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Is Pulp Vitality Threatened by Orthodontic Treatment? A Comprehensive Review

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Abstract

Orthodontic tooth movement relies on the biological response of dental tissues to externally applied mechanical forces. Although orthodontic treatment is generally considered safe, pulpal complications, including pulp canal obliteration and, more rarely, pulpal necrosis, have been reported, and the relationship between orthodontic forces and pulpal vitality remains debated. This narrative review aimed to provide a comprehensive overview of current evidence on pulpal responses to orthodontic forces. Relevant publications were identified through searches of PubMed, Scopus, the Cochrane Library, ScienceDirect, and Google Scholar, with emphasis on recent and relevant studies. The available evidence does not establish a direct causal relationship between orthodontic tooth movement and pulpal necrosis. Previously undiagnosed dental trauma may explain some reported cases, while excessive forces and pre-existing pulpal conditions may increase susceptibility. Appropriate force application and regular pulpal monitoring remain essential during orthodontic treatment.

Subject Areas

Dentistry

Keywords

Orthodontic Treatment, Orthodntics, Aseptic Necrosis, Endodontics, Interdisciplinary Approach

1. Introduction

Induced tooth movement can be defined as the biological response of the dento-periodontal system to the application of an external mechanical force. Orthodontic biomechanics relies on a complex sequence of biological reactions within the dental organ following force application, with the aim of correcting dental mal-

positions and thereby optimizing both aesthetics and occlusal function. Although orthodontic treatment is generally considered safe, pulpal complications such as root resorption, pulp canal obliteration, or, more rarely, pulpal necrosis may occur [1] [2].

Biologically, orthodontic tooth movement is based on a cascade of tissue remodeling processes within the periodontium. Under physiological conditions, tissue turnover involves continuous cellular and fibrillar renewal of the periodontal ligament and alveolar bone. This dynamic equilibrium allows natural tooth migration while maintaining the thickness of the lamina dura through a balance between bone apposition and resorption [3]. Therapeutic remodeling is initiated when orthodontic forces disrupt this equilibrium, triggering an acute inflammatory response associated with immediate hydrodynamic changes, including vascular compression and transient ischemia. This mechanical stress leads to the formation of a hyalinized zone characterized by localized tissue degeneration, whose removal by osteoclasts enables actual tooth displacement. When light forces are applied, bone resorption occurs directly (frontal resorption), whereas excessive forces result in indirect resorption originating from the medullary spaces [1] [3].

Mechanical force application affects not only the periodontal support tissues but also the pulpo-dentinal complex.

Studies using laser Doppler flowmetry [4]-[6] have demonstrated a transient reduction in pulpal blood flow (PBF) within the first 20 minutes following force activation, reaching a minimum between 48 and 72 hours. In most cases, PBF gradually returns to baseline levels within one to three months, suggesting physiological adaptation of the pulpal neurovascular system. At the molecular level, rapid but reversible changes in the expression of key proteins, including vascular endothelial growth factor (VEGF), fibronectin, and type I collagen, have been reported when light and controlled forces are applied [7].

Conversely, pathological remodeling may occur when the magnitude or duration of orthodontic forces exceeds the adaptive capacity of the tissues. Iatrogenic effects include root resorption involving dentin and cementum, epithelial attachment loss, and irreversible alveolar bone defects such as fenestrations and dehiscences [3].

Despite these advances, the direct relationship between orthodontic tooth movement (OTM) and pulpal necrosis remains one of the most debated topics in interdisciplinary dentistry. Although the prevalence of pulpal necrosis during orthodontic treatment has been estimated to range from 1% to 14%, no conclusive scientific evidence supports a direct causal relationship between orthodontic forces and irreversible pulp necrosis in teeth without pre-existing pathology [8].

