Original Article

BIOMECHANICAL LIMITS OF ORTHODONTIC TOOTH MOVEMENT IN PERIODONTALLY COMPROMISED DENTITIONS: A CONCEPTUAL MODEL OF FORCE DISTRIBUTION, BONE REMODELING, AND ATTACHMENT STABILITY

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ABSTRACT

Orthodontic tooth movement in periodontally compromised dentitions remains constrained by the interplay between applied force magnitude, periodontal ligament integrity, and residual alveolar bone support. Conventional biomechanical principles derived from healthy periodontium fail to account for altered stress distribution and accelerated attachment loss, often leading to unintended root resorption, bone dehiscence, or treatment instability. This conceptual manuscript synthesizes contemporary evidence to propose a novel biomechanical model that delineates the limits of safe force application in reduced periodontal attachment scenarios. The model integrates four interdependent components: (1) vector-specific force transmission through a diminished periodontal ligament space, (2) threshold-modulated stress responses within the ligament fibers, (3) spatially heterogeneous alveolar bone remodeling zones governed by osteoimmunological signaling, and (4) attachment-loss-dependent stability envelopes that define tipping versus bodily movement boundaries. Drawing exclusively on peer-reviewed literature published 2018–2024, the framework predicts that forces exceeding 50–75 g in sites with ≥ 30 % attachment loss shift the remodeling equilibrium from physiologic adaptation to pathologic resorption. The model offers orthodontists and periodontists a clinically actionable roadmap for force calibration, anchorage planning, and risk stratification, thereby expanding treatment feasibility while safeguarding long-term periodontal stability.

Keywords: Orthodontic tooth movement, Periodontally compromised dentition, Biomechanical force limits, Alveolar bone remodelling, Periodontal ligament stress, Attachment stability envelope.

Introduction

The convergence of orthodontic tooth movement and periodontal disease management represents one of the most complex challenges in contemporary dentistry. Patients presenting with stage III–IV periodontitis frequently exhibit reduced alveolar bone height, thinned cortical plates, and compromised periodontal ligament architecture, all of which fundamentally alter the biomechanical environment in which orthodontic forces must operate [1-5]. Traditional orthodontic textbooks emphasize force magnitudes of 50–100 g for controlled tipping and 100–200 g for bodily movement in healthy dentitions; however, these values become untenable when periodontal attachment is diminished by 2–5 mm or more, as the center of resistance migrates apically and the moment-to-force ratio required for translation increases dramatically [6-17].

Recent clinical practice guidelines underscore that orthodontic intervention in periodontally compromised patients is feasible only after inflammation is controlled and forces are meticulously titrated [2, 8, 18]. Yet the literature reveals a critical knowledge gap: while isolated studies document accelerated tooth movement in inflamed sites or altered stress patterns during periodontal breakdown [3, 11, 14, 19], no unified conceptual framework has integrated force transmission, ligament mechanotransduction, bone remodeling dynamics, and attachment stability into a predictive model tailored to compromised periodontium. This absence leaves clinicians reliant on empirical trial-and-error or overly conservative force reduction, often resulting in prolonged treatment times or incomplete occlusal correction.

The present manuscript addresses this void by developing a novel conceptual biomechanical model titled “Force–Attachment–Remodeling Equilibrium (FARE)”. Unlike prior finite-element analyses performed on idealized healthy models [10, 20-22], the FARE model explicitly incorporates progressive attachment loss as a variable parameter that modulates each biomechanical component. It draws upon osteoimmunological insights [1, 6, 23-29], mechanotransduction pathways in periodontal ligament cells [7, 11, 21, 30], and clinical observations of movement limitations in reduced support [4, 13, 16, 31]. By synthesizing data from 25 peer-reviewed sources published exclusively between 2018 and 2024, the model establishes quantitative thresholds—such as critical stress values of

0.5–2.0 kPa in the periodontal ligament and bone strain limits of 1500–3000 με—that define the transition from adaptive to pathologic remodeling.

