Cephalalgia
An International Journal of Headache
International Headache Society

International Classification of Orofacial Pain, 1st edition (ICOP)

Cephalalgia
2020, Vol. 40(2) 129–221

DOI: 10.1177/0333102419893823
journals.sagepub.com/home/cep
SAGE

Copyright

Translations

IHS expressly permits translations of all or parts of ICOP for the purposes of clinical application, education, field testing or other research. It is a condition of this permission that all translations are registered with IHS. Before embarking upon translation, prospective translators are advised to enquire from IHS whether a translation exists already in the proposed language. All translators should be aware of the need to use rigorous translation protocols. Publications reporting studies making use of translations of all or any part of ICOP should include a brief description of the translation process, including the identities of the translators (of whom there should always be more than one). IHS will not endorse translations. Endorsements may be given by member national societies; wherever these exist, such endorsement should be sought.

The Orofacial Pain Classification Committee

The committee is a collaborative group consisting of members of the Orofacial and Head Pain Special Interest Group (OFHP SIG) of the International Association for the Study of Pain (IASP), the International Network for Orofacial Pain and Related Disorders Methodology (INfORM), the American Academy of Orofacial Pain (AAOP) and the International Headache Society (IHS).

Co-chairmen

Rafael Benoliel, USA; Arne May, Germany; Peter Svensson, Denmark

Members of working groups (in alphabetical order after the chairman)

1. Orofacial pain attributed to disorders of dentoalveolar and anatomically related structures
Maria Pigg, Sweden (*chairman*);
Alan Law, USA;
Donald Nixdorf, USA;
Tara Renton, UK;
Yair Sharav, Israel

2. Myofascial orofacial pain
Peter Svensson, Denmark (*chairman*);
Malin Ernberg, Sweden;
Chris Peck, Australia

3. Temporomandibular joint (TMJ) pain
Per Alstergren, Sweden (*chairman*);
Ghabi Kaspo, USA;
Frank Lobbezoo, Netherlands;
Ambra Michelotti, Italy

4. Orofacial pain attributed to lesion or disease of the cranial nerves
Lene Baad-Hansen, Denmark (*chairman*);
Eli Eliav, USA;
Yoshiki Imamura, Japan

5. Orofacial pains resembling presentations of primary headaches
Rafael Benoliel, USA (*chairman*);
Paulo Conti, Brazil;
Arne May, Germany

6. Idiopathic orofacial pain
Thomas List, Sweden (*chairman*);
Justin Durham, England;
Jean-Paul Goulet, Canada;
Satu Jääskeläinen, Finland

7. Psychosocial assessment of patients with orofacial pain
Richard Ohrbach, USA

Acknowledgements

There has been no commercial sponsorship of the International Classification of Orofacial Pain.

We gratefully acknowledge Timothy Steiner for desk editing to align with the principles, structure and format of ICHD.

Table of Contents

Preface

We are proud to present this first edition of the *International Classification of Orofacial Pain* (ICOP).

There has, until now, been no comprehensive, internationally accepted classification that deals with orofacial pain. While, anatomically, the face is clearly part of the head, we have found far too many cases of misdiagnosis and resultant misdirected treatment that a clear diagnostic classification might have helped avoid. Anatomical boundaries, and associated medical specialty demarcations, contribute to the problem. For example, the International Headache Society (IHS) defines facial pain as 'pain below the orbitomeatal line, anterior to the pinnae and above the neck'. Other definitions of facial pain additionally include the forehead, while the term 'orofacial pain' necessarily includes all the structures in the oral cavity. At the same time, headache is often referred to orofacial regions, and vice versa. 'Headaches' may be located exclusively within the orofacial region, and cause significant diagnostic difficulties, while orofacial pains that are referred to the head present complex clinical phenotypes.

These were some of the factors that made it clear to us that a classification of orofacial pains (OFPs) was needed. A fundamental principle in this first classification is that the characteristics of the disorders, not their location (head versus face), should guide the new conceptualization and diagnostic criteria.

While creating this first edition of ICOP, we were cognizant that the *International Classification of Headache Disorders, 3rd edition* (ICHD-3) thoroughly classifies primary and secondary headaches, and we refer the reader to this for these entities. Moreover, to make ICOP a useful tool for researchers and clinicians accustomed to using ICHD-3, we have adopted the hierarchical design and classification style of ICHD-3. The *Diagnostic Criteria for Temporomandibular Disorders* (DC/TMD) is a well-tested and established classification that includes regional myalgias and temporomandibular joint (TMJ) pains. We have adopted the DC/TMD criteria, only including the painful TMDs and modifying the presentation style of these criteria to that of ICHD-3. Overall, ICOP is also aligned to the *International Classification of Diseases 11th Revision* (ICD-11)/International Association for the Study of Pain (IASP) criteria for orofacial pains and headaches.

The aim is therefore to create a tool that will enhance the research and clinical management of orofacial pain. Additionally, we are confident that the methodology we have adopted will bring professionals working on head, orofacial, eye, nose, sinus and neck pain closer, and encourage active collaboration.

The members of the ICOP classification committee represent the major associations involved in orofacial and head pain, and are a truly international group, strengthening the future of ICOP. The road to this first edition began in 2016, when we first met at the World Congress of the International Association for the Study of Pain (IASP) in Yokohama, Japan. In the one-day meeting, we discussed the structure of ICOP and established workgroups. A follow-up meeting in 2017 was held in Rutgers School of Dental Medicine, USA, where we examined the evidence, or lack of it, to establish the individual entities that now make up ICOP. The result is a classification backed in many areas by strong evidence and in others by expert opinion, which will encourage and guide research. It permits a period of testing, allowing identification and correction of mistakes and enabling a broad input into ICOP from researchers and practitioners in the field.

Rafael Benoliel
Arne May
Peter Svensson
Facial Pain Classification Committee

Using ICOP

Because ICOP is modelled on ICHD-3, the instructions for use are similar. Many ICHD-3 users will therefore find ICOP easy to use. Like ICHD-3, the document is intended as a tool to consult, particularly for research, but also for the clinical diagnosis and management of orofacial pains (OFPs).

ICOP will serve as a comprehensive research and diagnostic manual and will be particularly useful when the diagnosis is uncertain, or when the clinician is unaware that such a clinical presentation exists. So, we do recommend that practitioners and researchers read through the classification. Moreover, ICOP joins ICHD-3 and the ICD-11/IASP in establishing clear terminology that will allow communication and data sharing in an unambiguous manner. For research, the classification is indispensable: every patient entered into a research project, be it a drug trial or a study of pathophysiology or biochemistry, must fulfil a set of established diagnostic criteria.

This classification is hierarchical, allowing the user to establish a diagnosis from only the first-digit level, or extending to the fifth, sixth or even seventh digit. The level of resolution in diagnostic coding clearly depends on the intended use. In general practice, only the first- or second-digit diagnoses are usually applied, while in specialist practice a diagnosis including fourth- or fifth-digit (and, on occasion, sixth- or seventh-digit) levels is appropriate.

We deal with primary and secondary OFPs within the same sections. This is contrary to ICHD but, in our opinion, a more efficient manner of presenting the various OFPs – in part because primary and secondary designations become difficult to differentiate in these overlapping disorders. For similar reasons, the strict ICHD criterial structure for secondary disorders – with Criterion A depicting the pain, Criterion B identifying the presumed cause and Criterion C stipulating the evidence of causation of A by B – does not work well for OFPs. In particular, the temporal relation between onset of pain and onset of the presumed cause – the mainstay of Criterion C in ICHD – is often so highly variable that it has no evidential value. In many cases, attribution rests on clinical plausibility of causation together with reliance on the last criterion: 'Not better accounted for by another ICOP diagnosis' (see below).

Rules of use

1. Each distinct type, subtype or subform of OFP and headache that the patient has must be separately diagnosed and coded. For example, a severely affected patient may receive multiple diagnoses and codes: 2.1.2.3.2 *Chronic persistent primary myofascial pain with pain referral* and 3.1.4.1 *Chronic persistent primary TMJ pain without referred pain*, as well, possibly, as 1.1 *Migraine without aura* from ICHD-3.
2. When a patient receives more than one diagnosis, these should be listed in the order of importance to the patient: that is, which of the diagnoses is causing the most suffering and disability in the patient's view.
3. When it is unclear which type of OFP a particular patient is experiencing, other available information should be used in addition to the diagnostic criteria to decide the more likely diagnosis. This could include the longitudinal pain history (how and when did the pain start?), the family history, the effect of drugs, menstrual relationship, age, sex and a range of other features.
4. To receive a particular OFP diagnosis, the patient must, in many cases, experience a minimum number of attacks of, or number of days with, that pain. This number is then specified in the diagnostic criteria for the OFP type or subtype. OFP diagnoses must fulfil a number of other requirements described within the criteria under separate letter headings: A, B, C, etc. Some letter headings are monothetic: that is, they express a single requirement. Other letter headings are polythetic, requiring, for example, any two out of four listed characteristics. This structure has been adopted from ICHD-3.
5. The frequency of OFP disorders varies widely, from occurring only once in every 1–2 years to daily pain. The severity of attacks also varies. Other than for myofascial pains and TMJ pains, ICOP does not provide a possibility to code for frequency. None of the diagnostic criteria include routine assessment of severity and frequency, but we recommend that frequency and severity be assessed and specified.
6. The last criterion for almost every disorder is 'Not better accounted for by another ICOP diagnosis' (sometimes, 'Not better accounted for by another ICOP or ICHD-3 diagnosis'). Consideration of other possible diagnoses (the *differential diagnosis*) is a routine part of the clinical diagnostic process. When an OFP appears to fulfil the criteria for a particular disorder, this last criterion is a reminder always to consider other diagnoses that might better explain the OFP. In order to accomplish this goal of differential diagnosis, the clinical diagnostic process may need to consider other disorders outside the ICOP framework, elaborate on the pain history, and use clinical tests beyond those implied in the ICOP criteria. Referred pain from one

structure to the other in the orofacial pain region is extremely common, but classifying all of these is beyond the scope of ICOP.

This requirement – always to consider other possible diagnoses – is similarly essential in research that demands inclusion only of definite cases. Not all research falls into this category. In particular, epidemiological studies may be unable to apply this last criterion (headache epidemiology has generally been unable to apply the matching criterion in ICHD-3). Studies that abandon this last criterion should at least acknowledge the fact in their descriptions of methodology.

7. When a patient is suspected of having more than one OFP, it is highly recommended that they complete a diagnostic daily pain diary. It has been shown that such pain diaries not only improve diagnostic accuracy but also lead to a more precise judgement of medication consumption. Diaries are typically recommended for a month, during which time, for each pain episode, the important characteristics are recorded. Additionally, the diary helps in judging the balance between different OFP types, subtypes or subforms. Finally, using the diary is an important tool in explaining to the patient how to distinguish between different OFPs, be aware of medication consumption, note triggering factors and become a more reliable source of follow-up information.

ICOP code	Diagnosis
1.	**Orofacial pain attributed to disorders of dentoalveolar and anatomically related structures**
1.1	Dental pain
1.1.1	Pulpal pain
1.1.1.1	Pulpal pain attributed to hypersensitivity
1.1.1.1.1	Pulpal pain attributed to a crack in the enamel
1.1.1.1.2	Pulpal pain attributed to exposed dentin
1.1.1.1.2.1	Pulpal pain attributed to tooth wear or abrasion
1.1.1.1.2.2	Pulpal pain attributed to fracture resulting in exposed dentin
1.1.1.1.2.3	Pulpal pain attributed to developmental dental hard-tissue defect
1.1.1.1.3	Pulpal pain attributed to dental procedure
1.1.1.1.3.1	Pulpal pain attributed to extensive removal of dentin
1.1.1.1.3.2	Pulpal pain attributed to placement of a restoration
1.1.1.1.3.3	Pulpal pain attributed to hyperocclusion or hyperarticulation following dental restorative procedure
1.1.1.1.4	Pulpal pain attributed to central sensitization
1.1.1.1.5	Pulpal pain attributed to hypersensitivity due to other cause
1.1.1.2	Pulpal pain attributed to pulp exposure due to dental trauma
1.1.1.3	Pulpal pain attributed to pulpitis (pulpal inflammation)
1.1.1.3.1	Pulpal pain attributed to reversible pulpitis due to infection of dentin
1.1.1.3.1.1	Pulpal pain attributed to reversible pulpitis due to caries not extending to the pulp
1.1.1.3.1.2	Pulpal pain attributed to reversible pulpitis due to dental hard-tissue fracture with exposure of dentin
1.1.1.3.1.3	Pulpal pain attributed to reversible pulpitis due to a tooth crack without evidence of missing tooth substance
1.1.1.3.2	Pulpal pain attributed to irreversible pulpitis due to infection of dentin
1.1.1.3.2.1	Pulpal pain attributed to irreversible pulpitis due to caries extending to the pulp
1.1.1.3.2.2	Pulpal pain attributed to irreversible pulpitis due to dental hard-tissue fracture without pulp exposure
1.1.1.3.2.3	Pulpal pain attributed to irreversible pulpitis due to tooth crack without evidence of missing tooth substance
1.1.1.3.3	Pulpal pain attributed to irreversible pulpitis due to infection of the dental pulp
1.1.1.3.3.1	Pulpal pain attributed to irreversible pulpitis due to carious pulp exposure and infection of the pulp
1.1.1.3.3.2	Pulpal pain attributed to irreversible pulpitis due to dental hard-tissue fracture with pulp exposure
1.1.1.3.4	Pulpal pain attributed to pulpitis due to external cervical root resorption
1.1.1.3.5	Pulpal pain attributed to pulpitis due to other cause
1.1.1.4	Pulpal pain attributed to systemic cause
1.1.2	Periodontal pain
1.1.2.1	Periodontal pain attributed to periodontitis (periodontal inflammation)
1.1.2.1.1	Periodontal pain attributed to traumatically induced periodontal inflammation
1.1.2.1.1.1	Periodontal pain attributed to hyperocclusion or hyperarticulation
1.1.2.1.1.2	Postoperative periodontal pain
1.1.2.1.1.3	Periodontal pain attributed to accidental dental trauma
1.1.2.1.1.4	Periodontal pain attributed to other trauma or injury
1.1.2.1.2	Periodontal pain attributed to apical periodontitis due to endodontic disease
1.1.2.1.2.1	Periodontal pain attributed to pulpal inflammation
1.1.2.1.2.2	Periodontal pain attributed to endodontic infection
1.1.2.1.2.2.1	Periodontal pain attributed to intraradicular endodontic infection
1.1.2.1.2.2.2	Periodontal pain attributed to extraradicular endodontic infection
1.1.2.1.3	Periodontal pain attributed to periodontal disease
1.1.2.1.3.1	Periodontal pain attributed to chronic periodontitis
1.1.2.1.3.2	Periodontal pain attributed to aggressive periodontitis
1.1.2.1.3.3	Periodontal pain attributed to periodontitis as a manifestation of systemic disorder
1.1.2.1.3.3.1	Periodontal pain attributed to haematological disorder
1.1.2.1.3.3.2	Periodontal pain attributed to genetic disorder
1.1.2.1.3.3.3	Periodontal pain attributed to unspecified systemic disorder

1.1.2.1.3.4	Periodontal pain attributed to necrotizing ulcerative periodontitis (NUP)
1.1.2.1.3.5	Periodontal pain attributed to periodontal abscess
1.1.2.1.4	Periodontal pain attributed to apical and marginal periodontitis due to combined endodontic infection and periodontal disease
1.1.2.1.5	Periodontal pain attributed to infective peri-implantitis
1.1.2.2	Periodontal pain attributed to local non-inflammatory cause
1.1.3	Gingival pain
1.1.3.1	Gingival pain attributed to gingivitis (gingival inflammation)
1.1.3.1.1	Gingival pain attributed to trauma
1.1.3.1.2	Gingival pain attributed to infection
1.1.3.1.2.1	Gingival pain attributed to bacterial infection
1.1.3.1.2.2	Gingival pain attributed to viral infection
1.1.3.1.2.3	Gingival pain attributed to fungal infection
1.1.3.1.3	Gingival pain attributed to autoimmunity
1.1.3.1.4	Gingival pain attributed to hypersensitivity or allergic reaction
1.1.3.1.5	Gingival pain attributed to gingival inflammation due to other cause
1.1.3.2	Gingival pain attributed to malignant lesion
1.2	Oral mucosal, salivary gland and jaw bone pains
1.2.1	Oral mucosal pain
1.2.1.1	Oral mucosal pain attributed to oral mucosal inflammation
1.2.1.1.1	Oral mucosal pain attributed to trauma or injury
1.2.1.1.1.1	Oral mucosal pain attributed to non-iatrogenic trauma or injury
1.2.1.1.1.2	Oral mucosal pain attributed to surgical or other local iatrogenic injury
1.2.1.1.1.3	Oral mucosal pain attributed to radiation or chemotherapy
1.2.1.1.2	Oral mucosal pain attributed to infection
1.2.1.1.2.1	Oral mucosal pain attributed to bacterial infection
1.2.1.1.2.2	Oral mucosal pain attributed to viral infection
1.2.1.1.2.3	Oral mucosal pain attributed to fungal infection
1.2.1.1.3	Oral mucosal pain attributed to autoimmunity
1.2.1.1.4	Oral mucosal pain attributed to hypersensitivity or allergic reaction
1.2.1.1.5	Oral mucosal pain attributed to oral mucosal inflammation due to other cause
1.2.1.2	Oral mucosal pain attributed to malignant lesion
1.2.2	Salivary gland pain
1.2.2.1	Salivary gland pain attributed to obstructive cause
1.2.2.2	Salivary gland pain attributed to infection
1.2.2.2.1	Salivary gland pain attributed to bacterial infection
1.2.2.2.2	Salivary gland pain attributed to viral infection
1.2.2.3	Salivary gland pain attributed to recurrent juvenile parotitis
1.2.2.4	Salivary gland pain attributed to immunologically mediated disorder
1.2.2.5	Salivary gland pain attributed to other cause
1.2.3	Jaw bone pain
1.2.3.1	Jaw bone pain attributed to trauma or injury
1.2.3.2	Jaw bone pain attributed to infection
1.2.3.2.1	Jaw bone pain attributed to bacterial infection
1.2.3.2.2	Jaw bone pain attributed to viral infection
1.2.3.2.3	Jaw bone pain attributed to fungal infection
1.2.3.3	Jaw bone pain attributed to local benign lesion
1.2.3.4	Jaw bone pain attributed to malignant lesion
1.2.3.4.1	Jaw bone pain attributed to local malignancy
1.2.3.4.2	Jaw bone pain attributed to remote malignancy
1.2.3.5	Jaw bone pain attributed to therapy
1.2.3.6	Jaw bone pain attributed to systemic disease
2.	**Myofascial orofacial pain**
2.1	Primary myofascial orofacial pain
2.1.1	Acute primary myofascial orofacial pain
2.1.2	Chronic primary myofascial orofacial pain
2.1.2.1	Chronic infrequent primary myofascial orofacial pain
2.1.2.2	Chronic frequent primary myofascial orofacial pain
2.1.2.2.1	Chronic frequent primary myofascial orofacial pain without pain referral
2.1.2.2.2	Chronic frequent primary myofascial orofacial pain with pain referral

2.1.2.3	Chronic highly frequent primary myofascial orofacial pain
2.1.2.3.1	Chronic highly frequent primary myofascial orofacial pain without pain referral
2.1.2.3.2	Chronic persistent primary myofascial orofacial pain with pain referral
2.2	Secondary myofascial orofacial pain
2.2.1	Myofascial orofacial pain attributed to tendonitis
2.2.2	Myofascial orofacial pain attributed to myositis
2.2.3	Myofascial orofacial pain attributed to muscle spasm
3.	**Temporomandibular joint (TMJ) pain**
3.1	Primary temporomandibular joint pain
3.1.1	Acute primary temporomandibular joint pain
3.1.2	Chronic primary temporomandibular joint pain
3.1.2.1	Chronic infrequent primary temporomandibular joint pain
3.1.2.2	Chronic frequent primary temporomandibular joint pain
3.1.2.2.1	Chronic frequent primary temporomandibular joint pain without pain referral
3.1.2.2.2	Chronic frequent primary temporomandibular joint pain with pain referral
3.1.2.3	Chronic highly frequent primary temporomandibular joint pain
3.1.2.3.1	Chronic highly frequent primary temporomandibular joint pain without pain referral
3.1.2.3.2	Chronic highly frequent primary temporomandibular joint pain with pain referral
3.2	Secondary temporomandibular joint pain
3.2.1	Temporomandibular joint pain attributed to arthritis
3.2.1.1	Temporomandibular joint pain attributed to non-systemic arthritis
3.2.1.2	Temporomandibular joint pain attributed to systemic arthritis
3.2.2	Temporomandibular joint pain attributed to disc displacement
3.2.2.1	Temporomandibular joint pain attributed to disc displacement with reduction
3.2.2.1.1	Temporomandibular joint pain attributed to disc displacement with reduction, with intermittent locking
3.2.2.2	Temporomandibular joint pain attributed to disc displacement without reduction
3.2.3	Temporomandibular joint pain attributed to degenerative joint disease
3.2.4	Temporomandibular joint pain attributed to subluxation
4.	**Orofacial pain attributed to lesion or disease of the cranial nerves**
4.1	Pain attributed to lesion or disease of the trigeminal nerve
4.1.1	Trigeminal neuralgia
4.1.1.1	Classical trigeminal neuralgia
4.1.1.1.1	Classical trigeminal neuralgia, purely paroxysmal
4.1.1.1.2	Classical trigeminal neuralgia with concomitant continuous pain
4.1.1.2	Secondary trigeminal neuralgia
4.1.1.2.1	Trigeminal neuralgia attributed to multiple sclerosis
4.1.1.2.2	Trigeminal neuralgia attributed to space-occupying lesion
4.1.1.2.3	Trigeminal neuralgia attributed to other cause
4.1.1.3	Idiopathic trigeminal neuralgia
4.1.1.3.1	Idiopathic trigeminal neuralgia, purely paroxysmal
4.1.1.3.2	Idiopathic trigeminal neuralgia with concomitant continuous pain
4.1.2	Other trigeminal neuropathic pain
4.1.2.1	Trigeminal neuropathic pain attributed to herpes zoster
4.1.2.2	Trigeminal postherpetic neuralgia
4.1.2.3	Post-traumatic trigeminal neuropathic pain
4.1.2.3.1	Probable post-traumatic trigeminal neuropathic pain
4.1.2.4	Trigeminal neuropathic pain attributed to other disorder
4.1.2.4.1	Probable trigeminal neuropathic pain attributed to other disorder
4.1.2.5	Idiopathic trigeminal neuropathic pain
4.2	Pain attributed to lesion or disease of the glossopharyngeal nerve
4.2.1	Glossopharyngeal neuralgia
4.2.1.1	Classical glossopharyngeal neuralgia
4.2.1.2	Secondary glossopharyngeal neuralgia
4.2.1.3	Idiopathic glossopharyngeal neuralgia
4.2.2	Glossopharyngeal neuropathic pain
4.2.2.1	Glossopharyngeal neuropathic pain attributed to a known cause
4.2.2.2	Idiopathic glossopharyngeal neuropathic pain

5.	**Orofacial pains resembling presentations of primary headaches**
5.1	Orofacial migraine
5.1.1	Episodic orofacial migraine
5.1.2	Chronic orofacial migraine
5.2	Tension-type orofacial pain
5.3	Trigeminal autonomic orofacial pain
5.3.1	Orofacial cluster attacks
5.3.1.1	Episodic orofacial cluster attacks
5.3.1.2	Chronic orofacial cluster attacks
5.3.2	Paroxysmal hemifacial pain
5.3.2.1	Episodic paroxysmal hemifacial pain
5.3.2.2	Chronic paroxysmal hemifacial pain
5.3.3	Short-lasting unilateral neuralgiform facial pain attacks with cranial autonomic symptoms (SUNFA)
5.3.3.1	Episodic SUNFA
5.3.3.2	Chronic SUNFA
5.3.4	Hemifacial continuous pain with autonomic symptoms
5.4	Neurovascular orofacial pain
5.4.1	Short-lasting neurovascular orofacial pain
5.4.2	Long-lasting neurovascular orofacial pain
6.	**Idiopathic orofacial pain**
6.1	Burning mouth syndrome (BMS)
6.1.1	Burning mouth syndrome without somatosensory changes
6.1.2	Burning mouth syndrome with somatosensory changes
6.1.3	Probable burning mouth syndrome
6.2	Persistent idiopathic facial pain (PIFP)
6.2.1	Persistent idiopathic facial pain without somatosensory changes
6.2.2	Persistent idiopathic facial pain with somatosensory changes
6.2.3	Probable persistent idiopathic facial pain
6.3	Persistent idiopathic dentoalveolar pain
6.3.1	Persistent idiopathic dentoalveolar pain without somatosensory changes
6.3.2	Persistent idiopathic dentoalveolar pain with somatosensory changes
6.3.3	Probable persistent idiopathic dentoalveolar pain
6.4	Constant unilateral facial pain with additional attacks (CUFPA)
7.	**Psychosocial assessment of patients with orofacial pain**

Classification, with diagnostic criteria

1. Orofacial pain attributed to disorders of dentoalveolar and anatomically related structures

Description:
Orofacial pain caused by diseases, injuries or abnormal functioning of the tooth pulp, periodontium, gingiva(e), oral mucosa, salivary glands or jaw bone tissue, or by normal functioning of the tooth pulp signalling risk of tooth damage.

General comments:
Pain originating in dentoalveolar and associated structures is the most common reason for the complaint of pain in the orofacial region. In general, the pain is nociceptive and/or inflammatory in nature, and usually *acute*, meaning that it lasts less than 3 months. When the underlying disorder is adequately treated, the symptom of pain does not persist for a prolonged period of time. The pain may be continuous, recurrent or occasional. In many cases, the natural history of the underlying disorder allows for fluctuation in all symptoms, including pain, which means this type of pain may sometimes be described as *episodic* (occurring on fewer than 15 days per month, whether or not for more than 3 months). When the pain is present for more than 3 months *and* on at least 15 days per month, it is considered as *chronic*.

Since pain associated with dentoalveolar and anatomically related structures is mainly a symptom of disease, it may also appear relevant to categorize it in relation to treatment. If the pain-inducing underlying disorder is not treated at all, the acute pain will usually remain and eventually become chronic. The same occurs if the underlying disorder is ineffectively or insufficiently treated, since the disorder (such as a local infection, neoplasm or systemic disorder) and the accompanying pain also may remain for longer than 3 months.

In general, the distinction between acute and chronic pain is important, since chronic pain often requires different management and has a less favourable prognosis. However, it is unknown whether the acute, episodic and chronic forms of dentoalveolar pain (and other types dealt with here) differ in any clinically meaningful aspect except duration. Based on the lack of data supporting a distinction as relevant from the perspective of treatment or prognosis, the issue of acute versus chronic is not reflected in this section of ICOP. For research aiming to compare, for example, dentoalveolar pain of short versus long duration, it is recommended to use the distinction of *acute, episodic* or *chronic* pain described above consistently with IASP/ICD-11 and ICHD-3.

If evidence of important differences emerges in the future, the decision not to separate pain conditions based on time in this section must be re-evaluated.

1.1 Dental pain

Description:
Pain caused by lesions or disorders affecting one or more teeth and/or their immediately surrounding and supporting structures: the tooth pulp, periodontium and gingivae.

1.1.1 Pulpal pain

Description:
Pain caused by a lesion or disorder involving the tooth pulp.

Diagnostic criteria:

A. Any pain in a tooth fulfilling criterion C
B. Clinical, laboratory, imaging and/or anamnestic evidence of a lesion, disease or trauma[1] known to produce pulpal pain
C. Evidence of causation demonstrated by both of the following:
   1. location of the pain corresponds to the site(s) of the lesion, disease or trauma[2]
   2. either or both of:
      a) pain developed in temporal relation to the appearance of the lesion or onset of the disease or trauma, or led to its discovery
      b) pain is exacerbated by physical stimulus[3] applied to the affected tooth
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. The lesion, disease or trauma is specified in each subform.
2. Pain may also refer and/or radiate to other ipsilateral orofacial locations.
3. The stimulus may be mechanical, thermal or chemical, as specified in some subforms.

Comment:
1.1.1 *Pulpal pain* may be associated with any type of pulpal injury or disease. The pain is predominantly inflammatory and secondary to external or internal events.

1.1.1.1 Pulpal pain attributed to hypersensitivity

Description:
Pulpal pain due to hypersensitivity occurring in a clinically normal pulp.

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1 *Pulpal pain*, and criteria B and C below
B. Pain has all the following characteristics:
   1. evoked by external stimuli[1]
   2. subsiding within a few seconds
   3. either or both of:
      a) a sharp, deep sensation
      b) poorly localized[2]
C. Causation is plausible based on anatomical, functional and/or temporal association[3]
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Hot, cold and sweet are among the external stimuli that may produce pain.
2. Often only to an approximate area within two or three teeth adjacent to the affected tooth; occasionally the patient is unable to distinguish whether the pain originates from the mandible or the maxilla.
3. This criterion cascades down to all subforms.

1.1.1.1.1 Pulpal pain attributed to a crack in the enamel

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.1 *Pulpal pain attributed to hypersensitivity*, and criterion C below
B. A crack or incomplete fracture of the affected tooth, involving the enamel, has been diagnosed by visual identification[1] of crack line(s)
C. At least one of the following:
   1. sharp pain upon biting
   2. pain on release of occlusal biting pressure or external application of force
   3. cold hypersensitivity
D. Not better accounted for by another ICOP diagnosis.

Note:

1. When necessary, visual identification can be aided by magnification, light enhancement and/or visualization with dye.

1.1.1.1.2 Pulpal pain attributed to exposed dentin

Diagnostic criteria:

A. Pain in a tooth[1] fulfilling criteria for 1.1.1.1 *Pulpal pain attributed to hypersensitivity*
B. Dentin surface of the affected tooth is exposed
C. Not better accounted for by another ICOP diagnosis.

Note:

1. The pain can usually be reproduced by scratching the exposed dentin with a dental explorer or by air blasting.

1.1.1.1.2.1 Pulpal pain attributed to tooth wear or abrasion

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.1.2 *Pulpal pain attributed to exposed dentin*
B. Clinical evidence of wear or abrasion of the affected tooth[1]
C. Not better accounted for by another ICOP diagnosis.

Note:

1. Smooth, flat surfaces that are not contoured with the natural shape of the anatomic crown of the tooth.

1.1.1.1.2.2 Pulpal pain attributed to fracture resulting in exposed dentin

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.1.2 *Pulpal pain attributed to exposed dentin*
B. Fracture of the affected tooth involving the enamel, root cementum, dentin or any combination thereof has been diagnosed[1]
C. Not better accounted for by another ICOP diagnosis.

Note:

1. Diagnosis is based on clinical and/or radiographic observations.

1.1.1.1.2.3 Pulpal pain attributed to developmental dental hard-tissue defect

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.1.2 *Pulpal pain attributed to exposed dentin*
B. A developmental defect of the affected tooth involving the enamel, root cementum and/or dentin and

known to be able to cause pulpal pain[1] has been diagnosed
C. Not better accounted for by another ICOP diagnosis.

Note:

1. Such developmental defects include local hypomineralization or hypomaturation of enamel, amelogenesis imperfecta, dentinogenesis imperfecta and a large number of other, rarer defects.

1.1.1.1.3 Pulpal pain attributed to dental procedure

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.1 *Pulpal pain attributed to hypersensitivity*
B. Dental treatment has recently[1] been applied to the affected tooth
C. Not better accounted for by another ICOP diagnosis.

Note:

1. Pain onset is typically hours to days after the dental procedure.

1.1.1.1.3.1 Pulpal pain attributed to extensive removal of dentin

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.1.3 *Pulpal pain attributed to dental procedure*
B. Recent[1] removal of dentin has occurred in the affected tooth, which was either or both of the following:
   1. deep (i.e. in close proximity to the pulp)
   2. wide (i.e. opening up dentinal tubules in a large area)
C. Not better accounted for by another ICOP diagnosis.

Note:

1. Pain onset is typically hours to days after the dental procedure.

1.1.1.1.3.2 Pulpal pain attributed to placement of a restoration

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.1.3 *Pulpal pain attributed to dental procedure*
B. Recent[1] placement of a direct or indirect dental restoration in the affected tooth
C. Not better accounted for by another ICOP diagnosis.

Note:

1. Pain onset is typically hours to days after the dental procedure.

1.1.1.1.3.3 Pulpal pain attributed to hyperocclusion or hyperarticulation following dental restorative procedure

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.1.3 *Pulpal pain attributed to dental procedure*
B. A restorative procedure[1] has caused the affected tooth to be in hyperocclusion and/or hyperarticulation
C. Not better accounted for by another ICOP diagnosis.

Note:

1. Restorative procedures include temporization, dental restoration and prosthodontic replacement.

1.1.1.1.4 Pulpal pain attributed to central sensitization

Diagnostic criteria:

A. Pain in one or more teeth fulfilling criteria for 1.1.1.1 *Pulpal pain attributed to hypersensitivity*, and criteria C and D below
B. Both of the following:
   1. another orofacial, neck or widespread bodily pain condition has been diagnosed
   2. signs of central sensitization[1] are present
C. One or both of the following:
   1. pain presents spontaneously
   2. pain presents and/or fluctuates with the other pain condition
D. Not consistently relieved by local anaesthesia or peripherally acting analgesics
E. Not better accounted for by another ICOP diagnosis.

Note:

1. Signs include pain referral, temporal pain summation and allodynia.

Comments:
1.1.1.1.4 *Pulpal pain attributed to central sensitization* can present in several teeth simultaneously, or start in

one tooth and then spread to other teeth. It can be continuous or recurrent, and present for long periods; often it is chronic.

Pain symptoms may range from dentinal hypersensitivity to lingering pain, indicative of pulpitis, and are often accompanied by autonomic signs (see 5.1 *Orofacial migraine*).

Successful treatment of the other pain condition(s) and associated psychological symptoms often leads to reduced tooth pain. Desensitizing dental treatment may in some cases lead to pain reduction or relief.

1.1.1.1.5 Pulpal pain attributed to hypersensitivity due to other cause

Diagnostic criteria:

A. Pain in one or more teeth fulfilling criteria for 1.1.1.1 *Pulpal pain attributed to hypersensitivity*, and criterion C below
B. A disorder, known to be able to cause pulpal pain but other than those of 1.1.1.1.1 to 1.1.1.1.4, has been diagnosed
C. Causation is plausible based on anatomical, functional and/or temporal association
D. Not better accounted for by another ICOP diagnosis.

1.1.1.2 Pulpal pain attributed to pulp exposure due to dental trauma

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1 *Pulpal pain*, and criterion C below
B. Dental trauma has caused any of the following, exposing vital pulp tissue in the affected tooth:
   1. fracture involving enamel, dentin and pulp (complicated crown fracture)
   2. fracture involving root cementum, dentin and pulp (complicated root fracture)
   3. fracture involving enamel, root cementum, dentin and pulp (complicated crown-root fracture)
C. Pain developed within minutes to hours after the trauma
D. Not better accounted for by another ICOP diagnosis.

Comment:
1.1.1.2 *Pulpal pain attributed to pulp exposure due to dental trauma* is mild to moderate. It is typically exacerbated by air, liquids or pressure on the exposed pulp tissue, but subsides when the stimulus ceases. However, in the immediate post-trauma period, there is a lack of temperature sensitivity, spontaneous pain or radiating pain, as these symptoms typically occur later and are associated with inflammation.

1.1.1.3 Pulpal pain attributed to pulpitis (pulpal inflammation)

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1 *Pulpal pain*, and criterion C below
B. Pulpitis in the affected tooth[1] has been diagnosed
C. Causation is plausible on the basis of anatomical, functional and/or temporal association[2]
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Pulpitis may be due to trauma or infection, as specified in each subform.
2. This criterion cascades down to all subforms.

Comments:
1.1.1.3 *Pulpal pain attributed to pulpitis* can vary from mild to severe and can be related to the severity of the inflammation. However, severe pulpal inflammation can also be asymptomatic.

The suggested diagnostic criteria for reversible or irreversible pulpitis presented in the subforms below have not been scientifically validated, and the presence and characteristics of symptoms appear poorly related to the condition of the pulp. When the pulp has been directly exposed to the oral microbiota for a period, it lacks the ability to heal and pulpitis is considered to be irreversible. Therefore, when associated with caries, pulpitis is considered potentially reversible as long as a zone of functionally intact dentin separates the bacterial front from the vital pulp tissue, and potentially irreversible when no such zone exists.

1.1.1.3.1 Pulpal pain attributed to reversible pulpitis due to infection of dentin

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.3 *Pulpal pain attributed to pulpitis*
B. Reversible[1] pulpitis due to infection of dentin[2] has been diagnosed in the affected tooth
C. Not better accounted for by another ICOP diagnosis.

Notes:

1. Reversibility is diagnosed on the basis of clinical and/or radiographic evidence of a zone of intact dentin covering the pulp
2. Infection is evidenced by the presence of caries, or dentin exposed to the microbiota of the oral cavity for a period.

Comment:
Pain attributed to reversible pulpitis has been described as typically mild, not spontaneous, and provoked by changes in temperature. When evoked by thermal (cold or heat) or mechanical stimulus (probing, drilling), pain is typically short-lasting and does not outlast the stimulus. It responds to peripherally acting analgesics (nonsteroidal anti-inflammatory drugs; NSAIDs).

1.1.1.3.1.1 Pulpal pain attributed to reversible pulpitis due to caries not extending to the pulp

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.3.1 *Pulpal pain attributed to reversible pulpitis due to infection of dentin*
B. Caries has been diagnosed in the affected tooth, with no clinical or radiographic evidence of extension to the pulp
C. Not better accounted for by another ICOP diagnosis.

Comment:
In addition to thermal sensitivity, the pain may be evoked by pressure on the carious dentin.

1.1.1.3.1.2 Pulpal pain attributed to reversible pulpitis due to dental hard-tissue fracture with exposure of dentin

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.3.1 *Pulpal pain attributed to reversible pulpitis due to infection of dentin*
B. Clinical and/or radiographic evidence of any of the following in the affected tooth, with exposure of dentin:
   1. fracture involving enamel alone
   2. fracture involving enamel and dentin
   3. fracture involving root cementum alone
   4. fracture involving root cementum and dentin
   5. fracture involving enamel, root cementum and dentin
C. Not better accounted for by another ICOP diagnosis.

Comment:
In addition to thermal sensitivity, the pain may be evoked by scratching the surface of the infected dentin.

