

Persistent Post-Endodontic Dental Pain and its Relationship to Phantom Limb-Like Neuropathic Pain: A Narrative Review

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Abstract

Persistent post-endodontic pain affects a clinically significant proportion of patients despite technically successful root canal treatment. This narrative review examines the biopsychosocial mechanisms underlying this condition through five interrelated domains: peripheral sensitization, central sensitization, neuroplasticity and cortical reorganization, psychosocial factors, and the clinical entities of persistent idiopathic dentoalveolar pain and painful post-traumatic trigeminal neuropathy as classified by the International Classification of Orofacial Pain 2020. A conceptual parallel with phantom limb pain is explored, highlighting shared mechanisms of deafferentation, ectopic discharge, and maladaptive cortical remapping, while acknowledging the anatomical and clinical differences inherent to the trigeminal system. Psychosocial factors including pain catastrophizing, anxiety, and depression are framed as perpetuating and amplifying elements rather than alternative diagnoses. Evidence-based pharmacological and non-pharmacological management strategies are reviewed. The review is intended for clinicians in dentistry, orofacial pain, and pain medicine seeking an integrative reference for this complex condition.

Keywords: Post-endodontic pain; phantom tooth pain; central sensitization; neuropathic pain; persistent idiopathic dentoalveolar pain.

INTRODUCTION

Persistent pain following root canal treatment constitutes a well-documented clinical challenge. Nixdorf et al. reported that approximately 10% of patients continue to experience pain six months after endodontic therapy and that prolonged preoperative pain significantly increases the risk of persistent postoperative pain, with affected patients also incurring higher rates of additional radiographs, retreatments, medical consultations, and analgesic use. [1,2] The prevalence of non-odontogenic tooth pain occurring one to six years after root canal treatment has been estimated at 3.1 to 3.4%, with proposed origins including referred pain from temporomandibular disorders, sinusitis, and neuropathic pain secondary to nerve injury, as well as persistent idiopathic dentoalveolar pain of undetermined origin. [3]

This clinical scenario poses a significant diagnostic and therapeutic challenge, as the management of persistent post-endodontic pain frequently requires coordinated input from multiple dental and medical specialties. Berge demonstrated that peripheral nerve or tissue damage may initiate a pain state that extends well beyond the acute injury phase. [3] Once this occurs, changes in the electrical properties of sensory nerves disrupt the balance between central excitatory and inhibitory systems, leading to heightened neural excitability and the emergence of complex chronic neuropathic pain. [4,5]

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Chronic neuropathic pain is characteristically associated with damage or injury to peripheral nerves, whether from trauma or surgical procedures. Although symptoms may appear immediately, onset can be delayed by days, months, or even years [6,7]. Clinically, the presentation is often described as a dull, deep, aching sensation, occasionally accompanied by episodes of sharp, paroxysmal pain. A particularly instructive manifestation of neuropathic pain is phantom limb pain, in which individuals experience painful sensations perceived to originate from an amputated limb. Phantom limb pain typically begins within days of amputation but may be delayed considerably; it can present as tingling, shooting, stabbing, throbbing, burning, aching, pinching, clamping, or vise-like squeezing sensations. [5] The pain itself is real, even though its perceived location corresponds to a limb that no longer exists. [6,8]

The neuromatrix theory proposes that the brain maintains an internal representation of the body even after amputation, and that a mismatch between this representation and the absence of peripheral input may trigger persistent pain [9]. Experimental studies have demonstrated that visual and sensory conflicts can alter body perception and induce sensations similar to phantom pain, suggesting that disrupted interactions among cortical representation, sensory feedback, and visual input contribute to the development of phantom limb pain. [6]

The biopsychosocial model, originally proposed by Engel, provided a foundational framework for understanding how biological, psychological, and social factors interact in the experience of illness. [7,10] This model has since been extended by patient-centered and narrative medicine approaches, which emphasize that the same clinical condition may carry substantially different meanings for different individuals depending on their personal history, expectations, and psychological context. Applied to persistent post-endodontic pain, this framework enables clinicians to move beyond a purely mechanistic view and appreciate the multi-layered nature of pain persistence.[8]

