical characteristics of the 30 conditions considered in this chapter. Treatment of these 30 conditions is discussed, along with associated conditions, in various other chapters in this book and therefore is not covered here.

1.1.A Nociceptive versus neuropathic pain

When pain persists beyond the time expected for healing to occur, two explanations exist. First, long-standing chronic pain sensations may still be occurring via local disease

inducing pain mediators (e.g., inflammatory cytokines). Second, long-standing pain might be due to a “neuropathic conversion” due to sensitization of the peripheral and central nerves. The following five-step pathophysiologic process can be used to explain how this conversion occurs: (1) local cellular and humoral inflammation develops where tissue damage or ischemic injury occurs; (2) this inflammation means there is an accumulation of pain-inducing endogenous chemicals within the pain site; (3) altered peripheral neurogenic tissues develop because of these chemicals; (4) these altered nerves have lowered thresholds and even spontaneous activation; and (5) central sensitization and plasticity of the pain pathways from trigeminal nucleus or spinal cord to the cortex develop. Additional discussion of specific neuronal changes that occur in the nervous system with neuropathic pain is provided in Chapter 6, which focuses on neurogenic pain and anticonvulsant medications. This dichotomous etiology indicates that, in addition to making a diagnosis, you must also understand whether the pain is a typical nociceptive pain or an atypical neuropathic pain, because they have different prognoses and are treated quite differently.

1.1.B Differential diagnosis and etiology of chronic orofacial pain

When a patient attends a physician’s or dentist’s office with a complaint of orofacial pain, they hope fervently that they will be given a diagnosis and an effective plan of treatment. Most physicians and dentists will perform an examination, take a careful medical history, and order appropriate tests. Based on this information, a diagnosis is usually rendered. For example, if a patient has pain on function, has limited mouth opening, and notices a crunching sound coming from one of the jaw joints, a diagnosis of localized osteoarthritis

Table 1.1 The 30 most common orofacial-pain-related diseases and their distinguishing clinical features

Disease	Distinguishing clinical features
1 Myalgia	<p>Subjective pain in the muscle on function Pain that can be replicated by muscle palpation No discernable taut band or trigger point with referring pain <i>Note:</i> It is necessary to distinguish primary from secondary myalgia. Secondary myalgia sources include direct trauma to the muscle (injections) and regional painful pathology such as arthritic joint disease or disk derangement.</p>
2 Myofascial pain	<p>Subjective pain in the muscle on function Pain that can be replicated by muscle palpation Discernable taut band in the affected muscles Trigger point in this band that causes pain to radiate on sustained compression <i>Note:</i> Myalgia is labeled <i>myofascial pain</i> only when taut bands and trigger points are present.</p>
3 Fibromyalgia	<p>Subjective pain in multiple sites aggravated by function Widespread pain involving more than three body quadrants Continuous symptoms (>3 months in duration) Strong pain on muscle palpation in at least 11 of 18 established body sites <i>Note:</i> Myalgia is labeled <i>fibromyalgia</i> only when these criteria are met.</p>
4 TMJ DDWR	<p>Single noise—click or pop—from the TMJ on a single movement Noise may be reciprocal (on both open and close) No restriction or deflection of jaw motion after click</p>
5 TMJ DDNR	<p>Sudden onset, continuous loss of full jaw motion Pain in the affected joint on wide open attempt Prior history of clicking in the affected joint that has now stopped <i>DxTest:</i> MRI shows DDNR in both closed and open positions</p>
6 Local TMJ arthritis	<p>Subjective pain in preauricular area aggravated by function Pain that can be replicated by TMJ capsule palpation Joint motion often produces crepitation sounds <i>DxTest:</i> erosive or remodeling-type joint-surface changes on CT imaging</p>
7 Polyjoint OA affecting the TMJ	<p>Subjective pain in preauricular area aggravated by function Pain that can be replicated by TMJ capsule palpation Joint motion often produces crepitation sounds Multiple joints affected with pain beyond TMJ <i>DxTest:</i> erosive or remodeling-type joint-surface changes on CT imaging <i>DxTest:</i> negative serology for autoimmune markers of rheumatoid disease</p>
8 Rheumatic arthritis affecting the TMJ	<p>Subjective pain in preauricular area aggravated by function Pain that can be replicated by TMJ capsule palpation Joint motion often produces crepitation sounds Multiple joints affected with pain beyond TMJ <i>DxTest:</i> erosive or remodeling-type joint-surface changes on CT imaging <i>DxTest:</i> positive serology for autoimmune markers of rheumatoid disease</p>
9 Temporal arteritis	<p>New headache pain of a constant nature Tender, thickened, and pulseless scalp vessels <i>DxTest:</i> positive serology for autoimmune markers of an inflammatory disease <i>DxTest:</i> confirmed by blood vessel biopsy showing giant-cell infiltrate <i>Note:</i> Markers are elevated ESR and a C-reactive protein.</p>
10 Trigeminal sensory neuropathy	<p>Unilateral or bilateral sensory loss of one or more trigeminal nerve divisions Usually, also presence of pain in these same areas <i>DxTest:</i> negative MRI for pathology involving the CNS or trigeminal nerve <i>DxTest:</i> confirming diagnosis of associated CTD <i>Note:</i> Most commonly associated with an autoimmune CTD such as mixed or undifferentiated CTD, scleroderma, Sjögren’s syndrome, or lupus erythematosus. If so, there may also be complaints of Raynaud’s phenomenon, polyjoint arthritis, and sometimes muscle weakness.</p>
11 Migraine	<p>Unilateral headache location Pulsatile severe headache that lasts multiple hours Nausea associated with the headache pain Photophobia and phonophobia associated with headache pain <i>DxTest:</i> negative MRI for pathology involving the CNS <i>Note:</i> Pain episodes may be preceded by aura such as “flashing lights or dizziness.”</p>

Table 1.1 (Continued)

Disease	Distinguishing clinical features
12 Cluster headaches	<p>Rapid-onset intense paroxysmal headache pains One-sided retro-orbital, supraorbital, and temporal headache pains Pain episodes lasting from 15 to 180 minutes Headaches occur several times in a 24-hour period Pain that may occur at night, waking the patient from a sound sleep <i>DxTest:</i> negative MRI for pathology involving the CNS <i>Note:</i> Headache must be associated with ipsilateral autonomic signs, including conjunctival injection, ptosis, miosis, eyelid edema, facial flushing or blanching, forehead sweating, lacrimation, nasal congestion, and rhinorrhea.</p>
13 Tension-type headaches	<p>Dull aching bilateral, episodic pain of long-lasting duration (hours to days) Pain located in the suboccipital, temporal, and frontal regions Pain typically increasing slowly during the day to a later afternoon peak</p>
14 Chronic daily headaches	<p>Continuous or very frequent headache (4 or more days per week) Maybe with clinical features of both migraine and tension-type headache <i>DxTest:</i> negative MRI for pathology involving the CNS</p>
15 Acute trigeminal neuritis	<p>Injury- or infection-associated acute onset numbness or tingling Burning sensation in the affected nerve <i>DxTest:</i> CTs and MRI needed to check for pathology involving the involved nerve</p>
16 Trigeminal neuroma	<p>Movement- or touch-induced sharp often electric-like pain Pain occurring in an area of anesthesia that was induced after an injury or surgery that inadvertently transected a nerve</p>
17 Trigeminal neuralgia	<p>Sudden, usually unilateral, severe pain Brief (seconds), stabbing or electric-like pain Usually recurrent (multiple times a day) pain Pain occurring in one or at most two trigeminal nerve branches <i>DxTest:</i> MRI of trigeminal nerve and brain <i>Note:</i> In most (90%) cases MRI will not show pathology involving the trigeminal nerve; other cases will show CNS tumor or other pathology.</p>
18 Chronic trigeminal neuropathy	<p>Constant dental and gingival pain in a very focal oral region Usually, pain of unknown origin <i>DxTest:</i> negative radiographic finding indicative of pulpal pathology <i>DxTest:</i> negative endodontic thermal testing indicative of pulpal pathology</p>
19 Postherpetic neuralgia	<p>Burning, deep aching, tingling, itching, or stabbing pain of the skin Usually located on the V1 or V2 division Pain that is always located in area of prior viral infection where ulcerative lesion was located <i>Note:</i> Pain and preceding ulcerative lesion can be intraoral if it involves the V3 division.</p>
20 Burning mouth (not related to hyposalivation)	<p>Constant burning sensation of the anterior tissues of the mouth Pain often increasing throughout the day No clinically discernable oral pathology on examination</p>
21 Pemphigus vulgaris	<p>Blistering diseases of the skin and mucous membranes of the mouth <i>DxTest:</i> Biopsy will confirm the diagnosis of pemphigus.</p>
22 Benign mucous membrane pemphigoid	<p>Blistering diseases of the skin and mucous membranes of the mouth <i>DxTest:</i> Biopsy will confirm the diagnosis of BMMP.</p>
23 Erosive lichen planus	<p>Filamentous, white, lacy lines on the cheek or other oral tissues Erythema and ulceration of the mucosal tissues <i>DxTest:</i> Biopsy will confirm the diagnosis of LP. <i>Note:</i> LP becomes painful when it turns erythematous and erosive.</p>
24 Mucositis	<p>Painful inflammation and ulceration of the mucous membranes <i>Note:</i> This disorder almost always occurs as a result of chemotherapy and radiotherapy for cancer, although a severe allergic reaction to a medication or infection is possible.</p>
25 Ulcerative disease of the mucosa	<p>Ulcerative or severe inflammation of the mucous membrane <i>DxTest:</i> Biopsy will confirm the diagnosis of a nonspecific ulcerative disease. <i>Note:</i> Positive findings for the causative systemic or allergic disease</p>
26 Cancer pain in the jaw	<p>Trigeminal sensory disorder with variable presentation Neural deficit may be numbness or pain (continuous or episodic) <i>DxTest:</i> Positive MRI for cancer affecting trigeminal nerve</p>

(Continued)

Table 1.1 (Continued)

Disease	Distinguishing clinical features
27 Orofacial dyskinesia	Repetitive abnormal movement disorder involving the jaw, lip, and tongue <i>DxTest:</i> Negative MRI for any CNS pathology
28 Orofacial dystonia	Involuntary briefly sustained contraction of involved muscle <i>DxTest:</i> Negative MRI for any CNS pathology
29 Bruxism	Sleep-state-related motor hyperactivity causing repeated brief motor activation of the jaw closers, usually with resulting side-to-side motion of the jaw and tooth attrition <i>DxTest:</i> Wear patterns on full arch acrylic splint can prove whether bruxism is active.
30 Habitual parafunction and secondary masticatory hyperactivity	Conscious tooth clenching, habitual cheek chewing, or habitual lip biting Consider medication-induced hyperactivity if taking SSRI or stimulant Elevated masticatory and cervical muscle stiffness evident on palpation <i>DxTest:</i> Stop suspected medications if patient is on stimulants or SSRIs.