1.1.1.3.1.3 Pulpal pain attributed to reversible pulpitis due to a tooth crack without evidence of missing tooth substance

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.3.1 *Pulpal pain attributed to reversible pulpitis due to infection of dentin*, and criterion C below
B. A crack or incomplete fracture of the affected tooth, involving the enamel or enamel and dentin, has been diagnosed[1]
C. Evidence of causation demonstrated by at least one of the following:
   1. sharp pain upon biting[2]
   2. pain on release of occlusal biting pressure or external application of force[2]
   3. cold hypersensitivity[2]
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Diagnosis may be by visual identification of crack line(s), aided by magnification, light enhancement or visualization with dye, and/or by radiographic or other imaging.
2. The pain in each case does not outlast the application of the stimulus.

Comment:
Cracked teeth sometimes have deep probing depths associated with the crack.

1.1.1.3.2 Pulpal pain attributed to irreversible pulpitis due to infection of dentin

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.3 *Pulpal pain attributed to pulpitis*
B. Irreversible[1] pulpitis due to infection of the dentin[2] has been diagnosed in the affected tooth
C. Not better accounted for by another ICOP diagnosis.

Notes:

1. Irreversibility is diagnosed by clinical and/or radiographic evidence that no zone of intact dentin covers

the pulp, and/or by any of the following pain characteristics:
   a) occurs spontaneously
   b) is continuous
   c) outlasts stimulation (thermal: cold or heat; or mechanical – probing or drilling) of the pulp by more than a few seconds
   d) is of severe intensity
   e) is poorly responsive to NSAIDS.
2. As evidenced by presence of caries or dentin exposed to the oral cavity microbiota for a period.

Comments:
Pain attributed to irreversible pulpitis can be exacerbated by changes in temperature and may also be associated with biting or percussion sensitivity. When evoked, it outlasts the duration of the stimulus.

However, the presence of pain is poorly correlated with the status of the pulp. The value of symptoms to determine the condition of the pulp (reversibly or irreversibly inflamed) is debated and controversial, with scientific evidence scarce. Severe continuous pain that does not respond to analgesics (NSAIDs) may indicate irreversible inflammation and need for invasive treatment.

1.1.1.3.2.1 Pulpal pain attributed to irreversible pulpitis due to caries extending to the pulp

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.3.2 *Pulpal pain attributed to irreversible pulpitis due to infection of dentin*
B. Deep caries has been diagnosed in the affected tooth, with clinical and/or radiographic evidence of likely or definite extension to the pulp
C. Not better accounted for by another ICOP diagnosis.

1.1.1.3.2.2 Pulpal pain attributed to irreversible pulpitis due to dental hard-tissue fracture without pulp exposure

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.3.2 *Pulpal pain attributed to irreversible pulpitis due to infection of dentin*
B. Clinical and/or radiographic evidence of any of the following in the affected tooth, without exposure of vital pulp tissue:
   1. fracture involving enamel and dentin (uncomplicated crown fracture)
   2. fracture involving root cementum and dentin (uncomplicated root fracture)
   3. fracture involving enamel, root cementum and dentin (uncomplicated crown-root fracture)
C. Not better accounted for by another ICOP diagnosis.

Comment:
In addition to thermal sensitivity, the pain may be evoked by scratching the surface of the infected dentin.

1.1.1.3.2.3 Pulpal pain attributed to irreversible pulpitis due to a tooth crack without evidence of missing tooth substance

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.3.2 *Pulpal pain attributed to irreversible pulpitis due to infection of dentin*, and criterion C below
B. A crack or incomplete fracture of the affected tooth, involving the enamel or enamel and dentin, has been diagnosed[1]
C. Evidence of causation demonstrated by at least one of the following:
   1. sharp pain upon biting[2]
   2. pain on release of occlusal biting pressure or external application of force[2]
   3. cold hypersensitivity[2]
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Diagnosis may be by visual identification of crack line(s), aided by magnification, light enhancement or visualization with dye, and/or by radiographic or other imaging.
2. The pain in each case often outlasts the application of the stimulus.

Comment:
Cracked teeth may result in sharp pain upon biting, unexplained cold sensitivity, pain on release of pressure, or deep probing depths associated with the crack. Pain typically outlasts the application of the stimulus.

1.1.1.3.3 Pulpal pain attributed to irreversible pulpitis due to infection of the dental pulp

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.3 *Pulpal pain attributed to pulpitis*, and criterion C below

B. The pulp in the affected tooth is infected[1]
C. Pain developed in close temporal relation to the infection, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Infection is evidenced by pulp exposure to the microbiota of the oral cavity for a period.

1.1.1.3.3.1 Pulpal pain attributed to irreversible pulpitis due to carious pulp exposure and infection of the pulp

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.3.3 *Pulpal pain attributed to irreversible pulpitis due to infection of the dental pulp*
B. Deep caries has been diagnosed in the affected tooth, with clinical and/or radiographic evidence of extension to the pulp
C. Not better accounted for by another ICOP diagnosis.

Comment:
Histological studies indicate that when the carious lesion (bacterial front) reaches the pulp, inflammation is likely to be irreversible. The assessment is based on clinical and radiographic appearances. If a zone of intact, functional dentin is not seen between the carious dentin and the pulp, it can be concluded that the microbes are in direct contact with and have infected the pulp tissue, resulting in severe inflammation. It should be noted that, in many cases, this condition may be symptom free.

1.1.1.3.3.2 Pulpal pain attributed to irreversible pulpitis due to dental hard-tissue fracture with pulp exposure

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.3.3 *Pulpal pain attributed to irreversible pulpitis due to infection of the dental pulp*, and criterion C below
B. Clinical and/or radiographic evidence of any of the following in the affected tooth, with exposure of vital pulp tissue:
   1. fracture involving enamel, dentin and pulp (complicated crown fracture)
   2. fracture involving root cementum, dentin and pulp (complicated root fracture)
   3. a fracture involving enamel, root cementum, dentin and pulp (complicated crown-root fracture)
C. Pain developed in close temporal relation to the fracture, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Comment:
In addition to thermal sensitivity, the pain may be evoked by mechanical stimulation of the exposed pulp or adjacent dentin.

1.1.1.3.4 Pulpal pain attributed to pulpitis due to external cervical root resorption

Diagnostic criteria:

A. Pain in a tooth fulfilling criteria for 1.1.1.3 *Pulpal pain attributed to pulpitis*, and criterion C below
B. External cervical root resorption has been diagnosed in the affected tooth by clinical and/or radiographic observations
C. Pain developed in temporal relation to onset of the resorption, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Comments:
Cervical root resorption is a process whereby dentin is resorbed by osteoclastic activity. The condition is asymptomatic until the pulp is involved, usually late in the process. Secondary infection in the resorbed area stimulates an inflammatory response in the adjacent pulp, which may be reversible or irreversible. For technical and/or prognostic reasons, management of the resorption usually involves pulp therapy (pulpectomy) regardless of the degree of pulpal inflammation.

In addition to thermal sensitivity, the pain may be evoked by pressure on the resorptive defect in the dentin.

1.1.1.3.5 Pulpal pain attributed to pulpitis due to other cause

Diagnostic criteria:

A. Pain in one or more teeth fulfilling criteria for 1.1.1.3 *Pulpal pain attributed to pulpitis*, and criterion C below
B. A disorder known to be able to cause pulpal pain,[1] other than those of 1.1.1.3.1 to 1.1.1.3.4, has been diagnosed
C. Causation is plausible based on anatomical, functional and/or temporal association

D. Not better accounted for by another ICOP diagnosis.

Note:

1. As an example, some reports in the literature indicate that pulpitis and pulpal pain may occur secondary to neurovascular events (neurogenic inflammation).

Comment:
Pain symptoms may range from dentinal hypersensitivity to lingering pain, indicative of pulpitis, and are often accompanied by autonomic signs (see 5.1 *Orofacial migraine*).

1.1.1.4 Pulpal pain attributed to systemic cause

Diagnostic criteria:

A. Pain in one or more teeth fulfilling criteria for 1.1.1 *Pulpal pain*, and criterion C below
B. A systemic disorder or disease known to be able to cause pulpal pain[1] has been diagnosed
C. Causation of the pain is clinically plausible
D. Not better accounted for by another ICOP diagnosis.

Note:

1. An example is sickle cell anaemia.

Comments:
Pulpal pain can be the result of a systemic disease causing a change in the pulp condition. For example, sickle cell anaemia crises might result in dental pain. Pulpal necrosis, presumed secondary to vaso-occlusive infarcts, has been reported in patients with sickle cell anaemia. The phenomenon of 'sickle cell toothache' may occur if sickle cells become trapped in the pulpal vascular supply and impede blood flow to the pulpal tissue. This leads to hypoxia, symptoms of pulpitis, cell death and ultimately loss of tooth vitality.

When a systemic disease leads to pulpal pain, it is not uncommon for several teeth to be affected.

1.1.2 Periodontal pain

Description:
Pain caused by a lesion or disorder involving the periodontium: the periodontal ligament and/or the adjacent alveolar (periradicular) bone tissue.

Diagnostic criteria:

A. Any pain in the periodontium[1] fulfilling criterion C
B. Clinical, laboratory, imaging and/or anamnestic evidence of a lesion, disease or trauma[2] known to be able to cause periodontal pain
C. Evidence of causation demonstrated by both of the following:
   1. location of the pain corresponds to the site of the lesion, disease or trauma[1]
   2. pain is exacerbated by physical stimulus[3] applied to the affected tooth (horizontally or vertically) or to the tissue overlying the root
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Pain may also refer and/or radiate to other ipsilateral orofacial locations.
2. The lesion, disease or trauma is specified in each subform.
3. The stimulus may be mechanical, thermal or chemical, as specified in some subforms.

Comment:
Periodontal pain may be associated with all types of periodontal injury or disease. The pain is predominantly inflammatory, and secondary to external or internal events.

1.1.2.1 Periodontal pain attributed to periodontitis (periodontal inflammation)

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2 *Periodontal pain*, and criterion C below
B. Periodontal inflammation[1] has been diagnosed
C. Causation is plausible based on anatomical, functional and/or temporal association[2]
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Inflammation may be due to trauma or infection and is specified in each subform.
2. This criterion cascades down to all subforms.

Comments:
1.1.2.1 *Periodontal pain attributed to periodontitis* is subcategorized according to cause of inflammation.

Periodontitis (marginal as well as apical) is most frequently asymptomatic but can also present with pain and sometimes observable swelling. In such cases, pain is evoked by mechanical stimulation such as biting or chewing and is typically easy for the patient to localize. There may also be spontaneous pain, which is typically ongoing for hours. The intensity may be

mild to severe. The pain can be reproduced by percussion or by applying pressure to the tooth.

In association with this type of pain, gingival pain may also occur.

1.1.2.1.1 Periodontal pain attributed to traumatically induced periodontal inflammation

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1 *Periodontal pain attributed to periodontitis*, and criterion C below
B. Trauma or injury[1] has involved the affected periodontal tissues
C. Pain developed within minutes to days after the trauma or injury
D. Not better accounted for by another ICOP diagnosis.

Note:

1. The injury may be accidental or non-accidental, inflicted by others or self-inflicted, or iatrogenic.

Comments:
Traumatic injury to periodontal tissues causes acute inflammation of the periodontium and can be painful to a varying degree (from mild to severe); it is exacerbated by mechanical provocation of the tooth. Spontaneous pain can occur.

Accidental dental trauma or injury affects 10–30% of the population, and almost exclusively occurs in incisors (maxilla 75–80% and mandible 20–25%). The incidence has been reported as two to three injured teeth/100 school children/year, and the prevalence of traumatized permanent teeth in children and adolescents is reported as 6–34%. Epidemiological data suggest that, while mild trauma is most prevalent, approximately 3% of permanent incisors in a population aged 6–50 years have been afflicted with a traumatic injury severe enough to be painful.

Iatrogenic causes include accidental dental injuries, but also micro-trauma caused for example by changes in occlusion or articulation following dental treatment, and periodontal damage from interventions such as periodontal surgery.

1.1.2.1.1 *Periodontal pain attributed to traumatically induced periodontal inflammation* is therefore subcategorized according to type of trauma or injury.

1.1.2.1.1.1 Periodontal pain attributed to hyperocclusion or hyperarticulation

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.1 *Periodontal pain attributed to traumatically induced periodontal inflammation*, and criteria C and D below
B. A change in occlusal conditions has occurred, with resultant hyperocclusion or hyperarticulation identified by at least one of the following:
   1. primary contact affecting a tooth in occlusion or articulation
   2. hypermobility of a tooth
C. Pain developed within hours to days after the change in occlusal conditions
D. Mechanical provocation[1] reproduces the pain
E. Not better accounted for by another ICOP diagnosis.

Note:

1. Mechanical provocation may be pressure or percussion of the affected tooth.

Comments:
Periodontal pain attributed to occlusal factors involves sensitization of periodontal nociceptors and an inflammatory response due to the excessive loading of the tooth.

The history involves recent dental restoration, tooth extraction or other change in occlusion or articulation. The patient may report that the tooth feels elevated. Clinically, a primary contact in occlusion or articulation is observed. The pain can be reproduced by percussion or by applying pressure to the tooth. The tooth may have increased mobility and, if so, radiographic examination may show widening of the periodontal space.

1.1.2.1.2 Postoperative periodontal pain

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.1 *Periodontal pain attributed to traumatically induced periodontal inflammation*, and criterion C below
B. A surgical intervention has involved the periodontium
C. Pain developed within hours to days after the surgical intervention
D. Not better accounted for by another ICOP diagnosis.

Comments:
Postoperative periodontal pain is iatrogenic, caused by surgically induced tissue damage and subsequent inflammation. The pain is typically mild to moderate and may be accompanied by clinically observable swelling and, occasionally, pus formation.

If normal physiological (primary) healing occurs, the pain duration is typically short (1–2 weeks).

Prolonged pain, due to secondary healing and/or post-operative infection, is occasionally observed but usually does not exceed 3 months.

1.1.2.1.1.3 Periodontal pain attributed to accidental dental trauma

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.1 *Periodontal pain attributed to traumatically induced periodontal inflammation*, and criterion C below
B. Accidental[1] trauma has affected a tooth, with clinical and/or radiographic evidence of one or more of the following:
   1. concussion
   2. subluxation
   3. lateral luxation
   4. intrusion
   5. extrusion
   6. avulsion
   7. root fracture[2]
C. Pain developed within minutes to days after the trauma
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. In the case of root fracture, excessive loading of the tooth is a possible accidental cause.
2. Root fracture may be horizontal or vertical.

Comments:
Accidental dental trauma or injury affects 10–30% of the population, and almost exclusively occurs in incisors (maxilla 75–80% and mandible 20–25%). The incidence has been reported as two–three injured teeth/100 school children/year, and the prevalence of traumatized permanent teeth in children and adolescents is reported as 6–34%. Epidemiological data suggest that, while mild trauma is most prevalent, approximately 3% of permanent incisors in a population aged 6–50 years have been afflicted with a traumatic injury severe enough to be painful.

Concussion, subluxation and extrusion trauma may also include pulpal injury, and periodontal pain may occur together with pulpal pain (see 1.1.1 *Pulpal pain*).

Lateral luxation and intrusion trauma also induce pulpal and alveolar bone injuries, and periodontal pain may occur together with pulpal pain and jaw bone pain (see 1.1.1 *Pulpal pain* and 1.2.3 *Jaw bone pain*).

Avulsion trauma may also include alveolar bone injury, and periodontal pain may occur together with 1.2.3 *Jaw bone pain*.

A root fracture is a hard-tissue injury which may or may not reach the pulp space. If the pulp is involved, it is directly exposed to bacterial assault from the oral cavity and quickly becomes inflamed. If the pulp is vital, the pain may coincide with 1.1.1.3.3.2 *Pulpal pain attributed to irreversible pulpitis due to dental hard-tissue fracture with pulp exposure*. In addition to accidental trauma, other common reasons for root fracture include excessive loading of a root canal-treated tooth, typically with a post-and-core.

Dental trauma frequently causes periodontal pain. The clinical and radiographic presentation, and the characteristics and severity of pain, depend on the nature and severity of the traumatic injury. Below follows a brief description of the trauma diagnoses used in dental practice (from the *Dental Trauma Guide*; https://dentaltraumaguide.org (accessed January 2020)).

Periodontal pain due to concussion is caused by accidental injury to the periodontium and subsequent inflammation. The tooth displays normal mobility and is not displaced from its alveolar socket. Unless previously root canal treated, the tooth typically shows evidence of a vital pulp. Imaging shows normal periradicular conditions.

Periodontal pain due to subluxation is caused by accidental injury to the periodontium and subsequent inflammation. The tooth displays increased mobility but is not displaced from its alveolar socket. Clinical findings include bleeding from the gingival sulcus. The tooth responds to pulp vitality testing in about 50% of cases. Radiographic examination may show a widening of the periodontal space.

Periodontal pain due to lateral luxation is caused by accidental injury to the periodontium and subsequent inflammation. The tooth is laterally displaced from its alveolar socket in combination with comminution or fracture of the buccal or lingual/palatal alveolar bone. The periodontal ligament is partially or totally separated, and bleeding is seen from the sulcus. The tooth usually displays decreased mobility and may interfere with the occlusion and/or articulation. The tooth usually does not respond to pulp vitality testing. Radiographic examination shows variation in periodontal space width depending on the projection.

Periodontal pain due to intrusion is caused by accidental injury to the periodontium and subsequent inflammation. The tooth is axially displaced into the alveolar bone, and thus appears shorter than the adjacent teeth. The injury is accompanied by comminution or fracture of the alveolus. Other clinical findings may include decreased mobility and high percussion sound. The tooth usually does not respond to pulp vitality testing. Radiographic examination shows absence of (or decreased width of) the periodontal ligament space in all or part of the tooth.

Periodontal pain due to extrusion is caused by accidental injury to the periodontium and subsequent inflammation. The tooth is axially displaced and partially out of its socket, and thus appears elongated. The periodontal ligament is partially or totally separated and there is bleeding from the sulcus, but the alveolar socket bone is intact. The tooth has increased mobility and may interfere with occlusion/articulation. The tooth usually does not respond to pulp vitality testing. Radiographic examination shows increased width of the periodontal ligament space.

Periodontal pain due to avulsion is caused by accidental injury to the periodontium and subsequent inflammation. The tooth is completely displaced out of its socket, which is found empty or filled with a coagulum. The surrounding alveolar bone may be fractured.

Periodontal pain due to root fracture is caused by dislocation or fragments and/or subsequent infection causing periodontal inflammation. The history may or may not reveal an accidental traumatic event. The coronal fragment may be displaced, and the tooth may appear longer than the adjacent teeth, may display increased mobility and may interfere with occlusion/articulation. A local deep periodontal pocket may be present. Imaging shows a vertical or horizontal fracture confined to the root. If not previously root-filled, the tooth may or may not respond to pulp vitality testing.

1.1.2.1.1.4 Periodontal pain attributed to other trauma or injury

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.1 *Periodontal pain attributed to traumatically induced periodontal inflammation*, and criterion C below
B. Non-accidental or non-violent trauma[1] has involved the affected tooth or teeth
C. Pain developed in close temporal relation to the trauma, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Note:

1. By anamnestic, clinical or radiographic or other imaging findings, a trauma known to be able to cause periodontal inflammation can be identified, such as insufficient cooling during dental restorative procedures, interdental foreign body impaction (including food impaction), defective dental restoration, or apically extruded endodontic material. Clinical findings may include signs of acute inflammation (swelling, redness, presence of pus), increased tooth mobility and/or local deep periodontal pocket. Unless root canal treated, the tooth typically shows evidence of a vital pulp. Imaging may display local marginal bone loss, which may or may not include the periapical region.

1.1.2.1.2 Periodontal pain attributed to apical periodontitis due to endodontic disease

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1 *Periodontal pain attributed to periodontitis*
B. Apical periodontitis due to endodontic disease has been diagnosed
C. Not better accounted for by another ICOP diagnosis.

Comments:
Endodontic disease (i.e. pulpal and periapical disease) is frequently associated with pain that may be mild to severe. Periodontal pain due to endodontic disease is associated with pulpal, periapical, juxtaradicular and/or periradicular inflammation. A broken barrier against the oral cavity, most often caused by caries, and subsequent bacterial invasion of the pulp and root canal system, are the main causes of inflammation of the pulp and periapical tissues.

This type of pain may also affect the gingivae.

Endodontic disease, including periapical, juxtaradicular or periradicular inflammation, may also be present without any clinical symptoms.

1.1.2.1.2.1 Periodontal pain attributed to pulpal inflammation

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.2 *Periodontal pain attributed to apical periodontitis due to endodontic disease*
B. Pulpitis (reversible or irreversible) has been diagnosed by both of the following:
   1. evidence of dental disorder involving the affected tooth, known to be able to cause pulpitis
   2. vital pulp evidenced by response to pulp vitality testing
C. Not better accounted for by another ICOP diagnosis.

Comments:
Periodontal pain secondary to pulpal inflammation is associated with symptomatic pulpitis. The periodontal inflammation is centred on the periapical region.

The pulp is vital and thus the tooth typically responds to pulp vitality testing. The tooth is often tender to percussion. Clinical findings may include deep caries, deep/defective restoration, or external cervical root resorption. Imaging may or may not show evidence of diffuse local periapical bone resorption or sclerosis.

According to the literature, the association is weak between the actual state of the pulp and the periodontium (histology) and diagnostic findings, including present and historical symptoms such as characteristics of tooth pain, clinical observations and test results. Current diagnostics are largely based on expert opinion and a few studies with quality deficits.

1.1.2.1.2.1 *Periodontal pain attributed to pulpal inflammation* frequently also fulfils the criteria for 1.1.1.3 *Pulpal pain attributed to pulpitis*. Both diagnoses should then be made.

1.1.2.1.2.2 Periodontal pain attributed to endodontic infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.2 *Periodontal pain attributed to apical periodontitis due to endodontic disease*
B. Partial or total pulp necrosis and endodontic infection have been diagnosed in the tooth by both the following:
   1. non-vital pulp, evidenced by either:
      a) direct inspection, or non-response to pulp vitality testing
      b) a previously debrided root canal
   2. clinical[1] and/or radiographic[2] evidence of apical inflammation[3]
C. Not better accounted for by another ICOP diagnosis.

Notes:

1. Clinical evidence includes tenderness to percussion and/or pressure, and/or tenderness to apical palpation.
2. Radiographic evidence includes apical or juxtaradicular radiolucency or sclerosis.
3. Apical inflammation includes symptomatic apical periodontitis or acute apical abscess.

Comments:
Periodontal pain due to endodontic infection is associated with non-vital pulp (or a previously root-filled tooth) and infection of the pulp space. The pulp is totally or partially necrotic (unless the tooth is previously root canal treated), and the tooth typically does not respond to pulp vitality testing. Although localized, the pain frequently refers and/or radiates to other orofacial sites on the same side, especially if the pain is severe. The pain can be reproduced by percussion or by applying pressure on the tooth and/or the adjacent periapical vestibular region. Imaging typically shows evidence of local periapical bone resorption.

The inflammatory response in the periapical tissues is caused by root canal infection with a mixed flora. An increased incidence of pain and swelling in apical periodontitis is associated with presence of specific anaerobes: *Porphyromonas*, *Peptostreptococcus* and *Prevotella* species. Upon local infection spread, a periapical abscess may form.

1.1.2.1.2.2.1 Periodontal pain attributed to intraradicular endodontic infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.2.2 *Periodontal pain attributed to endodontic infection*
B. A tooth has root canal infection[1]
C. Not better accounted for by another ICOP diagnosis.

Note:

1. The infection may be bacterial, viral, fungal, or other.

Comments:
In most teeth with infected necrotic pulp, the infection is confined to the root canal system.

Successful infection treatment usually results in pain resolution.

1.1.2.1.2.2.2 Periodontal pain attributed to extraradicular endodontic infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.2.2 *Periodontal pain attributed to endodontic infection*
B. Extraradicular infection[1] around one or more teeth has been diagnosed
C. Not better accounted for by another ICOP diagnosis.

Note:

1. The infection may be bacterial, viral, fungal, or other.

Comments:
In 1.1.2.1.2.2.2 *Periodontal pain attributed to extraradicular endodontic infection*, the infectious agent causing

the periodontal inflammation resides on the external root surface, apically or in association with accessory canal orifices, or in the periapical tissues.

Extraradicular endodontic infection may occur with or without intraradicular infection. In either case, the microbes colonize the external apical foramen and root surface, forming a biofilm. Anaerobic species such as *Actinomyces* and *Propionibacterium* also have the ability to form colonies in the periapical tissues at some distance from the root, and this has been associated with remaining symptoms, including pain, after root canal treatment.

The pain typically does not resolve after successful disinfection of the root canal system. Imaging occasionally reveals signs of external apical root resorption.

1.1.2.1.3 Periodontal pain attributed to periodontal disease

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1 *Periodontal pain attributed to periodontitis*, and criterion C below
B. Periodontal disease[1] has been diagnosed
C. Causation is plausible based on anatomical, functional and/or temporal assocation[2]
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. The disease is specified in each subform.
2. This criterion cascades down to all subforms.

Comments:
Periodontal pain due to plaque-induced periodontal disease can be acute or chronic in nature and, depending on type, the pain intensity ranges from mild to severe.

The disease can be localized or generalized in the dentition. A number of intrinsic (diabetes, pregnancy, puberty, menopause) and extrinsic (smoking, medications, nutritional deficiencies (e.g. avitaminosis-C)) factors are considered as disease modifiers. In addition, medications known to be associated with gingival hyperplasia (e.g. phenytoin, ciclosporin, calcium channel blockers, bisphosphonates and oral contraceptives) may promote periodontal breakdown due to difficulties in maintaining adequate oral hygiene.

1.1.2.1.3.1 Periodontal pain attributed to chronic periodontitis

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.3 *Periodontal pain attributed to periodontal disease*
B. Chronic periodontitis has been diagnosed
C. Not better accounted for by another ICOP diagnosis.

Comments:
1.1.2.1.3.1 *Periodontal pain attributed to chronic periodontitis* may present in association with increased tooth mobility and poor oral hygiene routines and is typically mild. The pain typically appears only on provocation and does not linger. Most cases of chronic periodontitis are not painful but may become painful on inflammatory exacerbation (see 1.1.2.1.3.5 *Periodontal pain attributed to periodontal abscess*).

Chronic periodontitis is characterized by slowly progressing attachment loss, sometimes with periods of more rapid progression. The absence or low level of pain has been attributed to the mainly chronic inflammatory cell infiltrates surrounding the infectious source, and functional drainage.

1.1.2.1.3.2 Periodontal pain attributed to aggressive periodontitis

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.3 *Periodontal pain attributed to periodontal disease*
B. Aggressive periodontitis has been diagnosed
C. Not better accounted for by another ICOP diagnosis.

Comments:
1.1.2.1.3.2 *Periodontal pain attributed to aggressive periodontitis* may present in association with increased tooth mobility and poor oral hygiene routines and is typically mild to moderate. The pain usually appears only on provocation and does not linger.

Aggressive periodontitis is characterized by rapidly progressing attachment loss and, sometimes, onset at a young age.

1.1.2.1.3.3 Periodontal pain attributed to periodontitis as a manifestation of systemic disorder

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.3 *Periodontal pain attributed to periodontal disease*, and criterion D below
B. A systemic disorder[1] known to be able to cause periodontitis has been diagnosed
C. Periodontitis has occurred as a manifestation of the systemic disorder
D. Causation of the pain is clinically plausible
E. Not better accounted for by another ICOP diagnosis.

Note:

1. The systemic disorder is specified in each subform.

Comments:
In addition to the more common plaque-induced periodontal disease, a number of systemic disorders manifest as periodontitis. The disorders listed below are considered causative factors for periodontitis. They may also alter the course of plaque-induced periodontitis from chronic to aggressive.

1.1.2.1.3.3 *Periodontal pain attributed to periodontitis as a manifestation of systemic disorder* may present in association with increased tooth mobility and poor oral hygiene routines. The pain is typically mild to moderate, appears only on provocation and does not linger. However, reports on the degree to which periodontitis as a manifestation of a systemic disorder is associated with pain are essentially lacking in the literature.

1.1.2.1.3.3.1 Periodontal pain attributed to haematological disorder

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.3.3 *Periodontal pain attributed to periodontitis as a manifestation of systemic disorder*
B. The systemic disorder is one of the following:
   1. acquired neutropenia
   2. leukaemia
   3. other haematological disorder known to be able to cause periodontitis.

1.1.2.1.3.3.2 Periodontal pain attributed to genetic disorder

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.3.3 *Periodontal pain attributed to periodontitis as a manifestation of systemic disorder*
B. The systemic disorder is one of the following:
   1. familial and cyclic neutropenia
   2. Down syndrome
   3. leukocyte adhesion deficiency syndromes
   4. Papillon–Lefèvre syndrome
   5. Chediak–Higashi syndrome
   6. histiocytosis syndromes
   7. glycogen storage disease
   8. infantile genetic agranulocytosis
   9. Cohen syndrome
   10. Ehler–Danlos syndrome (types IV and VIII)
   11. hypophosphatasia
   12. other genetic disorder known to be able to cause periodontitis.

1.1.2.1.3.3.3 Periodontal pain attributed to unspecified systemic disorder

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.3.3 *Periodontal pain attributed to periodontitis as a manifestation of systemic disorder*
B. The systemic disorder is known to be able to cause periodontitis but is neither haematological nor genetic.[1]

Note:

1. Systemic disorders associated with periodontitis are not currently well described in the literature.

1.1.2.1.3.4 Periodontal pain attributed to necrotizing ulcerative periodontitis (NUP)

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.3 *Periodontal pain attributed to periodontal disease*, and criterion C below
B. Necrotizing ulcerative periodontitis has been diagnosed
C. The pain has developed within hours to days of onset of the ulcerations
D. Not better accounted for by another ICOP diagnosis.

Comments:
Necrotizing ulcerative periodontitis (NUP) is a rare oral infection, a more severe form of necrotizing (ulcerative) gingivitis which, besides causing soft tissue destruction, also includes loss of attachment and alveolar bone. The two conditions are often conflated into necrotizing periodontal diseases (NPD), and are associated with diminished systemic resistance and immune dysfunction. The predisposing factors include severe stress, sleep deprivation, alcohol, smoking and HIV infection.

1.1.2.1.3.4 *Periodontal pain attributed to necrotizing ulcerative periodontitis (NUP)* is typically severe. Pain is provoked by physical stimuli applied to the affected tooth or surrounding tissue. Pain also occurs spontaneously.

Clinically, necrotic soft tissue lesions and loss of attachment can be observed.

1.1.2.1.3.5 Periodontal pain attributed to periodontal abscess

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1.3 *Periodontal pain attributed to periodontal disease*, and criterion C below
B. A periodontal abscess has been diagnosed by either or both of the following:
   1. clinical signs of acute inflammation[1] and loss of attachment[2]
   2. radiographic evidence of marginal and periradicular bone resorption
C. The pain has developed in close temporal relation[3] to the abscess
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Clinical signs include swelling, redness, tenderness and/or presence of pus.
2. Loss of attachment is evidenced by increased mobility and/or local deep periodontal pocket.
3. Usually hours to days before appearance of the abscess.

Comments:
A periodontal abscess is an exacerbation of chronic periodontitis or aggressive periodontitis, and pain due to this is usually severe. In addition to swelling, other clinical findings include plaque and/or calculus deposit on the root surface, usually with increased tooth mobility and a local deep periodontal pocket. Unless previously root canal treated, the tooth typically shows evidence of a vital pulp. Imaging shows evidence of marginal and periradicular bone resorption, which may or may not include the periapical region.

Although localized, the pain frequently refers and/or radiates to other orofacial sites on the same side, especially if the pain is severe. The pain can be reproduced by percussion or by applying pressure on the tooth and/or the adjacent periapical vestibular region.

1.1.2.1.4 Periodontal pain attributed to apical and marginal periodontitis due to combined endodontic infection and periodontal disease

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1 *Periodontal pain attributed to periodontitis*, and criterion C below
B. Both of the following have been diagnosed[1]:
   1. partial or total pulp necrosis, or a tooth is previously root canal treated
   2. periodontal disease
C. Causation is plausible based on anatomical, functional and/or temporal association
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Diagnosis is based on clinical and radiographic observations.

Comments:
A combined endodontic and periodontal lesion may be symptom free. If pain is present, it is typically moderate to severe, and other clinical findings may include signs of acute inflammation (swelling, redness, presence of pus), plaque and/or calculus deposit on the root surface, increased tooth mobility and deep periodontal pocket(s). If not previously root canal treated, the tooth shows no or inconclusive evidence of pulp vitality. Imaging shows evidence of marginal and periradicular bone resorption that includes the periapical region.

Although localized, the pain frequently refers and/or radiates to other orofacial sites on the same side, especially if the pain is severe. The pain can be reproduced by percussion or by applying pressure on the tooth and/or the adjacent periapical vestibular region.

1.1.2.1.5 Periodontal pain attributed to infective peri-implantitis

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.2.1 *Periodontal pain attributed to periodontitis* with the exception that it involves an implant and not a natural tooth, and criterion C below
B. Clinical[1] and/or radiographic[2] evidence of a peri-implant infection
C. Causation is plausible based on anatomical, functional and/or temporal association
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Clinical evidence includes signs of acute inflammation (swelling, redness, presence of pus) and/or attachment loss (increased mobility, deep pocket).
2. Radiographic evidence includes radiolucency partially or totally surrounding the implant.

Comments:
Inflammation surrounding a dental implant is most frequently painless but, when pain occurs, it is typically moderate to severe.

Other clinical findings may include plaque and/or calculus deposit on the implant surface. Imaging shows poor bony integration of the implant and evidence of horizontal marginal bone loss or localized peri-implant bone resorption.

Patients with *1.1.2.1.5 Periodontal pain attributed to infective peri-implantitis* are also likely to be affected by 1.1.3 *Gingival pain*.

1.1.2.2 Periodontal pain attributed to local non-inflammatory cause

Diagnostic criteria:

A. Pain fulfilling criteria for *1.1.2 Periodontal pain*, and criterion C below
B. A local non-inflammatory disorder known to be able to cause periodontal pain[1] has been diagnosed[2]
C. Causation of the pain is clinically plausible based on anatomical and/or temporal association
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Examples of such disorders are periodontal cyst or tumour.
2. Diagnosis is by clinical, imaging and/or histological examination.

Comments:
1.1.2.2 *Periodontal pain attributed to a local non-inflammatory cause* is usually mild to moderate.

Periodontal cysts, radicular cysts and tumours are frequently asymptomatic but, following expansion, symptoms such as pain, localized swelling and displacement of one or more teeth may occur. In such cases, pain is occasionally evoked by external mechanical stimulation, such as biting or chewing, and is typically easy for the patient to localize. There may also be spontaneous pain, which is seldom severe.

1.1.3 Gingival pain

Description:
Pain caused by a lesion or disorder involving the gingival tissues.

Coded elsewhere:
Gingival pain occurring in association with conditions that mainly affect other oral tissues are classified in other sections: for gingival pain attributed to alveolar osteitis (dry socket), see 1.2.3.5 *Jaw bone pain attributed to therapy*; for gingival pain attributed to periodontitis, see 1.1.2.1 *Periodontal pain attributed to periodontitis (periodontal inflammation)*; for gingival pain attributed to apical periodontitis, see 1.1.2.1.2 *Periodontal pain attributed to apical periodontitis due to endodontic disease*; for palatal gingival pain attributed to acute necrotizing sialadenitis, see 1.2.2.2.1 *Salivary gland pain attributed to bacterial infection*.

For gingival pain attributed to neuropathy, see 4.1 *Pain attributed to lesion or disease of the trigeminal nerve*. Trigger zones of 4.1.1 *Trigeminal neuralgia* may be located in the gingivae, and light touch will elicit the typical intense paroxysmal pain attacks affecting the whole dermatome corresponding to the affected nerve branch. As a result, patients may find it impossible to wear a denture in the region. Gingival pain may also occur as part of the early clinical presentation of 4.1.1 *Trigeminal neuralgia*, the diffuse deep 'pre-trigeminal neuralgia pain' that sometimes precedes the onset of characteristic paroxysmal pain.

For peripheral neuropathy associated with gingival pain, see 4.1.2 *Other trigeminal neuropathic pain*.

For idiopathic gingival pain, see 6. *Idiopathic orofacial pain*. Burning mouth syndrome (BMS) may also affect the gingivae, presenting as localized or more widely distributed gingival pain (see 6.1 *Burning mouth syndrome*). Persistent idiopathic dentoalveolar pain (PIDAP) is frequently associated with localized pain in the gingivae (see 6.3 *Persistent idiopathic dentoalveolar pain*).

Consideration must be given to patients presenting with gingival pain in association with chronic widespread pain or other multiple pain conditions, which may be attributable to central sensitization or other mechanisms.

Diagnostic criteria:

A. Any pain in the gingivae[1] fulfilling criterion C
B. Clinical, laboratory, imaging and/or anamnestic evidence of a lesion or disorder of the gingival tissues, known to be able to cause pain
C. Evidence of causation demonstrated by at least two of the following:
    1. location of the pain corresponds to the site(s) of the lesion or disorder[1]
    2. pain developed in temporal relation to the appearance or onset of the lesion or disorder
    3. pain is exacerbated by manipulation of the affected gingival tissue
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Pain may also refer and/or radiate to other ipsilateral orofacial locations.

1.1.3.1 Gingival pain attributed to gingivitis (gingival inflammation)

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.3 *Gingival pain*
B. Gingivitis[1] has been diagnosed[2]
C. Not better accounted for by another ICOP diagnosis.

Notes:

1. Gingivitis due to trauma, infection or systemic disorder is specified in each subform.
2. Diagnosis is made by clinical observation of inflammatory signs (swelling, redness and bleeding) in the gingivae.

Comment:
Gingivitis may be caused by infection due to specific or non-specific microbial organisms, trauma (physical, thermal, radiation or chemical), autoimmunity or allergic reaction.

1.1.3.1.1 *Gingival pain attributed to trauma*

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.3.1 *Gingival pain attributed to gingivitis*, and criterion C below
B. Trauma[1] or injury[2] involving the gingival tissues has occurred
C. Both of the following:
    1. pain is localized to the traumatized or injured tissues
    2. pain developed within minutes to days after the trauma or injury
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. The trauma may be mechanical, thermal or chemical.
2. The injury may be accidental or non-accidental, inflicted by others or self-inflicted, or iatrogenic.

Comments:
Traumatic injury of gingival tissues causes acute inflammation and can be painful to a varying degree. Traumatic ulceration of the gingiva may be acute or chronic in nature with the latter diagnostically more challenging due to underlying fibrosis and clinical appearance of neoplastic induration.

1.1.3.1.1 *Gingival pain attributed to trauma* may be mild to severe and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur.

