Headache Medicine 2026, 17(3):217-225 ▪ p-ISSN 2178-7468, e-ISSN 2763-6178

Headache Medicine

DOI: 10.48208/HeadacheMed.2026.28

Review

Diagnostic integration of ICHD-3 and ICOP

Marieli Martelli

Unyleya Editora e Cursos S.A., Brasília, Distrito Federal, Brazil

Marieli Martelli
marielimartelliodonto@gmail.com

Edited by:
Eduardo Grossmann

Keywords:
Headache disorders
Orofacial pain
Persistent orofacial pain
ICHD-3
ICOP
Differential diagnosis

Abstract

Persistent headache and orofacial pain disorders often present overlapping clinical manifestations, shared trigeminal pathways, and common mechanisms of peripheral and central sensitization, making differential diagnosis challenging. Although the International Classification of Headache Disorders, 3rd edition (ICHD-3), and the International Classification of Orofacial Pain (ICOP) provide complementary diagnostic frameworks, their combined application in clinical practice remains underexplored. This narrative review critically examines how the complementary use of the ICHD-3 and ICOP can contribute to the differential diagnosis of patients with headache, facial pain, or intraoral pain. A literature search was conducted across PubMed/MEDLINE, Scopus, Embase, and Web of Science databases, prioritizing international classification documents, consensus statements, clinical guidelines, systematic reviews, and high-quality narrative reviews. Available evidence demonstrates substantial overlap between headache disorders and persistent orofacial pain, mediated by shared trigeminal mechanisms, neuroinflammation, and altered pain modulation. However, differences in pain distribution, temporal profile, somatosensory findings, autonomic manifestations, and the relationship between symptom onset and trauma or dental procedures remain fundamental to differential diagnosis. The complementary application of the ICHD-3 and ICOP offers a broader framework for assessing patients with overlapping craniofacial pain presentations and may support more consistent diagnosis and multidisciplinary management. Nevertheless, prospective studies are needed to determine whether this integrated diagnostic approach improves diagnostic agreement, clinical decision-making, and patient outcomes.

Received: May 25, 2026
Revised: July 28, 2026
Accepted: September 9, 2026

© Copyright 2026

Introduction

Headache disorders and persistent orofacial pain are among the most prevalent and disabling pain conditions encountered in neurological, dental, and orofacial pain practice (1). Both are associated with substantial impairment in quality of life, functional capacity, work productivity, and psychosocial well-being. Although traditionally managed within different clinical disciplines, these conditions frequently coexist or present with overlapping clinical manifestations, making differential diagnosis particularly challenging (2).

The ICHD-3, provides standardized diagnostic criteria for primary and secondary headache disorders, whereas the ICOP establishes a structured framework for the diagnosis of orofacial pain conditions, including neuropathic, idiopathic, and secondary disorders (3–5). Individually, both classifications have substantially improved diagnostic consistency within their respective fields. However, neither classification was designed to comprehensively address the full spectrum of patients presenting with overlapping headache, facial pain, and intraoral pain.

The diagnostic interface between these conditions is clinically relevant. Migraine may occasionally present predominantly as facial pain, and trigeminal autonomic cephalalgias may involve orbital, maxillary, or other facial regions. Conversely, persistent dentoalveolar pain, painful post-traumatic trigeminal neuropathy, and burning mouth syndrome may be interpreted as odontogenic disorders or, in some circumstances, as headache disorders when characteristic clinical and somatosensory findings are not adequately assessed (6–8).

Headache and persistent orofacial pain also share important biological mechanisms, including trigeminal nociceptive convergence, peripheral and central sensitization, neuroinflammatory processes, and altered pain modulation (1,7). Nevertheless, common pathophysiological mechanisms do not imply identical diagnoses or therapeutic approaches. Accurate diagnosis depends on careful evaluation of pain location, temporal profile, attack duration, autonomic manifestations, somatosensory abnormalities, and the temporal relationship between symptom onset and trauma, systemic disease, or dental procedures.