AIDS, acquired immunodeficiency syndrome; BMMP, benign mucous membrane pemphigoid; CDH, chronic daily headache; CNS, central nervous system; CT, computed tomography; CTD, connective tissue disease; ddC, dideoxycytidine; ddl, dideoxyinosine; DDNR, disk displacement with no reduction; DDWR, disk displacement with reduction; DxTest, diagnostic test; ESR, erythrocyte sedimentation rate; HIV, human immunodeficiency virus; LP, lichen planus; MRI, magnetic resonance imaging; NSAIDs, nonsteroidal anti-inflammatory drugs; OA, osteoarthritis; SSRIs, selective serotonin reuptake inhibitors; TMJ, temporomandibular joint.

is certainly probable. If the disease has progressed far enough, a radiograph of the temporomandibular joint (TMJ) will confirm and document the magnitude of the osseous changes. Unfortunately, simply reformulating the patient's complaint (painful, noisy joint) into medical nomenclature (osteoarthritis) is not sufficient. An expert clinician must strive to both find an etiology for the disease and understand the pathophysiologic basis for the pain itself, before this diagnosis is complete (Table 1.2). The discovery of the etiology is by far the most difficult part of the diagnostic process; later in this chapter and in several other chapters, we discuss what is currently known about the causation of most of the common orofacial pain disorders.

1.1.C Anatomic localization and age predilection

Another essential component of the differential diagnostic process is to fully understand and document the anatomic localization and extent of the pain site. In most cases this begins by having the patient outline the pain's outer borders and then pinpoint the pain's focal source (if one exists). The clinician must also palpate this source to verify it and see if, with simple pressure, the pain can be replicated. Based on the physical signs and symptoms as well as the anatomic location, pattern, and character of the pain, a list of diseases that cause pain in the orofacial region can usually be narrowed down to two or three likely pain disorders. This process is facilitated if, when creating the differential diagnosis list, the clinician has in mind the "age-based" predilections of the painful diseases that occur in the orofacial region. For example, trigeminal neuralgia is far more likely in someone over the age of 50 than under the age of 50.

1.1.D Diagnostic testing

Appropriate tests or diagnostic–treatment procedures may help narrow the list to the most likely diagnosis; however, due to the subjective nature of pain, there is no test that can measure the intensity of pain, nor any current clinically useful imaging device that can show pain. In most cases, clinicians must use the patient's own description of the type, duration, and location of the pain to get diagnostic clues. Certain pain-inducing pathologies are visible on a radiograph or a magnetic resonance image (MRI); however, because many are not, we must occasionally use innovative methods to confirm our diagnosis. These innovative methods are discussed in Chapter 10, but here we provide in table form the most frequently used diagnostic methods appropriate for the 30 diseases considered in this chapter (Table 1.3). More details on the pros and cons of these tests are provided in the various chapters where each disease entity is discussed.

1.1.E Prevalence of orofacial pain

Comparing the age predilection, the anatomic localization, and the character of the patient's problem with the known prevalence of orofacial pain disorders usually allows the clinician to make a reasonable diagnosis. The reported overall prevalence of general persistent pain in the adult population of the United States is quite high. For example, a Gallup survey of 2002 adults found that approximately 4 of 10 adults (42%) of those polled say they experience pain on a daily basis, while 89% admit to experiencing pain on a monthly basis.² These pains have diverse origins: chronic pain disorders such as arthritis, osteoporosis, diabetic neuropathy, migraine, and fibromyalgia; pain related to cancer;

Table 1.2 Probable etiologies associated with the 30 most common orofacial pain diseases

Disease	Etiology
1 Primary and secondary myalgia (all types)	Medications (stimulants or SSRIs) causing motor hyperactivity Stress (job or personal) causing muscular hypoperfusion and/or hyperactivity Waking and sleeping parafunctions (repetitive oral habits and behaviors) History of traumatic muscle injury (intramuscular local anesthetic injection) Local nonmuscle pathology (arthritis or derangement)
2 Myofascial pain (all types)	Common etiologies same as for myalgia Taut bands and trigger points, suggesting localized neuronal sensitization in muscle
3 Chronic widespread pain and fibromyalgia	Common etiologies same as for myalgia Multiple pain sites, allodynia, and mechanical hyperalgesia, suggesting central sensitization Unknown genetic susceptibility that predisposes to fibromyalgia
4 TMJ DDWR	Traumatically altered discal ligaments that attach it to the condyle Parafunction Joint hypermobility Acute macrotrauma to jaw
5 TMJ DDNR	Common etiologies that cause DDNR same as for DDWR
6 Localized TMJ arthritis	Trauma (either macrotrauma or repetitive microtrauma) A prior DDNR in the involved joint
7 Polyjoint osteoarthritis and TMJ	Idiopathic (but most likely genetic) Secondary polyjoint arthritis (e.g., psoriasis)
8 Rheumatic arthritis and TMJ	Autoimmune induced
9 Temporal arteritis	Giant-cell inflammation due to autoimmunity
10 Idiopathic trigeminal sensory neuropathy	Autoimmunity (seen with various CTDs such as Sjögren's syndrome, undifferentiated and mixed CTD, and scleroderma)
11 Migraine	Genetics
12 CH and autonomic cephalalgias	Genetics
13 Tension-type headaches	Stress
14 Chronic daily headaches	Neuronal sensitization due to frequent episodic headaches Genetic factors likely Stress factors likely Analgesic medication overuse may play a causative role in CDH.
15 Facial pain related to trigeminal neuritis	Viral-induced neural inflammation (e.g., HIV, <i>Cytomegalovirus</i> , <i>Poliovirus</i> , and hepatitis B or C infections) Trauma-induced neural inflammation Bacterial-induced neural inflammation (e.g., leprosy, diphtheria, Lyme disease, and trypanosomiasis) Diabetes may be involved if widespread Rare immune reactions (e.g., Guillain-Barré syndrome; chronic inflammatory demyelinating polyneuropathy; neuropathies associated with vasculitis) Metabolically induced and nutritional-imbalance-induced neuropathy (e.g., deficiency of vitamins B ₁₂ , B ₁ [thiamine], B ₆ [pyridoxine], and E) Renal-failure-induced polyneuropathy Toxin-induced polyneuropathy (e.g., alcohol and other toxins) Medication-induced neuritis (e.g., vincristine and cisplatin; ddC and ddI in AIDS; and dapsone, used to treat leprosy)
16 Facial pain related to trigeminal neuroma	Surgical or trauma-induced nerve trunk transection
17 Facial pain related to trigeminal neuralgia	Vascular compression Multiple sclerosis Acoustic neuroma (tumor) induced compression CNS neoplasia
18 Facial pain related to a chronic trigeminal neuropathy	Inflammation or trauma to alveolar nerve (e.g., traumatic injury, periodontal surgery, pulp extirpation, endodontic therapy, apicoectomy, tooth extraction, implant insertion) Maybe genetic factors

(Continued)

Table 1.2 (Continued)

Disease	Etiology
19 Facial pain related to postherpetic neuralgia	Herpes zoster infection
20 Burning mouth symptoms (not related to hyposalivation)	Inflammation- or age-related trigeminal small fiber dysfunction or atrophy in tongue and lip region
21 Pemphigus vulgaris	Autoimmunity against keratinocyte cell surfaces
22 Benign mucous membrane pemphigoid	Autoimmunity
23 Lichen planus	Autoimmunity Lichenoid reactions, allergic responses to an allergen Medication induced (e.g., antihypertensive drugs, NSAIDs, tetracycline, and several sulfonamides)
24 Mucositis	Chemotherapy Radiation therapy Allergic reaction to medication
25 Other chronic (nonmalignant) ulcerative conditions of the mouth	Autoimmunity Trauma Systemic disease with oral manifestations (e.g., Behçet's disease, celiac disease, GVHD, Crohn's disease, ulcerative colitis, lupus erythematosus, and neutropenia)
26 Cancer pain in the jaw	Neoplasia invasion of trigeminal nerve or base of brain at foramen ovale Jaw bone cancer due to primary or malignant–metastatic neoplasia
27 Dyskinesia	Idiopathic dysfunction of basal ganglia Medication induced (e.g., neuroleptic medications)
28 Dystonia	Idiopathic dysfunction of basal ganglia
29 Bruxism	Disinhibition disorder involving the jaw motor system during sleep
30 Habitual parafunction and spontaneous and secondary hypertonicity	Idiopathic extrapyramidal system hyperactivity Medication-induced motor hyperactivity (e.g., SSRIs or psychostimulants) Stress

AIDS, acquired immunodeficiency syndrome; CDH, chronic daily headache; CH, cluster headache; CNS, central nervous system; CTD, connective tissue disease; ddC, dideoxycytidine; ddI, dideoxyinosine; DDNR, disk displacement with no reduction; DDWR, disk displacement with reduction; GVHD, graft-versus-host disease; HIV, human immunodeficiency virus; NSAIDs, nonsteroidal anti-inflammatory drugs; SSRIs, selective serotonin reuptake inhibitors; TMJ, temporomandibular joint.