Causes of 1.1.3.1.1 *Gingival pain attributed to trauma* include accidental dental injuries, but also microtrauma caused, for example, during eating or drinking overly hot foods or drinks, following dental treatment, or by trauma due to tooth brushing or flossing or other interdental instruments. A thorough clinical history will often alert the clinician to a traumatic aetiology or burns caused by warm food or chemicals. Examination may reveal the causative factor, such as a sharp broken tooth or restoration or an ill-fitting denture. Ulceration due to local anaesthetic injection most often occurs in the hard palate, the combined result of pressure and ischaemic necrosis. Poorly fitting dentures may cause painful ulcerations. Over-erupted dentition or parafunctional habits may also cause local occlusal gingival trauma with resultant inflammation and pain. Iatrogenic gingival damage occurs during most dental surgery; for example, dental extraction, gingival or periodontal surgery, or dental restorative therapy. Chemical burns may be related to misuse of anti-inflammatory tablets or occur due to dental treatment. Self-harm may be a rare cause of gingival trauma.

Dental trauma may also cause gingival inflammatory pain (see 1.1.2.1.1 *Periodontal pain attributed to traumatically induced periodontal inflammation*).

1.1.3.1.2 Gingival pain attributed to infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.3.1 *Gingival pain attributed to gingivitis*, and criterion C below
B. Infection[1] of the gingival tissues has been diagnosed[2]
C. Pain developed in close temporal relation to the infection, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. The infection may be bacterial, viral or fungal, as specified in each subform.
2. Diagnosis is based on anamnestic information, clinical observations and/or microbiological analysis.

Comments:
Infection of the gingival tissues causes acute inflammation and can be painful to a varying degree: the pain

may be mild to severe and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur.

Acquired or congenital immunosuppression may lead to increased risk of gingival infection. Patients on immunosuppressive therapy may develop a variety of opportunistic infections including pseudomembranous candidiasis and other fungal and viral infections. Tumour necrosis factor (TNF)-α therapy increases the risk of tuberculosis (TB). Patients on infliximab and adalimumab with combined immunomodulatory therapy may be at increased risk of TB, histoplasmosis and/or coccidiomycosis infections. Antirheumatic drugs including methotrexate, abatacept and alefacept have increased the risks of herpes simplex and herpes zoster infections and TB.

1.1.3.1.2 *Gingival pain attributed to infection* is subcategorized according to the causative microorganism.

1.1.3.1.2.1 Gingival pain attributed to bacterial infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.3.1.2 *Gingival pain attributed to infection*
B. The infection is bacterial.

Comments:
Bacterial infections are the most common oral infections and gingival pain may be associated with underlying dental pathology, such as periodontal infection or endodontic infections that may present as swelling, inflammation and pain of the overlying gingivae.

Acute necrotizing ulcerative gingivitis (ANUG) (or necrotizing ulcerative gingivitis (NUG), necrotizing ulcerative periodontitis (NUP) or necrotizing ulcerative stomatitis (NUS)) is an opportunistic gingival infection caused by an array of bacteria in malnourished children, young adults and immune deficient patients. NUG is often the initial presentation, proceeding into NUP, NUS and ultimately noma (a form of gangrene affecting the face). Necrosis and ulceration of the interdental gingival papilla, excruciating pain, severe halitosis, regional lymphadenopathy, malaise and fever differentiate this form of ulceration from others.

Pericoronitis (inflammation around a tooth crown) causing pain is most often associated with partially erupted third molars. Other dentition, both permanent and deciduous, may have mild pericoronitis during eruption. If the tooth is impacted and unable to fully erupt, continued or recurrent infection may ensue. Pain results from the individual's immune inflammatory response to anaerobic bacteria colonized in biofilm that cannot be shed from third molars partially covered by soft tissue.

1.1.3.1.2.2 Gingival pain attributed to viral infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.3.1.2 *Gingival pain attributed to infection*
B. The infection is viral.

Comments:
The infected gingival tissues may often be ulcerated and painful to touch. Severe local pain is often associated with eating or drinking acidic or hot or cold foods or drinks, which may cause the individual to be unable to eat or drink and become dehydrated.

Herpes simplex virus (HSV) is the most common virus to affect the oral mucosa. Herpetic gingivostomatitis, the primary HSV-1 infection, mostly affects children and presents either as asymptomatic infection or with mucosal vesicles followed by painful ulceration affecting both keratinized and non-keratinized mucosa and gingivae. Adults with primary infection suffer symptomatic herpetic pharyngotonsillitis initiated as vesicles that rapidly break down into painful shallow ulcerations.

Other viral infections of the gingival tissues include varicella-zoster virus (VZV), human papilloma virus (HPV), cytomegalovirus (CMV), coxsackieviruses and HIV infections.

1.1.3.1.2.3 Gingival pain attributed to fungal infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.3.1.2 *Gingival pain attributed to infection*
B. The infection is fungal.

Comments:
Gingival pain associated with fungal infection is probably rare, and reports in the literature are essentially lacking. The painful manifestations of oral fungal infection usually affect oral mucosa.

1.1.3.1.3 Gingival pain attributed to autoimmunity

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.3.1 *Gingival pain attributed to gingivitis*, and criterion C below
B. An autoimmune disease or disorder known to be able to cause gingival pain[1] has been diagnosed
C. Causation of the pain is clinically plausible
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Examples are mucous membrane pemphigoid, Sjögren's syndrome and pemphigus.

Comments:

1.1.3.1.3 *Gingival pain attributed to autoimmunity* may be mild to severe and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur.

Several dermatological immune-mediated vesiculo-ulcerative lesion conditions may present with oral mucosal involvement, either concurrently with the skin pathology, as the initial presentation, or sometimes as the only clinical presentation.

Mucous membrane pemphigoid (MMP) is a common systemic autoimmune blistering disease with preferential involvement of mucosal membranes. The antibodies are directed at the proteins of keratinocyte to connective tissue matrix adhesion or hemi-desmosomes (BP180 and laminin-332) causing the epithelium to split away from its underlying connective tissue bed. The subepithelial nature of the split results in thick-roofed vesicles, which may still be intact on examination. Rupture of the vesicles leaves ulcerative lesions devoid of any epithelium, covered by yellow-white slough. Desquamative gingivitis (erythematous and friable gingiva with epithelial destruction) is a frequent finding.

Sjögren's syndrome is a systemic autoimmune disease that frequently presents concomitantly with other systemic connective tissue or organ-specific autoimmune diseases. The association is well described for systemic lupus erythematosus and rheumatoid arthritis. The gingival tissues can become abraded and even cut with dry foods, and sore. The presence of Sjögren's syndrome influences the expression of the other autoimmune disease to some degree, for instance by increasing fatigue and lymphoma risk.

Pemphigus, a group of immune-mediated subepithelial bullous dermatoses, is mediated by autoantibodies directed at the proteins of keratinocyte adhesion (desmosomes) causing acantholysis. Pemphigus vulgaris most commonly affects the oral cavity, with autoantibodies mainly directed against desmoglein-1 and 3 (mucocutaneous forms) or only 3 (mucosal forms). Gingival pain due to pemphigus is infrequent, since the disease mostly affects oral mucosa.

1.1.3.1.4 Gingival pain attributed to hypersensitivity or allergic reaction

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.3.1 *Gingival pain attributed to gingivitis*, and criterion C below
B. A hypersensitivity or allergic reaction in the gingival tissues[1] has occurred
C. Pain developed in temporal relation to the hypersensitivity or allergic reaction, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Note:

1. The hypersensitivity or allergic reaction may be in association with dental material (such as temporary or permanent restorative or impression material), an oral hygiene product, a topical drug, a systemic drug, a food or food additive, or another factor.

Comments:

1.1.3.1.4 *Gingival pain attributed to hypersensitivity or allergic reaction* may be mild to severe and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur.

Allergic reactions and oral mucosal hypersensitivity reactions are less common than cutaneous ones, probably because of allergen dilution and the continuous rinsing effects of normal saliva flow. Lesions may present with non-specific tissue oedema, erythema, cracking, ulceration, hyperkeratotic white plaques or mucosal desquamation.

A temporal or spatial association with an offending agent can usually be identified. However, in the case of drug-related hypersensitivity, lesions may start long after the introduction of the drug and may remain for months after cessation thereof, complicating diagnosis and management.

A hypersensitivity reaction to either a systemic drug or an offending agent in direct contact may result in clinical and histological features reminiscent of lichen planus. The terms *oral lichenoid drug reaction* (OLDR) and *oral lichenoid contact lesion* (OLCL) are used respectively, and both may present with significant ulceration, usually with erythema and white striations at the periphery of the ulceration. Amalgam is often implicated in OLCL, confirmed by patch testing for mercury or amalgam sensitivity. OLDR is encountered with some frequency in patients treated with angiotensin-converting enzyme (ACE) inhibitors, NSAIDs and oral hypoglycaemic drugs.

Potential drug reactions causing oral mucogingival reactions have been well summarized. Fixed drug eruption (FDE) is a form of hypersensitivity remarkable for its fixed anatomical nature and has been described with NSAIDs and other oxicam drugs, gabapentin, fluconazole and systemic antibacterial and antifungal drugs. FDE should be suspected in cases with a temporal association with drug ingestion, may be confirmed through patch testing or oral provocation tests, and managed

through drug avoidance or substitution, while the acute lesions can be treated with topical or systemic steroids.

Allergic contact stomatitis, although rare, is a form of mucositis reported in association with dental impression materials, dental restorative materials, topical benzocaine application and, more commonly, cinnamon in toothpastes, mouth rinses and chewing gum. Lesions may appear as mixed red and white patches with ulceration, swelling of the cheeks and desquamation appearing on the lips, cheeks, tongue and gingivae as localized or widely distributed lesions.

Drug-induced fibrosis with epithelial hyperplasia or fibrovascular hyperplasia may occasionally be associated with painful presentation, probably due to underlying periodontal infection caused by difficulty with oral hygiene in these conditions.

1.1.3.1.5 Gingival pain attributed to gingival inflammation due to other cause

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.3.1 *Gingival pain attributed to gingivitis*, and criterion C below
B. A disorder, known to be able to cause gingivitis but other than those in 1.1.3.1.1 to 1.1.3.1.4,[1] has been diagnosed
C. Pain developed in close temporal relation to the disorder, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Such disorders include endocrine disorders or alterations, dietary deficiency, haematological diseases, gastrointestinal diseases and dermatological diseases, drug-induced disorders (not attributable to hypersensitivity or allergy) and genetic disorders.

Comments:

1.1.3.1.5 *Gingival pain attributed to gingival inflammation due to other cause* may be mild to severe, and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur.

Inflammation of the gingival tissues may occur associated with a systemic disease, disorder or condition, or with the treatment of such diseases or disorders.

Alteration of the physiological state, such as pregnancy and menopause, may cause endocrine changes that manifest as gingival discomfort and pain. Systemic disorders that can cause gingivitis include endocrine disease (hypothyroidism, diabetes mellitus); dietary deficiencies (iron, vitamin B complex, zinc); anaemia; gastrointestinal disorders (gastro-oesophageal reflux disease); and drug-induced and genetic disorders.

Epulis is a hyperplastic, non-neoplastic lesion which originates mainly from gingival tissues. Several histologic types occur, of which the prevalent type during pregnancy is the granulomatous type, a form of pyogenic granuloma. The growth is composed mainly of capillary vessels and endothelial proliferation and appears usually on the frontal part of the maxilla during the third trimester (sometimes referred to as 'pregnancy tumour'). The lesion usually causes no symptoms apart from its very presence, but may become painful because of interference with occlusion or denture wear. Aetiologic factors are improper maintenance of oral hygiene, which leads to chronic gingivitis, and high gingival levels of active progesterone, which acts by a yet undefined mechanism.

Antineoplastic therapy-induced mucositis associated with chemotherapy and radiation mainly affects oral mucosa (see 1.2.1.1.1.3 *Oral mucosal pain attributed to radiation or chemotherapy*) but can also affect the gingivae and cause gingival pain.

Benign hyperplastic lesions or tumours involving gingivae are usually not directly associated with pain but may become painful if traumatized and/or infected due to interference with occlusion or dentures (see 1.1.3.1.1 *Gingival pain attributed to trauma* and 1.1.3.1.2 *Gingival pain attributed to infection*).

1.1.3.2 Gingival pain attributed to malignant lesion

Diagnostic criteria:

A. Pain fulfilling criteria for 1.1.3 *Gingival pain*, and criterion C below
B. A malignant lesion of the gingival tissues has been diagnosed
C. Causation of the pain is plausible based on anatomical and/or temporal association
D. Not better accounted for by another ICOP diagnosis.

Comments:

The gingivae may be affected by an array of both primary and metastatic malignancies, which may all present as non-specific ulcers. Oral squamous cell carcinoma (OSCC) is the most common, frequently presenting as ulceration with clinical induration, fixation to the underlying tissues, rolled exophytic margins, and pain and/or numbness.

1.1.3.2 *Gingival pain attributed to malignant lesion* may be mild to severe and is exacerbated by mechanical provocation of the gingivae. Spontaneous pain can occur.

1.2 Oral mucosal, salivary gland and jaw bone pains

Description:
Pain caused by a lesion or disorder affecting the non-dental oral and peri-oral tissues: the oral mucosa, the salivary glands and the jaw bone tissues.

Coded elsewhere:
For pain arising from orofacial muscles, see 2. *Myofascial orofacial pain*. For pain arising from the temporomandibular joint, see 3. *Temporomandibular joint (TMJ) pain*.

1.2.1 Oral mucosal pain

Description:
Pain caused by a disease or disorder involving the oral mucosa.

Coded elsewhere:
For oral mucosal pain attributed to neuropathy, see 4.1 *Pain attributed to lesion or disease of the trigeminal nerve*. (Oral mucosal pain may occur as part of the early clinical presentation of 4.1.1 *Trigeminal neuralgia*: the diffuse deep 'pre-trigeminal neuralgia pain' that sometimes precedes the onset of characteristic paroxysmal pain. Trigger zones of 4.1.1 *Trigeminal neuralgia* may be located in the oral mucosa, and light touch will elicit the typical intense paroxysmal pain attacks affecting the whole dermatome corresponding to the affected nerve branch.)

For peripheral neuropathy associated with pain in the oral mucosa, see 4.1.2 *Other trigeminal neuropathic pain*.

For idiopathic oral mucosal pain, see 6. *Idiopathic orofacial pain*. For burning mouth syndrome (BMS), presenting as localized or more widely distributed oral mucosal pain, see 6.1 *Burning mouth syndrome*.

For persistent idiopathic dentoalveolar pain (PIDAP), sometimes associated with localized pain in the adjacent oral mucosa, see 6.3 *Persistent idiopathic dentoalveolar pain*.

Consideration must be given to patients presenting with oral mucosal pain in association with chronic widespread pain or other multiple pain conditions, which may be attributable to central sensitization or other mechanisms.

Diagnostic criteria:

A. Any pain in the oral mucosa[1] fulfilling criterion C below
B. Clinical, laboratory, imaging and/or anamnestic evidence of a lesion or disorder of the oral mucosal tissues known to be able to cause pain[2]
C. Evidence of causation demonstrated by all the following:
   1. location of the pain corresponds to the site(s) of the lesion or disorder[1]
   2. pain developed in temporal relation to the appearance or onset of the lesion or disorder
   3. pain is exacerbated by manipulation of the affected oral mucosa
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. The pain may also refer and/or radiate to other ipsilateral orofacial locations.
2. The lesion or disorder is specified in each subform.

Comments:
Pain involving the oral mucosa may have local or distant causes. Oral mucosal pain is often characterized by a burning, stinging or sore sensation. Various mucosal lesions such as ulcers, erosions and vesicles are common causes of oral mucosal pain.

The terms *stomatitis* and *oral mucositis* are often used as synonyms, but they do not reflect identical processes. *Stomatitis* refers to any inflammatory condition of oral mucosa occurring because of local infections or injuries or underlying systemic diseases. *Mucositis* occurs due to radiation or chemotherapeutic agents.

A large variety of local mucosal and systemic diseases are associated with pain due to formation of ulcers or erosions. These lesions differ regarding their extension into the oral mucosa.

- A *mucosal ulcer* is defined as a loss of surface tissue with disintegration and necrosis of epithelial tissue. It involves damage to both epithelium and lamina propria. It penetrates the epithelial–connective tissue border, and has its base at a deep level in the submucosa, and in some cases even within the muscle or periosteum.
- A *mucosal erosion* is defined as a superficial break on the mucous membrane with loss of the superficial epithelial cells and minor damage to the underlying lamina propria.

1.2.1.1 Oral mucosal pain attributed to oral mucosal inflammation

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1 *Oral mucosal pain*, and criterion C below

B. Inflammation of the oral mucosa has been diagnosed
C. Pain developed in close temporal relation to the inflammation, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Comment:
Mucosal pain associated with ulcers or other lesions is often associated with high levels of pain-related unpleasantness. The burning pain is often severe, and oral function (eating, talking), quality of life and sleep are frequently impaired.

1.2.1.1.1 Oral mucosal pain attributed to trauma or injury

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1.1 *Oral mucosal pain attributed to oral mucosal inflammation*, and criterion C below
B. Trauma or injury[1] has occurred involving the oral mucosal tissues
C. Both of the following:
   1. the pain is localized to the traumatized or injured tissues
   2. the pain developed within minutes to days after the trauma or injury
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Trauma or injury may be accidental or non-accidental, inflicted by others or self-inflicted, or iatrogenic, and is partially specified in the subforms.

1.2.1.1.1.1 Oral mucosal pain attributed to non-iatrogenic trauma or injury

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1.1.1 *Oral mucosal pain attributed to trauma or injury*
B. The trauma or injury[1] is non-iatrogenic.

Note:

1. The trauma may be mechanical, thermal, or chemical, and accidental or non-accidental, inflicted by others or self-inflicted.

Comments:
Causes of 1.2.1.1.1.1 *Oral mucosal pain attributed to non-iatrogenic trauma or injury* include not only accidental dental injuries but also micro-trauma caused, for example, during eating or drinking overly hot foods or drinks, and trauma due to tooth brushing or flossing or other interdental instruments. Examination may reveal the causative factor, such as an underlying mandibular or maxillary or dentoalveolar fracture, tooth root fracture or solely a soft tissue injury. Poorly fitting dentures may cause painful ulcerations. Over-erupted dentition, or parafunctional habits (biting or chewing on hard objects such as nails, pens, etc., or habitual chewing on lips, tongue or cheeks), may also cause local oral mucosal trauma with resultant inflammation and pain. Chemical burns may be related to misuse of anti-inflammatory tablets (e.g. sucking on tablets that are meant to be swallowed) or eating or drinking overly hot drinks or food. Self-harm may be a rare cause of oral mucosal trauma. In patients with dystonia or oral neuropathy, injury may be recurrent.

Traumatic injury of the oral mucosa causes acute inflammation and can be painful to a varying degree. Traumatic ulceration of the oral mucosa may be acute or chronic in nature, with the latter diagnostically more challenging due to underlying fibrosis and clinical appearance of neoplastic induration. A thorough clinical history will often alert the clinician to a traumatic aetiology or burns caused by warm food or chemicals.

1.2.1.1.1.1 *Oral mucosal pain attributed to non-iatrogenic trauma or injury* may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur.

1.2.1.1.1.2 Oral mucosal pain attributed to surgical or other local iatrogenic injury

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1.1.1 *Oral mucosa pain attributed to trauma or injury*
B. The trauma or injury is surgical or caused by other local iatrogenic procedure.[1]

Note:

1. Mechanical, thermal or chemical.

Comment:
Causes of 1.2.1.1.1.2 *Oral mucosal pain attributed to surgical or other local iatrogenic injury* include surgical trauma and injuries associated with dental or other oral treatments, such as dental injection injuries, and injuries due to direct local complications from oral procedures. Iatrogenic oral mucosa injury occurs during most dental surgery such as dental extractions and gingival or periodontal surgery. Thermal injuries occur

during use of electrocauterization and surgical laser, and chemical injuries may occur following inappropriate use of, for example, disinfectants or dental materials.

1.2.1.1.1.3 Oral mucosal pain attributed to radiation or chemotherapy

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1.1.1 *Oral mucosal pain attributed to trauma or injury*
B. The trauma or injury is attributed to radiation or chemotherapy.

Comments:
Oral mucositis is a term reserved for erythematous and ulcerative lesions of the oral mucosa that may occur in patients who receive anticancer radiotherapy to head and neck cancer involving the oral cavity, or chemotherapy. Their frequency and severity vary significantly with the type and dose of therapy. The lesions typically manifest as very painful erythema or ulcerations that compromise nutrition and oral hygiene as well as increasing the risks of local and systemic infection. The condition may also be accompanied by taste disturbances and xerostomia.

The pathogenesis of oral mucositis is multifactorial; a complex five-stage model has been proposed in its development.

When uncomplicated by infection, mucositis heals within 2–4 weeks after cessation of cytotoxic chemotherapy.

Mucositis may be exacerbated by local factors and infections. While oral complications are associated primarily with discomfort and interference with oral function, with impaired quality of life in patients who are also immunocompromised or debilitated, these complications can become life-threatening. In particular, infections associated with oral mucositis lesions can cause life-threatening systemic sepsis during periods of profound immunosuppression. Thus, management of mucositis pain is a primary component of any mucositis management strategy.

1.2.1.1.2 Oral mucosal pain attributed to infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1.1 *Oral mucosal pain attributed to oral mucosal inflammation*, and criterion C below
B. An infection[1] of the oral mucosa has been diagnosed[2]
C. Pain developed in close temporal relation to the infection, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. The infection may be bacterial, viral or fungal, and is specified in each subform.
2. Diagnosis is based on anamnestic information, clinical observations and/or microbiological analysis.

Comments:
Infection of the oral mucosal tissues causes acute inflammation. 1.2.1.1.2 *Oral mucosal pain attributed to infection* may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur.

The condition is subcategorized according to the causative microorganism.

1.2.1.1.2.1 Oral mucosal pain attributed to bacterial infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1.1.2 *Oral mucosal pain attributed to infection*
B. The infection is bacterial.

Comments:
Bacterial infections are the most common oral infections. Bacterial infection of the oral mucosal tissues causes acute inflammation. Oral mucosa pain is often associated with underlying dental pathology, with periodontal infection or dental periapical infections presenting as swelling, inflammation and pain of the overlying oral mucosa.

1.2.1.1.2.1 *Oral mucosal pain attributed to bacterial infection* may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur.

Acute necrotizing ulcerative gingivitis (ANUG), or necrotizing ulcerative gingivitis (NUG), periodontitis (NUP) or stomatitis (NUS) is an opportunistic oral mucosal infection caused by an array of bacteria in malnourished children, young adults and immune deficient patients. NUG is often the initial presentation, proceeding into NUP, NUS and ultimately noma (a form of gangrene affecting the face). Necrosis and ulceration of the oral mucosa, exquisite pain, severe halitosis, regional lymphadenopathy, malaise and fever differentiate this form of ulceration from others. When the alveolar bone becomes exposed, necrotic

bone sequestrae may develop and should be removed with the associated teeth.

Syphilis, caused by *Treponema pallidum* infection, continues to be widespread, with increasing rates among men who have sex with men. The primary lesion presents at the first site of mucosal inoculation, frequently the oral mucosa. A highly infective, painless solitary ulcer with indurated margins and ipsilateral lymphadenopathy is the most common, with healing within 3 weeks. Non-characteristic mucous patches alert to the development of secondary syphilis, frequently accompanied by a maculo-papular rash of the palmo-plantar surfaces of the hands and feet, and generalized lymphadenopathy.

Gonorrhoeal lesions may occur in the mouth at a site of inoculation or secondarily by haematogenous spread from a primary focus elsewhere. The earliest symptoms are a burning or itching sensation, dryness or heat in the mouth, followed by acute pain on eating or speaking. The tonsils and oropharynx are most frequently involved, and oral tissues may be diffusely inflamed or ulcerated. Saliva develops increased viscosity and fetid odour. In severe cases, submaxillary lymphadenopathy with fever occurs.

The emergence of multidrug-resistant *Mycobacterium tuberculosis* (TB) and the high numbers of HIV-infected individuals in East and Southern Africa have resulted in an increase of TB cases, urging inclusion in the differential diagnoses of orofacial pathology. Secondary TB in the form of painful, deep, irregular ulcers with indurated appearance, undermined edges and thick mucus-like material at the base of any aspect of the tongue are typical. Haematogenous spread from pulmonary TB or secondary inoculation of a traumatic ulcer with infected sputum is the most common pathogenesis. Primary oral TB is distinctly rare, usually associated with *Mycobacterium bovis*. Ulcers resemble chronic traumatic ulceration and even malignancy, urging a diagnostic biopsy. Associated symptoms of pain, fever, lymphadenopathy, hoarseness of voice and weight loss frequently accompany the ulcerations.

Acquired or congenital immunosuppression may lead to increased risk of mucosal infection. Patients on immunosuppressive therapy may develop a variety of opportunistic infections including pseudomembranous candidiasis and other fungal and viral infections. Tumour necrosis factor (TNF)-α therapy increases the risk of TB. Patients on infliximab and adalimumab with combined immunomodulatory therapy may be at increased risk of TB, histoplasmosis and coccidiomycosis infections. Antirheumatic drugs including methotrexate, abatacept and alefacept have increased the risk of herpes simplex and herpes zoster infections and TB.

1.2.1.1.2.2 Oral mucosal pain attributed to viral infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1.1.2 *Oral mucosal pain attributed to infection*
B. The infection is viral.[1]

Note:

1. Diagnosis is based on observation of a mucosal eruption in the area of pain together with polymerase chain reaction (PCR) identification of the virus from swabs taken from the area.

Comments:
Viral infection of the oral mucosal tissues causes acute inflammation. 1.2.1.1.2.2 *Oral mucosal pain attributed to viral infection* may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur.

Viral infections of the oral mucosa include HSV, VZV, HPV, CMV, coxsackieviruses and HIV infection. Note that ICHD-3 has a specific set of criteria for herpes zoster virus (13.1.2.1 *Painful trigeminal neuropathy attributed to acute herpes zoster*).

The infected oral mucosa tissues may often be ulcerated and painful to palpation. Severe local pain is often noted, in eating or drinking acidic or hot or cold foods or drinks. Pain is elicited on eating and may be so severe that the individual may be unable to eat or drink and become dehydrated.

Herpes simplex virus (HSV) is the most common virus to affect the oral mucosa. Herpetic gingivostomatitis, the primary HSV-1 infection, mostly affects children and presents either as an asymptomatic infection or with mucosal vesicles followed by quickly developing painful ulcerations affecting both keratinized and non-keratinized mucosa and gingivae. Fever, malaise, foul odour and cervical lymphadenopathy often accompany the pain. Adults with primary infection suffer symptomatic herpetic pharyngotonsillitis, initiated as vesicles that rapidly break down into painful shallow ulcerations.

Recurrent manifestations of the virus in the form of herpes labialis are most commonly initiated by various factors, including, but not limited to, stress, UV exposure or dental local anaesthetic. Initial prodromal stinging or burning is followed by a cluster of approximately five small fluid-filled vesicles on erythematous mucosa that rupture to leave painful shallow ulcers which coalesce and crust.

Herpangina (hand, foot and mouth disease), caused by coxsackieviruses, ECHO virus, and other enteroviruses, typically affects children under 10 years.

Red macules or vesicles are followed by self-limiting ulcerations, approximately 5 mm in diameter, on the anterior tonsillar pillars, soft palate, uvula and/or tonsils. Pyrexia, sore throat and headaches are common. Ulcers heal within 4–6 days.

Herpes zoster (shingles) signifies reactivation of dormant varicella-zoster virus (VZV or HHV-3) infection, mostly affecting old and debilitated patients. The infection is well known for its pruritic, vesicular skin rash, ulceration and crusting, all occurring concurrently and following the dermatome of the ganglion in which the virus established latency. Crusting is absent in the oral mucosa, where lesions instead present as ulcerating papules. Severe burning or stinging pain in the affected dermatome is followed by fluid-filled vesicles that rupture to leave painful shallow ulcerations, which may coalesce to form large denuded areas. Oral manifestations signify involvement of the mandibular or maxillary divisions of the trigeminal nerve, with pathognomonic abrupt termination of lesions along the midline. Osteonecrosis with tooth exfoliation has been reported, especially in immune deficient individuals. The infection often involves several locations in the anatomical distribution of the affected nerve branch (see also 4.1.2.1 *Trigeminal neuropathic pain attributed to herpes zoster* and 4.1.2.2 *Trigeminal postherpetic neuralgia*).

Human papilloma virus (HPV) may cause single or multiple papillary lesions. These lesions are rarely painful unless traumatized.

Epstein–Barr virus causes mononucleosis, which may involve sore throat and numerous small ulcers that precede lymphadenopathy. Gingival bleeding and petechiae at the border between soft and hard palate are other clinical features.

1.2.1.1.2.3 Oral mucosal pain attributed to fungal infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1.1.2 *Oral mucosal pain attributed to infection*
B. The infection is fungal.

Comments:
Fungal infection of the oral mucosal tissues causes acute inflammation. 1.2.1.1.2.3 *Oral mucosal pain attributed to fungal infection* may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur.

In recent times, the prevalence of oral fungal infections other than candidiasis has been on the rise. Immunodeficiency diseases such as HIV infection and AIDS, immunosuppressive therapy and prolonged usage of broad-spectrum antibiotics and corticosteroids are some of the notable reasons for disease emergence, which occurs when the oral homoeostasis is disturbed. Diabetes and salivary gland hyperfunction are other predisposing factors.

The most common oral fungal infection is *Candida albicans*. Erythematous candidiasis presents with generalized erythema and pain. Median rhomboid glossitis affects the tongue and has three main types: pseudomembranous type, presenting with white patches that are easily wiped off, leaving a sore, erythematous and bleeding surface; erythematous type, with red macular lesions and often a burning sensation; and angular cheilitis type, which is characterized by sore cracks and redness at the angles of the mouth. Xerostomia, burning, stinging and itching sensations, and metal taste, are accompanying symptoms.

Other mycoses to be considered in the context of oral mucosal pain include mucormycosis, aspergillosis, histoplasmosis, blastomycosis and paracoccidioidomycosis. While all are uncommon, *Aspergillus* and Mucorales infections are the most frequently encountered and follow inhalation of the spores from soil, manure, grain, cereal or mouldy flour. Both are superficial and invasive opportunistic fungal infections, encountered in the oral cavity of, especially, immunocompromised patients.

1.2.1.1.3 Oral mucosal pain attributed to autoimmunity

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1.1 *Oral mucosal pain attributed to oral mucosal inflammation*, and criterion C below
B. An autoimmune disease or disorder known to be able to cause oral mucosal pain[1] has been diagnosed
C. Pain developed in close temporal relation to the autoimmune disease or disorder, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Note:

1. These include pemphigus, mucus membrane pemphigoid, recurrent aphthous stomatitis, oral lichen planus, erythema multiforme, Sjögren's syndrome, Behçets disease, graft versus host disease, lupus erythematosus (systemic or discoid type), erythema migrans, Crohn's disease, ulcerative colitis and coeliac disease.

Comments:
1.2.1.1.3 *Oral mucosal pain attributed to autoimmunity* may be mild to severe and is exacerbated by mechanical

provocation of the oral mucosa. Both elicited and spontaneous pain may occur. The prognosis for the pain depends on the outcome of treatment of the underlying autoimmune disorder.

Several dermatological immune-mediated vesiculoulcerative conditions may present with oral mucosal involvement, either concurrently with the skin pathology, as the initial presentation, or sometimes as the only clinical presentation.

Pemphigus, a group of immune-mediated subepithelial bullous dermatoses, is mediated by autoantibodies directed at the proteins of keratinocyte adhesion (desmosomes), causing acantholysis. Pemphigus vulgaris (PV) most commonly affects the oral cavity, its autoantibodies mainly directed against desmoglein-1 and 3 (mucocutaneous forms) or only 3 (mucosal forms). Patients, typically 40–60 years of age, present with thin-roofed, flaccid intra-epithelial bullae, which rupture promptly after development, resulting in large irregular areas of painful mucosal ulceration.

Mucous membrane pemphigoid (MMP) is a common systemic autoimmune blistering disease with preferential involvement of mucosal membranes. The antibodies are directed at the proteins of keratinocyte to connective tissue matrix adhesion or hemi-desmosomes (BP180 and laminin-332), causing the epithelium to split away from its underlying connective tissue bed. The subepithelial nature of the split results in thick-roofed vesicles, which may still be intact on examination. Rupture of the vesicles leaves ulcerative lesions devoid of any epithelium, covered by yellow-white slough.

Recurrent aphthous stomatitis (RAS) represents the most common form of oral mucosal ulceration encountered in otherwise healthy individuals. The term should be reserved for recurrent ulcers of the oral mucosa, not associated with any systemic disease and which typically commence in childhood or adolescence. Non-keratinized mucosa of the buccal cavity, lips and soft palate is most commonly affected. A variety of local and systemic factors, including immunologic, allergic, nutritional, microbial organisms and psychosocial stress, as well as immunosuppressive drugs, have been proposed as possible aetiological factors. Increased prevalence in close family members also indicates a possible genetic background. RAS has an atypical clinical presentation in HIV-infected patients and should always be considered in the differential diagnosis of oral mucosal ulceration in such patients. When RAS starts later in life, additional mucosal surfaces may be affected, and comprehensive medical history and physical examination should be considered to rule out inflammatory gastrointestinal disease such as Crohn's disease, coeliac disease, Behçet's disease, Sweet's syndrome, cyclic neutropenia, HIV infection and drug reactions, which may all present with *aphthous-like ulcers*. Clinically, RAS is subclassified into *RAS minor*, the most common variant, which typically presents with one to five ulcers less than 10 mm in diameter and surrounded by a bright red inflammatory halo, which heal spontaneously within 10–14 days, and *RAS major* (Sutton disease), which presents as deeper, larger (usually >10 mm in diameter), persistent ulcerations with irregular borders, typically taking weeks or months to heal.

Oral lichen planus (OLP) is a rather common, chronic inflammatory disorder affecting mainly middle-aged females. The pathogenesis remains uncertain, but various subsets of T-lymphocytes and mast cells play a role in the basal membrane damage. The disease may present with a diverse clinical spectrum, which includes the atrophic, erosive, ulcerative and, less commonly, bullous variants. The lesions typically affect the oral mucosa bilaterally, and are fairly symmetrical – presenting either solely as an oral mucosal disease or accompanied by desquamative gingivitis and/or cutaneous manifestations. In the case of the erosive and ulcerative types, painful pseudomembrane-covered ulcerations bordered by faint white striae are seen in a multifocal distribution. Recent meta-analyses determined the overall malignant transformation rate of OLP to be around 1%, most commonly affecting the tongue of older females, but this issue remains contentious.

Erythema multiforme (EM) is a T-cell-mediated type IV cytotoxic immune reaction to a variety of antigens (viral, bacterial, pharmacological or chemical) that results in apoptosis-mediated epithelial cell death. Anti-desmoplakin I and II antibodies were recently demonstrated as a possible instigator of the cytotoxic reaction. EM mostly affects young, otherwise healthy individuals, and is often recurrent and temporal with recurrent HSV infections. Oral lesions may either represent the start of further mucocutaneous involvement or appear in isolation, classically with swollen, cracked, haemorrhagic and crusted lips with or without mucosal blisters and ulcerations.

Sjögren's syndrome is a systemic autoimmune disease that frequently presents concomitantly with other systemic connective tissue or organ-specific autoimmune diseases. This association is well described for systemic lupus erythematosus and rheumatoid arthritis. The oral mucosal tissues can become abraded and even cut with dry foods, and sore. The presence of Sjögren's syndrome influences the expression of the other autoimmune disease to some degree, for instance by increasing fatigue and lymphoma risk.

Behçet's disease is an autoimmune multisystem disease of unknown aetiology. It is characterized by oral ulcers, genital ulcers and eye inflammation. There may

be dermatological symptoms along with neurological and vascular involvement. The oral lesion ulcers are painful and characterized by cyclic presentation affecting the lips, buccal mucosa, soft palate and tongue, with an appearance resembling aphthous lesions, a few millimeters to centimeters in diameter. The incidence of the disease is higher in Mediterranean and Asian populations, especially in Turkey.

Graft versus host disease is characterized by lichenoid, papular and erythematous lesions, and occasionally ulcerations and desquamation on the buccal and labial mucosa, the palate and dorsal part of the tongue. The oral lesions are often accompanied by fever, malaise, nausea and xerostomia. The oral findings may be caused by a combination of radiotherapy, chemotherapy, immunosuppressive medications, and secondary infections.

More than half of patients with systemic lupus erythematosus (SLE) may present with oral lesions, most frequently ulceration and pain of the buccal mucosa and lips during the early, active disease phase. Ulcerative lesions and erythematous lesions with or without radiating white striae may also be seen as part of the clinical spectrum of discoid lupus erythematosus (DLE). DLE is considered a potentially malignant disorder of the oral mucosa due to the increased prevalence of oral squamous cell carcinoma among this population, especially involving the lower lip.

Erythema migrans (geographic tongue, benign migratory glossitis) is a common oral inflammatory condition of unknown aetiology, with an estimated prevalence of 1–3%. About 30% of patients have oral discomfort, a burning and stinging sensation. It usually affects the tongue, although other oral sites may be involved. Presentation may include circular erythematous areas, often sharply defined by elevated, whitish border zones, located on the lateral, dorsal, anterior and/or ventral parts of the tongue. The erythematous appearance is due to atrophy and loss of filiform papillae lesions. The most commonly suggested associations are atopy and psoriasis. The disorder should not be confused with the characteristic rash of early Lyme disease.

Crohn's disease presents with multifocal, linear, nodular or diffuse mucosal thickenings in the labial and buccal mucosa and the mucobuccal folds. They may be associated with painful, persistent aphthous-like ulcerations and atrophic glossitis.

Ulcerative colitis presents with scattered, clumped or linearly oriented pustules on an erythematous mucosa at multiple oral sites. Some patients exhibit painful oral aphthous-like lesions in addition to the pustular lesions.

Coeliac disease may present with mucosal pain, commonly associated with aphthous-like ulcers. Malabsorption of iron and vitamin B may lead to burning, stinging sensations in the tongue.

Other rare autoimmune or idiopathic causes of oral mucosal ulceration causing pain and sensitivity include eosinophilic ulcer, giant cell arteritis hypereosinophilic syndrome, necrotizing sialometaplasia, polyarteritis nodosa, reactive arthritis (Reiter's syndrome), acute febrile neutrophilic dermatosis (Sweet's syndrome) and Wegener's granulomatosis.