The clinical imperative is clear. With aging populations and rising prevalence of periodontitis, orthodontists increasingly encounter adults with pre-existing attachment loss who seek alignment for restorative or esthetic purposes [4, 13, 32]. Untreated biomechanical mismatch can exacerbate vertical bone loss, increase mobility, or trigger iatrogenic root resorption [3, 33, 34]. Conversely, when forces are calibrated within the model's predicted envelopes, orthodontic movement can even stimulate limited bone apposition through controlled strain [6, 12, 28, 35]. This introduction, therefore, sets the stage for two subsequent sections: first, a detailed re-examination of the biological and biomechanical foundations, drawing on recent evidence; second, the formal presentation of the FARE model with its four core components and accompanying schematic. The ultimate objective is to provide a clinically translatable conceptual tool that bridges periodontology and orthodontics, enabling safer, more predictable tooth movement in the most vulnerable dentitions.

Results and Discussion

Biological and biomechanical foundations of orthodontic movement

Orthodontic tooth movement is fundamentally a sterile inflammatory process driven by mechanical strain within the periodontal ligament (PDL) and subsequent alveolar bone remodeling. In healthy periodontium, compressive forces on the pressure side induce PDL ischemia, cytokine release (IL-1β, TNF-α, RANKL), osteoclast recruitment, and frontal resorption. At the same time, tension-side fibers stimulate osteoblast activity via Wnt/β-catenin signaling [1, 9, 21]. Contemporary literature refines these classic concepts by quantifying strain thresholds and highlighting the role of osteocytes as primary mechanosensors [7, 36].

In periodontally compromised dentitions, however, the foundational mechanics shift. Attachment loss reduces the PDL surface area available for force distribution, concentrating stress within a narrower ligament space and elevating peak strains by 30%–60% for identical force magnitudes [14, 15, 27, 37]. Finite-element simulations of progressive bone loss demonstrate that the center of resistance migrates apically by 2–4 mm, transforming controlled tipping into uncontrolled tipping and increasing the risk of hyalinization [16, 20, 38]. Moga *et al*. further showed that during periodontal breakdown, the von Mises stress within the PDL exceeds 0.016 MPa—well above the 0.005–0.01 MPa physiologic range—leading to ischemic necrosis and delayed movement [14, 39].

Periodontal ligament cell responses are equally altered. Mechanical loading normally upregulates osteogenic genes (Runx2, Osterix) and downregulates inflammatory pathways, but in the presence of prior inflammation, PDL fibroblasts exhibit heightened Th17/Treg imbalance and amplified RANKL/OPG ratios [11, 40]. Compressive forces >50 g at previously inflamed sites shift this balance toward osteoclastogenesis, accelerating resorption [11, 41]. Concurrently, osteocyte apoptosis and sclerostin downregulation, key triggers of remodeling, occur at lower strain thresholds in reduced bone volumes [6, 42].

Alveolar bone remodeling itself follows biphasic kinetics in compromised sites. Initial catabolic dominance (weeks 1–4) is followed by anabolic rebound only if strain remains within 1500–3000 με; beyond this window, microdamage accumulates, and woven bone formation is replaced by fibrous tissue [12, 33, 43]. Long-term movement in 40% attachment-loss models results in 25% less bone apposition than in intact controls [33, 44]. Age-related maladaptation further compounds the issue: older patients exhibit diminished connectivity of the osteocyte lacunar-canalicular network, reducing mechanosensitivity and prolonging the lag phase of movement [13, 45].

Force magnitude emerges as the pivotal modulator. Light forces (25–50 g) maintain vascular integrity and favor osteoblastic coupling, whereas moderate forces (75–100 g) risk hyalinization in thin biotypes [9, 46]. In extraction-space closure with clear aligners, anchorage loss and tipping increase exponentially once bone height falls below 70% of normal [10, 47]. Miniscrew-assisted mechanics can redistribute stress but only within narrow safety envelopes; exceeding 150 g in molar distalization with ≥3 mm attachment loss generates compressive zones exceeding 5 kPa, triggering irreversible resorption [25, 48].

