Original Article

ORTHODONTIC ALIGNMENT AS A MODIFIER OF PERIODONTAL RISK: CONCEPTUALIZING THE INTERDEPENDENCE BETWEEN TOOTH POSITION, PLAQUE RETENTION, AND PERIODONTAL STABILITY

Claire Martin[1], Julien Robert[2], Sophie Bernard[1]*

[1]*Department of Orthodontics and Dentofacial Orthopedics, Faculty of Dentistry, University of Lyon, Lyon, France.*
[2]*Department of Periodontology and Oral Tissue Regeneration, Faculty of Dentistry, University of Strasbourg, Strasbourg, France. julien.robert@gmail.com*

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ABSTRACT

Orthodontic alignment is traditionally viewed as a means to improve aesthetics and function, yet its role as a modifiable determinant of periodontal risk remains underexplored in interdisciplinary theory. This conceptual manuscript proposes that tooth position directly modulates plaque retention patterns, which in turn govern periodontal tissue stability and long-term disease susceptibility. Through a synthesis of contemporary evidence, the authors construct an interdependent triad: malalignment creates sheltered niches for microbial accumulation; orthodontic correction disrupts these niches and redistributes biofilm; and the resulting tissue response either reinforces or erodes periodontal attachment. Four core propositions are advanced to explain these interactions. First, crowding and rotation increase interproximal and gingival plaque retention surfaces beyond the threshold for effective self-care. Second, alignment reduces these surfaces while simultaneously improving access for professional instrumentation. Third, the modified plaque ecology attenuates inflammatory cascades that drive attachment loss. Fourth, sustained alignment stabilizes periodontal architecture by minimising secondary occlusal trauma and facilitating physiologic bone remodelling. The model positions orthodontic alignment not as an adjunct but as an active modifier capable of lowering periodontal risk in both healthy and compromised dentitions. Clinical translation emphasises early risk stratification and sequenced ortho-perio care to harness alignment-induced stability. This theoretical framework reframes orthodontic intervention as a periodontal preventive strategy and calls for future interdisciplinary protocols that quantify alignment-specific risk reduction.

Key words: Orthodontic alignment, Periodontal risk modification, Plaque retention, Tooth malposition, Periodontal stability, Conceptual model.

Introduction

The interface between orthodontics and periodontology has evolved from sequential treatment to true interdependence. Tooth position is no longer regarded solely as an aesthetic or functional variable; it is increasingly recognised as a biomechanical and ecological determinant of periodontal health [1-7]. Malaligned teeth create microenvironments that favour plaque retention, alter gingival architecture, and modify occlusal load distribution [1, 2]. Conversely, orthodontic alignment can reshape these environments, redistribute biofilm, and enhance periodontal resilience [3, 4, 8-12]. This conceptual manuscript advances the thesis that orthodontic alignment functions as a modifiable risk factor for periodontal disease progression by directly influencing plaque retention dynamics and subsequent tissue stability.

Contemporary clinical practice frequently encounters adult patients presenting with both malocclusion and periodontal compromise. In such cases, traditional risk-assessment models—focused primarily on plaque index, smoking, and diabetes—underestimate the contribution of tooth position [1, 2]. Crowding, rotations, and drifting generate inaccessible interproximal surfaces and gingival crevices that evade routine mechanical plaque control [1, 13-17]. These sheltered zones promote subgingival biofilm maturation, elevate inflammatory mediators, and accelerate clinical attachment loss [2, 17]. Systematic analyses have consistently documented higher periodontal indices in malocclusion segments compared with aligned arches [4, 17, 18-21]. Yet the literature has stopped short of theorising alignment itself as a primary risk modifier.

The present framework addresses this gap by positioning orthodontic tooth movement within a three-domain causal chain: (1) tooth position and malalignment patterns, (2) plaque retention and microbial accumulation, and (3) periodontal tissue response and stability. Alignment intervenes at the first domain, producing cascading effects that attenuate risk at the second and reinforce resilience at the third [3, 4, 8, 12, 21]. This interdependence is not merely additive; it is multiplicative. Even modest changes in crown-root angulation can shift plaque ecology from pathogenic to commensal dominance, while sustained alignment preserves newly formed attachment and prevents secondary occlusal

trauma [3, 7, 8].