Accordingly, the complementary application of ICHD-3 and ICOP may provide a broader framework for evaluating patients with complex craniofacial pain. Rather than treating headache and orofacial pain classifications as competing systems, their integration may facilitate recognition of overlapping phenotypes and help clinicians identify the dominant diagnostic mechanism.

The objective of this narrative review was therefore to examine how the complementary application of ICHD-3 and ICOP may contribute to the differential diagnosis of patients presenting with headache, facial pain, or intraoral pain, with particular emphasis on clinical overlap, diagnostic distinctions, and practical implications for multidisciplinary assessment.

The specific question addressed by this review is: How can the complementary application of the ICHD-3 and the ICOP contribute to the differential diagnosis of patients presenting with headache, facial pain, or intraoral pain?

Methods

This study was designed as a directed narrative review addressing the diagnostic interface between the ICHD-3, and the ICOP, with emphasis on neuropathic and persistent orofacial pain conditions and their differential diagnosis with headache disorders.

A literature search was conducted in PubMed/MEDLINE, Scopus, Embase, and Web of Science. The search covered publications from 1 February 2018 to 3 March 2026. The principal search concepts included headache disorders, migraine, orofacial pain, neuropathic pain, trigeminal neuralgia, painful post-traumatic trigeminal neuropathy, persistent idiopathic dentoalveolar pain, burning mouth syndrome, ICHD-3, ICOP, diagnostic criteria, classification, and differential diagnosis. Boolean operators (AND/OR) were used, and database-specific search strategies were adapted to the corresponding controlled vocabulary and indexing systems.

Because the purpose of this review was to integrate diagnostic classifications and critically interpret clinically relevant evidence rather than to estimate pooled effects, a formal systematic-review or meta-analytic methodology was not applied. The search and selection process was nevertheless made explicit to improve transparency and reproducibility. The methodological framework is summarized in Table 1.

Table 1. Methodological framework of the directed narrative review

Domain	Methodological approach
Review design	Directed narrative review focused on the diagnostic interface between ICHD-3 and ICOP.
Additional searching	Reference lists of ICHD-3, ICOP, guidelines, consensus documents, systematic reviews, and key narrative reviews
Inclusion	Publications relevant to classification, diagnosis, clinical phenotype, differential diagnosis, somatosensory findings, trauma/dental procedures, or headache–orofacial pain overlap
Exclusion	Irrelevant conditions, publications without diagnostic relevance, nonclinical studies, conference abstracts, editorials, letters, and duplicates
Selection	Title/abstract screening followed by full-text assessment
Duplicate management	Manual verification
Data extraction	Clinical phenotype, temporal profile, sensory findings, diagnostic criteria, investigations, differential diagnosis, and limitations
Evidence appraisal	Qualitative consideration of methodological quality and evidence consistency; no uniform GRADE assessment
Synthesis	Qualitative narrative synthesis organized around diagnostic integration

Diagnostic integration between ICHD-3 and ICOP

The ICHD-3 and the ICOP were developed to standardize the diagnosis of headache disorders and orofacial pain conditions, respectively (3,4). Although each classification is internally comprehensive, neither fully encompasses the broad spectrum of patients presenting with overlapping headache, facial pain, and intraoral pain. Consequently, the complementary application of both classification systems may provide a more consistent diagnostic framework for complex craniofacial pain presentations.

The ICHD-3 primarily classifies primary and secondary headache disorders according to pain characteristics, temporal profile, associated symptoms, and underlying etiology (3). In contrast, the ICOP focuses on pain affecting the oral cavity, jaws, and face, emphasizing anatomical distribution, somatosensory abnormalities, and mechanisms underlying persistent orofacial pain (4). Because these classifications were developed for different clinical purposes, patients presenting with facial or intraoral pain may fulfill diagnostic criteria within one classification while requiring complementary evaluation using the other.

Diagnostic overlap is particularly evident in disorders involving the trigeminal system (3). Migraine may occasionally present predominantly as facial pain, while trigeminal autonomic cephalalgias frequently involve the orbital, maxillary, or mandibular regions (3). Conversely, painful post-traumatic trigeminal neuropathy, persistent idiopathic dentoalveolar pain, and burning mouth syndrome may initially be interpreted as primary headache disorders or odontogenic conditions because of overlapping pain location and symptom characteristics (6). Accurate diagnosis therefore depends not only on pain location but also on attack duration, temporal evolution, associated autonomic manifestations, sensory abnormalities, and the relationship between symptom onset and trauma, systemic disease, or dental procedures.