This review is organized around five interrelated mechanistic domains: peripheral sensitization, central sensitization and neuroplasticity, cortical reorganization, psychosocial factors, and phantom tooth pain as a distinct clinical entity [11,12]. This structure allows for a multi-layered understanding of pain that integrates peripheral neural processes, central nervous system adaptation, and the psychosocial context that shapes symptom perception and chronification, with particular attention to phantom tooth pain as the clinical manifestation emerging from these interacting systems. The review is intended to

provide clinicians, researchers, and students in dentistry and orofacial pain with a clear, integrative reference for understanding the mechanisms underlying persistent and phantom-like dental pain phenomena [11].

METHODS.

A structured literature search was conducted in major databases including PubMed/MEDLINE, Cochrane Library, Embase, and PsycINFO to ensure comprehensive coverage of the evidence on endodontic and neuropathic dental pain. The search was limited to studies published between January 2002 and June 2026 and restricted to English-language publications.

The search strategy combined MeSH terms and free-text keywords organized across key thematic domains, including endodontic pain, peripheral sensitization, central sensitization, neuroplasticity, cortical reorganization, and psychosocial factors, as well as clinical entities such as atypical odontalgia, phantom tooth pain, persistent dentoalveolar pain, painful post-traumatic trigeminal neuropathy, and persistent idiopathic dentoalveolar pain.

Inclusion criteria comprised randomized controlled trials, systematic reviews, meta-analyses, controlled clinical trials, and prospective cohort studies involving adult populations aged 18 years or older, with pain diagnoses classified according to ICOP 2020, IASP definitions, or RDC/TMD-compatible criteria. Exclusion criteria included case reports, editorials, animal studies, pediatric-only populations, and studies lacking standardized pain classification.

A narrative review design was selected given the broad, multi-level nature of the mechanistic domains addressed, allowing for an integrative clinical synthesis of heterogeneous evidence for a multidisciplinary audience.

PERIPHERAL SENSITIZATION IN POST-ENDODONTIC PAIN

According to the International Association for the Study of Pain, peripheral sensitization is defined as an increase in the responsiveness and a reduction in the activation threshold of peripheral nociceptors following tissue injury or inflammation. [8,12] Pulpal nociception is mediated by A δ and C trigeminal afferent fibers, which are responsible for the transmission of fast and slow pain respectively. These fibers respond to hydrodynamic, thermal, and chemical stimuli and, during pulpitis and apical periodontitis, exhibit a significant increase in excitability. [9]

This state of hyperexcitability is mediated by a neuroinflammatory microenvironment in which

prostaglandins, bradykinin, tumor necrosis factor- α , and neuropeptides such as substance P and calcitonin gene-related peptide participate. These mediators act on transient receptor potential channels and voltage-gated sodium channels, reducing the neuronal activation threshold and facilitating spontaneous activity of nociceptive fibers. [10, 11]

Following endodontic treatment, injury to trigeminal afferents may induce ectopic discharges originating in peripheral terminals or neuronal cell bodies within the trigeminal ganglion. This spontaneous activity is associated with dysfunction of sodium channel subtypes Nav1.7 and Nav1.8, which play a key role in the generation and maintenance of both inflammatory and neuropathic pain. [12,13] This irregular activity may sustain nociceptive input to second-order neurons in the trigeminal nucleus caudalis even in the absence of active inflammation or radiographic evidence of pathology, thereby explaining the persistence of pain in cases of technically successful endodontic treatment without an identifiable odontogenic source. [13]

Persistent post-endodontic pain should therefore be understood as a pathophysiological continuum in which peripheral sensitization and ectopic activity prolong afferent signaling beyond tissue repair, potentially facilitating the transition toward neuropathic pain states characterized by neuroplastic changes in trigeminal pathways. [14].

CENTRAL SENSITIZATION, NEUROPLASTICITY, AND CORTICAL REORGANIZATION.