2. Review-Methods

This narrative review aimed to provide a comprehensive overview of current evidence on pulpal responses to orthodontic forces. A literature search was con-

ducted using PubMed, Scopus, the Cochrane Library, ScienceDirect, and Google Scholar. The main search terms included "tooth movement", "pulp necrosis", "pulpal blood flow", "laser Doppler flowmetry", and "pulp sensibility". Prospective and retrospective studies done on humans, systematic reviews, finite element analysis studies, and molecular or in vitro investigations examining pulpal responses to mechanical stress were considered. Case reports, case series and articles lacking an explicit methodology were excluded. The selected literature was analyzed qualitatively and synthesized thematically, taking into account the nature and level of scientific evidence.

3. Orthodontic Force-Induced Pulpal Necrosis: Current Evidence and Associated Risk Factors

Recent systematic reviews consistently conclude that there is no strong scientific evidence directly linking orthodontic forces to pulpal necrosis in initially healthy teeth [9]-[11]. The overall certainty of the available evidence is rated as "very low" with regard to establishing a causal relationship [8].

From this perspective, pulpal necrosis associated with orthodontic treatment has been described by some authors as an etiological myth. According to Consolaro *et al.* [12], orthodontic forces are inherently dissipative and physiological; when pulpal necrosis occurs, it is almost exclusively attributable to an undiagnosed prior dental trauma [12]. Nevertheless, careful identification of predisposing factors remains essential to prevent irreversible pulpal damage. While orthodontic treatment is generally safe, several biological and mechanical factors may increase the vulnerability of the pulpo-dentinal complex.

3.1. History of Dental Trauma

The most significant and well-documented risk factor is a history of dental trauma, particularly concussion injuries. According to several authors, orthodontic tooth movement alone does not induce pulpal necrosis but rather reveals or exacerbates a latent apical neurovascular injury caused by a previous, often undiagnosed, traumatic event [12]. Teeth that have sustained severe periodontal injuries exhibit a markedly higher susceptibility to loss of vitality than intact teeth [10] [13]. Moreover, teeth subjected to repeated traumatic episodes show a substantially increased risk of pulpal and periapical complications [14].

3.2. Dental Maturity and Patient Age

Patient age at the onset of orthodontic treatment directly influences pulpal response. In older patients, the pulpal chamber volume is reduced due to physiological deposition of secondary dentin, and the apical foramen becomes narrower, limiting collateral circulation and slowing recovery of pulpal blood flow [6] [15]. In contrast, immature teeth with widely open apices benefit from enhanced vascular adaptability and exhibit a lower risk of vascular strangulation during orthodontic force application [16].

3.3. Magnitude and Type of Orthodontic Forces

Force magnitude represents a major determinant of tissue stress. The application of heavy forces, particularly those exceeding 224 - 250 g, results in a more pronounced reduction in pulpal blood flow and may lead to prolonged periodontal ligament hyalinization. This condition interferes with physiological remodeling and promotes cellular damage [6] [8] [11]. However, biomechanical evidence provides a more nuanced perspective. Finite element simulations indicate that even under forces ranging from 2 N to 4 N, the stress exerted on the neurovascular bundle remains below the maximum physiological hydrostatic pressure (16 - 22 kPa), indicating a potentially limited mechanical impact under modeled conditions [17]. This mechanical tolerance is further corroborated at the molecular level, transcriptional responses in periodontal ligament cells, including changes in *CPNE3* and *OPHN1* expression, suggest the activation of cellular pathways involved in adaptation and tissue homeostasis [18]. Collectively, these findings provide indirect mechanistic evidence regarding tissue responses to orthodontic loading but should not be interpreted as definitive proof of pulpal safety.

Moreover, the temporal pattern of force application also plays a critical role, continuous forces are considered more harmful than intermittent forces, as the latter allow periods of tissue recovery and vascular reperfusion [2] [9].

When evaluating pulp sensitivity changes during orthodontic treatment at different time periods, Briseño-Marroquín *et al.* [19] demonstrated that although electrical pulp sensitivity thresholds initially increased after force application, all tooth types retained their responsiveness, indicating a reversible physiological alteration rather than irreversible pulpal damage.

During the alignment and leveling phase, the pulpal vascular system responded rapidly. Salles *et al.* [5] reported a significant reduction in pulpal blood flow (PBF) within the first 20 minutes following activation, with a maximum decrease occurring between 48 and 72 hours. Nevertheless, PBF consistently returned to baseline within one month, confirming that this initial phase did not result in permanent pulpal injury.