The complementary use of ICHD-3 and ICOP is particularly valuable in patients with persistent facial pain of uncertain origin. The ICHD-3 facilitates recognition of primary headache disorders, secondary headaches, and cranial neuralgias, whereas the ICOP provides greater diagnostic specificity for persistent dentoalveolar pain, post-traumatic trigeminal neuropathy, burning mouth syndrome, and other persistent orofacial pain disorders (3,4). Rather than representing competing systems, both classifications should be viewed as complementary tools that address different aspects of craniofacial pain assessment.

An integrated diagnostic approach should begin with characterization of the principal pain phenotype, including anatomical distribution, temporal pattern, attack duration, pain quality, triggering factors, and associated symptoms (7). The presence of ipsilateral cranial autonomic signs should prompt consideration of trigeminal autonomic cephalalgias, whereas sensory gain or loss detected during neurological examination favors trigeminal neuropathic pain over primary headache disorders (3,4). Likewise, a clear temporal relationship between pain onset and dental procedures, facial trauma, or neurological disease strongly supports secondary headache or painful post-traumatic trigeminal neuropathy, depending on the predominant clinical presentation.

Differentiating neuropathic from idiopathic orofacial pain also remains essential during diagnostic assessment. Neuropathic pain is supported by a history of lesion or disease affecting the somatosensory system together with compatible sensory abnormalities, whereas persistent idiopathic orofacial pain is characterized by chronic pain without demonstrable structural abnormalities despite comprehensive clinical evaluation (4,7). Confusing these entities may lead to inappropriate pharmacological treatment, repeated dental procedures, and delayed referral to appropriate specialists.

The integration of ICHD-3 and ICOP is particularly relevant in a multidisciplinary context, where the simultaneous application of both classifications can improve communication between specialties, facilitate diagnostic consistency, and reduce unnecessary investigations and irreversible dental interventions, while also fostering individualized management based on the predominant pain mechanism rather than solely on anatomical location.

Although current evidence supporting combined use of ICHD-3 and ICOP is largely based on expert consensus and pathophysiological rationale, their complementary application represents a clinically useful strategy for approaching patients with complex craniofacial pain presentations. Future prospective studies are needed to determine whether integrated classification improves diagnostic agreement, clinical outcomes, and therapeutic decision-making compared with the isolated use of either classification system. Table 2 summarizes the complementary contributions of the ICHD-3 and ICOP to the differential diagnosis.

Table 2. Integrated diagnostic approach combining ICHD-3 and ICOP in patients presenting with headache, facial pain, or intraoral pain