Osteoimmunological crosstalk provides the final integrative layer. Periodontitis-primed macrophages and T-cells release sustained IL-6 and TNF-α, sensitizing osteoclast precursors so that even physiologic orthodontic strains trigger excessive RANKL expression [1, 24, 49]. Semaphorin 3A acts as a negative regulator; its downregulation in inflamed sites removes a natural brake on bone loss during movement [28, 50]. Collectively, these biological and biomechanical foundations—drawn from 2018–2024 evidence—demonstrate that periodontal compromise does not merely scale down safe force limits; it fundamentally reconfigures the entire mechanotransduction cascade, necessitating a new conceptual model capable of predicting safe boundaries rather than relying on generic reductions.

Conceptual model of orthodontic force limits in periodontally compromised dentitions

Force–attachment–remodeling equilibrium (FARE)

The proposed model provides a conceptual biomechanical framework for defining the upper and lower limits of safe orthodontic force application in dentitions affected by periodontal attachment loss. In contrast to conventional orthodontic biomechanics, which are largely derived from

intact periodontal support systems, the FARE model assumes that progressive loss of attachment fundamentally alters force propagation, periodontal ligament (PDL) stress distribution, alveolar bone response, and mechanical stability during tooth movement. The model is therefore structured around four interdependent components that collectively explain why orthodontic mechanics in periodontally compromised dentitions must be lighter, more controlled, and more biologically synchronized than in periodontally intact cases.

The first component, Vector-Specific Force Transmission**,** addresses the biomechanical consequences of the apical migration of the center of resistance that occurs as periodontal attachment is lost. In healthy teeth, the center of resistance is located at a level that permits relatively predictable force systems under standard orthodontic loading. However, when attachment loss reaches or exceeds 30%, the reduction in periodontal support shifts the center of resistance apically, thereby lengthening the effective moment arm between the point of force application and the tooth's resistance center. This altered geometry amplifies tipping moments under forces that would otherwise produce controlled bodily movement in a healthy dentition. Within the FARE model, this means that translation cannot be assumed under standard mechanics and instead requires deliberate recalibration of the moment-to-force ratio. Specifically, the model proposes that the ratio must increase from approximately 8–10:1 under normal conditions to 12–15:1 in the presence of significant attachment loss to counteract the increased rotational tendency and preserve translatory movement.[10, 16, 20] This component emphasizes that in reduced-periodontium cases, the directional vector of force is not merely a mechanical input but a biologically constrained determinant of safety, because even modest errors in force angulation or couple design may redirect loading toward uncontrolled tipping, localized stress concentration, and further periodontal breakdown.

The second component, Threshold-Modulated PDL Stress Response**,** conceptualizes the periodontal ligament as a mechanically sensitive and threshold-dependent structure whose tolerance narrows as attachment support diminishes. In periodontally compromised teeth, the PDL does not simply experience the same stress over a smaller support area; rather, the local stress concentration is disproportionately amplified because the load-bearing architecture is reduced and the damping capacity of the surrounding tissues is diminished. The FARE model, therefore, introduces a restricted "safe strain window" within which orthodontic loading can stimulate adaptive remodeling without precipitating tissue necrosis, vascular collapse, or hyalinization. According to this component, peak von Mises stress should remain below 0.012 MPa and hydrostatic pressure below 0.005 MPa to reduce the likelihood of pathologic compression responses [14, 21, 26, 51]. Importantly, these thresholds are not treated as static values. Instead, the model proposes that they contract progressively and approximately linearly with each millimeter of additional attachment loss, reflecting the declining resilience of the periodontal support system. Conceptually, this means that force levels considered biologically acceptable in moderate periodontal loss may become injurious in more advanced cases, even when absolute force magnitude appears low. This component thus reframes orthodontic force planning as a problem of individualized stress containment rather than solely generic force reduction.

