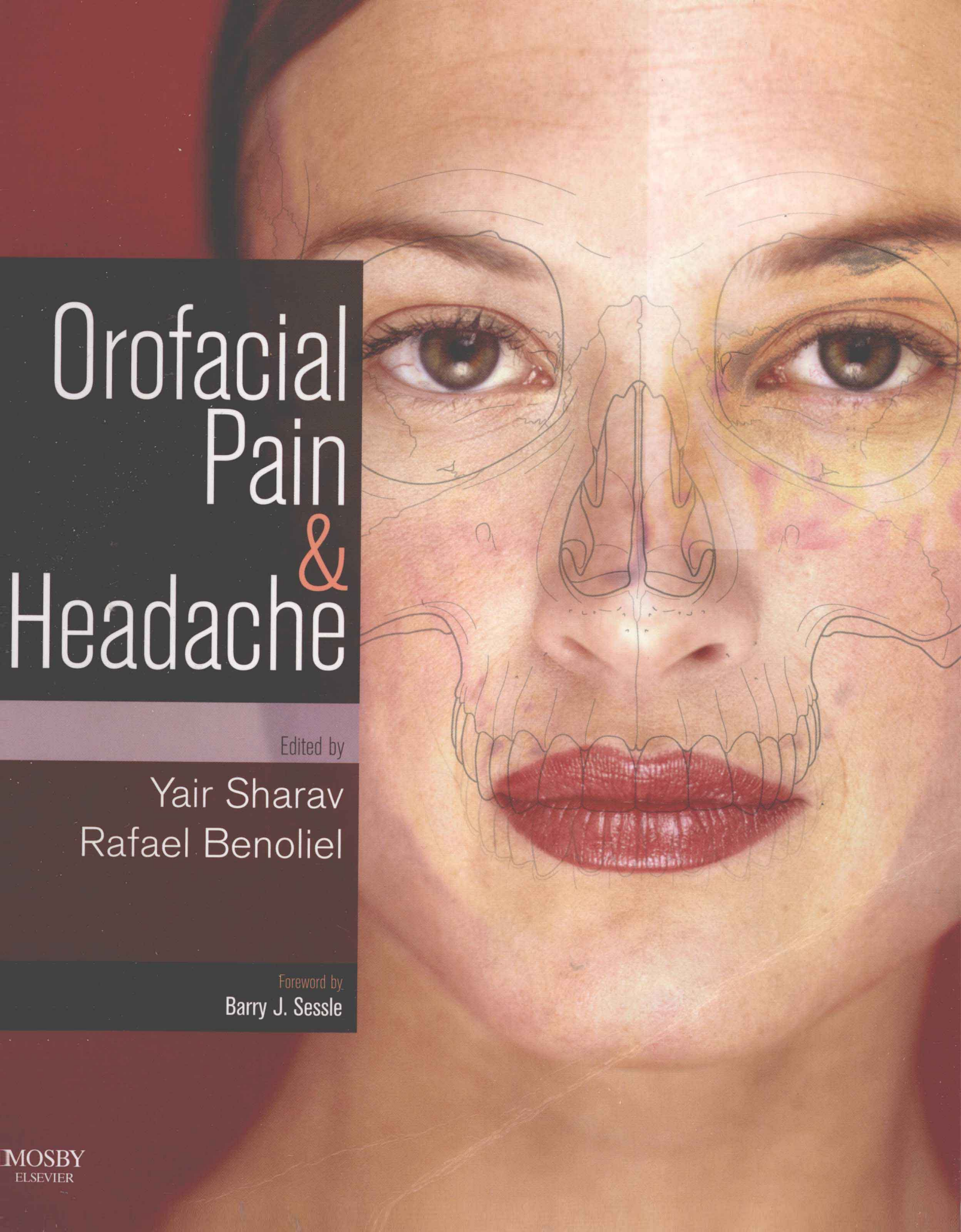

Orofacial Pain & Headache

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Foreword by

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Orofacial Pain And Headache

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Foreword

Orofacial Pain and Headache is a timely and comprehensive addition to the important area of the diagnosis and treatment of craniofacial pain. It is true to state that the diagnosis and management of acute pain conditions are readily achieved; this is one of the characteristic features of the practice of dentistry. However, this is sadly not the case for many chronic pain conditions. A patient with chronic orofacial pain can represent a significant challenge to the clinician, leading to repeated and usually unsuccessful interventions. Why is this? Unfortunately, the curriculum of most dental and medical schools has only a limited emphasis on pain mechanisms, diagnosis and management. Additionally, there is at present only an incomplete understanding of the mechanisms underlying the aetiology and pathogenesis of chronic orofacial pain conditions. Furthermore, chronic orofacial pain can take many forms, with a wide variety of apparently successful treatment options; many of these do not have a strong scientific or evidence basis. To further complicate matters, pain is a multidimensional experience involving physical, cognitive and emotional aspects and chronic pain in particular recruits active involvement of these dimensions. An important related factor is that the orofacial region has special meaning to each of us since we communicate with and express our feelings to others through this body region. We recognize our acquaintances by their facial features, and from the moment of birth, we sustain our life through the intake of air, fluids and foodstuffs through orofacial structures.

