Orofacial Pain and Headache:

A Comprehensive Guide

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Edited by

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Cambridge Scholars Publishing

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Professor Benoliel has published extensively on the clinical phenotypes of orofacial pain and their interface with headache and continues to publish in both clinical and basic related sciences. He is presently the Editor-in-Chief of the *Journal of Oral and Facial Pain and Headache* and co-edited the award-winning textbook "Orofacial Pain and Headache". He serves in many scientific committees including the Classification Committees of the International Headache Society, the joint IASP/WHO classification workforce for chronic pain in ICD-11 and was key in the establishment of the first 'International Classification of Orofacial Pain'.

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Professor Sharav established the first Orofacial Pain Clinic in Israel in 1974 at the Hadassah Medical Center and was co-founder and director of the Hebrew University Center for Research on Pain.

Professor Sharav served as and Head of the Department of Oral Medicine in Hadassah Medical Center for almost 25 years. During 1979-1985 he was the Dean of the Faculty Dental Medicine at the Hebrew University.

Internationally, he was twice a Visiting Scientist at the National Institute of Health (NIH) in Bethesda Maryland, and a Vising Professor at Toronto University.

Professor Sharav's work has widely contributed to the knowledge and understanding of diagnosis, classification, management and mechanisms of orofacial pain.

PREFACE TO THE 3RD EDITION

Diagnosis and treatment of orofacial pain is an intricate process exacerbated by the density of anatomical structures, and the psychological significance to the facial area. Management of this pain therefore demands the services of clinicians from many specialties. Hence the patient may wander frustratingly from one specialist to another.

We have come a long way from our 2008 first edition. At that time, we questioned the need for another textbook on Orofacial Pain; and especially its integration with Headache. The question was not necessary. The book was accepted with great enthusiasm and the British Medical Association (BMA) book competition highly commended this first edition as one of the best books published in Medicine for that year.

When the 2nd Edition, published in 2015, was again awarded by the BMA book competition, as one of best books in Neurology, we knew we were on the right track. Reviewers pointed to our success at integrating the two broad topics of orofacial pain and headache.

Two years ago, our hope of integrating Orofacial Pain and Headache culminated with the introduction of the new International Classification of Orofacial Pain, 1st edition (ICOP) published in 2022 in Cephalalgia, the official Journal of the International Headache Society. The classification delineates the boundaries of our subject matter, enhances precise communication between professionals involved in the area, orofacial pain specialists as well as headache neurologists, proving to be very valuable in refining the diagnostic process.

As the first and second editions were well accepted and highly praised, we felt that we were not in a position to disappoint our faithful readers. We very much hope that we have succeeded in this mission.

Finally, for this edition Rafael Benoliel will be first co-editor and Yair Sharav second co-editor.

Yair Sharav, Rafael Benoliel. 2025

CHAPTER 1

DIAGNOSIS OF THE PATIENT WITH OROFACIAL PAIN

YAIR SHARAV, RAFAEL BENOLIEL

Diagnosis and treatment of orofacial pain is an intricate process compounded by the density of anatomical structures, and the prominent emotional significance attributed to this region. Management of orofacial pain demands the services of clinicians from various specialties, such as dentistry, otolaryngology, ophthalmology, neurology, neurosurgery and psychology/psychiatry. Complex referral patterns to and from adjacent structures are common in orofacial pain adding to the complexity of the diagnosis of pain in this area. Consequently, the patient with orofacial pain may wander from one specialist to the other in order to get adequate help. We believe that an integrative knowledge encompassing ***all regional craniofacial pains*** is mandatory in order to give the best service to our patients. The third edition of our textbook therefore continues to integrate all craniofacial pains, based on contributions from various disciplines, having extensive clinical experience and a thorough understanding of pain mechanisms specific to the trigeminal system. Since we wrote the previous, second, edition of this book, many changes have occurred, in particular the definition of orofacial pain disorders; refining both its boundaries and relation with headaches. This culminated with the introduction of the International Classification of Orofacial Pain, 1st edition (ICOP) (1). The classification enhances precise communication between professionals involved in the area, and is very valuable for research and in refining the diagnostic process.