Central sensitization is defined by the International Association for the Study of Pain as an increased responsiveness of nociceptive neurons within the central nervous system to normal or subthreshold sensory input. [15] In this state, pain-processing pathways become hyperexcitable, amplifying nociceptive signals and producing pain responses to stimuli that are normally non-painful or only mildly painful. Central sensitization is now recognized as a key mechanism underlying the transition from acute to chronic pain across a range of musculoskeletal and orofacial conditions [16].

A principal mechanism in central sensitization is the wind-up phenomenon within the trigeminal nucleus caudalis. Wind-up refers to a progressive increase in the excitability of wide dynamic range neurons following repetitive activation of primary afferent C-fibers, and it corresponds clinically to temporal summation, in which pain perception progressively intensifies during repetitive stimulation of identical intensity [17]. This

represents a form of activity-dependent neural plasticity that contributes to the development and maintenance of persistent facial pain. [16,18]

Synaptic plasticity is fundamental to the establishment of central sensitization. Persistent nociceptive input induces structural and functional changes that strengthen synaptic transmission through the activation of N-methyl-D-aspartate receptors, which increases calcium influx and triggers intracellular signaling cascades that enhance neuronal excitability [19]. These changes are mediated through long-term potentiation and long-term depression, which alter nociceptive processing and promote chronic pain states. [16,20] The descending pain regulatory system also plays a critical role; under normal conditions, descending inhibitory pathways suppress nociceptive transmission through the release of serotonin and norepinephrine, but dysfunction of these pathways, combined with reduced gamma-aminobutyric acid-mediated inhibition, increases neuronal excitability and facilitates pain persistence. [17, 18]

Within the trigeminal system, persistent nociceptive input can induce neuroplastic changes resulting in the expansion of receptive fields within the trigeminal subnucleus caudalis [21]. Neurons that originally responded to a specific tooth may begin responding to adjacent teeth or nearby facial structures, impairing pain localization and producing diffuse or referred orofacial pain. [18] Activation of astrocytes and microglia further promotes the release of pro-inflammatory mediators that enhance neuronal excitability and sustain pain transmission [22].

Neuroimaging studies have demonstrated reductions in gray matter volume in regions involved in pain processing and modulation, including the prefrontal cortex, thalamus, insula, and anterior cingulate cortex. [19] Altered activity within the amygdala and hippocampus further reinforces pain-related memory traces and emotional distress, contributing to pain persistence across time. [16,23]

Dental deafferentation following pulp extirpation provides a clinically relevant example of maladaptive neuroplasticity. The loss of pulpal sensory input can induce cortical remapping within the primary somatosensory cortex, allowing neighboring cortical representations to occupy the deafferented area. [20] This process closely resembles the mechanisms observed in phantom limb phenomena, where cortical reorganization following sensory loss contributes to persistent abnormal sensations and pain. [21, 22] Although cortical adaptation is normally compensatory, excessive reorganization may alter sensory perception and contribute to chronic pain despite resolution of the original peripheral injury [24].

The parallel between dental deafferentation and phantom limb pain is instructive but not complete. Both conditions involve deafferentation, ectopic discharge, and maladaptive plasticity; however, phantom limb pain involves the spinal somatosensory system and a far more extensive deafferentation, whereas dental deafferentation is limited to a small trigeminal territory [25]. The degree of cortical representation and the clinical reversibility of the reorganization also differ substantially. [21, 23] Neuroimaging studies confirm that chronic orofacial pain conditions, including temporomandibular disorders and trigeminal neuropathic pain, involve widespread alterations in brain structure, function, and connectivity that extend beyond peripheral pathology. [24, 25]

Once central sensitization becomes established, pain may persist even after complete tissue healing. As a direct clinical consequence, additional dental procedures frequently fail to provide relief, because the primary driver of pain is no longer peripheral but is instead maintained by maladaptive changes within central nociceptive pathways. [18, 26]

PSYCHOSOCIAL DIMENSIONS AND CHRONIC PAIN PERPETUATION.