The conceptual model developed herein draws upon evidence spanning cross-sectional observations [1, 2], systematic reviews of combined ortho-perio therapy [4, 12, 17, 21], and longitudinal retention studies [13, 14, 16, 19]. It reframes orthodontic intervention as a targeted ecological and biomechanical therapy rather than an elective aesthetic procedure. In stage III–IV periodontitis, for example, pathologic migration exacerbates plaque retention; realignment reverses this migration and simultaneously reduces pocket depths through improved tissue tone [2, 8, 12, 21]. In healthy periodontium, alignment prevents future risk by eliminating anatomical plaque traps before inflammation becomes established [5, 7].

Importantly, the model acknowledges temporal dynamics. Immediate post-alignment changes include reduced plaque indices and improved gingival margin contours [4, 17]. Long-term stability depends on retention protocols that maintain the new position without introducing additional plaque-retentive hardware [13, 14, 16, 19]. Thus, alignment is both an acute modifier and a chronic stabiliser. The framework also integrates host susceptibility: patients with genetic or systemic risk factors derive amplified benefit from alignment-mediated risk reduction because the intervention lowers the microbial challenge below the individual's inflammatory threshold [5, 7].

By constructing this triad and deriving testable propositions, the manuscript provides clinicians with a theoretical scaffold for interdisciplinary decision-making. Orthodontists can incorporate periodontal risk scoring into treatment planning, while periodontists can leverage alignment to optimise maintenance outcomes [3, 7, 8]. The ultimate aim is to shift periodontal risk management from reactive maintenance to proactive biomechanical redesign. The following sections first examine the mechanistic links between tooth position and plaque retention, then formalise the conceptual model through explicit propositions and a diagrammatic representation.

Results and Discussion

Tooth position and plaque retention dynamics in periodontal disease development

Tooth position dictates the spatial architecture of the dentition and, by extension, the topography available for plaque adhesion. In aligned arches, interproximal contacts are tight and convex, limiting sheltered surfaces to thin embrasure spaces that remain accessible to interdental brushes and floss. Malalignment disrupts this geometry. Crowding produces overlapping contacts that create narrow, inaccessible crevices; rotations expose concave proximal surfaces; and vertical discrepancies generate ledge-like overhangs [1, 2, 17]. Each of these configurations increases the surface area protected from mechanical disruption, allowing undisturbed biofilm maturation [1, 17].

Empirical observations confirm that sites with crowding greater than 2 mm exhibit significantly elevated plaque scores and deeper probing depths [1, 2]. The mechanism is twofold. First, the physical niche reduces shear forces from mastication and tongue movement, permitting early colonisers to transition into mature, anaerobic communities. Second, the altered gingival contour compresses papillary tissue, forming pseudo-pockets that trap food debris and promote subgingival extension of supragingival plaque [1, 2, 17]. These dynamics are particularly pronounced in the anterior mandible, where saliva flow is lower, and self-cleansing is already compromised [1].

Beyond static anatomy, tooth position influences dynamic plaque accumulation through altered neuromuscular patterns. Patients with anterior crowding often adopt compensatory tongue postures that further shield lingual surfaces [22-29]. Rotated premolars create buccal concavities inaccessible to standard brushing strokes [1, 17]. Over time, these microenvironments select for periodontopathic species—Porphyromonas gingivalis, Tannerella forsythia, and Treponema denticola—whose virulence factors exacerbate gingival inflammation [17]. The resulting cytokine surge (IL-1β, IL-6, TNF-α) activates osteoclastogenesis, initiating connective-tissue attachment loss precisely at the sites of maximal plaque retention [17].