Incisor retraction, despite involving large-magnitude tooth movements, appeared to be safe for the dentin-pulp complex. Guo *et al.* [20] found no significant difference in pulpal blood flow of anterior teeth after en masse retraction compared with untreated controls. A transient reactive hyperemia was observed one month after appliance removal, followed by a complete return to baseline values within three months, demonstrating the resilience of apical vascularization to prolonged retraction forces.

Throughout the space closure phase, ongoing mechanical stresses continued to influence pulpal neurophysiology. According to Golež *et al.* [21], orthodontic tooth movement led to increased sensory response thresholds, reflecting transient hypoxia of Aδ nerve fibers. This temporary reduction in sensitivity should not have been interpreted as loss of pulp vitality, as normal sensory function was restored after the active movement period.

3.4. Type of Orthodontic Tooth Movement

Among orthodontic movements, intrusion is consistently identified as the most critical. By concentrating compressive forces directly at the narrow apical foramen, intrusion increases the risk of pulpal ischemia or vascular strangulation [8] [9] [21] [22]. This movement is particularly hazardous in teeth presenting with complete pulp canal calcification, as their compromised vascular network cannot compensate for the force-induced reduction in blood flow [1] [13]. A recent study of Cai *et al.* [17] indicated that tooth rotation is in fact the movement that generates the highest level of stress on the neurovascular bundle, followed by intrusion and extrusion.

4. Diagnosis of Pulp Necrosis during Orthodontic Treatment

Management of pulp necrosis occurring during orthodontic treatment requires close interdisciplinary collaboration between orthodontists and endodontists to preserve the tooth on the arch while minimizing the risk of further complications [2] [23].

Diagnosis represents a major clinical challenge, as orthodontic forces transiently alter the physiological status of the pulpo-dentinal complex [11] [16]. Therefore, systematic pulpal assessment should be integrated into orthodontic follow-up to distinguish reversible adaptive responses from irreversible pathological changes [4] [23].

4.1. Clinical Warning Signs: Pain and Discoloration

Orthodontic monitoring should be particularly attentive to clinical "red flags". Crown discoloration or darkening is a nonspecific warning sign that may be associated with aseptic pulpal necrosis or calcific metamorphosis, potentially involving the diffusion of hemoglobin breakdown products into dentinal tubules [12] [16]. Although orthodontic tooth movement may induce short-term sensitivity, persistent or atypical pain should raise suspicion of endodontic pathology, even though symptoms may be confounded with appliance activation discomfort [16] [23].

4.2. Limitations of Conventional Sensibility Tests

Diagnosis traditionally relies on thermal and electric pulp tests; however, their reliability is compromised during orthodontic treatment [4] [11]. Orthodontic forces transiently increase the electric pulp test threshold, likely due to apical nerve fiber compression or pulpal hypoxia, leading to false-negative results [10] [16] [22]. While healthy teeth typically respond to EPT throughout treatment, sensitivity thresholds fluctuate depending on wire type and force magnitude [21]. Cold testing appears more stable than EPT during orthodontic treatment, although both methods assess neural response rather than true vascular vitality [19] [21].

4.3. Blood-Flow Assessment

Assessment of vascular vitality is considered more critical than neural response, as pulpal healing depends on the maintenance of blood circulation [4] [11]. Laser

Doppler flowmetry (LDF) is currently the most accurate non-invasive method for real-time monitoring of pulpal blood flow [4] [20] [22]. Studies demonstrate an immediate decrease in PBF following force application, followed by normalization within one to three months [4] [6]. However, persistent alterations in blood-flow measurements alone cannot establish pulpal necrosis without validated diagnostic thresholds and corroborating clinical findings [6] [22].

4.4. Structural Imaging

Conventional two-dimensional radiography has limited ability for early detection of pulpal necrosis or resorption processes [2] [11]. Cone Beam Computed Tomography (CBCT) provides three-dimensional assessment of dental and periapical structures and may help identify structural changes, including external root resorption; however, it does not directly assess pulpal vitality, and its diagnostic superiority should be established for the specific condition investigated [1] [24].

