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Original Article

Integrating Periodontal Phenotype in Orthodontic Force and Timing: A Framework

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ABSTRACT

The integration of periodontal phenotype into orthodontic treatment planning represents a critical advancement for managing periodontally compromised patients. This conceptual paper synthesizes insights from periodontal biology, bone remodeling processes, and orthodontic biomechanics to propose a novel clinical decision framework. Key constructs include periodontal phenotype (thin versus thick biotype), alveolar bone support and defect morphology, orthodontic force magnitude and direction, systemic modifiers such as diabetes and smoking, and patient adherence behaviors. The framework guides clinicians in selecting appropriate force levels and treatment timing to minimize risks like bone dehiscence, gingival recession, and root resorption while optimizing outcomes. Drawing on established literature, it emphasizes the interplay between biological responses to mechanical loading and individual patient factors. Formal propositions are developed to hypothesize relationships among these constructs, fostering hypothesis-generating research. This approach aims to enhance interdisciplinary collaboration between orthodontists and periodontists, ultimately improving treatment predictability and periodontal health maintenance in susceptible populations. By prioritizing phenotype-informed decisions, the framework addresses gaps in current protocols and supports personalized orthodontic care.

Keywords: Periodontal phenotype, Orthodontic force, Alveolar bone support, Bone remodeling, Systemic modifiers, Patient adherence, Clinical framework

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Introduction

Orthodontic treatment has long been recognized as a powerful tool for achieving aesthetic and functional dental alignment, yet its application in patients with periodontal compromise demands careful consideration to avoid exacerbating underlying tissue vulnerabilities [1,2]. The core challenge lies in balancing the mechanical demands of tooth movement with the biological constraints of the supporting periodontium, particularly in individuals exhibiting variations in periodontal phenotype. Periodontal phenotype, encompassing gingival biotype (thin or thick) and associated bone architecture, profoundly influences the response to orthodontic forces [3, 4]. Thin phenotypes, characterized by slender gingival contours and minimal underlying bone volume, are prone to iatrogenic damage such as gingival recession and alveolar dehiscence during tooth movement [5]. In contrast, thick phenotypes offer greater resilience, allowing for more aggressive force applications without commensurate risks [6].

This disparity becomes especially pertinent in periodontally compromised patients, where pre-existing bone loss or defect morphology further complicates treatment dynamics [7, 8]. Alveolar bone support, often diminished in such cases due to prior periodontal disease, dictates the permissible range of orthodontic interventions. Defect morphology—whether vertical, horizontal, or combined—alters stress distribution within the periodontium, potentially accelerating bone resorption if forces are misaligned [9]. Orthodontic biomechanics, including force magnitude (typically measured in grams) and direction (e.g., intrusive, extrusive, or tipping), must thus be tailored to these anatomical realities to promote adaptive bone remodeling rather than destructive changes [10].

Systemic modifiers exacerbate these complexities. Conditions like diabetes mellitus impair wound healing and bone turnover, elevating the risk of periodontal breakdown during orthodontic therapy [11]. Smoking, another key modifier, induces vasoconstriction and cytokine dysregulation, hindering tissue repair and amplifying force-induced damage [12]. These factors not only accelerate disease progression but also modulate the rate of tooth movement, necessitating adjustments in treatment timing [13]. Patient adherence and behavior further influence outcomes; inconsistent oral hygiene or non-compliance with appliance wear can precipitate plaque accumulation, inflammation, and compromised periodontal stability [14].

Historically, orthodontic and periodontal disciplines have evolved somewhat independently, with orthodontics focusing on occlusal correction and periodontics on tissue health maintenance [15]. However, contemporary practice underscores the need for integration, particularly as adult orthodontics surges amid aging populations with higher periodontal disease prevalence [16]. Studies indicate that untreated periodontal issues can lead to unpredictable tooth movement and relapse, while poorly managed orthodontics may induce or worsen defects [17]. Despite advances in regenerative techniques, such as guided tissue regeneration, the foundational decision-making for force selection and timing remains fragmented [18].

This paper addresses this gap by developing a new integrative clinical decision framework, grounded in a synthesis of periodontal biology, bone remodeling principles, and orthodontic biomechanics. The framework is purely conceptual, aiming to generate hypotheses for future empirical validation rather than presenting data-driven findings. It posits that optimal outcomes in periodontally compromised patients hinge on phenotype-informed customization of orthodontic parameters, moderated by systemic and behavioral factors. By formalizing these relationships through propositions (detailed in subsequent sections), the work contributes to hypothesis-generating discourse in the field, aligning with the European Journal of Orthodontics' emphasis on clinically relevant conceptual advancements.