Table 1.3 Confirmatory or exclusionary diagnostic methods

Disease	Diagnostic testing
1 Localized myalgia	History, palpation findings
2 Myofascial pain	History, palpation findings
3 Fibromyalgia	History, palpation findings
4 TMJ DDWR	Auscultation, jaw ROM assessment
5 TMJ DDNR	Palpation, jaw ROM assessment, MRI
6 Local TMJ arthritis	Palpation, cone beam CT of TMJ
7 Polyjoint OA (affecting the TMJ)	Palpation, cone beam CT of TMJ, clinical review of all joints
8 Rheumatic arthritis (affecting the TMJ)	Cone beam CT, serologic testing (RF, ESR, ANA), clinical review of all joints
9 Temporal arteritis	Serologic testing (ESR, CRP), scalp vessel palpation, blood vessel biopsy
10 Trigeminal sensory neuropathy	MRI imaging (to rule out CNS pathology), serologic testing for CTDs (ANA, CRP)
11 Migraine	History, MRI (to rule out CNS pathology)
12 Cluster headaches	History, MRI (to rule out CNS pathology)

Table 1.3 (Continued)

Disease	Diagnostic testing
13 Tension-type headaches	History
14 Chronic daily headaches	History, MRI (to rule out CNS pathology)
15 Acute trigeminal neuritis	History, neurologic exam, MRI (to rule out CNS pathology)
16 Trigeminal neuroma	MRI (to rule out CNS pathology), neurologic exam, anesthetic testing
17 Trigeminal neuralgia	History, MRI (to rule out CNS pathology), neurologic exam
18 Chronic trigeminal neuropathy	History, dental radiographs, MRI, anesthetic testing
19 Postherpetic neuralgia	History, anesthetic testing
20 Burning mouth (not due to hyposalivation)	History, MRI (to rule out CNS pathology)
21 Pemphigus vulgaris	Clinical exam, biopsy
22 Benign mucous membrane pemphigoid	Clinical exam, biopsy
23 Lichen planus	Clinical exam, biopsy
24 Mucositis	History, clinical exam
25 Other ulcerative disease of the mucosa	History, clinical exam, serologic testing
26 Cancer pain in the jaw	MRI and CT
27 Orofacial dyskinesia	Brain MRI (to rule out CNS pathology)
28 Orofacial dystonia	Brain MRI (to rule out CNS pathology)
29 Bruxism	History and examination, occlusal appliance wear pattern
30 Oral parafunction and spontaneous and secondary hypertonicity	History, palpation

ANA, antinuclear antibody; CT, computed tomography; CNS, central nervous system; CRP, C-reactive protein; CTD, connective tissue disease; DDNR, disk displacement with no reduction; DDWR, disk displacement with reduction; ESR, erythrocyte sedimentation rate; HIV, human immunodeficiency virus; MRI, magnetic resonance imaging; NSAIDs, nonsteroidal anti-inflammatory drugs; OA, osteoarthritis; RF, rheumatic factor; ROM, range of motion; SSRIs, selective serotonin reuptake inhibitors; TMJ, temporomandibular joint.

postsurgical pain; and pain caused by accidents and burns. Whether it is cancer pain or noncancer pain, opioid treatment of pain is common. For example, 73% of hospitalized patients receiving opioid therapy still reported moderate distress and 75% of postsurgical patients were in either moderate or marked distress.^{3,4} When you look more closely at elderly patients (defined as over the age of 65), the prevalence of general persistent pain is much higher than in those under the age of 65. The prevalence of persistent pain in the elderly ranges from 25% to 88%, depending on the definition used and the subset of elderly patients being studied.^{5,6} For example, another study conducted telephone interviews of community-dwelling north Floridians ($n = 1636$) who were over 65 years of age and found that 17.4% reported some form of current or recent (with the last year) orofacial pain.⁷

1.2 Facial pain related to muscle pain

Muscle pain comes in many forms, from the widespread types such as fibromyalgia to the local and regional forms

of myalgia. Myofascial pain and the more generalized fibromyalgia syndrome (FMS) are common chronic pain problems that predominantly affect middle-aged women.⁸⁻¹¹ While local myalgia and myofascial pain are more prevalent in the middle aged, fibromyalgia increases with age and is substantially more evident in the elderly population. Each of the myalgia subtypes is discussed in the following subsections. A detailed discussion of these disorders and their appropriate management is presented in Chapter 16.

1.2.A Disease 1a: primary myalgia

Myalgia can be separated into local and regional, with a distinction between primary and secondary myalgia also made. The term “primary myalgia” indicates that if a biopsy were performed, there would be no microscopic evidence of inflammation. Histologically evident myositis is discussed in Section 1.2.B on secondary myalgia.^{12,13} The pain-inducing changes seen in primary myalgia are most likely due to sensitization of muscle nociceptors.

Clinical criteria

When a direct muscle injury that explains the muscle pain cannot be found and the patient does not have another adjacent pathology in the area that would cause secondary muscle guarding effects (e.g., arthritis of the TMJ or internal derangement of the TMJ), then one of the criteria for a primary myalgia is satisfied. The actual diagnosis of myalgia (all types) requires the following additional criteria to be satisfied: (1) the patient is aware of pain in the muscle on function; (2) this pain must be replicated by palpation; and (3) there is no discernable taut band or trigger point in the muscle that causes pain to radiate on palpation.

Etiology

If a primary myalgia is suspected, the clinician must seek to find the etiology by asking about (1) medications, (2) stress, and (3) parafunctions (both waking and sleeping). If a patient is using psychological stimulant medication or is using a serotonin selective reuptake inhibitor (SSRI), then a medication-induced myalgia would be suspected. The various medications that can induce muscle pain are reviewed in Chapter 19 and are not discussed here. Second, a stress-associated myalgia should be suspected if a patient reports a prolonged increase in environmental (job or personal) stress levels. A discussion of how stress can induce muscle pain without the presence of histologically evident inflammation is given in Chapter 16. With regard to stress, psychological factors have been associated with chronic facial and jaw pain.¹⁴ Third, a parafunction-induced myalgia should be suspected when a patient admits to repetitive oral habits or if such habits are observed. In this case the clinician will typically diagnose a primary myalgia due to parafunction. Sometimes the parafunction is very specific and the pain it produces in the jaw muscles is limited to one or two muscles. Oral parafunctions may be present both during waking and sleeping hours and during specific activities such as chronic gum chewing.¹⁵

Several studies have reported that there is a moderately strong positive association between self-reported clenching and chronic masticatory myofascial pain (MMFP).^{16–18} Unfortunately, these studies do not specify whether the clenching is occurring during waking or sleeping periods because to do so accurately would require an actual recording of the jaw motor behaviors over moderately long periods of time (minimum 2 weeks). One study used a case–control design including 83 patients with MMFP, selected from the patients at a hospital dental service, and 100 concurrent controls. Using unconditional logistic regression analysis they found that self-reported clenching–grinding either in association with an elevated anxiety score (OR = 8.48) or an elevated depression score (OR = 8.13) was statistically associated with chronic MMFP. They concluded that tooth

clenching, trauma, and female gender strongly contribute to the presence of chronic MMFP even when other psychological symptoms are similar between subjects. Interestingly, grinding-only behaviors, age, and household income and education were not related to chronic MMFP. This report showed no association between tooth grinding and chronic muscle pain, which is in conflict with other studies. For example, one study performed a questionnaire-based epidemiologic cross-sectional study and another used a clinically based case–control design.^{19,20} These two studies found a positive relationship between self-reported nocturnal tooth grinding and self-reported jaw pain. This conflict will require additional data to resolve.

1.2.B Disease 1b: secondary myalgia due to active local pathology (e.g., temporomandibular joint disease)

Direct muscle injury is not common in the masticatory system, but when present it can produce a quite dramatic change in normal function causing strong focal pain and severely limited opening; this limitation is due to co-contraction of the openers and closers and is called trismus.²¹

Clinical criteria

The term “secondary myalgia” implies the presence of some extrinsic direct trauma or local (nonmuscle) pathology that is inducing myalgia.

Etiology

The two most common causes of a secondary myalgia are (1) a traumatic muscle injury and (2) a local nonmuscle pathology that induces a change in muscle function. The most common traumatic cause of a focal myositis in the jaw system is an inadvertent intramuscular injection of local anesthetic during dental treatment. In these cases, the nature of the injury is influenced by the amount of injected material, the type of anesthetic used, and more important, whether a vasoconstrictor such as epinephrine was included in the anesthetic solution. Several authors have described and documented the effect of an inadvertent anesthetic injection into muscle tissue.^{22–25} In some cases, acute traumatic trismus can convert to chronic contracture of the involved muscle.²⁶ Other forms of local muscle injury can occur from trauma (e.g., neck musculature can be injured during a low-velocity rear-end collision) that produces a regional cervical muscle strain, which then causes a secondary cervical and sometimes even masticatory myalgia. Current data suggests that the jaw closing and opening muscles themselves are not overstretched or torn during a low-velocity rear-end motor vehicle collision,^{27,28} but they may become involved when a guarding–trismus response develops in concert with the

injured craniocervical muscles.²⁹ If a direct muscle trauma is suspected as the etiology, then the traumatic event is usually easily identified in the history. Fortunately, most such traumatic injuries are self-resolving without long-term consequences.

When a local pain-inducing pathology is present, localized and even regional myalgia will develop in response. For example, acute TMJ arthritis can cause an associated muscle pain in the masseter and temporalis on the side ipsilateral to the involved joint. The pain in the muscle tissue is secondary, but it may generate an equal or greater degree of tenderness to palpation than elicited by palpating the involved joint. That the nociceptors inside a joint or even inside a tooth can induce a secondary motor reaction in the anatomically adjacent muscle has been clearly demonstrated in the literature.^{30,31} The most likely secondary jaw and cervical motor activation occurs with a painful arthritis or internal derangement of the TMJ.³² However, these reactions are also likely to occur with acute pulpal pain, osteomyelitis, or other mandibular bone or soft-tissue infections in the region. When a patient presents with one-sided muscle pain in the absence of trauma or a strong stress or parafunction history, the clinician should carefully examine the TMJ for local disease or dysfunction. When a patient presents with both a local pathologic process and muscle pain that seems to have developed after the pathology began, it would be appropriate to consider that the myogenous process is a secondary myalgia not a primary one. In these cases it is logical and appropriate to manage or minimize the local pathology first and then re-examine the myogenous pain for resolution or persistence.

1.2.C Disease 2: myofascial pain (focal or regional)

While many consider myalgia and myofascial pain to be similar, the International Association for the Study of Pain Subcommittee on Taxonomy has classified myofascial pain (MFP) as pain in any muscle with trigger points that are very painful to compression during palpation and cause referred pain sensations.³³ Essentially the term myofascial pain is used only when specific criteria are satisfied.

Clinical criteria

The criteria for myofascial pain are both subjective (history based) and objective (examination based). The three subjective criteria that patients should endorse are (1) spontaneous dull aching pain and localized tenderness in the involved muscle(s), (2) stiffness in the involved body area, and (3) easily induced fatigueability with sustained function. The four objective criteria are (1) a hyperirritable spot within a palpably taut band of skeletal muscle or muscle fascia,

(2) reports by the patient, upon sustained compression of this hyperirritable spot, of new or increased dull aching pain in a nearby site, (3) decreased range of unassisted movement of the involved body area, and (4) weakness without atrophy and no neurological deficit explaining this weakness. Many have included the presence of referred autonomic phenomena upon compression of the hyperirritable spot and/or a twitch response to snapping palpation of the taut bands as additional diagnostic criteria.^{34–38} However, inclusion of the last criterion is not endorsed by all since it is not a reliably present physical finding.³⁹

Etiology

The common etiologies that cause myofascial pain are the same as those given for myalgia (see Secs. 1.2.A and 1.2.B).

