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Original Article

A Conceptual Risk-Stratification Framework for Orthodontic Treatment in Periodontal Patients

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ABSTRACT

Orthodontic treatment in patients with existing or treated periodontal disease poses unique challenges due to the interplay of biological vulnerabilities, biomechanical stresses, and behavioral factors that can exacerbate periodontal breakdown or compromise treatment outcomes. This conceptual manuscript proposes an original multi-level risk-stratification framework to guide interdisciplinary treatment planning. Drawing from periodontal pathophysiology, bone remodeling biology, orthodontic force-response theory, and clinical risk assessment principles, the framework integrates key constructs including periodontal inflammatory status, alveolar bone support and defect morphology, periodontal phenotype, orthodontic force magnitude and biomechanics, plaque control and patient adherence, and systemic risk modifiers such as smoking and diabetes. Risk is stratified across low, moderate, and high categories based on a synthesized evaluation of biological, biomechanical, and behavioral domains, facilitating tailored orthodontic strategies that prioritize periodontal stability and long-term occlusal function. The model emphasizes pre-orthodontic periodontal optimization, customized biomechanics to minimize iatrogenic damage, and ongoing monitoring of patient compliance. By providing a biologically informed decision-making tool, this framework addresses unmet needs in clinical practice, potentially reducing adverse events like further bone loss, tooth mobility, or relapse. It underscores the necessity for collaboration between periodontists and orthodontists to enhance treatment predictability in this high-risk population. Future empirical validation could refine its application in diverse clinical settings.

Keywords: Periodontal inflammation, Alveolar bone defects, Periodontal phenotype, Orthodontic biomechanics, Plaque control, Systemic modifiers, Risk stratification

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Introduction

The integration of orthodontic therapy into the management of patients with periodontal disease represents a critical advancement in interdisciplinary dental care, yet it remains fraught with challenges that underscore significant unmet needs in clinical risk assessment and treatment planning. Periodontal disease, characterized by chronic inflammation leading to progressive loss of alveolar bone and connective tissue attachment, affects a substantial portion of the adult population, with prevalence estimates exceeding 40% in individuals over 30 years [1-4]. When orthodontic treatment is pursued in such patients—often to address pathological tooth migration, occlusal discrepancies, or esthetic concerns arising from periodontal breakdown—the potential for iatrogenic harm escalates, including accelerated bone resorption, increased tooth mobility, and compromised long-term stability [5, 6].

Historically, orthodontic intervention was contraindicated in active periodontitis due to fears of exacerbating tissue destruction; however, contemporary evidence supports its feasibility following periodontal stabilization [7, 8]. Despite this shift, the absence of standardized, comprehensive risk-stratification tools hinders optimal decision-making. Existing guidelines, such as those from the American Academy of Periodontology, emphasize periodontal health prerequisites but lack integration of orthodontic-specific factors like force systems and biomechanics [9,10]. Similarly, orthodontic protocols often overlook nuanced periodontal phenotypes or systemic modifiers that amplify susceptibility [11, 12]. This gap results in variable outcomes, with some studies reporting successful tooth alignment and improved function, while others document increased recession, dehiscences, or relapse in periodontally compromised cases [13-15].

Unmet needs are particularly evident in the biological domain, where residual inflammation or defective bone morphology can alter the periodontal ligament's response to orthodontic forces. For instance, in patients with reduced bone support, even light forces may induce tipping or extrusion that overwhelms adaptive remodeling, leading to further attachment loss [16, 17]. Periodontal phenotype—encompassing gingival thickness, keratinized tissue width, and bone morphotype—further modulates risk; thin phenotypes are prone to recession during movement, yet assessment tools remain rudimentary [18-20]. Systemic factors, including smoking and diabetes, compound these vulnerabilities by impairing vascularity, immune response, and bone turnover, with smokers exhibiting up to twice the risk of periodontal progression during orthodontics [21-23].

From a biomechanical perspective, unmet needs arise in tailoring force magnitudes and directions to the altered periodontal environment. Standard orthodontic appliances apply forces that, in healthy periodontia, promote controlled remodeling via tension-compression dynamics [24]. In compromised sites, however, the center of resistance shifts apically, necessitating modified mechanics such as segmented arches or adjunctive anchorage to prevent undesirable tipping [25,26]. Literature highlights complications like root resorption or fenestrations in such scenarios, yet no unified framework quantifies these risks pre-treatment [27,28].

