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When we published the first edition of this book, we felt there was a true need to bridge the fields of orofacial pain and headache with a textbook that could integrate the knowledge from both fields. While at the time we questioned the necessity for an additional book in the area of craniofacial pain, this worry soon became unnecessary. The book was accepted with great enthusiasm, and in 2009, the British Medical Association highly commended the first edition as one of the best books published in the medical field. Reviews were complimentary: “This textbook is a joy to read,” proclaimed a 2008 review in the *Journal of Orofacial Pain* (Quintessence Publishing). The reviewers praised our success in integrating the broad topics of orofacial pain and headache. Furthermore, we were glad to see that this most important journal in the orofacial field changed its name in 2014 from the *Journal of Orofacial Pain* to the *Journal of Oral & Facial Pain & Headache*, pointing to what was already obvious to us; these two fields must continue to be integrated.

Integration ensures that we are consistent and relate all regional craniofacial pains to each other, thereby presenting the wider picture of craniofacial pain syndromes and the overlap between primary headaches and primary orofacial pain entities. Truly, the years since the first edition was published have seen some integration of the two fields, particularly through close collaboration between dental and medical professionals for the preparation of the 2013 International Classification of Headache Disorders. In spite of this, the International Headache Society’s classification system does not yet adequately cover all currently accepted orofacial pain entities. Therefore, we added the recently reviewed classification of the Diagnostic Criteria for Temporomandibular Disorders (DC-TMD) and drew from our wide experience in the field. Certainly, in the diagnosis of acute dental and otolaryngological pain, we have continued to stress the importance of accurate, evidence-based diagnosis. This may seem oversimplistic at first, but consider the reports of misdiagnosis of cluster headache, paroxysmal hemicrania, migraine, and trigeminal neuralgia as dental pains or sinus headaches. The results to our patients are often devastating and unnecessary. Clearly as a profession, we still have hurdles to overcome.

While we preserved the well-structured format of the last edition, we updated the chapters to reflect current knowledge and added a new chapter on orofacial pain and sleep, as data continues to point to their interconnection. We have also made this edition friendlier to clinicians. In many chapters, we first address the clinical picture and treatment strategies and follow with a discussion of the underlying mechanisms. In addition, the design has been updated, and we have found that the esthetic layout of the present edition, made possible by the excellent editors and production staff of the Quintessence Publishing house, makes the excursion through the pages of this book a most pleasant experience.

Finally, as the first edition was highly praised, we felt that we were not in a position to disappoint our faithful readers and had to keep to the high standards that are expected. We very much hope we have succeeded in this mission.
For many years, the area of orofacial pain was completely dominated by the concept that most facial pains were due to “disturbed function” of the temporomandibular joint (TMJ).

This was an approach established by an otolaryngologist named James B. Costen who linked etiology to derangements of the dental occlusion; facial pain was thus handed over to dentistry. As a profession, we enthusiastically adopted the treatment of facial pain but have for many years concentrated our efforts on a mechanistic approach to treatment. These events essentially segregated facial pain from headache and, in effect, from mainstream medicine. As a result, ideal conditions were established in each of the two disciplines for the development of different approaches to the understanding of mechanisms and therapy of craniofacial pain. However, as our understanding of pain mechanisms, and in particular chronic pain, developed, it became clear that facial pain has underlying neurophysiologic mechanisms common to headaches and other body areas. Masticatory muscle pain was examined in light of other regional muscle pains, and management of the TMJ was related to, and brought in line with, basic orthopedic principles. Most importantly, features of some facial pain entities are very similar to those of some headaches. Examples include masticatory myofascial pain and tension-type headache and a facial equivalent of migraine.

The dental profession has been slow in adopting medically based classification and approaches to therapy. In a similar fashion, the medical profession has been very resistant to incorporating established facial pains into current classifications; temporomandibular disorders are a prime example and currently unrecognized by the International Headache Society.

One may correctly claim that toothache is unique, but is it really? On a mechanistic level, pulpitis is an inflammatory process within a confined space—not very different from the inflammatory process of migraine confined within the skull. Indeed, we believe that migraine-like mechanisms exist within the pulp chamber mimicking pulpitis, in the paranasal sinuses imitating sinusitis, and in other confined cranial structures causing atypical symptomatology. In each of these cases, anti-migraine medications are the correct treatment.

Clearly the task required is integration of knowledge in this anatomically dense region, traditionally divided between many medical disciplines. Based on our extensive clinical experience with patients suffering from orofacial pain and headache as well as our thorough understanding of pain mechanisms specific to the trigeminal system, we feel that we are well equipped to fulfill this task. This textbook therefore deals with oral and facial pain as well as with headaches and aims to integrate the knowledge across these disciplines. We hope we have succeeded.

We appreciate the contribution of our teachers, colleagues, and students. Throughout our professional lives, we have interacted with many professionals worldwide, and each has enriched our understanding of pain mechanisms and our clinical knowledge. Being in the “business” of teaching, both undergraduates and residents, we have been consistently challenged by curious students with difficult questions. These have kept us up to date and enabled us to re-examine and reassess the way orofacial pain is understood and taught.

Last but not least, our warm gratitude and appreciation to our families for bearing with us through the long process of preparing, writing, editing, and publishing this book.
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The Diagnostic Process

Yair Sharav, DMD, MS
Rafael Benoliel, BDS

Diagnosis and treatment of orofacial pain is a complex process compounded by the density of anatomical structures and the prominent psychologic significance attributed to this region. Management of orofacial pain thus demands the services of clinicians from various specialties, such as dentistry, otolaryngology, ophthalmology, neurology, neurosurgery, psychiatry, and psychology. Complex referral patterns to adjacent structures are common in orofacial pain and, indeed, one person’s headache is another person’s facial pain. In clinical practice, the two types of pain are often intimately related. Consequently, a patient with orofacial pain may wander from one specialist to another to try to find adequate help.

The second edition of this textbook continues to integrate the issue of orofacial pain with headache through contributions from practitioners in different disciplines, all of whom have extensive clinical experience and a thorough understanding of pain mechanisms specific to the trigeminal system. Accordingly, the authors address all regional craniofacial pains together and aim to present a wider picture of orofacial pain syndromes, including the overlap between primary headaches and primary orofacial pain entities. Many patients with chronic orofacial pain suffer primary headache variants in the orofacial region, and a lack of familiarity with these syndromes is likely a factor in misdiagnosis by dental practitioners and medical specialists. Other patients may suffer from primary orofacial pain entities that remain unclassified by the International Headache Society (IHS) and are unknown to neurologists, otolaryngologists, other medical practitioners, and even dentists.1–3 The integration of headache and orofacial pain classifications is of paramount importance. In the past, about half of the patients in tertiary-care craniofacial pain clinics were labeled as “idiopathic” or “undiagnosable” when the previous IHS classification was applied.2–5 The hope is that the current classification, which has witnessed a novel collaboration between orofacial pain and headache specialists, will improve the situation.