1.2.D Disease 3: chronic widespread pain and fibromyalgia

Chronic widespread pain and fibromyalgia are quite similar conditions in that the patient has complaints of multiquadrant muscle pain, but only fibromyalgia has an accepted set of specific physical examination criteria. Fibromyalgia affects up to 2% of the population and can start at any age; it is at least 7 times more common in women than in men.⁴⁰ By the time the diagnosis is made, patients have often had symptoms for many years.

Clinical criteria

Patients with fibromyalgia complain of muscular and sometimes joint pain all over and, by definition, have pain on both sides of the body, above and below the waist, and in both the trunk and extremities. There are specific clinical history and examination criteria that must be met before a diagnosis of fibromyalgia is rendered. These criteria, adopted by the American College of Rheumatology (ACR), specify that a diagnosis of fibromyalgia is made when there is widespread pain lasting for at least 3 months accompanied by tenderness at discrete locations.⁴¹ According to the ACR criteria, patients must have at least 11 tender points of a possible 18 but, in practice, the diagnosis can be made in patients with fewer tender points if there is widespread pain and many of the other characteristic symptoms. Patients with fibromyalgia are often tender all over; the presence of tenderness other than at the classic locations does not exclude the diagnosis. These findings suggest and most researchers agree that an aberrant central pain processing mechanism produces a state of sensitized pain perception in FMS.⁴² Because of the widespread muscle and joint pain, fibromyalgia patients usually have poor-quality nonrestorative sleep. They also frequently report irritable bowel syndrome and headaches. Because of

the negative effect fibromyalgia has on activities of daily living, it usually induces depression and anxiety, and it often accompanies other chronic painful disorders.⁴³

Etiology

It is likely that patients who develop chronic widespread pain and/or fibromyalgia have a genetic factor that predisposes them to sensitization of the central nervous system (CNS). For the local factors that trigger pain, see Sections 1.2.A and 1.2.B; the common etiologies that cause fibromyalgia are the same as those given for myalgia.

1.3 Facial pain due to derangement and non-autoimmune arthritis or capsulitis of the temporomandibular joint

The second subgroup of conditions is facial pain due to joint and disk derangements as well as the non-autoimmune arthrogenous diseases. “Derangement” is a nonspecific term that means abnormal function of the intra-articular structures (displacement of the disk), but in this section we also include abnormal joint function (dislocation and locking), as described in Section 1.3.C. Disk derangement of the TMJ is more common in the 20- to 50-year-old population.⁴⁴ Localized osteoarthritis is characterized by focal degeneration of joint cartilage with osseous erosion and sclerosis; sometimes osteophyte formation at the joint margins occurs in an older cohort of patients.^{45,46} In addition to osteoarthritis, there are a number of polyarthritic diseases in which the TMJ is involved in the arthrogenous process. These various conditions are described in Sections 1.3.D and 1.3.E and in the next subgroup of orofacial pain disorders (Sec. 1.4).

1.3.A Disease 4: disk displacement with reduction

Disk displacement with reduction (DDWR) is more of a dysfunction than a pain disorder, but if the joint tissue is inflamed, a click can be painful.

Clinical criteria

Evidence for disk displacement with reduction is transient jaw movement interference or clear joint noise, noted clinically as a single joint sound (usually described as a click or pop) emanating from one or both joints. A diagnosis of DDWR is not appropriate if the opening or closing movement noise is only an asynchronous eminence translation. If the click is associated with a clear loss of maximum opening

ability or if the noises are a result of arthritic changes in the joint (i.e., crepitus or multiple noises in a single movement), then the diagnosis of disk displacement without reduction (DDNR, Sec. 1.3.B) or osteoarthritis will supersede the diagnosis of DDWR.

Etiology

For a TMJ disk to be displaced, the ligaments that attach it to the condyle must be stretched to such a degree that the disk has additional mobility. This process can occur from parafunction, joint remodeling, acute trauma, and joint hypermobility syndrome. These etiologies and how they cause DDWR and DDNR are discussed in Chapter 20.

1.3.B Disease 5: disk displacement with no reduction

Disk displacement with no reduction (DDNR) is definitely painful when the patient attempts to open wide in the early stages.

Clinical criteria

The appropriate historical evidence for DDNR is a clear TMJ movement restriction or hypomobility that began suddenly and has continued since that time without remission. The appropriate clinical evidence for this disorder is maximum passive stretch mouth opening (interincisal distance including overbite) of less than 38 mm. This opening is often accompanied by a deflection to the side that is locked during maximum opening. The patient will also have only a small limitation of lateral motion if any loss is evident. Finally, the affected joint often has a history of joint noises that stopped at the time of the movement restriction. If the acute onset hypomobility becomes chronic (i.e., greater than 6 months), the opening may increase by several millimeters (up to 42 mm) and crepitus noises may develop. Magnetic resonance imaging is needed to see the disk since it is a soft-tissue structure that cannot be seen on computerized tomography (CT).

Etiology

The common etiologies that cause DDNR are the same as those given for DDWR (see Sec. 1.3.A).

1.3.C Open dislocations and locking problems seen in the temporomandibular joint

Because they are relatively rare and generally unmistakable when present, these three TMJ internal derangement subcat-

egories are not included in this group of 30 most common disorders.

Clinical criteria

- 1 A *true open dislocation* is present when the condyle undergoes excessive translation, moving to a position that is well beyond where it would normally go to even with a very wide open movement. In this position the jaw will be unable to close and usually requires that manual manipulation of the jaw be performed to reduce the problem.
- 2 A *simple open locking* is often mistakenly diagnosed as a dislocation when the patient's jaw is actually only locked open and not truly dislocated. An open locking is present when the condyle becomes stuck or locked in a wide open position (condyle anterior to eminence) but is not in a position of excessive condyle translation. Similar to true dislocation, open locking is a situation in which the patient is unable to close, but most times the patient is able to self-reduce the locked jaw without assistance.
- 3 A *posterior disk displacement* of the TMJ disk causes an inability to fully close after opening or a partial-open locking. Actually this condition should not be confused with the prior problem of wide-open locking of the condyle. These patients complain of the inability to close their jaw after opening but the condyle is not anterior to the articular eminence. If only one joint is involved, the jaw may be in an extreme lateral position but again not in a wide open position. The likely cause of this condition is a posterior DDNR, preventing the condyle from returning to its original position or full closure. Spasm of the lateral pterygoid can also cause the posterior teeth not to articulate.

It should be noted that dysfunction, not pain, is clearly the main problem when derangements occur, because a disk derangement of the jaw (clicking, locking, and/or dislocation) is normally not painful when the jaw is not moving. On the other hand, osteoarthritis does cause spontaneous pain and certainly pain on function.

A detailed discussion of derangement-type disorders and their appropriate management is presented in Chapter 20.

Etiology

For a true dislocation to occur, the ligaments that restrict condyle motion (i.e., the TMJ ligament) must be stretched to such a degree that the condyle has additional mobility. For an open locking to occur, the various ligaments of the jaw do not need to be stretched or torn, but jaw elevator muscles must tighten (i.e., develop trismus) to such a degree

that the joint is jammed anterior to the eminence. For a posterior disk displacement this dysfunction develops due to the same process as for anterior disk displacement, namely the disk ligaments are stretched. These etiologies and how they cause DDWR and DDNR are discussed in Chapter 20.

1.3.D Disease 6: local temporomandibular joint arthritis

As the name implies, arthritis of the TMJ is a painful inflammation of the joint. Osteoarthritis (OA) is the most common degenerative disease that affects the TMJ. It is considered a disease of the bone, cartilage, and supporting tissues and is the result of both mechanical and biologic events that destabilize the normal coupling of degradation and synthesis of articular cartilage and subchondral bone.⁴⁷

Clinical criteria

A painful joint without any osseous changes is described as arthralgia, which is considered to be present when the joint tissues exhibit increased tenderness to palpation pressure. Other terms for arthralgia are capsulitis, retrodiscitis, synovitis, and joint effusion. When a crunching or grinding type of noise is produced by motion of the jaw and/or if TMJ radiographs confirm the presence of bony surface deterioration, then the diagnosis switches to localized OA. If the condition involves joints other than the TMJ, then it is called a polyarthritic osteoarthritis assuming no other arthritic disease process is identified. Osteoarthritis also requires the presence of joint pain confirmed by palpation and/or detectable crepitus coming from the involved joint. If only bony surface changes are present and normal function exists and no pain is elicited, this condition is described as osteoarthrosis. Radiographic findings that indicate degenerative arthrotic changes of the TMJ are loss of joint space, flattening of the articulating surfaces, bony spurs, sclerosis of bony surfaces, or discrete erosive bony lesions. Once pain, swelling, and dysfunction are found in other body joints beyond the TMJ, then polyarthritis is considered to be present. The polyjoint form of OA has no serologic markers but almost always there are clear radiographic indications (e.g., flattening, loss of space, spurs, erosive lesions, and sclerosis) of arthrotic changes of the TMJs.

Etiology

Localized OA is usually thought to be traumatic in nature (either macrotrauma or repetitive microtrauma) but could also be due to a rare infective arthritic disease. When an elderly patient attends a dentist's office with a complaint of jaw pain, the most likely diagnosis is localized arthritis

(assuming he or she does not exhibit polyjoint arthritic disease). This can usually be discovered with palpation, auscultation, and radiographic examination of the joint. When a patient has such complaints, is in his or her twenties or thirties, and there is no clear-cut traumatic injury to explain the localized arthritis, the most likely trauma is a prior DDNR of the involved joint. In a study based on a European population, the reported prevalence of OA was approximately 12% for subjects between 25 and 50 years of age, but in patients over 60 years this prevalence reached as high as 95%.⁴⁸ Osteoarthrotic changes in the TMJs are much less prevalent than the study's data might suggest for all body sites. Specifically, as reported for a random sample of elderly Finnish subjects (between 76 and 86 years of age).⁴⁹ Aging, in and of itself, is not thought to cause osteoarthritis, but if a combination of several age-related changes occurs in the same individual, then OA will result. Specifically, forceful repetitive function (e.g., bruxism) and/or disk displacement along with synovial fluid alterations of the TMJ will predispose the elderly individual to OA. Arthritic disorders and their management are presented in Chapter 18.

1.3.E Disease 7: polyjoint osteoarthritis and the temporomandibular joint

Polyjoint osteoarthritis may also involve the TMJ; the difference between polyjoint and localized osteoarthritis lies mostly in etiology and prognosis. Polyjoint OA of the TMJ is less likely to be due to a local traumatic event and the odds of improvement are lower. There are several polyjoint arthritic conditions that affect the TMJ but OA is the most common.