The International Association for the Study of Pain describes pain as an unpleasant sensory and emotional experience associated with real or possible tissue damage, or sensations similar to such damage. This definition explicitly acknowledges the subjective and multidimensional nature of pain and forms the conceptual basis for understanding why persistent orofacial pain cannot be fully explained by biological mechanisms alone [26]. The biopsychosocial model provides a complementary framework for evaluating how biological, psychological, and social factors interact to initiate, perpetuate, or amplify pain. Within this model, the ICOP Axis II framework, analogous to the Axis II component of the Diagnostic Criteria for Temporomandibular Disorders, enables clinicians to systematically assess the psychosocial burden of patients presenting with chronic orofacial pain conditions. [27,28]

Pain catastrophizing, defined as a negative cognitive and emotional response involving rumination, magnification, and helplessness in the context of pain, has been consistently associated with greater perceived pain intensity, higher disability, and poorer treatment outcomes in persistent orofacial pain populations. [28] Anxiety is another well-established contributor: preoperative dental anxiety is a significant predictor of postoperative pain intensity following endodontic treatment, and patients with elevated anxiety levels report more severe and more prolonged pain experiences. [29,30]

The relationship between depression and chronic pain is bidirectional and neurobiologically grounded. Shared serotonergic and noradrenergic pathways underlie both mood regulation and descending pain inhibition, which helps explain why patients with depressive symptoms are disproportionately represented among those with persistent post-endodontic pain [30]. Somatization and high somatic symptom burden also constitute independent risk factors for the development of persistent dentoalveolar pain following endodontic treatment; patients who report multiple unexplained physical symptoms across organ systems are more likely to develop chronic pain after dental procedures. [31]

Prior chronic pain history is clinically one of the most significant risk factors. Patients with pre-existing conditions such as fibromyalgia, temporomandibular disorders, or headache disorders demonstrate heightened central sensitization at baseline, substantially increasing their vulnerability to persistent post-endodontic pain. [32] A cross-sectional study using ICOP diagnostic criteria confirmed that pain intensity in patients with orofacial pain is associated with reduced self-efficacy and mildly decreased quality of life, underscoring the importance of evaluating the psychosocial burden as part of routine assessment. [33]

In a meta-analysis examining pain modulation profiles in patients with temporomandibular disorders coexisting with migraine and fibromyalgia, the co-occurrence of these conditions was shown to amplify pain burden and complicate management, emphasizing the importance of a comprehensive and coordinated treatment approach. [34] The PHQ-4, a validated four-item screening instrument encompassing two depression items and two anxiety items, provides a practical and time-efficient option for preoperative psychosocial screening in dental settings. [35,36] Additional validated tools include the Pain Catastrophizing Scale and the Graded Chronic Pain Scale.

It is important to frame psychosocial factors as perpetuating and amplifying elements rather than as evidence that the patient's pain is imaginary or purely psychological [37]. These factors do not eliminate the biological basis of the pain experience; rather, they interact with peripheral and central mechanisms to influence the trajectory of pain over time. Clinicians who dismiss the psychosocial dimension risk both misdiagnosis and therapeutic failure. [27, 32]

PHANTOM TOOTH PAIN AND ATYPICAL ODONTALGIA: CLINICAL PRESENTATION, DIAGNOSIS, AND MANAGEMENT

Persistent pain after endodontic treatment may arise from the biological mechanisms and psychosocial factors

discussed in preceding sections [38,39]. Historically termed atypical odontalgia or phantom tooth pain, these conditions are now classified under the ICOP 2020 framework as persistent idiopathic dentoalveolar pain and painful post-traumatic trigeminal neuropathy. [39,40] This modern classification builds on early twentieth-century observations of persistent tooth pain without identifiable dental pathology, which Marbach subsequently linked in 1993 to deafferentation mechanisms analogous to those observed in phantom limb pain. [39, 41]