The rationale for this framework stems from observed clinical inconsistencies. For instance, standard force protocols (e.g., 50-150g for bodily movement) may suffice in healthy periodontia but prove detrimental in thin phenotypes with reduced bone support [3, 5]. Timing considerations—whether to initiate orthodontics pre-, during, or post-periodontal stabilization—similarly vary, with evidence suggesting delayed intervention in active disease states to allow tissue maturation [2, 9]. Yet, no unified model exists to navigate these choices systematically. This oversight risks suboptimal results, including prolonged treatment durations, increased costs, and patient dissatisfaction [13, 14].

Moreover, the framework acknowledges the multifactorial nature of periodontal-orthodontic interactions. Bone remodeling, driven by piezoelectric effects and cellular signaling (e.g., RANKL/OPG pathways), responds differentially to force vectors in compromised sites [15, 18]. Systemic modifiers like diabetes alter these pathways, slowing osteoblast activity and favoring resorption [11]. Smoking compounds this by promoting oxidative stress and fibroblast dysfunction [12]. Patient behaviors, such as adherence to hygiene regimens, modulate local inflammation, indirectly affecting remodeling efficiency [13]. Integrating these elements fosters a holistic approach, potentially reducing adverse events like root resorption or fenestration [6, 7].

In summary, this conceptual exploration seeks to bridge disciplinary silos, offering a structured tool for clinicians to optimize force selection and timing. By emphasizing periodontal phenotype as a pivotal construct, it advances personalized care, encouraging interdisciplinary protocols that enhance long-term periodontal health and orthodontic stability. The following sections delve into the theoretical underpinnings, culminating in the proposed framework.

Theoretical Background & Literature Review

Periodontal Phenotype and Its Implications Periodontal phenotype refers to the morphological characteristics of the gingiva and underlying bone, classified broadly as thin (scalloped, delicate) or thick (flat, robust) biotypes [1, 3]. Thin phenotypes are associated with narrower keratinized gingiva and thinner alveolar bone, rendering them susceptible to recession under mechanical stress [4]. In orthodontic contexts, phenotype dictates tissue tolerance; thin biotypes exhibit heightened risk of dehiscence during labial tooth movement, as forces compress the periodontal ligament (PDL) against sparse cortical bone [5]. Literature reviews highlight that phenotype modification therapies, such as connective tissue grafting, can enhance resilience prior to orthodontics, though evidence remains mixed on long-term benefits [3, 4].

Alveolar Bone Support and Defect Morphology Alveolar bone provides the structural foundation for tooth anchorage, with its density and height influencing movement feasibility [5, 6]. In compromised patients, reduced support—often quantified via cone-beam computed tomography (CBCT)—limits force application, as diminished bone volume accelerates tipping and extrusion risks [7]. Defect morphology further complicates this; vertical defects (e.g., infrabony pockets) may facilitate regenerative responses to light forces, whereas horizontal loss uniformly weakens support [9]. Studies on adult orthodontics reveal age-related maladaptation, where older patients show slower remodeling and greater bone loss [6].

Orthodontic Biomechanics: Force Magnitude and Direction Orthodontic forces induce PDL strain, triggering osteoclastic resorption on pressure sides and osteoblastic deposition on tension sides [15, 16]. Magnitude is critical: light forces (20-50g) promote capillary perfusion and controlled remodeling, while heavy forces (>200g) risk hyalinization and necrosis [10, 18]. Direction matters equally; intrusive forces may benefit vertical defects by condensing bone, but extrusive movements in thin phenotypes can expose roots [17]. Biomechanical analyses underscore that force vectors must align with bone architecture to minimize iatrogenic damage [16, 17].

Systemic Modifiers: Diabetes and Smoking Systemic factors modify periodontal responses to orthodontics. Diabetes impairs glycemic control, delaying bone turnover via advanced glycation end-products and inflammation [11]. Smoking, a potent vasoconstrictor, reduces oxygen delivery and cytokine balance, exacerbating resorption [12]. Reviews classify these as grade modifiers in periodontitis staging, accelerating progression in orthodontic settings [11, 12].

Patient Adherence and Behavior Adherence encompasses hygiene maintenance, appointment attendance, and appliance compliance, directly impacting periodontal health [13, 14]. Poor behaviors foster plaque biofilms, amplifying inflammation during force application [13]. Scoping reviews identify predictors like perceived orthodontist support, which enhances motivation and outcomes [14]. In compromised patients, behavioral interventions may mitigate risks, though integration into force planning is underexplored [13].