Clinical criteria

The clinical findings in polyjoint OA of the TMJ do not differ from the findings described in Section 1.3.D, except that the patient must have other body joints involved. For example, one easily recognizable clinical marker of polyjoint OA is the formation of Heberden's nodes on the distal interphalangeal joint of the hand. The proximal interphalangeal joint, first carpometacarpal joint, spine, and knee and hip joints are also common OA sites.

Etiology

Primary polyjoint osteoarthritis is more or less considered idiopathic, although genetic defects are suspected strongly in this disease especially when a familial pattern of OA is present.⁵⁰ Secondary polyjoint osteoarthritis is defined as joint damage or cartilage changes characteristic of osteoarthritis caused by other identified congenital or developmen-

tal disorders.⁵¹ Prior trauma, surgery, inflammatory disease, bone disease, blood dyscrasias, neuropathic joint diseases, excessive frequent intra-articular steroid injections, endocrinopathies, and metabolic disorders may damage joint surfaces and cartilage.⁵² Finally, with severe and very aggressive polyjoint OA, it is necessary to also have a negative serologic test for rheumatoid factors before the diagnosis of polyjoint or generalized OA can be confirmed. It is likely that molecular-genetic defects in type 2 cartilage collagen binding proteins are involved since they are critical to joint health. A recent review on the genetic risk factors for OA discussed the findings from twin studies, segregation analyses, linkage analyses, and candidate gene association studies and summarized inheritance patterns and the location in the genome of potentially causative mutations.⁵³ However, the various studies do not always provide a consensus on the genetic factors that are etiologic for this condition.

1.4 Autoimmune arthritic, connective tissue, and vascular disorders causing facial pain

The third subgroup of orofacial pain conditions is facial pain due to chronic autoimmune-related disorders of joints (including the TMJ), connective tissues, or vascular tissues. In this subgroup and by far the most common is rheumatoid arthritis (RA). Second most prevalent is the vascular disease temporal arteritis, characterized by inflammation of large and middle-sized blood vessels with giant cell-type inflammatory cells inside the arteries.⁵⁴ Third, an uncommon inflammation of the trigeminal nerve causes a combination of pain and numbness in the trigeminal nerve. This sensory neuropathy has been associated with a variety of connective tissue autoimmunities, such as Sjögrens syndrome, lupus erythematosus, scleroderma, and mixed and undifferentiated connective tissue disease.

1.4.A Disease 8: rheumatic arthritis and the temporomandibular joint

Rheumatoid arthritis is a polyjoint disease that affects the TMJ. RA is the most common chronic, systemic, autoimmune, inflammatory disease that affects the TMJ; other polyjoint diseases include lupus erythematosus and psoriatic arthritis, but they are not included in this group of 30 most common disorders.

Clinical criteria

Rheumatoid arthritis is characterized by joint inflammation, erosive properties, and symmetric multiple joint involve-

ment. RA can involve other body organs and in some patients can be an aggressive disease causing progressive joint damage, decreased function, and increased impairment. The main serologic marker, rheumatoid factor (RF), an immunoglobulin M (IgM) autoantibody against the Fc portion of an IgG molecule, is found in 75–80% of patients. Edema, hyperplasia of synovial lining, and inflammatory infiltrate are early components of the clinical presentation. Chronic RA is characterized by hyperplasia of Type A synovial cells and subintimal mononuclear cell infiltration resulting in the massive damage of cartilage, bone, and tendons by the pannus, an infiltrating inflammatory synovial tissue mass.^{55–58} Rheumatoid arthritis is found in the temporomandibular joint in more than 50% of adults and children with RA,⁵⁹ but the TMJ appears to be one of the last joints attacked by RA. Clinical findings include dull aching pain associated with function, joint edema, and limited mandibular range of motion. When severe, an anterior open bite can result but typically the patient has morning stiffness and has stiffness and pain at rest. Radiographic findings range from flattening of the condylar head to severe, irregular condyle deformity.

Etiology

While the etiology is unknown, certain genetic markers, *HLA-DR4* and *HLA-DRI*, are found in approximately 30% of patients with RA.

1.4.B Disease 9: temporal arteritis

This giant-cell-based inflammatory disease of the vasculature occurs when the cranial and scalp vessels become inflamed.

Clinical criteria

Patients with temporal arteritis have palpable vessels of the scalp that are sore, tender, thickened, and sometimes pulseless because of the inflammation.⁶⁰ The mean age of onset for temporal arteritis is 70 years and it is rare in people less than 50 years of age.⁶¹ A study examining the influence of age on the clinical expression of biopsy-proven giant cell arteritis reported this disorder as more common in women (female-to-male ratio 1.58:1.00) and as occurring in patients with an age greater than or equal to 50 years.⁶² Systemic symptoms (e.g., fever) occur in about half of patients, and in about 15% of patients it may be the presenting clinical manifestation. In approximately two-thirds of all patients, headache is the most frequent seminal symptom. The onset is more often gradual, but it can also be abrupt with new headache pain such as scalp tenderness

as a primary complaint. The pain symptoms are usually confined to the temporal and sometimes the occipital arteries, but the occipital arteries are less often involved. Occasionally, intermittent claudication (fatigue or pain on function) may occur in the muscles of the jaw or even tongue. In rare cases, more marked vascular narrowing may lead to infarction of the scalp or the tongue. One serious complication of temporal arteritis is permanent partial or complete loss of vision in one or both eyes. Affected patients typically report partially obscured vision in one eye, which may progress to total blindness. If untreated, the other eye is likely to become affected within 1–2 weeks. Warning signals for temporal arteritis include onset of a new headache after the age of 50, the progressive course and systemic symptoms of malaise, and jaw claudication on function. The screening investigations usually ordered for clinically suspected temporal arteritis are (1) complete blood count, (2) erythrocyte sedimentation rate (ESR), (3) C-reactive protein, (4) urea electrolytes, (5) liver function, (6) bone biochemistry, (7) glucose, (8) thyroid function, (9) rheumatoid factor, (10) electrophoresis, and (11) a chest X-ray. If the ESR is elevated, a biopsy of a clinically affected scalp vessel is confirmatory.⁶³

Etiology

The cause of temporal arteritis is thought to be related to multiple environmental and genetic factors that trigger this autoimmune-type inflammatory reaction.

1.4.C Disease 10: idiopathic trigeminal sensory neuropathy

Trigeminal sensory neuropathy (TSN) is a multifactorial inflammatory disorder of the trigeminal nerve causing sensory dysfunction (numbness, pain).

Clinical criteria

The TSN patient usually presents with unilateral or bilateral sensory loss of one or more divisions of the trigeminal nerve. The numbness can be either painful or nonpainful. Because of the association with mixed and undifferentiated connective tissue disease there may also be complaints of Raynaud's phenomenon, polyjoint arthritis, and sometimes muscle weakness.

Etiology

This condition is associated with Sjögren's syndrome, undifferentiated and mixed connective tissue disease, and scleroderma, which are all considered to be connective tissue

disorders.^{64–70} The source of the underlying neural dysfunction is thought to be autoimmune because of this association.⁷¹ The sensory deficits of facial pain and numbness can occur several years before a clear serologic confirmed clinical diagnosis of one of these connective tissue diseases, requiring vigilance for cancer-induced neural dysfunction.

1.5 Headache pains that cause orofacial pain

The fourth subgroup of 30 orofacial pain conditions is facial pain due to headaches. Approximately 90% of headache pain in the adult population is caused by migraines or tension-type headaches.⁷² However, of the new headaches that develop in people over 50 years old, approximately one-third are due to intracranial lesions or some other systemic disease. The overall prevalence of headaches declines with age and it has been reported that the prevalence of headaches declines from 83% of individuals between ages 21 and 34 to 59% between ages 55 and 74.⁷³ One exception to this generalization is migraines, which sometimes occur for the first time after age 50; in fact, about 2% of all migraines start at this late age.⁷⁴ The following subsections discuss episodic headaches as well as those that have converted to the chronic form.

1.5.A Disease 11: migraine

This common disorder is considered to be a neurovascular dysfunction of the trigeminal nerve.

Clinical criteria

The main criteria for migraine with or without aura are (1) unilateral headache location, (2) a pulsatile headache, (3) nausea associated with the pain, and (4) photophobia and phonophobia. If an aura is present, it occurs before the headache pain develops and is described as a “flashing light or dizziness.” Migraines occur slightly more often in women than men, and mostly in people under 40 years old. When the headache develops, it usually lasts 2–6 hours, but never more than several days. There are several migraine variants, such as: (a) hemiplegic migraine (head pain, transient motor–sensory changes); (b) ophthalmoplegic migraine (eye pain, transient optic nerve palsy with diplopia–ptosis); (c) complicated migraine (cerebral vascular ischemia with resulting infarction and cerebral tissue damage); (d) midface migraine (orodental pain, duration 4–72 hours, with nausea, vomiting, phonophobia, photophobia). When diagnosing systemic and intracranial diseases and other disorders that are often a cause of headaches in old age, it is prudent to obtain a CT scan or MRI of the head. A good general rule

is that an unusual initial presentation or a change in symptomatology (other than frequency or intensity) of migraine is a “red flag” that calls for consideration of imaging studies.

Etiology

The fact that most migraine patients have a strong familial history of migraine indicates it has a genetic basis. A detailed discussion of migraine is provided in Chapter 15.

1.5.B Disease 12: cluster headaches and autonomic cephalalgias

Cluster headache (CH) is the most common of the trigeminal autonomic cephalalgias, but this headache group also includes paroxysmal hemicrania as well as short-lasting unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT).

Clinical criteria

Cluster headaches are one-sided retro-orbital, supraorbital, and temporal pain lasting from 15 minutes up to 3 hours when untreated. The headaches often occur at night, waking the patient from a sound sleep with severe pain. With CH, the patients are very agitated during the attack (pacing and head pounding), have no preheadache aura, and, usually, have no associated nausea or vomiting. Among CH patients, the afflicted are mostly men (5–6 times greater prevalence than women), are mostly smokers, and have an age of onset between 20 and 40 years. Cluster headache patients must exhibit, on the affected side, one of the following autonomic signs: conjunctival injection; ptosis; miosis; eyelid edema; flushing or blanching of the face; forehead or nasal sweating; lacrimation; nasal congestion and rhinorrhea. The headaches occur in clusters and will often repeat several times in a 24-hour period (one attack every other day to as many as eight per day). The cluster period frequently lasts for weeks to months and is usually present in specific seasons of the year (greater in winter and spring) and can go into remission for months.^{75,76}

Etiology

The etiology is unknown but a genetic defect is suspected as the basis of this disorder. A detailed discussion of CH and autonomic cephalalgia is provided in Chapter 15.

1.5.C Disease 13: tension-type headaches

Tension-type headaches (TTHAs) are the most common headache in society, with a lifetime prevalence in the general population of 30–78%. Even though this is the most

frequent type of headache, the symptoms are somewhat nonspecific.