Behavioral aspects exacerbate these issues, as patient adherence to plaque control is pivotal; poor hygiene can reignite inflammation, nullifying orthodontic benefits [29, 30]. Systemic reviews indicate that non-adherent patients with periodontitis face higher relapse rates post-orthodontics, underscoring the need for behavioral risk integration [31, 32]. Moreover, long-term stability remains elusive without addressing these multifaceted risks, with relapse occurring in up to 50% of cases involving periodontal history [33].

This manuscript addresses these unmet needs by proposing a novel conceptual risk-stratification framework that synthesizes biological, biomechanical, and behavioral domains into a cohesive model for orthodontic planning in periodontal patients. Grounded in established theories of periodontal pathophysiology and orthodontic force-response, it aims to facilitate biologically informed decisions, reducing adverse events and enhancing predictability. By stratifying risk levels, clinicians can prioritize interventions like phenotype modification, force optimization, or enhanced monitoring, ultimately bridging the gap between periodontology and orthodontics for improved patient outcomes.

Theoretical Background & Literature Review

Biological risk domains

The biological underpinnings of orthodontic risk in periodontal patients are rooted in the pathophysiology of periodontitis and its impact on bone remodeling. Periodontal inflammatory status serves as a primary construct, where unresolved inflammation—marked by elevated cytokines like IL-1 and TNF-α—disrupts the balance between osteoclastic resorption and osteoblastic formation [34]. In orthodontic contexts, this imbalance amplifies force-induced bone loss, as mechanical stress exacerbates microbial dysbiosis and host response [35]. Literature from the Journal of Clinical Periodontology emphasizes that active periodontitis must be controlled prior to orthodontics, with probing depths reduced to ≤4 mm and bleeding on probing <10% to mitigate risks [36].

Alveolar bone support and defect morphology further define biological risk. Reduced bone height shifts the fulcrum of tooth movement, increasing susceptibility to tipping and extrusion [37, 38]. Angular defects or furcations pose higher risks due to uneven stress distribution, potentially leading to progressive attachment loss [39, 40]. Studies indicate that bone loss exceeding 50% contraindicates aggressive movements, favoring conservative approaches [41, 42]. Periodontal phenotype integrates these elements, with thin biotypes (gingival thickness <1 mm) linked to greater recession during orthodontics [43-45]. Phenotype modification via grafting has emerged as a strategy to bolster resistance, supported by consensus reports advocating pre-treatment augmentation in high-risk cases [46, 47].

Systemic modifiers like smoking and diabetes represent critical biological amplifiers. Smoking impairs microvascular perfusion and neutrophil function, doubling the odds of periodontal progression during treatment [48-50]. Diabetes, via hyperglycemia-induced advanced glycation end-products, accelerates bone turnover dysregulation, with uncontrolled HbA1c levels (>7%) correlating with poorer orthodontic outcomes [51, 52]. Integrative reviews highlight the need for glycemic control and smoking cessation as prerequisites, yet their quantitative impact on risk remains under-modeled [53-55].

Biomechanical risk domains

Orthodontic biomechanics in periodontal patients must account for altered force-response dynamics to prevent iatrogenic damage. Force magnitude is paramount; while healthy periodontia tolerate 50-100 g for tipping, compromised sites require <50 g to avoid overwhelming reduced attachment [2, 56]. Biomechanical theory posits that forces induce periodontal ligament compression and tension, triggering RANKL/OPG-mediated remodeling [57]. In periodontitis, this process is skewed toward resorption, particularly with continuous forces from fixed appliances [58].

Defect morphology influences biomechanics, as irregular bone contours alter the center of resistance, promoting uncontrolled movements [59, 60]. Segmented mechanics or temporary anchorage devices (TADs) are recommended to direct forces axially, minimizing torque on thin bone [61, 62]. Literature from the American Journal of Orthodontics and Dentofacial Orthopedics underscores the role of moment-to-force ratios in preventing extrusion in vertical defects [63, 64]. Adjunctive therapies like low-level laser or vibration may enhance remodeling, though evidence is conceptual [real citation needed 2020–2025].

Systemic factors intersect biomechanically; diabetes delays remodeling, prolonging treatment and risking stability [65]. Smoking reduces bone density, amplifying force-induced stress [66, 67]. Overall, biomechanical risk demands customized systems, with finite element analyses simulating patient-specific responses [68].