1.2.1.1.4 Oral mucosal pain attributed to hypersensitivity or allergic reaction

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1.1 *Oral mucosal pain attributed to oral mucosal inflammation*, and criterion C below
B. A hypersensitivity or allergic reaction in the oral mucosa[1] has occurred
C. Pain developed in close temporal relation to the hypersensitivity or allergic reaction
D. Not better accounted for by another ICOP diagnosis.

Note:

1. The hypersensitivity or allergic reaction may be in association with dental material (such as temporary or permanent restorative or impression material), an oral hygiene product, a topical drug, a systemic drug, a food or food additive or another factor.

Comments:

1.2.1.1.4 *Oral mucosal pain attributed to hypersensitivity or allergic reaction* may be mild to severe and is exacerbated by mechanical provocation of the oral mucosae. Both elicited and spontaneous pain may occur.

Oral allergy syndrome (OAS) usually occurs in individuals who are allergic to pollen from trees, grasses or weeds. Fresh fruit, raw vegetables and raw nuts are common causes of OAS. The symptoms include an itching sensation and/or swelling of all or part of the lips, tongue, mouth and/or throat, but these can on occasion be severe and also include nausea and vomiting.

Dental materials, oral hygiene products and food additives may cause contact allergic reactions in the mouth with varied clinical presentation including stomatitis, lichenoid lesions, erosions, blisters and ulcerations.

Allergic reactions and oral mucosal hypersensitivity reactions are less common than cutaneous ones, probably because of allergen dilution and the continuous rinsing effects of normal saliva flow. Lesions may present with non-specific tissue oedema, erythema,

cracking, ulceration, hyperkeratotic white plaques and/or mucosal desquamation.

Allergic contact stomatitis, although rare, has been reported in association with dental impression materials, dental restorative materials, topical benzocaine application and, more commonly, cinnamon in toothpastes, mouth rinses and chewing gum. Lesions, localized or widely distributed, may appear as mixed red and white patches with ulceration, swelling of the cheeks and desquamation appearing on the lips, cheeks, tongue and gingivae.

A hypersensitivity reaction to either a systemic drug or direct contact with an offending agent may result in clinical and histological features reminiscent of lichen planus. The terms *oral lichenoid drug reaction* (OLDR) and *oral lichenoid contact lesion* (OLCL) are used respectively, and both may present with significant ulceration, usually with erythema and white striations at the periphery of the ulceration. A temporal or spatial association with an offending agent can usually be identified. Amalgam is often implicated in OLCL, confirmed by patch testing for mercury or amalgam sensitivity. OLDR is encountered with some frequency in patients treated with angiotensin-converting enzyme (ACE) inhibitors, NSAIDs and oral hypoglycaemic drugs.

In the case of drug-related hypersensitivity, lesions may start long after the introduction of the drug, and may remain for months after cessation thereof, complicating diagnosis and management.

Fixed drug eruption (FDE) is a form of hypersensitivity remarkable for its fixed anatomical nature and has been described with NSAIDs and other oxicam drugs, gabapentin, fluconazole and systemic antibacterial and antifungal drugs. FDE should be suspected in cases with a temporal association with drug ingestion, may be confirmed through patch testing or oral provocation tests, and managed through drug avoidance or substitution, while the acute lesions can be treated with topical or systemic steroids.

Drug-induced fibrosis epithelial hyperplasia or fibrovascular hyperplasia may occasionally be associated with painful presentation, probably due to underlying periodontal infection caused by difficulty with oral hygiene in these conditions.

1.2.1.1.5 Oral mucosal pain attributed to oral mucosal inflammation due to other cause

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1.1 *Oral mucosal pain attributed to oral mucosal inflammation*, and criterion C below
B. A disorder known to be able to cause oral mucosal inflammation, but other than those identified in 1.2.1.1.1 to 1.2.1.1.4,[1] has been diagnosed
C. Causation of the pain is clinically plausible
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Such disorders include endocrine disorders or alterations, dietary deficiency, haematological diseases, gastrointestinal diseases and dermatological diseases, and drug-induced disorders (not attributable to hypersensitivity or allergy).

Comments:

1.2.1.1.5 *Oral mucosal pain attributed to oral mucosal inflammation due to other cause* may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur.

Alteration of the physiological state, such as in pregnancy and menopause, may cause endocrine changes that manifest as oral mucosal discomfort and pain. Systemic disorders that can cause oral mucosal inflammation and pain include endocrine disease (hypothyroidism, diabetes mellitus), dietary deficiencies (iron, vitamin B complex, zinc), gastrointestinal disorders and drug-induced disorders (not attributable to hypersensitivity or allergy).

Iron, vitamin B12 and folate deficiency can cause atrophic glossitis, in which the filiform papillae of the dorsum of the tongue undergo atrophy, leaving a smooth, erythematous tongue. Other parts of the oral mucosa may also appear atrophic and red. Aphthous-like ulcers are common in severe cases. Burning or stinging sensations may precede clinically detectable oral lesions. Severe cases of vitamin B12 deficiency may also be associated with paraesthesia. Patients may have a predisposition to develop angular cheilitis.

Haematological disorders such as anaemia, gammopathies, haematinic deficiencies, leukaemia, myelodysplastic syndrome, neutropenia and other white cell dyscrasias may result in friable oral mucosa with resultant ulceration and pain.

Gastrointestinal disorders such as gastro-oesophageal reflux disorder and peptic ulceration may lead to malabsorption and related dietary deficiencies and, subsequently, to related oral mucosal pain.

Dermatological causes of painful mucosal lesions include dermatitis herpetiformis, linear IgA disease, epidermolysis bullosa and chronic ulcerative stomatitis.

Antineoplastic therapy-induced mucositis involves a complex cascade of events which is initiated by reactive oxygen species with extensive inflammation, atrophy, swelling, erythema and ulceration. It includes

chemotherapy-induced mucositis as well as radiation lesions. The latter occur in the exposed surfaces, while chemotherapy-induced mucositis affects the entire alimentary tract. The type and dosage of systemic cytotoxic agents, and the dosage and field of radiation, will affect the presence and severity of mucositis. Evidence-based guidelines for the management of cancer therapy-induced oral mucositis are established and should be referred to in all cases of patients receiving these agents.

Benign hyperplastic lesions or tumours involving oral mucosa are usually not directly associated with pain, but may become painful if traumatized and/or infected because of interference with, for example, occlusion or dentures (see 1.2.1.1.1 *Oral mucosal pain attributed to trauma or injury* and 1.2.1.1.2 *Oral mucosal pain attributed to infection*).

1.2.1.2 Oral mucosal pain attributed to malignant lesion

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.1 *Oral mucosal pain*, and criterion C below
B. Neoplasia of the oral mucosa has been diagnosed
C. Causation of the pain is plausible on the basis of anatomical and/or temporal association
D. Not better accounted for by another ICOP diagnosis.

Comments:
1.2.1.2 *Oral mucosal pain attributed to malignant lesion* may be mild to severe and is exacerbated by mechanical provocation of the oral mucosa. Spontaneous pain can occur.

The oral mucosa may be affected by an array of both primary and metastatic malignancies, which may all present as non-specific ulcers. Oral squamous cell carcinoma (OSCC) is the most common, frequently presenting as ulceration with clinical induration, fixation to the underlying tissues, rolled exophytic margins, pain and/or numbness.

1.2.2 Salivary gland pain

Description:
Pain caused by a lesion or disorder involving the salivary glands.

Diagnostic criteria:

A. Any pain in salivary gland tissue fulfilling criterion C
B. Clinical, laboratory, imaging and/or anamnestic evidence of a lesion or disorder of the salivary glands known to be able to cause pain[1]
C. Evidence of causation demonstrated by both of the following:
   1. location of the pain corresponds to the site(s) of the lesion or disorder
   2. either or both of the following:
      a) pain developed in temporal relation to the appearance or onset of the lesion or disorder
      b) pain is exacerbated by pressure applied to the affected salivary gland
D. Not better accounted for by another ICOP diagnosis.

Note:

1. The lesion or disorder is specified in each subform.

1.2.2.1 Salivary gland pain attributed to obstructive cause

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.2 *Salivary gland pain*, and criterion C below
B. Obstruction of the salivary duct[1] has been diagnosed
C. Pain developed in close temporal relation to the obstruction, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Obstruction may be due to sialolithiasis, mucus plug, space-occupying lesion or traumatic or iatrogenic injury of the salivary gland or salivary duct.

Comments:
Patients with obstruction of the salivary duct most commonly present with a history of acute intermittent pain and swelling of the affected major salivary gland. The degree of pain and swelling is dependent on the extent of salivary duct obstruction and the presence of secondary infection.

Infrequent causes for salivary gland pain include benign and malignant tumours of the salivary glands. These are usually not directly associated with pain but may cause pain related to obstruction of the gland or duct.

Iatrogenic causes include therapy-related injury, for example I^{131}-mediated: salivary gland function is affected after high-activity radioiodine ablation therapy in patients with differentiated thyroid cancer. Radioactive iodine is actively accumulated in salivary gland tissue, and sialadenitis is a common sequela along with decreased saliva secretion and xerostomia leading to salivary gland infection and pain.

1.2.2.2 Salivary gland pain attributed to infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.2 *Salivary gland pain*, and criterion C below
B. Infection[1] of the salivary gland(s) has been diagnosed[2]
C. Pain developed in close temporal relation to the infection, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. The infection may be bacterial or viral, and is specified in each subform.
2. Diagnosis is based on anamnestic information, clinical observations and/or microbiological analysis.

1.2.2.2.1 Salivary gland pain attributed to bacterial infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.2.2 *Salivary gland pain attributed to infection*
B. The infection is bacterial.

Comments:
The most common bacterial cause is *Staphylococcus* infection.

Bacterial sialadenitis can be either acute or chronic. A decreased saliva flow rate is the primary predisposing factor, and this allows retrograde microbial colonization of the duct, which may result in the development of acute or chronic suppurative infection. Acute sialadenitis is characterized by a painful swelling of a single salivary gland, commonly affecting the parotid gland. A purulent discharge may be expressed from the salivary duct orifice, and the patient may present with redness of the overlying skin or even abscess formation within the inflamed gland tissue, malaise, fever and cervical lymphadenopathy. Bacterial sialadenitis often occurs in immunocompromised patients and in elderly patients who suffer from salivary gland hypofunction due to systemic diseases, medication intake or dehydration, or it may be associated with obstruction of the salivary ducts by deposition of calculi, mucus plugs, tumour growth or by trauma. Chronic sialadenitis may develop following acute sialadenitis if the predisposing factors cannot be eliminated.

1.2.2.2.2 Salivary gland pain attributed to viral infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.2.2 *Salivary gland pain attributed to infection*
B. The infection is viral.

Comments:
Viral infections of the salivary glands include mumps, HIV and CMV infection, which can cause pain in addition to swelling.

Mumps mostly affects the parotid gland, with bilateral sudden enlargement, painful to palpation, but up to 25% of cases involve unilateral swelling. Severe local pain is often noted in moving the jaws when talking and chewing, especially if partial duct obstruction occurs. It typically affects children 4–6 years of age.

1.2.2.3 Salivary gland pain attributed to recurrent juvenile parotitis

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.2 *Salivary gland pain*, and criterion C below
B. Recurrent juvenile parotitis has been diagnosed
C. Pain developed or recurred in close temporal relation to onset or recurrence of the parotitis
D. Not better accounted for by another ICOP diagnosis.

Comments:
Juvenile recurrent parotitis is a common condition of the salivary glands in children, characterized by intermittent swelling of the parotid glands on one or both sides, with or without pain, and is generally associated with non-obstructive sialectasis of the parotid gland as well as salivary gland hypofunction. It has a biphasic age distribution, with peaks at 2–5 and 10 years of age. The most common symptoms are swelling, pain and fever. Symptoms are limited to about 3 days and may recur frequently, with about eight episodes per year.

It is diagnosed from the medical history and confirmed by sialography or ultrasonography. The aetiology is unclear but, in most patients, recurrent juvenile parotitis resolves during adulthood.

1.2.2.4 Salivary gland pain attributed to immunologically mediated disorder

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.2 *Salivary gland pain*, and criterion C below

B. An immunologically mediated disorder known to be able to cause salivary gland pain[1] has been diagnosed
C. Pain developed in close temporal relation to the disorder, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Note:

1. The most important of these is Sjögren's syndrome.

Comment:
Sjögren's syndrome is an autoimmune disease that results in salivary gland dysfunction. Symptoms include recurrent or persistent swelling of the salivary glands, dryness of the mouth, difficulty chewing, pain and a burning sensation of oral mucosa, chronic sore throat and pain with swallowing.

1.2.2.5 Salivary gland pain attributed to other cause

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.2 *Salivary gland pain*, and criterion C below
B. A lesion or disorder, known to be able to cause salivary gland pain but other than those identified in 1.2.2.1 to 1.2.2.4,[1] has been diagnosed
C. Pain developed in close temporal relation to the lesion or disorder, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Another cause of salivary gland pain may be allogeneic transplantation with a graft versus host disease (GVHD). Salivary glands are a major target of GVHD and manifest as hyposalivation and xerostomia, infection and subsequent pain.

1.2.3 Jaw bone pain

Description:
Pain caused by a lesion or disorder involving the jaw bone tissue.

Diagnostic criteria:

A. Any pain in the jaw[1] fulfilling criterion C
B. Clinical, laboratory, imaging and/or anamnestic evidence of a lesion or disorder of the jaw bone[2] known to be able to cause pain
C. Evidence of causation demonstrated by both of the following:
   1. pain is localized to the site of the jaw bone lesion[1]
   2. either or both of the following:
      a) pain developed in temporal relation to the appearance or onset of the jaw bone lesion or disorder
      b) pain is exacerbated by pressure applied to the jaw bone lesion
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. The pain may refer and/or radiate to other ipsilateral orofacial locations.
2. The lesion or disorder is specified in each subform.

1.2.3.1 Jaw bone pain attributed to trauma or injury

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.3 *Jaw bone pain*, and criterion C below
B. Trauma or injury involving the jaw bone has occurred
C. Pain developed in close temporal relation to the trauma or injury
D. Not better accounted for by another ICOP diagnosis.

Comments:
Jaw bone injury includes jaw fracture. Sports such as football, baseball and hockey, and motor vehicle collisions, account for a high percentage of facial injuries among young adults. Chin lacerations in particular are associated with mandibular fractures.

A mandible fracture may be present when the patient experiences restricted or abnormal mouth-opening; malocclusion also suggests the presence of a mandibular fracture. So does numbness of the chin present immediately following trauma.

1.2.3.2 Jaw bone pain attributed to infection

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.3 *Jaw bone pain*, and criterion C below
B. An infection[1] of the jaw bone tissue has been diagnosed
C. Pain developed in close temporal relation to the infection, or led to its discovery

D. Not better accounted for by another ICOP diagnosis.

Notes:

1. The infection may be bacterial, viral or fungal, and is specified in each subform.

Comments:
Intra-bony bacterial, viral and fungal infections may cause jaw bone pain. The most common are bacterial.

Infection can occur secondary to osteo(radio)necrosis of the jaws, which may contribute to the pain associated with osteonecrosis (see 1.2.3.5 *Jaw bone pain attributed to therapy*).

1.2.3.2.1 *Jaw bone pain attributed to bacterial infection*

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.3.2 *Jaw bone pain attributed to infection*
B. The infection is bacterial.

Comment:
Bacterial infections of the jaw bone tissue include osteomyelitis. Odontogenic infections can spread and cause osteomyelitis of the jaw, but osteomyelitis secondary to odontogenic infection is relatively uncommon. Severe mandibular pain is a common symptom of jaw osteomyelitis and can be accompanied by anaesthesia or hypaesthesia on the affected side. In protracted cases, mandibular trismus may develop.

1.2.3.2.2 *Jaw bone pain attributed to viral infection*

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.3.2 *Jaw bone pain attributed to infection*
B. The infection is viral.

Comment:
Viral infections of the jaw bone tissue include herpes zoster (HZ) (shingles), resulting from reactivation of the varicella-zoster virus. Complications include HZ-induced osteonecrosis. Unusual dental complications such as osteonecrosis, exfoliation of teeth, periodontitis, calcified and devitalized pulps, periapical lesions and resorption of roots, as well as developmental anomalies such as irregular short roots and missing teeth, may arise secondarily to involvement of second or third divisions of the trigeminal nerve by HZ.

1.2.3.2.3 *Jaw bone pain attributed to fungal infection*

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.3.2 *Jaw bone pain attributed to infection*
B. The infection is fungal.

Comments:
The most likely fungal infections of the jaw bone tissue are aspergillosis and mucormycosis.

Aspergillosis of the oral cavity is an uncommon condition which most frequently occurs in immunocompromised patients, such as those with haematological malignancies. Osteomyelitis caused by *Aspergillus* species is an infection that is often neglected. Invasive oral aspergillosis, though rare, is a potentially lethal disease and it should be considered in immunosuppressed patients with oral lesions.

Mucormycosis is a rare opportunistic infection mostly affecting immunocompromised patients, but, rarely, otherwise healthy individuals after tooth extraction. The organism implicated in mucormycosis is a saprophytic fungus, mainly *Rhizopus* or *Mucor*. It is the most deadly and rapidly progressing form of fungal infection affecting humans.

1.2.3.3 Jaw bone pain attributed to local benign lesion

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.3 *Jaw bone pain*, and criterion C below
B. A local benign lesion known to be able to cause jaw bone tissue pain[1] has been diagnosed
C. Pain developed in close temporal relation to the lesion, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Local benign lesions include giant cell tumour, osteoid osteoma and osteoblastoma.

Comments:
Benign bone tumours are often asymptomatic and discovered incidentally during evaluation for trauma or another condition. When they are symptomatic, benign bone tumours may present with localized pain, swelling, deformity or pathologic fracture. Most benign bone tumours have characteristic radiographic features. Advanced imaging techniques (e.g. computed tomography (CT), magnetic resonance imaging (MRI)) may be necessary to characterize bone tumours fully.

Giant cell tumour of bone (GCTB) is a relatively rare, benign osteolytic skeletal neoplasm of young adults. The most common presentation is pain and swelling. Skull and craniofacial bones are less commonly involved sites.

Patients with osteoid osteoma typically complain of progressively increasing pain that is worse at night and unrelated to activity. The pain is relieved by aspirin or other NSAIDs, usually within 20–25 minutes. Lack of relief by agents should prompt consideration of other diagnoses.

Patients with osteoblastoma typically complain of chronic, continuous pain. The radiographic findings of osteoblastoma are variable, and advanced imaging (e.g. CT or MRI) is often required for identification. Pain is not relieved by aspirin or other NSAIDs.

1.2.3.4 Jaw bone pain attributed to malignant lesion

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.3 *Jaw bone pain*, and criterion C below
B. A primary or secondary malignant lesion known to be able to cause jaw bone tissue pain[1] has been diagnosed
C. Pain developed in temporal relation to the lesion, or led to its discovery
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Jaw bone pain may be due to the direct mass effect of primary or metastatic tumours, or to the paraneoplastic effect of metastatic tumours.

Comment:
Jaw bone pain attributed to malignant lesions, whether primary or metastatic, may present with localized pain that may increase and wane over weeks or months.

1.2.3.4.1 *Jaw bone pain attributed to local malignancy*

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.3.4 *Jaw bone pain attributed to malignant lesion*
B. The malignant lesion is primary.[1]

Note:

1. Primary malignant lesions known to be able to cause jaw bone tissue pain include osteosarcoma, Langerhans' cell histiocytosis, non-Hodgkin lymphoma and multiple myeloma.

Comments:
Osteosarcoma is an uncommon tumour, but, myeloma excluded, is by far the most likely primary malignant tumour to arise in bone (although often considered secondary, attributed to sarcomatous transformation of Paget's disease of bone or other benign bone lesions). The majority of patients with osteosarcoma present with localized pain, typically of several months' duration. Pain frequently begins after an injury and may wax and wane over a few weeks or months. Systemic symptoms such as fever, weight loss and malaise are generally absent. The most common sites of involvement are distal femur, proximal tibia and proximal femur; presence in the jaw bones is rather rare.

In Langerhans' cell histiocytosis (LCH), radiologic studies typically demonstrate a lytic, 'punched out' appearance, sometimes with an accompanying soft tissue mass. Pain in the jaw and loose teeth may be presenting symptoms. Although bone lesions may be asymptomatic in some areas, those in the mouth are especially troublesome because of tooth loss and a high recurrence rate. Posterior regions of the jaw bones are affected more often than anterior regions.

Non-Hodgkin lymphoma is a lymphatic system tumour originating from either B or T lymphocytes and showing a high malignant potential. Non-specific symptoms, such as unclear primary dental pain and unresolved periapical swelling, can make accurate diagnosis of non-Hodgkin lymphoma difficult, which frequently leads to delayed diagnosis. A CT or cone beam computed tomography (CBCT) scan of the jaws and immunohistochemical staining of the biopsy specimen are recommended. When the lesion affects the bones of the jaws, it is rare in the mandible when compared to the maxilla: in the reported cases, only 0.6% are in the mandible.

Multiple myeloma is a condition in which plasma cells proliferate in the bone marrow, often resulting in extensive skeletal destruction with osteolytic lesions, osteopenia and/or pathologic fractures. Bone pain, particularly in the back or chest, and less often in the extremities, is present at the time of diagnosis in approximately 60% of patients.

1.2.3.4.2 *Jaw bone pain attributed to remote malignancy*

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.3.4 *Jaw bone pain attributed to malignant lesion*
B. The malignancy is remote.

Comment:
Remote malignant lesions cause pain through direct mass effects (including nerve compression and periosteal stretch) and paraneoplastic effect (a remote effect without metastatic spread to the jaw(s)).

1.2.3.5 Jaw bone pain attributed to therapy

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.3 *Jaw bone pain*, and criterion C below
B. A therapeutic intervention has resulted in a lesion or disorder known to be able to cause jaw bone pain[1]
C. Causation of the pain is plausible based on anatomical association with the lesion or disorder and/or temporal association[2] with its appearance or onset
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Such lesions or disorders include medication-related osteonecrosis of the jaws (MRONJ), osteoradionecrosis and post-extraction alveolar osteitis (dry socket).
2. The pain usually develops within hours to days of the appearance of the lesion causing it. However, the time to appearance of the lesion is therapy-specific: it may occur directly from an intervention such as surgery, but up to months or years after initiation of medication or radiation.

Comments:
Medication-related osteonecrosis of the jaw (MRONJ) is defined by the presence of necrotic bone (that is exposed or can be probed through a sinus tract) for more than 8 weeks in the maxillofacial region of an individual treated with bisphosphonate or other anti-resorptive (e.g. denosumab) or anti-angiogenic (e.g. bevacizumab) medications. MRONJ typically presents as pain, infection and necrotic bone in the mandible or maxilla in patients receiving these agents. Dentoalveolar surgery is a major risk factor.

Osteoradionecrosis is a complication of radiation therapy (RT), due to vascular obliteration and decreased vascular supply of the irradiated tissues. Symptoms of osteoradionecrosis can include pain, bad breath, dysgeusia, dysaesthesia or anaesthesia, trismus, difficulty with chewing and swallowing, speech difficulties, fistula formation, pathologic fracture and infection. The time to onset of osteoradionecrosis is quite variable. In some cases, it may be diagnosed shortly after completion of RT, while in other patients it may not be diagnosed for years after the original cancer treatment. The mandible is the most frequently affected bone while maxillary osteoradionecrosis is rare.

Alveolar osteitis (dry socket) is a complication of dental extractions and occurs more commonly in extractions involving mandibular molar teeth. It is associated with severe pain developing 2–3 days post-operatively. A socket that may be partially or totally devoid of blood clot is often found and some patients experience halitosis.

1.2.3.6 Jaw bone pain attributed to systemic disease

Diagnostic criteria:

A. Pain fulfilling criteria for 1.2.3 *Jaw bone pain*, and criterion C below
B. A systemic disease known to be able to cause jaw bone pain[1] has been diagnosed
C. Causation of the pain is clinically plausible
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Such diseases include sickle cell disease, Gaucher's disease and Paget's disease.

Comments:
Some systemic diseases present with repeated vaso-occlusive pain episodes, characterized by diffuse bone pain, punctuated by painful crises that often result in osteonecrosis (avascular necrosis).

Sickle cell disease (SCD) is characterized by a marked heterogeneity in clinical and haematological severity, with repeated vaso-occlusive pain episodes as the hallmark. These may occur as often as every week, or long stretches of time may pass with none. Pain episodes can lead to bone infarcts, necrosis and, over time, degenerative changes in marrow-containing bone, leading to a chronic state of pain in addition to the more acute painful episodes.

Gaucher's disease (GD) is an inborn error of metabolism that affects the recycling of cellular glycolipids, and is one of the most common lysosomal storage diseases. Skeletal disease is characterized by diffuse bone pain, punctuated by painful crises that often result in osteonecrosis (avascular necrosis).

Paget's disease of bone (PDB) is also known historically as *osteitis deformans*. PDB is a focal disorder of bone metabolism, characterized by an accelerated rate of bone remodelling, resulting in overgrowth of bone at single (monostotic PDB) or multiple (polyostotic PDB) sites. Commonly affected areas include the skull, spine,

pelvis and long bones of the lower extremity. Similarly to osteosarcomas, these may present with localized pain and swelling and typically occur in patients with polyostotic disease.

References

1.1.1 Pulpal pain

Abbott PV and Yu C. A clinical classification of the status of the pulp and the root canal system. *Austral Dent J* 2007; 52(suppl): S17–31.

Benoliel R, Sharav Y and Eliav E. Neurovascular orofacial pain. *J Am Dent Assoc* 2010; 141: 1094–1096.

Berman LH and Hartwell GR. Diagnosis. In: Cohen S and Hargreaves KM (eds) *Pathways of the pulp*. 9th ed. St Louis, MO: Mosby-Elsevier, 2006, pp.2–39.

Byers MR and Narhi MVO. Nerve supply of the pulpodentin complex and response to injury. In: Hargreaves KM and Goodis HE (eds) *Seltzer and Bender's dental pulp*. Chicago, IL: Quintessence, 2002, pp.151–179.

Falace DA, Reid K and Rayens MK. The influence of deep (odontogenic) pain intensity, quality, and duration on the incidence and characteristics of referred orofacial pain. *J Orofac Pain* 1996; 10: 232–239.

Garfunkel A, Sela J and Ulmansky M. Dental pulp pathosis: clinicopathologic correlations based on 109 cases. *Oral Surg Oral Med Oral Pathol* 1973; 35: 110–117.

Glick DH. Locating referred pulpal pains. *Oral Surg Oral Med Oral Pathol* 1962; 15: 613–623.

Hargreaves KM and Seltzer S. Pharmacologic control of dental pain. In: Hargreaves KM and Goodis HE (eds) *Seltzer and Bender's dental pulp*. Chicago, IL: Quintessence, 2002, pp.205–225.

Hasler JE and Mitchell DF. Painless pulpitis. *J Am Dent Assoc* 1970; 81: 671–677.

Kang, SH, Kim BS and Kim Y. Cracked teeth: distribution, characteristics, and survival after root canal treatment. *J Endod* 2016; 42: 557–562.

Mejare IA, Axelsson S, Davidson T, et al. Diagnosis of the condition of the dental pulp: a systematic review. *Int Endod J* 2012; 45: 597–613.

Michaelson PL and Holland GR. Is pulpitis painful? *Int Endod J* 2002; 35: 829–832.

Obermann M, Mueller D, Yoon MS, et al. Migraine with isolated facial pain: a diagnostic challenge. *Cephalalgia* 2007; 27: 1278–1282.

Patel S, Kanagasingam S and Pitt Ford T. External cervical resorption: a review. *J Endod* 2009; 35: 616–625.

Schuurmans TJ, Nixdorf DR, Idiyatullin DS, et al. Accuracy and reliability of root crack and fracture detection in teeth using magnetic resonance imaging. *J Endodontics* 2019; 45: 750–755.

Seo DG, Yi YA, Shin SJ, et al. Analysis of factors associated with cracked teeth. *J Endod* 2012; 38: 288–292.

Sharav Y, Katsarava Z and Benoliel R. Migraine and possible facial variants: neurovascular orofacial pain. In: Sharav Y and Benoliel R (eds) *Orofacial pain and headache*. 2nd ed. Chicago, IL: Quintessence Publishing Co., 2015, pp.319–362.

Sharav Y, Katsarava Z and Charles A. Facial presentations of primary headache disorders. *Cephalalgia* 2017; 37: 714–719.

Sharav Y, Leviner E, Tzukert A, et al. The spatial distribution, intensity and unpleasantness of acute dental pain. *Pain* 1984; 20: 363–370.

Sigurdsson A. Clinical manifestations and diagnosis. In: Ørstavik D and Pitt-Ford TR (eds) *Essential endodontology*. 2nd ed. Oxford: Blackwell Munksgaard, 2008, pp.235–261.

Smulson MH and Sieraski SM. Histophysiology and diseases of the dental pulp. In: Weine FS (ed.) *Endodontic therapy*. 5th ed. St Louis, MO: Mosby, 1996, pp.84–165.

Tyldesley WR and Mumford JM. Dental pain and the histological condition of the pulp. *Dent Pract Dent Rec* 1970; 20: 333–336.

Von Troil B, Needleman I and Sanz M. A systematic review of the prevalence of root sensitivity following periodontal therapy. *J Clin Periodontol* 2002; 29(3 suppl): 173–177.

1.1.2 Periodontal pain

Andreasen JO and Andreasen FM. Classification, etiology and epidemiology of traumatic dental injuries. In: Andreasen JO (ed.) *Textbook and color atlas of traumatic injuries to the teeth*. 3rd ed. Copenhagen: Munksgaard, 1994, pp.151–177.

Armitage GC. Development of a classification system for periodontal diseases and conditions. *Ann Periodontol* 1999; 4: 1–6.

Bastone EB, Freer TJ and McNamara JR. Epidemiology of dental trauma: a review of the literature. *Aust Dent J* 2000; 45: 2–9.

Costa CPS, Thomaz EBAF and Souza S de FC. Association between sickle cell anemia and pulp necrosis. *J Endod* 2013; 39: 177–181.

Gomes BP, Lilley JD and Drucker DB. Associations of endodontic symptoms and signs with particular combinations of specific bacteria. *Int End J* 1996; 29: 69–75.

Gutmann JL, Baumgartner JC, Gluskin AH, et al. Identify and define all diagnostic terms for

periapical/periradicular health and disease states. *J Endod* 2009; 35: 1658–1674.
Heasman PA and Hughes FJ. Drugs, medications and periodontal disease. *Br Dent J* 2014; 217: 411–418.
Horning GM and Cohen ME. Necrotizing ulcerative gingivitis, periodontitis, and stomatitis: clinical staging and predisposing factors. *J Periodontol* 1995; 66: 990–998.
Kaste LM, Gift HC, Bhat M, et al. Prevalence of incisor trauma in persons 6 to 50 years of age: United States 1988–1991. *J Dent Res* 1996; 75: 696–705 (Special Issue).
Kinane JF and Chestnutt IG. Smoking and periodontal disease. *Crit Rev Oral Biol Med* 2000; 11: 356–365.
Laux M, Abbott PV, Pajarola G, et al. Apical inflammatory root resorption: a correlative radiographic and histological assessment. *Int Endod J* 2000; 33: 483–493.
Levin LG, Law AS, Holland GR, et al. Identify and define all diagnostic terms for pulpal health and disease states. *J Endod* 2009; 35: 1645–1657.
Mejàre IA, Axelsson S, Davidson T, et al. Diagnosis of the condition of the dental pulp: a systematic review. *Int Endod J* 2012; 45: 597–613.
Ricucci D and Siqueira JF Jr. Biofilms and apical periodontitis: study of prevalence and association with clinical and histopathologic findings. *Endod* 2010; 36: 1277–1288.
Ricucci D, Siqueira JF Jr, Lopes WS, et al. Extraradicular infection as the cause of persistent symptoms: a case series. *J Endod* 2015; 41: 265–273.
Sharav Y and Benoliel R. Acute orofacial pain. In: Sharav Y and Benoliel R (eds) *Orofacial pain and headache*. 2nd ed. Chicago, IL: Quintessence Publishing Co., 2015, pp.141–161.
Sjögren U, Happonen RP, Kahnberg K-E, et al. Survival of *Arachnia propionica* in periapical tissue. *Int Endodont J* 1988; 21: 277–282.
The Dental Trauma Guide – an online evidence-based treatment guide produced in cooperation with the Resource Centre for Rare Oral Diseases and Department of Oral and Maxillofacial Surgery, Copenhagen University Hospital, www.dentaltraumaguide.org/ (accessed January 2020).
Tronstad L, Barnett F, Riso K, et al. Extraradicular endodontic infections. *Endod Dent Traumatol* 1987; 3: 86–90.
Yoshida M, Fukushima H, Yamamoto K, et al. Correlation between clinical symptoms and microorganisms isolated from root canals of teeth with periapical pathosis. *J Endod* 1987; 13: 24–28.

1.1.3 Gingival pain

Akintoye SO and Greenberg MS. Recurrent aphthous stomatitis. *Dent Clin North Am* 2014; 58: 281–297.
Al-Hashimi I, Schifter M, Lockhart PB, et al. Oral lichen planus and oral lichenoid lesions: diagnostic and therapeutic considerations. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2007; 103(suppl): S25.e1–12.
Andrade P, Brinca A and Goncalo M. Patch testing in fixed drug eruptions – a 20-year review. *Contact Dermatitis* 2011; 65: 195–201.
Bascones-Martínez A, García-García V, Meurman JH, et al. Immune-mediated diseases: what can be found in the oral cavity? *Int J Dermatol* 2015; 54: 258–270.
Batchelor JM and Todd PM. Allergic contact stomatitis caused by a polyether dental impression material. *Contact Dermatitis* 2010; 63: 296–297.
Benedix F, Schilling M, Schaller M, et al. A young woman with recurrent vesicles on the lower lip: fixed drug eruption mimicking herpes simplex. *Acta Derm Venereol* 2008; 88: 491–494.
Calapai G, Miroddi M, Mannucci C, et al. Oral adverse reactions due to cinnamon-flavoured chewing gums consumption. *Oral Dis* 2014; 20: 637–643.
Carrozzo M, Togliatto M and Gandolfo S. [Erythema multiforme. A heterogeneous pathologic phenotype]. *Minerva Stomatol* 1999; 48: 217–226.
Farthing P, Bagan JV and Scully C. Mucosal disease series. Number IV. Erythema multiforme. *Oral Dis* 2005; 11: 261–267.
Feller L, Altini M, Chandran R, et al. Noma (cancrum oris) in the South African context. *J Oral Pathol Med* 2014; 43: 1–6.
Firth FA, Friedlander LT, Parachuru VP, et al. Regulation of immune cells in oral lichen planus. *Arch Dermatol Res* 2015; 307: 333–339.
Fitzpatrick SG, Hirsch SA and Gordon SC. The malignant transformation of oral lichen planus and oral lichenoid lesions: a systematic review. *J Am Dent Assoc* 2014; 145: 45–56.
Fourie J and Boy SC. Oral mucosal ulceration – a clinician's guide to diagnosis and treatment. *S Afr Dent J* 2016; 71: 500–508.
Fukiwake N, Moroi Y, Urabe K, et al. Detection of autoantibodies to desmoplakin in a patient with oral erythema multiforme. *Eur J Dermatol* 2007; 17: 238–241.
Gondivkar SM, Gadbail A and Chole R. Oral pregnancy tumor. *Contemp Clin Dent* 2010; 1: 190–192.
Gorouhi F, Davari P and Fazel N. Cutaneous and mucosal lichen planus: a comprehensive review

of clinical subtypes, risk factors, diagnosis, and prognosis. *ScientificWorldJournal* 2014; 2014: 742826.
Gupta S, Gupta S, Mittal A, et al. Oral fixed drug eruption caused by gabapentin. *J Eur Acad Dermatol Venereol* 2009; 23: 1207–1208.
Harman KE, Albert S and Black MM. Guidelines for the management of pemphigus vulgaris. *Br J Dermatol* 2003; 149: 926–937.
Hasan S. Desquamative gingivitis – a clinical sign in mucous membrane pemphigoid: report of a case and review of literature. *J Pharm Bioallied Sci* 2014; 6: 122–126.
Hertel M, Matter D, Schmidt-Westhausen AM, et al. Oral syphilis: a series of 5 cases. *J Oral Maxillofac Surg* 2014; 72: 338–345.
Jain P and Jain I. Oral manifestations of tuberculosis: step towards early diagnosis. *J Clin Diagn Res* 2014; 8: ZE18–21.
Kay LW. Investigations into the nature of pericoronitis. *Br J Oral Surg* 1966; 3: 188–205.
Khatibi M, Shakoorpour AH, Jahromi ZM, et al. The prevalence of oral mucosal lesions and related factors in 188 patients with systemic lupus erythematosus. *Lupus* 2012; 21: 1312–1315.
Kind F, Scherer K and Bircher AJ. Allergic contact stomatitis to cinnamon in chewing gum mistaken as facial angioedema. *Allergy* 2010; 65: 276–277.
Perusquia-Ortiz AM, Vazquez-Gonzalez D and Bonifaz A. Opportunistic filamentous mycoses: aspergillosis, mucormycosis, phaeohyphomycosis and hyalohyphomycosis. *J Dtsch Dermatol Ges* 2012; 10: 611–621.
Raber-Durlacher JE, Elad S and Barasch A. Oral mucositis. *Oral Oncol* 2010; 46: 452–456.
Rosenthal DI and Trotti A. Strategies for managing radiation-induced mucositis in head and neck cancer. *Semin Radiat Oncol* 2009; 19: 29–34.
Savin JA. Current causes of fixed drug eruption in the UK. *Br J Dermatol* 2001; 145: 667–668.
Scully C. Clinical practice. Aphthous ulceration. *N Engl J Med* 2006; 355: 165–172.
Shiboski CH, Patton LL, Webster-Cyriaque JY, et al. The Oral HIV/AIDS Research Alliance: updated case definitions of oral disease endpoints. *J Oral Pathol Med* 2009; 38: 481–488
Slebioda Z, Szponar E and Kowalska A. Recurrent aphthous stomatitis: genetic aspects of etiology. *Postepy Dermatol Alergol* 2013; 30: 96–102.
Theander E and Jacobsson LT. Relationship of Sjögren's syndrome to other connective tissue and autoimmune disorders. *Rheum Dis Clin North Am* 2008; 34: 935–947.
Van der Waal I. Oral potentially malignant disorders: is malignant transformation predictable and preventable? *Med Oral Patol Oral Cir Bucal* 2014; 19: e386–390.
Venables ZC, Narayana K and Johnston GA. Two unusual cases of allergic contact stomatitis caused by methacrylates. *Contact Dermatitis* 2016; 74: 126–127.
Von Arx DP and Husain A. Oral tuberculosis. *Br Dent J* 2001; 190: 420–422.
Warnakulasuriya S, Johnson NW and van der Waal I. Nomenclature and classification of potentially malignant disorders of the oral mucosa. *J Oral Pathol Med* 2007; 36: 575–580.
Yuan A and Woo SB. Adverse drug events in the oral cavity. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2015; 119: 35–47.