Moreover, there is considerable overlap in the clinical presentation of headaches, such as tension type with regional myofascial pains of the face, and generalized pain syndromes, such as
fibromyalgia (see chapter 8). The relationship between isolated facial neurovascular pain (see chapter 10) and migraines or trigeminal autonomic cephalalgias remains unclear and is not accounted for by the recent IHS classification. Furthermore, a growing patient population has chronic craniofacial pain from trauma associated with traffic accidents or from invasive dental procedures, such as dental implants, which demands a multidisciplinary approach. This book bridges the gap between medically trained headache and dentally trained orofacial pain specialists. It will be useful to readers at different stages of their careers—undergraduate students, residents, practitioners, and dental and medical pain specialists.

Epidemiology: The Silent Crisis

Statistics from the United States indicate that 100 million adults suffer from chronic pain at an estimated annual cost of around $600 billion—higher than the cost for heart disease, cancer, or diabetes. However, chronic pain is a worldwide epidemic that has been termed “the silent crisis.” Examining relevant prevalence estimates gives important insight into the scope of the problem. Orofacial pain, of which about 10% is chronic, affects around a quarter of the general population. Painful temporomandibular disorders (TMDs) are quite prevalent; 4.6% of the population reports this type of pain (6.3% of women, 2.8% of men). This finding is in agreement with the 2009 National Health Interview Survey, which found that 5% of adults reported pain in the face or jaw over a 3-month period. Persistent facial pain, which has a reported incidence of 38.7 per 100,000 person-years, is more common in women and increases with age. Syndromes identified included trigeminal neuralgia and cluster headache, which are the most common forms. Paroxysmal hemicrania and glossopharyngeal neuralgia were among the rare syndromes. Clearly, orofacial pain is more prevalent than previously thought.

Therefore, diagnosis and management of orofacial pain and headache have become important subjects in medicine and dentistry. Both acute and chronic presentations may be benign or may signify serious underlying disease. The emphasis of this book is on the four major clinical families of orofacial pain: acute orofacial, neurovascular, musculoskeletal, and neuropathic (see chapters 6 and 8 to 12). In these chapters, the current etiology, diagnosis, and treatment are reviewed. The book includes many case presentations that are largely virtual, that is, created by integrating data from a number of cases seen in the clinic; thus, any resemblance to specific cases is purely coincidental. They are real, however, in that they reliably duplicate the type of cases seen in orofacial pain clinics. Typical textbook cases are rare, and each relevant section includes information related to the changes in presentation that may cause diagnostic confusion. Atypical cases may be difficult to manage; many have superimposed trauma and consequent neuropathic pain. Some of these cases present patients with a history of misdiagnosed acute pains in the orofacial region who have undergone repeated and unsuccessful interventions that slowly escalated and resulted in dental extractions and surgeries. Accurate diagnosis of acute dental and orofacial conditions is therefore essential (see chapter 6). The importance of acute and chronic otolaryngologic syndromes in the differential diagnosis of facial pain, particularly migraines and cluster headache, is paramount (see chapter 7). The growing number of older, often medically compromised, patients with orofacial pain deserves special attention (see chapter 14): Is orofacial pain in these patients related to their medical condition? Although this is essentially a clinical book, anatomy and neurophysiology are covered in a manner specifically relevant to the topic of orofacial pain (see chapter 2).
optimal patient care, and some of the best drugs offer notable relief for only a fraction of our patients, with some having disturbing side effects. Many patients inquire about complementary and alternative medicine and often actively search out these practitioners independently (see chapter 17). Neurosurgical approaches, including neuromodulation, remain relevant options for selected syndromes (see chapters 11 to 13). No diagnosis and treatment of orofacial pain would be complete without understanding its emotional undercurrents and having a thorough knowledge of its psychologic aspects and treatment possibilities, which are covered in chapter 4. A novel and welcome addition to this second edition is a description of the interactions between sleep and orofacial pain and headaches.

Chronic Pain Is a Disease

Pain is a multifaceted experience with physical, cognitive, and emotional aspects (Table 1-1). Three mechanistically distinct types of pain are distinguishable: nociceptive, inflammatory, and neuropathic. Nociceptive pain is the baseline defensive mechanism that protects us from potential harm. Inflammatory and neuropathic pains are characterized by altered and often aberrant function of the nervous system as a result of persistent pathology or plastic changes in the nervous system.

Thus, although we tend to call any sensation that hurts “pain,” many types of pain exist that subserve various biologic functions. For example, acute pain from extreme heat initiates a reflex withdrawal and ensures minimal tissue damage (nociceptive pain). This type of pain is a survival mechanism and may be termed “good” pain. Consequently, if tissue has been damaged, the local inflammatory response causes increased sensitivity in peripheral nociceptors (peripheral sensitization) and dorsal horn neurons (central sensitization) associated with pain transmission. As a result, the hand is sensitive to touch and more sensitive to pain (allodynia and hyperalgesia; see Table 1-1) so that the person protects and immobilizes the limb to aid rapid healing. Essentially, the system has been altered to behave differently. In most cases, tissue injury is followed by a healing period associated with ongoing pain that ultimately resolves with no residual problems.

In contrast, pain with no biologic advantage to the person is termed “bad” pain. For example, chronic pain that is not associated with ongoing tissue damage, but inflicts severe physical and emotional suffering on the person, offers no survival value. Chronic pain is often the result of primary or reactive changes in the nervous system that are associated with neuronal plasticity but are unable to modulate and thus actually serve to perpetuate the sensation of pain; in short, the system has malfunctioned, and maladaptive pain remains. Chronic pain is, therefore, a disease in its own right and often not a symptom. Additionally, chronic pain responds to therapy differently from acute pain and is associated with emotional and social behavioral changes (see chapter 4). Acute and chronic pains differ in many respects, and some of the major differences are presented in Table 1-2.