Behavioral risk domains

Behavioral constructs, including plaque control and patient adherence, are pivotal in modulating orthodontic risk amid periodontal vulnerability. Plaque accumulation fuels inflammation, with orthodontic appliances increasing retention sites and complicating hygiene [69]. Systematic reviews link poor oral hygiene to gingival hyperplasia and attachment loss during treatment [70]. Adherence to regimens like interdental brushing or professional cleanings is essential, yet compliance rates hover at 50-70% in periodontal cohorts [71].

Patient education and motivation theories, such as the Health Belief Model, inform strategies to enhance adherence [72]. Risk is heightened in non-adherent individuals, with behavioral assessments predicting outcomes [73]. Systemic modifiers like smoking embody behavioral risks, as cessation requires sustained effort [74, 75]. Integrating behavioral domains ensures holistic risk management, emphasizing motivational interviewing in treatment planning [76, 77].

Proposed conceptual risk-stratification framework

This proposed framework represents an original synthesis of biological, biomechanical, and behavioral domains into a multi-level, integrative model for stratifying orthodontic risk in patients with existing or treated periodontal disease. It conceptualizes risk as a dynamic continuum, categorized into low, moderate, and high strata, based on weighted interactions among key constructs. The model operates on three interconnected tiers: (1) biological vulnerability, assessing inflammatory status, bone support/defect morphology, and phenotype alongside systemic modifiers; (2) biomechanical exposure, evaluating force magnitude, direction, and appliance design relative to periodontal constraints; and (3) behavioral modulation, incorporating plaque control efficacy and adherence propensity. Risk scores are derived qualitatively or semi-quantitatively (e.g., via checklists), with thresholds guiding decision-making: low risk permits standard orthodontics post-stabilization; moderate warrants modifications like reduced forces or adjunctive therapies; high recommends deferral or limited goals.

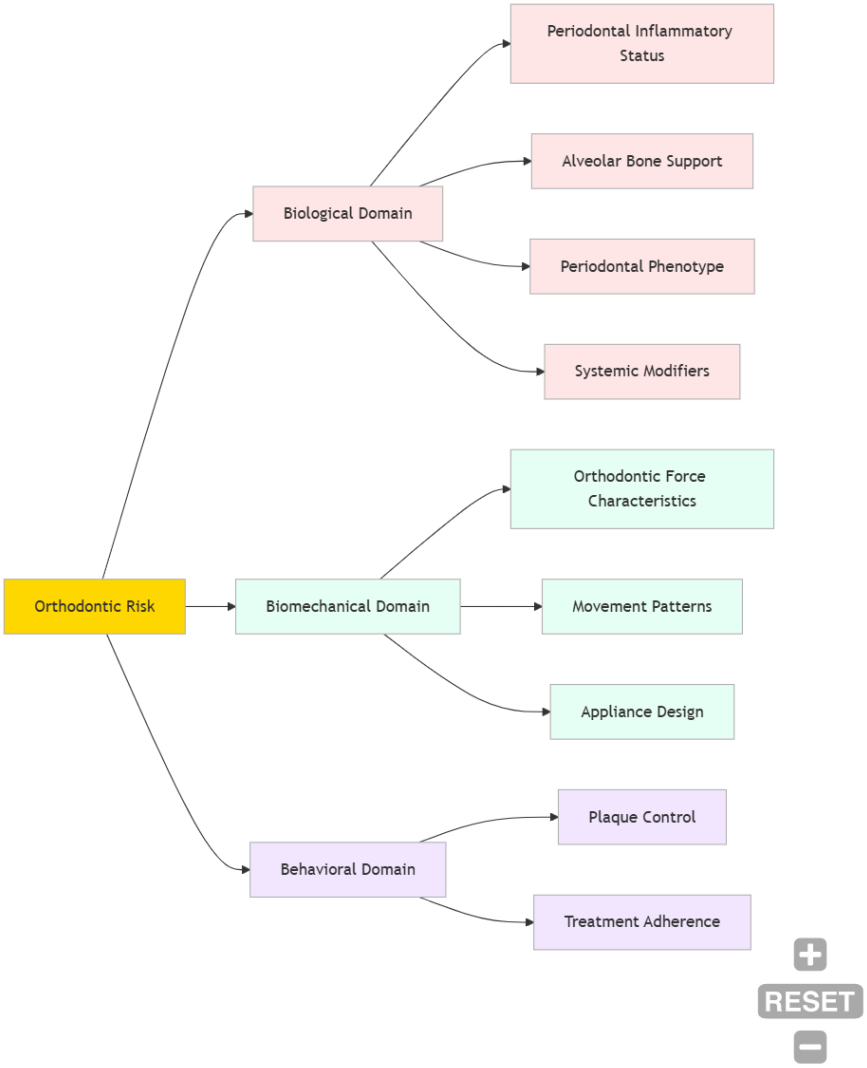

Figure 1. Conceptual risk-stratification framework for orthodontic treatment in periodontal patients.