Clinical criteria

A TTHA is generally a dull aching bilateral pain that is long lasting and increases slowly during the day to reach peak intensity near late afternoon. It may last for 1–2 days, but it must not occur more often than three times per week or it is not considered episodic. In most cases it is episodic and the pain is located in the suboccipital, temporal, and frontal regions. It is described as a “tight head band” and may be associated with pericranial tenderness. The headache may vary from a short duration to lasting hours, and it may increase slowly during the day to reach peak intensity near late afternoon.⁷⁷ Episodic tension-type headache (ETTH) does not present with migraine signs (e.g., throbbing, photophobia, nausea).

Etiology

Many theories have been put forth to explain the causation and pathogenesis of ETTH. An important and moderately controversial one is the role that pericranial muscle and fascial tenderness plays in the causation or triggering of ETTHs. The questions that need addressing are (1) “Does jaw or facial muscle tension cause an ETTH?” and (2) “If muscle tension is not causative, does muscle nociception from the jaw, face, and neck potentially assist in the triggering process for ETTHs and migraines?” These issues are important to the role that myofascial pain and local myalgia play in the overall headache management program.

1.5.D Disease 14: chronic daily headaches

The group of conditions called chronic daily headache (CDH) includes chronic migraine, chronic cluster headache, hemicrania continua, chronic tension-type headache (CTTH), and new daily persistent headache (NDPH). Migraine, cluster, and tension-type headaches initially present as episodic headaches but they all have the potential to convert or transform into a continuous headache.

Clinical criteria

The criteria for each chronic form are the same as for the episodic form, but to be considered a “transformed acute-to-chronic” headache requires that these disorders exist first in the episodic form and then over time transform to a more frequent or continuous headache. Once they convert, they are called CDH if present 4 or more days per week. Most of the time in CDH, the pain symptoms are present all of the time with only fluctuations up and down in intensity. One

subcategory of the CDH headaches is medication overuse headache, also known as an analgesic rebound headache. The criteria are a steady head or midface pain with frequent–intermittent or continuous multiple pain foci; the headaches improve when analgesics are withdrawn. The most commonly overused medications are over-the-counter (OTC) analgesics, ergotamines, barbiturates, benzodiazepines, and opioids.

Etiology

In addition to using too many analgesics, there are genetic and behavioral factors that likely facilitate the neuropathic conversion from an episodic to a chronic headache. This process is discussed in more detail in Chapter 15.

1.6 Orofacial neurogenous pain: neuralgia, neuropathy, burning mouth

The common mechanism for this subgroup of orofacial pain conditions is trigeminal nerve damage. The trigeminal nerve, if injured or stimulated strongly and long enough, will undergo sensitization. There are also idiopathic neuropathic pain conditions since it is not uncommon that the triggering injury cannot be identified. Regardless of the cause, when neuropathic changes develop, pain can take many forms, such as sharp brief lancinating pains or more continuous sustained pains. The multiple neurogenic diseases that affect the trigeminal nerve are presented in the following subsections.

1.6.A Disease 15: facial pain related to trigeminal neuritis

This multifactorial disorder presents as a continuous burning pain, numbness, tingling, and hypersensitivity along the distribution of the involved trigeminal nerve. When an individual nerve or nerve trunk is inflamed, this is described as a mononeuritis.

Clinical criteria

Mononeuritis pains have an acute onset and the cause is usually obvious based on the examination and history. Those caused by neural compression are also easy to figure out if the source is exogenous (i.e., dental implant) or due to neural abrasion from a compressive osseous growth. The three most common infections to affect the trigeminal nerves are dental abscess, sinus infection, and herpes zoster (shingles). Herpes zoster infection causes small skin vesicles along the distribution of the affected nerve, although vesicles and ulcers can be seen intraorally. Often these vesicles follow

the pain and may present 1–5 days after its onset. If the inflammation occurs in two or more nerve trunks in separate body areas, this is called polyneuritis disorder. The causes of a polyneuritis are diabetes, adverse medication reactions, infection, and immune-mediated neuritis. The symptoms of neuritis, regardless of cause, are a combination of numbness, tingling, weakness, and burning sensation in the affected nerve.

Etiology

Inflammation can be due to neural trauma, bacteria, viruses, or toxins that are damaging the nerve. For mononeuritis, it is most commonly caused by trauma (e.g., fracture, intra-neural injection, third-molar extraction, orthognathic surgical manipulations) or infection (bacterial or viral). Diabetic neuropathy is the most common known cause of polyneuritis and it can produce both an acute (usually reversible) nerve inflammation and chronic (irreversible) neuropathic changes in the trigeminal nerve. The diabetic neuritis patient will complain of numbness, tingling, and weakness in the fingers and toes. Immune-mediated neuritis occurs when the immune system turns against the body and causes an autoimmune reaction (e.g., Guillain–Barré syndrome, chronic inflammatory demyelinating polyneuropathy, neuropathies associated with vasculitis, neuropathies associated with monoclonal gammopathies). Viral-induced polyneuritis is caused by human immunodeficiency virus (HIV), *Cytomegalovirus*, *Poliovirus*, and hepatitis B or C infections causing vasculitic neuropathy. Bacterial-induced polyneuritis includes leprosy, diphtheria, Lyme disease, and trypanosomiasis. Nutritional-imbalance polyneuropathies are caused by deficiency of vitamins B₁₂, B₁ (thiamine), B₆ (pyridoxine), and E. Renal failure polyneuropathy can cause degeneration of peripheral nerve axons as a result of accumulated toxins. Toxin-induced polyneuropathy is caused by alcohol and other toxins (megadoses of vitamin B₆, lead, arsenic, mercury, thalium, organic solvents, and insecticides). Medication-induced neuritis and neuropathies include those caused by vincristine and cisplatin in treating cancer; nitrofurantoin, in pyelonephritis; amiodarone, in cardiac arrhythmias; dideoxycytidine (ddC) and dideoxyinosine (ddI), in acquired immunodeficiency syndrome (AIDS); and dapsone, in leprosy.

1.6.B Disease 16: facial pain related to trigeminal neuroma

Peripheral neural injury will result in trigeminal neuroma formation if the neural injury transects the nerve. The initial injury may only be briefly painful but, as a result of the injury, the nerve forms a chronically painful neuroma.

Clinical criteria

Peripheral neuroma occurs when a nerve is transected, causing sprouting of the proximal nerve trunk to form a bundle of nerves (neuroma) that can be spontaneously active. In the area supplied by the severed nerve there is numbness. The resulting neuroma causes symptoms such as hypersensitivity to light touch and spontaneous pain.⁷⁸

Etiology

The most common locations in the jaw where the nerve is transected are the lingual nerve, inferior alveolar nerve, and auriculotemporal nerve; it is most commonly due to a surgical intervention.

1.6.C Disease 17: facial pain related to trigeminal neuralgia

Trigeminal neuralgia (TN) often presents as severe lancinating pain located in the jaw. Patients present to the dental office with a sharp tooth-region pain and will inappropriately seek dental therapy (endodontics or extraction) as a first line of treatment.

Clinical criteria

Trigeminal neuralgia presents as a sudden, usually unilateral, severe, brief, stabbing, recurrent pain in the distribution of one or more branches of the fifth cranial nerve.⁷⁹ In 1988 the International Headache Society suggested the criteria for the diagnosis of TN, and a complete discussion of this disorder is presented in Chapter 6.⁸⁰

Etiology

While recent evidence points to vascular injury (abrasion) of the trigeminal nerve root inside the cranial vault, this alteration is not usually visible using current imaging modalities. However, in up to 15% of patients there may be an underlying cause such as a benign or malignant tumor of the posterior fossa or multiple sclerosis.⁸¹

1.6.D Disease 18: facial pain related to a chronic trigeminal neuropathy

Local sustained pain in a tooth or gingival site without evidence of local dental or periodontal pathology is labeled as a trigeminal neuropathy. This diagnosis assumes you are now dealing with a neuropathic pain not a pulpal or periodontal disease. Over the years, many different terms have been used to describe dental pain of unknown origin. The most common is “atypical odontalgia”.^{82–87} Once the tooth

is extracted and the pain continues, then the term “phantom tooth pain” is used.^{88–91}

Clinical criteria

The diagnosis of chronic trigeminal neuropathy is essentially a clinical process. The most prominent and sometimes the only symptom that is evident is pain. It is more commonly described as a continuous and spontaneous dull ache localized in a tooth or tooth region. The location may change to an edentulous area or entire parts of the maxilla or mandible. The pain also can be described as burning, sharp, or throbbing. It usually persists for months or years being continuous and persistent, but oscillating in intensity with episodes when the pain is more acute and severe. For a diagnosis of trigeminal neuropathy to be made, other pathologies characterized by tooth pain need to be ruled out. Several have been listed: pulpal toothache, trigeminal neuralgia, myofascial pain, sinusitis, cracked tooth syndrome, and migrainous neuralgia. Probably the most difficult task is to distinguish between trigeminal neuropathy and toothache from pulpal origin. Characteristics that are common to trigeminal neuropathy, but not common to pulpal toothache, are addressed in Chapter 17. A detailed discussion of the persistent orofacial pain due to neuropathy and the appropriate management is presented in Chapter 17.

Etiology

The most accepted theory regarding what causes these pain phenomena is that trauma to the orofacial structures (traumatic injury, periodontal surgery, pulp extirpation, endodontic therapy, apicoectomy, tooth extraction, implant insertion), or even minor trauma (crown preparation, inferior alveolar nerve block) might alter the neural continuity of the tissues, creating sensitization of the peripheral nociceptive nerves. Multiple mechanisms are involved in the pathogenesis of neuropathic pain but the common process is that, following a nerve injury or regional inflammation, the afferent nociceptive fibers become sensitized showing a lower activation threshold and sometimes developing spontaneous ectopic activity as a result of increased expression or redistribution of sodium channels on the axon. This sensitization could easily explain some of the clinical manifestations of oral neuropathic pain such as the clear-cut mechanical or thermal allodynia and persistent spontaneous pain.

1.6.E Disease 19: facial pain related to postherpetic neuralgia

Infection with herpes zoster can lead to the development of continuous pain in the skin or sometimes mucosal tissues of

the mouth during and following the viral infection. Herpes zoster infection strikes millions of older adults annually worldwide and disables a substantial number of them as postherpetic neuralgia. This event is more likely to occur in elderly people, partly because of age-related decline in specific cell-mediated immune responses to varicella-zoster virus.