The emphasis of this book is on the three major clinical families of chronic orofacial pain: musculoskeletal, neurovascular, and neuropathic. Clustering diagnostic signs and symptoms according to these clinical entities, is a very robust way of handling information (see Boxes 1-1,1-2,1-3). In the individual chapters we review current approaches to etiology, diagnosis, and treatment, including case presentations. Some of the cases have a history of

misdiagnosed pains, leading to repeated and unsuccessful interventions with superimposed trauma and consequent neuropathic pain. A short chapter on acute orofacial pain is also included, as this is often the first encounter with a patient presenting with a confusing clinical phenotype. We also include a chapter on pharmacotherapy of acute and chronic pain, paying particular attention to the impact of the opioid epidemic and offer appropriate therapeutic alternatives.

1. Chronic Pain is a Disease

Pain is a multifaceted experience involving physical, cognitive, and emotional aspects. The International Association for the Study of Pain has redefined pain as "an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage." Acute pain is usually a *symptom* associated with trauma or an inflammatory response defined as a localized sensation of discomfort, distress, or agony, resulting from the stimulation of specialized nerve ending. It serves as a protective mechanism insofar as it induces the sufferer to remove or withdraw from the source. 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 in dorsal horn neurons (central sensitization) associated with pain transmission. As a result, the affected area is painful to touch and an increased pain response (allodynia and hyperalgesia, Table 1-1) so that the individual protects and immobilizes the affected area to aid rapid healing. In most cases, tissue injury followed by a healing period associated with ongoing pain, ultimately resolves with no residual problems.

Chronic pain has no biological advantage to the individual and is 'bad' pain. It is mostly not associated with ongoing tissue damage and inflicts severe physical and emotional suffering on the individual, with no survival value. Many chronic pain conditions (e.g. low back pain, irritable bowel syndrome) have an obscure etiology and pathophysiology, but they are characterized by a complex interplay of biological, psychological, and social factors, which invariably include vague and ambiguous terms such as "nonspecific," "somatoform," or "functional." The term chronic primary pain (CPP) was chosen after extensive consultation and is expected to have widespread acceptability, especially from a nonspecialist perspective (2). The definition of the new diagnosis of CPP is intended to be diagnostic with regard to etiology, or the lack of a clear etiology. It aims to avoid the

obsolete dichotomy of "physical" versus "psychological," as well as exclusionary terms that define something by what is absent, such as "nonspecific." The meaning of "functional" is also ambiguous. The introduction of "chronic primary pain" eliminates this ambiguity (2).

Chronic pain may also be 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; the system has malfunctioned and maladaptive pain remains.

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 headaches.
Hyperalgesia	An increased response to a stimulus which is normally painful.	Associated with neuropathy or inflammation.
Hyperesthesia	Increased sensitivity to stimulation, excluding the special senses. Includes both allodynia and hyperalgesia.	Associated with neuropathy or inflammation.
Hypoalgesia	Diminished pain in response to a normally painful stimulus.	Typical of neural damage.
Analgesia	Absence of pain in response to stimulation which would normally be painful.	Commonly observed after complete axotomy or nerve block. Not unpleasant.

Chronic pain is, therefore, often a disease in its own right and not a symptom. Chronic pain has devastating effects on patient wellbeing, often associated with catastrophizing mechanisms resulting in severe and incapacitating pain. The application of behavioral modalities for pain management is consequently often necessary. No diagnosis and treatment of orofacial pain is therefore complete without understanding its emotional undercurrents and a thorough knowledge of its psychological aspects and

treatment possibilities. Additionally chronic pain responds to therapy differently from acute pain and is associated with emotional and social behavioral changes (see Table 1-2).

Patients, sometimes also physicians, find it hard to distinguish between pain as a disease, in contrast to 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* may result in repeated and unsuccessful interventions, attempting "to eradicate the cause of pain".

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)
Etiology	Peripheral (inflammatory)	Central (neuropathic)
Behavioral response	Anxiety, "guarding"	Depression, "illness behavior"
Local intervention	Good	Poor
Analgesic drugs	Good	Poor
Psychotropic drugs	Poor	Moderate to good

2. Epidemiology of Orofacial Pain

Orofacial pain, of which about 10% is chronic, affects around a quarter of the general population (3-5). Painful temporomandibular disorders are quite prevalent with 4.6% of the population reporting this type of pain (6.3% in women, 2.8% in men) (6). A national survey found that 5% of adults reported pain in the face or jaw over a 3-month period. The prevalence of orofacial pain in dental patients was 16.1%, of which the most prevalent pain locations were dentoalveolar (9.1%) and temporomandibular pain (6.6%); the former decreasing with age but not the latter (7). The incidence ratio of persistent facial pain is 38.7 per 100,000 person years, and more common in women and increased with age (8). It is clear that orofacial pain is more prevalent than previously thought.