Orthodontic alignment reverses these processes through three interrelated biomechanical actions. First, derotation and derotation eliminate overlapping contacts, restoring convex proximal contours and widening embrasures. Second, controlled intrusion or extrusion repositions gingival margins to physiologic levels, eliminating pseudo-pockets [3]. Third, arch coordination reduces bucco-lingual discrepancies that previously harboured food impaction [3, 4]. Collectively, these movements decrease the total plaque-retentive surface area by an estimated 30%–40% in moderate crowding cases [4, 17]. The reduction is not merely quantitative; it qualitatively alters biofilm architecture by exposing surfaces to salivary buffering, oxygen diffusion, and mechanical disruption [4, 17].

The downstream periodontal response is equally position-dependent. Aligned teeth transmit occlusal forces more axially, minimising lateral stress that could otherwise exacerbate bone loss in inflamed sites [3, 7]. Improved alignment also enhances gingival blood flow by relieving tissue compression, facilitating immune cell infiltration and collagen turnover [3]. Longitudinal data demonstrate that these tissue-level adaptations persist when retention is maintained, suggesting that alignment induces a new homeostatic equilibrium [8, 13]. In contrast, relapse into malalignment re-establishes the original risk topography within months, underscoring the necessity of stable positioning for sustained periodontal benefit [13, 14].

Importantly, the relationship is bidirectional. Pre-existing periodontal destruction can secondarily cause pathologic

migration, further worsening plaque retention [2, 12, 21]. This feedback loop explains why stage IV periodontitis patients frequently exhibit drifting incisors and open contacts that accelerate disease progression [2, 12]. Orthodontic realignment interrupts the cycle by repositioning teeth into bone-supported envelopes, thereby reducing pocket depths through both mechanical and biologic mechanisms [8, 12, 21]. The resulting decrease in clinical attachment loss risk is independent of plaque control compliance alone, offering a structural solution for patients with suboptimal hygiene [4, 8, 21].

Retention phase dynamics further illustrate the position–plaque–stability triad. Fixed lingual retainers, when properly contoured, can maintain alignment without adding plaque traps; however, poorly adapted retainers introduce new niches [13, 14, 16]. Vacuum-formed retainers, conversely, cover entire surfaces but are removable, enabling thorough cleaning [14, 19]. The choice of retention modality must therefore be guided by the same principles that drove alignment: minimal addition of retentive surfaces and maximal preservation of self-cleansing anatomy. Studies comparing retainer types confirm that periodontal indices remain stable only when the underlying alignment geometry is preserved [13, 14, 16, 19].

In summary, tooth position functions as the upstream regulator of plaque ecology. Malalignment creates and perpetuates high-risk microenvironments; orthodontic correction systematically dismantles those environments, redistributes microbial load, and reprograms tissue-level responses [3, 4, 8, 12, 17, 21]. These dynamics provide the mechanistic foundation for the conceptual model presented next.

Conceptual model of orthodontic alignment as a modifier of periodontal risk

The proposed model conceptualises orthodontic alignment as an active modifier within a closed-loop system linking tooth position, plaque retention, and periodontal stability. At its core lies a dynamic triad: malalignment patterns generate plaque retention zones that amplify microbial challenge, which, in turn, trigger inflammatory pathways that lead to attachment loss unless interrupted by alignment [1, 2, 17]. Orthodontic intervention acts at the first node, producing cascading reductions in risk across all subsequent nodes [3, 4, 8, 12, 21]. **Figure 1** illustrates the conceptual triad linking tooth position, plaque retention dynamics, and periodontal tissue stability, highlighting orthodontic alignment as a primary modifier of periodontal risk.