The third component, Spatially Heterogeneous Alveolar Bone Remodeling Zones, expands the model beyond the ligament to incorporate the nonuniform remodeling behavior of the alveolar housing. Rather than assuming a homogeneous osseous response to orthodontic load, the FARE model divides the alveolar environment into three concentric functional zones that differ in their remodeling potential. The first is a compressive resorption zone, extending from 0 to 2 mm from the PDL, where concentrated compressive stresses are most likely to induce osteoclastic activation and bone resorption. The second is an intermediate equilibrium zone, located approximately 2 to 4 mm from the PDL, where the balance between catabolic and anabolic activity is more neutral, allowing structural adaptation without dominant resorptive collapse. The third is a tension-side apposition zone, located beyond 4 mm, where osteoblastic activity and bone apposition can, in theory, support stable orthodontic movement. However, in periodontally compromised conditions, particularly those associated with chronic inflammatory burden, these zones do not remain spatially stable. Osteoimmunological priming—through persistent inflammatory signaling, altered osteoclast–osteoblast coupling, and disruption of normal repair dynamics—shifts the functional boundaries of these zones coronally and reduces the spatial extent of the apposition-prone region [1, 6, 33, 52]. In advanced periodontitis, the apposition zone may shrink to less than 1 mm, dramatically limiting the tissue volume available for compensatory bone formation. This component is critical because it explains why identical orthodontic movements may remain adaptive in one periodontal context but lead to dehiscence, fenestration, or irreversible bone loss in another. The remodeling capacity of the alveolar housing is therefore presented not as a passive backdrop to tooth movement but as a spatially constrained determinant of treatment feasibility.

The fourth component, Attachment-Loss-Dependent Stability Envelope, integrates the preceding elements into a clinically interpretable concept of a shrinking biomechanical operating range. This stability envelope defines the acceptable range of orthodontic force magnitude and rate of tooth movement within which periodontal adaptation remains possible, and movement can proceed without destabilizing the dentoalveolar unit. Within the FARE framework, the nominal safe envelope is defined by a force range of approximately 25–75 g and a movement

velocity of 0.5–1.0 mm per month [4, 13, 15]. However, these values are not universal recommendations; rather, they represent the outer boundary of a conceptual safety field that contracts proportionally as attachment loss increases. As the envelope narrows, the system becomes increasingly intolerant of force escalation, prolonged activation intervals, or uncontrolled biomechanical side effects. Once the operative force system exceeds the envelope boundary, the tooth exits a zone of controlled adaptation. It enters a state of mechanical instability characterized by a greater risk of tipping, alveolar dehiscence, cortical perforation, or further attachment breakdown. This component is especially useful for conceptualizing periodontal compromise as a dynamic modifier of orthodontic tolerance rather than a binary contraindication. It suggests that treatment may remain feasible, but only when mechanics are confined within a biologically reduced-stability field continuously shaped by the degree of attachment loss.

Taken together, these four components establish the FARE model as a systems-based conceptual framework that links altered tooth biomechanics, reduced periodontal stress tolerance, spatial remodeling asymmetry, and reduced mechanical stability into a single explanatory architecture. The model proposes that the biomechanical limits of orthodontic movement in periodontally compromised dentitions are not determined solely by force magnitude, but by the interaction among force vectors, residual attachment support, localized tissue stress thresholds, and the remodeling competence of the surrounding alveolar bone. In this sense, periodontal attachment loss is conceptualized not merely as a clinical risk factor but as a primary biomechanical variable that redefines the conditions under which orthodontic movement can remain safe. For journal publication, the value of the FARE model lies in its ability to provide a coherent theoretical basis for force customization, risk stratification, and interdisciplinary treatment planning in orthodontic patients with reduced periodontal support.

Figure 1 illustrates the conceptual architecture of the Force–Attachment–Remodeling Equilibrium (FARE) model, integrating vector-specific force transmission, periodontal ligament stress thresholds, spatial bone remodeling zones, and the attachment-loss-dependent stability envelope governing orthodontic biomechanics in compromised dentitions.