The trigeminal system provides most of the craniofacial sensory innervation, and is associated with specific physiological qualities and pain conditions. For example, pain syndromes such as trigeminal neuralgia or migraine are specific to the area, and trigeminal nerve injury responses differ from those in spinal nerves. Furthermore, the trigeminal nerve innervates anatomically related but functionally diverse organs such as the meninges, the craniofacial vasculature, the eyes, the ears, the teeth, oral soft tissues, muscles and the temporomandibular joint. In the brainstem, the trigeminal sensory nucleus overlaps with upper cervical dermatomes. Taken together, these features account for the complex and extensive pain referral patterns that often make clinical diagnosis so difficult.

The philosophy and design of this book make it a timely and instructive addition to the pain literature. Management of orofacial pain demands the services of clinicians from various specialties due to the anatomical density of the region. Based on their extensive clinical experience and a thorough understanding of pain mechanisms specific to the trigeminal system, the editors, Professors Yair Sharav and Rafael Benoliel, are well equipped to integrate knowledge across the various disciplines. They have written the major part of this comprehensive textbook and have successfully integrated knowledge from the areas of headache and craniofacial pain. In particular, they have succinctly explained common mechanisms involved in the two regions, with important implications for pain diagnosis and management.

The anatomy and neurophysiology relevant to orofacial pain are covered in Chapter 2, and this provides a solid basic science underpinning for subsequent chapters that present current knowledge of aetiologic and pathophysiologic mechanisms. The book emphasizes the four major clinical entities of orofacial pain: acute dental (Chapter 5), neurovascular (Chapters 9, 10), musculoskeletal (Chapters 7, 8) and neuropathic (Chapter 11). Diagnostic and management strategies are emphasized in these chapters and in Chapter 1, supplemented by expert contributions on otolaryngology and facial pain (Chapter 6) and neurosurgical procedures (Chapter 12), pharmacotherapy for acute and chronic pain (Chapters 15, 16), complementary and alternative medicine (Chapter 17) and psychologically based interventions (Chapter 4). Novel in its approach is the chapter on the occurrence of craniofacial pain in systemically complex patients (Chapter 14). The clinical chapters are complemented by several informative case reports that offer insight into the complexity of orofacial pain diagnosis and management.

As such, this book should be an invaluable resource for dental or medical students, dental practitioners, pain specialists from all fields and basic and clinical pain scientists who are interested in an up-to-date and comprehensive review of the diagnostic and management issues in the orofacial pain field.

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Preface

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'. This was an approach established by an otolaryngologist named Costen who linked aetiology 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 neurophysiological mechanisms common to headaches and other body areas. Masticatory muscle pain was examined in light of other regional muscle pains, and management of the temporomandibular joint was related to, and brought in line with, basic orthopaedic 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, and our thorough understanding of pain mechanisms specific to the trigeminal system, we feel that we are well equipped to fulfil 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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Contents

CHAPTER 1

The diagnostic process

Yair Sharav and Rafael Benoliel

1. The Problem

Diagnosis and treatment of orofacial pain is a complex process compounded by the density of anatomical structures and the prominent psychological 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 man's headache is another man's facial pain. In clinical practice, they are often intimately related. Consequently, the patient with orofacial pain may wander from one specialist to the other in order to get adequate help.

There are excellent textbooks available on orofacial pain or headache; each with its own individual emphasis, aimed at specific reader groups. Do we need another textbook? A textbook that integrates orofacial pain with headache based on contributions by various disciplines, all with extensive clinical experience and a thorough understanding of pain mechanisms specific to the trigeminal system, is indeed required. These, in essence, are the foundations for writing this book. Accordingly, we relate to *all regional craniofacial pains* and aim at presenting the wider picture of orofacial pain syndromes, including the overlap between primary headaches and primary orofacial pain entities. Many of the patients with chronic orofacial pain are primary headache variants, present in the orofacial region, and a lack of familiarity with these syndromes probably underlies misdiagnosis by dental practitioners as well as by medical specialists. Other patients that remain unclassified by the International Headache Society (IHS) and are unknown to neurologists, otolaryngologists, other medical practitioners and even to dentists are probably primary orofacial pain entities (Benoliel *et al* 1997; Czerninsky *et al* 1999; Sharav and Benoliel 2001; Benoliel and Sharav 2006). We believe the integration between headache and orofacial pain classifications to be of paramount importance, especially since about half of patients in tertiary care craniofacial pain clinics are still labelled as 'idiopathic' or 'undiagnosable' under IHS classification (Zebenholzer *et al* 2005, 2006; Benoliel and Sharav 2006). 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 (Chapter 7). There is also a growing patient population with chronic craniofacial pain due to trauma associated with traffic accidents (Benoliel *et al* 1994) and with invasive dental procedures such as dental implants, which demands a multidisciplinary approach. We hope that by applying this approach the gap between medically trained headache specialists and dentally trained orofacial pain specialists will be bridged. Additionally we have attempted to compile a book that will be useful to readers at different stages of their careers: undergraduate students, residents and practitioners, as well as dental and medical pain specialists. The chapters are clearly subdivided to allow quick navigation through the text and selection of relevant sections. Although each chapter is independent, we have no doubt that working through the book will serve the reader best.