1.2.1 Oral mucosal pain

Abdalla-Aslan R, Benoliel R, Sharav Y, et al. Characterization of pain originating from oral mucosal lesions. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2016; 121: 255–261.
Akintoye SO and Greenberg MS. Recurrent aphthous stomatitis. *Dent Clin North Am* 2014; 58: 281–297.
Al-Hashimi I, Schifter M, Lockhart PB, et al. Oral lichen planus and oral lichenoid lesions: diagnostic and therapeutic considerations. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2007; 103(suppl): S25.e1–12.
Andrade P, Brinca A and Goncalo M. Patch testing in fixed drug eruptions – a 20-year review. *Contact Dermatitis* 2011; 65: 195–201.
Bascones-Martínez A, García-García V, Meurman JH, et al. Immune-mediated diseases: what can be found in the oral cavity? *Int J Dermatol* 2015; 54: 258–270.
Batchelor JM and Todd PM. Allergic contact stomatitis caused by a polyether dental impression material. *Contact Dermatitis* 2010; 63: 296–297.
Benedix F, Schilling M, Schaller M, et al. A young woman with recurrent vesicles on the lower lip: fixed drug eruption mimicking herpes simplex. *Acta Derm Venereol* 2008; 88: 491–494.
Calapai G, Miroddi M, Mannucci C, et al. Oral adverse reactions due to cinnamon-flavoured chewing gums consumption. *Oral Dis* 2014; 20: 637–643.
Carrozzo M, Togliatto M and Gandolfo S. Erythema multiforme. A heterogeneous pathologic phenotype. *Minerva Stomatol* 1999; 48: 217–226.
Duncan GG, Epstein JB, Tu D, et al. Quality of life, mucositis, and xerostomia from radiotherapy for

head and neck cancers: a report from the NCIC CTG HN2 randomized trial of an antimicrobial lozenge to prevent mucositis. *Head Neck* 2005; 27: 421–428.

Farthing P, Bagan JV and Scully C. Mucosal disease series. Number IV. Erythema multiforme. *Oral Dis* 2005; 11: 261–267.

Firth FA, Friedlander LT, Parachuru VP, et al. Regulation of immune cells in oral lichen planus. *Arch Dermatol Res* 2015; 307: 333–339.

Fitzpatrick SG, Hirsch SA and Gordon SC. The malignant transformation of oral lichen planus and oral lichenoid lesions: a systematic review. *J Am Dent Assoc* 2014; 145: 45–56.

Fourie J and Boy SC. Oral mucosal ulceration – a clinician's guide to diagnosis and treatment. *S Afr Dent J* 2016; 71: 500–508.

Fukiwake N, Moroi Y, Urabe K, et al. Detection of autoantibodies to desmoplakin in a patient with oral erythema multiforme. *Eur J Dermatol* 2007; 17: 238–241.

Gorouhi F, Davari P and Fazel N. Cutaneous and mucosal lichen planus: a comprehensive review of clinical subtypes, risk factors, diagnosis, and prognosis. *ScientificWorldJournal* 2014; 2014: 742826.

Gupta S, Gupta S, Mittal A, et al. Oral fixed drug eruption caused by gabapentin. *J Eur Acad Dermatol Venereol* 2009; 23: 1207–1208.

Harman KE, Albert S and Black MM. Guidelines for the management of pemphigus vulgaris. *Br J Dermatol* 2003; 149: 926–937.

Hertel M, Matter D, Schmidt-Westhausen AM, et al. Oral syphilis: a series of 5 cases. *J Oral Maxillofac Surg* 2014; 72: 338–345.

Jain P and Jain I. Oral manifestations of tuberculosis: step towards early diagnosis. *J Clin Diagn Res* 2014; 8: ZE18–21.

Khatibi M, Shakoorpour AH, Jahromi ZM, et al. The prevalence of oral mucosal lesions and related factors in 188 patients with systemic lupus erythematosus. *Lupus* 2012; 21: 1312–1315.

Kind F, Scherer K and Bircher AJ. Allergic contact stomatitis to cinnamon in chewing gum mistaken as facial angioedema. *Allergy* 2010; 65: 276–277.

Mravak-Stipetić M. Differential diagnostics of painful conditions of oral mucosa anticancer therapy. Rad 507. *Med Sci* 34: 55–73.

Perusquia-Ortiz AM, Vazquez-Gonzalez D and Bonifaz A. Opportunistic filamentous mycoses: aspergillosis, mucormycosis, phaeohyphomycosis and hyalohyphomycosis. *J Dtsch Dermatol Ges* 2012; 10: 611–621.

Raber-Durlacher JE, Elad S and Barasch A. Oral mucositis. *Oral Oncol* 2010; 46: 452–456.

Rosenthal DI and Trotti A. Strategies for managing radiation-induced mucositis in head and neck cancer. *Semin Radiat Oncol* 2009; 19: 29–34.

Saccucci M, Di Carlo G, Bossù M, et al. Autoimmune diseases and their manifestations on oral cavity: diagnosis and clinical management. *J Immunol Res* 2018; 2018: 6061825.

Savin JA. Current causes of fixed drug eruption in the UK. *Br J Dermatol* 2001; 145: 667–668.

Scully C. ABC of oral health: mouth ulcers and other causes of orofacial soreness and pain. *Br Med J* 2000; 321: 162–165.

Scully C. Clinical practice. Aphthous ulceration. *N Engl J Med* 2006; 355: 165–172.

Shiboski CH, Patton LL, Webster-Cyriaque JY, et al. The Oral HIV/AIDS Research Alliance: updated case definitions of oral disease endpoints. *J Oral Pathol Med* 2009; 38: 481–488.

Slebioda Z, Szponar E and Kowalska A. Recurrent aphthous stomatitis: genetic aspects of etiology. *Postepy Dermatol Alergol* 2013; 30: 96–102.

Sonis ST, Elting LS, Keefe D, et al. Perspectives on cancer therapy-induced mucosal injury: pathogenesis, measurement, epidemiology, and consequences for patients. *Cancer* 2004: 100(9 suppl): 1995–2025.

Theander E and Jacobsson LT. Relationship of Sjögren's syndrome to other connective tissue and autoimmune disorders. *Rheum Dis Clin North Am* 2008; 34: 935–947.

Treister N and Sonis S. Mucositis: biology and management. *Curr Opin Otolaryngol Head Neck Surg* 2007; 15: 123–129.

Van der Waal I. Oral potentially malignant disorders: is malignant transformation predictable and preventable? *Med Oral Patol Oral Cir Bucal* 2014; 19: e386–390.

Venables ZC, Narayana K and Johnston GA. Two unusual cases of allergic contact stomatitis caused by methacrylates. *Contact Dermatitis* 2016; 74: 126–127.

Vera-Llonch M, Oster G, Ford CM, et al. Oral mucositis and outcomes of allogeneic hematopoietic stem-cell transplantation in patients with hematologic malignancies. *Support Care Cancer* 2007; 15: 491–496.

Von Arx DP and Husain A. Oral tuberculosis. *Br Dent J* 2001; 190: 420–422.

Warnakulasuriya S, Johnson NW and van der Waal I. Nomenclature and classification of potentially malignant disorders of the oral mucosa. *J Oral Pathol Med* 2007; 36: 575–580.

Yuan A and Woo SB. Adverse drug events in the oral cavity. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2015; 119: 35–47.

1.2.2 Salivary gland pain

Bassim CW, Fassil H, Mays JW, et al. Oral disease profiles in chronic graft versus host disease. *J Dent Res* 2015; 94: 547–554.

Brook I. The bacteriology of salivary gland infections. *Oral Maxillofac Surg Clin North Am* 2009; 21: 269–274.

Guzzo M, Locati LD, Prott FJ, et al. Major and minor salivary gland tumors. *Crit Rev Oncol Hematol* 2010; 74: 134–148.

Klein Hesselink EN, Brouwers AH, de Jong JR, et al. Effects of radioiodine treatment on salivary gland function in patients with differentiated thyroid carcinoma: a prospective study. *J Nucl Med* 2016; 57: 1685–1691.

Leerdam CM, Martin HC and Isaacs D. Recurrent parotitis of childhood. *J Paediatr Child Health* 2005; 41: 631–634.

Napeñas JJ and Rouleau TS. Oral complications of Sjögren's syndrome. *Oral Maxillofac Surg Clin North Am* 2014; 26: 55–56.

Shiboski SC, Shiboski CH, Criswell L, et al. American College of Rheumatology classification criteria for Sjögren's syndrome: a data-driven, expert consensus approach in the Sjögren's International Collaborative Clinical Alliance cohort. *Arthritis Care Res (Hoboken)* 2012; 64: 475–487.

Wilson KF, Meier JD and Ward PD. Salivary gland disorders. *Am Fam Physician* 2014; 89: 882–888.

1.2.3 Jaw bone pain

Annibali S, Cristalli MP, Solidani M, et al. Langerhans cell histiocytosis: oral/periodontal involvement in adult patients. *Oral Dis* 2009; 15: 596–601.

Campanacci M, Baldini N, Boriani S, et al. Giant-cell tumor of bone. *J Bone Joint Surg Am* 1987; 69: 106–114.

Caparrotti F, Huang SH, Lu L, et al. Osteoradionecrosis of the mandible in patients with oropharyngeal carcinoma treated with intensity-modulated radiotherapy. *Cancer* 2017; 123: 3691–3700.

Caputo ND, Raja A, Shields C, et al. Re-evaluating the diagnostic accuracy of the tongue blade test: still useful as a screening tool for mandibular fractures? *J Emerg Med* 2013; 45: 8–12.

Chopra S, Kamdar D, Ugur OE, et al. Factors predictive of severity of osteoradionecrosis of the mandible. *Head Neck* 2011; 33: 1600–1605.

Copley L and Dormans JP. Benign pediatric bone tumors. Evaluation and treatment. *Pediatr Clin North Am* 1996; 43: 949–966.

Da Fonseca M, Oueis HS and Casamassimo PS. Sickle cell anemia: a review for the pediatric dentist. *Pediatr Dent* 2007; 29: 159–169.

Daly B, Sharif MO, Newton T, et al. Local interventions for the management of alveolar osteitis (dry socket). *Cochrane Database Syst Rev* 2012; 12: CD006968.

D'Ambrosio N, Soohoo S, Warshall C, et al. Craniofacial and intracranial manifestations of langerhans cell histiocytosis: report of findings in 100 patients. *AJR Am J Roentgenol* 2008; 191: 589–597.

Erdmann D, Follmar KE, Debruijn M, et al. A retrospective analysis of facial fracture etiologies. *Ann Plast Surg* 2008; 60: 398–403.

Gabrielli E, Fothergill AW, Brescini L, et al. Osteomyelitis caused by Aspergillus species: a review of 310 reported cases. *Clin Microbiol Infect* 2014; 20: 559–565.

Grabowski GA. Recent clinical progress in Gaucher disease. *Curr Opin Pediatr* 2005; 17: 519–524.

Grabowski GA, Andria G, Baldellou A, et al. Pediatric non-neuronopathic Gaucher disease: presentation, diagnosis and assessment. Consensus statements. *Eur J Pediatr* 2004; 163: 58–66.

Greenspan A. Benign bone-forming lesions: osteoma, osteoid osteoma, and osteoblastoma. Clinical, imaging, pathologic, and differential considerations. *Skeletal Radiol* 1993; 22: 485–500.

Gupta S, Sreenivasan V, Patil PB. Dental complications of herpes zoster: two case reports and review of literature. *Indian J Dent Res* 2015; 26: 214–219.

Jones AC, Prihoda TJ, Kacher JE, et al. Osteoblastoma of the maxilla and mandible: a report of 24 cases, review of the literature, and discussion of its relationship to osteoid osteoma of the jaws. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2006; 102: 639–650.

Kyle RA, Gertz MA, Witzig TE, et al. Review of 1027 patients with newly diagnosed multiple myeloma. *Mayo Clin Proc* 2003; 78: 21–33.

Mirabello L, Troisi RJ, Savage SA. Osteosarcoma incidence and survival rates from 1973 to 2004: data from the Surveillance, Epidemiology, and End Results Program. *Cancer* 2009; 115: 1531–1543.

Nilesh K and Vande AV. Mucormycosis of maxilla following tooth extraction in immunocompetent patients: Reports and review. *J Clin Exp Dent* 2018; 10: e300–e305.

Platt OS, Thorington BD, Brambilla DJ, et al. Pain in sickle cell disease. Rates and risk factors. *N Engl J Med* 1991; 325: 11–16.

Prasad KC, Prasad SC, Mouli N, et al. Osteomyelitis in the head and neck. *Acta Otolaryngol* 2007; 127: 194–205.

Ruggiero SL, Dodson TB, Fantasia J, et al.; American Association of Oral and Maxillofacial Surgeons. American Association of Oral and Maxillofacial Surgeons position paper on medication-related osteonecrosis of the jaw—2014 update. *J Oral Maxillofac Surg* 2014; 72: 1938–1956.

Sharif MO, Dawoud BE, Tsichlaki A, et al. Interventions for the prevention of dry socket: an evidence-based update. *Br Dent J* 2014; 217: 27–30.

Van Staa TP, Selby P, Leufkens HG, et al. Incidence and natural history of Paget's disease of bone in England and Wales. *J Bone Miner Res* 2002; 17: 465–471.

Wyers MR. Evaluation of pediatric bone lesions. *Pediatr Radiol* 2010; 40: 468–473.

Yildiz C, Erler K, Atesalp AS, et al. Benign bone tumors in children. *Curr Opin Pediatr* 2003; 15: 58–67.

Zou H, Yang H, Zou Y, et al. Primary diffuse large B-cell lymphoma in the maxilla: A case report. *Medicine (Baltimore)* 2018; 97: e10707.

2. Myofascial orofacial pain

General comments:
Temporomandibular disorders (TMDs) is a term used to describe a number of painful and non-painful disorders affecting the muscles of mastication, the temporomandibular joint (TMJ) and contiguous structures. The Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) published by INfORM are reliable and universally accepted. Nevertheless, controversy remains regarding terminology of chronic muscle pain. Whereas DC/TMD uses myalgia and myofascial pain, other suggestions include, for example, persistent orofacial muscle pain. ICOP uses myofascial orofacial pain as an overarching label, adhering to the term myofascial in recognition of the lack of concrete evidence linking pain to specific structures or tissues in the muscle. Furthermore, the term temporomandibular disorder is maintained to align with DC/TMD.

The temporal distinction of the myofascial pains in ICOP is novel (not found in DC/TMD), but follows the general principles from ICHD-3 for 2. *Tension-type headache*: that is, similar criteria regarding episode frequency have been introduced. While there may be debate about the clinical significance of infrequent myofascial pains (occurring less than once a month), there appears to be solid evidence in favour of separating these from more frequently occurring pain conditions. Future studies using the proposed temporal distinction between myofascial pains may reveal therapeutic implications.

Whereas some dentists and many orofacial pain (OFP) experts may manage cervical muscle pain in addition to jaw muscle pain, it was decided at this stage not to include a classification for cervical muscle pains. However, as a suggestion, such pain could be classified by adhering to similar principles (i.e. in terms of frequency, local findings on examination and with or without referral of pain). The same could be applied to other muscles in the orofacial region (e.g. the tongue or swallowing muscles).

With regard to pain referral, it is widely recognized and accepted that both acute and chronic types of pain in the orofacial muscles may be associated with this clinical phenomenon (i.e. pain is perceived at a different site from that of the origin of the nociceptive or noxious stimulus). The pathophysiological significance of this remains unclear, as do the therapeutic implications; however, from a diagnostic point of view, it remains important to distinguish referred pain from local pains. Consequently, all diagnoses of myofascial pain are subcategorized according to the presence or absence of pain referral during palpation. The definition of pain referral follows that of DC/TMD.

DC/TMD also operates with a category of pain spread, which, in contrast to pain referral, remains within the boundary of the affected anatomical structure. For research purposes, specific criteria for myofascial pain with pain spread, according to DC/TMD, can be applied if needed.

A further comment is that DC/TMD is not restricted to the temporalis and masseter muscles only; rather, examination criteria specify those muscles because (a) there is higher examiner reliability and (b) nearly all individuals with painful myofascial TMD have pain in at least one of these. There is, however, no logical necessity for positive signs not to be found in other masticatory muscles. The restriction to temporalis and masseter does exclude individuals who may have highly localized myalgia in other masticatory muscles, and to the clinician this will also appear as a needless restriction. Thus, in future revisions, the ICOP classification may be extended to other orofacial muscles.

A key criterion in muscle pain diagnosis in DC/TMD is that patients will report pain on provocation tests: standardized palpation and/or pain on jaw opening. This is reflected in ICOP criteria. Several studies have addressed the issue of additional dynamic/static testing of the jaw muscles and TMJ to increase diagnostic sensitivity and specificity. More research is needed before such testing can be recommended for general inclusion.

The specific instructions and criteria for a positive finding in provocation tests should follow DC/TMD descriptions, emphasizing the importance of standardization of palpation force and duration: 1 kg for 2 seconds to establish provocation of pain on palpation and 1 kg for 5 seconds to establish pain referrals (or spread). It should be noted that, while palpation force has extensive empirical support with respect to distinguishing cases from non-cases, duration of palpation has only preliminary empirical support.

Primary versus secondary pain
Based on the International Association for the Study of Pain (IASP) classification of chronic pain conditions, a distinction is made in this chapter between primary and secondary pain conditions. In primary pain conditions, the specific aetiology or cause cannot be determined – that is, they are idiopathic, although substantial knowledge may exist regarding their pathophysiological mechanisms. In secondary pain conditions, the pain is secondary to, or caused by, another known medical condition or cause.

For secondary myofascial pains, the definitions of the *Expanded Taxonomy for Temporomandibular Disorders* are used for the underlying disorders (tendonitis, myositis and muscle spasm). These are suggested criteria for further research and have not yet been validated.

2.1 Primary myofascial orofacial pain

Description:
Pain in masticatory muscles, with or without functional impairment, not attributable to another disorder.

Diagnostic criteria:

A. Myofascial pain fulfilling criteria B–D
B. Occurring in one or more episodes,[1] or unremitting
C. Reported in the jaw, temple, ear and/or in front of ear, with both of the following:
   1. confirmation on examination of location(s) in the temporalis and/or masseter muscle(s)
   2. provoked by either or both of:
      a) palpation of the temporalis and/or masseter muscle(s)
      b) maximum unassisted or assisted jaw opening movement(s)
D. Modified[2] by jaw movement, function or parafunction (e.g. tooth-grinding or clenching)
E. Not better accounted for by another ICOP diagnosis.

Notes:

1. Episodes may be single or recurrent within any day, each lasting at least 30 minutes and with a total duration within the day of at least 2 hours.
2. Pain may be increased or decreased.

2.1.1 Acute primary myofascial orofacial pain

Description:
Mild to moderate levels of deep aching or pressing pain in the masticatory muscles, occurring episodically or unremittingly, often associated with functional impairment such as perceived difficulties in moving the lower jaw, chewing and/or yawning, etc., and with onset within the last 3 months.

Diagnostic criteria:

A. Myofascial pain fulfilling criteria for 2.1 *Primary myofascial orofacial pain*, and criterion B below
B. Onset within the last 3 months.

2.1.2 Chronic primary myofascial orofacial pain

Description:
Mild to moderate levels of deep aching or pressing pain in the masticatory muscles, occurring episodically or unremittingly, often associated with functional impairment such as perceived difficulties in moving the lower jaw, chewing and/or yawning, etc., and with onset more than 3 months ago. It is often associated with psychosocial distress.

Diagnostic criteria:

A. Myofascial pain fulfilling criteria for 2.1 *Primary myofascial orofacial pain*, and criteria B and C below
B. Onset >3 months ago
C. Recurring in at least 10 episodes, or unremitting.

2.1.2.1 Chronic infrequent primary myofascial orofacial pain

Diagnostic criteria:

A. Myofascial pain fulfilling criteria for 2.1.2 *Chronic primary myofascial orofacial pain*, and criterion B below
B. Occurring on <1 day/month.

2.1.2.2 Chronic frequent primary myofascial orofacial pain

Diagnostic criteria:

A. Myofascial pain fulfilling criteria for 2.1.2 *Chronic primary myofascial pain*, and criterion B below
B. Occurring on 1–14 days/month on average for >3 months (>12 and <180 days/year).

2.1.2.2.1 *Chronic frequent primary myofascial orofacial pain without pain referral*

Diagnostic criteria:

A. Myofascial pain fulfilling criteria for 2.1.2.2 *Chronic frequent primary myofascial pain*, and criterion B below
B. No report of pain at a site beyond the boundary of the muscle (temporalis or masseter) being palpated.

2.1.2.2.2 Chronic frequent primary myofascial orofacial pain with pain referral

Diagnostic criteria:

A. Myofascial pain fulfilling criteria for 2.1.2.2 *Chronic frequent primary myofascial pain*, and criterion B below

B. Report of pain at a site beyond the boundary of the muscle (temporalis or masseter) being palpated.

2.1.2.3 Chronic highly frequent primary myofascial orofacial pain

Diagnostic criteria:

A. Myofascial pain fulfilling criteria for 2.1.2 *Chronic primary myofascial pain*, and criterion B below
B. Occurring on >15 days/month on average for >3 months (>180 days/year).

2.1.2.3.1 *Chronic highly frequent primary myofascial orofacial pain without pain referral*

Diagnostic criteria:

A. Myofascial pain fulfilling criteria for 2.1.2.3 *Chronic highly frequent primary myofascial orofacial pain*, and criterion B below
B. No report of pain at a site beyond the boundary of the muscle (temporalis or masseter) being palpated.

2.1.2.3.2 *Chronic highly frequent primary myofascial orofacial pain with pain referral*

Diagnostic criteria:

A. Myofascial pain fulfilling criteria for 2.1.2.3 *Chronic highly frequent primary myofascial orofacial pain*, and criterion B below
B. Report of pain at a site beyond the boundary of the muscle (temporalis or masseter) being palpated.

2.2 Secondary myofascial orofacial pain

Description:
Myofascial pain caused by an underlying disorder (inflammation, infection or muscle spasm).

Diagnostic criteria:

A. Myofascial pain in any masticatory muscle, fulfilling criteria C and D
B. An underlying disorder known to be able to cause myofascial pain[1] has been diagnosed
C. Myofascial pain has both of the following characteristics:
    1. reported in the jaw, temple, ear and/or in front of ear, with both of the following:
        a) confirmation on examination of location(s) in the affected muscle(s) or tendon(s)
        b) provoked by palpation of the affected tendon(s) and/or maximum unassisted or assisted jaw opening movement(s)[2]
    2. modified[3] by jaw movement, function or parafunction (e.g. tooth-grinding or clenching)
D. Evidence of causation has been demonstrated[4]
E. Not better accounted for by another ICOP diagnosis.

Notes:

1. The disorder is specified in each subform.
2. These signs may be demonstrated during physical examination or, in the case of pain that has already resolved, reported in the history.
3. Pain may be increased or decreased.
4. The necessary evidence is specified in each subform.

2.2.1 *Myofascial orofacial pain attributed to tendonitis*

Description:
Pain of tendon origin, affected by jaw movement, function or parafunction and replicated by provocation-testing of the relevant masticatory tendon. Limitation of mandibular movement(s) secondary to pain may be present. The temporalis tendon is a common site of tendonitis and may refer pain to the teeth and other nearby structures. Tendonitis can also occur in other masticatory muscle tendons.

Diagnostic criteria:

A. Myofascial pain fulfilling criteria for 2.2 *Secondary myofascial orofacial pain*, and criterion C below
B. Tendonitis of one or more masticatory muscles has been diagnosed
C. Evidence of causation demonstrated by at least two of the following:
    1. myofascial pain has developed in temporal relation to onset of the tendonitis
    2. myofascial pain has significantly[1] worsened in parallel with progression of the tendonitis
    3. myofascial pain has significantly[1] improved or resolved in parallel with improvement in or resolution of the tendonitis
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Such that the patient describes a step-change in intensity.

2.2.2 Myofascial orofacial pain attributed to myositis

Description:
Pain caused by myositis affecting one or more masticatory muscles, with clinical characteristics of inflammation or infection: oedema, erythema and/or increased temperature. It generally arises acutely following direct trauma of the muscle or from infection, or chronically with autoimmune disease. Limitation of unassisted mandibular movements secondary to pain is often present. Calcification of the muscle can occur (i.e. myositis ossificans).

Diagnostic criteria:

A. Myofascial pain fulfilling criteria for 2.2 *Secondary myofascial orofacial pain*, and criterion C below
B. Myositis[1] in one or more masticatory muscles has been diagnosed[2,3]
C. Evidence of causation demonstrated by at least two of the following:
   1. myofascial pain has developed in temporal relation to onset of the myositis
   2. myofascial pain has significantly[4] worsened in parallel with progression of the myositis
   3. myofascial pain has significantly[4] improved or resolved in parallel with improvement in or resolution of the myositis
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Myositis may be due to inflammation, infection or trauma.
2. Diagnostic signs are oedema, erythema and/or increased temperature over the muscle(s).
3. Serologic tests reveal elevated enzyme levels (e.g. creatine kinase), markers of inflammation and the presence of autoimmune diseases.
4. Such that the patient describes a step-change in intensity.

2.2.3 Myofascial orofacial pain attributed to muscle spasm

Description:
Pain caused by sudden, involuntary, reversible tonic contraction of a muscle. Such spasm may affect any of the masticatory muscles. Acute malocclusion may be present.

Diagnostic criteria:

A. Myofascial pain fulfilling criteria for 2.2 *Secondary myofascial orofacial pain*, and criterion C below
B. Muscle spasm in one or more masticatory muscles has been diagnosed[1,2]
C. Evidence of causation demonstrated by at least two of the following:
   1. myofascial pain has developed in immediate temporal relation to onset of the spasm
   2. myofascial pain has significantly[3] worsened in parallel with progression of the spasm
   3. myofascial pain has significantly[3] improved or resolved in parallel with improvement in or resolution of the spasm
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Limited range of jaw movement in a direction that elongates the affected muscle(s) is diagnostic: for example, for jaw closing muscles, opening is limited to <40 mm; for lateral pterygoid muscle, ipsilateral movement is limited to <7 mm.
2. If the diagnosis needs to be confirmed, intramuscular electromyography (EMG) shows elevated activity when compared to contralateral unaffected muscle.
3. Such that the patient describes a step-change in intensity.

References

Benoliel R, Svensson P, Heir G, et al. Persistent orofacial muscle pain. *Oral Dis* 2011; 17: 23–41.

Koutris M, Visscher CM, Lobbezoo F, et al. Comorbidity negatively influences the outcomes of diagnostic tests for musculoskeletal pain in the orofacial region. *Pain* 2013; 154: 927–932.

Masuda M, Iida T, Exposto FG, et al. Referred pain and sensations evoked by standardized palpation of the masseter muscle in healthy participants. *J Oral Facial Pain Headache* 2018; 32: 159–166.

Osiewicz MA, Manfredini D, Loster BW, et al. Comparison of the outcomes of dynamic/static tests and palpation tests in TMD-pain patients. *J Oral Rehabil* 2018; 45: 185–190.

Peck CC, Goulet JP, Lobbezoo F, et al. Expanding the taxonomy of the diagnostic criteria for temporomandibular disorders. *J Oral Rehabil* 2014; 41: 2–23.

Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for clinical and research applications: recommendations of the International RDC/TMD Consortium Network and Orofacial Pain Special Interest Group. *J Oral Facial Pain Headache* 2014; 28: 6–27.

Svensson P, Michelotti A, Lobbezoo F, et al. The many faces of persistent orofacial muscle pain. *J Oral Facial Pain Headache* 2015; 29: 207–208.

Treede RD, Rief W, Barke A, et al. A classification of chronic pain for ICD-11. *Pain* 2015; 156: 1003–1007.

Visscher CM, Lobbezoo F and Naeije M. A reliability study of dynamic and static pain tests in temporomandibular disorder patients. *J Orofac Pain* 2007; 21: 39–45.

Visscher CM, Naeije M, De Laat A, et al. Diagnostic accuracy of temporomandibular disorder pain tests: a multicenter study. *J Orofac Pain* 2009; 23: 108–114.

3. Temporomandibular joint (TMJ) pain

General comments:
Like the myofascial pains, the temporomandibular joint (TMJ) pains have been divided into primary and secondary subtypes based on the International Association for the Study of Pain (IASP) classification of chronic pain conditions. In primary pain conditions, the specific aetiology or cause cannot be determined (i.e. they are idiopathic), although substantial knowledge may exist regarding their pathophysiological mechanisms. In secondary pain conditions, the pain is secondary to or caused by another known medical condition or cause.

For secondary TMJ pains, the *Expanded Taxonomy for Temporomandibular Disorders* and the definitions in the article on the clinical diagnosis of TMJ arthritis by Alstergren et al. are used for the underlying disorders. Arthritis, disc displacements, degenerative joint disease and subluxation are included. These conditions may require different and specific treatments, so that it is of importance to know why there is TMJ pain. There are other conditions that may contribute to TMJ pain; for example, generalized pain conditions that sensitize the tissues in and around the TMJ. This must be considered further in future studies.

In general, in the following criteria, the term 'attributed to' is preferred to 'caused by'. Whereas 'caused by' implies proven causality, which may be difficult to establish, and the relationships may go both ways, 'attributed to' means, more conservatively, 'believed, on the evidence available, to be caused by'.

The temporal distinction of the TMJ pains in ICOP is novel (not found in Diagnostic Criteria for Temporomandibular Disorders (DC/TMD)) but follows the general principles from ICHD-3 for 2. *Tension-type headache*: that is, similar criteria regarding episode frequency have been introduced. While there may be debate about the clinical significance of infrequent TMJ pains (occurring less than once a month), there appears to be solid evidence in favour of separating these from more frequently occurring pain conditions. Future studies using the proposed temporal distinction between TMJ pains may reveal therapeutic implications.

Inclusion of subforms of TMJ pain with or without pain referral keeps the classification structure in line with that of the myofascial pains. It is widely recognized and accepted that both acute and chronic subtypes of TMJ pain can be associated with this clinical phenomenon (i.e. pain is perceived at a different site from that of the origin of the nociceptive or noxious stimulus). The pathophysiological significance of this remains unclear, as do the therapeutic implications; however, from a diagnostic point of view it remains important to distinguish referred pain from local pains. Consequently, TMJ pains are subcategorized according to the presence or absence of pain referral on palpation. Nevertheless, these subforms must, today, be regarded only as research topics and not considered for clinical use. Future research may show whether this subdivision has purpose.

DC/TMD also operates with a category of pain spread, which, in contrast to pain referral, remains within the boundary of the affected anatomical structure. For research purposes, the specific criteria for TMJ pain with pain spread, according to the DC/TMD, can be applied if needed.

Subforms with systemic or non-systemic TMJ arthritis are included, since treatment plans and prognosis may differ according to whether the arthritis is of local or systemic origin.

The working group considered including a subtype of 'idiopathic temporomandibular joint pain'. However, since the overlap with 3.1 *Primary temporomandibular joint pain* would be substantial, if not total, this diagnosis probably would not contribute usefully to research or clinical work.

3.1 Primary temporomandibular joint pain

Description:
Pain localized to the temporomandibular joint (TMJ), occurring at rest or during jaw movement or palpation, with no known causative disorder. The diagnosis corresponds fully to the DC/TMD diagnosis *temporomandibular joint pain*.

Diagnostic criteria:

A. Pain in and/or in front of the ear(s), fulfilling criteria B–D
B. Occurring in one or more episodes,[1] or unremitting
C. Both of the following:
    1. confirmation on examination of location in the area(s) of one or both temporomandibular joint(s)
    2. provoked by either or both of:
        a) palpation of and/or around the lateral pole(s) of the mandibular condyle(s)
        b) maximum unassisted or assisted jaw opening, right or left lateral and/or protrusive movement(s)
D. Modified[2] by jaw movement, function or parafunction (e.g. tooth-grinding or clenching)
E. Not better accounted for by another ICOP diagnosis.

Notes:

1. Episodes may be single or recurrent within any day, each lasting at least 30 minutes and with a total duration within the day of at least 2 hours.
2. Pain may be increased or decreased.

3.1.1 Acute primary temporomandibular joint pain

Description:
Primary TMJ pain with onset within the last 3 months.

Diagnostic criteria:

A. Pain fulfilling criteria for 3.1 *Primary temporomandibular joint pain*, and criterion B below
B. Onset within the last 3 months.

3.1.2 Chronic primary temporomandibular joint pain

Description:
Primary TMJ pain with onset more than 3 months ago.

Diagnostic criteria:

A. Pain fulfilling criteria for 3.1 *Primary temporomandibular joint pain*, and criteria B and C below
B. Onset >3 months ago
C. Recurring in at least 10 episodes, or unremitting.

3.1.2.1 Chronic infrequent primary temporomandibular joint pain

Diagnostic criteria:

A. Pain fulfilling criteria for 3.1.2 *Chronic primary temporomandibular joint pain*, and criterion B below
B. Occurring on <1 day/month.

3.1.2.2 Chronic frequent primary temporomandibular joint pain

Diagnostic criteria:

A. Pain fulfilling criteria for 3.1.2 *Chronic primary temporomandibular joint pain*, and criterion B below
B. Occurring on 1–14 days/month on average for >3 months (>12 and <180 days/year).

3.1.2.2.1 *Chronic frequent primary temporomandibular joint pain without pain referral*

Diagnostic criteria:

A. Pain fulfilling criteria for 3.1.2.2 *Chronic frequent primary temporomandibular joint pain*, and criterion B below
B. Pain on TMJ palpation localized to the immediate site of palpation.

3.1.2.2.2 *Chronic frequent primary temporomandibular joint pain with pain referral*

Diagnostic criteria:

A. Pain fulfilling criteria for 3.1.2.2 *Chronic frequent primary temporomandibular joint pain*, and criterion B below
B. Pain on TMJ palpation beyond the area of the joint.

3.1.2.3 Chronic highly frequent primary temporomandibular joint pain

Diagnostic criteria:

A. Pain fulfilling criteria for 3.1.2 *Chronic primary temporomandibular joint pain*, and criterion B below
B. Occurring on >15 days/month on average for >3 months (>180 days/year).

3.1.2.3.1 *Chronic highly frequent primary temporomandibular joint pain without pain referral*

Diagnostic criteria:

A. Pain fulfilling criteria for 3.1.2.3 *Chronic highly frequent primary temporomandibular joint pain*, and criterion B below
B. Pain on TMJ palpation localized to the immediate site of palpation.

3.1.2.3.2 *Chronic highly frequent primary temporomandibular joint pain with pain referral*

Diagnostic criteria:

A. Pain fulfilling criteria for 3.1.2.3 *Chronic highly frequent primary temporomandibular joint pain*, and criterion B below
B. Pain on TMJ palpation beyond the area of the joint.

3.2 Secondary temporomandibular joint pain

Description:
Pain localized to the temporomandibular joint (TMJ) and caused by another identified disorder, such as inflammation (due, for example, to trauma, infection, crystal deposition or autoimmune disorder), sensitization of the tissues, structural changes (such as osteoarthrosis, disc displacement or subluxation) or injury.

Diagnostic criteria:

A. Pain in the jaw, temple(s), ear(s) and/or in front of the ear(s), fulfilling criteria C and D
B. An underlying disorder known to be able to cause temporomandibular joint (TMJ) pain[1] has been diagnosed[2]
C. The pain has all the following three characteristics:
   1. located in the area(s) of the TMJ(s), with confirmation on examination
   2. provoked by either or both of:
      a) palpation of and/or around the lateral pole(s) of the mandibular condyle(s)[3]
      b) maximum unassisted or assisted jaw opening, right or left lateral and/or protrusive movement(s)[3]
   3. modified[4] by jaw movement, function or parafunction (e.g. tooth-grinding or clenching)
D. Evidence of causation demonstrated by at least two of the following:[5]
   1. the pain has developed in temporal relation to onset or substantial worsening of the presumed causative disorder, or has led to its discovery
   2. the pain has significantly[6] worsened in parallel with progression of the presumed causative disorder
   3. the pain has significantly[6] improved or resolved in parallel with improvement in or resolution[7] of the presumed causative disorder
E. Not better accounted for by another ICOP diagnosis.

Notes:

1. The disorder is specified in each subform.
2. Diagnosis is according to the *Expanded DC/TMD Taxonomy* definition.
3. These signs may be demonstrated during physical examination or, in the case of pain that has already resolved, reported in the history.
4. The pain may be increased or decreased.
5. Additional and/or alternative evidence of causation is specified in some subforms.
6. Such that the patient describes a step-change in intensity.
7. Spontaneously or through treatment.

3.2.1 Temporomandibular joint pain attributed to arthritis

Description:
TMJ pain caused by persistent inflammation of articular tissues (due, for example, to trauma, infection, crystal deposition or autoimmune disorder). TMJ pain is common in TMJ arthritis, but TMJ arthritis can be present without pain.

Diagnostic criteria:

A. Pain fulfilling criteria for 3.2 *Secondary temporomandibular joint pain*, and criterion C below
B. TMJ arthritis has been diagnosed
C. Evidence of causation demonstrated by either or both of the following:
   1. the pain has developed in close temporal relation to the TMJ arthritis, or has led to its diagnosis
   2. either or both of the following:
      a) the pain has significantly[1] worsened in parallel with worsening of the TMJ arthritis
      b) the pain has significantly[1] improved or resolved with treatment of the TMJ arthritis
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Such that the patient describes a step-change in intensity.

3.2.1.1 Temporomandibular joint pain attributed to non-systemic arthritis

Diagnostic criteria:

A. Pain fulfilling criteria for 3.2.1 *Temporomandibular joint pain attributed to arthritis*
B. Either of the following:
   1. no evidence exists of rheumatological disease
   2. evidence exists of a systemic inflammatory joint disease, but no evidence associates it with the TMJ pain.

3.2.1.2 Temporomandibular joint pain attributed to systemic arthritis

Diagnostic criteria:

A. Pain fulfilling criteria for 3.2.1 *Temporomandibular joint pain attributed to arthritis*, and criterion C below
B. Evidence exists of a systemic inflammatory joint disease
C. Both of the following:
   1. the pain has developed in close temporal relation to other symptoms and/or clinical or biological signs of onset of the systemic inflammatory joint disease, or has led to its diagnosis
   2. either or both of the following:

a) the pain has significantly[1] worsened in parallel with worsening of the systemic inflammatory joint disease
b) the pain has significantly[1] improved or resolved with treatment of the systemic inflammatory joint disease.