Box 1-1 Typical clusters of signs and symptoms
Musculoskeletal Orofacial pain

TMJ (temporomandibular joint pain)
- ❑ Pain fairly localized to TMJ area
- ❑ Click/crepitating of TMJ
- ❑ Deviation of mouth opening toward affected joint
- ❑ TMJ painful on palpation and on function

MMP (masticatory myofascial pain)
- ❑ Pain, mostly unilateral, at angle of mandible and front of ear, diffuse
- ❑ Masticatory muscles tenderness on palpation, mostly on affected side
- ❑ Jaw dysfunction (limited opening, tiredness on chewing)
- ❑ Pain on function (such as: yawning, chewing, talking)

TTH (tension-type headache)
- ❑ Pain, bilateral, at temples and occipital areas, pressing and annoying
- ❑ Periodic or chronic
- ❑ Anorexia
- ❑ Nausea

3. Approach to Diagnosis

The presentation of knowledge in the present one is in a “linear”, disease-based, manner. We describe pain syndromes and outline their signs, symptoms, and associated features. In this respect, this is very different to the “circular” process of clinical data collection *and indeed patients present with complaints rather than diseases*. Knowledge of a disease does not therefore automatically guarantee the ability to identify it from a given set of signs and symptoms presented by the patient. The process of accumulating clinical data in order to reach a diagnosis is as much a science as it is an art, and we will devote part of this chapter to the understanding and application of this process.

4. The diagnostic process

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 etiology. The patient's decision to seek medical help is the first step in the diagnostic chain. 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, if the patient's pain is referred to the oral cavity from a remote organ such as the heart or is associated with migraine-like mechanisms driving the patient to seek help for a toothache. The patient has clearly, and understandably, missed or misinterpreted the 'Where' or the 'What.' It is the responsibility of the clinician to reach the correct diagnosis, relating to the source of pain and to its pathological process.

Box 1-2 Typical clusters of signs and symptoms
Neurovascular Craniofacial Pain

Migraine

- ❑ Strong, unilateral headache, throbbing
- ❑ Periodic
- ❑ Occasionally wakes from sleep
- ❑ Photophobia/phonophobia
- ❑ Nausea and vomiting
- ❑ Tearing, occasional

CHA (Cluster headache**)**

- ❑ Periorbital unilateral strong pain
- ❑ Clusters of active periods of pain
- ❑ At active period 1-2 attacks/24 hours,
- ❑ Typical attack 45-60 minutes duration
- ❑ Wakes from sleep (REM locked?)
- ❑ Tearing, one eye on affected side
- ❑ Rhinorrhea, one nostril on affected side
- ❑ Redness, ptosis, miosis of eye on affected side, possible
- ❑ Patient paces around restlessly

CPH (chronic paroxysmal hemicrania)

- ❑ Periorbital and temporal, unilateral st pain
- ❑ Short (minutes) paroxysmal pain attacks
- ❑ Occasionally wakes from sleep
- ❑ Tearing, conjunctival injection, one eye on affected side
- ❑ Rhinorrhea, one nostril on affected side
- ❑ Head movement may trigger pain

NVOP (neurovascular orofacial pain)

- ❑ Mid-face, peri-, and intra-oral pain
- ❑ Spontaneous or evoked (mostly by cold food ingestion)
- ❑ Occasional swelling or redness of cheek
- ❑ Nausea
- ❑ Tearing, one eye on affected side
- ❑ Mostly periodic but may be chronic

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. 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 medical disorders (e.g. migraine, diabetes). We proceed with the physical examination, supplemented by other tests as needed. Once we complete this process, we need to generate a working hypothesis, namely a diagnosis. Gathering information is a starting point, but does not on its own make a diagnosis! We will describe below the process of utilizing the patient's clinical data to generate diagnostic hypotheses.