Clinical criteria

The disease begins with localized abnormal skin sensations, ranging from itching or tingling to severe pain, which precede the skin lesions by 1–5 days. Healing of the skin lesions occurs over a period of 2–4 weeks, and often results in scarring and permanent changes in pigmentation. The cutaneous eruption is unilateral and does not cross the midline. Along with the rash, most patients experience a dermatomal pain syndrome caused by acute neuritis. The neuritis is described as burning, deep aching, tingling, itching, or stabbing pain, and ranges from mild to severe. This pain continues after the rash has healed in as many as 60–70% of patients over the age of 60 and is then considered postherpetic neuralgia, the more frequent and debilitating complication of herpes zoster in the elderly.⁹²

Etiology

The most well established risk factors for postherpetic neuralgia are older age, immunocompromised status, greater severity of acute pain during zoster, and a more severe rash. The patient with postherpetic neuralgia may experience constant pain (described as burning, aching, or throbbing), intermittent pain (described as stabbing or shooting), and stimulus-evoked pain such as allodynia (described as tender). Furthermore, postherpetic neuralgia can impair the elderly patient’s functional status by interfering with basic activities of daily life, such as dressing, bathing, and mobility, and instrumental activities of daily life, such as traveling, shopping, cooking, and housework. The appearance of herpes zoster is sufficiently distinctive that a clinical diagnosis is usually accurate. A direct immunofluorescence assay if needed would be the best and only way (other than culture) to distinguish herpes simplex virus infections from varicella-zoster virus infections. Polymerase-chain-reaction techniques are useful for detecting varicella-zoster virus DNA in fluid and tissues.^{93,94}

1.6.F Disease 20: burning mouth symptoms (not related to hyposalivation)

Continuous pain on the surface of the tongue, mucosa of the lips, and sometimes anterior gingival tissues is commonly

called burning mouth syndrome (BMS; stomatopyrosis) and its variant, burning tongue (glossopyrosis).

Clinical criteria

The sufferers are typically within an age range from 38 to 78 years.^{95,96} Occurrence below the age of 30 is rare, and the female-to-male ratio is about 7:1. Presence of burning sensations is the main complaint, usually described as constant, gradually increasing throughout the day, or intermittent, without any reliable alleviating agents. Diagnosis of BMS is one of exclusion since, like other neurosensory disorders, there are measurable physical signs other than pain. Over two-thirds of BMS patients report a bitter, metallic taste sensation as well as the burning.⁹⁷⁻⁹⁹ The BMS patient typically reports pain onset ranging from 3 years before to 12 years after menopause and approximately 50% of BMS patients complain of dry mouth (xerostomia) but do not exhibit measurable hyposalivation. The pain symptoms of BMS are invariably bilateral, and usually in multiple areas of the mouth. These symptoms often increase in intensity at the end of each day but seldom interfere with sleep. To be considered BMS, the patient should have had the pain continuously for at least 4–6 months. Pain levels may vary from mild to severe, but moderate pain is the most frequent presentation. The pain should be described as daily bilateral oral burning (or painlike) sensations deep within the oral mucosa, unremitting for at least 4–6 months. The symptoms should generally be continuous throughout all or almost all the day and should not interfere with sleep. Like many of the idiopathic diseases, it is a diagnosis made by taking a detailed history and then carefully going through the process to exclude other causes or diseases. The abnormalities that must be excluded are local pathology of the mucosal tissues, nutritional deficiencies (vitamin B₁, B₂, B₆, B₁₂, or B_c [folic acid]), salivary hypofunction, and diabetic neuropathy. If any of these problems are discovered or if oral lesions are present, the diagnosis is not stomatopyrosis. The frequent observation of taste changes and/or sensory–chemosensory dysfunctions in BMS patients suggests that this syndrome could reflect a neuropathic disorder.¹⁰⁰

Etiology

The hypothesized underlying etiology of BMS is an idiopathic small afferent fiber atrophy disorder. The concept that BMS is due to psychogenic or psychosomatic factors has generally not been supported by scientific evidence, and the reverse is the case.^{101,102} A detailed discussion of the burning mouth syndrome and its appropriate management is presented in Chapter 14.

1.7 Facial pain related to chronic oral inflammatory disease

Orofacial pain can arise from a persistent oral inflammatory disease, including blistering diseases, some of which can be extremely debilitating and even fatal. Many of these diseases are autoimmune in nature and may also be associated with certain human leukocyte antigen types. Some bullous diseases have serious sequelae, necessitating early treatment and intervention to prevent further morbidity or mortality. Autoimmune blistering diseases include pemphigus vulgaris, paraneoplastic pemphigus, bullous pemphigoid, cicatricial pemphigoid, dermatitis herpetiformis, linear IgA dermatosis, and graft-versus-host disease (GVHD). Here we focus on pemphigus, pemphigoid, and erosive lichen planus.

1.7.A Disease 21: pemphigus vulgaris

Pemphigus describes a disorder that causes pain because it produces blistering and sloughing of the oral mucosal tissues; it is one of a group of autoimmune blistering diseases that affects the skin and mucous membranes. Pemphigus vulgaris is a serious and deadly diagnosis in that it may be fatal if not treated with appropriate immunosuppressive agents.

Clinical criteria

Characteristically, lesions start in the oral mucosa, followed by the appearance of skin lesions months later. The bullae on the skin may remain localized for 6–12 months, then subsequently become widespread. Rarely, the lesions may arise as a generalized acute eruption. The lesions can be pruritic but are usually painful and accompanied by a burning sensation. Mouth lesions may be tender, preventing adequate food intake, which leads to weight loss. Its onset is slow to develop and the first lesions occur in the oral cavity. As the disease progresses, skin lesions will occur too. On the skin, the bullae last longer before rupture, a feature that makes diagnosis easier. Given the nonspecific nature of the intraoral ulcers, it is not uncommon for progression to skin lesions to occur before the true nature of the disease is appreciated. The microscopic features of intact bullae are usually specific enough to render a diagnosis of pemphigus vulgaris. The most significant of these is the finding of a cleft within the stratum spinosum (intraepithelial clefting) a finding that corresponds to the desmosome destruction. The cells of the stratum basale are unaffected and remain attached by the basement membrane to the underlying connective tissue. This finding creates an unusual appearance that pathologists call tombstoning, in reference to tombstonelike

basal cells. The lesions may be accompanied by weakness and malaise, and a history of epistaxis, dysphagia, and hoarseness. Pemphigus vulgaris is a serious autoimmune systemic dermatologic disease that may affect the oral mucous membrane and skin, manifesting as large fluid-filled rupture-prone bullae; it has a distinctive histologic appearance; anti-inflammatory agents are the only effective therapy and, unfortunately, pemphigus vulgaris has a high mortality rate.

Etiology

The presence of circulating antibodies against keratinocyte cell surfaces suggests that pemphigus is an autoimmune disease. Pemphigus vulgaris is equally prevalent in men and women, and the mean age of onset is between 40 and 60 years. Pemphigus vulgaris is also more common in persons of Jewish and Mediterranean descent.

1.7.B Disease 22: benign mucous membrane pemphigoid

Another painful blistering disorder, but far less morbid than pemphigus, is benign mucous membrane pemphigoid (BMMP); it is also an uncommon autoimmune condition that affects the oral mucosa (gingiva), manifesting as bullae and ulcers. BMMP has a distinctive histology and runs a benign course; improvement usually occurs with anti-inflammatory therapy; pemphigoid, unlike pemphigus, is not fatal.

Clinical criteria

Benign mucous membrane pemphigoid, “pemphigoid” for brevity, was also known as desquamative gingivitis, a designation that recognized its location (gingiva) and its basic lesion (surface sloughing). Recently the name “benign mucous membrane pemphigoid” has been adopted to recognize that it is not fatal (benign), that it occurs only on mucous membranes (mucous membrane), and that it superficially resembles pemphigus vulgaris (pemphigoid). In pemphigoid, autoantibodies attack basement membranes of the gingiva. The destruction of basement membrane proteins damages the attachment of the gingival epithelium to the underlying connective tissue, allowing the epithelium to become detached and form bullae and ulcers.

Etiology

Like other diseases of autoimmune origin, pemphigoid affects females more than males. It does not appear in people

under 40 years of age and, unlike pemphigus, there is little potential for pemphigoid to cause death. However, it is important for patients with this disease to be treated by an ophthalmologist for potential eye involvement.

1.7.C Disease 23: lichen planus

This disorder will cause oral pain if it ulcerates. It is not nearly as dangerous as pemphigus and pemphigoid; it does not form blisters, but it is also an autoimmune disorder affecting the skin and mucosal surfaces of the mouth.

Clinical criteria

Lichen planus (LP) appears as a series of filamentous, white, lacy lines on the inside of the cheeks or other oral tissue and it can cause ulcerative changes in these tissues. LP lesions can occur on other parts of the body, most notably on the skin of the antecubital space (inside of the elbows). Most of these lesions are painless. If the patient has the erosive form of lichen planus, these lesions can be quite painful when eating spicy or sharp-edged foods.

Etiology

Lichen planus is a dermatological autoimmune disease but is often first diagnosed by a dentist due to its characteristic appearance in the mouth. The diagnosis of LP is confused in some patients who develop a lichenoid mucositis due to exposure to a local chemical in the mouth to which the patient may be sensitive (e.g., cinnamon). It is especially associated with certain antihypertensive drugs, nonsteroidal anti-inflammatory drugs (NSAIDs), tetracycline, and several sulfonamides, as well as a number of illegal drugs. The condition often improves with the cessation of the offending drug. The condition is more of a severe nuisance than a disability.

1.7.D Disease 24: mucositis

Mucositis is a very painful mucosal disease most commonly seen after radiotherapy and chemotherapy.

Clinical criteria

When a patient exhibits a painful inflammation and ulceration of the oral mucous membranes, it is called oral mucositis (OM). When severe it can lead to significant complications, including dysphagia, malnutrition, electrolyte imbalance, systemic infection, and death. OM manifests initially as painful, erythematous mucosa that transforms

into more painful ulcerations.¹⁰³ The nonkeratinized mucosa of the oral cavity is typically affected with chemotherapy and radiotherapy. Lesions typically occur 1–2 weeks following chemotherapy or after radiotherapy (measured in grays [Gy]) greater than 30 Gy. The healing period is usually 2–4 weeks after cessation of either therapy. The extent and severity depend on the dosage and regimen of the therapy.^{104,105} One major problem with severe mucositis is that it can cause patients to terminate their radiotherapy or chemoradiotherapy.

Etiology

The most common reason for a severe oral mucositis is as a side effect of chemotherapy and radiotherapy treatment for cancer.^{106,107} The likelihood of oral mucositis depends on the chemotherapy regime being used.¹⁰⁸

1.7.E Disease 25: other chronic (nonmalignant) ulcerative conditions of the mouth

Members of this group of oral inflammatory and ulcerative problems are chronic and painful. The pain seen with an oral ulcer is because it is an open sore inside the mouth caused by a break in the mucous membrane or the epithelium on the lips or surrounding the mouth.

