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The Physiological Misunderstanding of Pain: Why Patients Perceive Pain during Exodontia Despite Profound Local Anaesthesia

Dr. Althaaf Khan
BDS (SMU), AssocFCGDent (UK)
Private Practice, South Africa

Corresponding Author

Abstract

Objective: The clinical observation that some patients report intra-operative pain during exodontia despite objective signs of profound local anaesthesia represents a persistent and often misunderstood phenomenon in everyday dental practice. This narrative review examines the physiological, pharmacological, anatomical and psychological bases of this discrepancy, with the aim of clarifying the distinction between a true failure of anaesthesia and a physiological misunderstanding of non-nociceptive sensation as pain.

Methodology: A narrative review of the peer-reviewed dental, anaesthesiology and pain-science literature was conducted. Sources were identified through targeted searches of biomedical databases and were synthesised under four physiological domains: (1) pharmacological and biochemical determinants of local anaesthetic efficacy in inflamed tissue; (2) anatomical variation and accessory or cross-innervation; (3) the neurophysiology of differential nerve fibre blockade and residual proprioception; and (4) the psychological and central modulation of pain perception, including anxiety, catastrophising and nocebo phenomena.

Major Findings: Profound local anaesthesia abolishes conduction in nociceptive Aδ and C fibres but does not, and is not intended to, abolish conduction in the larger, more resistant Aβ fibres that mediate pressure, tension, stretch and proprioception. Consequently, the mechanical forces of elevation, luxation and forceps application during exodontia continue to be transmitted to the central nervous system as pressure and movement. In patients with heightened anxiety, prior traumatic dental experience, pain catastrophising or nocebo expectation, this legitimate non-nociceptive afferent input is frequently misattributed and reported as pain, even when nociceptive transmission is fully blocked. True anaesthetic failure, driven by inflammatory acidosis, tetrodotoxin-resistant sodium channel upregulation, accessory or cross-innervation, and anatomical barriers to diffusion, must be differentiated from this physiological misunderstanding, as the two require fundamentally different clinical responses.

Conclusion: The perception of pain during exodontia under profound anaesthesia is frequently a physiological and psychological misunderstanding of preserved proprioceptive and pressure sensation rather than evidence of inadequate nociceptive blockade. Pre-operative communication that explicitly distinguishes pressure from pain, together with anxiety-reducing strategies and vigilant clinical differentiation from genuine anaesthetic failure, may reduce patient distress, improve informed consent, and refine clinical decision-making during extraction.

Keywords

Local Anaesthesia; Exodontia; Pain Perception; Proprioception; Dental Anxiety; Differential Nerve Blockade.

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1. Introduction

Profound local anaesthesia is the cornerstone of atraumatic exodontia. Yet a well-recognised and clinically disruptive scenario persists across dental practice: a patient with objectively confirmed soft-tissue anaesthesia (numb lip, absent response to probing, negative response to electric pulp testing where applicable) nonetheless reports pain, flinches, vocalises or withdraws during tooth elevation and luxation. This discordance between the clinician's objective assessment of anaesthetic depth and the patient's subjective report is a source of anxiety for both parties and, when misinterpreted, can lead to unnecessary re-injection, prolonged procedure time, escalating patient distress, and erosion of trust in the clinician [1].

The International Association for the Study of Pain (IASP) defines pain as an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage [2]. This definition is instructive for the present discussion: pain is not synonymous with nociception, and the subjective report of pain can arise even where classical nociceptive pathways are pharmacologically silenced. During exodontia, the mechanical forces required to expand the alveolar socket and disengage the periodontal ligament, namely pressure, tension, stretch and rotational movement, are transduced by a dense population of periodontal and periosteal mechanoreceptors whose afferent fibres are not reliably blocked by standard concentrations of local anaesthetic [3]. The clinical question this review addresses is therefore not simply "why does anaesthesia fail?" but the frequently overlooked corollary: "why do patients sometimes interpret a legitimate, non-painful physiological sensation as pain when anaesthesia has, in fact, succeeded?"