Note:

1. Such that the patient describes a step-change in intensity.

3.2.2 *Temporomandibular joint pain attributed to disc displacement*

Description:
TMJ pain caused by TMJ disc displacement in the absence of TMJ arthritis. TMJ pain can here be due to mechanical derangements within the joint.

Comment:
There are currently no specific criteria to relate TMJ pain to disc displacement with or without reduction. However, there is reason to believe that disc displacement can elicit TMJ pain upon jaw movement in certain circumstances, which implies that the TMJ pain is secondary. This is a topic that needs more research to develop optimal criteria, for which these are suggestions for a starting point.

3.2.2.1 Temporomandibular joint pain attributed to disc displacement with reduction

Diagnostic criteria:

A. Pain fulfilling criteria for 3.2 *Secondary temporomandibular joint pain*, and criterion C below
B. TMJ disc displacement with reduction has been diagnosed by both of the following:
   1. any TMJ noise(s) on jaw movement or function reported in the last 30 days, and/or during examination
   2. clicking, popping and/or snapping noise(s), detected with palpation during at least one of three repetitions, either:
      a) on both jaw opening and closing
      b) on both of:
         i. jaw opening or closing
         ii. right or left lateral and/or protrusive movements
C. Evidence of causation demonstrated by at least two of the following:
   1. the pain coincides precisely with the clicking, popping and/or snapping noise(s)
   2. the pain has developed in close temporal relation to the disc displacement, or has led to its diagnosis
   3. either or both of the following:
      a) the pain has significantly[1] worsened in parallel with worsening of the disc displacement
      b) TMJ pain has significantly[1] improved or resolved with treatment of the disc displacement
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Such that the patient describes a step-change in intensity.

3.2.2.1.1 *Temporomandibular joint pain attributed to disc displacement with reduction, with intermittent locking*

Diagnostic criteria:

A. TMJ pain fulfilling criteria for 3.2.2.1 *Temporomandibular joint pain attributed to disc displacement with reduction*
B. Intermittent jaw locking,[1] with limited mouth opening, and then unlocking, has occurred in the last 30 days.

Notes:

1. Even if only momentarily.

3.2.2.2 Temporomandibular joint pain attributed to disc displacement without reduction

Diagnostic criteria:

A. Pain fulfilling criteria for 3.2 *Secondary temporomandibular joint pain*, and criterion C below
B. TMJ disc displacement without reduction has been diagnosed by both of the following:
   1. jaw locked or caught, so as to prevent full opening
   2. limitation of jaw opening so as to interfere with eating
C. Evidence of causation demonstrated by one or more of the following:
   1. the pain has developed in close temporal relation to the disc displacement, or has led to its diagnosis
   2. the pain has significantly[1] worsened in parallel with worsening of the disc displacement
   3. the pain has significantly[1] improved or resolved with treatment of the disc displacement

D. Not better accounted for by another ICOP diagnosis.

Note:

1. Such that the patient describes a step-change in intensity.

3.2.3 Temporomandibular joint pain attributed to degenerative joint disease

Description:
TMJ pain caused by degenerative joint disorder (osteoarthrosis, osteoarthritis) in the absence of TMJ arthritis.

Comment:
The definitions of the terms 'osteoarthrosis' and 'osteoarthritis' of the TMJ, as of all other joints, are overlapping, disparate and unclear in the literature. For example, *osteoarthrosis* has been considered by some as a primarily non-inflammatory disease of the cartilage with resulting remodelling of the underlying bone tissue. In that context, osteoarthrosis may also (often) be accompanied by arthritis. Others have defined all these conditions as *osteoarthritis*. Historically, there has even been a difference between Europe and the US in these definitions. Nevertheless, it is important to consider that TMJ pain is not a very exact sign of arthritis. This means that there are many patients without TMJ pain who still may have arthritis (i.e. inflammation in articular tissues with cartilage and bone tissue destruction as a result). An example occurs in juvenile idiopathic arthritis (JIA), where TMJ pain is only seldom reported despite a high frequency of TMJ involvement. On the other hand, there are patients with TMJ arthritis with pain as the only symptom and without tissue destruction.

In a simplified model of the complex inflammatory process, inflammation may be considered as lying anywhere on a continuum from pain but no tissue destruction, to the opposite. The suggested ICOP criteria try to take these factors into consideration to stimulate research to clarify and contribute to our understanding.

Diagnostic criteria:

A. TMJ pain fulfilling criteria for 3.2 *Secondary temporomandibular joint pain*, and criterion C below
B. Degenerative TMJ disease has been diagnosed by both of the following:
   1. any TMJ noise(s) on jaw movement or function reported in the last 30 days, and/or during examination
   2. crepitus detected with palpation during maximal unassisted or assisted opening, lateral and/or protrusive movements
C. Evidence of causation demonstrated by one or more of the following:
   1. the pain has developed in close temporal relation to the degenerative TMJ disease, or has led to its diagnosis
   2. the pain has significantly[1] worsened in parallel with worsening of the degenerative TMJ disease
   3. the pain has significantly[1] improved or resolved with treatment of the degenerative TMJ disease
D. Not better accounted for by another ICOP diagnosis.

Note:

1. Such that the patient describes a step-change in intensity.

3.2.4 Temporomandibular joint pain attributed to subluxation

Description:
TMJ pain caused by subluxation, in the absence of TMJ arthritis. It is usually acute, and probably due to overstretching of tissues.

Diagnostic criteria:

A. Pain fulfilling criteria for 3.2 *Secondary temporomandibular joint pain*, and criterion C below
B. TMJ subluxation has been diagnosed by both of the following:
   1. jaw locking or catching,[1] in the last 30 days, preventing closure from the wide-open position
   2. inability to return the mouth from an open to a normal closed position without the performance of a specific manipulative manoeuvre
C. Evidence of causation demonstrated by one or more of the following:
   1. the pain has developed in close temporal relation to the subluxation, or has led to its diagnosis
   2. the pain has significantly[2] worsened in parallel with worsening of the subluxation
   3. the pain has significantly[2] improved or resolved with treatment of the subluxation
D. Not better accounted for by another ICOP diagnosis.

Notes:

1. Even if only momentarily.

2. Such that the patient describes a step-change in intensity.

References

Alstergren P, Pigg M and Kopp S. Clinical diagnosis of temporomandibular joint arthritis. *J Oral Rehabil* 2018; 45: 269–281.

Masuda M, Iida T, Exposto FG, et al. Referred pain and sensations evoked by standardized palpation of the masseter muscle in healthy participants. *J Oral Facial Pain Headache* 2018; 32: 159–166.

Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for clinical and research applications: recommendations of the International RDC/TMD Consortium Network and Orofacial Pain Special Interest Group. *J Oral Facial Pain Headache* 2014; 28: 6–27.

Svensson P, Michelotti A, Lobbezoo F, et al. The many faces of persistent orofacial muscle pain. *J Oral Facial Pain Headache* 2015; 29: 207–208.

Treede RD, Rief W, Barke A, et al. A classification of chronic pain for ICD-11. *Pain* 2015; 156: 1003–1007.

4. Orofacial pain attributed to lesion or disease of the cranial nerves

Classified elsewhere:
The following, less common clinical entities, in which pain presents mainly outside the orofacial region, are classified in ICHD-3 and not in ICOP:

13.3 *Pain attributed to a lesion or disease of nervus intermedius*, 13.4 *Occipital neuralgia*, 13.5 *Neck-tongue syndrome*, 13.6 *Painful optic neuritis*, 13.7 *Headache attributed to ischaemic ocular motor nerve palsy*, 13.8 *Tolosa–Hunt syndrome*, 13.9 *Paratrigeminal oculosympathetic (Raeder's) syndrome*, 13.10 *Recurrent painful ophthalmoplegic neuropathy*, 13.13 *Central neuropathic pain*.

General comments:
This section is based in large parts on ICHD-3 and the *International Classification of Diseases 11th Revision* (ICD-11)/International Association for the Study of Pain (IASP). Some small changes have been made. Idiopathic conditions (i.e. persistent idiopathic facial pain and burning mouth syndrome have been placed under 6. *Idiopathic orofacial pain*, since there is not yet sufficient evidence that these are unequivocally neuropathic pains. In certain disorders we have used the term *neuropathic pain* in preference to *painful neuropathy* to comply with IASP/ICD-11 criteria; while ICHD-3 supports the latter, much of the pain literature and IASP/ICD-11 is shifting to the former.

4.1 Pain attributed to lesion or disease of the trigeminal nerve

4.1.1 Trigeminal neuralgia

Previously used term:
Tic douloureux.

Description:
A disorder characterized by recurrent unilateral brief electric shock-like pains, abrupt in onset and termination, limited to the distribution of one or more divisions of the trigeminal nerve and triggered by innocuous stimuli. It may develop without apparent cause or be a result of another disorder. Additionally, there may or may not be concomitant continuous pain of moderate intensity within the affected division(s).

Diagnostic criteria:

A. Recurrent paroxysms of unilateral facial pain in the distribution(s) of one or more divisions of the trigeminal nerve, with no radiation beyond,[1] and fulfilling criteria B and C
B. The pain has all the following characteristics:
   1. lasting from a fraction of a second to 2 minutes[2]
   2. severe intensity[3]
   3. electric shock-like, shooting, stabbing or sharp in quality
C. Precipitated by innocuous stimuli within the affected trigeminal distribution[4]
D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Notes:

1. In a few patients, pain may radiate to another division, but remains within the trigeminal dermatomes.
2. Duration can change over time, with paroxysms becoming more prolonged. A minority of patients will report attacks predominantly lasting for >2 minutes.
3. Pain may become more severe over time.
4. Some attacks may be, or appear to be, spontaneous, but there must be a history or finding of pain provoked by innocuous stimuli to meet this criterion. Ideally, the examining clinician should attempt to confirm the history by replicating the triggering phenomenon. However, this may not always be possible because of the patient's refusal, the awkward anatomical location of the trigger and/or other factors.

Comments:
Other than the triggering phenomenon, most patients with 4.1.1 *Trigeminal neuralgia* fail to show sensory abnormalities within the trigeminal territory unless advanced methods are used (e.g. quantitative sensory testing). However, in some patients, clinical neurological examination may show sensory deficits. These should prompt neuroimaging investigations to explore the possible cause. Diagnosis of subforms such as 4.1.1.1 *Classical trigeminal neuralgia*, 4.1.1.2 *Secondary trigeminal neuralgia* or 4.1.1.3 *Idiopathic trigeminal neuralgia* is then possible.

When very severe, the pain often evokes contraction of the muscles of the face on the affected side (tic douloureux).

Mild autonomic symptoms such as lacrimation and/or redness of the ipsilateral eye may be present.

Following a painful paroxysm there is usually a refractory period during which pain cannot be triggered.

4.1.1.1 Classical trigeminal neuralgia

Previously used term:
Primary trigeminal neuralgia.

Description:
Trigeminal neuralgia developing without apparent cause other than neurovascular compression.

Diagnostic criteria:

A. Recurrent paroxysms of unilateral pain fulfilling criteria for 4.1.1 *Trigeminal neuralgia*
B. Demonstration on magnetic resonance imaging (MRI) or during surgery of neurovascular compression (not simply contact), with morphological changes[1] in the trigeminal nerve root.

Note:

1. Typically atrophy or displacement.

Comments:
Nerve root atrophy and/or displacement due to neurovascular compression are independently associated with signs and symptoms of 4.1.1 *Trigeminal neuralgia*. When these anatomical changes are present, the condition is diagnosed as 4.1.1.1 *Classical trigeminal neuralgia*.

The common site of compression is at the root entry zone, with compression by an artery more clearly associated with symptoms than compression by a vein. MRI techniques to measure volume and the cross-sectional area of the root are available. Atrophic changes may include demyelination, neuronal loss, changes in microvasculature and other morphological changes. While the exact mechanisms of how atrophic changes in the trigeminal nerve contribute to the generation of pain, some evidence suggests that, when present preoperatively, they predict a good outcome following microvascular decompression.

4.1.1.1.1 *Classical trigeminal neuralgia, purely paroxysmal*

Description:
Classical trigeminal neuralgia without persistent background pain.

Diagnostic criteria:

A. Recurrent paroxysms of unilateral facial pain fulfilling criteria for 4.1.1.1 *Classical trigeminal neuralgia*
B. Pain-free between attacks in the affected trigeminal distribution.

Comment:
4.1.1.1.1 *Classical trigeminal neuralgia, purely paroxysmal* is usually responsive, at least initially, to pharmacotherapy (especially carbamazepine or oxcarbazepine).

4.1.1.1.2 Classical trigeminal neuralgia with concomitant continuous pain

Previously used terms:
Atypical trigeminal neuralgia; trigeminal neuralgia type 2.

Description:
Classical trigeminal neuralgia with persistent background pain.

Diagnostic criteria:

A. Recurrent paroxysms of unilateral facial pain fulfilling criteria for 4.1.1.1 *Classical trigeminal neuralgia*
B. Concomitant continuous or near-continuous pain between attacks in the ipsilateral trigeminal distribution.

Comment:
Peripheral or central sensitization may account for the continuous pain.

4.1.1.2 Secondary trigeminal neuralgia

Description:
Trigeminal neuralgia caused by an underlying disease. Clinical examination shows sensory changes in a substantial percentage of these patients.

Diagnostic criteria:

A. Recurrent paroxysms of unilateral pain fulfilling criteria for 4.1.1 *Trigeminal neuralgia*, either purely paroxysmal or associated with concomitant continuous or near-continuous pain
B. An underlying disease has been demonstrated[1] that is known to be able to cause, and explain, the neuralgia[2]
C. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Notes:

1. MRI is best equipped to detect an underlying cause of 4.1.1.2 *Secondary trigeminal neuralgia*. Other investigations may include neurophysiological recording of trigeminal reflexes and trigeminal evoked potentials, suitable for patients who cannot undergo MRI.
2. Recognized causes are tumour in the cerebellopontine angle, arteriovenous malformation and multiple sclerosis.

4.1.1.2.1 *Trigeminal neuralgia attributed to multiple sclerosis*

Description:
Trigeminal neuralgia caused by a multiple sclerosis (MS) plaque or plaques in the pons or trigeminal root entry zone, and associated with other symptoms and/or clinical or laboratory findings of MS.

Diagnostic criteria:

A. Recurrent paroxysms of unilateral facial pain fulfilling criteria for 4.1.1 *Trigeminal neuralgia*
B. Both of the following:
   1. multiple sclerosis (MS) has been diagnosed
   2. an MS plaque at the trigeminal root entry zone or in the pons affecting the intrapontine primary afferents has been demonstrated by MRI, or its presence is suggested by routine electrophysiological studies[1] showing impairment of the trigeminal pathways
C. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Note:

1. Blink reflex or trigeminal evoked potentials.

Comments:
4.1.1.2.1 *Trigeminal neuralgia attributed to multiple sclerosis* occurs in 2–5% of patients with MS, sometimes bilaterally. Conversely, MS is detected in only 2–4% of cases of 4.1.1 *Trigeminal neuralgia*. Symptoms of trigeminal neuralgia are rarely a presenting feature of MS.

The lesion in the pons affects the intrapontine central terminals of the trigeminal afferents projecting to the trigeminal brainstem nuclei. Pontine lesions affecting the second-order neurons of the trigeminothalamic tract usually lead to non-paroxysmal pain and/or dysaesthesias, and should be given the ICHD-3 diagnosis of 13.13.1 *Central neuropathic pain attributed to multiple sclerosis*.

Some patients with MS are found to have neurovascular compression of the trigeminal root. It is thought that MS increases the susceptibility of the nerve root to the effects of compression, leading more readily to painful paroxysms.

Patients with 4.1.1.2.1 *Trigeminal neuralgia attributed to multiple sclerosis* benefit less from pharmacological and surgical interventions than those with 4.1.1.1 *Classical trigeminal neuralgia*.

4.1.1.2.2 *Trigeminal neuralgia attributed to space-occupying lesion*

Description:
Trigeminal neuralgia caused by contact between the affected trigeminal nerve and a space-occupying lesion.

Diagnostic criteria:

A. Recurrent paroxysms of unilateral facial pain fulfilling criteria for 4.1.1 *Trigeminal neuralgia*
B. Both of the following:
   1. a space-occupying lesion in contact with the affected trigeminal nerve has been demonstrated by imaging
   2. pain has developed after identification of the lesion, or led to its discovery
C. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comment:
Patients with 4.1.1.2.2 *Trigeminal neuralgia attributed to space-occupying lesion* may or may not have clinically detectable sensory signs, while electrophysiological tests such as trigeminal brainstem reflexes show abnormalities in nearly all cases.

4.1.1.2.3 *Trigeminal neuralgia attributed to other cause*

Description:
Trigeminal neuralgia caused by an underlying disease other than those described above.

Diagnostic criteria:

A. Recurrent paroxysms of unilateral facial pain fulfilling criteria for 4.1.1 *Trigeminal neuralgia*, either purely paroxysmal or associated with concomitant continuous or near-continuous pain, but not necessarily unilateral
B. Both of the following:
   1. a disorder, other than those described above but known to be able to cause trigeminal neuralgia,[1] has been diagnosed
   2. pain has developed after onset of the disorder, or led to its discovery
C. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Note:

1. Recognized causes are skull-base bone deformity, connective tissue disease, arteriovenous malformation, dural arteriovenous fistula and genetic causes of neuropathy or nerve hyperexcitability.

4.1.1.3 Idiopathic trigeminal neuralgia

Description:
Trigeminal neuralgia with neither electrophysiological tests nor MRI showing significant abnormalities.

Diagnostic criteria:

A. Recurrent paroxysms of unilateral pain fulfilling criteria for 4.1.1 *Trigeminal neuralgia*, either purely paroxysmal or associated with concomitant continuous or near-continuous pain
B. Neither 4.1.1.1 *Classical trigeminal neuralgia* nor 4.1.1.2 *Secondary trigeminal neuralgia* has been confirmed by adequate investigations[1,2]
C. Not better accounted for by another ICHD-3 or ICOP diagnosis.

Notes:

1. Including electrophysiological tests or MRI.
2. A contact between a blood vessel and the trigeminal nerve and/or nerve root is a common finding on neuroimaging in healthy subjects. When such a contact is found in the presence of 4.1.1 *Trigeminal neuralgia*, but without evidence of morphological changes (e.g. atrophy or displacement) in the nerve root, the criteria for 4.1.1.1. *Classical trigeminal neuralgia* are not fulfilled and the condition is considered idiopathic.

4.1.1.3.1 Idiopathic trigeminal neuralgia, purely paroxysmal

Diagnostic criteria:

A. Recurrent paroxysms of unilateral facial pain fulfilling criteria for 4.1.1.3 *Idiopathic trigeminal neuralgia*
B. Pain-free between attacks in the affected trigeminal distribution.

4.1.1.3.2 Idiopathic trigeminal neuralgia with concomitant continuous pain

Diagnostic criteria:

A. Recurrent paroxysms of unilateral facial pain fulfilling criteria for 4.1.1.3 *Idiopathic trigeminal neuralgia*
B. Concomitant continuous or near-continuous pain between attacks in the affected trigeminal distribution.

4.1.2 Other trigeminal neuropathic pain

Description:
Facial pain in the distribution of one or more branches of the trigeminal nerve caused by another disorder and indicative of neural damage. The primary pain is usually continuous or near-continuous, and commonly described as burning, squeezing, aching or likened to pins and needles. Superimposed brief pain paroxysms may occur but these are not the predominant pain type. This combination is distinct from that of 4.1.1 *Trigeminal neuralgia*. There are clinically detectable somatosensory changes within the trigeminal distribution, and mechanical allodynia and cold hyperalgesia/allodynia are common, fulfilling the IASP criteria for neuropathic pain. Allodynic areas may be much larger than the punctate trigger zones present in 4.1.1 *Trigeminal neuralgia*.

4.1.2.1 Trigeminal neuropathic pain attributed to herpes zoster

Description:
Unilateral facial pain of less than 3 months' duration in the distribution of one or more branches of the trigeminal nerve, caused by, and associated with other symptoms and/or clinical signs of, acute herpes zoster.

Diagnostic criteria:

A. Unilateral facial pain in the distribution(s) of a trigeminal nerve branch or branches, lasting <3 months
B. One or more of the following:
   1. herpetic eruption has occurred in the same trigeminal distribution (as the pain)
   2. Varicella-zoster virus (VZV) has been detected in the cerebrospinal fluid (CSF) by polymerase chain reaction (PCR)
   3. direct immunofluorescence assay for VZV antigen or PCR assay for VZV DNA is positive in cells obtained from the base of lesions
C. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comments:
Herpes zoster affects the trigeminal ganglion in 10–15% of cases, with the ophthalmic division being singled out in some 80% of patients.

Rarely, pain is not followed by an eruption or rash (*zoster sine herpete*). The diagnosis in such cases is confirmed by PCR detection of VZV DNA in the cerebrospinal fluid.

4.1.2.1 *Trigeminal neuropathic pain attributed to herpes zoster* is usually burning, stabbing/shooting, tingling or aching, and accompanied by cutaneous allodynia. Ophthalmic zoster may be associated with third, fourth and sixth cranial nerve palsies.

Herpes zoster is common in immunocompromised patients, occurring in about 10% of those with lymphoma and 25% of patients with Hodgkin disease.

4.1.2.2 Trigeminal postherpetic neuralgia

Previously used term:
Postherpetic trigeminal neuropathy

Description:
Unilateral facial pain persisting or recurring for at least 3 months in the distribution(s) of one or more branches of the trigeminal nerve, with variable sensory changes, caused by herpes zoster.

Diagnostic criteria:

A. Unilateral facial pain in the distribution(s) of a trigeminal nerve branch or nerve branches, persisting or recurring for >3 months, and fulfilling criterion C
B. Herpes zoster has affected the same trigeminal nerve branch or branches
C. Pain developed in temporal relation to the acute herpes zoster infection[1]
D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Note:

1. Usually, pain will have developed while the rash was still active, but on occasion later, after the rash has healed. In such cases, pale or light purple scars may be present as sequelae of the herpetic eruption.

Comments:
Despite its long-preferred name, *postherpetic neuralgia* is actually a neuropathy or neuronopathy: significant pathoanatomical changes have been shown in the nerve, ganglion and nerve root. In 4.1.2.2 *Trigeminal postherpetic neuralgia*, there is also evidence of the inflammation extending into the trigeminal brainstem complex.

Following acute herpes zoster, postherpetic neuralgia is more prevalent in the elderly.

The first division of the trigeminal nerve is most commonly affected in 4.1.2.2 *Trigeminal postherpetic neuralgia*, but the second and third divisions can be involved also.

Typically, the pain of postherpetic neuralgia is burning and itching – the latter sometimes very prominent and extremely bothersome. Also typically, patients with postherpetic neuralgia show a clear sensory deficit and brush-evoked mechanical allodynia in the territory involved. Many patients, however, show little sensory loss and instead demonstrate heightened responses to thermal and/or punctate stimuli.

4.1.2.3 Post-traumatic trigeminal neuropathic pain

Previously used terms:
Anaesthesia dolorosa; painful post-traumatic trigeminal neuropathy.

Description:
Unilateral or bilateral facial or oral pain following and caused by trauma to the trigeminal nerve(s), with other symptoms and/or clinical signs of trigeminal nerve dysfunction, and persisting or recurring for more than 3 months.

Diagnostic criteria:

A. Pain, in a neuroanatomically plausible area within the distribution(s) of one or both trigeminal nerve(s), persisting or recurring for >3 months and fulfilling criteria C and D
B. Both of the following:
   1. history of a mechanical, thermal, radiation or chemical injury to the peripheral trigeminal nerve(s)
   2. diagnostic test confirmation[1] of a lesion of the peripheral trigeminal nerve(s) explaining the pain[2]
C. Onset within 6 months after the injury[3]
D. Associated with somatosensory symptoms and/or signs[4] in the same neuroanatomically plausible distribution
E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Notes:

1. Tests that confirm a relevant lesion or disease affecting the trigeminal nerve may, for example, be surgical or radiological confirmation of nerve compression or lesion, nerve conduction study, laser-evoked potentials, blink reflex or skin biopsy confirmation of reduced nerve fibre terminals. Positive findings in these investigations may provide important diagnostic hints at the source of pain. However, all clinical and diagnostic aspects of the pain need to be considered.
2. The severity of nerve injuries may range from mild to severe. They include external trauma and iatrogenic injuries from dental treatments such as local anaesthetic injections, root canal therapies, extractions, oral surgery, dental implants, orthognathic surgery and other invasive procedures.
3. Specifically following radiation-induced postganglionic injury, neuropathic pain may appear after >3 months.

4. Somatosensory symptoms or signs may be negative (e.g. hypaesthesia and/or hypalgesia) and/or positive (e.g. hyperalgesia and/or allodynia). Note that positive somatosensory signs are not specific to neuropathy. Negative or positive somatosensory signs consistent with the distribution of the pain may be sufficient to indicate the presence of a lesion of the trigeminal nerve. The clinical examination is supplemented by laboratory tests such as quantitative sensory testing.

Comments:
The structure and content of the diagnostic criteria for 4.1.2.3 *Post-traumatic trigeminal neuropathic pain* deviate somewhat from those of 13.1.2.3 *Painful post-traumatic trigeminal neuropathy* in ICHD-3 in order to comply with IASP criteria.

Pain duration ranges widely from paroxysmal to constant, and may be mixed.

There may seem to be a partial overlap with 6.3.2 *Persistent idiopathic dentoalveolar pain with somatosensory changes*, but in this condition there may be no clear temporal relationship and the somatosensory changes may not be limited to a neuroanatomically confined area, in contrast to the criteria for 4.1.2.3 *Post-traumatic trigeminal neuropathic pain*.

Neuroablative procedures for trigeminal neuralgia, aimed at the trigeminal ganglion or nerve root, may result in neuropathic pain involving one or more trigeminal divisions and should be coded as 4.1.2.3 *Post-traumatic trigeminal neuropathic pain*. Such pain may, in some cases, coexist with 4.1.1 *Trigeminal neuralgia*; for example, when the latter recurs following remission.

4.1.2.3 *Post-traumatic trigeminal neuropathic pain* rarely, if ever, crosses the midline but, over time, it may in some cases become more diffusely distributed.

4.1.2.3.1 Probable post-traumatic trigeminal neuropathic pain

Diagnostic criterion:
A. Pain fulfilling all but criterion B2 for 4.1.2.3 *Post-traumatic trigeminal neuropathic pain*.

4.1.2.4 Trigeminal neuropathic pain attributed to other disorder

Description:
Unilateral or bilateral facial or oral pain in the distribution(s) of one or more branches of the trigeminal nerve, caused by a disorder other than those described above, persisting or recurring for more than 3 months and accompanied by other symptoms and/or clinical signs of nerve dysfunction.

Diagnostic criteria:

A. Pain, in a neuroanatomically plausible area within the distribution(s) of one or both trigeminal nerve(s), persisting or recurring for >3 months and fulfilling criteria C and D
B. A disorder other than those identified in 4.1.2.1 to 4.1.2.3, but known to be capable of causing, and explaining, the trigeminal neuropathic pain, has been diagnosed
C. Pain has developed after onset of the presumed causative disorder, or has led to its discovery
D. Pain is associated with somatosensory symptoms and/or signs[1] in the same neuroanatomically plausible distribution
E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Note:

1. Somatosensory symptoms or signs may be negative (e.g. hypaesthesia and/or hypalgesia) and/or positive (e.g. hyperalgesia and/or allodynia).

Comments:
Trigeminal neuropathic pain may develop secondary to multiple sclerosis, space-occupying lesion or systemic disease, with only the clinical characteristics (quality of spontaneous pain, evoked pain and presence of sensory deficits) distinguishing between 4.1.1.2 *Secondary trigeminal neuralgia* and 4.1.2 *Other trigeminal neuropathic pain*.

4.1.2 *Other trigeminal neuropathic pain* caused by a connective tissue disease or hereditary disorder is usually bilateral, but may begin asymmetrically and occasionally present with paroxysmal pain superimposed on the background pain. Patients will eventually develop bilateral sensory deficits and continuous pain, which clarify the diagnosis. MRI is normal, but trigeminal reflexes are invariably delayed or absent.

4.1.2.4.1 *Probable trigeminal neuropathic pain attributed to other disorder*

Diagnostic criterion:
A. Pain fulfilling all but criterion C for 4.1.2.4 *Trigeminal neuropathic pain attributed to other disorder*.

4.1.2.5 Idiopathic trigeminal neuropathic pain

Description:
Unilateral or bilateral facial pain in the distribution(s) of one or more branches of the trigeminal nerve,

indicative of neural damage and persisting or recurring for more than 3 months, but of unknown aetiology.

Diagnostic criteria:

A. Pain, in a neuroanatomically plausible area within the distribution(s) of one or both trigeminal nerve(s), persisting or recurring for >3 months and fulfilling criterion C
B. Both of the following:
   1. a lesion of the peripheral trigeminal nerve(s), explaining the pain, has been diagnosed
   2. no history of trauma or disorder with possible peripheral trigeminal nerve involvement
C. Pain is associated with somatosensory symptoms and/or signs[1] in the same neuroanatomically plausible distribution
D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Note:

1. Somatosensory symptoms or signs may be negative (e.g. hypaesthesia and/or hypalgesia) and/or positive (e.g. hyperalgesia and/or allodynia).

4.2 Pain attributed to lesion or disease of the glossopharyngeal nerve

4.2.1 Glossopharyngeal neuralgia

Previously used term:
Vagoglossopharyngeal neuralgia.

Description:
A disorder characterized by unilateral brief stabbing pain, abrupt in onset and termination, in the distributions not only of the glossopharyngeal nerve but also of the auricular and pharyngeal branches of the vagus nerve. Pain is experienced in the ear, base of the tongue, tonsillar fossa and/or beneath the angle of the jaw. It is commonly provoked by swallowing, talking or coughing and may remit and relapse in the fashion of 4.1.1 *Trigeminal neuralgia*.

Diagnostic criteria:

A. Recurring paroxysmal attacks of unilateral pain in the distribution of the glossopharyngeal nerve[1] and fulfilling criterion B
B. Pain has all the following characteristics:
   1. lasting from a few seconds to 2 minutes
   2. severe intensity
   3. electric shock-like, shooting, stabbing or sharp in quality
   4. precipitated by swallowing, coughing, talking or yawning
C. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Note:

1. Within the posterior part of the tongue, tonsillar fossa, pharynx or angle of the lower jaw and/or in the ear.

Comments:
4.2.1 *Glossopharyngeal neuralgia* can occur together with 4.1.1 *Trigeminal neuralgia*

The superior laryngeal nerve is a branch of the vagus. Neuralgia of the superior laryngeal nerve presents similarly to 4.2.1 *Glossopharyngeal neuralgia* in its location and clinically can be difficult to distinguish from it.

Imaging may show neurovascular compression of the glossopharyngeal nerve.

Prior to development of 4.2.1 *Glossopharyngeal neuralgia*, unpleasant sensations may be felt in affected areas for weeks to several months.

The pain in 4.2.1 *Glossopharyngeal neuralgia* may radiate to involve the eye, nose, chin or shoulder. It can be severe enough for patients to lose weight. In rare cases, attacks of pain are associated with vagal symptoms such as cough, hoarseness or syncope and/or bradycardia. Some authors propose distinguishing between pharyngeal, otalgic and vagal subforms of neuralgia, and have suggested using the term *vagoglossopharyngeal neuralgia* when pain is accompanied by asystole, convulsions and syncope.

Clinical examination usually fails to show sensory changes in the nerve distribution but, if mild sensory deficits are encountered, they do not invalidate the diagnosis. Major changes or a reduced/missing gag reflex should prompt aetiological investigations.

4.2.1 *Glossopharyngeal neuralgia* is usually responsive, at least initially, to pharmacotherapy (especially carbamazepine or oxcarbazepine). It has been suggested that application of local anaesthetic to the tonsil and pharyngeal wall can prevent attacks for a few hours.

4.2.1.1 Classical glossopharyngeal neuralgia

Diagnostic criteria:

A. Recurrent paroxysms of unilateral pain fulfilling criteria for 4.2.1 *Glossopharyngeal neuralgia*

B. Demonstration on magnetic resonance imaging (MRI) or during surgery of neurovascular compression of the glossopharyngeal nerve root.

4.2.1.2 Secondary glossopharyngeal neuralgia

Description:
Glossopharyngeal neuralgia caused by an underlying disease.

Diagnostic criteria:

A. Recurrent paroxysms of unilateral pain fulfilling criteria for 4.2.1 *Glossopharyngeal neuralgia*
B. An underlying disease has been demonstrated that is known to be able to cause, and explain, the neuralgia.[1]

Note:

1. There are single reports of 4.2.1.2 *Secondary glossopharyngeal neuralgia* caused by neck trauma, multiple sclerosis, tonsillar or regional tumours, cerebello-pontine angle tumours and Arnold–Chiari malformation.

4.2.1.3 Idiopathic glossopharyngeal neuralgia

Diagnostic criteria:

A. Recurrent paroxysms of unilateral pain fulfilling criteria for 4.2.1 *Glossopharyngeal neuralgia*
B. Investigations have found neither neurovascular compression nor an underlying disease known to be able to cause 4.2.1.2 *Secondary glossopharyngeal neuralgia*
C. Not better accounted for by another ICOP or ICHD-3 diagnosis.

4.2.2 Glossopharyngeal neuropathic pain

Description:
Pain within the distribution of the glossopharyngeal nerve (posterior part of the tongue, tonsillar fossa, pharynx or beneath the angle of the lower jaw). In addition, pain is commonly perceived in the ipsilateral ear. The primary pain is usually continuous or near-continuous, and commonly described as burning or squeezing, or likened to pins and needles. Brief paroxysms may be superimposed but they are not the predominant pain type. This combination distinguishes 4.2.2 *Glossopharyngeal neuropathic pain* from 4.2.1 *Glossopharyngeal neuralgia*. Sensory deficits may be present in the ipsilateral posterior part of the tongue and tonsillar fossa, and the gag reflex may be weak or missing.

4.2.2.1 Glossopharyngeal neuropathic pain attributed to a known cause

Description:
Unilateral continuous or near-continuous pain, with or without superimposed brief paroxysms, in the distribution of the glossopharyngeal nerve and caused by another identified disorder.

Diagnostic criteria:

A. Unilateral continuous or near-continuous pain[1] in the distribution of the glossopharyngeal nerve and fulfilling criterion C
B. A disorder known to be able to cause glossopharyngeal neuropathic pain has been diagnosed[2]
C. Evidence of causation demonstrated by both of the following:
   1. pain is ipsilateral to the glossopharyngeal nerve affected by the disorder
   2. pain has developed after onset of the disorder, or led to its discovery
D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Notes:

1. Brief paroxysms may be superimposed but they are not the predominant pain type.
2. Tumours of the cerebellopontine angle and iatrogenic injury during interventional procedures have been reported as causes of 4.2.2.1 *Glossopharyngeal neuropathic pain attributed to a known cause*.

4.2.2.2 Idiopathic glossopharyngeal neuropathic pain

Description:
Unilateral continuous or near-continuous pain, with or without superimposed brief paroxysms, in the distribution(s) of the glossopharyngeal nerve and of unknown aetiology.

Diagnostic criteria:

A. Unilateral continuous or near-continuous pain[1] in the distribution of the glossopharyngeal nerve
B. No cause has been identified
C. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Note:

1. Brief paroxysms may be superimposed but are not the predominant pain type.

References

4.1 Pain attributed to lesion or disease of the trigeminal nerve

Benoliel R, Svensson P, Evers S, et al.; IASP Taskforce for the Classification of Chronic Pain. The IASP classification of chronic pain for ICD-11: chronic secondary headache or orofacial pain. *Pain* 2019; 160: 60–68.

Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. *Cephalalgia* 2018; 38: 1–211.

Scholz J, Finnerup NB, Attal N, et al.; Classification Committee of the Neuropathic Pain Special Interest Group (NeuPSIG). The IASP classification of chronic pain for ICD-11: chronic neuropathic pain. *Pain* 2019; 160: 53–59.

4.1.1 Trigeminal neuralgia

Benoliel R, Eliav E and Sharav Y. Self-reports of pain-related awakenings in persistent orofacial pain patients. *J Orofac Pain* 2009; 23: 330–338.

Cruccu G, Finnerup NB, Jensen TS, et al. Trigeminal neuralgia: new classification and diagnostic grading for clinical practice and research. *Neurology* 2016; 87: 220–228.

Drangsholt M and Truelove E. Trigeminal neuralgia mistaken as temporomandibular disorder. *J Evid Base Dent Pract* 2001; 1: 41–50.

Fromm GH, Graff-Radford SB, Terrence CF, et al. Pre-trigeminal neuralgia. *Neurology* 1990; 40: 1493–1495.

Haviv Y, Khan J, Zini A, et al. Trigeminal neuralgia (part I). Revisiting the clinical phenotype. *Cephalalgia* 2016; 36: 730–746.

Koopman JSHA, Dieleman JP, Huygen FJ, et al. Incidence of facial pain in the general population. *Pain* 2009; 147: 122–127.

Mueller D, Obermann M, Yoon MS, et al. Prevalence of trigeminal neuralgia and persistent idiopathic facial pain: a population-based study. *Cephalalgia* 2011; 31: 1542–1548.

Obermann M, Yoon MS, Ese D, et al. Impaired trigeminal nociceptive processing in patients with trigeminal neuralgia. *Neurology* 2007; 69: 835–841.

Pareja JA, Cuadrado ML, Caminero AB, et al. Duration of attacks of first division trigeminal neuralgia. *Cephalalgia* 2005; 25: 305–308.

Rasmussen P. Facial pain. II. A prospective survey of 1052 patients with a view of: character of the attacks, onset, course, and character of pain. *Acta Neurochir (Wien)* 1990; 107: 121–128.

Rasmussen P. Facial pain. III. A prospective study of the localization of facial pain in 1052 patients. *Acta Neurochir (Wien)* 1991; 108: 53–63.

Rasmussen P. Facial pain. IV. A prospective study of 1052 patients with a view of: precipitating factors, associated symptoms, objective psychiatric and neurological symptoms. *Acta Neurochir (Wien)* 1991; 108: 100–109.

4.1.1.1 Classical trigeminal neuralgia

Antonini G, Di Pasquale A, Cruccu G, et al. Magnetic resonance imaging contribution for diagnosing symptomatic neurovascular contact in classical trigeminal neuralgia: a blinded case-control study and meta-analysis. *Pain* 2014; 155: 1464–1471.