1.1. The Scope

The diagnosis and management of orofacial pain, both acute and chronic, have become important subjects in dentistry (Sharav 2005). Additionally orofacial pain is quite prevalent in the general population: around 17–26%, of which 7–11% are chronic (Goulet *et al* 1995; Riley *et al* 1998; Macfarlane *et al* 2002a; Ng *et al* 2002; McMillan *et al* 2006). Naturally therefore, the emphasis of this book is on the four major clinical families of orofacial pain: acute dental, neurovascular, musculoskeletal and neuropathic (Chapters 5, 7–11). In these chapters, we review

current aetiology, diagnosis and treatment. Although the many case presentations included are largely 'virtual', i.e. created from integrated data, the cases are real in that they reliably duplicate the type seen in orofacial pain clinics, and any resemblance to specific cases is purely coincidental. However, as any experienced clinician will attest, the 'typical' textbook cases are rare and we relate to the changes in presentation that may cause diagnostic confusion under each relevant section. Atypical cases may be difficult to manage; many have superimposed traum and consequent neuropathic pain. Some of these cases often have a history of misdiagnosed acute pains in the orofacial region leading to repeated and unsuccessful interventions that slowly escalate and result in dental extractions and surgeries. Accurate diagnosis of acute dental and orofacial conditions is therefore essential and covered in Chapter 5. The importance of acute and chronic otolaryngological syndromes in the differential diagnosis of facial pain is paramount and our colleagues cover this area in Chapter 6. The growing number of older, often medically compromised, patients with orofacial pain deserves special attention: Is orofacial pain in these patients related to their medical condition? The answer is found in Chapter 14.

One of the mainstays of pain management is indisputably pharmacotherapy. Because many of the drugs used are common to many of the syndromes, we have included two separate chapters on pharmacotherapy: acute and chronic (Chapters 15 and 16). The management of pain relies on accurate diagnosis and on reliable follow-up that demonstrates objective improvement. Chapter 3 covers the important area of pain measurement as well as the assessment of peripheral nerve function. Unfortunately, we are a long way from optimal patient care and some of the best drugs will offer substantial relief (not absolute) for only about 75% of patients, many with disturbing side effects. Many patients therefore enquire about neurosurgical options; see Chapter 12. In parallel, there is rising popularity and demand among orofacial pain patients for complementary and alternative medicine (CAM), and we must know the available treatment options in this field (Chapter 17). No diagnosis and treatment of orofacial pain is complete without understanding its emotional undercurrents or having a thorough knowledge of its psychological aspects and treatment possibilities, which are covered in Chapter 4. Although this is essentially a clinical book, anatomy and neurophysiology are covered in a manner specifically relevant to the topic of orofacial pain (Chapter 2).

1.2. The Philosophy

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

As in most other medical textbooks, the presentation of knowledge here is done in a 'linear', disease-based, manner. We describe pain syndromes and outline their signs, symptoms and associated features, which is very different to the 'circular' process of clinical data collection and indeed how 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 the understanding and application of this process.

The great strength of classification systems, especially if they have been field-tested, lies in their ability to predefine into a recognizable and universally accepted entity the sign and symptom complex that patients present. We employ criteria published by the IHS (Olesen *et al* 2004), the American Academy of Orofacial Pain (AAOP) (Okeson 1996) and the International Association for the Study of Pain (IASP) (Merskey and Bogduk 1994). None of these are perfect, nor are they individually comprehensive. Therefore, we integrate all these systems according to their strengths in the following manner. For headaches the IHS classification reigns supreme and is used throughout this book for all 'headache' entities. However, it is not detailed enough for orofacial pain entities and so we have used the AAOP's criteria. For temporomandibular disorders (TMDs), the Research Diagnostic Criteria for TMD (RDC-TMD) are often referred to (Dworkin and LeResche 1992). The IASP's strength lies in its regional and systems approach to pain classification (e.g. musculoskeletal, neurovascular pain) and its excellent approach to neuropathic pain entities. The integration of such internationally accepted systems into pain clinics and research papers is essential, and ultimately an enriching endeavour.