This physiological misunderstanding sits alongside, and must be carefully distinguished from, genuine anaesthetic failure. True failure has well-described mechanistic bases, including inflammatory tissue acidosis, upregulation of anaesthetic-resistant sodium channels in inflamed pulp, anatomical barriers to diffusion, and accessory or cross-innervation of the dental arches [4,5,6]. Failure to differentiate between these two distinct clinical entities, one pharmacological and one perceptual, risks either the under-treatment of a patient in genuine pain or the over-treatment (with repeated injections and unnecessary anaesthetic load) of a patient who is, in physiological terms, already profoundly anaesthetised.

The objective of this review is threefold: first, to characterise the pharmacological and anatomical determinants of genuine local anaesthetic failure during exodontia; second, to describe the neurophysiological basis by which proprioceptive and pressure sensation persists under profound anaesthesia and may be misinterpreted as pain; and third, to examine the psychological and central modulatory factors, including anxiety, catastrophising and nocebo expectation, that amplify this misinterpretation. The hypothesis underpinning this review is that a substantial proportion of reported intra-operative "pain" during exodontia under confirmed profound anaesthesia represents a physiological misunderstanding of preserved mechanosensory and proprioceptive input rather than a true breakthrough of nociceptive transmission.

2. Materials and Methods

This article was prepared as a narrative review rather than a systematic review, reflecting its objective of synthesising mechanistic and conceptual literature drawn from dental anaesthesiology, oral surgery, neurophysiology and pain science, rather than pooling quantitative outcome data from a homogenous set of clinical trials.

2.1 Search Strategy

A targeted literature search was conducted using PubMed/MEDLINE, Google Scholar and publisher databases (Wiley Online Library, Dove Medical Press, PubMed Central) for articles published in English. Search terms included combinations of: "local anaesthesia failure", "hot tooth", "inflammatory acidosis", "tetrodotoxin-resistant sodium channels", "differential nerve blockade", "proprioception periodontal ligament", "mylohyoid accessory innervation", "dental anxiety pain perception", "pain catastrophising dentistry", "nocebo dental extraction" and "IASP definition of pain". Reference lists of retrieved articles were hand-searched for additional relevant sources.

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2.2 Selection Criteria

Peer-reviewed original research, narrative and systematic reviews, and authoritative position statements addressing the pharmacology of local anaesthetic action, the anatomy of trigeminal innervation relevant to exodontia, the neurophysiology of nerve fibre blockade, and the psychological modulation of pain perception in dental settings were included. Non-peer-reviewed opinion pieces were excluded except where used to illustrate clinical framing rather than to support a mechanistic claim.

2.3 Analytical Approach

Findings were organised thematically into four physiological domains, namely pharmacological/biochemical, anatomical, neurophysiological, and psychological/central, to allow the discrepancy between objective anaesthetic depth and subjective pain report to be examined from complementary perspectives. Because this review synthesises previously published, de-identified literature and does not report new data derived from identifiable patients, no original clinical investigation, patient recruitment or biological sampling was undertaken by the author, and formal institutional ethical approval was therefore not applicable. Where illustrative clinical scenarios are described, these are composite and non-identifying, consistent with standard editorial practice for narrative reviews of this kind.

3. Results and Discussion

3.1 Genuine Anaesthetic Failure: The Pharmacological and Biochemical Domain

A meaningful proportion of reported intra-operative pain reflects true, pharmacologically incomplete nociceptive blockade rather than misperception, and this must be excluded before any misunderstanding hypothesis is invoked. Local anaesthetics are weak bases that must cross the nerve sheath and axonal membrane in their uncharged, lipid-soluble form before re-ionising intracellularly to block voltage-gated sodium channels from within the axoplasm [4,5]. In inflamed tissue, the local extracellular pH falls, shifting the equilibrium toward the charged, poorly diffusible form of the drug and reducing the concentration reaching the nerve membrane; this "inflammatory acidosis" theory remains the most frequently cited explanation for failure in a "hot tooth" [4,6]. Ueno and colleagues, however, demonstrated experimentally that local anaesthetics retain substantial membrane-fluidising activity even at pH 6.4, and proposed that inflammatory peroxynitrite, rather than acidosis alone, may impair drug-membrane interaction, indicating that the biochemical mechanism of failure in inflamed tissue is more complex than pH shift in isolation [7].

