Orofacial Pain
A Guide to Medications and Management

Editors

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Frequently asked questions and answers, recommendations for medication prescribing, and figures and tables are available for download at www.wiley.com/go/clarkdionne.
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This book begins by describing the 30 most common conditions that a dentist or physician may encounter when patients present with orofacial pain and dysfunction (not due to a dental infection). Chapter 1 provides a short description of the clinical characteristics of these 30 conditions. The majority of these conditions are also described in greater detail along with recommendations about the best evidence-based treatment approach in Chapters 12 through 20. Chapter 2 introduces the 60 most common medications that are used by clinicians who treat orofacial pain. These medications and how they are used are then described in detail in Chapters 3 through 11.

In all instances we have attempted, where possible, to collect and present the scientific evidence that supports or refutes the use of a specific medication for a specific condition. Obviously this book has a clear focus on medications because so many of the pain disorders that occur in the orofacial region are treated with medications. However, this focus should not diminish the fact that there are several other options that should be used in combination with medications, including behavioral (psychosocial) and various physical medicine methods. These interventions can help a patient gain a sense of control over his or her pain and should be introduced early in the course of pain management.

The creation of a body of work such as this takes a good deal of time and effort. First we want to thank our spouses for the support and tolerance they have given us during this effort. Next we thank all of the chapter authors, all of whom are good friends and colleagues who trusted us to produce a book that they would be proud to have contributed to. Finally, at the end of Chapters 3–20 we provide a few key recommendations based on the content covered in these chapters. We have put all of the tables, figures, and end-of-chapter recommendations in a website maintained by Wiley (our publisher) for anyone who has enough curiosity to go to the website (www.wiley.com/go/clarkdionne). As an added benefit we have included a set of 187 questions and answers that should be valuable to the readers. We hope this website is considered a valuable addition to the book.

Glenn T. Clark
Raymond A. Dionne
Chapter 1

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

Glenn T. Clark, DDS, MS

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. 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