2. The Revolution: Pain as a Disease

Pain is a multifaceted experience involving physical, cognitive and emotional aspects (see Table 1.1). There are three mechanistically distinct types of pain: nociceptive, inflammatory and neuropathic. Nociceptive pain is the baseline defence mechanism that protects us from potential harm. Inflammatory and neuropathic pain 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 term any sensation that hurts 'pain', many types of pain exist that subserve various biological functions. For example, acute pain from extreme heat initiates a reflex withdrawal and ensures minimal tissue damage (nociceptive pain). This type of

Table 1.1 Definition of Pain Terms

Term	Definition	Clinical Implication
Pain	An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.	Some patients may be unable to communicate verbally. Pain is an individually subjective experience.
Allodynia	Pain due to a stimulus which does not normally provoke pain (e.g. touch, light pressure, or moderate cold or warmth).	Associated with neuropathy, inflammation and certain headache states; see Chapters 5, 9 and 11. A lowered threshold where the stimulus (e.g. light touch) and response mode (pain) differ from the normal state.
Hyperalgesia	An increased response to a stimulus which is normally painful.	Associated with neuropathy or inflammation—reflects increased pain on suprathreshold stimulation. The stimulus and response modes are basically the same.
Hyperaesthesia	Increased sensitivity to stimulation, excluding the special senses. Includes both allodynia and hyperalgesia.	Associated with neuropathy or inflammation. See Chapter 11
Hyperpathia	A painful syndrome characterized by an abnormally painful reaction to a stimulus, especially a repetitive stimulus, as well as an increased threshold.	Typical of neuropathic pain (Chapter 11)
Hypoalgesia	Diminished pain in response to a normally painful stimulus.	Typical of neural damage. Raised threshold: stimulus and response mode are the same but with a lowered response.
Analgesia	Absence of pain in response to stimulation which would normally be painful.	Commonly observed after complete axotomy or nerve block. Not unpleasant.
Hyperpathia	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, hyperaesthesia, hyperalgesia or dysaesthesia.	Typical of neuropathic pain syndromes (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.
Paraesthesia	An abnormal sensation, whether spontaneous or evoked.	Typical of neuropathic pain syndromes; see Chapter 11.
Hypoaesthesia	Decreased sensitivity to stimulation, excluding the special senses.	
Dysaesthesia	An unpleasant abnormal sensation, whether spontaneous or evoked. Hyperalgesia and allodynia are forms of dysaesthesia.	

pain is a survival mechanism termed 'good' pain (Iadarola and Caudle 1997). Consequently, if tissue has been damaged, the local inflammatory response in the injured tissue causes increased sensitivity in peripheral nociceptors (peripheral sensitization) and in 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, Table 1.1) so that the individual protects and immobilizes the limb to aid rapid healing. Essentially the system has been altered to behave differently. In most cases, tissue injury followed by a healing period associated with ongoing pain resolves with no residual problems for the individual (still good pain).

In contrast, pain with no biological advantage to the organism is 'bad' pain. For example, chronic pain not associated with ongoing tissue damage, that inflicts severe physical and emotional suffering on the individual, offers no survival value. Such chronic pain syndromes are typical of neuropathic pain but also include other syndromes, for instance chronic orofacial pain syndromes and headaches. Chronic pain is often the result of primary or reactive changes in the nervous system associated with neuronal plasticity, which are unable to modulate and actually serve to perpetuate the sensation of pain (see post-traumatic neuropathic pain, Chapters 2 and 11); 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 behavioural changes (see Chapter 4). Acute and chronic pains differ from each other in many respects, and some of the major differences are presented in Table 1.2.

Patients, and sometimes physicians, find it hard to distinguish between pain as a disease and pain as a symptom. The latter signifies an expression of a pathological process that if treated will cause the pain to disappear. Unfortunately, the inability to perceive pain as a *disease*

Table 1.2 Major Features of Acute and Chronic Pain

Features	Acute Pain	Chronic Pain
Time course	Short (Hours to days)	Long (months to years)
Aetiology	Peripheral mechanisms	Central mechanisms
Behavioural response	Anxiety, 'guarding'	Depression, 'illness behaviour'
Response to treatment:		
Local intervention	Good	Poor
Analgesic drugs	Good	Poor
Psychotropic drugs	Poor	Moderate to good

may result in repeated and unsuccessful interventions, all in an attempt 'to eradicate the cause of pain'.

3. The Process: Diagnosis of Orofacial Pain

Faced with a patient with a pain complaint we must answer three major questions: Where, What and Why; and if possible in the order presented. The first, *where*, is concerned with the location, such as the anatomical structure or system affected. The second, *what*, deals primarily with the pathological process. The third, *why*, is about the aetiology. 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 that the patient's pain is referred to the oral cavity from a remote organ (such as the heart, see Chapter 13), or is associated with migraine-like mechanisms (Chapter 9) and he consults his dentist. The patient has clearly, and understandably, missed or misinterpreted the 'where' or the 'what'. It is the responsibility of the clinician to analyse the patient's complaints and to reach the correct diagnosis. In other words, the clinician must rigorously apply the diagnostic process in order to accurately define the location, identify the pathological process and ideally establish the aetiology of the pain.

Our natural starting point is a comprehensive gathering of information. We 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 below). In addition, a thorough personal history should include details on medical, drug and psychosocial history, occupation, stress and a family history relating to marital status, recent events (e.g. bereavement) and any history of familial disorders (e.g. migraine, diabetes). We proceed with the physical examination, supplemented by other tests as needed. Once we have gone through this process, we then generate a working hypothesis, namely a diagnosis. Gathering information is a starting point, but on its own does not make a diagnosis! We will describe in the following the process of utilizing the patient's clinical data to generate diagnostic hypotheses.