Patients, and sometimes physicians, find it hard to distinguish pain as a disease from pain as a symptom. The latter signifies an expression of a pathologic process that, if treated, will cause the pain to disappear. Unfortunately, the inability to perceive pain as a disease may result in repeated and unsuccessful interventions, all in an attempt to eradicate the cause of pain.
### Table 1-1 Definition of pain terms

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
<th>Clinical implication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain</td>
<td>An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.</td>
<td>Some patients may be unable to communicate verbally. Pain is an individually subjective experience.</td>
</tr>
<tr>
<td>Alloodynia</td>
<td>Pain due to a stimulus that does not normally provoke pain (eg, touch, light pressure, or moderate cold or warmth).</td>
<td>Associated with neuropathy, inflammation, and certain headache states (see chapters 5, 9, and 11). A lowered threshold where the stimulus and response mode differ from the normal state.</td>
</tr>
<tr>
<td>Hyperalgesia</td>
<td>An increased response to a stimulus that is normally painful.</td>
<td>Associated with neuropathy or inflammation. Reflects increased pain on suprathreshold stimulation. The stimulus and response mode are basically the same.</td>
</tr>
<tr>
<td>Hyperesthesia</td>
<td>Increased sensitivity to stimulation, excluding the special senses. Includes both allodynia and hyperalgesia.</td>
<td>Associated with neuropathy or inflammation (see chapter 11).</td>
</tr>
<tr>
<td>Hypoalgesia</td>
<td>Diminished pain in response to a normally painful stimulus.</td>
<td>Typical of neural damage. Raised threshold: stimulus and response mode are the same (lowered response).</td>
</tr>
<tr>
<td>Analgesia</td>
<td>Absence of pain in response to stimulation that would normally be painful.</td>
<td>Commonly observed after complete axotomy or nerve block. Not unpleasant.</td>
</tr>
<tr>
<td>Hyperpathia</td>
<td>A painful syndrome characterized by an abnormally painful reaction to a stimulus, especially a repetitive stimulus, as well as an increased threshold. May occur with allodynia, hyperesthesia, hyperalgesia, or dysesthesia.</td>
<td>Typical of neuropathic pain syndromes (see chapter 11). Faulty identification and localization of the stimulus, delay, radiating sensation, and after-sensation may be present, and the pain is often explosive in character.</td>
</tr>
<tr>
<td>Paresthesia</td>
<td>An abnormal sensation, whether spontaneous or evoked.</td>
<td>Typical of neuropathic pain syndromes (see chapter 11).</td>
</tr>
<tr>
<td>Hypoesthesia</td>
<td>Decreased sensitivity to stimulation, excluding the special senses.</td>
<td></td>
</tr>
<tr>
<td>Dysesthesia</td>
<td>An unpleasant abnormal sensation, whether spontaneous or evoked. Hyperalgesia and allodynia are forms of dysesthesia.</td>
<td></td>
</tr>
</tbody>
</table>
Table 1-2  Major features of acute and chronic pain

<table>
<thead>
<tr>
<th>Features</th>
<th>Acute pain</th>
<th>Chronic pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time course</td>
<td>Short (hours to days)</td>
<td>Long (months to years)</td>
</tr>
<tr>
<td>Etiology</td>
<td>Peripheral (inflammatory)</td>
<td>Central (neuropathic)</td>
</tr>
<tr>
<td>Behavioral response</td>
<td>Anxiety, “guarding”</td>
<td>Depression, “illness behavior”</td>
</tr>
<tr>
<td>Response to treatment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Local intervention</td>
<td>Good</td>
<td>Poor</td>
</tr>
<tr>
<td>Analgesic drugs</td>
<td>Good</td>
<td>Poor</td>
</tr>
<tr>
<td>Psychotropic drugs</td>
<td>Poor</td>
<td>Moderate to good</td>
</tr>
</tbody>
</table>

Approach to Diagnosis and Management

The need to base therapeutic approaches on evidence-based medicine is obvious, and the authors wholeheartedly agree with this approach. Thus, this book cites state-of-the-art research to support statements whenever possible. However, evidence-based medicine is a tasteless science unless peppered by clinical experience and judgment, careful appraisal of drug side effects and complications (especially in the medically compromised patient), the individual variability of patients, and a respectful approach to a patient’s autonomy. Therefore, expert opinion also enriches this text.

As in most other textbooks of medicine, this book presents knowledge in a linear, disease-based manner. Pain syndromes are described and their signs, symptoms, and associated features outlined. This is very different from the circular process of clinical data collection; indeed, patients present with complaints rather than diseases. Knowledge of a disease does not automatically guarantee the ability to identify it from a given set of signs and symptoms. The process of accumulating clinical data in order to reach a diagnosis is as much a science as it is an art, and part of this chapter is devoted to understanding and applying this process.

Classification, Disease, and Diagnosis

In the clinical setting as much as in the research setting, classification systems are important. Diagnoses (based on classifications) usually dictate therapeutic options and indicate a prognosis. A number of relevant classifications are available for orofacial pain and headache, though they are not in complete accord.\(^\text{14}\) The IHS,\(^\text{6}\) the American Academy of Orofacial Pain (AAOP),\(^\text{15}\) and the Research Diagnostic Criteria for Temporomandibular Disorders (RDC-TMD)\(^\text{16}\) have all recently reviewed their classifications.

These classifications often have offshoots based on specific characteristics. Thus, chronic facial pain\(^\text{17}\) or chronic daily headache\(^\text{18}\) may be subclassified as temporal, and indomethacin-responsive headaches\(^\text{19}\) may be subclassified as therapeutic. The former is probably most useful in epidemiologic and disease-burden studies, whereas the latter presents a treatment-dependent diagnostic challenge. However, both classification approaches offer little advantage in guiding clinical diagnosis and therapy.\(^\text{17}\)

The concept that diseases are identifiable through their symptomatology is the basis of classifications.\(^\text{20}\) Classifications aim to organize orofacial pains, some headaches, and TMDs into a logical and applicable system. The lev-
el of detail of the classification depends on its planned use or requirements. Over the years, research has enriched medical practice with specific diagnostic biomarkers, but these are largely unavailable in the field of orofacial pain and headache. In the absence of adequate biomarkers, the diagnosis of orofacial pain and headache is based on a clinician’s ability to recognize a particular combination of signs and symptoms in a patient. Thus, diagnosis remains heavily reliant on the patient’s story—the way it is related and how the clinician interprets it.