<table>
<thead>
<tr>
<th>Disease</th>
<th>Distinguishing clinical features</th>
</tr>
</thead>
</table>
| 1  Myalgia                    | 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  
|                               | Note: 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. |
| 2  Myofascial pain            | 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  
|                               | Note: Myalgia is labeled *myofascial pain* only when taut bands and trigger points are present. |
| 3  Fibromyalgia               | 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  
|                               | Note: Myalgia is labeled *fibromyalgia* only when these criteria are met. |
| 4  TMJ DDWR                   | 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 |
| 5  TMJ DDNR                   | 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  
|                               | **DxTest:** MRI shows DDNR in both closed and open positions |
| 6  Local TMJ arthritis        | Subjective pain in preauricular area aggravated by function  
|                               | Pain that can be replicated by TMJ capsule palpation  
|                               | Joint motion often produces crepitation sounds  
|                               | **DxTest:** erosive or remodeling-type joint-surface changes on CT imaging |
| 7  Polyjoint OA affecting the TMJ | Subjective pain in preauricular area aggravated by function  
|                               | Pain that can be replicated by TMJ capsule palpation  
|                               | Joint motion often produces crepitation sounds  
|                               | **DxTest:** erosive or remodeling-type joint-surface changes on CT imaging  
|                               | **DxTest:** negative serology for autoimmune markers of rheumatoid disease |
| 8  Rheumatic arthritis affecting the TMJ | 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  
|                               | **DxTest:** erosive or remodeling-type joint-surface changes on CT imaging  
|                               | **DxTest:** positive serology for autoimmune markers of rheumatoid disease |
| 9  Temporal arteritis         | New headache pain of a constant nature  
|                               | Tender, thickened, and pulseless scalp vessels  
|                               | **DxTest:** positive serology for autoimmune markers of an inflammatory disease  
|                               | **DxTest:** confirmed by blood vessel biopsy showing giant-cell infiltrate  
|                               | Note: Markers are elevated ESR and a C-reactive protein. |
| 10 Trigeminal sensory neuropathy | Unilateral or bilateral sensory loss of one or more trigeminal nerve divisions  
|                               | Usually, also presence of pain in these same areas  
|                               | **DxTest:** negative MRI for pathology involving the CNS or trigeminal nerve  
|                               | **DxTest:** confirming diagnosis of associated CTD  
|                               | Note: Most commonly associated with an autoimmune CTD such as mixed or undifferentiated CTD, scleroderma, Sjögren’s syndrome, or lupus erythematosis. If so, there may also be complaints of Raynaud’s phenomenon, polyjoint arthritis, and sometimes muscle weakness. |
| 11 Migraine                   | Unilateral headache location  
|                               | Pulsatile severe headache that lasts multiple hours  
|                               | Nausea associated with the headache pain  
|                               | Photophobia and phonophobia associated with headache pain  
|                               | **DxTest:** negative MRI for pathology involving the CNS  
<p>|                               | Note: Pain episodes may be preceded by aura such as “flashing lights or dizziness.” |</p>
<table>
<thead>
<tr>
<th>Disease</th>
<th>Distinguishing clinical features</th>
</tr>
</thead>
</table>
| 12 Cluster headaches | 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  
**DxTest:** negative MRI for pathology involving the CNS  
**Note:** Headache must be associated with ipsilateral autonomic signs, including conjunctival injection, ptosis, miosis, eyelid edema, facial flushing or blanching, forehead sweating, icrimation, nasal congestion, and rhinorrhea. |
| 13 Tension-type headaches | 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 |
| 14 Chronic daily headaches | Continuous or very frequent headache (4 or more days per week)  
Maybe with clinical features of both migraine and tension-type headache  
**DxTest:** negative MRI for pathology involving the CNS |
| 15 Acute trigeminal neuritis | Injury- or infection-associated acute onset numbness or tingling  
Burning sensation in the affected nerve  
**DxTest:** CTs and MRI needed to check for pathology involving the involved nerve |
| 16 Trigeminal neuroma | 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 |
| 17 Trigeminal neuralgia | 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  
**DxTest:** MRI of trigeminal nerve and brain  
**Note:** In most (90%) cases MRI will not show pathology involving the trigeminal nerve; other cases will show CNS tumor or other pathology. |
| 18 Chronic trigeminal neuropathy | Constant dental and gingival pain in a very focal oral region  
Usually, pain of unknown origin  
**DxTest:** negative radiographic finding indicative of pulpal pathology  
**DxTest:** negative endodontic thermal testing indicative of pulpal pathology |
| 19 Postherpetic neuralgia | 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  
**Note:** Pain and preceding ulcerative lesion can be intraoral if it involves the V3 division. |
| 20 Burning mouth (not related to hyposalivation) | Constant burning sensation of the anterior tissues of the mouth  
Pain often increasing throughout the day  
No clinically discernable oral pathology on examination |
| 21 Pemphigus vulgaris | Blistering diseases of the skin and mucous membranes of the mouth  
**DxTest:** Biopsy will confirm the diagnosis of pemphigus. |
| 22 Benign mucous membrane pemphigoid | Blistering diseases of the skin and mucous membranes of the mouth  
**DxTest:** Biopsy will confirm the diagnosis of BMMP. |
| 23 Erosive lichen planus | Filamentous, white, lacy lines on the cheek or other oral tissues  
Erythema and ulceration of the mucosal tissues  
**DxTest:** Biopsy will confirm the diagnosis of LP.  
**Note:** LP becomes painful when it turns erythematous and erosive. |
| 24 Mucositis | Painful inflammation and ulceration of the mucous membranes  
**Note:** 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. |
| 25 Ulcerative disease of the mucosa | Ulcerative or severe inflammation of the mucous membrane  
**DxTest:** Biopsy will confirm the diagnosis of a nonspecific ulcerative disease.  
**Note:** Positive findings for the causative systemic or allergic disease |
| 26 Cancer pain in the jaw | Trigeminal sensory disorder with variable presentation  
Neural deficit may be numbness or pain (continuous or episodic)  
**DxTest:** Positive MRI for cancer affecting trigeminal nerve |
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 currently 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.