5. The diagnostic hypotheses

Routines in medicine are very effective, as 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. Yet, the gathering of clinical information is a back-and-forth process, mainly dictated by the diagnostic process and the possible differential diagnoses considered. Indeed, the experienced clinician often formulates initial *diagnostic hypotheses* very early on in the clinical setting. At a certain point in time, 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” answer to questions such as “where do you feel your pain?”, on the other hand most “hypothesis-generated questions” aim at a “closed” yes or no answer. For example, “does bending your head aggravate pain?” in patients where sinusitis is suspected. “Does the tooth react painfully to my application of a cold stimulus?” when a carious lesion in a vital tooth, as the source of evoked pain, is suspected. The answer expected to such questions is a *yes or no*. If the answer, whether to an oral question or to a physical test, “satisfies” the hypothesis the examiner usually proceeds with another hypothesis-generated question, but if the answer leads to a dead-end, clinicians often return to the routine methodology. Ultimately, we cluster enough “positive” pieces of information to confirm our hypothesis (diagnosis), and usually also 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 (see Boxes 1-1,1-2,1-3). The specific clustering of signs, symptoms, and other information leads to a diagnosis based on classification systems later discussed. 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 this information is real. Often connecting the collected information with a diagnosis or a set of criteria in a classification is difficult.

6. Diagnosis for beginners: A guide for the perplexed

Over the years, we have developed a clustering system for diagnostic entities that is useful for the more difficult diagnostic process of *chronic* orofacial pain. We have found it extremely useful for trainees and students. This system divides chronic orofacial pain into three main symptomatic classes: musculoskeletal, neurovascular, and neuropathic (detailed in Boxes 1-1,1-2,1-3). 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 1-3 for a description of the diagnostic process generated by hypotheses based on *pain location*, and then go to Table 1-4 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. It is entirely "legitimate", after considering a diagnosis, to keep testing it by gathering further information. Referring to points addressed in the column of "Information critical for hypothesis testing" of tables 1-3 and 1-4 for further specific information is requested at all levels; history, physical examination, and ancillary tests. The ability to start the diagnostic process from pain-location (Table 1-3) or from pain temporal-characteristics (Table 1-4) allows for versatility of the interview method and for cross checking our hypothesis generation in more than one way.

All of the above may initially seem quite confusing, but when reading the chapters dealing with chronic orofacial pain of musculoskeletal, neurovascular, or neuropathic origin, and gathering more clinical experience, the usefulness of this guide will become apparent. Therefore, while presented at the introductory part of the book, it is a recommended reference to consult with when reading subsequent chapters.

7. The Pain History

Patients are normally willing to tell their "story" or pain history, but there is usually a need to supplement this information with specific questions such as location, temporal behavior, intensity, and relation to function and to sensory modalities. The essentials of an orofacial pain history includes the following: location, onset, duration, frequency, severity, quality, associated signs, aggravating factors, alleviating factors, impact on daily function, and medical and family history. A structured intake form for the clinical interview and examination findings is useful; practitioners can design their own forms based on these essentials.

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 in 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. 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 pain patients endure.

8. 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.

The routine examination. 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. We look for facial asymmetry, change in color, and deviation or limitation of mouth opening. We palpate cervical and submaxillary lymph nodes, parotid and submandibular salivary glands, masticatory and neck muscles, TMJ, and detect any abnormality in texture, mobility, or tenderness.

Box 1-3 Typical clusters of signs and symptoms
Neuropathic orofacial Pain

Trigeminal neuralgia (TN)

- ❑ Unilateral pain trigeminal nerve area (mostly 2nd and 3rd divisions)
- ❑ Paroxysmal electric-like very short (seconds) strong pain
- ❑ Pain attack accompanied by facial tic
- ❑ Triggered by light touch, vibration, and other non-painful stimuli
- ❑ After triggering there is a refractory period
- ❑ No sensory deficit

Traumatic Neuropathies (CRPS-I/II)

- ❑ Pain location associated trauma
- ❑ Pain continuous, mostly burning
- ❑ Allodynia
- ❑ Sensory deficit (in CRPS/II)
- ❑ Dysesthesia (in CRPS/II)

Table 1-3. Diagnostic process and hypothesis generation based on pain location

Location (unilateral)	**Diagnostic hypothesis**	**Information critical for hypothesis testing**
Fronto-temporal	Migraine Hemicrania continua	Pain attack duration Patterns of periodicity Photo- and/or phono-phobia Nausea
Orbital and periorbital	Cluster headache Paroxysmal hemicrania SUNCT	Pain attack duration Attacks/day Periodicity Tearing, rhinorrhea SUNCT triggered by touch
Preauricular, angle of mandible	Temporomandibular joint (TMJ) pain Masticatory muscle pain	Aggravates by chewing Mouth opening dysfunction TMJ tenderness, click Masticatory muscle tenderness
Mid-face, peri-oral, or intra-oral	Trigeminal neuralgia Neurovascular orofacial pain (NVOP)	Attack duration Triggered by touch, vibration Evoked by cold/hot foods Tearing, rhinorrhea

An intra-oral 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. Physical findings should be summarized on a standardized form, devised by the clinician according to his/her personal preferences.