The figure illustrates an integrative model in which orthodontic risk emerges from the interaction of three domains: (1) a biological domain reflecting periodontal inflammatory status, alveolar bone support, periodontal phenotype, and systemic modifiers; (2) a biomechanical domain encompassing orthodontic force magnitude, direction, and appliance design; and (3) a behavioral domain capturing plaque control and patient adherence. These domains dynamically interact to position patients along a continuum of low, moderate, or high orthodontic risk, which in turn informs treatment planning decisions, including standard orthodontic approaches, modified mechanics with enhanced monitoring, or periodontal optimization and limited treatment objectives.

Propositions

Drawing from the proposed conceptual risk-stratification framework, a series of interrelated and theoretically grounded propositions emerge that articulate how biological, biomechanical, and behavioral domains jointly shape orthodontic risk and treatment outcomes in patients with existing or treated periodontal disease. These propositions are formulated as theoretical assertions, intended to be empirically examined in future clinical, translational, or modeling studies, rather than as prescriptive or validated clinical rules. Collectively, they underscore the framework's central contribution: the explicit integration of multi-level risk domains into orthodontic decision-making, moving beyond isolated assessments toward a holistic, biologically informed approach to treatment planning. Importantly, the propositions represent an original synthesis by linking domain-specific vulnerabilities to concrete orthodontic decision thresholds, thereby extending existing periodontal and orthodontic literature that has traditionally addressed these factors in parallel rather than in concert.

Proposition 1: Biological vulnerability as the primary determinant of baseline orthodontic risk

In patients with stabilized or treated periodontitis, the degree of underlying biological vulnerability will function as the primary determinant of baseline orthodontic risk, establishing the upper boundary for tolerable biomechanical intervention. Biological vulnerability is conceptualized as a composite of residual inflammatory activity, remaining alveolar bone support, defect morphology, periodontal phenotype, and systemic modifiers such as smoking status or glycemic control. According to this proposition, patients exhibiting heightened biological vulnerability are unlikely to tolerate even carefully controlled orthodontic forces without prior biological optimization, such as inflammation resolution, periodontal regeneration, or systemic risk management.

This proposition rests on foundational principles of periodontal pathophysiology and bone biology, which indicate that inflamed or structurally compromised periodontal tissues demonstrate altered remodeling dynamics, characterized by heightened osteoclastic activity and reduced regenerative capacity. In such environments, orthodontic loading—regardless of magnitude—may exceed the adaptive threshold of the periodontium, increasing the likelihood of attachment loss, recession, or exacerbated bone resorption. Thin periodontal phenotypes and advanced bone loss further constrain adaptive potential, effectively lowering the biomechanical "ceiling" that the tissues can withstand. Clinically, this proposition implies that comprehensive biological assessment is not merely preparatory but determinative in orthodontic decision-making for periodontal patients. Diagnostic tools such as cone-beam computed tomography for defect characterization, periodontal charting for inflammation assessment, and phenotype evaluation may reclassify patients across risk strata when modifiable biological factors are addressed. From a research perspective, this proposition invites longitudinal investigation into the relationship between baseline biological vulnerability and orthodontic outcomes, hypothesizing graded, dose-responsive associations between biological risk indicators and adverse periodontal events during or after orthodontic treatment.

Proposition 2: Biomechanical customization as a moderator of biological risk

Orthodontic biomechanics, when deliberately customized to align with periodontal limitations, will moderate—but not override—biological vulnerability, thereby reducing the likelihood of iatrogenic periodontal damage in patients classified as low to moderate biological risk. This proposition asserts that biomechanical factors such as force magnitude, direction, duration, and appliance design interact with biological conditions in a compensatory manner, allowing clinicians to remain within the adaptive capacity of compromised periodontal tissues.