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

<table>
<thead>
<tr>
<th>Disease</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Primary and secondary myalgia (all types)</td>
<td>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)</td>
</tr>
<tr>
<td>2 Myofascial pain (all types)</td>
<td>Common etiologies same as for myalgia, Taut bands and trigger points, suggesting localized neuronal sensitization in muscle</td>
</tr>
<tr>
<td>3 Chronic widespread pain and fibromyalgia</td>
<td>Common etiologies same as for myalgia, Multiple pain sites, allodynia, and mechanical hyperalgesia, suggesting central sensitization, Unknown genetic susceptibility that predisposes to fibromyalgia</td>
</tr>
<tr>
<td>4 TMJ DDWR</td>
<td>Traumatically altered discal ligaments that attach it to the condyle, Parafuction, Joint hypermobility, Acute macrotrauma to jaw</td>
</tr>
<tr>
<td>5 TMJ DDNR</td>
<td>Common etiologies that cause DDNR same as for DDWR</td>
</tr>
<tr>
<td>6 Localized TMJ arthritis</td>
<td>Trauma (either macrotrauma or repetitive microtrauma), A prior DDNR in the involved joint</td>
</tr>
<tr>
<td>7 Polyjoint osteoarthritis and TMJ</td>
<td>Idiopathic (but most likely genetic), Secondary polyjoint arthritis (e.g., psoriasis)</td>
</tr>
<tr>
<td>8 Rheumatic arthritis and TMJ</td>
<td>Autoimmune induced</td>
</tr>
<tr>
<td>9 Temporal arteritis</td>
<td>Giant-cell inflammation due to autoimmunity</td>
</tr>
<tr>
<td>10 Idiopathic trigeminal sensory neuropathy</td>
<td>Autoimmunity (seen with various CTDs such as Sjögren’s syndrome, undifferentiated and mixed CTD, and scleroderma)</td>
</tr>
<tr>
<td>11 Migraine</td>
<td>Genetics</td>
</tr>
<tr>
<td>12 CH and autonomic cephalalgias</td>
<td>Genetics</td>
</tr>
<tr>
<td>13 Tension-type headaches</td>
<td>Stress</td>
</tr>
<tr>
<td>14 Chronic daily headaches</td>
<td>Neuronal sensitization due to frequent episodic headaches, Genetic factors likely, Stress factors likely, Analgesic medication overuse may play a causative role in CDH.</td>
</tr>
<tr>
<td>15 Facial pain related to trigeminal neuritis</td>
<td>Viral-induced neural inflammation (e.g., HIV, <em>Cytomegalovirus</em>, <em>Poliavirus</em>, 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 B12, B1 [thiamine], B6 [pyridoxine], and E), Renal-failure-induced polyneuropathy, Toxin-induced polyneuropathy (e.g., alcohol and other toxins), Medication-induced neuritis (e.g., vincristine and cisplatinum; ddC and ddI in AIDS; and dapson, used to treat leprosy)</td>
</tr>
<tr>
<td>16 Facial pain related to trigeminal neuroma</td>
<td>Surgical or trauma-induced nerve trunk transection</td>
</tr>
<tr>
<td>17 Facial pain related to trigeminal neuralgia</td>
<td>Vascular compression, Multiple sclerosis, Acoustic neuroma (tumor) induced compression, CNS neoplasia</td>
</tr>
<tr>
<td>18 Facial pain related to a chronic trigeminal neuropathy</td>
<td>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)</td>
</tr>
</tbody>
</table>
Table 1.2  (Continued)

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

Table 1.3  Confirmatory or exclusionary diagnostic methods

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

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.
postsurgical pain; and pain caused by accidents and burns. Whether it is cancer pain or noncancer pain, opioid treat-
ment of pain is common. For example, 73% of hospitalized
patients receiving opioid therapy still reported moderate dis-
tress and 75% of postsurgical patients were in either moder-
ate or marked distress.3,4 When you look more closely at
elderly patients (defined as over the age of 65), the preva-
lence 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 fibro-
myalgia syndrome (FMS) are common chronic pain prob-
lems that predominantly affect middle-aged women.8–11
While local myalgia and myofascial pain are more preva-
ient in the middle aged, fibromyalgia increases with age
and is substantially more evident in the elderly popula-
ion. Each of the myalgia subtypes is discussed in the
following subsections. A detailed discussion of these dis-
orders 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 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. 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. The most likely secondary jaw and cervical motor activation occurs with a painful arthritis or internal derangement of the TMJ. However, these reactions are also likely to occur with acute pulp 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. 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. However, inclusion of the last criterion is not endorsed by all since it is not a reliably present physical finding.

**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. 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. 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. 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-
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.

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 arthritic 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 arthritic 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%.51 Osteoarthritic 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 erythematosis, 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 erythematosis 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.\textsuperscript{55–58} Rheumatoid arthritis is found in the temporomandibular joint in more than 50% of adults and children with RA,\textsuperscript{60} 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, \textit{HLA-DR4} and \textit{HLA-DR1}, 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.\textsuperscript{60} The mean age of onset for temporal arteritis is 70 years and it is rare in people less than 50 years of age.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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. The source of the underlying neural dysfunction is thought to be autoimmune because of this association. 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. 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. 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. 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 pulsetile 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.

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...
The 30 most prevalent chronic painful conditions

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 cisplatinum in treating cancer; nitrofurantoin, in pyelonephritis; amiodarone, in cardiac arrhythmias; dideoxycytidine (ddC) and dideoxyinosine (ddl), 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.\(^7^8\)

**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.\(^7^9\) 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.\(^8^0\)

**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.\(^8^1\)

**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”.\(^8^2–8^7\) 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 orodontal 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.\textsuperscript{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.\textsuperscript{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 measureable 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 Bc [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.\textsuperscript{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.\textsuperscript{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...