Persistent idiopathic dentoalveolar pain presents as dentoalveolar pain lasting longer than three months in the absence of clinical or radiographic evidence of local disease [42]. Painful post-traumatic trigeminal neuropathy, by contrast, arises from documented trigeminal nerve trauma and presents with somatosensory abnormalities such as allodynia, hyperalgesia, hypoesthesia, or paresthesia. [38, 42] Both conditions predominantly affect women aged 30 to 50 years and frequently co-occur with anxiety, depression, pain catastrophizing, or a history of repeated local dental procedures. [41, 43]

Patients typically describe a continuous or near-continuous dull, burning, deep, or pressing discomfort. Although initially localized to a specific tooth or area, the pain can radiate to adjacent structures over time. [43,44] A critical diagnostic feature is that local anesthetic blocks often yield inconsistent or only partial relief, which is a key finding that helps distinguish these neuropathic entities from conventional odontogenic pain. [44, 45]

Misdiagnosis is common and carries significant

consequences. Clinicians who do not recognize these entities frequently proceed with endodontic retreatment, additional procedures, or extractions, none of which provide relief and all of which may worsen the underlying neuropathy by adding further trigeminal injury. [46, 42] The differential diagnosis must therefore include cracked tooth syndrome, persistent periapical pathology, referred myofascial pain, neurovascular toothache, and burning mouth syndrome. [47]

When persistent idiopathic dentoalveolar pain or painful post-traumatic trigeminal neuropathy is confirmed, management requires a conservative and multidisciplinary approach [48]. Tricyclic antidepressants, particularly amitriptyline and nortriptyline, remain first-line pharmacological therapies based on available evidence. Serotonin-norepinephrine reuptake inhibitors, gabapentinoids including gabapentin and pregabalin, and topical capsaicin represent additional options for selected cases. [44,49] Non-pharmacological strategies, including patient education, reassurance, cognitive behavioral therapy, and the avoidance of further irreversible dental interventions, are equally important for achieving successful long-term outcomes. [46,50]

Early recognition of these conditions is essential to prevent unnecessary treatments and promote evidence-based care. The diagnostic framework provided by ICOP 2020, combined with an appreciation of the psychosocial and neurobiological factors described in earlier sections, enables a comprehensive and individualized clinical approach. [46]

Diagnostic Summary: PIDAP vs. PTTN

Table 1. *Diagnostic and clinical comparison of persistent idiopathic dentoalveolar pain (PIDAP) and painful post-traumatic trigeminal neuropathy (PTTN) according to ICOP 2020. [38, 42, 43]*

Feature	PIDAP	PTTN
Pain duration	More than 3 months	More than 3 months
Precipitating event	No identifiable cause; often follows dental procedure	Documented trigeminal nerve trauma
Somatosensory changes	Absent or minimal	Present (allodynia, hyperalgesia, hypoesthesia, paresthesia)
Response to local block	Partial or inconsistent relief	Partial or inconsistent relief
Demographics	Women aged 30-50; history of anxiety, depression, or multiple dental procedures	Women aged 30-50; similar psychosocial risk profile
ICOP 2020 basis	No clinical or radiographic odontogenic pathology	Pain arising from injury to trigeminal nerve or its branches
First-line treatment	TCAs, SNRIs, CBT, patient education	TCAs, gabapentinoids, SNRIs, topical capsaicin, CBT

CONCLUSION

Persistent post-endodontic dental pain is most accurately conceptualized as a multifactorial neuropathic pain condition rather than a residual inflammatory or purely mechanical problem. The five mechanistic domains reviewed in this paper converge on a unified model: peripheral sensitization initiates the process through nerve injury and neuroinflammation at the site of endodontic treatment; central sensitization perpetuates and amplifies nociceptive signaling within the trigeminal nucleus caudalis; maladaptive neuroplasticity and cortical reorganization sustain pain despite the resolution of peripheral pathology; psychosocial factors including catastrophizing, anxiety, and depression amplify the subjective experience and reinforce chronification; and the clinical result of this interacting cascade is the diagnostic entities described under ICOP 2020 as persistent idiopathic dentoalveolar pain and painful post-traumatic trigeminal neuropathy.