3.1. The Methodology

Patients are normally willing to tell their 'story' or pain history, but there is usually a need to supplement this information with specific questions concerning the location, temporal behaviour, intensity and relation to function and to sensory modalities. We find that a structured intake for the clinical interview and examination findings is useful (Boxes 1.1 and 1.2 and Fig. 1.1), particularly for teaching and training. The intake systematically records the basic information needed in a pain history (Box 1.3) 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 7–11)—for this, we draw on accepted classification systems such as those of the IHS, the AAOP and the IASP.

3.1.1. The Pain History

Location. Precisely identifying location is a complex issue when specifically dealing with orofacial or craniofacial pain; the region is compact, with many important structures 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 extra-oral and intraoral regions (Fig. 1.1); these are very 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, on both sides. Often pain is unilateral but may change sides from attack to attack (migraine) whereas in others 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 that the pain felt in a certain point spreads in a vector-like fashion or it may spread in all directions. Pain radiation and pain spread are usually associated with severe

Box 1.1 Pain History

1. **Patient's details:** Name.....
   Age.....
   Sex: Male/Female 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:**
   a. Pain location (also marked on pre-prepared drawing, see Fig. 1.1) .....
   b. Pain onset and duration .....
   c. Age at onset of pain attacks .....
   d. Pain attack frequency (mark continuous if no pain free periods) .....
   e. Pain attack duration .....
   f. Pain severity- mark on scale below (10cm line)

   

   No Pain Worst Pain
   g. Factors that precipitate/aggravate pain .....
   h. Pain is eased by .....
   i. Pain quality: pressing/piercing/throbbing/burning/electric/sharp/other .....
4. **Accompanying signs and symptoms:**
   a. Systemic: nausea/vomiting/photophobia/phonophobia/dizziness
   b. 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: /night**
    Comment: .....

pain; see Chapter 5 (Sharav *et al* 1984). When the source of pain is in one location but felt in another, remote location, the pain is called referred. In many such 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 (e.g. myofascial trigger points; see Chapter 7). The craniofacial symptoms may be associated with other body pains and these are best recorded on a body drawing.

Temporal behaviour. Another valuable descriptor is the behaviour of pain in relation to time. The temporal behaviour 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 whilst trigeminal neuralgia affects older subjects.

Pain may occur at specific times of the day, such as the morning or evening. Moreover pain onset may be associated with weekly (e.g. weekends), monthly (e.g. menstruation) or even yearly (e.g. seasonal) events. Pain can be *intermittent*, such as in pulpitis, or *continuous*, as in muscular pain. Episodic pain, also termed *periodic*, appears during certain periods; otherwise the patient is 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 9, 10). Pain may become inactive for prolonged periods and be in *remission*, such as observed in cluster headache and in trigeminal neuralgia. Of diagnostic significance is whether the pain *wakes* the patient from sleep since this is common in neurovascular-type pain or pulpitis; see section on sleep disruption below.

Pain *duration* is often included in the classification of orofacial pain syndromes. Masticatory myofascial pain, for example, may last from some hours to the best part of a day with a mean of about 5–6 hours (see Chapter 7). Very short pain attacks—from a few seconds to 2 minutes—characterize trigeminal neuralgia. At the other end of the spectrum tension-type headaches may last a few days and in the chronic form are often continuous. Overlap in pain duration is common among related facial pain syndromes such as the trigeminal autonomic cephalalgias (see Chapter 10, Fig. 10.9A).

A further temporal aspect of pain behaviour 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 below 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 10, Fig. 10.9B). Frequency of attacks is easily obtained from conscientiously kept pain diaries (see below and Box 1.4).

3.1.1.1. Modes of Onset

When strong pain develops very rapidly in an aggressive fashion such as in pulpitis or trigeminal neuralgia, it is termed *paroxysmal*. Pain is *evoked* when it occurs only after stimulation, e.g. cold application to a tooth with a carious lesion; *spontaneous* when it occurs on its own with no external stimulus (e.g. 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 of valuable diagnostic information, and we ask patients to evaluate how strong their pain feels. A simple and quick way is to ask the

Box 1.2 Physical Examination

(Continued from Box 1.1, pain history)
(Including, diagnostic considerations and treatment plan)

1. **Extra-oral examination**
   a. **Head and Neck** (mark any asymmetry, change in color, swellings, etc.) .....
   b. **Lymph nodes** .....
2. **TMJ and masticatory muscles examination** (mark tenderness to palpation on a scale from: 0 = no tenderness, to 3 = very tender)

Muscles	Right	Left	TMJ	Right	Left	Opening	In mm
Masseter			Lat. tenderness			Max. open	
Temporalis			Ext. Miatus Tend.			Deviation (R, L)	
Med. Pterygoid			Right Occ loading			Lat. Move (R)	
Lat. Pterygoid			Left Occ loading			Lat. Move (L)	
Sub-occipital			Click*				
SC mastoid			Reciprocal click*				
Trapezius			Crepitation				