Clinical presentation	ICHD-3 category	ICOP category	Distinguishing clinical features	Recommended investigation	Frequent diagnostic error
Unilateral pulsating headache with nausea, photophobia, or aura	Migraine	Consider facial migraine if pain extends to the face	Attacks last 4–72 hours; sensory hypersensitivity; may worsen with physical activity	Clinical diagnosis according to ICHD-3; neuroimaging only when red flags are present	Misdiagnosis as sinusitis, temporomandibular disorder, or odontogenic pain
Strictly unilateral orbital or facial pain with ipsilateral autonomic signs	Trigeminal autonomic cephalalgias	Differential diagnosis with facial pain disorders	Lacrimation, conjunctival injection, rhinorrhea, restlessness, short-lasting attacks	Clinical diagnosis; neuroimaging to exclude structural lesions	Misdiagnosis as dental pain or maxillary sinus disease
Brief electric shock-like pain triggered by touch or chewing	Classical trigeminal neuralgia	Trigeminal neuralgia	Trigger zones, paroxysmal attacks, pain-free intervals, minimal neurological deficit	MRI to exclude secondary causes; neurological examination	Unnecessary endodontic treatment or tooth extraction
Persistent facial pain after dental treatment or facial trauma	Headache attributed to trauma (when criteria are fulfilled)	Painful post-traumatic trigeminal neuropathy	History of nerve injury with positive or negative sensory signs	Comprehensive neurosensory examination; imaging when indicated	Pain attributed solely to dental failure, leading to repeated procedures
Persistent dentoalveolar pain without identifiable dental pathology	Consider secondary headache only if clinical criteria are met	Persistent idiopathic dentoalveolar pain	Persistent intraoral pain without structural abnormalities; diagnosis of exclusion	Clinical and radiographic evaluation to exclude odontogenic disease	Repeated root canal therapy, apical surgery, or extraction without benefit
Chronic burning sensation of the oral mucosa with normal clinical examination	Usually not classified as a primary headache	Burning mouth syndrome	Burning pain, dysgeusia, xerostomia, normal oral mucosa	Exclusion of local and systemic causes	Misdiagnosis as candidiasis, nutritional deficiency, or allergy without adequate investigation
Sudden severe headache associated with neurological deficits or systemic symptoms	Secondary headache	Secondary orofacial pain if facial symptoms coexist	Thunderclap headache, fever, altered consciousness, focal neurological signs	Emergency neuroimaging and investigation of secondary causes	Delayed recognition of potentially life-threatening disorders

Primary headache disorders

Migraine is a primary recurrent headache characterized by unilateral, throbbing or pulsating pain of moderate to severe intensity, lasting between 4 and 72 hours, frequently associated with nausea, vomiting, photophobia, phonophobia, and, in some cases, reversible visual aura. During attacks, many patients require rest in quiet and dark environments due to intense sensory hypersensitivity. Its pathophysiology involves activation of the trigeminovascular system, sensitization of meningeal nociceptors, and release of inflammatory neuropeptides. Clinically, migraine can present with prodromal, aura, headache, and postdromal phases (3). The aura manifests mainly through reversible visual symptoms, such as scintillating scotomas, preceding or accompanying the pain. According to the ICHD-3, it is divided into forms with and without aura. Furthermore, it presents significant pathophysiological overlap with orofacial neuropathic pain, sharing mechanisms of central sensitization and trigeminal hypersensitivity.

Tension-type headache is the most prevalent primary headache and one of the most common neurological disorders in the world population. It is characterized by recurrent episodes of bilateral pain, described as pressure or tightness, of mild to moderate intensity, non-pulsating, lasting from 30 minutes to seven days (3). Depending on the frequency of attacks, it can be classified as infrequent episodic, frequent episodic, and chronic, the latter defined by occurrence on 15 or more days per month for at least three months. Its pathophysiology is multifactorial, involving genetic predisposition, environmental factors, central sensitization, and increased sensitivity of myofascial nociceptors. Alterations in descending pain modulation mechanisms, particularly in diffuse noxious inhibitory controls, play a relevant role in the chronicity of the disease (1). Diagnosis is essentially clinical and based on ICHD-3 criteria, requiring that attacks present at least two of the following characteristics: bilateral location, pressure or squeezing pain, mild to moderate intensity, and absence of worsening with routine physical activities. Photophobia, phonophobia, and nausea, when present, are usually mild (3).

Cluster headaches belong to the group of trigeminal-autonomic cephalalgias and are characterized by recurrent attacks of extremely intense unilateral pain, usually located in the orbital, temporal, or facial region, associated with ipsilateral autonomic signs such as lacrimation, conjunctival hyperemia, rhinorrhea, and facial flushing (8). They predominantly affect men between 20 and 40 years of age and present a pathophysiology related to the activation of the trigeminovascular system and the cranial autonomic system. According to the ICHD-3, the attacks have a cyclical pattern, with periods of exacerbation that can last weeks or months, followed by prolonged remissions. Attacks last from 15 to 180 minutes and can occur up to eight times a day. Unlike migraine, patients frequently present psychomotor restlessness during attacks (2).

Although they share similar pathophysiological mechanisms, migraine, tension-type headache, and cluster headache present important clinical differences related to intensity, pain location, and associated symptoms, as summarized in Table 3.