Table 1-4. Diagnostic process and hypothesis generation based on temporal pain characteristics

Temporal pain behavior and characteristics	**Diagnostic hypothesis**	**Information critical for hypothesis testing**
Short, paroxysmal	Trigeminal neuralgia Paroxysmal hemicrania SUNCT	Pain location and attack duration Triggering/evoking stimuli Autonomic signs Wakes from sleep
Periodic, throbbing	Migraine Cluster headache Neurovascular orofacial pain (NVOP)	Pain location and attack duration Periodicity Autonomic signs Nausea
Continuous, pressing	TTH, TMJ, MMP, HC	Pain location, laterality, aggravated by chewing, TMJ or masticatory muscle tenderness, click, mouth opening dysfunction, history of trauma, sensory changes

TTH=tension type headache, MMP=masticatory muscle pain, TMJ=temporomandibular joint, HC=hemicrania continua

9. Confirmatory Tests

Several additional 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. 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 and the more sophisticated, neuroimaging

techniques such as computerised tomography (CT) or magnetic resonance imaging (MRI). The indication whether to choose CT or MRI imaging may depend on clinical considerations discussed specifically under the relevant chapters. The American Headache Society's Board of Directors made clear recommendations among which, "not to perform neuroimaging studies in patients with stable headaches that meet criteria for migraine and not to perform computed tomography imaging for headache when magnetic resonance imaging is available, except in emergency settings" (9). Thus, they adopt the Choosing Wisely recommendations (10) (see Box 1-5).

Sometimes, in individual cases with pronounced anxiety, imaging may be indicated to alleviate emotional distress.

10. Choosing wisely

The use of CT increased threefold from 1993-2007 and concerns have been raised over the negative health effects these may have (11). The authors of a risk assessment study estimated that approximately 29000 future cancers could be related to CT scans performed in the US in 2007. The largest contributions were from scans of the abdomen and pelvis (n = 14 000), chest (n = 4100), and head (n = 4000) (11). One-third of the projected cancers were due to scans performed at the ages of 35 to 54.

Additionally, over testing adds a significant economic burden to the healthcare system. Many physicians worry about malpractice liability and order too many tests for fear of overlooking anything that could conceivably contribute to a lawsuit, resulting in tests and treatments that are inappropriate, unnecessary, wasteful, or redundant. To help reduce such waste in the US and promote physician and patient conversations on choosing treatments and tests wisely, 9 medical specialty societies have joined the American Board of Internal Medicine (ABIM) Foundation and Consumer Reports in the first phase of the Choosing Wisely campaign (12). These nine organizations were asked to pick 5 tests or treatments within their purview that they believed were overused. The Choosing Wisely Website (www.choosingwisely.org) lists these 45 tests and treatments, of which 8 list at least 1 imaging test (10). At the time of our book update, August 2024, there were 80+ Specialty Society Partners, 700+ Recommendations of Tests and Treatment that were considered overused or unnecessary, and 30+ countries reached. The American Headache Society's Board of Directors made clear recommendations among which not to perform neuroimaging studies in patients with stable headaches that meet

criteria for migraine and not to perform computed tomography imaging for headache when magnetic resonance imaging is available, except in emergency settings (9). American College of Radiology Choosing Wisely recommendation, updated June 29, 2017, says, "Don't do imaging for uncomplicated headache. Imaging headache patients absent specific risk factors for structural disease is not likely to change management or improve outcome… Also, incidental findings lead to additional medical procedures and expense that do not improve patient well-being."