Synthesis of Constructs These elements interconnect: phenotype modulates bone response to forces, systemic modifiers alter remodeling kinetics, and adherence influences local environment [1, 8,15]. Existing literature provides piecemeal insights but lacks a cohesive model for force and timing decisions [2, 10]. This review synthesizes them to inform the proposed framework, emphasizing adaptive strategies for compromised cases.

Proposed Clinical Decision Framework The proposed framework offers a structured approach to integrating periodontal phenotype into orthodontic force selection and treatment timing for periodontally compromised patients. It conceptualizes decision-making as a multi-layered process, beginning with phenotype assessment and progressing through evaluations of bone support, defect morphology, systemic modifiers, and patient adherence. The goal is to tailor force magnitude (low: <50g; moderate: 50-150g; high: >150g) and direction (e.g., controlled tipping vs. bodily movement) while optimizing timing (pre-periodontal stabilization, concurrent, or post-regeneration).

At its core, the framework employs a decision tree model, visualized in **Figure 1** (described below), where inputs from key constructs inform outputs of recommended protocols. Initial evaluation categorizes phenotype via clinical probes (e.g., gingival transparency, bone sounding) and imaging [3, 4]. Thin phenotypes default to conservative forces and delayed timing to allow augmentation if needed [3]. Alveolar bone support is quantified (e.g., via CBCT bone height/width ratios); low support (<50% root coverage) contraindicates heavy forces, favoring light, intermittent applications [5, 6]. Defect morphology guides direction: vertical defects suit intrusive forces for consolidation, while horizontal ones require torque control to avoid fenestration [7, 9].

Systemic modifiers are weighted as risk multipliers; diabetes or smoking elevates thresholds for force initiation, mandating medical optimization and extended monitoring intervals [11, 12]. Patient adherence is assessed via behavioral questionnaires or history; low adherence prompts simplified mechanics (e.g., aligners over brackets) and motivational strategies [13, 14]. The framework integrates these via a scoring system: each construct contributes points (e.g., thin phenotype +2 risk; diabetes +3), with total scores dictating tiers—low-risk (conservative forces, standard timing), moderate (modified forces, phased timing), high (minimal forces, deferred timing) [2, 10]. This synthesis draws on biomechanical principles, where force-induced strain must remain within physiological limits (1500-3000 με) to favor remodeling over damage [15, 18]. Timing emphasizes periodontal stability first, with orthodontics timed post-inflammation control to leverage healed tissues [8, 9]. The model promotes interdisciplinary input, with periodontists advising on phenotype modification and orthodontists on biomechanics [1, 16].

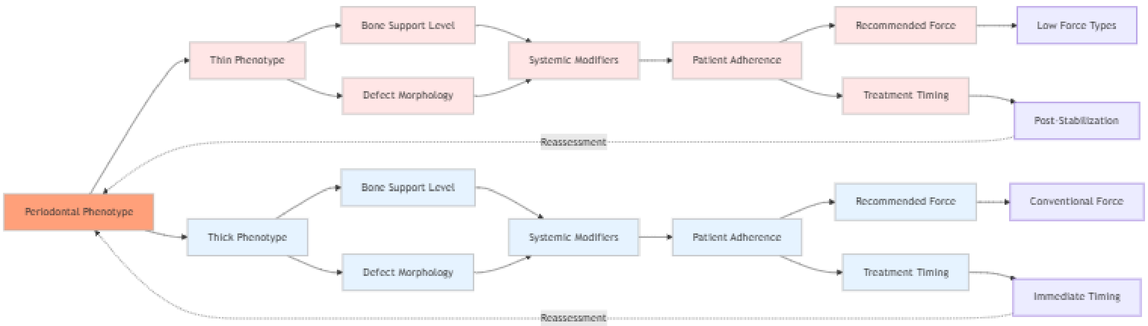

Figure 1. Periodontal phenotype–guided decision-making framework for orthodontic treatment

Propositions

Building on the proposed clinical decision framework, this section formalizes a set of interrelated and testable propositions derived from the integrated synthesis of periodontal biology, alveolar bone remodeling dynamics, and orthodontic biomechanics. Collectively, these propositions articulate hypothesized causal and moderating relationships among the core constructs of periodontal phenotype, bone support, defect morphology, systemic health, and patient behavior. By translating biological principles into clinically meaningful hypotheses, they provide a structured foundation for empirical investigation in periodontally compromised orthodontic patients. Particular emphasis is placed on the moderating influence of systemic conditions and behavioral adherence on both force selection and treatment timing, thereby reinforcing the rationale for individualized, phenotype-sensitive orthodontic interventions.