Of course, diagnosis should not be confused with disease, as in all areas of medicine diagnoses are often made for unknown underlying processes. Consider migraine, which was once thought to be a vascular headache. After further scientific investigations, we now appreciate the complexity of the underlying central nervous system events leading to a migraine and the vascular changes understood as epi-phenomena. As we elucidate exact processes underlying a disorder, diagnosis approaches etiology and, ultimately, the true disease.

Clearly, in orofacial pain and headache the aim of diagnosing all entities is unattainable. Therefore, classifications commonly have one or more “other” diagnoses or categories. Indeed, we all have patients with chronic orofacial pain whose diagnosis remains elusive and whose signs and symptoms cannot be neatly pigeonholed into established diagnoses. Many of these entities share temporal features, such as pain for most or all of the day that is long-standing or chronic (> 3 months). Past attempts at terminology have left us with diagnoses of such diseases as atypical odontalgia, atypical facial pain, and persistent idiopathic facial pain; these classifications are inadequate. More recently, Nixdorf et al exercised the classification of such cases based on ontologic principles and suggested the term persistent dentoalveolar pain disorder. These general terms may be accurate in the symptomatic description but may also tend to lump together a number of underlying diagnostic entities that may present with similar, but subtly different, clinical phenotypes; thus, their contribution to management may be minimal.

Existing classifications are not always accurate or adequate. Often syndromes overlap in their clinical phenotype, and these are discussed in the clinical chapters. For example, a tension-type headache (TTH) may be extremely difficult to differentiate from a mild migraine without aura because of overlap in the appearance of ostensibly diagnostic features. Mild nausea and photo- and phonophobia may form part of the TTH phenotype, TTH may be aggravated by exercise, regional muscle tenderness is equally prevalent in both, and even headache precipitants are identical between migraine and TTH.

Problems occur even with specifically tailored classifications; recent reliability studies on the RDC-TMD conclude that the specified clinical tests identified as independent diagnostic criteria would be unacceptably susceptible to diagnostic misclassification. The more common diagnoses had good examiner reliability, but some lack of agreement was clearly present, even when well-trained examiners perform these procedures.

Field-testing of classifications often reveal novel subtypes of the same diagnosis or new diagnoses hidden within previous ones. Thus, cluster headache was extracted from migraines, and paroxysmal hemicrania was subsequently subclassified from cluster headache. In the field of TMDs, the recognition and classification of separate joint and muscle disorders in the late 1980s and early 1990s opened an opportunity to revise old and irrelevant terminology. Much of this work has been admirably completed in regard to joint disorders but is still lacking in regard to muscle pain, particularly chronic masticatory muscle pain, which is often termed myofascial masticatory muscle pain, a term based on an outdated premise that the muscle and surrounding fascia are the origins of pain.

Multi-axis systems recognize the biopsychosocial model of pain, which reflects the inherent complexity of the pain experience and the clear relationship between onset, treatment response, and psychosocial issues. For example, the RDC-TMD includes a separate axis for the classification of psychosocial dysfunction/suffering. It would clearly be an advantage to have an integrated classification of orofacial pain and headache that takes into account psychosocial comorbidity. The assessment of psychologic distress may be performed with the RDC-TMD questionnaire or with established alternatives.
As health care providers, we have become increasingly dependent on a wide array of laboratory and imaging studies to diagnose and subsequently manage patients’ diseases.\textsuperscript{42} We must, however, appreciate the limitation of diagnostic tests in any clinical setting but particularly in the diagnosis of orofacial pain and headache. Not only are diagnostic tests inherently limited as diagnostic tools, but there are also few biomarkers in current use for the diagnosis of primary orofacial pain\textsuperscript{43–45} and headache\textsuperscript{46–49} disorders. In the absence of biomarkers, classifications are self-defining and difficult to validate. However, as biotechnology improves, we may be able to incorporate specific biomarkers into classification criteria, whether as a separate axis or integrated. Biomarkers will aid in diagnosis and enable assessment of disease control or severity, much as levels of fasting glucose levels and glycated hemoglobin are used for diabetes. Ongoing research may change the situation, but currently the predictive value of available biomarkers in primary orofacial pain/headache diagnosis is very low.

All of these classification systems are integrated in this book according to their strengths in the following manner. The authors have no doubt that for headaches the IHS is the most comprehensive, so it is used throughout this book for all headache entities. For orofacial pain entities, the IHS classification is not detailed enough; thus, the AAOP’s criteria is used, and specifically for TMDs, the Diagnostic Criteria for TMD (DC/TMD) is used. The strength of the International Association for the Study of Pain (IASP) lies in its regional and systems approach to pain classification (eg, musculoskeletal, neurovascular pain) and to the excellent approach to neuropathic pain entities. The integration of such internationally accepted systems into pain clinics and research studies is essential and ultimately an enriching endeavor.

Diagnosis of Orofacial Pain

Faced with a patient with a pain complaint, clinicians have to answer three major questions—where, what, and why—and if possible, ask them in this order. The first, where, is concerned with the location, such as the anatomical structure or system affected. The second, what, deals primarily with the pathologic process. The third, why, is about the etiology. The patient’s decision to seek medical help is the first step in the diagnostic chain; surprisingly, not all patients with significant pain seek treatment. Based mostly on the pain location, patients will choose which specialist to consult. Naturally, if it is a toothache, the patient decides to consult the dentist, and most times the choice will be correct. However, suppose the patient’s pain is referred to the oral cavity from a remote organ (such as the heart; see chapter 14) or is associated with migraine-like mechanisms (see chapter 10), and he or she consults a dentist. The patient has clearly, and understandably, missed or misinterpreted the “where” or the “what.” The clinician’s responsibility is to analyze the patient’s complaints and reach the correct diagnosis. In other words, the clinician has to rigorously apply the diagnostic process to accurately define the location, identify the pathologic process, and ideally establish the etiology of the pain.

The natural starting point is a comprehensive gathering of information. Clinicians routinely start with history taking, the strongest tool when it comes to the diagnosis of pain. Pain symptoms should specify location, duration, pain characteristics, and other pertinent data (see the section titled “The Pain History”). In addition, a thorough personal history should include details on medical, drug, and psycho-social history; occupation; stress; family history relating to marital status and recent events (eg, bereavement); and any history of familial disorders (eg, migraine, diabetes). The physical examination is next, supplemented by other tests as needed. Once this process has been completed, a working hypothesis needs to be generated, namely, a diagnosis. Gathering information is a starting point but does not on its own make a diagnosis. In the sections that follow, the process of using the patient’s clinical data to generate diagnostic hypotheses is described.
The Diagnostic Process

Getting to Know Your Patient and the Patient’s Pain

Patients are normally willing to tell their story or pain history, but the clinician usually needs to supplement this information with specific questions concerning the location, temporal behavior, intensity, relation to function, and sensory modalities. A structured intake for the clinical interview and examination findings is useful (Fig 1-1 and Forms 1-1 and 1-2), particularly for teaching and training. The intake systematically records the basic information needed in a pain history (Box 1-1), and practitioners can design their own forms based on these principles. Additionally, the structured intake, or form, presents questions and examination procedures vital to the diagnosis of the more common clinical conditions (see chapters 8 to 12); for this, accepted classification systems such as those from the IHS, the AAOP, and the IASP are relied upon.