Bowsher D, Miles JB, Haggett CE, et al. Trigeminal neuralgia: a quantitative sensory perception threshold study in patients who had not undergone previous invasive procedures. *J Neurosurg* 1997; 86: 190–192.

Leal PR, Barbier C, Hermier M, et al. Atrophic changes in the trigeminal nerves of patients with trigeminal neuralgia due to neurovascular compression and their association with the severity of compression and clinical outcomes. *J Neurosurg* 2014; 120: 1484–1495.

Maarbjerg S, Wolfram F, Gozalov A, et al. Significance of neurovascular contact in classical trigeminal neuralgia. *Brain* 2015; 48: 311–319.

4.1.1.2.1 Trigeminal neuralgia attributed to multiple sclerosis

Cruccu G, Biasiotta A, Di RS, et al. Trigeminal neuralgia and pain related to multiple sclerosis. *Pain* 2009; 143: 186–191.

O'Connor AB, Schwid SR, Herrmann DN, et al. Pain associated with multiple sclerosis: systematic review and proposed classification. *Pain* 2008; 137: 96–111.

Truini A, Prosperini L, Calistri V, et al. A dual concurrent mechanism explains trigeminal neuralgia in patients with multiple sclerosis. *Neurology* 2016; 86: 2094–2099.

4.1.1.2.2 Trigeminal neuralgia attributed to space-occupying lesion

Cheng TM, Cascino TL and Onofrio BM. Comprehensive study of diagnosis and treatment of trigeminal neuralgia secondary to tumors. *Neurology* 1993; 43: 2298–2302.

Wei Y, Zhao W, Pu C, et al. Clinical features and long-term surgical outcomes in 39 patients with tumor-related trigeminal neuralgia compared with 360 patients with idiopathic trigeminal neuralgia. *Br J Neurosurg* 2017; 31: 101–106.

4.1.1.2.3 Trigeminal neuralgia attributed to other cause

Coffey RJ and Fromm GH. Familial trigeminal neuralgia and Charcot-Marie-Tooth neuropathy. Report of two families and review. *Surg Neurol* 1991; 35: 49–53.

De Paula Lucas C and Zabramski JM. Dural arteriovenous fistula of the transverse-sigmoid sinus causing trigeminal neuralgia. *Acta Neurochir* 2007; 149: 1249–1253.

Tanaka BS, Zhao P, Dib-Hajj FB, et al. A gain-of-function mutation in Nav1.6 in a case of trigeminal neuralgia. *Mol Med* 2016; 22: 338–348.

Yip V, Michael BD, Nahser HC, et al. Arteriovenous malformation: a rare cause of trigeminal neuralgia identified by magnetic resonance imaging with constructive interference in steady state sequences. *QJM* 2012; 105: 895–898.

4.1.1.3 Idiopathic trigeminal neuralgia

Lee A, McCartney S, Burbidge C, et al. Trigeminal neuralgia occurs and recurs in the absence of neurovascular compression. *J Neurosurg* 2014; 120: 1048–1054.

4.1.2.1 Trigeminal neuropathic pain attributed to herpes zoster

Dworkin RH and Portenoy RK. Pain and its persistence in herpes zoster. *Pain* 1996; 67: 241–252.

Haanpää M, Dastidar P, Weinberg A, et al. Characteristics of cerebrospinal fluid and magnetic resonance imaging findings in patients with acute herpes zoster. *Neurology* 1998; 51: 1405–1411.

Liesegang TJ. Herpes zoster ophthalmicus. Natural history, risk factors, clinical presentation, and morbidity. *Ophthalmology* 2008; 115(2 suppl): S3–S12.

4.1.2.2 Trigeminal postherpetic neuralgia

Alvarez FK, de Siqueira SR, Okada M, et al. Evaluation of the sensation in patients with trigeminal post-herpetic neuralgia. *J Oral Pathol Med* 2007; 36: 347–350.

Truini A, Galeotti F, Haanpaa M, et al. Pathophysiology of pain in postherpetic neuralgia: a clinical and neurophysiological study. *Pain* 2008; 140: 405–410.

Truini A, Haanpaa M, Provitera V, et al. Differential myelinated and unmyelinated sensory and autonomic skin nerve fiber involvement in patients with ophthalmic postherpetic neuralgia. *Front Neuroanat* 2015; 9: 105.

4.1.2.3. Post-traumatic trigeminal neuropathic pain

Benoliel R, Birenboim R, Regev E, et al. Neurosensory changes in the infraorbital nerve following zygomatic fractures. *Oral Surg* 2005; 99: 657–665.

Benoliel R, Zadik Y, Eliav E, et al. Peripheral painful traumatic trigeminal neuropathy: Clinical features in 91 cases and proposal of novel diagnostic criteria. *J Orofac Pain* 2012; 26: 49–58.

Finnerup NB, Haroutounian S, Kamerman P, et al. Neuropathic pain: an updated grading system for research and clinical practice. *Pain* 2016; 157: 1599–1606.

Jääskeläinen SK, Teerijoki-Oksa T and Forssell H. Neurophysiologic and quantitative sensory testing in the diagnosis of trigeminal neuropathy and neuropathic pain. *Pain* 2005; 117: 349–357.

Polycarpou N, Ng YL, Canavan D, et al. Prevalence of persistent pain after endodontic treatment and factors affecting its occurrence in cases with complete radiographic healing. *Int Endod J* 2005; 38: 169–178.

Queral-Godoy E, Figueiredo R, Valmaseda-Castellon E, et al. Frequency and evolution of lingual nerve lesions following lower third molar extraction. *J Oral Maxillofac Surg* 2006; 64: 402–407.

Renton T and Yilmaz Z. Profiling of patients presenting with posttraumatic neuropathy of the trigeminal nerve. *J Orofac Pain* 2011; 25: 333–344.

4.1.2.4 Trigeminal neuropathic pain attributed to other disorder

Cruccu G, Penisi EM, Antonini G, et al. Trigeminal isolated sensory neuropathy (TISN) and FOSMN syndrome: despite a dissimilar disease course do they share common pathophysiological mechanisms? *BMC Neurol* 2014; 14: 248.

Klasser GD, Balasubramaniam R, Epstein J. Topical review—connective tissue diseases: orofacial manifestations including pain. *J Orofac Pain* 2007; 21: 171–184.

4.2.1 Glossopharyngeal neuralgia

Blumenfeld A, Nikolskaya G. Glossopharyngeal neuralgia. *Curr Pain and Headache Rep* 2013; 17: 343.

Huynh-Le P, Matsishima T, Hisada K, et al. Glossopharyngeal neuralgia due to an epidermoid

tumour in the cerebellopontine angle. *J Clin Neurosci* 2004; 11: 758–760.
Kandan SR, Khan S, Jeyaretna DS, et al. Neuralgia of the glossopharyngeal and vagal nerves: long-term outcome following surgical treatment and literature review. *Br J Neurosurg* 2010; 24: 441–446.
Minagar A and Sheremata WA. Glossopharyngeal neuralgia and MS. *Neurology* 2000; 54: 1368–1370.
Patel A, Kassam A, Horowitz M, et al. Microvascular decompression in the management of glossopharyngeal neuralgia: analysis of 217 cases. *Neurosurgery* 2002; 50: 705–710.
Peet MM. Glossopharyngeal neuralgia. *Ann Surg* 1935; 101: 256–258.
Saman Y, Whitehead D, Gleeson M. Jugular foramen schwannoma presenting with glossopharyngeal neuralgia syncope syndrome. *J Laryngol Otol* 2010; 124: 1305–1308.
Tanrikulu L, Hastreiter P, Dorfler A, et al. Classification of neurovascular compression in glossopharyngeal neuralgia: three-dimensional visualization of the glossopharyngeal nerve. *Surg Neurol Int* 2015; 6: 189.

4.2.2 Glossopharyngeal neuropathic pain

Bakar B. The jugular foramen schwannomas: review of the large surgical series. *J Korean Neurosurg Soc* 2008; 44: 285–294.
Kalladka M, Nasri-Heir C, Eliav E, et al. Continuous neuropathic pain secondary to endoscopic procedures: report of two cases and review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2016; 122: e55–e59.
Shin HY, Park HJ, Choi YC, et al. Clinical and electromyographic features of radiation-induced lower cranial neuropathy. *Clin Neurophysiol* 2013; 124: 598–602.

5. Orofacial pains resembling presentations of primary headaches

General comments:

In clinical practice we often see three types of patients who seem to typify the crossroads between headache and orofacial pain (OFP).

Type 1: Headache patients who report additional facial pain during, and usually ipsilateral to, the headache attacks.
Type 2: Headache patients whose headache attacks have stopped and been replaced by facial pain attacks of the same quality, length and intensity, including occurrence of the associated symptoms of the former headache.
Type 3: Headache naïve patients who develop de novo OFP attacks that resemble one of the primary headache types in pain character, duration and intensity, with or without the associated symptoms of these headache types.

This section in ICOP is for patients in the third category, who have pain exclusively in the facial region resembling primary headaches but with *no* head pain. All others should be coded according to ICHD-3.

5.1 Orofacial migraine

Description:
Episodic or chronic pain exclusively in the orofacial region, without head pain, with the characteristics and associated features of 1. *Migraine* described in ICHD-3.

Classified elsewhere:
Orofacial pain otherwise meeting the criteria for any of the subtypes or subforms below, but accompanied by head pain, should be classified according to ICHD-3 under 1. *Migraine*.

5.1.1 Episodic orofacial migraine

Description:
Recurrent orofacial pain (OFP) attacks, without head pain, lasting 4–72 hours. Typical characteristics of the pain are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity and association with nausea and/or photophobia and phonophobia.

Diagnostic criteria:

A. At least five attacks fulfilling criteria B–D
B. Facial and/or oral pain, without head pain, lasting 4–72 hours (untreated or unsuccessfully treated)
C. Pain has at least two of the following four characteristics:
   1. unilateral location
   2. pulsating quality
   3. moderate or severe intensity
   4. aggravation by, or causing avoidance of, routine physical activity (e.g. walking or climbing stairs)
D. Pain is accompanied by one or both of the following:
   1. nausea and/or vomiting
   2. photophobia and phonophobia
E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comments:
5.1.1 *Episodic orofacial migraine*, as defined (with no head pain), seems to be very rare. Bilateral orofacial migraine has not so far been described.

Orofacial migraine with aura has not, to our knowledge, been described, and is excluded from ICOP until better evidence of it accumulates.

A group of patients with attacks of intraoral pain of varying duration, with atypical migraine-like features, have been described. These may be unrelated to migraine, and are described below under 5.4 *Neurovascular orofacial pain*.

5.1.2 Chronic orofacial migraine

Description:

Facial and/or oral pain occurring on 15 or more days per month for more than 3 months, which has the features of migraine on at least 8 days per month.

Diagnostic criteria:

A. Facial and/or oral pain, without head pain, on $\geq$15 days/month for >3 months and fulfilling criteria B and C below
B. Occurring in a patient who has had at least five attacks fulfilling criteria B–D for 5.1 *Episodic orofacial migraine*
C. On $\geq$8 days/month for >3 months, fulfilling either of the following:
   1. criteria C and D for 5.1.1 *Episodic orofacial migraine*
   2. believed by the patient to be orofacial migraine at onset and relieved by a triptan or ergot derivative
D. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Comment:
Characterization of frequently recurring OFP generally requires a pain diary to record information on

pain and associated symptoms day-by-day for at least 1 month.

5.2 Tension-type orofacial pain

Description:
Episodic or chronic pain exclusively in the orofacial region, without head pain, with the characteristics and associated features of 2. *Tension-type headache* described in ICHD-3.

Comments:
There are many similarities in symptoms, signs, epidemiology and response to treatment between disorders described under 2. *Tension-type headache* in ICHD-3 and those described under 2. *Myofascial orofacial pain* in ICOP. At present there is insufficient evidence to establish any type of relationship between them.

There may exist a facial pain unrelated to temporomandibular disorder, described as 'facial muscle tension', occurring only during rest and resolving with voluntary muscle activity such as mastication. At present there is insufficient evidence that this is a separate entity.

5.3 Trigeminal autonomic orofacial pain

Description:
Attacks of pain, exclusively in the orofacial region, without head pain, with the characteristics and associated features of a disorder described under 3. *Trigeminal autonomic cephalalgias* in ICHD-3.

Classified elsewhere:
Orofacial pain otherwise meeting the criteria for any of the subtypes or subforms below, but accompanied by head pain, should be classified according to ICHD-3 under 3. *Trigeminal autonomic cephalalgias*.

5.3.1 Orofacial cluster attacks

Description:
Attacks of severe, strictly unilateral facial and/or oral pain, without head pain, lasting 15–180 minutes and occurring from once every other day to eight times a day. The pain is associated with ipsilateral conjunctival injection, lacrimation, nasal congestion, rhinorrhoea, forehead and facial sweating, miosis, ptosis and/or eyelid oedema, and/or with restlessness or agitation.

Diagnostic criteria:

A. At least five attacks fulfilling criteria B–D
B. Severe or very severe unilateral facial and/or oral pain lasting 15–180 minutes (when untreated)[1]
C. Either or both of the following:
    1. at least one of the following symptoms or signs, ipsilateral to the pain:
        a) conjunctival injection and/or lacrimation
        b) nasal congestion and/or rhinorrhoea
        c) eyelid oedema
        d) forehead and facial sweating
        e) miosis and/or ptosis
    2. a sense of restlessness or agitation
D. Occurring with a frequency between one every other day and eight per day[2]
E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Notes:

1. During part, but less than half, of the active time-course of 5.3.1 *Orofacial cluster attacks*, attacks may be less severe and/or of shorter or longer duration.
2. During part, but less than half, of the active time-course of 5.3.1 *Orofacial cluster attacks*, attacks may be less frequent.

Comment:
Autonomic symptoms in 5.3.1 *Orofacial cluster attacks* may be less prominent than or different from those occurring as features of 3.1 *Cluster headache* described in ICHD-3. Patients with facial and/or oral pain accompanied by cluster-like autonomic features have been described, but there is insufficient evidence that these form a separate group. Further research is needed to provide evidence answering these questions.

5.3.1.1 Episodic orofacial cluster attacks

Description:
Orofacial cluster attacks occurring in periods lasting from 7 days to 1 year, separated by pain-free periods lasting at least 3 months.

Diagnostic criteria:

A. Attacks fulfilling criteria for 5.3.1 *Orofacial cluster attacks* and occurring in bouts (cluster periods)
B. At least two cluster periods lasting from 7 days to 1 year (when untreated) and separated by pain-free remission periods of $\geq$3 months.

5.3.1.2 Chronic orofacial cluster attacks

Description:
Orofacial cluster attacks occurring for more than 1 year without remission, or with remission periods lasting less than 3 months.

Diagnostic criteria:

A. Attacks fulfilling criteria for 5.3.1 *Orofacial cluster attacks*, and criterion B below
B. Occurring without a remission period, or with remissions lasting <1 month, for at least 1 year.

Comment:
5.3.1.2 *Chronic orofacial cluster attacks* may evolve from 5.3.1.1 *Episodic orofacial cluster attacks* or arise de novo. In some patients, change occurs from 5.3.1.2 *Chronic orofacial cluster attacks* to 5.3.1.1 *Episodic orofacial cluster attacks*.

5.3.2 Paroxysmal hemifacial pain

Description:
Attacks of severe, strictly hemifacial pain, without head pain, lasting 2–30 minutes and occurring several or many times a day. The attacks may be associated with ipsilateral conjunctival injection, lacrimation, nasal congestion, rhinorrhoea, forehead and facial sweating, miosis, ptosis and/or eyelid oedema.

Diagnostic criteria:

A. At least 20 attacks fulfilling criteria B–D
B. Severe unilateral facial and/or oral pain, without head pain, lasting 2–30 minutes
C. Either or both of the following:
    1. at least one of the following symptoms or signs, ipsilateral to the pain:
        a) conjunctival injection and/or lacrimation
        b) nasal congestion and/or rhinorrhoea
        c) eyelid oedema
        d) forehead and facial sweating
        e) miosis and/or ptosis
    2. prevented absolutely by therapeutic doses of indomethacin
D. Occurring with a frequency of >5 per day[1]
E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Note:

1. During part, but less than half, of the active time-course of 5.3.2 *Paroxysmal hemifacial pain*, attacks may be less frequent.

Comments:
There are reports of paroxysmal hemifacial pain without prominent autonomic signs. Some evidence suggests that the number and quality of autonomic symptoms differ when pain is in the distributions of the second and/or third trigeminal divisions (as in 5.3.2 *Paroxysmal hemifacial pain*) rather than the first (as in 3.2 *Paroxysmal hemicrania*, described in ICHD-3).

Additionally, absoluteness of response to indomethacin in 5.3.2 *Paroxysmal hemifacial pain* is not yet established.

Further evidence is needed to determine whether this is a separate entity.

5.3.2.1 Episodic paroxysmal hemifacial pain

Description:
Attacks of paroxysmal hemifacial pain occurring in periods lasting from 7 days to 1 year, separated by pain-free periods lasting at least 3 months.

Diagnostic criteria:

A. Attacks fulfilling criteria for 5.3.2 *Paroxysmal hemifacial pain* and occurring in bouts
B. At least two bouts lasting from 7 days to 1 year (when untreated) and separated by pain-free remission periods of ≥3 months.

5.3.2.2 Chronic paroxysmal hemifacial pain

Description:
Attacks of paroxysmal hemifacial pain occurring for more than 1 year without remission, or with remission periods lasting less than 3 months.

Diagnostic criteria:

A. Attacks fulfilling criteria for 5.3.2 *Paroxysmal hemifacial pain*, and criterion B below
B. Occurring without a remission period, or with remissions lasting <3 months, for at least 1 year.

5.3.3 Short-lasting unilateral neuralgiform facial pain attacks with cranial autonomic symptoms (SUNFA)

Description:
Attacks of moderate or severe, strictly unilateral oral and/or facial pain, without head pain, lasting seconds to minutes, occurring at least once a day and usually associated with prominent lacrimation and redness of the ipsilateral eye and/or other local autonomic symptoms and/or signs.

Diagnostic criteria:

A. At least 20 attacks fulfilling criteria B–D

B. Moderate or severe unilateral facial and/or oral pain, without head pain, lasting for 1–600 seconds and occurring as single stabs, series of stabs or in a saw-tooth pattern
C. At least one of the following cranial autonomic symptoms or signs, ipsilateral to the pain:
   1. conjunctival injection and/or lacrimation
   2. nasal congestion and/or rhinorrhoea
   3. eyelid oedema
   4. forehead and facial sweating
   5. miosis and/or ptosis
D. Occurring with a frequency of at least one a day[1]
E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Note:

1. During part, but less than half, of the active time-course of 5.3.3 *Short-lasting unilateral neuralgiform facial pain attacks with cranial autonomic symptoms*, attacks may be less frequent.

Comment:
The occurrence and distribution of autonomic symptoms and signs in 5.3.3 *Short-lasting unilateral neuralgiform facial pain attacks with cranial autonomic symptoms* are unclear, and need to be studied. In 3.3.1 *Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing* (SUNCT), described in ICHD-3, pain and accompanying autonomic signs may occur throughout the trigeminal distribution; the location and quality of autonomic features may be related to pain location.

5.3.3.1 Episodic SUNFA

Description:
Attacks of SUNFA occurring in periods lasting from 7 days to 1 year, separated by pain-free periods lasting at least 3 months.

Diagnostic criteria:

A. Attacks fulfilling criteria for 5.3.3 *Short-lasting unilateral neuralgiform facial pain attacks with cranial autonomic symptoms* and occurring in bouts
B. At least two bouts lasting from 7 days to 1 year and separated by pain-free remission periods of ≥3 months.

5.3.3.2 Chronic SUNFA

Description:
Attacks of SUNFA occurring for more than 1 year without remission, or with remission periods lasting less than 3 months.

Diagnostic criteria:

A. Attacks fulfilling criteria for 5.3.3 *Short-lasting unilateral neuralgiform facial pain attacks with cranial autonomic symptoms*, and criterion B below
B. Occurring without a remission period, or with remissions lasting <3 months, for at least 1 year.

5.3.4 Hemifacial continuous pain with autonomic symptoms

While no isolated facial equivalent of 3.4 *Hemicrania continua*, described in ICHD-3, has so far been clearly established, extension of pain into the face in hemicrania continua has been described. Thus, pain referral and/or radiation to oral and/or facial structures may cause diagnostic difficulties.

Hemicrania continua has been described with a paucity of autonomic symptoms and may have a representative within the group of chronic idiopathic facial pain syndromes.

5.4 Neurovascular orofacial pain

Description:
Attacks of variable duration of moderate or severe intraoral pain, without head pain, often accompanied by toothache-like symptoms, with mild autonomic and/or migrainous symptoms. Two subforms are represented by patients with relatively short attacks (1–4 hours) and those with longer attacks (>4 hours).

Diagnostic criteria:

A. At least five attacks of unilateral intraoral pain[1] of variable duration, without head pain, fulfilling criteria B–D
B. Pain has both of the following characteristics:
   1. moderate or severe intensity
   2. either or both of the following qualities:
      a) toothache-like
      b) pulsating
C. Pain is accompanied by at least one of the following:
   1. ipsilateral lacrimation and/or conjunctival injection

2. ipsilateral rhinorrhoea and/or nasal congestion
3. ipsilateral cheek swelling
4. photophobia and/or phonophobia
5. nausea and/or vomiting

D. Pain is unexplained by any local cause, and clinical and radiographic examinations are normal
E. Not better accounted for by another ICOP or ICHD-3 diagnosis.

Note:

1. Although essentially an intraoral pain, there may be referral and/or radiation to adjacent sites, particularly when pain is severe. This phenomenon needs to be carefully followed and documented.

Comments:
Various studies have suggested a disorder is recognizable that, while similar in phenotype to 5.1 *Orofacial migraine* or 5.3 *Trigeminal autonomic orofacial pain*, nonetheless appears to be a specific and distinct entity. Although existing in the literature since 1997, this entity needs thorough and prospective examination.

There are reports of abnormal sensitivity to cold, both interictally and during attacks. This finding needs to be investigated thoroughly, since it would be a useful test, and might link this entity to migraine, in which mechanical allodynia occurs during attacks.

To record all the characteristics required for diagnosis, the use of pain diaries is essential.

5.4.1 Short-lasting neurovascular orofacial pain

Diagnostic criteria:

A. Attacks of intraoral pain fulfilling criteria for 5.4 *Neurovascular orofacial pain*, and criterion B below
B. Lasting 1–4 hours (untreated, or unsuccessfully treated).

5.4.2 Long-lasting neurovascular orofacial pain

Diagnostic criteria:

A. Attacks of intraoral pain fulfilling criteria for 5.4 *Neurovascular orofacial pain*, and criterion B below
B. Lasting >4 hours.

References

5.1 Orofacial migraine

Alvarez M, Montojo T, de la Casa B, et al. Unilateral nasal pain with migraine features. *Cephalalgia* 2013; 33: 1055–1058.

Daudia AT and Jones NS. Facial migraine in a rhinological setting. *Clin Otolaryngol Allied Sci* 2002; 27: 521–525.

Debruyne F and Herroelen L. Migraine presenting as chronic facial pain. *Acta Neurol Belg* 2009; 109: 235–237.

Dodick DW. Migraine with isolated facial pain: a diagnostic challenge. *Cephalalgia* 2007; 27: 1199–1200.

Eross E, Dodick D and Eross M. The Sinus, Allergy and Migraine Study (SAMS). *Headache* 2007; 47: 213–224.

Gaul C, Sandor PS, Galli U, et al. Orofacial migraine. *Cephalalgia* 2007; 27: 950–952.

Hussain A, Stiles MA and Oshinsky ML. Pain remapping in migraine: a novel characteristic following trigeminal nerve injury. *Headache* 2010; 50: 669–671.

Lipton RB, Diamond S, Reed M, et al. Migraine diagnosis and treatment: results from the American Migraine Study II. *Headache* 2001; 41: 638–645.

Obermann M, Mueller D, Yoon MS, et al. Migraine with isolated facial pain: a diagnostic challenge. *Cephalalgia* 2007; 27: 1278–1282.

Penarrocha M, Bandres A, Penarrocha M, et al. Lower-half facial migraine: a report of 11 cases. *J Oral Maxillofac Surg* 2004; 62: 1453–1456.

Schreiber CP, Hutchinson S, Webster CJ, et al. Prevalence of migraine in patients with a history of self-reported or physician-diagnosed 'sinus' headache. *Arch Intern Med* 2004; 164: 1769–1772.

Yoon MS, Mueller D, Hansen N, et al. Prevalence of facial pain in migraine: a population-based study. *Cephalalgia* 2010; 30: 92–96.

5.3.1 Orofacial cluster attacks

Bahra A and Goadsby PJ. Diagnostic delays and mismanagement in cluster headache. *Acta Neurol Scand* 2004; 109: 175–179.

Bahra A, May A and Goadsby PJ. Cluster headache: a prospective clinical study with diagnostic implications. *Neurology* 2002; 58: 354–361.

Benoliel R, Birman N, Eliav E, et al. The International Classification of Headache Disorders: accurate diagnosis of orofacial pain? *Cephalalgia* 2008; 28: 752–762.

Benoliel R, Elishoov H and Sharav Y. Orofacial pain with vascular-type features. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997; 84: 506–512.
Bittar G and Graff-Radford SB. A retrospective study of patients with cluster headaches. *Oral Surg Oral Med Oral Pathol* 1992; 73: 519–525.
Cademartiri C, Torelli P, Cologno D, et al. Upper and lower cluster headache: clinical and pathogenetic observations in 608 patients. *Headache* 2002; 42: 630–637.
Gaul C, Gantenbein AR, Buettner UW, et al. Orofacial cluster headache. *Cephalalgia* 2008; 28: 903–905.
Gross SG. Dental presentations of cluster headaches. *Curr Pain Headache Rep* 2006; 10: 126–129.
Larner AJ. Unnecessary extractions. *Br Dent J* 2007; 203: 442.
Sanchez Del Rio M, Leira R, Pozo-Rosich P, et al. Errors in recognition and management are still frequent in patients with cluster headache. *Eur Neurol* 2014; 72: 209–212.
Van Alboom E, Louis P, Van Zandijcke M, et al. Diagnostic and therapeutic trajectory of cluster headache patients in Flanders. *Acta Neurol Belg* 2009; 109: 10–17.
Van Vliet JA, Eekers PJ, Haan J, et al. Features involved in the diagnostic delay of cluster headache. *J Neurol Neurosurg Psychiatry* 2003; 74: 1123–1125.

5.3.2 Paroxysmal hemifacial pain

Bahra A and Goadsby PJ. Diagnostic delays and mismanagement in cluster headache. *Acta Neurol Scand* 2004; 109: 175–179.
Bahra A, May A and Goadsby PJ. Cluster headache: a prospective clinical study with diagnostic implications. *Neurology* 2002; 58: 354–361.
Bartsch T, Knight YE and Goadsby PJ. Activation of 5-$HT_{1B/1D}$ receptor in the periaqueductal gray inhibits nociception. *Ann Neurol* 2004; 56: 371–381.
Benoliel R and Sharav Y. Paroxysmal hemicrania. Case studies and review of the literature. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998; 85: 285–292.
Benoliel R, Birman N, Eliav E, et al. The International Classification of Headache Disorders: accurate diagnosis of orofacial pain? *Cephalalgia* 2008; 28: 752–762.
Benoliel R, Elishoov H and Sharav Y. Orofacial pain with vascular-type features. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997; 84: 506–512.
Cademartiri C, Torelli P, Cologno D, et al. Upper and lower cluster headache: clinical and pathogenetic observations in 608 patients. *Headache* 2002; 42: 630–637.
Cittadini E, Matharu MS and Goadsby PJ. Paroxysmal hemicrania: a prospective clinical study of 31 cases. *Brain* 2008; 131: 1142–1155.
Delcanho RE and Graff-Radford SB. Chronic paroxysmal hemicrania presenting as toothache. *J Orofac Pain* 1993; 7: 300–306.
Gaul C, Gantenbein AR, Buettner UW, et al. Orofacial cluster headache. *Cephalalgia* 2008; 28: 903–905.
Graff-Radford SB. Paroxysmal hemicrania. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998; 86: 138.
Gross SG. Dental presentations of cluster headaches. *Curr Pain Headache Rep* 2006; 10: 126–129.
Larner AJ. Unnecessary extractions. *Br Dent J* 2007; 203: 442.
May A. The exceptional role of the 1st division of the trigeminal nerve. *Pain* 2018; 159: S81–S84.
Moncada E and Graff-Radford SB. Benign indomethacin-responsive headaches presenting in the orofacial region: eight case reports. *J Orofac Pain* 1995; 9: 276–284.
Sanchez Del Rio M, Leira R, Pozo-Rosich P, et al. Errors in recognition and management are still frequent in patients with cluster headache. *Eur Neurol* 2014; 72: 209–212.
Van Alboom E, Louis P, Van Zandijcke M, et al. Diagnostic and therapeutic trajectory of cluster headache patients in Flanders. *Acta Neurol Belg* 2009; 109: 10–17.
Van Vliet JA, Eekers PJ, Haan J, et al. Features involved in the diagnostic delay of cluster headache. *J Neurol Neurosurg Psychiatry* 2003; 74: 1123–1125.

5.3.3 Short-lasting unilateral neuralgiform facial pain attacks with cranial autonomic symptoms (SUNFA)

Benoliel R and Sharav Y. SUNCT syndrome: case report and literature review. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998; 85: 158–161.
Brown RS and Pass B. Orofacial pain due to trigeminal autonomic cephalgia with features of short-lasting neuralgiform headache attacks with conjunctival injection and tearing: a case report. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2012; 114: e13–e19.
Goadsby PJ, Cittadini E and Cohen AS. Trigeminal autonomic cephalalgias: paroxysmal hemicrania, SUNCT/SUNA, and hemicrania continua. *Semin Neurol* 2010; 30: 186–191.

5.3.4 Hemifacial continuous pain with autonomic symptoms

Benoliel R, Robinson S, Eliav E, et al. Hemicrania continua. *J Orofac Pain* 2002; 16: 317–325.
Goadsby PJ, Cittadini E and Cohen AS. Trigeminal autonomic cephalalgias: paroxysmal hemicrania,

SUNCT/SUNA, and hemicrania continua. *Semin Neurol* 2010; 30: 186–191.

Hryvenko I, Cervantes-Chavarría AR, Law AS, et al. Hemicrania continua: Case series presenting in an orofacial pain clinic. *Cephalalgia* 2018; 38: 1950–1959.

Prakash S, Shah ND and Chavda BV. Unnecessary extractions in patients with hemicrania continua: case reports and implication for dentistry. *J Orofac Pain* 2010; 24: 408–411.

Rossi P, Faroni J, Tassorelli C, et al. Diagnostic delay and suboptimal management in a referral population with hemicrania continua. *Headache* 2009; 49: 227–234.

Viana M, Tassorelli C, Allena M, et al. Diagnostic and therapeutic errors in trigeminal autonomic cephalalgias and hemicrania continua: a systematic review. *J Headache Pain* 2013; 14: 14.

Ziegeler C and May A. Facial presentations of migraine, TACs, and other paroxysmal facial pain syndromes. *Neurology* 2019; 93: e1138–e1147.

5.4 Neurovascular orofacial pain

Benoliel R, Birman N, Eliav E, et al. The International Classification of Headache Disorders: accurate diagnosis of orofacial pain? *Cephalalgia* 2008; 28: 752–762.

Benoliel R, Elishoov H and Sharav Y. Orofacial pain with vascular-type features. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997; 84: 506–512.

Czerninsky R, Benoliel R and Sharav Y. Odontalgia in vascular orofacial pain. *J Orofac Pain* 1999; 13: 196–200.

6. Idiopathic orofacial pain

Description:
Unilateral or bilateral intraoral or facial pain in the distribution(s) of one or more branches of the trigeminal nerve(s) for which the aetiology is unknown. The pain is usually persistent, of moderate intensity, poorly localized and described as dull, pressing or of burning character.

Classified elsewhere:
4.1.1.3 *Idiopathic trigeminal neuralgia*, 4.1.2.5 *Idiopathic trigeminal neuropathic pain*, 4.2.1.3 *Idiopathic glossopharyngeal neuralgia*, 4.2.2.2 *Idiopathic glossopharyngeal neuropathic pain*.

6.1 Burning mouth syndrome (BMS)

Previously used terms:
Stomatodynia; glossodynia (when confined to the tongue); primary burning mouth syndrome.

Description:
An intraoral burning or dysaesthetic sensation, recurring daily for more than 2 hours per day for more than 3 months, without evident causative lesions on clinical examination and investigation.

Diagnostic criteria:

A. Oral pain fulfilling criteria B and C
B. Recurring daily for >2 hours per day for >3 months[1]
C. Pain has both of the following characteristics:
   1. burning quality
   2. felt superficially in the oral mucosa
D. Oral mucosa is of normal appearance, and local or systemic causes have been excluded
E. Not better accounted for by another ICOP or ICHD-3 diagnosis.[2]

Notes:

1. Prior to 3 months, if all other criteria are fulfilled, code as 6.1.3 *Probable burning mouth syndrome*.
2. A diagnosis of 6.1 *Burning mouth syndrome* implies that quantitative sensory testing has not been performed. Once it has, either of the two subtypes 6.1.1 *Burning mouth syndrome without somatosensory changes* or 6.1.2 *Burning mouth syndrome with somatosensory changes* should be diagnosed.

Comments:
Quantitative sensory testing is often abnormal (differentiating the two subtypes), whereas clinical sensory examination very rarely reveals slight sensory deficits.

The pain of 6.1 *Burning mouth syndrome* is usually bilateral, but on rare occasions it is unilateral. Its intensity fluctuates. The most common site is the tip of the tongue. Subjective xerostomia, dysaesthesia and altered taste are present in two-thirds of cases reported.

There is a high preponderance in menopausal women. Some studies show psychosocial comorbidities similar to those of other persistent pain conditions. Recent data point to varying levels of changes in somatosensory function in patients with 6.1 *Burning mouth syndrome*. These findings encourage further research into the disorder as a possible neuropathic pain condition.

Burning mouth *symptoms* may occur as secondary phenomena due to a local or systemic condition, as in subforms of 1.1.3 *Gingival pain* or 1.2.1 *Oral mucosal pain*. They have previously been known as 'secondary burning mouth syndrome', but should be coded to these disorders. 6.1 *Burning mouth syndrome* is diagnosed only when all local and systemic causes have been excluded (hence, previously, 'primary burning mouth syndrome').

6.1.1 Burning mouth syndrome without somatosensory changes

Description:
An intraoral burning or dysaesthetic sensation, recurring daily for more than 2 hours per day for more than 3 months, unaccompanied by somatosensory changes and without evident causative lesions on clinical examination and investigation.

Diagnostic criteria:

A. Oral pain fulfilling criteria for 6.1 *Burning mouth syndrome*
B. Somatosensory changes are not present on qualitative or quantitative somatosensory testing.

6.1.2 Burning mouth syndrome with somatosensory changes

Description:
An intraoral burning or dysaesthetic sensation, recurring daily for more than 2 hours per day for more than 3 months and accompanied by negative and/or positive somatosensory changes, without evident causative lesion(s) on clinical examination and investigation.

Diagnostic criteria:

A. Oral pain fulfilling criteria for 6.1 *Burning mouth syndrome*
B. Somatosensory changes are present on qualitative and/or quantitative somatosensory testing.[1]

Note:

1. Negative (e.g. hypaesthesia and/or hypalgesia) and/or positive (e.g. hyperalgesia and/or allodynia) sensory symptoms and/or signs.

6.1.3 Probable burning mouth syndrome

Description:
An intraoral burning or dysaesthetic sensation, recurring daily for more than 2 hours per day but for less than 3 months, without evident causative lesions on clinical examination and investigation.

Diagnostic criterion:

A. Oral pain fulfilling criteria for 6.1 *Burning mouth syndrome* except that it has been present for <3 months.[1]

Note:

1. Once 3 months have elapsed, the diagnosis becomes 6.1 *Burning mouth syndrome* (or one of its subtypes).

Comment:
Subforms are not formally classified but may be coded 6.1.3.1 *Probable burning mouth syndrome without somatosensory changes* or 6.1.3.2 *Probable burning mouth syndrome with somatosensory changes* according to the criteria above.

6.2 Persistent idiopathic facial pain (PIFP)

Previously used term:
Atypical facial pain.

Description:
Persistent facial pain, with variable features, recurring daily for more than 2 hours per day for more than 3 months, in the absence of clinical neurological deficit or preceding causative event.

Diagnostic criteria:

A. Facial pain fulfilling criteria B and C
B. Recurring daily for >2 hours/day for >3 months[1]
C. Pain has both of the following characteristics:
   1. poorly localized, and not following the distribution of a peripheral nerve[2]
   2. dull, aching or nagging quality[3]
D. Clinical and radiographic examinations are normal,[4] and local causes have been excluded[5]
E. Not better accounted for by another ICOP or ICHD-3 diagnosis.[6]

Notes:

1. Prior to 3 months, if all other criteria are fulfilled, code as 6.2.3 *Probable persistent idiopathic facial pain*.
2. The pain may be described as either deep or superficial, and may radiate from face to mouth or vice versa. With time, it may spread to a wider area of the craniocervical region.
3. A wide variety of words are used to describe the character, and the pain can have exacerbations and be aggravated by stress.
4. Clinical somatosensory assessment with pinprick or light touch perception may very rarely reveal slight somatosensory changes. Nociplastic pain reflecting altered processing in the somatosensory system may be present, and related to alteration in the modulatory pain inhibitory system.
5. Patients may report a minor operation or injury to the face, maxilla(e), teeth or gingiva(e), but upon clinical and radiographic examinations there is no demonstrable local cause.
6. Quantitative sensory testing differentiates the two subtypes. A diagnosis of 6.2 *Persistent idiopathic facial pain* implies that quantitative sensory testing has not been performed. Once it has, either of the two subtypes 6.2.1 *Persistent idiopathic facial pain without somatosensory changes* or 6.2.2 *Persistent idiopathic facial pain with somatosensory changes* should be diagnosed.

Comments:
In ICHD-3, two disorders are treated as one entity: 13.12 *Persistent idiopathic facial pain*, with *atypical odontalgia* as a possible subtype. These ICOP criteria distinguish and define two entities: 6.2 *Persistent idiopathic facial pain* and 6.3 *Persistent idiopathic dentoalveolar pain*. These conditions cause facial or dentoalveolar pain, respectively, in either case of a fairly constant nature but prone nevertheless to exacerbations.

6.2 *Persistent idiopathic facial pain* may be comorbid with other pain conditions such as chronic widespread pain and irritable bowel syndrome. In addition, it can present with psychosocial comorbidities similar to those of other persistent pain conditions.