Box 1-5
Indications for neuroimaging in headache

Clear Indication
- Unexplained abnormal neurological finding (e.g. numbness)

Possible Indication
- Headache worsened by Valsalva maneuver
- Headache causing awakening from sleep
- New headache in the older population
- Progressively worsening headache or rapidly increasing headache frequency

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

11. Smartphone-assisted diagnosis

With the escalating use of smartphone devices in everyday life, there has been a rapid growing trend for adapting them into diagnostic needs related to medical health care (13). Certain information, such as medications taken or underlying medical problems, can be readily obtained from a smartphone app. Medscape or Epocrates are among such very useful apps, that contain useful medical information and can be downloaded free of charge. These are also very useful when checking for possible drug interactions, especially when prescribing medications to our patients. Pain diaries, very useful for follow-up for monitoring change in pain and behavior, are available on follow-up smartphones daily diaries (14). Finally, with online health information becoming increasingly popular among patients, it became clear that health information seeking could improve the patient-physician relationship, if the patient discusses the information with the physician.

References

1. International Classification of Orofacial Pain, 1st edition (ICOP). Cephalalgia. 2020;40(2):129-221.
2. Nicholas M, Vlaeyen JWS, Rief W, Barke A, Aziz Q, Benoliel R, et al. The IASP classification of chronic pain for ICD-11: chronic primary pain. Pain. 2019;160(1):28-37.
3. Macfarlane TV, Blinkhorn AS, Davies RM, Kincey J, Worthington HV. Oro-facial pain in the community: prevalence and associated impact. Community Dent Oral Epidemiol. 2002;30(1):52-60.
4. Ng KF, Tsui SL, Chan WS. Prevalence of common chronic pain in Hong Kong adults. The Clinical journal of pain. 2002;18(5):275-81.
5. McMillan AS, Wong MC, Zheng J, Lam CL. Prevalence of orofacial pain and treatment seeking in Hong Kong Chinese. J Orofac Pain. 2006;20(3):218-25.
6. Isong U, Gansky SA, Plesh O. Temporomandibular joint and muscle disorder-type pain in U.S. adults: the National Health Interview Survey. J Orofac Pain. 2008;22(4):317-22.
7. Horst OV, Cunha-Cruz J, Zhou L, Manning W, Mancl L, DeRouen TA. Prevalence of pain in the orofacial regions in patients visiting general dentists in the Northwest Practice-based REsearch Collaborative in Evidence-based DENTistry research network. Journal of the American Dental Association. 2015;146(10):721-8 e3.
8. Koopman JS, Dieleman JP, Huygen FJ, de Mos M, Martin CG, Sturkenboom MC. Incidence of facial pain in the general population. Pain. 2009;147(1-3):122-7.
9. Loder E, Weizenbaum E, Frishberg B, Silberstein S, American Headache Society Choosing Wisely Task F. Choosing wisely in headache medicine: the American Headache Society's list of five things physicians and patients should question. Headache. 2013;53(10):1651-9.
10. Rao VM, Levin DC. The overuse of diagnostic imaging and the Choosing Wisely initiative. Ann Intern Med. 2012;157(8):574-6.
11. Berrington de Gonzalez A, Mahesh M, Kim KP, Bhargavan M, Lewis R, Mettler F, et al. Projected cancer risks from computed tomographic scans performed in the United States in 2007. Arch Intern Med. 2009;169(22):2071-7.
12. Cassel CK, Guest JA. Choosing wisely: helping physicians and patients make smart decisions about their care. JAMA. 2012; 307(17):1801-2.

13. Hernandez-Neuta I, Neumann F, Brightmeyer J, Ba Tis T, Madaboosi N, Wei Q, et al. Smartphone-based clinical diagnostics: towards democratization of evidence-based health care. J Intern Med. 2019;285(1):19-39.
14. Gamwell KL, Kollin SR, Gibler RC, Bedree H, Bieniak KH, Jagpal A, et al. Systematic evaluation of commercially available pain management apps examining behavior change techniques. Pain. 2021;162(3):856-65.

CHAPTER 2

ACUTE DENTAL PAIN

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Acute orofacial pain is most frequently dental in origin, associated with the teeth and their supporting structures; the periodontium. Dental pain is primarily due to dental caries. Other oral pains are periodontal or gingival in origin. Acute dental and periodontal pain is moderate to severe in intensity or from 60 –100 on a 100 mm visual analogue scale (VAS) [1]. In about 60% of cases pain is not localized but spreads into remote areas of the head and face, and reported in sites that differ from the pain source [1, 2]. There is considerable overlap in pain referral patterns for maxillary and mandibular sources [1]. These observations indicate that the spatial distribution of acute dental pain is not sufficient as a diagnostic tool for identifying pain source, and the source of pain cannot be predicted from the pain location. Pain spread is correlated with pain intensity and stronger pain