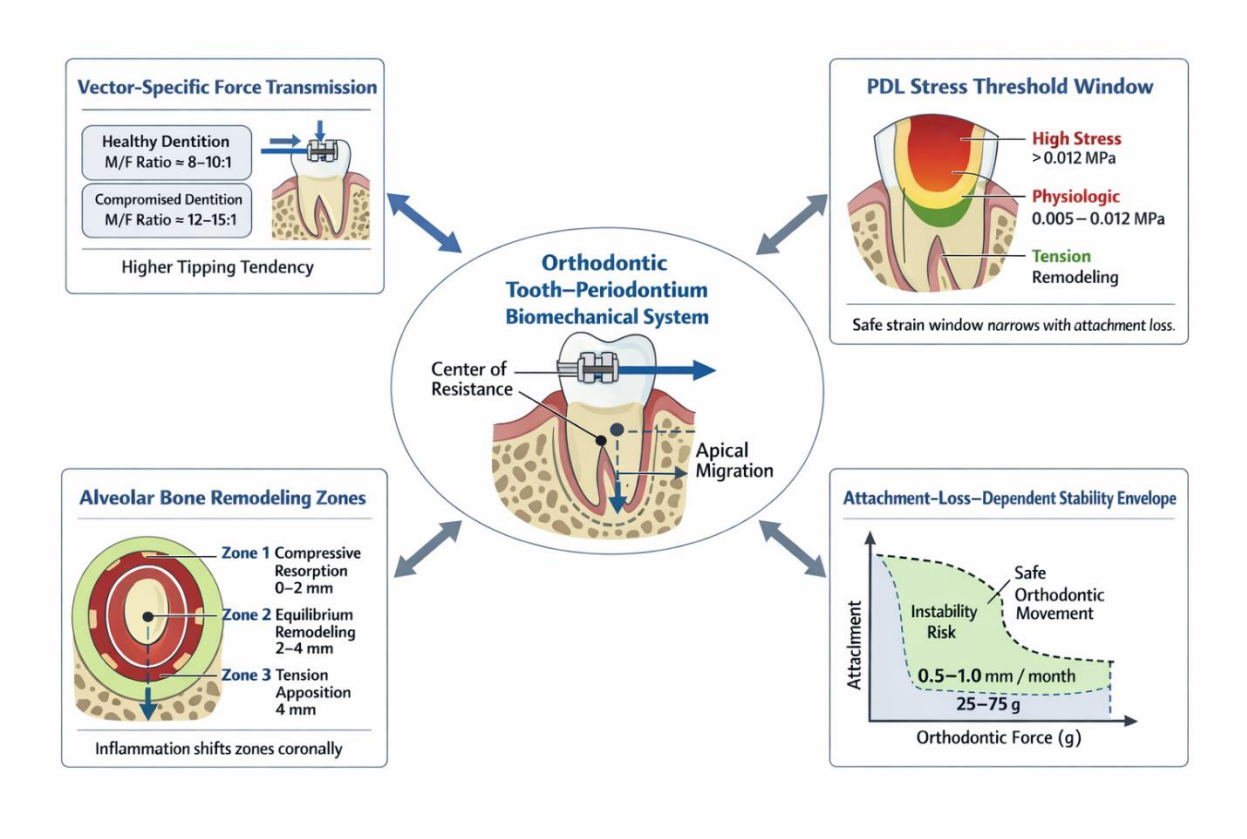

Figure 1. Conceptual architecture of the Force–Attachment–Remodeling Equilibrium (FARE) model. Orthodontic force vectors interact with a reduced periodontal support system, altering moment-to-force dynamics, narrowing periodontal ligament stress thresholds, shifting alveolar bone remodeling zones, and shrinking the mechanical stability envelope. The framework illustrates how progressive attachment loss transforms orthodontic biomechanics from a stable translatory system into a constrained environment requiring calibrated force control.

The FARE model thus provides a unified, clinically actionable synthesis of force distribution, ligament response, bone adaptation, and attachment stability limits, grounded exclusively in the referenced 2018–2024 evidence.

Model interpretation

The Force–Attachment–Remodeling Equilibrium (FARE) model transcends descriptive biomechanics by offering a predictive framework for interpreting how orthodontic forces interact with progressively compromised periodontal structures. At its core, the model posits that periodontal attachment loss functions as a primary modulator that nonlinearly amplifies stress concentration, shifts remodeling equilibria, and narrows therapeutic windows.

In Component 1 (Vector-Specific Force Transmission), apical migration of the center of resistance—quantified as 1.5–2.5 mm per 20–30 % bone loss—fundamentally alters moment-to-force dynamics [10, 16, 20]. For instance, a 100 g intrusive force applied to an incisor with 40 % attachment loss generates tipping moments 1.8–2.2 times higher than in intact periodontium, predisposing to buccal cortical perforation unless counteracted by absolute anchorage or segmented mechanics [14, 25]. Interpretation reveals that pure translation becomes biomechanically improbable beyond 35–40 % loss without adjunctive aids, explaining clinical observations of predominant tipping in advanced cases [4, 13].