Fig 1-1 Suggested diagram for indicating pain location.
1. **Patient’s details**
   - Name __________________________ Age ____ Sex (M/F)
   - Marital status ___________ Occupation _______________________

2. **Medical status**  
Summary of relevant medical conditions, medications, etc (patient must complete a detailed medical questionnaire, not shown here) ____________________________________________

3. **Pain complaint**
   - Pain location (also marked on pre-prepared drawing, see Fig 1-1) ___________________________
   - Pain onset and duration _________________________________________________________________
   - Age at onset of pain attacks ____________________________________________________________
   - Pain attack frequency (mark continuous if no pain-free periods) ___________________________
   - Pain duration _______________________________________________________________________
   - Pain severity (mark on scale below (10-cm line)
     - No pain
     - Worst pain

   Factors that precipitate/aggravate pain ____________________________________________________
   - Pain is eased by _________________________________________________________________
   - Pain quality (pressing/piercing/throbbing/burning/electric/sharp/other) ____________________

4. **Accompanying signs and symptoms**
   - Systemic: nausea/vomiting/photophobia/phonophobia/dizziness
   - Local: tearing/rhinorrhea/swelling/redness

5. **History of trauma** (Yes/No)
   - If yes: date ______________ Description  __________________________________________________

6. **Pain history summary** (additional details including response to previous treatments) ______

7. **Pain in other body regions** (also mark on Fig 1-1) __________________________________________

8. **How does your pain affect your quality of life?**
   - No effect
   - Extremely

9. **How well do you sleep?**
   - Very well
   - Extremely badly

10. **Does the pain wake you?** (Yes/No) Frequency: times / night
    - Comments: ________________________________________________________________

---

Form 1-1 Pain history.
The Diagnostic Process

1. Extraoral examination
   • Head and neck (mark any asymmetry, change in color, swellings, etc)
   • Lymph nodes

2. TMJ and masticatory muscles examination (mark tenderness to palpation on a scale from 0 to 3: 0 = no tenderness, 3 = very tender)

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Right</th>
<th>Left</th>
<th>TMJ</th>
<th>Right</th>
<th>Left</th>
<th>Opening</th>
<th>(mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Masseter</td>
<td></td>
<td></td>
<td>Lateral tenderness</td>
<td></td>
<td></td>
<td>Maximum open</td>
<td></td>
</tr>
<tr>
<td>Temporalis</td>
<td></td>
<td></td>
<td>External auditory meatus tenderness</td>
<td></td>
<td></td>
<td>Deviation (right, left)</td>
<td></td>
</tr>
<tr>
<td>Medial pterygoid</td>
<td></td>
<td></td>
<td>Right occlusal loading</td>
<td></td>
<td></td>
<td>Lateral movement (right)</td>
<td></td>
</tr>
<tr>
<td>Lateral pterygoid</td>
<td></td>
<td></td>
<td>Left occlusal loading</td>
<td></td>
<td></td>
<td>Lateral movement (left)</td>
<td></td>
</tr>
<tr>
<td>Suboccipital</td>
<td></td>
<td></td>
<td>Click*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sternocleidomastoid</td>
<td></td>
<td></td>
<td>Reciprocal click*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trapezius</td>
<td></td>
<td></td>
<td>Crepitation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Mark presence and the interincisal opening at which click occurs.

3. Cranial nerves (mark if examined and intact; findings to be summarized under “Remarks”)
   • Corneal reflex
   • Pupillary reflex
   • III, IV, VI eye movements
   • Vth sensory
   • Vth motor
   • Facial (VII)
   • IX
   • XI
   • XII
   • Remarks:

4. Intraoral examination (summary)

5. Ancillary tests, radiographs (modality and summary of findings)

6. Discussion of findings and suggested diagnosis

7. Treatment plan (medications, other treatment modalities, follow-up planning)

Form 1-2 Physical examination. TMJ, temporomandibular joint.
The Pain History

Location

Precisely identifying location is a complex issue when specifically dealing with orofacial or craniofacial pain; the region is compact, and many important structures are close together (brain, eyes, nose, sinuses, and teeth), so pain spread is common. Notwithstanding, certain craniofacial pain syndromes have a propensity for particular areas and specific referral patterns. In order to record location, patients should point to the area where they feel the pain. Pain should also be marked on pre-prepared drawings of extraoral and intraoral regions (see Fig 1-1); these are helpful for communicating with the patient and serve as an important reference at a later stage. Pain can be unilateral, meaning on one side of the face, head, or mouth, or bilateral, that is, on both sides. Often pain is unilateral but may change sides from attack to attack (migraine), whereas in other conditions it may predominantly affect one side or even be side-locked (always on the same side). The patient should describe, and outline by finger pointing, whether the pain is localized or diffuse. Diffuse implies a large area with ill-defined borders and is usually outlined by patients with the whole hand rather than by finger pointing. Pain may radiate, which means the pain felt in a certain point spreads in a vectorlike fashion, or may spread in all directions. Pain radiation and pain spread are usually associated with severe pain (see chapter 6). When the source of pain is in one location but felt in another remote location, the pain is called referred. In many cases, the patient is usually aware only of the pain in the area of referral, and the primary source or location is identified by the clinician at a later stage (eg, myofascial trigger points; see chapter 8). The craniofacial symptoms may be associated with other body pains, and these are best recorded on a body drawing.

Temporal behavior

Another valuable descriptor is the behavior of pain in relation to time. The temporal behavior of the pain, once established, may be crucial in diagnosis. One of the essential features of many craniofacial pains is the age of onset; migraine typically begins early in life, whereas trigeminal neuralgia affects older subjects.