The phantom limb pain analogy is useful as a conceptual and communicative framework precisely because it highlights that pain can be real, disabling, and neurobiologically grounded even in the absence of identifiable peripheral pathology. Both phantom limb pain and persistent post-endodontic pain share mechanisms of deafferentation, ectopic discharge, and maladaptive cortical remapping. However, the analogy has important limitations that clinicians must bear in mind. Amputation produces extensive, permanent, and unambiguous deafferentation; root canal treatment produces a far more limited trigeminal deafferentation, and the degree of cortical representation and potential for reversal differ substantially between the two systems. Additionally, dental pain involves diagnostic uncertainty that phantom limb pain does not, because residual odontogenic pathology must always be excluded before a neuropathic diagnosis is assigned.

For the general dentist, the most immediate clinical implication of this review is the importance of recognizing psychosocial and central sensitization risk factors before initiating endodontic treatment. Preoperative screening using validated tools such as the PHQ-4, the Pain Catastrophizing Scale, and the Graded Chronic Pain Scale can identify patients at elevated risk of chronification. When a patient presents with persistent pain following technically successful treatment and no identifiable odontogenic cause, the clinician should resist the temptation to perform retreatment, further procedures, or extraction, as these interventions are unlikely to resolve centrally driven pain and may worsen the neuropathy. Clear referral pathways to orofacial pain specialists and pain medicine clinicians should be established before the

patient undergoes additional unnecessary interventions.

For the pain medicine clinician, this review underscores the importance of understanding the dental context in which these conditions develop. The ICOP 2020 classification provides a standardized diagnostic framework that bridges dental and medical pain medicine, and familiarity with its criteria for persistent idiopathic dentoalveolar pain and painful post-traumatic trigeminal neuropathy is essential. Pharmacological management with tricyclic antidepressants, serotonin-norepinephrine reuptake inhibitors, and gabapentinoids should be combined with cognitive behavioral therapy and patient education as part of a multidisciplinary strategy. Irreversible dental procedures should not be recommended for patients who present without clear evidence of active odontogenic pathology.

The current evidence base retains significant limitations. Prospective cohort studies using standardized ICOP-based outcome measures remain scarce, neuroimaging data specifically from patients with persistent post-endodontic pain are limited, and head-to-head randomized controlled trials comparing pharmacological interventions for persistent dentoalveolar pain have not yet been conducted. Existing studies also suffer from heterogeneous diagnostic criteria, particularly those predating ICOP 2020, and underrepresentation of diverse populations. Future research should prioritize prospective risk stratification studies using validated psychosocial screening tools, cortical neuroimaging studies in persistent dentoalveolar pain populations, and randomized controlled trials for combined pharmacological and behavioral interventions using ICOP 2020 diagnostic criteria. Development of validated patient-reported outcome measures specific to post-endodontic pain would also substantially advance the field.

This narrative review is subject to several inherent limitations. No pooled quantitative analysis was performed, selection bias in the literature reviewed cannot be excluded, the search was restricted to English-language publications, and older studies in this field employed heterogeneous diagnostic criteria that limit comparability. Nevertheless, the integrative framework presented here provides a clinically useful synthesis of the current understanding.

In conclusion, persistent post-endodontic pain should be understood as a neuropathic, biopsychosocial condition in which an initial dental injury may trigger a cascade of peripheral and central neuroplastic changes that are subsequently amplified by psychosocial factors. For the general dentist, the key takeaway is to screen

for neuropathic and psychosocial risk factors before treatment, avoid repeated irreversible procedures when symptoms suggest a centrally driven pain condition, and refer promptly to an orofacial pain specialist when the clinical picture does not fit a conventional odontogenic diagnosis. For the pain medicine clinician, the key takeaway is to integrate the dental history and ICOP 2020 diagnostic framework into the assessment and to approach management with a multidisciplinary strategy that combines pharmacological therapy with behavioral intervention.

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