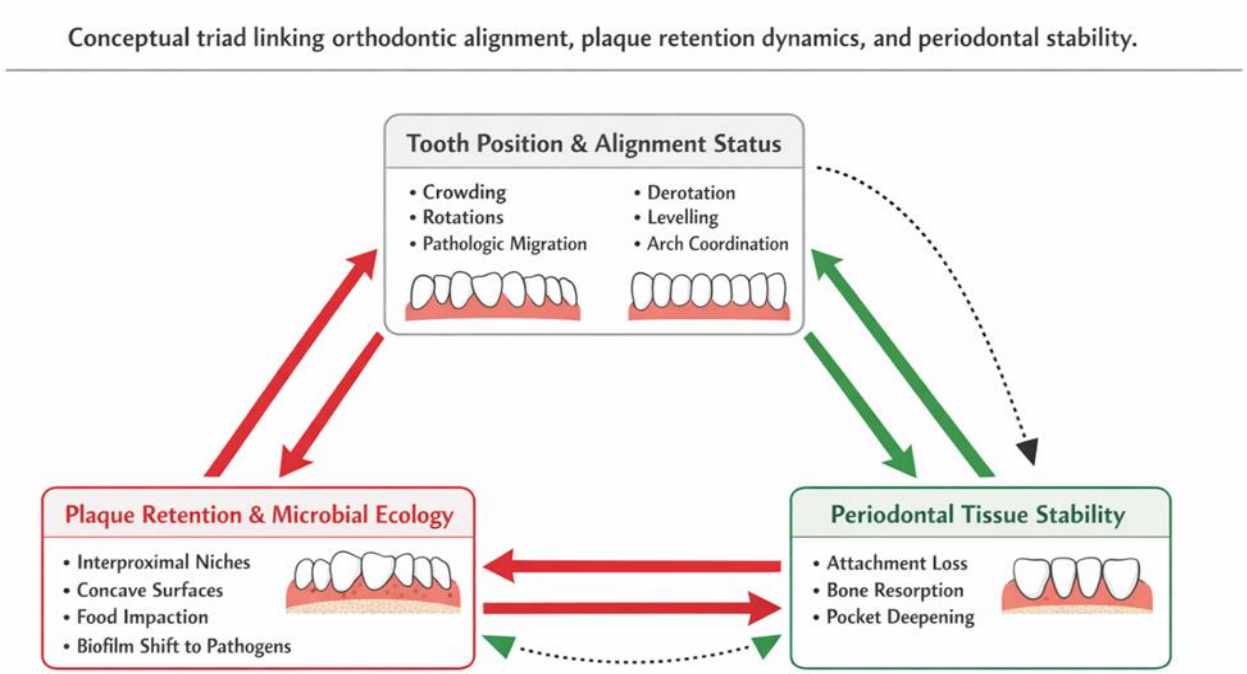

Figure 1. Conceptual triad linking orthodontic alignment, plaque retention dynamics, and periodontal stability.

Five propositions formalise the core relationships within this framework.

Proposition 1 states that malaligned tooth positions create anatomically sheltered plaque-retention zones that exceed the mechanical disruption threshold of routine oral hygiene, thereby elevating the risk of subgingival biofilm maturation and subsequent periodontal inflammation [1, 2, 17]. This proposition is supported by cross-sectional evidence linking greater crowding severity with higher plaque accumulation and increased bleeding indices [1, 2].

Proposition 2 holds that orthodontic alignment reduces the total surface area and structural complexity of plaque-retentive niches through derotation, levelling, and arch coordination, thereby decreasing microbial accumulation and shifting the biofilm toward a less pathogenic composition [3, 4, 17]. Systematic reviews of combined orthodontic-periodontal therapy further suggest that alignment alone can contribute to measurable reductions in probing depth under controlled maintenance conditions [4, 12, 21].

Proposition 3 proposes that the reduction in plaque retention following alignment attenuates host inflammatory responses, thereby limiting cytokine-driven osteoclast activation and helping preserve connective-tissue attachment [3, 4, 17]. Longitudinal observations in periodontally compromised patients indicate that realignment may stabilise attachment levels even beyond the effects of non-surgical debridement alone [8, 21].

Proposition 4 asserts that sustained orthodontic alignment minimises secondary occlusal trauma and supports more physiologic bone remodelling, thereby enhancing long-term periodontal architectural stability and reducing the likelihood of disease recurrence [3, 7, 8]. Retention studies beyond 5 years report lower attachment loss rates in well-aligned segments than in relapsed dentitions [13, 14].