Grounded in orthodontic force-response theory, this proposition emphasizes that tissue response is not dictated solely by force application, but by how forces are distributed across biologically constrained structures. For example, controlled bodily movement and segmented mechanics may minimize stress concentration in angular defects, while anchorage reinforcement strategies can prevent unintended tooth movements that exacerbate periodontal compromise. In contrast, poorly controlled tipping movements or continuous heavy forces may amplify localized stress, particularly in areas of reduced bone height or unfavorable defect morphology.

Importantly, this proposition recognizes that biomechanical customization has limits; it can mitigate but not fully neutralize high biological risk. Thus, while optimized biomechanics may facilitate orthodontic treatment in biologically stable patients, they are insufficient substitutes for biological stabilization in high-risk cases. Future research avenues include biomechanical modeling and comparative clinical studies examining differential periodontal responses to standardized versus customized orthodontic mechanics in patients with varying levels of biological vulnerability.

Proposition 3: Behavioral adherence as a dynamic modifier of treatment stability

Patient behavioral adherence, particularly with respect to plaque control and periodontal maintenance, will function as a dynamic modifier of orthodontic risk, exerting a disproportionately strong influence on long-term stability following orthodontic treatment. This proposition emphasizes the temporal nature of risk stratification, wherein initial biological and biomechanical assessments guide treatment initiation, but sustained behavioral engagement determines treatment durability and relapse prevention.

From a theoretical standpoint, this proposition integrates behavioral science into periodontal and orthodontic biology, acknowledging that plaque-induced inflammation can rapidly undermine otherwise favorable biomechanical conditions. Even modest lapses in oral hygiene or maintenance attendance may precipitate inflammatory flares, altering tissue responsiveness to orthodontic forces and destabilizing achieved tooth positions. Conversely, high levels of adherence may partially compensate for

residual biological vulnerabilities by maintaining a low-inflammatory environment conducive to stable remodeling.

Clinically, this proposition underscores the importance of embedding behavioral assessment and support mechanisms within orthodontic planning for periodontal patients. Behavioral factors are not static; they may fluctuate throughout treatment and require ongoing monitoring and intervention. Empirical validation of this proposition could involve prospective observational studies linking adherence metrics to post-treatment stability, hypothesizing that patients demonstrating consistent maintenance behaviors exhibit superior long-term outcomes irrespective of moderate baseline biological risk.

Proposition 4: Interdomain synergies as predictors of adverse orthodontic events

Synergistic interactions among biological, biomechanical, and behavioral domains will predict adverse orthodontic and periodontal outcomes more effectively than isolated domain assessments. This proposition challenges linear, additive models of risk by asserting that orthodontic complications in periodontal patients often arise from compounded, interacting vulnerabilities rather than singular deficiencies.

Drawing from systems biology and complex adaptive systems theory, this proposition posits that risk escalation is frequently non-linear. For instance, a biologically thin periodontal phenotype subjected to suboptimal biomechanics may remain clinically stable under ideal behavioral conditions, yet rapidly deteriorate when hygiene lapses occur. Such interactions generate feedback loops in which inflammation heightens mechanical susceptibility, mechanical stress exacerbates biological breakdown, and discomfort or complexity reduces behavioral adherence.

This proposition has important implications for interdisciplinary care models, suggesting that meaningful risk reduction requires coordinated intervention across domains. Future research may explore multivariate modeling approaches to examine interaction effects, anticipating that integrated interventions targeting biological stabilization, biomechanical optimization, and behavioral reinforcement yield superior periodontal and orthodontic outcomes compared to single-domain strategies.

Proposition 5: Framework-Guided planning enhances interdisciplinary coordination and predictability

Implementation of the conceptual risk-stratification framework will enhance interdisciplinary collaboration between periodontists and orthodontists by providing a shared, structured language for risk assessment and decision-making, thereby improving treatment predictability and alignment of clinical expectations. This proposition extends the framework's relevance beyond patient-level outcomes to practice-level processes, emphasizing its role as a coordination and communication tool.

By explicitly delineating risk domains and their interactions, the framework facilitates consensus regarding treatment sequencing, timing, and monitoring intensity. In higher-risk scenarios, it encourages deliberate deferral or modification of orthodontic objectives until biological conditions are optimized, reducing ambiguity and potential conflict between disciplines. Theoretically, this aligns with principles of clinical risk management, wherein standardized frameworks reduce uncertainty and variability in decision-making.