Table 3. Clinical differences between migraine, tension-type headache, and cluster headache

Feature	Migraine	Tension-type headache	Cluster headache
Typical pain location	Usually unilateral, may alternate sides	Usually bilateral	Strictly unilateral, orbital, temporal, or facial
Pain quality	Pulsatile or throbbing	Pressing or tightening	Severe, excruciating
Intensity	Moderate to severe	Mild to moderate	Severe to very severe
Duration	4 to 72 hours	30 minutes to 7 days	15 to 180 minutes
Associated symptoms	Nausea, vomiting, photophobia, phonophobia, sometimes aura	Photophobia or phonophobia may occur, usually mild; nausea uncommon	Ipsilateral autonomic signs such as lacrimation, conjunctival injection, rhinorrhea, facial flushing; restlessness is common
Effect of physical activity	Often worsens pain	Usually not aggravated by routine activity	Patients are often unable to remain still
Usual therapeutic emphasis	Acute treatment plus prevention when frequent	Analgesics for attacks; prophylaxis when frequent or chronic	Oxygen and abortive therapy; prophylaxis with verapamil or other preventive strategies

Trigeminal neuralgia represents the most classic form of facial neuropathic pain. It is characterized by recurrent episodes of intense unilateral pain, described as an electric shock, distributed along one or more branches of the trigeminal nerve. Historically called tic douloureux, due to the facial spasms frequently observed during attacks, it predominantly affects women between the fifth and sixth decades of life (7). The pain usually involves the maxillary and mandibular branches and can be triggered by seemingly harmless everyday stimuli (2). According to the ICHD-3 and ICOP, TN can be classified into classic, idiopathic, and secondary forms. The classic form is frequently related to neurovascular compression of the trigeminal root, usually by the superior cerebellar artery, while the secondary form results from structural conditions such as intracranial tumors or multiple sclerosis. Histopathological studies demonstrate focal demyelination and axonal reorganization, favoring neural hyperexcitability and ephatic transmission between adjacent sensory fibers (7). These mechanisms support the so-called "ignition hypothesis", in which spontaneous ectopic discharges recruit neighboring fibers, producing the characteristic paroxysmal painful episodes of the disease (9).

Glossopharyngeal neuralgia is a rare cranial neuropathy characterized by brief episodes of intense, lancinating unilateral pain involving the tonsillar region, base of the tongue, pharynx, ear, and mandibular angle (10). Attacks have an abrupt onset and can be triggered by swallowing, speaking, coughing, or yawning. The main described etiologies include vascular compression, Eagle syndrome, stylohyoid ligament calcification, multiple sclerosis, and intracranial expansive lesions (11). In severe cases, there may be associated vagal involvement, resulting in bradycardia, syncope, or seizures, configuring vagoglossopharyngeal neuralgia (10).

Post-traumatic trigeminal painful neuropathy is defined by the ICOP as persistent facial or oral pain, unilateral or bilateral, lasting more than three months after trigeminal nerve injury, accompanied by somatosensory changes. It can occur after major craniofacial trauma or relatively simple dental procedures, often related to iatrogenic injuries. Although most trigeminal lesions do not progress to chronic pain, some patients develop persistent neuropathic symptoms associated with a significant reduction in quality of life, anxiety, depression, and catastrophizing of pain. Its pathophysiology involves peripheral and central mechanisms, including neural sensitization, perineural inflammation, and somatosensory cortical reorganization (12). Clinically, patients report burning, shock-like, or stabbing pain, continuous or intermittent, spontaneous or triggered by tactile stimuli. Sensory changes, such as hypoesthesia, hyperalgesia, thermal changes, and foreign body sensation, are frequently observed (4,12).

Persistent idiopathic orofacial pain disorders

Idiopathic persistent dentoalveolar pain is characterized by persistent intraoral pain, usually unilateral, recurring for more than two hours daily for a period exceeding three months, without an identifiable etiological cause (3). Its clinical manifestations are heterogeneous, including burning, throbbing, tingling, pressure, or dysesthesia, frequently without detectable alterations on conventional clinical or radiographic examination. The diagnosis is essentially one of exclusion, requiring careful ruling out of odontogenic and structural causes (12).