*mark presence and the interincisal opening at which click occurs; SC, sternoeleido; Occ, occsulal

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. **Intra-oral examination (summary)**
   .....
5. **Ancillary tests, X-rays** (modality and summary of findings)
   .....
6. **Discussion of findings and suggested diagnosis**
   .....
7. **Treatment plan** (medications, other treatment modalities, follow-up planning):
   .....

patient to assess pain intensity on a scale of 0–10 (verbal analogue scale, where 0 means no pain at all, and 10 the most excruciating pain imagined). The use of a visual analogue scale (VAS), where the patient can mark the pain intensity, is also useful and there are a number of such scales available. Chapter 3 gives detailed descriptions of the methods that can be used to evaluate and measure pain intensity and unpleasantness. It is important to note, however, that there is tremendous overlap between intensities reported for craniofacial pain syndromes (Fig. 1.3).

Pain quality. Patients suffering from particular pain syndromes more often employ certain descriptive terms. Trigeminal neuralgia presents with pain that is sharp or electric, whereas other neuropathies have burning pain (Fig. 1.2). Neurovascular pain is usually throbbing in character, although some forms of dental pathologies also possess this quality (Fig. 1.4). We therefore try to elucidate specific descriptions from pain patients by verbal interview or by employing established questionnaires such as the McGill Pain Questionnaire (see Chapter 3).

Aggravating or alleviating factors. We attempt to elucidate if the pain is aggravated by specific factors. These may be local factors such as chewing, ingestion of 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: rest or sleep often alleviates pain for patients with migraine. The response to simple analgesics or specific medications may often aid in diagnosis (see, for example, Chapters 10, 11).

Impact on daily function and quality of life. Pain often interferes with basic orofacial functions such as chewing, speaking or even tooth brushing. Secondary results may include detrimental dietary changes, social isolation and dental neglect with ensuing pathology. Additionally most chronic pain states induce 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 chronic pain patients (Moldofsky 2001a). Prolonged periods of disturbed sleep induce daytime fatigue, sleepiness, difficulties with concentration and reduced coping abilities (Lavigne *et al* 1999; Brousseau *et al* 2003). Additionally disturbed sleep per se may induce generalized muscle pain and reduced pain thresholds and endurance (Moldofsky 2001a). These are important factors to consider in the management of chronic orofacial pain.

Sleep disorders may occur directly because of pain or medical comorbidity (Nicholson and Verma 2004;