A second, molecular mechanism involves the upregulation of tetrodotoxin-resistant (TTXr) sodium channels, such as Nav1.8, on nociceptor terminals within inflamed pulp. These channels are comparatively resistant to blockade by amide local anaesthetics at clinically used concentrations, so that even where diffusion is adequate, a greater proportion of channels remains available to generate action potentials [4,6]. Inflammatory mediators such as prostaglandin E2 further lower the excitability threshold of peripheral nociceptors and alter their resting membrane potential, compounding the problem [4]. Increased local blood flow secondary to inflammatory vasodilation also accelerates systemic clearance of the anaesthetic from the injection site, shortening the time available for adequate nerve block to develop [6,8].

These biochemical mechanisms explain why the inferior alveolar nerve block, in particular, is reported to fail in a substantial minority of cases, with pulpal anaesthesia failure rates documented at approximately 17% for mandibular first molars and even higher for teeth with symptomatic irreversible pulpitis [9]. Clinically, this category of failure is characterised by inadequate soft-tissue numbness, incomplete response suppression on probing prior to elevation, or a history of acute infective or inflammatory pathology at the extraction site, and constitutes a true indication for supplemental anaesthetic technique rather than reassurance alone.

3.2 Anatomical Variation, Accessory and Cross-Innervation

A second category of genuine failure arises independently of tissue inflammation and reflects anatomical variability in trigeminal innervation. The nerve to the mylohyoid, traditionally regarded as a purely motor branch of the inferior alveolar nerve, has been shown by dissection and clinical studies to provide accessory sensory innervation to the mandibular molars and, in some individuals, the

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anterior teeth, via small lingual foramina that bypass the standard field of an inferior alveolar nerve block [10]. Because this branch may leave the main trunk proximal to the mandibular foramen and can be shielded from anaesthetic solution by the sphenomandibular ligament, a conventional block may anaesthetise the lip and tongue, both reliable clinical indicators of inferior alveolar nerve blockade, while leaving pulpal sensation to the molars intact [11,12]. This dissociation between subjective soft-tissue numbness and objective pulpal anaesthesia is a frequent source of clinician confusion, since the patient may appear, by every external sign, to be profoundly anaesthetised.

Cross-innervation from the contralateral inferior alveolar nerve across the midline has also been documented, particularly at the mandibular incisors, where terminal fibres of the incisive nerve may extend beyond the midline sufficiently to maintain sensation despite unilateral block [11,12]. Additional anatomical contributors include variation in the position of the mandibular foramen relative to the occlusal plane, the thickness and density of the overlying cortical bone (a particular barrier in the posterior mandible compared with the more porous maxilla), and buccal or lingual nerve contributions in specific individuals [6,13]. Supplemental techniques, including the Gow-Gates approach, intraligamentary injection, or a modified mylohyoid infiltration adjacent to the lingual plate, have each been shown to improve anaesthetic success where these anatomical factors are suspected [11,14], underscoring that this category, like inflammatory failure, represents a genuine and remediable pharmacological deficit rather than a perceptual phenomenon.

3.3 Differential Nerve Fibre Blockade: The Neurophysiological Basis of the Misunderstanding

The central physiological explanation for pain perception under otherwise successful anaesthesia lies in the differential susceptibility of nerve fibre types to local anaesthetic blockade. Peripheral nerve fibres are classified according to diameter, myelination and conduction velocity into Aα (motor, proprioception), Aβ (light touch, pressure, vibration, position sense), Aδ (sharp, well-localised pain, temperature) and C fibres (dull, poorly localised pain, temperature) [15,16]. In vivo peripheral nerve blockade studies indicate that small myelinated Aδ (and Aγ motor) fibres are generally the most susceptible to local anaesthetic conduction block, followed by the larger myelinated Aα and Aβ fibres, with small unmyelinated C fibres, counter-intuitively, often the most resistant at a given drug concentration [16]. The clinically important consequence is that a concentration of anaesthetic sufficient to abolish sharp, well-localised nociceptive transmission (the pain a patient would report from, for example, a needle stick or a burn) does not reliably or completely abolish conduction in the larger Aβ fibres that convey pressure, stretch and proprioceptive information from the periodontal ligament, periosteum and adjacent soft tissue [3,17].