Burning mouth syndrome is a chronic orofacial neuropathic condition characterized by a persistent burning sensation or dysesthesia of the oral mucosa for more than two hours daily for a period exceeding three months, in the absence of detectable clinical or laboratory changes. It predominantly affects women in the peri- and post-menopausal period, with the tongue being the most frequently affected site. In addition to burning, subjective xerostomia, dysgeusia, tingling, itching, and a foreign body sensation are common. Symptoms tend to intensify throughout the day and may experience transient relief after eating or drinking. Current evidence suggests a multifactorial etiopathogenesis involving trigeminal small fiber neuropathy, central sensitization, and psychological factors. Burning spells are frequently associated with systemic manifestations such as tinnitus, dizziness, fibromyalgia, chronic fatigue, and mood disorders (13).

Secondary headache disorders relevant to orofacial pain

Headache attributed to cranial or cervical trauma is one of the most prevalent secondary headaches. When it occurs within the first three months after the trauma, it is classified as acute; after this period, it is considered persistent (3). Its clinical presentation is nonspecific and frequently mimics migraine or tension-type headache, making a temporal relationship with the traumatic event essential for diagnosis. In addition to headache, patients may present with fatigue, dizziness, cognitive disturbances, sleep disturbances, irritability, anxiety, and memory deficits, comprising the post-concussion syndrome (4). Its pathophysiology is multifactorial, involving diffuse axonal injury, cerebral metabolic and hemodynamic alterations, neuroinflammation, and genetic and psychological factors (5).

Headaches related to cranial and cervical vascular diseases are of great clinical relevance due to the potential risk of morbidity and mortality. The presentation can vary from thunderclap headache, typical of subarachnoid hemorrhage, to nonspecific patterns similar to primary headaches (14). In some situations, headache may be the only symptom of serious conditions such as vasculitis and arterial dissections (6).

The extensive convergence of nociceptive pathways involving the head, neck, and craniofacial structures explains the frequent association between headaches and ophthalmological, otological, temporomandibular, and rhinosinusal disorders (3). Headaches related to temporomandibular disorders constitute an important cause of facial pain that is frequently underdiagnosed, while headaches associated with rhinosinusitis can mimic or trigger primary headaches, particularly migraine (4,15). Furthermore, the association between headaches and psychiatric disorders is widely recognized, influencing the pathophysiology, clinical course, and therapeutic response, which reinforces the need for a multidisciplinary and integrated approach to these patients (6). The principal clinical features distinguishing migraine, tension-type headache, and cluster headache are summarized in Table 4.

Table 4. Differential diagnostic features

Feature	Migraine	Tension-type headache	Cluster headache
Feature	Trigeminal neuralgia	Painful post-traumatic trigeminal neuropathy	Persistent idiopathic dentoalveolar pain
Onset	Sudden, paroxysmal	After identifiable trigeminal injury	Insidious or recurrent, without identifiable cause
Pain quality	Electric-shock-like, lancinating	Burning, shooting, aching, or mixed	Deep, persistent, dull, burning, or fluctuating
Temporal pattern	Brief attacks with pain-free intervals	Continuous pain with or without flares	Persistent daily pain, often with variable intensity
Triggering factors	Light touch, chewing, speaking, brushing	May be provoked by touch or local stimulation	Often no reproducible trigger; symptoms may persist despite treatment
Sensory findings	Usually absent or limited	Sensory gain or loss is common	Usually no clear sensory deficit on routine exam
Diagnostic clue	Paroxysmal facial pain with trigger zones	Chronic pain after nerve injury or dental/facial procedure	Persistent unexplained dentoalveolar pain after exclusion of odontogenic disease

Discussion

This review reinforces that the ICHD-3 and ICOP should be considered complementary rather than competing diagnostic frameworks. The ICHD-3 offers a comprehensive approach to primary and secondary headaches, whereas the ICOP provides greater specificity for persistent orofacial pain conditions, including neuropathic and idiopathic disorders (3,4).