4.5. Investigational Biochemical Markers

Aspartate aminotransferase (AST) has been investigated as a potential biochemical marker of cellular injury in dental tissues. As an intracellular enzyme, its release into surrounding fluids may reflect cellular damage associated with mechanical stress. In the context of orthodontic tooth movement, changes in AST activity may therefore provide indirect information about tissue responses to applied forces [8] [10] [11]. However, AST elevation is not specific to pulpal injury and cannot independently distinguish reversible tissue stress from irreversible damage or pulpal necrosis.

4.6. Importance of Trauma History

A diagnosis of pulpal necrosis during orthodontic treatment necessitates a thorough investigation of prior dental trauma, particularly concussion injuries [10] [12]. According to Consolaro *et al.* [12], orthodontic movement alone does not cause necrosis but reveals a pre-existing apical neurovascular lesion resulting from an undiagnosed traumatic event. Comprehensive pre-orthodontic assessment therefore remains the cornerstone of differential diagnosis [16] [20].

5. Therapeutic Strategies to Manage Pulp Necrosis during Orthodontic Tooth Movement

When pulpal necrosis is diagnosed during active orthodontic treatment, immediate endodontic intervention is indicated [2]. Canal cleaning and shaping should be performed, followed by placement of a temporary calcium hydroxide dressing to control infection and stabilize inflammatory root resorption [2] [16]. The timing of definitive obturation should be determined by the endodontic diagnosis, adequacy of infection control, and presence and progression of root resorption, rather than routinely postponed until completion of orthodontic treatment. Obturation may proceed once appropriate clinical criteria are met; persistent infec-

tion or active inflammatory resorption may warrant continued medication and reassessment in accordance with established endodontic guidelines.

The management of traumatized or necrotic teeth requires precise therapeutic scheduling and careful biomechanical control by the orthodontist. In teeth that have undergone endodontic treatment prior to orthodontic intervention, a waiting period of approximately six months was recommended before initiating tooth movement, provided that the tooth remained asymptomatic and radiographic evidence of periapical healing was present [22]. Similarly, in cases of severe orthodontically induced inflammatory root resorption, clinicians often suspended active treatment for three to six months and employed passive archwires in order to reduce mechanical loading and promote cemental repair.

Within this clinical context, the choice of orthodontic appliance primarily influenced force delivery and modulation rather than pulpal vascular outcomes. When comparing clear aligners with fixed orthodontic appliances, Alhaija *et al.* [4] reported no statistically significant difference between the two modalities with respect to changes in pulpal blood flow, suggesting that pulpal responses were more closely related to the magnitude and control of applied forces than to the type of appliance itself.

Orthodontists must inform patients of the increased risks affecting teeth with a history of trauma, while endodontists should ensure dense three-dimensional obturation, as properly treated endodontic teeth present a risk of apical resorption comparable to that of vital teeth [1] [2]. This interdisciplinary synergy enables mechanical objectives to be tailored, favoring light and intermittent forces, according to pulpal biological response, thereby ensuring a favorable long-term prognosis [2] [23].

6. Conclusions

Current evidence does not establish a direct causal relationship between orthodontic tooth movement and pulpal necrosis in teeth without pre-existing pathology. Previously undiagnosed dental trauma may explain some reported cases of loss of pulp vitality during orthodontic treatment, although its role is not definitively established. Within biological limits, orthodontic forces can induce transient alterations in pulpal blood flow, but their long-term implications remain incompletely understood.

However, factors including a history of trauma, advanced age, intrusive movements, heavy or continuous forces, and compromised apical vascular supply may increase pulpal susceptibility. Diagnosis remains challenging due to the limited reliability of conventional sensibility tests during treatment. Therefore, systematic monitoring and close interdisciplinary collaboration are essential to ensure early detection, appropriate management, and favorable long-term outcomes.

Author Contributions

A. EL Mabrak and H. Mansy: Conceptualization, Methodology, Writing—Origi-

nal Paper. F. EL Quars: general supervision. All authors reviewed and approved the final version of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

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