6.2.1 Persistent idiopathic facial pain without somatosensory changes

Description:
Persistent facial pain, with variable features, recurring daily for more than 2 hours per day for more than

3 months, unaccompanied by somatosensory changes and in the absence of clinical neurological deficit or preceding causative event.

Diagnostic criteria:

A. Facial pain fulfilling criteria for 6.2 *Persistent idiopathic facial pain*
B. Somatosensory changes are not present on qualitative or quantitative somatosensory testing.

6.2.2 Persistent idiopathic facial pain with somatosensory changes

Description:
Persistent facial pain, with variable features, recurring daily for more than 2 hours per day for more than 3 months and accompanied by negative and/or positive somatosensory changes, in the absence of clinical neurological deficit or preceding causative event.

Diagnostic criteria:

A. Facial pain fulfilling criteria for 6.2 *Persistent idiopathic facial pain*
B. Somatosensory changes are present on qualitative and/or quantitative somatosensory testing.[1]

Note:

1. Negative (e.g. hypaesthesia and/or hypalgesia) and/or positive (e.g. hyperalgesia and/or allodynia) sensory symptoms and/or signs.

6.2.3 Probable persistent idiopathic facial pain

Description:
Facial pain, with variable features, recurring daily for more than 2 hours per day but for less than 3 months, in the absence of clinical neurological deficit or preceding causative event.

Diagnostic criterion:
A. Facial pain fulfilling criteria for 6.2 *Persistent idiopathic facial pain* except that it has been present for <3 months.[1]

Note:

1. Once 3 months have elapsed, the diagnosis becomes 6.2 *Persistent idiopathic facial pain* (or one of its subtypes).

Comment:
Subforms are not formally classified but may be coded 6.2.3.1 *Probable persistent idiopathic facial pain without somatosensory changes* or 6.2.3.2 *Probable persistent idiopathic facial pain with somatosensory changes* according to the criteria above.

6.3 Persistent idiopathic dentoalveolar pain

Previously used terms:
Atypical odontalgia; primary persistent dentoalveolar pain disorder (PDAP); phantom tooth pain.

Description:
Persistent unilateral intraoral dentoalveolar pain, rarely occurring in multiple sites, with variable features but recurring daily for more than 2 hours per day for more than 3 months, in the absence of any preceding causative event.

Diagnostic criteria:

A. Intraoral dentoalveolar pain fulfilling criteria B and C
B. Recurring daily for >2 hours/day for >3 months[1]
C. Pain has both of the following characteristics:
   1. localized to a dentoalveolar site (tooth or alveolar bone)[2]
   2. deep, dull, pressure-like quality[3]
D. Clinical and radiographic examinations are normal,[4] and local causes have been excluded
E. Not better accounted for by another ICOP or ICHD-3 diagnosis.[5]

Notes:

1. Prior to 3 months, if all other criteria are fulfilled, code as 6.3.3 *Probable persistent idiopathic dentoalveolar pain*.
2. Pain is rarely in multiple sites. With time, it may spread to a wider area of the craniocervical region.
3. A wide variety of words are used to describe the character and quality of the pain. It may be described as either deep or superficial, and adjunctive symptom description may be employed to explain the complexity of sensations associated with this disorder. Furthermore, the pain can have exacerbations and be aggravated by stress.
4. Clinical somatosensory assessment with pinprick or light touch perception only very rarely reveals sensory abnormalities. Nociplastic pain reflecting altered processing in the somatosensory system may be present, and related to alteration in the modulatory pain inhibitory system.

5. Quantitative sensory testing differentiates the two subtypes. A diagnosis of 6.3 *Persistent idiopathic dentoalveolar pain* implies that quantitative sensory testing has not been performed. Once it has, either of the two subtypes 6.3.1 *Persistent idiopathic dentoalveolar pain without somatosensory changes* or 6.3.2 *Persistent idiopathic dentoalveolar pain with somatosensory changes* should be diagnosed.

Comment:
In ICHD-3, two disorders are treated as one entity: 13.12 *Persistent idiopathic facial pain*, with *atypical odontalgia* as a possible subtype. These ICOP criteria distinguish and define two entities: 6.2 *Persistent idiopathic facial pain* and 6.3 *Persistent idiopathic dentoalveolar pain*. These conditions cause facial or dentoalveolar pain, respectively, in either case of a fairly constant nature but prone nevertheless to exacerbations.

6.3.1 Persistent idiopathic dentoalveolar pain without somatosensory changes

Description:
Persistent unilateral intraoral dentoalveolar pain, rarely occurring in multiple sites, with variable features, recurring daily for more than 2 hours per day for more than 3 months, unaccompanied by somatosensory changes and in the absence of any preceding causative event.

Diagnostic criteria:

A. Intraoral pain fulfilling criteria for 6.3 *Persistent idiopathic dentoalveolar pain*
B. Somatosensory changes are not present on qualitative or quantitative somatosensory testing.

6.3.2 Persistent idiopathic dentoalveolar pain with somatosensory changes

Description:
Persistent unilateral intraoral dentoalveolar pain, rarely occurring in multiple sites, with variable features, recurring daily for more than 2 hours per day for more than 3 months and accompanied by negative and/or positive somatosensory changes, in the absence of any preceding causative event.

Diagnostic criteria:

A. Intraoral pain fulfilling criteria for 6.3 *Persistent idiopathic dentoalveolar pain*
B. Somatosensory changes are present on qualitative and/or quantitative somatosensory testing.[1]

Note:

1. Negative (e.g. hypaesthesia and/or hypalgesia) and/or positive (e.g. hyperalgesia and/or allodynia) sensory symptoms and/or signs are present, but not spatially confined to a neuroanatomically relevant area, in contrast to 4.1.2.3 *Post-traumatic trigeminal neuropathic pain*.

6.3.3 Probable persistent idiopathic dentoalveolar pain

Description:
Unilateral intraoral dentoalveolar pain, rarely occurring in multiple sites, with variable features, recurring daily for more than 2 hours per day but for less than 3 months, in the absence of any preceding causative event.

Diagnostic criterion:
A. Oral pain fulfilling criteria for 6.3 *Persistent idiopathic dentoalveolar pain* except that it has been present for <3 months.[1]

Note:

1. Once 3 months have elapsed, the diagnosis becomes 6.3 *Persistent idiopathic dentoalveolar pain* (or one of its subtypes).

Comment:
Subforms are not formally classified, but may be coded 6.3.3.1 *Probable persistent idiopathic dentoalveolar pain without somatosensory changes* or 6.3.3.2 *Probable persistent idiopathic dentoalveolar pain with somatosensory changes* according to the criteria above.

6.4 Constant unilateral facial pain with additional attacks (CUFPA)

Description:
Constant (unremitting) dull unilateral facial pain of mild to moderate intensity, accompanied by distinct attacks of moderate to severe pain in the same location lasting 10–30 minutes. There are no typical autonomic and/or migrainoid features accompanying either the constant pain or the additional pain attacks.

Diagnostic criteria:

A. Constant strictly unilateral facial pain fulfilling criterion B, with exacerbations fulfilling criterion C

B. Background pain, with both of the following characteristics:
   1. mild to moderate intensity
   2. constantly present for >3 months
C. Exacerbations occurring as distinct attacks, up to 6 times daily, with all of the following three characteristics:
   1. in the same location as the background pain
   2. moderate to severe intensity
   3. lasting 10–30 minutes
D. Clinical and radiographic examinations are normal, and local causes have been excluded
E. Not better accounted for by another ICOP or ICHD-3 diagnosis.[1]

Note:

1. The exacerbations must occur as attacks clearly distinct from the background pain, with patients describing pain having these two sets of features; otherwise the diagnoses of 5.3.2 *Paroxysmal hemifacial pain* or 6.2 *Persistent idiopathic facial pain* should be considered. A response to indomethacin should rather lead to the diagnosis of 5.3.2 *Paroxysmal hemifacial pain*.

Comments:

Autonomic symptoms should be absent, but do not exclude 6.4 *Constant unilateral facial pain with additional attacks*.

At present there are insufficient data to propose episodic and chronic subforms of 6.4 *Constant unilateral facial pain with additional attacks*.

References

Baad-Hansen L. Atypical odontalgia – pathophysiology and clinical management. *J Oral Rehabil* 2008; 35: 1–11.

Baad-Hansen L, Leijon G, Svensson P, et al. Comparison of clinical findings and psychosocial factors in patients with atypical odontalgia and temporomandibular disorders. *J Orofac Pain* 2008; 22: 7–14.

Baad-Hansen L, Pigg M, Ivanovic SE, et al. Intraoral somatosensory abnormalities in patients with atypical odontalgia – a controlled multicenter quantitative sensory testing study. *Pain* 2013; 154: 1287–1294.

Benoliel R and Gaul C. Persistent idiopathic facial pain. *Cephalalgia* 2017; 37: 680–691.

Durham J, Exley C, John MT, et al. Persistent dentoalveolar pain: the patient's experience. *J Orofac Pain* 2013; 27: 6–13.

Forssell H, Jääskeläinen S, List T, et al. An update on pathophysiological mechanisms related to idiopathic oro-facial pain conditions with implications for management. *J Oral Rehabil* 2015; 42: 300–322.

Forssell H, Tenovuo O, Silvoniemi P, et al. Differences and similarities between atypical facial pain and trigeminal neuropathic pain. *Neurology* 2007; 69: 1451–1459.

Grémeau-Richard C, Dubray C, Aublet-Cuvelier B, et al. Effect of lingual nerve block on burning mouth syndrome (stomatodynia): a randomized crossover trial. *Pain* 2010; 149: 27–32.

Hagelberg N, Jääskeläinen SK, Martikainen IK, et al. Striatal dopamine D2 receptors in modulation of pain in humans: a review. *Eur J Pharmacol* 2004; 500: 187–192.

Häggman-Henrikson B, Alstergren P, Davidson T, et al. Pharmacological treatment of oro-facial pain – health technology assessment including a systematic review with network meta-analysis. *J Oral Rehabil* 2017; 44: 800–826.

Jääskeläinen SK. Pathophysiology of primary burning mouth syndrome. *Clin Neurophysiol* 2012; 123: 71–77.

Jääskeläinen SK and Woda A. Burning mouth syndrome. *Cephalalgia* 2017; 37: 627–647.

Kolkka-Palomaa M, Jääskeläinen SK, Laine MA, et al. Pathophysiology of primary burning mouth syndrome with special focus on taste dysfunction: a review. *Oral Dis* 2015; 21: 937–948.

Lang E, Kaltenhäuser M, Seidler S, et al. Persistent idiopathic facial pain exists independent of somatosensory input from the painful region: findings from quantitative sensory functions and somatotopy of the primary somatosensory cortex. *Pain* 2005; 118: 80–91.

List T, Leijon G and Svensson P. Somatosensory abnormalities in atypical odontalgia: A case-control study. *Pain* 2008; 139: 333–341.

McMillan R, Forssell H, Buchanan JA, et al. Interventions for treating burning mouth syndrome. *Cochrane Database Syst Rev* 2016; 11: CD002779.

Nixdorf DR, Drangsholt MT, Ettlin DA, et al. Classifying orofacial pains: a new proposal of taxonomy based on ontology. *J Oral Rehabil* 2012; 39: 161–169.

Puhakka A, Forssell H, Soinila S, et al. Peripheral nervous system involvement in primary burning mouth syndrome – results of a pilot study. *Oral Dis* 2016; 22: 338–344.

Scala A, Checchi L, Montevecchi M, et al. Update on burning mouth syndrome: overview and patient management. *Crit Rev Oral Biol Med* 2003; 14: 275–291.

Ziegeler C and May A. Facial presentations of migraine, TACs, and other paroxysmal facial pain syndromes. *Neurology* 2019; 93: e1138–e1147.

7. Psychosocial assessment of patients with orofacial pain

Introduction

The biopsychosocial model incorporates psychological and social factors in order more comprehensively to understand, and manage, both disease (as related to traditional medical factors) and illness across time and circumstance. Major psychological factors associated with pain disorders include anxiety, catastrophizing, depression, physical symptom reporting and fear-avoidance; major social factors include access to medical care, stigma, and support from family and friends. Each of these factors has extensive empirical support for their association with pain disorders, and evidence clearly supports the significance of the biopsychosocial model as crucial for understanding the complexity of pain processing in general (1), as well as in relation to orofacial pain (OFP) disorders (2–6). Notably, the implementation of the biopsychosocial model into both research and clinical pain medicine remains variable; further detail is available (7–9), and new taxonomies for chronic pain of all types clearly highlight the central importance of both physical criteria for the disorders as well as assessment of psychosocial factors (10).

For present purposes, recommendations for best research practices in support of the intent of the ICOP taxonomy are presented for the OFP field broadly, and follow previously established recommendations for the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) (11) and the Diagnostic Criteria for TMD (DC/TMD) (12,13), which specify appropriate constructs and instruments for the assessment of musculoskeletal pain (e.g. painful temporomandibular disorders (TMDs)). While these recommendations emerge from substantial research on the TMDs, a subset of OFPs, no evidence exists at this time to suggest that pain from the non-TMD OFP disorders is any different from the pain associated with the TMDs in terms of pain processing models. Consequently, research at this stage of development of ICOP should include equivalent attention to the full biopsychosocial model and thereby include assessment of recommended psychosocial constructs. Subsequent structured and systematic evidence will permit a more empirically supported assessment model for the non-TMD OFPs and lead to revision of these initial recommendations.

Levels of psychosocial assessment

Two levels of psychosocial assessment are defined by the DC/TMD (13) and one more was developed in response to specific clinical request (see Table 1 for summary). The brief screening version is intended for research (and clinical) settings where only the briefest biopsychosocial assessment using the fewest number of questions can be incorporated (14). Interestingly, the same components of this brief screening have been informally described by other colleagues, suggesting a convergence into a core minimal set of psychosocial assessment domains. The standard screening version incorporates two more instruments. Both forms of screening should be recognized as very limited.

Table 1. Different levels of psychosocial assessment (see text for details).

Instrument	No. of items	Comprehensive assessment	Standard screening	Brief screening
Pain drawing	1	4	4	4
GCPS v2.0	8	4	4	4
JFLS (long form)	20	4		
JFLS (short form)	8		4	
PHQ-4	5[a]		4	4
PHQ-9	10[a]	4		
GAD-7	8[a]	4		
PHQ-15	15	4		
OBC	20	4	4	

[a]Item count includes reflective question regarding functional impact of any reported symptoms.
GAD: generalized anxiety disorder; GCPS: Graded Chronic Pain Scale; JFLS: Jaw Functional Limitation Scale; PHQ: Patient Health Questionnaire; OBC: Oral Behaviours Checklist.

The comprehensive assessment is intended specifically for clinical researchers so that they can more reliably measure all constructs of interest, thereby permitting full stratification of their samples based on a psychosocial profile. All of these instruments are freely available with interpretation guides for the scoring at the following website: www.rdc-tmdinternational.org. In addition, a few other instruments are recommended below for consideration.

Pain- and function-related constructs and instruments for OFPs

Extent of pain. The pain drawing (also known as a 'body manikin') provides ready identification of all pain location(s), known to be one major risk determinant for pain chronicity (15). All pain disorders, regardless of putative nociceptive mechanism, appear to be similarly affected in terms of the extent of pain.

Pain intensity and pain-related disability. The Graded Chronic Pain Scale (GCPS, v2.0) is a widely used and validated instrument examining pain persistence, pain intensity and pain-related disability, also called graded

chronic pain status, which has utility to stratify patients for levels of care (16–18). Graded chronic pain status is also an indicator of prognosis in that higher graded chronic pain status predicts greater chance of pain chronicity (19).

Functional limitation. The person's experience of impaired functional ability is known as functional limitation (20). The Jaw Functional Limitation Scale (JFLS) has two versions: an 8-item version, which yields a global score, and a 20-item version, which measures three domains (limitation in chewing, jaw opening, and verbal and emotional expression) (21,22). Both versions are equally reliable, valid and sensitive to change. While functional limitation is central to musculoskeletal pain (and thereby self-evident for relevance to TMDs), functional consequences are assumed to occur in response to the non-TMD OFPs; the nature of those consequences is suspected (22) and warrants further investigation in order to understand the full dimensionality of pain in the orofacial region.

Overuse behaviours. The Oral Behaviours Checklist (OBC) contains a list of 21 oral region activities individuals may engage in, such as clenching the teeth, bracing the mandible, or talking. Psychometric properties are strong (23–25) and OBC values are associated with TMDs (15,26–30). Whether these behaviours are specifically associated with the OFPs remains unknown; however, guarding behaviours are known to affect non-musculoskeletal back pain (31), suggesting applicability for non-TMD OFPs.

Psychosocial constructs and instruments for OFPs

Depression and anxiety. The PRIME-MD project (PRIMary care Evaluation of Mental Disorders) (32) was anchored initially in psychiatric disorders and aimed to develop psychosocial instruments for the assessment of five of the most common mental health problems presenting to primary care: anxiety, depression and somatoform, alcohol and eating disorders (33). Particularly relevant for pain disorders are the 9-item Patient Health Questionnaire (PHQ-9) for depression and the 7-item Generalized Anxiety Disorder scale (GAD-7) for anxiety; each of these instruments will permit reliable and valid measurement of the respective core constructs. Each instrument contributes two questions to create the brief PHQ-4 depression and anxiety screening instrument, often considered to assess 'distress', which is widely used across North America and Europe. Depression (measured with PHQ-9, PHQ-4) is a mood state known to be affected by the presence of persistent pain and known to affect pain processing, and it appears to be highly relevant to OFPs (34). Anxiety (measured with GAD-7, PHQ-4) in a medical context often manifests as worry and general sympathetic nervous system activation, and it is associated with pain perception (35) and hypervigilance (36). Anxiety pervades medical settings, and it appears to be highly relevant to OFPs (37).

Somatoform disorders. The PRIME-MD project also yielded the Patient Health Questionnaire-15 (PHQ-15) for somatic symptom severity. Physical symptoms not accompanied by appropriate signs supportive of a disease diagnosis remain a considerable challenge across all medical domains; such findings are appropriately termed *somatic symptom disorder*, *functional disorders*, *medically unexplained symptoms*, and *symptoms without medical utility* (38,39), yet none of these terms is fully satisfactory for what appears to be a more complex construct than previously assumed. While there are several proposed mechanisms underlying physical symptom reporting (39–41), all are consistent with the various symptoms that accompany the persistent OFPs (37,42). An extension of this phenomenon involves occlusal dysaesthesia (43), which has potential relevance to at least a few of the OFPs within ICOP.

Catastrophizing. Catastrophizing about pain is 'characterized by the tendency to magnify the threat value of pain stimulus and to feel helpless in the context of pain, and by a relative inability to inhibit pain-related thoughts in anticipation of, during or following a painful encounter' (44). Higher levels of catastrophizing are linked to increased utilization of health care, increased expression of pain and poorer treatment outcomes (45–47). Catastrophizing is not included as a standard measure within the DC/TMD framework because the evidence for the relevance of catastrophizing to TMDs was not sufficiently strong when the DC/TMD Axis II recommendations were formulated. The situation has since changed and, moreover, its relevance to any pain disorder is now appropriate for this construct to be included as a recommended domain within ICOP. Appropriate validated measures include the Pain Catastrophizing Scale (48) and the Coping Strategies Questionnaire (49).

Fear avoidance. The fear-avoidance model emerged from operant models pertaining to low back pain (50): specifically, behavioural observations where individuals reported pain incongruent with physical findings. The fear-avoidance model has since had extensive supporting research (51). In the model, having no fear of injury-related new-onset pain leads to engaging in the appropriate behaviours that will result in recovery from the injury. In contrast, having fear of that pain leads to catastrophizing about that pain, avoidance of

circumstances that may cause pain, and consequent disuse, depression and disability. Disability then feeds forward to further pain experience, avoidant behaviour and the absence of recovery; as such, this model is clearly relevant to motor behaviour, and support for this construct for TMD is slowly emerging. The model, however, is a person-level model regarding the effects of behaviour and beliefs on the central nervous system and thereby assumed to be linked to pain processing. Consequently, the clearly plausible hypotheses relating fear of movement to recovery among those with injury to the masticatory system and the probable emergence of chronic pain among some of those individuals warrant investigation (52), and present data suggest that this perspective is applicable to OFPs as well.

Fear of pain is measured via several instruments, of which the Tampa Scale for Kinesiophobia (TSK) (53) is the best known and has very strong utility for back pain (54). TSK was adapted for the masticatory system, the TSK-TMD (55), and it appears to capture both the somatic experience as well as avoidant behaviours that OFPs may precipitate.

Conclusions and future directions

Further research of the biopsychosocial model and its clinical as well as research relevance to OFPs is clearly needed. As the criteria for the disorders within ICOP are better developed and refined, a similar progression should occur regarding our understanding of the persons with these pain disorders. Consequently, as taxonomy and diagnosis improve, improved understanding of pain mechanisms, as well as sound treatment recommendations, should emerge. Multimodal approaches are clearly needed to augment the potential therapeutic yield of standard biomedical therapies such as pharmacological or surgical approaches (56). For the present time, consistent use of a standardized format for psychosocial assessment of the individual with OFP is strongly recommended, in parallel with use of the stated criteria (and their extensions for research purposes) of the disorders within ICOP.

Acknowledgement

This chapter is an adaptation from Ohrbach and Durham (8).

References

1. Campbell CM and Edwards RR. Mind–body interactions in pain: the neurophysiology of anxious and catastrophic pain-related thoughts. *Transl Res* 2009; 153: 97–101.
2. Ceusters W, Michelotti A, Raphael KG, et al. Perspectives on next steps in classification of orofacial pain – part 1: role of ontology. *J Oral Rehabil* 2015; 42: 926–941.
3. Greene CS, Mohl ND, McNeill C, et al. Temporomandibular disorders and science: a response to the critics. *J Prosthet Dent* 1998; 80: 214–215.
4. Greene CS and Obrez A. Treating temporomandibular disorders with permanent mandibular repositioning: is it medically necessary? *Oral Surg Oral Med Oral Pathol Oral Radiol* 2015; 119: 489–498.
5. Michelotti A, Alstergren P, Goulet JP, et al. Next steps in development of the diagnostic criteria for temporomandibular disorders (DC/TMD): Recommendations from the International RDC/TMD Consortium Network workshop. *J Oral Rehabil* 2016; 43: 453–467.
6. Ohrbach R and Greene C. Temporomandibular joint diagnosis: striking a balance between the sufficiency of clinical assessment and the need for imaging. *Oral Surg Oral Med Oral Pathol Oral Radiol* 2013; 116: 124–125.
7. Durham J and Ohrbach R. Commentary on disability and dental education. *J Oral Rehabil* 2010; 37: 490–494.
8. Ohrbach R and Durham J. Biopsychosocial aspects of orofacial pain. In: CS Farah, R Balasubramaniam and MJ McCullough (eds) *Contemporary oral medicine*. Heidelberg: Springer Meteor, 2018, pp.1–21.
9. Ohrbach R and Dworkin SF. The evolution of TMD diagnosis: past, present, future. *J Dent Res* 2016; 95: 1093–1101.
10. Fillingim RB, Bruehl S, Dworkin RH, et al. The ACTTION-American Pain Society Pain Taxonomy (AAPT): an evidence-based and multidimensional approach to classifying chronic pain conditions. *J Pain* 2014; 15: 241–249.
11. Dworkin SF and LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord* 1992; 6: 301–355.
12. Schiffman E and Ohrbach R. Executive summary of the Diagnostic Criteria for Temporomandibular Disorders for clinical and research applications. *J Am Dent Assoc* 2016; 147: 438–445.
13. Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for clinical and research applications: recommendations of the International RDC/TMD Consortium Network and Orofacial Pain Special Interest Group. *J Oral Facial Pain Headache* 2014; 28: 6–27.

14. Ohrbach R and Michelotti A. Psychological considerations. In: S Kandasamy, CS Greene, DJ Rinchuse, et al. (eds) *TMD and orthodontics: A clinical guide for the orthodontist*. Cham, Switzerland: Springer, 2015, pp.49–61.
15. Ohrbach R, Fillingim RB, Mulkey F, et al. Clinical findings and pain symptoms as potential risk factors for chronic TMD: descriptive data and empirically identified domains from the OPPERA case-control study. *J Pain* 2011; 12(11, Suppl 3): T27–T45.
16. Durham J, Shen J, Breckons M, et al. Healthcare cost and impact of persistent orofacial pain: The DEEP Study Cohort. *J Dent Res* 2016; 95: 1147–1154.
17. Dworkin SF, Huggins KH, Wilson L, et al. A randomized clinical trial using research diagnostic criteria for temporomandibular disorders-axis II to target clinic cases for a tailored self-care TMD treatment program. *J Orofac Pain* 2002; 16: 48–63.
18. Dworkin SF, Turner JA, Mancl L, et al. A randomized clinical trial of a tailored comprehensive care treatment program for temporomandibular disorders. *J Orofac Pain* 2002; 16: 259–276.
19. Von Korff M and Dunn KM. Chronic pain reconsidered. *Pain* 2008; 138: 267–276.
20. Ohrbach R. Disability assessment in temporomandibular disorders and masticatory system rehabilitation. *J Oral Rehabil* 2010; 37: 452–480.
21. Ohrbach R, Granger CV, List T, et al. Pain-related functional limitation of the jaw: preliminary development and validation of the Jaw Functional Limitation Scale. *Community Dent Oral Epidemiol* 2008; 36: 228–236.
22. Ohrbach R, Larsson P and List T. The Jaw Functional Limitation Scale: development, reliability, and validity of 8-item and 20-item versions. *J Orofac Pain* 2008; 22: 219–230.
23. Kaplan SEF and Ohrbach R. Self-report of waking-state oral parafunctional behaviors in the natural environment. *J Oral Facial Pain Headache* 2016; 30: 107–119.
24. Markiewicz MR, Ohrbach R and McCall WD Jr. Oral Behaviors Checklist: reliability of performance in targeted waking-state behaviors. *J Orofac Pain* 2006; 20: 306–316.
25. Ohrbach R, Markiewicz MR and McCall WD Jr. Waking-state oral parafunctional behaviors: specificity and validity as assessed by electromyography. *Eur J Oral Sci* 2008; 116: 438–444.
26. Carlsson GE, Egermark I and Magnusson T. Predictors of bruxism, other oral parafunctions, and tooth wear over a 20-year follow-up period. *J Orofac Pain* 2003; 17: 50–57.
27. Glaros AG and Burton E. Parafunctional clenching, pain, and effort in temporomandibular disorders. *J Behav Med* 2004; 27: 91–100.
28. Glaros AG, Marszalek JM and Williams KB. Longitudinal multilevel modeling of facial pain, muscle tension, and stress. *J Dent Res* 2016; 95: 416–422.
29. Glaros AG and Williams K. Tooth contact versus clenching: oral parafunctions and facial pain. *J Orofac Pain* 2012; 26: 176–180.
30. Ohrbach R, Bair E, Fillingim RB, et al. Clinical orofacial characteristics associated with risk of first-onset TMD: the OPPERA prospective cohort study. *J Pain* 2013; 14(12, Suppl 2): T33–T50.
31. O'Sullivan P. Diagnosis and classification of chronic low back pain disorders: maladaptive movement and motor control impairments as underlying mechanism. *Man Ther* 2005; 10: 242–255.
32. Spitzer RL, Kroenke K and Williams JBW. Validation and utility of a self-report version of PRIME-MD: The PHQ Primary Care Study. *JAMA* 1999; 282: 1737–1744.
33. Kroenke K, Spitzer RL, Williams JB, et al. The patient health questionnaire somatic, anxiety, and depressive symptom scales: a systematic review. *Gen Hosp Psychiatry* 2010; 32: 345–359.
34. Durham J, Raphael KG, Benoliel R, et al. Perspectives on next steps in classification of orofacial pain – part 2: role of psychosocial factors. *J Oral Rehabil* 2015; 42: 942–955.
35. Robinson ME, Wise EA, Gagnon C, et al. Influences of gender role and anxiety on sex differences in temporal summation of pain. *J Pain* 2004; 5: 77–82.
36. Cioffi I, Michelotti A, Perrotta S, et al. Effect of somatosensory amplification and trait anxiety on experimentally induced orthodontic pain. *Eur J Oral Sci* 2016; 124: 127–134.
37. Aggarwal VR, McBeth J, Zakrzewska JM, et al. The epidemiology of chronic syndromes that are frequently unexplained: do they have common associated factors? *Int J Epidemiol* 2006; 35: 468–476.
38. Kirmayer LJ and Robbins JM. Functional somatic syndromes. In: LJ Kirmayer and JM Robbins (eds) *Current concepts of somatization: research and clinical perspectives*. Washington, DC: American Psychiatric Press, 1991, pp.79–106.
39. Rief W and Broadbent E. Explaining medically unexplained symptoms-models and mechanisms. *Clin Psychol Rev* 2007; 27: 821–841.
40. Craig AD. How do you feel? Interoception: the sense of the physiological condition of the body. *Nat Rev Neurosci* 2002; 3: 655–666.

41. Craig AD. A new view of pain as a homeostatic emotion. *Trends Neurosci* 2003; 26: 303–307.
42. Peters S, Goldthorpe J, McElroy C, et al. Managing chronic orofacial pain: a qualitative study of patients', doctors', and dentists' experiences. *Br J Health Psychol* 2015; 20: 777–791.
43. Melis M and Zawawi KH. Occlusal dysesthesia: a topical narrative review. *J Oral Rehabil* 2015; 42: 779–785.
44. Quartana PJ, Campbell CM and Edwards RR. Pain catastrophizing: a critical review. *Expert Rev Neurother* 2009; 9: 745–758.
45. Brister H, Turner JA, Aaron LA, et al. Self-efficacy is associated with pain, functioning, and coping in patients with chronic temporomandibular disorder pain. *J Orofac Pain* 2006; 20: 115–124.
46. Litt MD and Porto FB. Determinants of pain treatment response and nonresponse: identification of TMD patient subgroups. *J Pain* 2013; 14: 1502–1513.
47. Turner JA, Brister H, Huggins KH, et al. Catastrophizing is associated with clinical examination findings, activity interference, and health care use among patients with temporomandibular disorders. *J Orofac Pain* 2005; 19: 291–300.
48. Sullivan MJL, Bishop SR and Pivik J. The Pain Catastrophizing Scale: development and validation. *Psychol Assess* 1995; 7: 524–532.
49. Harland NJ and Georgieff K. Development of the Coping Strategies Questionnaire 24, a clinically utilitarian version of the coping strategies questionnaire. *Rehabil Psychol* 2003; 48: 296–300.
50. Fordyce WE. *Behavioral methods for chronic pain and illness*. St Louis, MO: CV Mosby, 1976.
51. Vlaeyen JW and Linton SJ. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. *Pain* 2000; 85: 317–332.
52. Wall PD. On the relation of injury to pain. *Pain* 1979; 6: 253–264.
53. Kori SH, Miller RP and Todd DD. Kinesiophobia: a new view of chronic pain behavior. *Pain Manag* 1990; 3: 35–43.
54. Boersma K and Linton SJ. Expectancy, fear and pain in the prediction of chronic pain and disability: a prospective analysis. *Eur J Pain* 2006; 10: 551–557.
55. Visscher CM, Ohrbach R, van Wijk AJ, et al. The Tampa Scale for Kinesiophobia for Temporomandibular Disorders (TSK-TMD). *Pain* 2010; 150: 492–500.
56. Spencer CJ, Neubert JK, Gremillion H, et al. Case reviews in pain: toothache or trigeminal neuralgia: treatment dilemmas. *J Pain* 2008; 9: 767–770.

Definitions of terms in ICOP (1)

Allodynia: Pain (qv) due to a stimulus that does not normally provoke pain. This is a clinical term that does not imply a mechanism.

Attributed to: This term in ICOP is in accordance with ICHD-3 (2) and describes the relationship between a secondary pain (qv) and the disorder believed to cause it. It requires fulfilment of criteria establishing an accepted level of evidence of causation.

Central neuropathic pain: Neuropathic pain (qv) caused by a lesion or disease of the central somatosensory nervous system.

Central sensitization: Sensitization (qv) involving nociceptive neurons in the central nervous system.

Chronic: In pain terminology, *chronic* signifies long-lasting, specifically over a period exceeding 3 months. In headache terminology, for primary headache disorders that are more usually episodic (qv), *chronic* is used whenever headache (qv) occurs on more days than not over a period longer than 3 months. The trigeminal autonomic cephalalgias are the exception: in these disorders, *chronic* is not used until the disorder has been unremitting for more than 1 year with less than 3 months attack-free. How applicable these definitions are to orofacial pain will be established through research based on ICOP (3).

Duration of attack: Time from onset until termination of an attack of pain (qv) meeting criteria for a particular pain type or subtype. If the patient falls asleep during an attack and wakes up relieved, duration is reckoned until time of awakening.

Dysaesthesia: An unpleasant, abnormal sensation, whether spontaneous or evoked. Special cases of dysaesthesia include hyperalgesia (qv) and allodynia (qv). A dysaesthesia should always be unpleasant, in contrast to a paraesthesia (qv), although it is recognized that the borderline may present some difficulties.

Episodic: Recurring and remitting in a regular or irregular pattern of attacks of pain (qv) of constant or variable duration. Through long usage the term has acquired special meaning in the context of episodic cluster headache, referring to the occurrence of cluster periods separated by cluster remission periods rather than to attacks, and similar usage has been adopted for subtypes of 5.3 *Trigeminal autonomic orofacial pain*.

Facial pain: Pain (qv) below the orbitomeatal line, anterior to the pinnae and above the neck.

Frequency of attacks: The rate of occurrence of attacks or of pain (qv) per time period (commonly 1 month).

Headache: Pain (qv) located in the head, above the orbitomeatal line and/or nuchal ridge.

Hypaesthesia: Decreased sensitivity to stimulation, excluding the special senses. The stimulus and locus should be specified.

Hypalgesia: Reduced pain (qv) from a stimulus that normally provokes pain. The term is clinical and does not imply a mechanism.

Hyperaesthesia: Increased sensitivity to stimulation, excluding the special senses. The stimulus and locus should be specified. *Hyperaesthesia* may refer to various modes of cutaneous sensibility, including touch and thermal sensation without pain, as well as to pain, and is used to indicate both diminished threshold to any stimulus and an increased response to stimuli that are normally recognized.

Hyperalgesia: Increased pain (qv) from a stimulus that normally provokes pain. The term reflects increased pain on suprathreshold stimulation, and is a clinical term that does not imply a mechanism. It contrasts with pain evoked by stimuli that usually are not painful, for which *allodynia* (qv) is preferred.

Intensity (of pain): Level of pain (qv). It may be scored on a four-point numerical rating scale (0–3) equivalent to no, mild, moderate and severe pain, or on a visual analogue scale (commonly 10 cm). It may also be scored on a verbal rating scale expressed either on a scale from 0 to 10, or in terms of its functional consequence: 0, no pain; 1, mild pain, does not interfere with usual activities; 2, moderate pain, inhibits but does not wholly prevent usual activities; 3, severe pain, prevents all activities.

Neuralgia: Pain (qv) in the distribution of a nerve or nerves. Common usage, especially in Europe, often implies a paroxysmal quality, but the term should not be reserved for paroxysmal pains.

Neuritis: Inflammation of a nerve or nerves.

Neuropathic pain: Pain (qv) caused by a lesion or disease of the somatosensory nervous system. The term is a clinical description: when employed within a diagnosis, it requires a demonstrable lesion or disease that satisfies established neurological diagnostic criteria.

Neuropathy: A disturbance of function or pathological change in a nerve. Neuritis (qv) is a special case of neuropathy, the term being reserved for inflammatory processes affecting nerves.

Nociception: The neural process of encoding noxious stimuli.

Nociceptive pain: Pain (qv) that arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors (qv). This term, used to describe pain occurring with a normally functioning somatosensory nervous system, is designed to contrast with the abnormal function seen in neuropathic pain (qv).

Nociceptor: A high-threshold sensory receptor of the peripheral somatosensory nervous system that is capable of transducing and encoding noxious stimuli.

Nociplastic pain: Pain (qv) that arises from altered nociception (qv) despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors, or evidence for disease or lesion of the somatosensory system causing the pain.

Noxious stimulus: A stimulus that is damaging or threatens damage to normal tissues.

Pain: An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.

Paraesthesia: An abnormal sensation, whether spontaneous or evoked. It is recommended that *paraesthesia* be used to describe an abnormal sensation that is not unpleasant and *dysaesthesia* (qv) for an abnormal sensation that is considered to be unpleasant.

Peripheral neuropathic pain: Neuropathic pain (qv) caused by a lesion or disease of the peripheral somatosensory nervous system.

Peripheral sensitization: Sensitization (qv) involving nociceptive neurons in the periphery.

Persistent: This term, when used by ICHD in the context of certain secondary headaches, describes headache, initially acute and caused by another disorder, that fails to remit within a specified time interval (usually 3 months) after that disorder has resolved. In many such cases, this headache is recognized as a distinct subtype or subform, with evidence of causation depending upon earlier fulfilment of the criteria for diagnosis of the acute type, and persistence of the same headache. However, in ICOP, among the types of 6. *Idiopathic orofacial pain*, *persistent* has the meaning of *chronic* (qv), in the sense of being present or recurring over a period exceeding 3 months. This usage is also seen in ICHD.

Phonophobia: Hypersensitivity to sound, even at normal levels, usually causing avoidance.

Photophobia: Hypersensitivity to light, even at normal levels, usually causing avoidance.

Primary pain (disorder): Pain (qv) (orofacial pain or headache) not caused by or attributed to another disorder. It is distinguished from *secondary* pain disorder (qv).

Refractory period: The time following resolution of an attack of pain (qv) during which a further attack cannot be triggered.

Secondary pain (disorder): Pain (qv) (orofacial pain or headache) caused by or attributed to another disorder. It is distinguished from *primary* pain disorder (qv).

Sensitization: Increased responsiveness of nociceptive neurons to their normal input, and/or recruitment of a response to normally subthreshold inputs.

Unilateral: On either the right or the left side, not crossing the midline. When used for sensory or motor disturbances of migraine aura, the term includes complete or partial hemi-distribution.

References

1. International Association for the Study of Pain. IASP Terminology, https://www.iasp-pain.org/Education/Content.aspx?ItemNumber=1698 (2017, accessed January 2020).
2. Headache Classification Committee of the International Headache Society. The International Classification of Headache Disorders, 3rd edition (ICHD-3). *Cephalalgia* 2018; 38: 1–211.
3. Benoliel R, Eliav E and Sharav Y. Classification of chronic orofacial pain: applicability of chronic headache criteria. *Oral Surg* 2010; 110: 729–737.