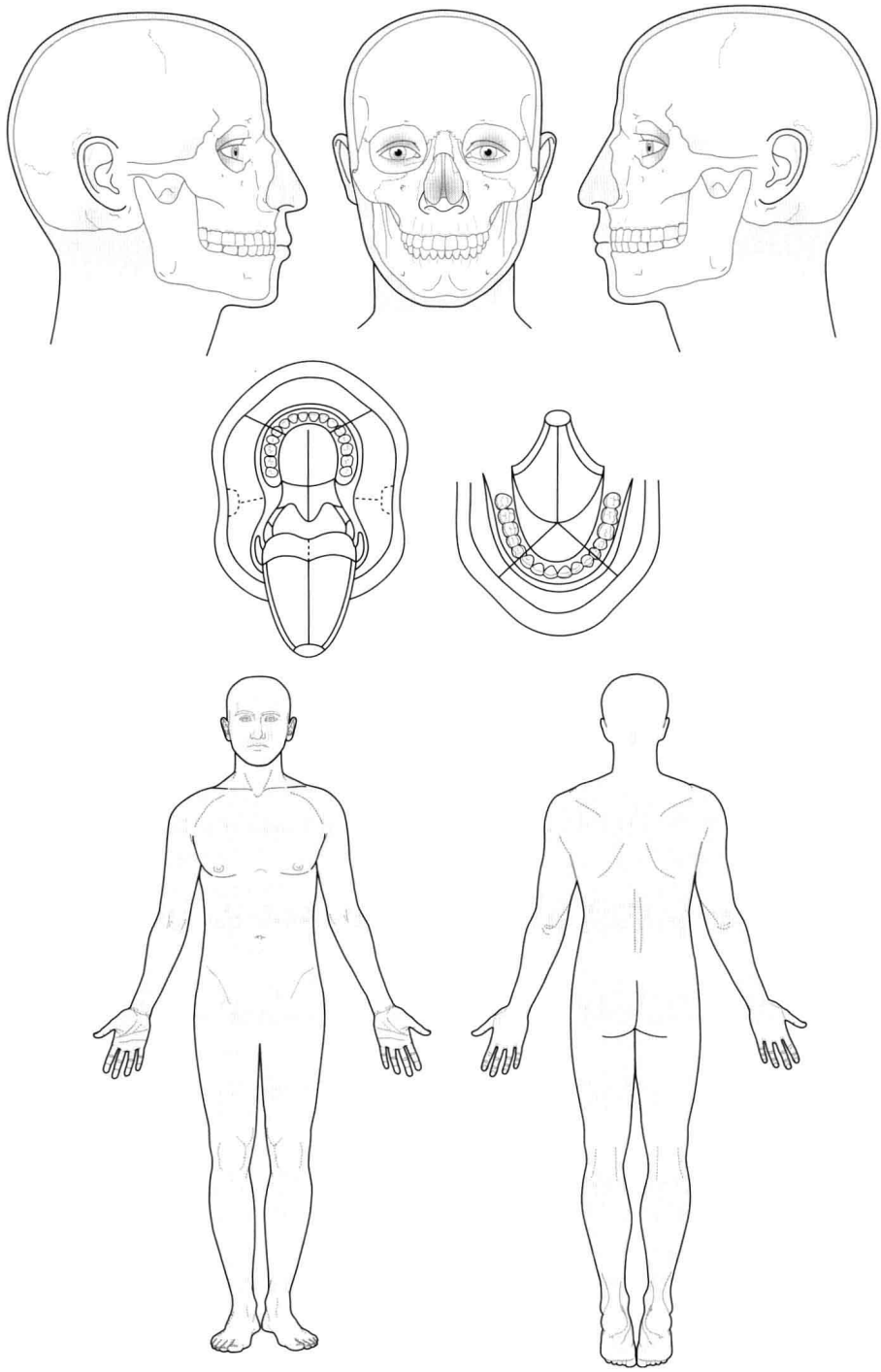

Fig. 1.1 • Suggested diagram for drawing pain location.

Sabatowski *et al* 2004; Kelman and Rains 2005; Zelman *et al* 2006). Acute dental conditions such as irreversible pulpitis or acute dentoalveolar 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 (Sharav *et al* 1984). Certain pain syndromes such as the trigeminal autonomic cephalgias (Chapter 10) and fibromyalgia (Chapter 7) may be pathophysiologically related to specific sleep disorders (Moldofsky 2001b; Rains and Poceta 2005). Pain diaries, where the patients record nighttime pain, are often the first sign that patients suffer from disturbed sleep. However, patients may often report getting a full night's sleep but awaken feeling not rested or unrefreshed. This pattern of unrefreshing sleep may itself 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 (Vgontzas and Chrousos 2002; Hirshkowitz 2004; Harris 2005).

Associated features. A number of local or general features often consistently accompany pain attacks. These

Box 1.3 Essentials of an Orofacial Pain History

- Location
  - Local: Head, neck, intra oral
  - Other body regions
- Attack Onset
  - Time of day; morning, midday, evening.
  - Month- menstrual
  - Year- seasonal
- Attack duration
  - Seconds, minutes, hours, days
- Attack frequency
  - 24 hour distribution
  - Use of pain diaries
- Onset of present problem
  - Age at onset, associated events, trauma
- Severity
  - Verbal or visual analogue scales
- Quality
  - Verbal descriptions; stabbing, burning
  - Structured questionnaires: McGill
- Associated features or signs
  - Local, systemic
- Aggravating factors
  - Local: thermal, function
  - Systemic: dialysis, stress
- Alleviating factors
  - Endogenous; sleep
  - Exogenous; analgesics, massage
- Impact on daily function
  - Lost work days, marital relations, wakes
- Personal and Social History
  - Occupation, stress, function. Psychosocial evaluation
- Family History
  - Headache, facial pain, bereavement
- Medical status
  - Hypertension
- Drug History
  - Analgesic drug abuse

may be localized as in swelling, redness, sweating, tearing, rhinorrhea, and ptosis, or generalized such as nausea, photophobia and dizziness. There may be sensory changes associated with the pain complaint. Some patients may not be aware of a neurological deficit and we recommend a basic examination of the cranial nerves, as outlined in Table 1.3. If there are findings, the specific modes of sensory changes should be evaluated, as discussed in detail in Chapter 3, Measuring and Assessing Pain.

Drug history as pertains to the pain condition. Patients often forget the drugs and dosages they are taking, and should bring documentation with them on their first visit. The most reliable method is to request a physician's summary or a drug card; medical alert bracelets and hospital release notes are also valuable sources of information. It is imperative to record what drugs the patient has tried to alleviate pain. These may be over-the-counter (OTC) drugs or physician-prescribed. Exact dosage, schedule and duration for each drug trial will indicate whether the full therapeutic potential was exploited.

Listening to the 'language of pain'. Patients with similar pain conditions may describe their pain in very different terms (Zborowsky 1969). 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, severity and quality. Thus, a patient with trigeminal neuralgia may relate that their pain is severe and electric, or sharp. Additionally some patients may choose terms that describe an 'emotional' dimension; the same patient with trigeminal neuralgia may add that their pain is unbearable to live with, frightening or depressing. The choice of words to describe pain is therefore important and offers insight into the complete experience that pain patients endure (see Chapters 3 and 4). Psychosocial assessment of pain patients 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.