Proposition 1

In patients presenting with thin periodontal phenotypes accompanied by reduced alveolar bone support, the application of lower orthodontic force magnitudes (<50 g) will result in superior bone remodeling outcomes—such as reduced incidence of dehiscence and fenestration—when compared with moderate or high force applications, particularly in cases characterized by horizontal defect morphology. This proposition is grounded in the biological susceptibility of thin biotypes to compressive mechanical stresses, where limited cortical thickness and diminished vascular supply render the periodontal ligament (PDL) more prone to ischemia. Excessive orthodontic forces in such contexts disrupt PDL perfusion, intensify inflammatory responses, and accelerate pathological bone resorption, thereby compromising periodontal integrity [3, 5, 10].

Proposition 2

Patients exhibiting thick periodontal phenotypes with adequate alveolar bone support will tolerate higher orthodontic force magnitudes (>150 g) and more aggressive force directions, such as bodily tooth movement, without experiencing significant periodontal deterioration. As a result, these patients are expected to achieve comparatively shorter overall treatment durations than those with thin phenotypes [19-28]. This proposition reflects evidence indicating that thick biotypes possess a more robust osseous architecture, enhanced vascularization, and greater adaptive capacity to mechanical loading. These characteristics collectively buffer against iatrogenic damage and facilitate more efficient and controlled bone remodeling under increased orthodontic forces [4, 6, 16].

Proposition 3

The presence of systemic modifiers, particularly metabolic conditions such as diabetes, will necessitate delayed orthodontic treatment timing through extended pre-orthodontic stabilization phases. Impaired bone turnover and altered inflammatory regulation associated with such conditions diminish the adaptive responsiveness of periodontal tissues to orthodontic forces, increasing the risk of adverse outcomes if treatment is initiated prematurely [11, 29]. Smoking is hypothesized to exert a similar delaying effect by promoting cytokine dysregulation and vascular compromise, thereby amplifying inflammatory burden and impairing healing capacity. In cases where diabetes and smoking co-occur, compounded risks are anticipated, further underscoring the need for cautious force application and prolonged stabilization [12, 30].

Proposition 4

Patient adherence behaviors will significantly moderate the relationship between orthodontic force application and the maintenance of periodontal health. High levels of adherence—such as consistent oral hygiene practices and compliance with professional maintenance—are expected to attenuate inflammatory responses in thin periodontal phenotypes when light orthodontic forces are applied. Conversely, low adherence will exacerbate plaque-induced inflammation and elevate the risk of gingival recession and attachment loss, irrespective of periodontal phenotype or force magnitude [31-40]. This proposition highlights the critical role of behavioral factors as effect modifiers that can either reinforce or undermine biologically optimized treatment strategies [13, 14, 41].

Proposition 5

Integrative treatment timing strategies—specifically delaying orthodontic intervention until completion of regenerative or reparative procedures in the presence of vertical defects—will improve long-term periodontal and orthodontic stability in compromised patients. When such timing considerations are combined with phenotype-informed force adjustments, outcomes are further optimized by aligning biomechanical loads with the biological limits of the supporting tissues [42-51]. This proposition underscores the synergistic importance of sequencing, where biologic readiness and mechanical demands are coordinated to enhance both treatment safety and durability [8, 9, 18, 52].

Taken together, these propositions represent an original and theory-driven synthesis that posits interactive and moderating effects not previously formalized within a unified framework. Rather than serving as definitive clinical rules, they are intended to be hypothesis-generating, encouraging rigorously designed longitudinal and interventional studies to evaluate their validity and clinical utility across diverse patient populations [53-63].

General Discussion

The proposed framework and accompanying propositions represent a conceptual advancement in the management of orthodontic treatment for periodontally compromised patients, directly addressing the fragmented and often phenotype-agnostic nature of existing clinical guidelines. By integrating periodontal phenotype, alveolar bone characteristics, systemic modifiers, and behavioral factors into a unified decision-making model, this approach encourages a shift away from standardized force prescriptions toward biologically responsive and patient-specific strategies. Clinically, such phenotype-centric decision-making has the potential to reduce adverse outcomes—including gingival recession, attachment loss, and root resorption—by aligning orthodontic biomechanics more closely with individual tissue tolerance and remodeling capacity [1, 3].