Pain may occur at specific times of the day, such as the morning or evening; thus, times
of pain onset should be recorded. Moreover, pain onset may be associated with weekly (eg, weekends), monthly (eg, menstruation), or even yearly (eg, seasonal) events. Pain can be intermittent when it comes and goes, such as in pulpitis, or continuous when it lasts for long periods, such as in muscular pain. Episodic pain, also termed periodic, appears only during certain periods, and the patient is otherwise pain free. For example, pain appears for a day or two a couple of times in a month, as in migraine, or for a couple of weeks once a year, as in cluster headache (see chapters 10 and 11). Pain may become inactive for prolonged periods and be in remission, such as observed in cluster headache and trigeminal neuralgia. Of diagnostic significance is whether the pain wakes the patient from sleep, because this is related to pain intensity and often specifically to certain diagnoses.

Pain duration is often included in the classification of orofacial pain syndromes. Masticatory myofascial pain, for example, may last from a few hours to the best part of a day, with a mean of about 5 or 6 hours (see chapter 8). Very short pain attacks—from a few seconds to 2 minutes—are characteristic of trigeminal neuralgia. At the other end of the spectrum, TTHs may last a few days, though in the chronic form they are often continuous. Overlap in pain duration is common among related facial pain syndromes, such as the trigeminal autonomic cephalalgias (see chapter 11 and Fig 11-12a).

A further temporal aspect of pain behavior relates to the frequency of pain attacks. Frequency is the number of attacks over a defined period—per day, week, month, or months and in very frequent attacks in units of minutes to hours. As described later, pain may be evoked or initiated by external stimuli, in which case the frequency of pain is related to the frequency of the stimulus application. Although specific entities are associated with a characteristic frequency of attacks, there may be significant overlap (for example, see chapter 11 and Fig 11-12b). Frequency of attacks is easily obtained from conscientiously kept pain diaries (Form 1-3).

**Modes of onset**

When strong pain develops very rapidly and aggressively, such as in pulpitis or trigeminal neuralgia, it is termed paroxysmal. Pain is evoked when it occurs only after stimulation, for example, cold application to a tooth with a caries lesion; spontaneous when it occurs on its own with no external stimulus, such as pulpitis; or triggered when the pain response is out of proportion to the stimulus, such as is typical for trigeminal neuralgia. Pain is termed progressive when it becomes more severe, or stronger, over time.

**Pain intensity**

Pain intensity is valuable diagnostic information, and thus patients are asked to evaluate how strong their pain feels. A simple and quick way is to ask the patient to assess pain intensity on a scale of 0 to 10 (a verbal analog scale, where 0 means no pain at all, and 10 is the most excruciating pain imagined). The use of a visual analog scale, where the patient can mark the pain intensity, is also useful; a number of such scales are available. Chapter 3 gives detailed descriptions of the methods for evaluating and measuring pain intensity and unpleasantness. Note, however, that there is tremendous overlap between intensities reported for craniofacial pain syndromes (Fig 1-2).

**Pain quality**

Patients suffering from particular pain syndromes more often use certain descriptive terms. Trigeminal neuralgia presents with pain that is sharp or electric, and other neuropathies are characterized by burning pain (Fig 1-3). Neurovascular pain is usually throbbing in nature, although some forms of dental pathologies also possess this quality (Fig 1-4). Therefore, we as clinicians try to elucidate specific descriptions from patients with pain by conducting a verbal interview or using established questionnaires, such as the McGill Pain Questionnaire (see chapter 3).
The Pain History

Patient’s name:__________________________________

On a scale of 0 to 10, when 0 = no pain and 10 = worst pain imaginable, mark your pain for four periods in the day (morning, midday, afternoon, and night [only if it wakes you]).

<table>
<thead>
<tr>
<th>Day and date</th>
<th>Pain intensity</th>
<th>Medication prescribed</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Morning</td>
<td>Midday</td>
<td>Afternoon</td>
</tr>
<tr>
<td>1</td>
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<td>28</td>
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</tr>
</tbody>
</table>

Form 1-3 Model of a pain diary used in a clinical setting.
The Diagnostic Process

Fig 1-2 Mean pain severity in various craniofacial pain disorders. PH, paroxysmal hemicrania; HC, hemicrania continua; IP, irreversible pulpitis; TN, trigeminal neuralgia; MMP, masticatory myofascial pain.

Fig 1-3 Symptomatic, system-based classification of chronic craniofacial pain.

Fig 1-4 Percent of patients reporting a throbbing quality in various craniofacial pain disorders. PH, paroxysmal hemicrania; HC, hemicrania continua; IP, irreversible pulpitis.
Aggravating or alleviating factors

Another part of the pain history is an attempt to elucidate if the pain is aggravated by specific factors. These may be local factors, such as chewing; ingesting cold or hot drinks; or more generalized stimuli, such as exposure to cold air, bending down, physical activity, stress, or excitement. Certain syndromes are characterized by what alleviates or reduces the pain severity; for example, rest or sleep often alleviates pain for patients with migraine. The response to simple analgesics or specific medications may often aid in diagnosis (see chapters 11 and 12).

Impact on daily function and quality of life

Pain often interferes with basic orofacial functions, such as chewing, speaking, or tooth brushing. Secondary results may include detrimental dietary changes, social isolation, and dental neglect with ensuing pathology. Additionally, most chronic pain states produce an increasingly negative impact on the patient’s general physical function and quality of life. This may reduce the patient’s work capacity and affect the function of the surrounding family members.

Sleep disruption

Pain-related sleep disorders are very common and underlie many of the affective and cognitive problems in patients with chronic pain. Prolonged periods of disturbed sleep induce daytime fatigue, sleepiness, difficulties with concentration, and reduced coping abilities. Additionally, disturbed sleep per se may induce generalized muscle pain and reduced pain thresholds and endurance. These are important factors to consider in the management of chronic orofacial pain.

Sleep disorders may occur directly because of pain or medical comorbidity. Acute dental conditions, such as irreversible pulpitis or acute periapical abscess, may cause disturbed sleep. The association between such dental conditions and sleep is based on the intensity of pain and not a specific diagnosis. Certain pain syndromes, such as the trigeminal autonomic cephalalgias (see chapter 11) and fibromyalgia (see chapter 8) may be pathophysiologically related to specific sleep disorders. Pain diaries, where the patients record nighttime pain, are often the first sign that they suffer from disturbed sleep. However, patients often report getting a full night’s sleep but awakening feeling not rested or unrefreshed. This pattern of unrefreshing sleep may aggravate the pain condition, and a vicious cycle is set up. Referral for a sleep study will determine the nature of the sleep disorder and help formulate a more comprehensive management approach. The orofacial pain specialist must be cognizant of the structure, control, and function of normal sleep and the effects of pain- or stress-related disruption. The relationship between pain and sleep is detailed in chapter 5.