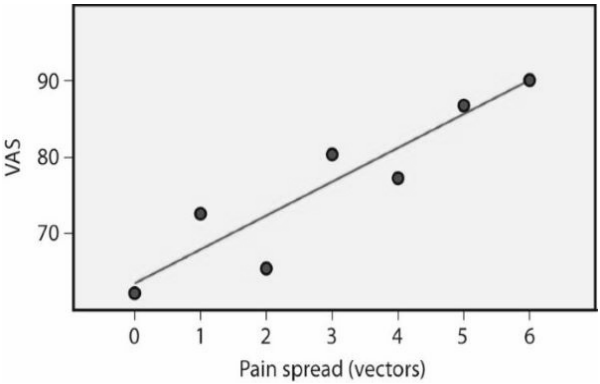

Figure 2-1: Spread of acute dental pain as a function of pain intensity. Pain intensity is described on a 0 - 100 mm visual analogue scale (VAS). Pain spread denotes the number of locations on the face that the pain spread to (vectors). It is clear that pain spread is a function of pain intensity. (Based on data from Sharav *et al.* [1]).

tends to spread more (Figure 2-1), but neither duration nor quality of pain influences the incidence of referred pain [3]. Pain spread is not dependent on the tissue affected by the pathological process, e.g. dental or periodontal structures [1].

The mechanisms responsible for pain spread are of central origin, resulting from interactions between primary nociceptive afferents and trigemino thalamic neurons. Factors which may be important for the extensive pain spread patterns in the facial area include: convergence of primary afferents from different areas, such as cutaneous, tooth pulp, visceral, neck, and muscle afferents onto nociceptive and non-nociceptive neurons in trigeminal subnucleus caudalis. Spatial overlap of pain from maxillary and mandibular pain sources is most probably due to the large receptive fields of wide dynamic range neurons, that can extend beyond one trigeminal division [4, 5]. Glial cell activation has been proposed to be also involved in the phenomenon of spread of pain sensation [6]. As pain intensity increases, neurons whose receptive field center lies within the source of pain would increase their activity activating a larger receptive field, and also activating somatotopically adjacent neurons [5].

Recently, the classification of dental pain was summarized, and forms a basis for research data reporting as well as for clinical observation [7]. A clear classification and terminology will avoid misdiagnosis and dental mutilation, and indeed it was recently addressed [8].

1. Caries and symptom progression

Caries confined to the enamel is not associated with any pain. As the caries lesion progresses to the superficial layer of the dentine, pain can be evoked to various stimuli such as change in temperature and sweet substances. With deeper caries penetration into the tooth the pain to these stimuli becomes stronger and lasts longer. When the carious lesion approaches the tooth pulp, a strong, spontaneous, paroxysmal pain develops, that is usually intermittent in nature. Finally, as microorganisms and products of tissue disintegration invade the area around the root apex, the tooth becomes very sensitive to chewing, touch, and percussion. Usually at that stage the paroxysmal, intermittent pain acquires a continuous dull nature and the tooth is no longer sensitive to changes in temperature. The development of symptoms follows the progression of pathology and the dental structures involved; initially dentine, followed by pulp and ultimately the periapical periodontal tissues (Figure 2-2). In clinical practice the demarcation between these various stages is sometimes indistinct; the tooth may be sensitive simultaneously to

temperature changes and to chewing. The anamnestic details of dental and periodontal pain are described in Table 2-1 and the physical and radiographic signs in Table 2-2.

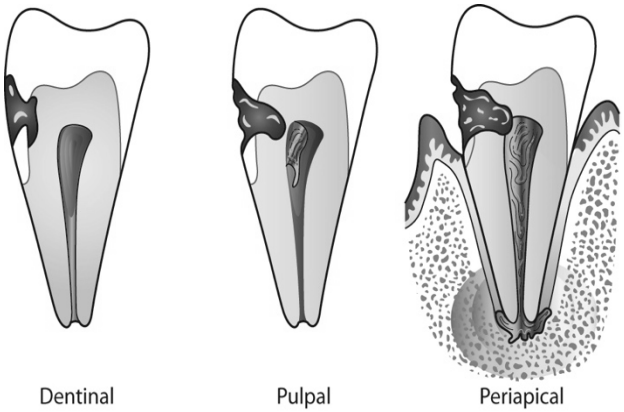

Figure 2-2: Acute dental pain presented at three progressive consecutive stages of caries penetration: (a) **Dentinal** pain is associated with caries penetrating into the dentine, (b) **Pulpal** pain is associated with deep caries penetration approaching the dental pulp, (c) **Periapical** periodontitis occurs when the inflammatory process invades into the periapical area.