Proposition 5 states that, in patients with pre-existing stage III–IV periodontitis, orthodontic correction of pathologic migration interrupts the self-reinforcing cycle of drifting-induced plaque retention and progressive attachment loss, thereby transforming a progressive risk state into a more stable maintenance state [2, 8, 12, 21]. An interdisciplinary case series supports this reversal when orthodontic alignment is appropriately sequenced after initial periodontal therapy [8, 12].

Collectively, these propositions position orthodontic alignment not as an ancillary periodontal intervention, but as a primary modifier of periodontal risk [3, 4, 7, 8, 12, 21]. The model is inherently dynamic: alignment can be titrated according to baseline periodontal vulnerability, while retention protocols must be individualised to preserve the newly established dental geometry over time [13, 14, 16, 19]. Although future empirical validation will require longitudinal trials capable of isolating alignment effects from confounding variables, the present conceptual architecture already offers meaningful clinical value for periodontal risk stratification and interdisciplinary treatment sequencing [30-39].

Theoretical interpretation of the triad: from mechanical reconfiguration to biologic homeostasis

The conceptual model advanced in Part 1 posits orthodontic alignment as a pivotal modifier within the tooth position–plaque retention–periodontal stability triad. Theoretically, this triad operates as a self-regulating system in which mechanical inputs (tooth repositioning) elicit biological outputs (altered microbial ecology and tissue remodeling). Malalignment functions as a persistent disequilibrium state: it imposes structural barriers to plaque disruption, thereby sustaining a high microbial burden that perpetuates chronic low-grade inflammation [1, 2, 17]. Orthodontic forces, by contrast, introduce controlled disequilibrium that favors the restoration of equilibrium [40- 49].

At the mechanistic level, alignment achieves this by biomechanically reprogramming the periodontal ligament and alveolar bone. Controlled tooth movement induces transient compression and tension zones that stimulate osteoblast–osteoclast coupling, favoring bone apposition on tension sides and resorption on compression sides without net loss in stable cases [3, 8]. This process is amplified in previously compromised sites, where realignment repositions roots within residual bony envelopes, reducing moment arms for occlusal forces and converting potentially pathologic lateral loads into axial vectors [3, 7, 21]. The reduction in lateral stress limits micro-trauma to inflamed periodontal ligament fibers, permitting fibroblast proliferation and collagen reorganization [3, 8].

Ecologically, the model interprets plaque retention zones as selective niches that enrich for dysbiotic consortia. Crowding and rotations create anaerobic microhabitats with reduced redox potential, favoring obligate anaerobes [17]. Alignment dismantles these niches, increasing oxygen exposure and salivary access, which shifts community composition toward aerotolerant commensals [4, 17]. This microbial shift downregulates virulence factor expression and attenuates pattern recognition receptor activation in gingival epithelial cells, thereby reducing NF-κB signaling and pro-inflammatory cytokine release [17]. The attenuated inflammatory milieu limits RANKL expression, curbing osteoclastogenesis and preserving attachment [3, 4].

From a systems perspective, the triad exhibits hysteresis: the path from malalignment to disease progression is easier than reversal, owing to irreversible attachment loss and adaptive microbial changes. Orthodontic intervention overcomes this hysteresis by providing a structural reset that lowers the activation energy for biologic recovery [8, 12, 21]. Sustained stability requires retention to prevent relapse-induced re-emergence of niches, creating a new attractor state of low-risk homeostasis [13, 14, 19]. In high-susceptibility individuals, alignment lowers the microbial load below the inflammatory threshold, effectively increasing the buffer capacity against systemic modifiers [5, 7].

This interpretation reframes orthodontics from a morphologic discipline to an applied ecology and biomechanics intervention. It explains why alignment yields disproportionate periodontal benefits in compromised cases: the intervention targets the upstream cause (anatomical plaque traps) rather than downstream symptoms

(inflammation) [2, 8, 12, 21]. The model also predicts non-linear dose–response relationships—modest alignment corrections in mild malocclusion may yield minimal benefit, whereas targeted repositioning in severe pathologic migration produces amplified stabilization [2, 12, 21].