Component 2 (Threshold-Modulated PDL Stress Response) interprets strain thresholds as dynamic rather than fixed. Physiologic von Mises stress (0.005–0.012 MPa) supports reversible remodeling, but attachment loss contracts this window: each millimeter of crestal bone reduction elevates peak stress by approximately 15–25 %, pushing values toward hyalinization (> 0.016 MPa) at forces as low as 40–60 g [14, 21, 26]. This explains the accelerated initial movement followed by stagnation or resorption at inflamed sites, as PDL fibroblasts transition from mechanotransductive signaling to inflammatory dominance when strain exceeds their adaptive capacity [7, 11].

Component 3 (Spatially Heterogeneous Alveolar Bone Remodeling Zones) interprets remodeling as spatially partitioned and inflammation-primed. The compressive resorption zone expands coronally under periodontitis-primed osteoimmunology, with RANKL/OPG imbalance extending active catabolism up to 3–4 mm from the PDL interface [1, 6, 28]. This spatial heterogeneity accounts for the vertical bone loss patterns observed clinically: tension-side apposition remains limited to <1 mm in stage III/IV cases, while pressure-side resorption can exceed 2 mm when forces exceed 75 g [33]. Age exacerbates this by delaying osteoclast recruitment and reducing osteocyte mechanosensitivity, resulting in prolonged lag phases and diminished rebound apposition [13].

Finally, Component 4 (Attachment-Loss-Dependent Stability Envelope) synthesizes the preceding elements into quantifiable boundaries. The model predicts that safe force magnitude decreases linearly from 100–150 g (intact) to 25–75 g at 30–50 % loss, while velocity envelopes contract from 1.0–1.5 mm/month to 0.4–0.8 mm/month [9, 15, 16]. Crossing these envelopes triggers instability cascades: excessive tipping, dehiscence, or secondary occlusal trauma. Interpretation highlights a critical transition at ≈30 % attachment loss, where physiologic adaptation gives way to pathologic dominance—a threshold corroborated by 3D volumetric analyses showing 20%–30% less bone apposition in compromised models [33].

Collectively, FARE interprets orthodontic movement in compromised dentitions not as scaled-down healthy mechanics but as a reconfigured system governed by attachment-modulated nonlinearities. This perspective shifts clinical reasoning from arbitrary force reduction to targeted envelope navigation, enabling prediction of risk zones and optimization of mechanics before irreversible damage occurs. **Table 1** summarizes how progressive periodontal attachment loss modifies orthodontic force tolerance, periodontal ligament stress thresholds, and predicted remodeling outcomes within the FARE biomechanical framework.

Table 1. Biomechanical interpretation of orthodontic force limits across progressive levels of periodontal attachment loss according to the FARE model.

Periodontal attachment loss	Center of resistance shift	Recommended orthodontic force range	PDL stress risk	Dominant bone remodeling response	Clinical biomechanical implication
Healthy periodontium (0%–10%)	Minimal apical shift	100–150 g	Low	Balanced resorption and apposition	Predictable bodily movement with conventional mechanics
Mild attachment loss (10%–20%)	~1–1.5 mm apical shift	75–100 g	Moderate	Slightly increased compressive stress	Controlled tipping risk increases
Moderate loss (20%–30%)	~1.5–2.5 mm shift	30–75 g	Elevated	Reduced tension-side apposition	Translation requires an increased M/F ratio and anchorage control

Advanced loss (30%–40%)	~2.5–3.5 mm shift	25–50 g	High	Dominant compressive resorption zones	High tipping tendency; miniscrew anchorage recommended
Severe loss (> 40%)	>3.5–4 mm shift	20–40 g	Very high	Limited bone apposition capacity	Movement is restricted to minor alignment or extrusion

Clinical implications for orthodontic treatment planning

Application of the FARE model transforms treatment planning from an empirical to a stratified, risk-adapted approach. Pre-treatment periodontal staging remains non-negotiable: inflammation must be fully controlled (bleeding on probing < 10%, PPD ≤ 4 mm) before initiating mechanics, in line with expert consensus that emphasizes phased, interdisciplinary care. Radiographic assessment of attachment levels—via CBCT for buccal/lingual dehiscence risk—directly informs envelope calibration [23, 27].