Clinical criteria

These nonmalignant ulcerative disorders present as ulcers or inflammation of the mucous membranes inside the mouth. Distinction of a Behçet's ulcer from a chronic aphthous ulcer is difficult. Chapter 14 provides more detail on these problems.

Etiology

There are many reasons the tissue ulcerates, including trauma (physical or chemical) and infection from microorganisms and viruses. Various medical conditions and medications cause mucositis. After excluding the previously discussed vesiculobullous conditions of pemphigus vulgaris, benign mucous membrane pemphigoid, and ulcerative lichen planus, some of the medical conditions associated with chronic oral ulcers include Behçet's disease, celiac disease (also known as gluten sensitivity), graft-versus-host disease, Crohn's disease, ulcerative colitis, lupus erythematosus, and neutropenia.

1.7.F Disease 26: cancer pain in the jaw

The painful symptoms that are associated with a neoplastic invasion of the trigeminal nerve and/or a metastatic bone

lesion of the jaw will present as pain that produces symptoms that mimic neuritis, neuralgia, and neuropathy or simply induce sensory loss if a cancer invades a nerve sheath or root. There will also be bone pain when patients have metastatic jaw bone lesions (e.g., multiple myeloma).

Clinical criteria

Orofacial pain not only has been the initial complaint of primary oral cancer patients but also has been reported to be one of the earliest indicators of recurrent cancer. A recent study described 12 patients who experienced recurrence of primary head and neck cancers that were preceded by severe orofacial pain.¹⁰⁹ When the pain was reported, the authors described their patients as not demonstrating other evidence of malignant disease despite clinical examination, plain radiography, CT scans, and even MRIs of the area.

Etiology

The most common cancers associated with the trigeminal nerve are posterior tongue–lateral pharyngeal cancer, causing pain in the lingual nerve, and cancer of the nasopharynx invading the infratemporal region and affecting the trigeminal nerve as it exits the foramen ovale.

1.8 Facial pain related to oral motor disorders

Orofacial “movement disorders” can be broadly classified as either “hypokinetic” or “hyperkinetic” conditions. The hypokinetic disorders (e.g., Parkinsonian rigidity) are not usually associated with pain. In contrast, the hyperkinesias spasm and trismus can produce pain. They can be further subclassified into the stereotypic dyskinesias, tremors, dystonias, tics, myoclonus, and choreas. Some might even add the parasomnias (e.g., habitual tooth clenching, sleep bruxism) and secondary spasms to this group. With the exceptions of tooth clenching and sleep bruxism, involuntary oral movement disorders are more common in older age. For example, Bourgeois and colleagues examined 270 elderly subjects in a residential nursing facility for dyskinesias, both spontaneous and drug induced.¹¹⁰ They reported that females exhibited twice the likelihood of having a dyskinesia (27%) as males (12%). Within those who had dyskinesia, they were two-thirds of the time related to neuroleptic medications and one-third of the time of spontaneous onset. While some oral motor disorders do induce pain, in general, the link between pain and abnormal motor function is not strong. The hyperkinetic disorders that produce pain are discussed in the following subsections.

1.8.A Disease 27: dyskinesia

Orofacial dyskinesia, which usually presents more as a dysfunction than as a pain disorder, can be drug related or can occur spontaneously. The prevalence rate of drug-induced dyskinesia (tardive form) is approximately 15–30% in patients who receive long-term treatment with neuroleptic medications.¹¹¹ For spontaneous dyskinesias, the prevalence rate is 1.5–38% of elderly individuals, depending on age and definition.

Clinical criteria

The word “dyskinesia” means abnormal movement and is used to describe a continuous repetitive movement disorder of the jaw, lips, or tongue that can be drug induced (tardive) or can occur without clear cause (spontaneous). By definition, orofacial dyskinesias are involuntary, repetitive, stereotypical movement of the lips, tongue, and sometimes jaw during the day.^{112,113} Sometimes the dyskinesia is medication induced (called tardive) or it can occur spontaneously. The spontaneous form of dyskinesia often affects the elderly. The tardive form of dyskinesia typically occurs in mentally ill patients who have had long-term exposure to medications used to treat the mental illness.¹¹⁴ By definition, tardive dyskinesia requires at least 3 months of total cumulative drug exposure, which can be continuous or discontinuous. Moreover the dyskinesia must persist more than 3 months after cessation of the medications in question. Most dopamine receptor antagonists cause oral tardive dyskinesia to one degree or another. The typical antipsychotics and in recent years even the atypical antipsychotics, including clozapine (Clozaril™), olanzapine (Zyprexa™), and risperidone (Risperdal™), have been reported to cause both tardive dystonia and tardive dyskinesia. No adequate epidemiologic data exist regarding whether any particular psychiatric diagnosis constitutes a risk factor for the development of tardive reactions to medications, but the duration of exposure to antipsychotics required to cause tardive reaction is from months to years. Unfortunately, there is no more specific diagnostic test for dyskinesia other than clinical observation and history.

Etiology

Risk factors for the development of tardive dyskinesia are older age, female gender, and the presence of affective disorders.¹¹⁵ Elderly women are twice as likely to develop the disorder.¹¹⁶ When this disorder is associated with a drug use, the medications most commonly implicated are the neuroleptic medications now in widespread use as a component of behavioral therapy. There are isolated reports in the literature that implicate dental treatment as an etiologic factor for

the onset of spontaneous orofacial dyskinesia. Exposure to antipsychotics need not be long, and a minimum safe period is not apparent. This duration of neuroleptic exposure seems to be shorter for women. A longer duration of exposure to neuroleptics does not correlate with the severity of the reaction. Treatment of orofacial dyskinesia is largely with medications, which unfortunately are not highly successful.

1.8.B Disease 28: dystonia

Dystonia usually presents more as a dysfunction than as a painful disorder; however, if the dystonic contraction is strong and frequent enough, pain can result.

Clinical criteria

Dystonia presents as an involuntary briefly sustained contraction of muscles. When the dystonia involves only one or two areas of the body it is labeled a focal dystonia. For example, some patients exhibit an involuntary repetitive contraction of the orbicularis oculi muscles, which produces eye closure. This disorder has been called blepharospasm. If the cervical muscles (usually sternocleidomastoid and trapezius) contract, this is called a torticollis. There are several focal dystonic patterns involving some combination of jaw, neck, tongue, and perioral muscles and they are described as focal orofacial, orolingual, oromandibular, and cervical dystonia.¹¹⁷ Frequently the patient with a significant oromandibular dystonia will have compromised mastication and be unable to function with a removable dental prosthesis (especially mandibular full dentures). Some of the severe orofacial dystonias may actually create such difficulty that patients will be unable to eat and may lose weight. If the dystonia strongly affects the tongue musculature, this may compromise the patient’s ability to speak clearly. If the patient exhibits a combination of blepharospasm and jaw opening dystonia, this has been labeled Meige’s syndrome.^{118,119}

Etiology

Most dystonia cases are spontaneous in origin.

1.8.C Disease 29: bruxism

Bruxism is a sleep-related motor disorder that if severe can induce pain and dysfunction in the jaw structures. These changes include broken or worn teeth, TMJ derangement, TMJ arthritis, and jaw muscle pain. Between 6% and 20% of the population has been reported to exhibit bruxism. This disorder is more common in children (14%), and then generally decreases after the age of 50 years.¹²⁰ The distinction between tooth grinding and tooth clenching is not clear-cut

but the latter is thought to occur more frequently, and more in women than in men.

Clinical criteria

It is somewhat difficult to confirm or refute the presence of bruxism or clenching since patients often do not know they are grinding and certainly may not know they are clenching. Moreover, there has been no population-based study involving a large number of patients where polysomnography has been performed, and unfortunately dental wear or attrition is not always a good indicator of current bruxism or clenching.

Etiology

The etiology of bruxism is unknown.

1.8.D Disease 30: habitual parafunction and spontaneous and secondary hypertonicity

Oral parafunction includes tooth clenching, habitual cheek chewing, and gum chewing as well as generalized jaw and neck muscle tension. Sometimes patients can be seen repeatedly clenching their masseter muscles during the clinical interview.

Clinical criteria

From a scientific perspective, little definitive information exists on the problem of tooth clenching because most habitual behaviors are not deliberate acts, but are performed at the edge of consciousness. What is known is that habitual tooth clenching for nonfunctional purposes is a common behavior, it appears to be of short duration and low amplitude, and it is exhibited several times per hour in healthy control subjects of both genders. The daytime habitual jaw muscle activity reported by nonpain, healthy controls suggest the peak amplitude of these activities probably ranges between 5% and 20% of maximum voluntary contraction and the duration of tooth contact periods is probably low (less than 5 seconds per event). However, in myogenous pain subjects, this behavior increases substantially in frequency, duration, and amplitude; at present we do not have adequate data to understand or describe the range of values (frequency, duration, and amplitude) exhibited by these subjects.^{121,122} Based on self-reported tooth contact data, patients with muscle pain may exhibit approximately 4 times more tooth-contact-related activities than controls without pain. While experimental studies of clenching at high or low level force can produce pain during and for a short while after the clenching task, they do not support the idea that clenching

alone produces any long-term pain, and the process of myogenous pain must be more complex than simple clenching-induced muscle injury. Some patients report chronic muscle stiffness (often unilateral) that has not been classified as a specific entity. This sensation would involve small changes in stiffness and muscle tone and not be easily distinguished from background normal activity, but any clinician who examines the jaw and neck muscles in patients with chronic pain will not infrequently discover that some patients have elevated firmness and reduced mobility of their muscles.

Etiology

Like bruxism there is no specific etiology for this problem. In the orofacial region, an example of this would be what is described as elevated masticatory and cervical muscle stiffness.^{123,124} The obvious neurologic conditions (e.g., stiff-man syndrome) that include muscular rigidity are usually thought to be due to an elevated level of extrapyramidal activity.¹²⁵ Moreover, normal aging has also been associated with some development of spontaneous muscle activity,¹²⁶ as have primary psychiatric disorders such as major depression and schizophrenia. There are times that this elevated jaw muscle stiffness is centrally generated and involves medication-induced alteration in extrapyramidal motor neuron activity. The drug class most often reported is chronic exposure to neuroleptic drugs.¹²⁷ Another class of medication more recently associated with motor side effects is the serotonin selective reuptake inhibitors (e.g., paroxetine, fluoxetine), which are discussed in both Chapters 8 and 19.

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