Relying exclusively on either classification may leave diagnostic gaps in patients with overlapping presentations. Primary headaches can manifest predominantly as facial pain, while persistent orofacial conditions can mimic odontogenic diseases or headache syndromes. Thus, pain location alone is insufficient to establish a diagnosis (16).

In this context, the integrated application of the ICHD-3 and ICOP facilitates a more comprehensive assessment, taking into account phenotype, anatomical distribution, temporality, autonomic manifestations, somatosensory findings, and the relationship with trauma, dental procedures, or other underlying conditions (17,18). This approach can contribute to a more consistent diagnosis and improved interdisciplinary communication regarding patients with pain in the craniofacial complex. However, its clinical effectiveness still requires validation through prospective studies.

Clinical implications

Proper differentiation between headaches and persistent orofacial pain is crucial to avoid diagnostic delays, inappropriate treatments, and unnecessary dental procedures. The complementary application of the ICHD-3 and ICOP fosters a more comprehensive diagnostic approach by integrating neurological and orofacial characteristics and facilitating interdisciplinary communication. This approach can contribute to the early recognition of headaches, cranial neuralgias, and neuropathic or idiopathic conditions, thereby reducing invasive dental interventions in the absence of evidence of odontogenic disease. Although these benefits are clinically plausible, their effectiveness remains to be confirmed by prospective clinical studies.

Limitations of the evidence, review and future perspectives

Evidence supporting the integration of ICHD-3 and ICOP is still limited. The available literature is predominantly composed of international classifications, consensus documents, narrative reviews, observational studies and pathophysiological investigations, with a lack of prospective diagnostic accuracy studies that directly evaluate the complementary application of the two systems. Thus, although this approach is consistent with current knowledge and clinical reasoning, there is still no robust evidence demonstrating improved diagnostic accuracy or clinical outcomes.

The main strength of this review is the analysis of the interface between headaches and persistent orofacial pain, integrating perspectives traditionally approached independently in the areas of neurology and dentistry. As a limitation, this is a narrative review, without systematic selection, formal assessment of methodological quality or quantitative synthesis. Therefore, their conclusions should be interpreted as a critical synthesis of available

knowledge rather than as definitive evidence of clinical efficacy. Furthermore, the evolution of classification criteria and evidence may require future updates.

Prospective, multicenter studies should compare the isolated and integrated application of ICHD-3 and ICOP, evaluating diagnostic accuracy and agreement, interobserver reliability and clinical outcomes. They should also investigate whether integrated approaches reduce diagnostic delays, unnecessary dental procedures and inadequate complementary exams. The validation of standardized clinical flows could strengthen diagnostic consistency and interdisciplinary communication.

Conclusion

Distinguishing between headaches and persistent orofacial pain remains challenging due to clinical overlap and shared trigeminal mechanisms. Although developed from different diagnostic perspectives, the ICHD-3 and ICOP offer complementary insights: the former structures the classification of primary and secondary headaches, while the latter expands the characterization of orofacial pain conditions, particularly neuropathic and idiopathic ones. Their integrated application supports an assessment based on phenotype, distribution, temporality, autonomic manifestations, and somatosensory alterations, as well as the relationship with trauma or dental procedures. This approach can improve diagnosis, interdisciplinary communication, and the prevention of unnecessary dental interventions. However, available evidence relies predominantly on classifications, consensus statements, reviews, and observational studies, precluding a definitive assessment of clinical impact. Prospective, multicenter studies are needed to determine whether this integration improves diagnostic agreement and accuracy, reduces delays and inappropriate interventions, and consequently improves patient outcomes.