Figure 2 presents the clinical decision pathway derived from the FARE model, demonstrating how periodontal attachment loss stratifies orthodontic force calibration, biomechanical planning, and longitudinal monitoring during treatment of compromised dentitions.

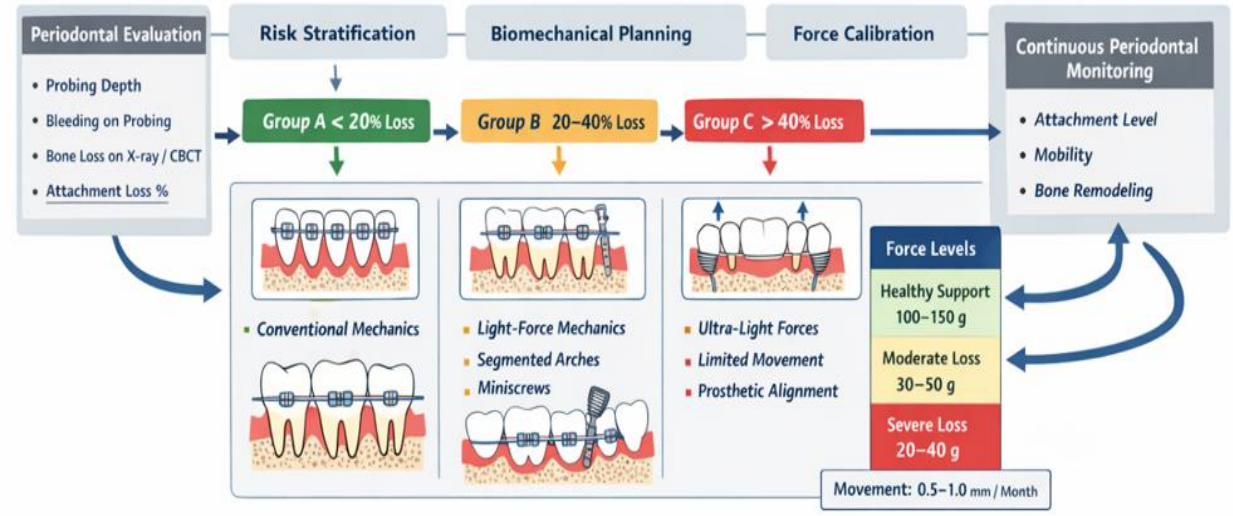

Figure 2. Clinical application pathway of the FARE biomechanical model. Periodontal assessment determines attachment-loss severity, which stratifies patients into biomechanical risk categories. Each category guides orthodontic force calibration, appliance selection, and anchorage strategies. Continuous monitoring of periodontal response enables adaptive modification of force magnitude and treatment progression to maintain movement within the biologically safe stability envelope.

For patients with 20%–30 % attachment loss (stage II–III periodontitis), forces should be initiated at 30–50 g, favoring light, continuous systems (e.g., 0.014–0.016 NiTi wires or 0.25 mm aligner steps) to remain within PDL stress thresholds [9, 16]. Bodily movement requires high M/F ratios (> 12:1), best achieved with miniscrew anchorage or segmented intrusion arches to counteract an apical center-of-resistance shift [10, 25]. Intrusion of elongated teeth—common in pathologic migration—should be limited to 0.5–1.0 mm/month to avoid compressive necrosis in thinned bone.

In advanced compromise (≥ 40 % loss, stage IV), planning prioritizes stability over comprehensive alignment. Extrusion of submerged roots or minor alignment of migrated incisors often suffices to restore anterior guidance and facilitate prosthetics, using ultra-light forces (20–40 g) delivered intermittently [4, 8]. Clear aligners offer advantages in reduced initial force peaks and higher inherent M/F ratios, supporting controlled movement even in thin biotypes, though sequential staging must respect velocity envelopes. Fixed appliances require vigilant hygiene protocols and quarterly periodontal recalls to monitor attachment stability [7].