Table 1 summarises the mechanistic pathways through which orthodontic alignment modifies periodontal risk by influencing plaque retention, microbial ecology, and periodontal tissue response.

Table 1. Mechanistic pathways linking orthodontic alignment to periodontal risk modification.

Domain	Mechanism	Biological/Clinical effect	Periodontal outcome
Tooth position	Crowding, rotation, pathologic migration	Creation of sheltered plaque niches	Increased microbial accumulation
Plaque ecology	Undisturbed biofilm maturation	Dysbiotic microbial community	Gingival inflammation
Host inflammatory response	Cytokine release (IL-1β, TNF-α, RANKL)	Osteoclast activation and connective-tissue degradation	Clinical attachment loss
Orthodontic alignment	Derotation, levelling, and arch coordination	Reduction of plaque-retentive surfaces	Lower microbial burden
Tissue remodeling	Improved axial load distribution and gingival architecture	Reduced lateral stress and improved blood flow	Stabilised periodontal attachment
Retention phase	Maintenance of aligned geometry	Prevention of relapse-induced plaque traps	Long-term periodontal stability

Clinical implications for orthodontic treatment planning

Translating the conceptual model into practice requires integration of periodontal risk assessment at every orthodontic decision point. Treatment planning should begin with comprehensive periodontal staging using the 2017 World Workshop classification, prioritizing the achievement of periodontal stability (probing depths ≤ 4 mm, bleeding on probing <10%, no suppuration) before initiating tooth movement [5, 7, 50-52]. In stage III–IV periodontitis, initial non-surgical therapy must resolve active inflammation, followed by radiographic confirmation of bone level stabilization before alignment [8, 12, 21].

Risk stratification informs sequencing and mechanics. Patients with thin biotypes or generalized recession warrant low-force, controlled mechanics (e.g., clear aligners for precise root torque without excessive tipping) to minimize further dehiscence [3, 5]. In cases of pathologic migration, uprighting and intrusion of elongated anterior teeth reduce pocket depths by repositioning the gingival margins and eliminating pseudo-pockets [2, 12, 21]. Interdisciplinary protocols should include baseline CBCT for root proximity assessment and periodic monitoring of attachment levels during active treatment [3, 8]. **Figure 2** depicts the clinical risk-modification pathway demonstrating how orthodontic alignment alters plaque retention dynamics and inflammatory signaling to influence periodontal outcomes.