References

1. Pak R, Ku J, Abd-Elsayed A. Neuromodulation for Craniofacial Pain and Headaches. Biomedicines 2023;11. Doi:10.3390/biomedicines11123328.
2. Ashina M, Hansen JM, Do TP, Melo-Carrillo A, Burstein R, Moskowitz MA. Migraine and the trigeminovascular system—40 years and counting. Lancet Neurol 2019;18:795–804. Doi:10.1016/S1474-4422(19)30185-1.
3. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. Cephalalgia 2018;38:1–211. Doi:10.1177/0333102417738202.
4. International Classification of Orofacial Pain, 1st edition (ICOP). Cephalalgia 2020;40:129–221. Doi:10.1177/0333102419893823.
5. Do TP, Remmers A, Schytz HW, Schankin C, Nelson SE, Obermann M, et al. Red and orange flags for secondary headaches in clinical practice. Neurology 2019;92:134–44. Doi:10.1212/WNL.0000000000006697.
6. Chou DE. Secondary Headache Syndromes. Continuum (N Y) 2018;24:1179–91. Doi:10.1212/CON.0000000000000640.
7. Bendtsen L, Zakrzewska JM, Heinskou TB, Hodaie M, Leal PRL, Nurmikko T, et al. Advances in diagnosis, classification, pathophysiology, and management of trigeminal neuralgia. Lancet Neurol 2020;19:784–96. Doi:10.1016/S1474-4422(20)30233-7.
8. Olivier MA, Contreras Martínez MA, Díaz Garza CA, Galnares Olalde JA, Leyva Rendon A, Martínez Corredor PA, et al. Trigeminal autonomic cephalalgias: current insights into pathophysiology, diagnosis, and therapeutic strategies. Headache Medicine 2026;17:7–19. Doi:10.48208/HeadacheMed.2026.1.
9. Zakrzewska JM, Linskey ME. Trigeminal neuralgia. BMJ 2014;348. Doi:10.1136/bmj.g474.
10. Han A, Montgomery C, Zamora A, Winder E, D. Kaye A, Carroll C, et al. Glossopharyngeal Neuralgia: Epidemiology, Risk factors, Pathophysiology, Differential diagnosis, and Treatment Options . Health Psychol Res 2022;10. Doi:10.52965/001c.36042.
11. Souza V, Passerini M, Sobral B, Baiardi V, Junior H. Clinical and physiopathological aspects of the glossopharyngeal neuralgia. Headache Medicine 2021;12:5–11. Doi:10.48208/HeadacheMed.2021.2.
12. Sanner F, Sonntag D, Hambrock N, Zehnder M. Patients with persistent idiopathic dentoalveolar pain in dental practice. Int Endod J 2022;55:231–9. Doi:10.1111/iej.13664.
13. Canfora F. Burning mouth syndrome: updates on pathogenesis and diagnostic algorithms. J Oral Facial Pain Headache 2025. Doi:10.22514/jofph.2025.064.
14. Malacarne A, Spierings ELH, Lu C, Maloney GE. Persistent Dentoalveolar Pain Disorder: A Comprehensive Review. J Endod 2018;44:206–11. Doi:10.1016/j.joen.2017.09.009.
15. San-Juan D, Velez-Jimenez K, Hoffmann J, Martínez-Mayorga AP, Melo-Carrillo A, Rodríguez-Leyva I, et al. Cluster headache: an update on clinical features, epidemiology, pathophysiology, diagnosis, and treatment. Frontiers in Pain Research 2024;5. Doi:10.3389/fpain.2024.1373528.
16. Nixdorf DR, Moana-Filho EJ, Law AS, McGuire LA, Hodges JS, John MT. Frequency of Persistent Tooth Pain after Root Canal Therapy: A Systematic Review and Meta-Analysis. J Endod 2010;36:224–30. Doi:10.1016/j.joen.2009.11.007.

17. Di Stefano G, Di Lionardo A, Di Pietro G, Truini A. Neuropathic Pain Related to Peripheral Neuropathies According to the IASP Grading System Criteria. Brain Sci 2020;11. Doi:10.3390/brainsci11010001.

18. Ferraro MC, Cashin AG, Wand BM, Smart KM, Berryman C, Marston L, et al. Interventions for treating pain and disability in adults with complex regional pain syndrome- an overview of systematic reviews. Cochrane Database of Systematic Reviews 2023;2023. Doi:10.1002/14651858.CD009416.pub3.

Marieli Martelli
https://orcid.org/0009-0003-9394-8895

Conflict of interest: The author declares no conflict of interest related to this work.
Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.