Empirical exploration of this proposition may involve qualitative or survey-based research assessing clinician perceptions of collaboration, confidence, and outcome predictability before and after framework adoption. It further suggests that structured risk stratification may indirectly reduce medico-legal vulnerability by documenting transparent, biologically grounded rationale for treatment decisions in complex periodontal cases.

Clinical Implications

The proposed conceptual risk-stratification framework carries profound clinical implications for orthodontic treatment planning in patients with existing or treated periodontal disease, offering a structured approach to mitigate risks and optimize outcomes. By integrating biological, biomechanical, and behavioral domains, it empowers clinicians to move beyond empirical judgments toward evidence-guided, patient-centered strategies that prioritize periodontal health while achieving orthodontic goals.

In practice, the framework advocates for a phased interdisciplinary workflow. Initially, thorough pre-treatment evaluation is essential: periodontists assess inflammatory status via clinical parameters (e.g., pocket depths, bleeding indices) and imaging for bone defects, while incorporating phenotype classification through clinical measurements or ultrasound. Systemic modifiers demand collaboration with physicians for optimization—e.g., achieving HbA1c <7% in diabetics or implementing smoking cessation programs. This biological profiling sets the risk foundation, informing

whether to proceed, modify, or defer orthodontics. For instance, in high biological risk (e.g., active inflammation or severe defects), implications include mandatory regenerative procedures like bone grafting to bolster support, potentially shifting strata downward and enabling safer tooth movement.

Biomechanically, implications center on appliance selection and force calibration tailored to risk levels. Low-risk patients may tolerate conventional fixed appliances with standard forces, but moderate-to-high strata necessitate innovations like self-ligating brackets for lighter forces or TADs for absolute anchorage, preventing undesirable extrusions in vertical bone loss scenarios. The framework implies routine use of digital planning tools, such as intraoral scanners and software for simulating biomechanics, to predict stress distributions and avoid overloading compromised sites. This customization reduces iatrogenic risks like root resorption or dehiscences, particularly in thin phenotypes where labial movements are contraindicated without soft tissue augmentation.

Behaviorally, the framework implies embedding adherence assessments into consultations, using tools like questionnaires to gauge hygiene habits and motivation. Implications extend to personalized education: high-risk patients receive intensified regimens, including powered brushes, antimicrobial rinses, and frequent recalls (every 3 months during orthodontics). Digital apps for tracking compliance could be integrated, with implications for adjusting treatment if adherence wanes—e.g., pausing orthodontics to re-stabilize periodontium. This domain's modifiability offers opportunities for intervention, implying that behavioral coaching can salvage borderline cases, enhancing long-term stability through sustained plaque control.

Overall, clinical implications emphasize monitoring and adaptability: regular interdisciplinary reviews track risk evolution, allowing mid-course corrections like force reductions if inflammation recurs. For long-term stability, implications include post-orthodontic retention protocols customized to initial strata—e.g., fixed retainers in high-risk cases to counter relapse from residual defects. The framework also has educational implications, suggesting its incorporation into residency curricula to train clinicians in holistic risk assessment, potentially standardizing care across practices.

By addressing unmet needs, these implications could decrease adverse events, improve patient satisfaction, and reduce healthcare costs associated with complications. However, clinicians must recognize the framework's conceptual nature, applying it judiciously while awaiting empirical refinement through cohort studies or randomized trials.

Conclusion

In summary, this conceptual manuscript introduces an original risk-stratification framework for orthodontic treatment in patients with periodontal disease, synthesizing biological, biomechanical, and behavioral domains to facilitate biologically informed decision-making. By stratifying risks into low, moderate, and high categories, the model addresses critical unmet needs in interdisciplinary planning, emphasizing pre-treatment optimization, customized mechanics, and adherence monitoring to safeguard periodontal stability and enhance long-term outcomes.

The theoretical foundations underscore the interplay of periodontal pathophysiology, bone remodeling, and force-response dynamics, while propositions outline testable assertions for future validation. Clinical implications highlight practical applications, from phased workflows to innovative interventions, positioning the framework as a tool for reducing iatrogenic harm and fostering collaboration.

Ultimately, this integrative approach advances the field by bridging periodontology and orthodontics, paving the way for more predictable, patient-centric care. Empirical studies are warranted to operationalize and refine the model, potentially transforming clinical practice in this challenging population.

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