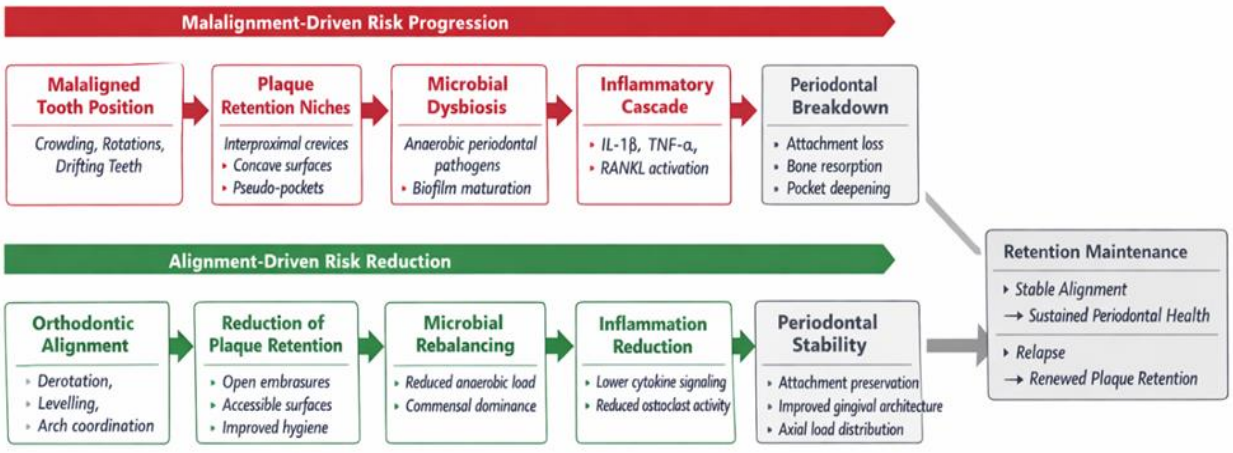

Figure 2. Clinical pathway illustrating how orthodontic alignment modifies periodontal risk across stages of plaque retention and tissue response.

Biomechanical considerations emphasize force systems that respect periodontal limits. Light, continuous forces (50–100 g) minimize hyalinization and maximize periodontal ligament remodeling without exacerbating inflammation

[3]. Anchorage planning avoids reactive forces on compromised units; skeletal anchorage or palatal miniscrews distribute load away from vulnerable teeth [3, 12]. Retention design is critical: fixed retainers should feature smooth, low-profile contours to avoid plaque accumulation, with regular ultrasonic debridement [13, 14, 16, 19]. Removable vacuum-formed retainers offer superior hygiene access but require high compliance [14, 19].
Treatment duration and expectations must be adjusted. Periodontally compromised cases often require extended active phases to allow tissue adaptation, with slower movement rates to prevent flare-ups [4, 8]. Patient education focuses on enhanced hygiene protocols during alignment, including the use of interdental aids tailored to newly widened embrasures [5, 7]. Shared decision-making incorporates patient values: esthetic gains from alignment may motivate improved compliance, indirectly supporting periodontal maintenance [6].

Preventive strategies leveraging alignment-induced stability
The model supports proactive orthodontic intervention as a preventive modality in select populations. In adolescents with moderate crowding and emerging gingivitis, early alignment eliminates plaque traps before subgingival colonization establishes, potentially reducing lifetime periodontal risk [1, 5, 17]. For adults in maintenance with drifting or flaring secondary to prior periodontitis, interceptive alignment prevents progression by closing open contacts and restoring self-cleansing contours [2, 8, 12, 21].

Preventive retention protocols include bonded lingual retainers with periodic replacement (every 3–5 years) and the application of chlorhexidine varnish at high-risk sites [13, 14, 16]. Digital monitoring tools enable remote plaque assessment, facilitating early intervention to prevent relapse [according to recent trends]. Long-term maintenance integrates ortho-perio recalls every 3–6 months, with probing and radiographic surveillance to detect early attachment changes [5, 7, 8].

Population-level prevention could incorporate alignment status into periodontal risk calculators, elevating malocclusion as a modifiable factor alongside smoking and diabetes [1, 2, 7]. Educational campaigns targeting general dentists should emphasize referral for ortho-perio evaluation in patients with combined malalignment and inflammation [3, 7].

Conclusion

This conceptual theory manuscript synthesizes the interdependence between tooth position, plaque retention, and periodontal stability into a coherent framework that positions orthodontic alignment as a primary modifier of periodontal risk. The proposed triad and five propositions provide a mechanistic rationale for observed clinical phenomena: alignment dismantles pathologic niches, reprograms microbial ecology, attenuates inflammation, and stabilizes architecture through biomechanical and biologic synergy.

Theoretical interpretation reveals the system as dynamic and homeostatic, with hysteresis overcome by timely intervention.

Clinically, the model advocates sequenced, interdisciplinary care that prioritizes periodontal stability, employs conservative mechanics, and customizes retention to preserve gains. Preventive applications extend benefits to at-risk populations, reframing orthodontics as a periodontal health strategy. Future research should quantify alignment-specific risk reduction using prospective cohorts that isolate tooth-position effects. Until then, this framework equips clinicians to harness orthodontic alignment for enhanced long-term periodontal outcomes, ultimately preserving natural dentition in an aging population with increasing periodontal vulnerability.

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