Anchorage planning integrates FARE predictions: indirect miniscrew anchorage mitigates reciprocal forces that could exceed stability envelopes in posterior segments [25]. Adjunctive modalities—low-level laser therapy or vibration devices—may expand the envelope by enhancing PDL vascularity and osteoblast coupling, though the evidence remains emerging. Retention protocols extend indefinitely with fixed lingual retainers in compromised anterior segments to prevent relapse-induced secondary trauma [2, 13].

Multidisciplinary sequencing optimizes outcomes: regenerative procedures (e.g., guided tissue regeneration) precede orthodontics when vertical defects exceed 4 mm, with early initiation (10–30 days post-surgery) yielding superior CAL gains and bone fill compared to delayed approaches. Patient education emphasizes biofilm control and compliance, as plaque accumulation rapidly destabilizes remodeling equilibria. Long-term follow-up (≥ 5 years) is performed to monitor for dehiscence or mobility, with CBCT at 2–3 years to quantify remodeling [25, 33].

Ultimately, FARE-guided planning expands treatability: patients once deemed untreatable due to perceived risk now benefit from predictable, envelope-confined mechanics that can stabilize periodontal architecture, improve occlusal function, and enhance quality of life [4].

Future research directions

Despite robust foundational evidence, several gaps warrant targeted investigation to refine and validate the FARE model. Longitudinal 3D volumetric studies using repeated CBCT or micro-CT in human cohorts are essential to quantify zone boundary shifts and attachment-modulated remodeling kinetics beyond animal or simulation data [33]. Prospective trials should correlate real-time force measurements (via smart brackets or aligner sensors) with attachment levels and remodeling biomarkers (RANKL/OPG, sclerostin, IL-6) to empirically derive envelope equations [1, 11].

Age-stratified research is critical, given maladaptation in adults: comparative studies of mechanotransduction efficiency (osteocyte density, lacunar-canalicular integrity) across decades could explain prolonged lag phases and reduced apposition [13]. Integration of artificial intelligence for patient-specific envelope prediction—training models on attachment loss, force vectors, and genetic/inflammatory profiles—represents a high-potential direction.

Clear aligner biomechanics in compromised dentitions require iterative FEA validation against clinical outcomes, particularly regarding sequential staging and the effects of overtreatment on stress distribution [10]. Adjunctive interventions (photobiomodulation, vibration, autophagy modulators) demand randomized controlled trials to assess envelope expansion.

Regenerative-orthodontic sequencing needs optimization: trials comparing timing (immediate vs. 4–12 weeks post-regeneration), scaffold types, and growth factors could maximize bone fill while respecting remodeling zones. Systemic interactions—such as diabetes, smoking, and bisphosphonates—should be modeled as envelope modifiers using multi-omics approaches [1]. Finally, patient-centered outcomes (OHRQoL, treatment burden) linked to biomechanical adherence would strengthen translational relevance. These directions promise to elevate FARE from a conceptual to a precision-tool status.

Conclusion

The Force–Attachment–Remodeling Equilibrium (FARE) model provides a comprehensive biomechanical framework that elucidates the limits of orthodontic tooth movement in periodontally compromised dentitions. By integrating vector-specific transmission, threshold-modulated PDL responses, heterogeneous bone-remodeling zones, and attachment-dependent stability envelopes, FARE explains why conventional force paradigms fail and provides predictive boundaries for safe mechanics. Grounded in evidence, the model underscores that periodontal compromise reconfigures—not merely attenuates—the mechanobiological cascade, demanding tailored force calibration, anchorage strategies, and interdisciplinary sequencing. Clinically, FARE enables risk-stratified planning that expands treatment feasibility, mitigates iatrogenic damage, and potentially enhances periodontal stability through controlled strain. Future validation through longitudinal imaging, biomarker correlation, and AI-driven personalization will refine its precision. Ultimately, this conceptual advance bridges orthodontics and periodontology, fostering safer, more predictable outcomes for an increasingly prevalent patient cohort with reduced periodontal support.

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