2. Epidemiology of dental pain

Epidemiological data on dental pain are sparse and of poor quality and its reported prevalence in community-dwelling children or adults has a very high range, depending on the description used for dental pain or the methods of data gathering [9, 10].

More than half of the publications reported the lifetime prevalence of dental pain, while few studies reported the current prevalence of dental pain. As a result, heterogeneity was high among the included publications (e.g., 01.33-87.7%). Overall pooled prevalence of dental pain was 32.7% (CI = 29.6–35.9) [11].

3. Dental Pain

3.1 Dentinal pain

The anamnestic details of dentinal pain are described in Table 2-1. Pain is evoked, of mild to moderate intensity, and is usually aggravated by cold or

sweet foods and beverages. The physical and radiographic signs of dentinal pain are described in Table 2-2. Bitewing X-Rays are most useful in detecting proximal caries, especially if located at the distal aspect (Figure 2-3).

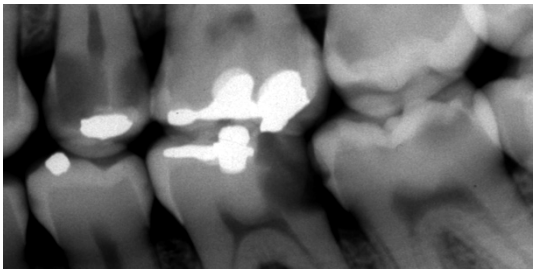

Figure 2-3: Bite-wing radiograph of left side tooth crowns demonstrates multiple locations with deep caries.

3.2 The cracked-tooth "syndrome"

Symptoms. In addition to the symptoms typical for dentinal pain, the patient may also complain of a sharp pain, elicited by biting, that resolves immediately. Localization of the source of pain is not precise, but aided by the biting location. The patient also complains of pain and discomfort associated with cold and hot foods. These complaints indicate a crack in the dentine, the so-called 'cracked tooth syndrome' [12, 13]. These incompletely fractured teeth may be associated with long lasting diffuse orofacial pain and are difficult to diagnose [14]. Assessment, prognosis, and predictable management strategies of the cracked tooth syndrome were recently reviewed [15].

Physical and radiographic signs. The main diagnostic challenge is localizing the affected tooth, especially as the crack is not readily detected and radiographs are not helpful. Localization can be achieved by causing the crack to widen, and thus duplicate the pain, by the following techniques:

1. Percussion or pressure on the cusps of the suspected teeth at different angles (Figure 2-4a).
2. Asking the patient to bite on individual cusps using a fine wooden stick or a predesigned bite stick (available commercially).
3. Probing firmly around margins of fillings and in suspected fissures (Figure 2-4b).

The bite test was found to be the most reliable for reproducing symptoms [13].

Table 2-1: Anamnestic details of dental and periodontal pain

Pain origin	Localiz-ation	Character	Intensity	Aggravated by
Dental				
Dentinal	Poor	Evoked, does not outlast stimulus	Mild to moderate	Hot, cold, sweet, or sour foods
Pulpal	Very poor	Spontaneous, paroxysmal, intermittent	Moderate to severe	Hot, cold, sometimes chewing
Periodontal				
Periapical Lateral	Good	Continues for hours, deep, boring	Moderate to severe	Chewing

3.3 Treatment of Dentinal Pain

Dentinal pain due to caries is best treated by removal of the carious lesion and restoring the tooth. Sensitivity usually disappears within a day or two, although when the carious lesion is deep the tooth may remain sensitive to cold stimulation for a week or two. Treatment of the 'cracked tooth' depends on the state of the tooth (existing restorations, periodontal condition) and the extent of the fracture. Crowning the tooth is inevitable but root canal treatment is usually not indicated. Of 127 patients that were crowned because of a crack tooth and specifically diagnosed with reversible pulpitis, 100 cases did not require root canal treatment within 6 years of follow up. It was concluded that root canal treatment will be necessary in about 20% of these cases within a 6-month period [16].