This is not an incidental limitation of anaesthetic technique; profound local anaesthesia is neither designed nor intended to abolish proprioception, since a degree of preserved position sense is what allows the surgical team, and in principle the patient, to register that manipulation is occurring at all. The periodontal ligament and periosteum are richly supplied with mechanoreceptors that mediate the fine proprioceptive discrimination required for normal mastication, and these afferents continue to signal the pressure, torque and stretch generated by elevator and forceps application even when nociceptive Aδ and C fibre transmission from the same field has been fully blocked [3]. A patient experiencing this preserved but entirely non-noxious mechanosensory input, often accompanied by the low-frequency vibration and cracking sounds of periodontal ligament rupture and bone expansion, may reasonably but incorrectly interpret the sensation as pain, particularly in the absence of prior explanation that such pressure is an expected and harmless feature of a technically successful extraction.

This distinction is supported by observations in regional and neuraxial anaesthesia more broadly, where the differential blockade of autonomic, sensory and proprioceptive fibres is a well-recognised, reproducible clinical phenomenon, with motor and proprioceptive function returning before pinprick sensation as a block regresses, and being the last modalities lost as a block is established [15]. Applied to exodontia, this hierarchy predicts precisely the clinical picture seen at the chair: a patient with a fully numb lip and no response to a sharp probe (Aδ/C fibre blockade confirmed) may nonetheless feel, and accurately report feeling, the pressure of elevation (Aβ fibre transmission preserved). The clinician's task is therefore not to eliminate this sensation, which may not be pharmacologically achievable or even desirable, but to correctly identify it as pressure rather than pain, and to ensure the patient shares that same interpretive framework before the procedure begins.

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3.4 Psychological Modulation and Central Amplification of Perceived Pain

The peripheral persistence of proprioceptive and pressure afferents provides the physiological raw material for the misunderstanding, but whether that raw material is consciously labelled as "pressure" or as "pain" is substantially shaped by central and psychological factors. Because pain is, by definition, always a personal and subjective experience influenced by biological, psychological and social factors and cannot be inferred solely from peripheral nociceptor activity [2], ambiguous mechanosensory input is particularly susceptible to top-down reinterpretation in the anxious or fearful patient.

Dental anxiety is common, multifactorial, and strongly associated with expectation of pain; studies using the Corah Dental Anxiety Scale and similar instruments have repeatedly found a significant positive correlation between pre-operative anxiety and intra-operative pain scores reported during extraction, independent of objective anaesthetic depth [1,18,19]. Pain catastrophising, characterised by rumination, magnification and feelings of helplessness in anticipation of a painful stimulus, has been shown to function as an independent predictor of both dental anxiety and heightened pain reporting, and appears to act, at least in part, through mechanisms distinct from simple fear [20]. Cognitive models of dental fear further emphasise that perceived unpredictability and lack of control over the procedure can be more influential in driving distress than the objective intensity of any peripheral stimulus, which is directly relevant to exodontia, an inherently unpredictable procedure from the supine patient's perspective, involving sound, vibration and force that cannot easily be visually anticipated [20].

Expectation itself can amplify or dampen perceived pain through well-documented placebo and nocebo mechanisms. In a cross-sectional study of post-extraction patients, both hypoalgesic (placebo) and hyperalgesic (nocebo) effects were demonstrated, with amplification of the emotional component of pain reporting occurring even in the absence of corresponding clinical signs of inadequate anaesthesia or complication [21]. Negatively framed language from the clinician or dental team, for example emphasising that a procedure "might hurt" rather than that the patient "will feel pressure and movement," has been shown more broadly in the pain literature to increase both anticipatory anxiety and reported pain intensity, whereas carefully framed, expectation-setting communication can reduce it [22,23]. Reassurance strategies such as structured pre-procedural explanation of expected sensations, permission to signal discomfort, planned rest breaks, and relaxation breathing have each been associated with reduced anxiety and reduced perceived pain during extraction in case-level and controlled observations [24].