Associated features

A number of local or general features consistently accompany pain attacks. These may be localized (as in swelling, redness, sweating, tearing, rhinorrhea, or ptosis) or generalized (such as nausea, photophobia, and dizziness). In addition, sensory changes may be associated with the pain complaint. Some patients may not be aware of a neurologic deficit, and thus the authors recommend a basic examination of the cranial nerves, outlined in Table 1-3. If there are findings, the specific modes of sensory changes are evaluated later, as discussed in detail in chapter 3.

Drug history as it pertains to the pain condition

Patients often forget the drugs and dosages they are taking, so they should bring documentation with them on their first visit. The most reliable method is to request a physician’s summary, but a drug card, medical alert bracelets, and hospital release notes are also valuable sources of information. Recording what drugs the patient has tried in order to alleviate pain is imperative. These may be over-the-counter drugs or physician-prescribed drugs. Exact dosage, schedule, and duration for each drug will indicate whether the full therapeutic potential was exploited.
### Table 1-3  Basic cranial nerve examination

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Pathway/cranial nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group A (Cranial nerves I, II, III, IV, VI, and VIII)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shine a light into the patient’s pupil (test both sides).</td>
<td>Tests the pupillary light reflex: Pupils should constrict bilaterally</td>
<td>Afferent is the optic nerve (I) and efferent the oculomotor (III)</td>
</tr>
<tr>
<td>Ask the patient to close his eyes, seal one nostril, and smell coffee, tobacco, or eugenol packed in unmarked containers.</td>
<td>Positive identification of smell</td>
<td>Tests olfactory nerve function (II); note that patients with colds or allergies may have a reduced ability</td>
</tr>
<tr>
<td>Ask the patient to seal one ear and whisper numbers contralaterally.</td>
<td>Accurate repetition</td>
<td>Tests the vestibulocochlear nerve (VIII)</td>
</tr>
<tr>
<td>Ask the patient to follow your finger with his eyes in the following directions:</td>
<td>Accurate and smooth tracking of finger movements by both eyes simultaneously</td>
<td>Tests the oculomotor (III), trochlear (IV), and abducens (VI) nerves</td>
</tr>
<tr>
<td><strong>Group B (Cranial nerves V and VII)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ask the patient to look laterally and up with his eyes: gently stimulate the cornea with a wisp of cotton wool.</td>
<td>Tests the corneal reflex; causes immediate closure of the eyelids</td>
<td>Afferent is the trigeminal nerve and efferent the facial</td>
</tr>
<tr>
<td>With his eyes closed, ask the patient to identify sharp, blunt, and thermal stimuli in the upper, middle, and lower face.</td>
<td>Accurate identification of stimuli and area tested</td>
<td>Sensory branches of the trigeminal (V) nerve</td>
</tr>
<tr>
<td>Ask the patient to clench teeth.</td>
<td>Feel for symmetric contraction of masticatory (eg, masseter) muscles</td>
<td>Motor branch of the trigeminal (V) nerve</td>
</tr>
<tr>
<td>Ask patient to raise his eyebrows, close his eyes, smile, and whistle.</td>
<td>Symmetric movement; good muscle strength</td>
<td>Facial (VII) nerve function</td>
</tr>
<tr>
<td>Place sweet, salty, and sour stimuli on the patient’s tongue.</td>
<td>Accurate identification</td>
<td>Chorda tympani branch of facial nerve</td>
</tr>
<tr>
<td><strong>Group C (Cranial nerves IX, X, XI, and XII)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ask the patient to rotate his head to both sides, then raise his shoulders (all against mild manual resistance).</td>
<td>Symmetric movement; good muscle strength</td>
<td>Accessory (XI) nerve</td>
</tr>
<tr>
<td>Ask the patient to say “aah.”</td>
<td>The uvula and soft palate should be raised symmetrically</td>
<td>Glossopharyngeal (IX) nerve</td>
</tr>
<tr>
<td>Ask the patient to perform tongue movements.</td>
<td></td>
<td>Hypoglossal (XII) nerve</td>
</tr>
</tbody>
</table>
Confirmatory Tests

Listening to the language of pain

Patients with similar pain conditions may describe their pain in very different terms. This may reflect differences in culture, education, or the actual physical experience of pain, no doubt influenced by genetic factors. Patients most often describe their pain in the physical dimension, for example, by severity and quality. Thus, a patient with trigeminal neuralgia may relate that the pain is severe and electric, or sharp. On the other hand, some patients may choose terms that describe an emotional dimension; the same patient with trigeminal neuralgia may add that the pain is unbearable to live with, frightening, or depressing. This multidimensionality of pain underlies its definition as an experience rather than as a sensation. The choice of words to describe pain is therefore important and offers an insight into the complete experience that a patient with pain endures (see chapters 3 and 4). Psychosocial assessment of patients with pain is therefore important (see chapter 4). The application of questionnaires typically used in such assessments is time-consuming but may be invaluable in preparing a treatment plan and assessing prognosis.

Physical Examination

The physical examination of a patient who complains of pain aims to identify the source and cause of pain, that is, the affected structure and the pathophysiologic process. Routine physical examination builds on the history to formulate a differential diagnosis and may require further special tests.

A routine physical examination of the head and neck should include observation, clinical examination (eg, palpation), and detection of functional and sensory deviations from the normal. Clinicians should look for facial asymmetry, change in color, and deviation or limitation of mouth opening. They should also palpate cervical and submaxillary lymph nodes, parotid and submandibular salivary glands, masticatory and neck muscles, and the temporomandibular joint to detect any abnormality in texture, mobility, or tenderness. A routine, basic examination of the cranial nerves (see Table 1-3) should also be performed. An intraoral examination seeks possible sources of pain (eg, caries lesions, mucosal erosions, or ulcerations) and includes examination modalities such as inspection, probing, palpation, and percussion. The authors summarize physical findings on a standardized form (see Form 1-2), though clinicians may want to devise their own form of examination according to personal preferences.

Confirmatory Tests

Several other tests, in addition to the routine physical examination, may be required to confirm or refute the suspected diagnosis. These may be as simple as the application of a cold stimulus to a tooth with suspected pulpitis or more elaborate sensory testing (see chapter 3). Radiographs and other means of imaging are still by far the most useful ancillary tests. These include the simple, relatively cheap, bitewing or periapical dental radiographs (see chapter 6) and more sophisticated, neuroimaging techniques such as computed tomography (CT) or magnetic resonance imaging (MRI).