3.2. The Physical Examination

The physical examination of a patient who complains of pain aims at identifying the source and cause of pain, i.e. the affected structure and the pathophysiological process. Routine physical examination builds upon 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 (e.g. palpation) and detection of functional and sensory deviations from the normal. One should look for facial asymmetry, change in colour and deviation or limitation of mouth opening. We palpate cervical and submaxillary lymph nodes, parotid and submandibular salivary glands, masticatory and neck muscles and the TMJ to detect any abnormality in texture, mobility or tenderness. A routine, basic examination of the cranial nerves is also performed (Table 1.3). An intraoral examination seeks possible sources of pain, e.g. carious lesions, mucosal erosions or ulcerations, and includes examination modalities such as inspection, probing, palpation and percussion. We summarize physical findings on a standardized form (Box 1.2), but the clinician may devise his/her own form of examination according to personal preferences.

3.3. The Differential: Confirmatory Tests

In addition to the routine physical examination, several tests 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, 'bite-wing' or periapical dental radiographs

Box 1.4 Model of Pain Diary Employed in Clinical Setting. QOL = Quality of Life

Patient's name:

On a scale of 0 to 10, when: 0 = No pain, and 10 = Strongest pain possible, mark 4 times a day your pain for that period (AM = morning, Mid. = midday, PM = afternoon. Ni. = night, only if wakes).

Day and date	Pain intensity				Medication prescribed	Remarks	
	AM	Mid.	PM	Ni.		Side effects, escape drugs (No., type)	Effect on QOL
1							
2							
3							
4							
5							
6							
20							
21							
22							
23							
24							
25							
26							
27							
28							

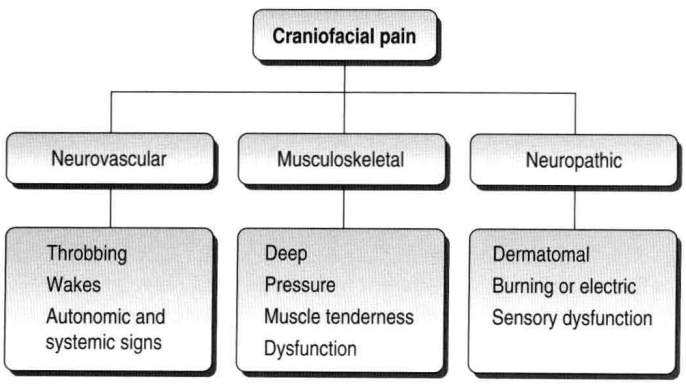

Fig. 1.2 • Symptomatic, system-based classification of chronic craniofacial pain.

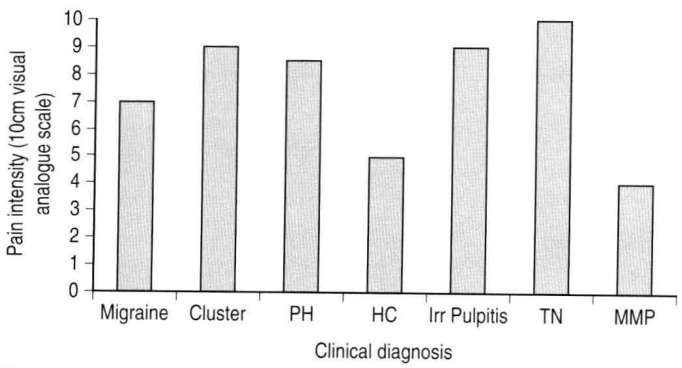

Fig. 1.3 • Mean pain severity in various craniofacial pain disorders. PH = paroxysmal hemicrania, HC = hemicrania continua, Irr Pulpitis = irreversible pulpitis, TN = trigeminal neuralgia, MMP = masticatory myofascial pain.

(Chapter 5) and the more sophisticated, neuroimaging techniques such as computerized 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 healthcare systems. Most studies dealing with this issue relate specifically to headache or trigeminal neuralgia, but the guidelines may easily be adopted for orofacial pain in general (Frishberg *et al* 2000; Sandrini *et al* 2004).

Among patients with normal neurological examinations and headaches diagnosed as migraine or tension type, the prevalence of significant intracranial abnormalities on neuroimaging is approximately 0.2 and 0%, respectively (Frishberg *et al* 2000). Undiagnosable headaches have a higher prevalence of intracranial abnormalities but studies report varying, inconsistent figures ranging from 0 to 6.7% (Frishberg *et al* 2000). Positive neurological findings are intuitively suggestive of an intracranial abnormality; see also trigeminal neuralgia discussion in Chapter 11. However, the predictive value of an intracranial abnormality by a positive neurological exam is surprisingly low at around 3%; this is due to the very low initial probability of intracranial abnormalities. Patient complaints of neurological symptoms will significantly increase this risk (Frishberg *et al* 2000). The absence of findings on the neurological examination significantly decreases (but does not eliminate) the likelihood of finding a significant lesion on neuroimaging (Frishberg *et al* 2000). When considering neuroimaging the orofacial pain practitioner should