3.5 Reconciling the Two Categories: A Framework for Clinical Differentiation

Taken together, the pharmacological/anatomical domain (Sections 3.1–3.2) and theneurophysiological /psychological domain (Sections 3.3–3.4) describe two clinically distinct but frequently conflated causes of the same presenting complaint, namely a patient who reports pain during extraction despite an apparently successful local anaesthetic technique. Distinguishing between them is of direct clinical consequence, since true anaesthetic failure requires further pharmacological intervention (supplemental infiltration, alternative block technique, buffered or higher-concentration anaesthetic, or staged treatment of acute inflammation), whereas a physiological misunderstanding of preserved pressure sensation requires communication, reassurance and behavioural support rather than repeated needle exposure and escalating drug dose [1,9].

Practical clinical indicators favouring true failure include a positive response to sharp probing of the gingival sulcus or tooth prior to elevation, absent soft-tissue numbness in the expected distribution, a history of acute pulpal or periapical inflammation, and reported pain that is sharp, well-localised and reproducible with light contact. Indicators favouring a physiological misunderstanding of preserved proprioception include confirmed soft-tissue numbness and a negative response to sharp stimulus testing, pain described as pressure, tightness or a dull ache that accompanies but does not precede mechanical force, heightened pre-operative anxiety or a history of dental trauma, and rapid resolution of distress with verbal reassurance rather than with additional anaesthetic. In practice these categories are not always cleanly separable, and mixed presentations, for example a genuinely anxious patient with a partially inflamed pulp, are common; the framework is intended to guide clinical reasoning rather than to replace it.

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From a broader perspective, this reconciliation reinforces a point implicit in the revised IASP definition of pain: that pain and nociception are not interchangeable, and that a report of pain, however sincerely and accurately conveyed by the patient, does not by itself indicate a breach of nociceptive blockade [2]. Respecting the validity of the patient's subjective report while correctly attributing its physiological source is therefore central to both good clinical decision-making and to the ethical obligation to take patient-reported pain seriously.

4. Conclusion

The perception of pain during exodontia despite profound, objectively confirmed local anaesthesia is not a single phenomenon but the product of at least two distinct physiological processes that are frequently, and understandably, conflated at the chairside. Genuine anaesthetic failure, driven by inflammatory acidosis, tetrodotoxin-resistant sodium channel upregulation, and anatomical variation such as mylohyoid accessory innervation, remains a real and remediable cause of breakthrough nociception that must always be excluded first. Separately, and arguably more frequently in cases where soft-tissue anaesthesia is confirmed, the differential susceptibility of nerve fibre types to local anaesthetic blockade means that pressure, stretch and proprioceptive sensation mediated by relatively resistant Aβ fibres persists even when nociceptive Aδ and C fibre transmission is fully abolished. Where this preserved, non-noxious mechanosensory input is amplified by anxiety, pain catastrophising or nocebo expectation, patients may reasonably but mistakenly label it as pain.

Clinically, this distinction supports a practical approach in which objective testing (soft-tissue numbness, response to sharp probing) is used to differentiate true failure from misperception before further anaesthetic is administered, and in which explicit pre-operative communication, distinguishing expected pressure and movement from pain, and inviting the patient to signal true pain rather than pressure, is used as a first-line strategy for the latter. Such an approach may reduce unnecessary re-injection, improve the validity of informed consent, and support a more trusting clinician-patient relationship during a procedure that remains, for many patients, a significant source of anxiety. Future prospective research quantifying the relative contribution of pharmacological failure versus proprioceptive misattribution to reported intra-operative pain, using objective sensory testing alongside validated anxiety and catastrophising instruments, would help to refine this framework further.

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7. Conflict of Interest

The author declares no financial, professional, institutional or personal conflict of interest that could have influenced the conduct, interpretation or reporting of this review.

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