The decision when to refer a patient with chronic orofacial pain for advanced neuroimaging is often complex, particularly under current financial constraints in health care systems. Most studies dealing with this issue relate specifically to headache or trigeminal neuralgia, but the guidelines may be easily adopted for orofacial pain in general. Among patients with normal neurologic examinations and headaches diagnosed as migraine or tension type, the prevalence of significant intracranial abnormalities on neuroimaging is approximately 0.2% and 0%, respectively. Undiagnosable headaches have a higher prevalence of intracranial abnormalities, but studies report varying, inconsistent figures ranging from 0% to 6.7%. Positive neurologic findings are intuitively suggestive of an intracranial abnormality (see also chapter 12). However, the predictive value of an intracranial abnormality by a positive neurologic exam is surprisingly low, around 3%; this is due to the very low initial probability of intracranial abnormalities. Patient complaints of neurologic symptoms will significantly increase this risk. The absence of findings on a neurologic examination notably decreases (but does not eliminate) the likelihood of finding a signif-
icant lesion on neuroimaging. When ordering neuroimaging, the orofacial pain practitioner should specifically ask that extracranial areas be examined (jaws, submandibular space), as these are often excluded on routine imaging.

The indications for advanced neuroimaging are shown in Box 1-2. For individual patients with pronounced anxiety, imaging may be indicated to alleviate emotional distress. The number of studies comparing CT with MRI is limited, but they suggest that MRI may be more sensitive than CT for identifying clinically insignificant abnormalities. However, MRI may not be more sensitive for identifying clinically significant pathology that is relevant to the cause of headache. Thus, the choice of modality and the region to be scanned need to be based on a differential diagnosis.

Choosing wisely

The use of CT increased threefold from 1993 to 2007, and concerns have been raised over the negative health effects of CT. The authors of a risk-assessment study estimated that approximately 29,000 future cancers could be related to CT scans performed in the United States in 2007. The largest contributions were from scans of the abdomen and pelvis (n = 14,000), chest (n = 4,100), and head (n = 4,000). One-third of the projected cancers were due to scans performed at the ages of 35 to 54 years. These data indicate that risk-reduction efforts are warranted.

Additionally, overtesting adds a large economic burden to the health care system. To help reduce such waste in the United States and promote physician and patient conversations on choosing treatments and tests wisely, nine medical specialty societies have joined the American Board of Internal Medicine Foundation and Consumer Reports in the first phase of the Choosing Wisely campaign. These nine organizations were asked to pick five tests or treatments within their purview that they believed are overused. The Choosing Wisely website (www.choosingwisely.org) lists 45 such tests and treatments, and eight organizations list at least one imaging test. The American Headache Society’s board of directors recently made clear recommendations about not performing neuroimaging studies in patients with stable headaches that meet criteria for migraine and not performing CT imaging for headache when MRI is available, except in emergency settings.

Clearly, organizations that bring together orofacial pain experts need to formulate similar recommendations. Meanwhile, clinicians are advised to carefully consider the use of confirmatory tests and the cost (monetary and healthwise) to benefit ratio of such tests.
Establishing a Diagnosis

Routines in medicine are very effective in that they add confidence, especially to the inexperienced; sometimes save time; and ensure a comprehensive gathering of clinical information. In principle, the diagnosis should follow the history, physical examination, and ancillary tests. Clinical information gathering is a back-and-forth process, mainly dictated by the diagnostic process and the possible differential diagnoses considered.

Indeed, an experienced clinician often formulates initial diagnostic hypotheses very early on in the clinical setting. At a certain point, and usually quite early, we start to depart from the routine and to consider diagnostic hypotheses. We start to test these hypotheses by asking specific questions. The difference is that while routine questions expect an open-ended answer to such questions as “Where do you feel your pain?” most hypothesis-generated questions aim at a closed-ended yes or no answer. For example, asking “Does bending your head aggravate pain?” is useful when a patient is suspected to have sinusitis. “Does the tooth react painfully to a cold stimulus?” may be asked when a caries lesion is suspected in a vital tooth. The answer expected to such questions is yes or no. If the answer, whether to an oral question or a physical test, satisfies the hypothesis, the examiner usually proceeds with another hypothesis-generated question. If the answer leads to a dead end, however, clinicians often return to the routine methodology.

Ultimately, we cluster enough positive pieces of information to confirm our hypothesis (diagnosis) and usually some negative pieces of information that enable us to refute other possible diagnoses.

Clustering of information is a useful tool in the decision-making process in that it reduces the number of fragments of information and facilitates the process. The specific clustering of signs, symptoms, and other information leads to a diagnosis based on classification systems, as discussed earlier.

However, gathering information on its own does not make a diagnosis. For the beginner or inexperienced clinician, the question of what to do with all of this information is real. Often, connecting the collected information with a diagnosis or a set of criteria in a classification is difficult.

Diagnosis for beginners

Over the years, the authors have developed a clustering system for diagnostic entities that is useful for the more difficult diagnostic process of chronic orofacial pain and thus useful for trainees and students. The system divides chronic orofacial pain into three main symptomatic classes: musculoskeletal, neurovascular, and neuropathic (detailed in Fig 1-3 and Box 1-3). The authors advise the beginner to examine these entities and the cluster of signs and symptoms relevant for each class of these diagnostic entities. Then proceed to Table 1-4 for a description of the diagnostic process generated by hypotheses based on pain location, and then go to Table 1-5 for the diagnostic process based on the temporal behavior and characteristics of the pain. This system proceeds from signs and symptoms presented by the patient to the disease process hypothesized by the clinician (i.e., diagnostic hypothesis). This allows the beginner to get an initial feel for the diagnosis and see which family of entities it belongs to.

After considering a diagnosis, it is legitimate to keep testing it by gathering further information. In reference to points addressed in the “Information critical for hypothesis testing” column of Tables 1-4 and 1-5, further specific information is requested at all levels: history, physical examination, and ancillary tests. The ability to start the diagnostic process from pain location (see Table 1-4) or from pain characteristics (see Table 1-5) demonstrates the versatility of the interview method and allows the clinician to cross-check the hypothesis generation in more than one way. The authors recommend that the reader refer back to this method of diagnosing chronic orofacial pain when reading subsequent chapters, especially the chapters dealing with chronic orofacial pain of musculoskeletal, neurovascular, or neuropathic origin.