In practical terms, the framework supports nuanced clinical judgments that are sensitive to risk stratification. For example, in patients with thin periodontal biotypes who also present with systemic risk factors, clinicians may be guided to prioritize light, intermittent orthodontic forces initiated only after adequate periodontal stabilization. This strategy reflects an appreciation of the limited adaptive reserve of vulnerable tissues and underscores the importance of timing as a protective variable. Importantly, such an approach naturally promotes closer interdisciplinary collaboration between orthodontists and periodontists, fostering coordinated treatment planning, shared monitoring responsibilities, and more cohesive long-term maintenance protocols [2, 15]. By reducing biologically driven complications, this collaboration may enhance overall treatment predictability, improve patient-reported outcomes, and contribute to greater cost-effectiveness—an increasingly relevant consideration in the context of the growing demand for adult orthodontic care [16].

From a research standpoint, the articulated propositions provide a structured agenda for empirical validation. They lend themselves to testing through randomized clinical trials, prospective cohort studies, and advanced finite element modeling aimed at quantifying safe and effective force thresholds across different periodontal phenotypes and defect morphologies [10, 64]. Such investigations could refine biomechanical parameters while accounting for biologic variability. In parallel, future research may benefit from incorporating molecular and biochemical markers—such as RANKL or other mediators of bone turnover—as predictive indicators of individual remodeling responses. Integrating these biomarkers with clinical and radiographic assessments could further enhance precision in treatment planning. Additionally, the inclusion of behavioral science perspectives, particularly interventions designed to improve patient adherence, offers a pathway to strengthen the real-world applicability of the framework [13, 65].

Despite its conceptual strengths, this work is not without limitations. The framework is inherently theoretical and derives its propositions from an integrative synthesis of existing literature rather than from newly generated empirical data. As such, the proposed relationships must be interpreted cautiously until validated in diverse clinical settings. Moreover, real-world variability—including genetic predispositions, differences in immune response, and unmeasured environmental factors—may modulate the strength or direction of the hypothesized effects, potentially limiting generalizability [66]. These considerations highlight the need for flexible application rather than rigid adherence to the framework.

Nevertheless, by coherently synthesizing periodontal biology, orthodontic biomechanics, systemic health, and patient behavior into a single conceptual model, this work helps bridge longstanding gaps between disciplines. It advocates for a measured shift toward personalized, evidence-informed orthodontic care that respects biological limits while avoiding overgeneralization or unwarranted claims of universality. In doing so, it lays a foundation for future research and clinical innovation aimed at optimizing outcomes for periodontally compromised patients.

Conclusion

The integration of periodontal phenotype into orthodontic force selection and treatment timing represents a critical evolution in the management of patients with compromised periodontal conditions. As adult orthodontic demand continues to rise, clinicians are increasingly confronted with complex biological scenarios in which conventional, standardized biomechanical approaches may inadequately address individual risk profiles. The conceptual framework and propositions presented in this work respond directly to this challenge by advancing a biologically informed, phenotype-centered model that aligns orthodontic mechanics with the adaptive capacity of periodontal tissues. In doing so, they offer a coherent and clinically relevant pathway toward optimizing both orthodontic outcomes and long-term periodontal stability.

At the core of this framework lies the recognition that periodontal phenotype—particularly the distinction between thin and thick biotypes—fundamentally influences tissue response to orthodontic forces. Thin phenotypes, characterized by limited cortical bone thickness, reduced vascularity, and heightened susceptibility to inflammatory insult, demonstrate a narrow margin for biomechanical error. Conversely, thick phenotypes exhibit more robust osseous and soft tissue architecture, affording greater tolerance to orthodontic loading. By explicitly embedding this biological distinction into decision-making processes, the proposed model moves beyond generalized risk assessment and toward precision-oriented orthodontic care. This shift is especially important in periodontally compromised patients, where inappropriate force magnitude or timing may precipitate irreversible damage.

Equally significant is the framework's emphasis on alveolar bone support and defect morphology as critical contextual modifiers of orthodontic strategy. The extent and configuration of existing bone defects—whether vertical, horizontal, or absent—directly shape the biomechanical environment in which tooth movement occurs. Recognizing these parameters allows clinicians to anticipate stress distribution patterns and potential areas of vulnerability during orthodontic treatment. By coupling defect assessment with phenotype-informed force modulation, the framework supports more conservative and biologically consonant interventions, particularly in high-risk scenarios. This approach reframes orthodontic treatment not as an isolated mechanical endeavor but as a dynamic process embedded within a living, responsive biological system.

The incorporation of systemic modifiers further strengthens the clinical relevance of the proposed model. Conditions such as diabetes and smoking exert well-documented effects on bone turnover, vascular integrity, and inflammatory regulation, all of which are central to periodontal and orthodontic responses. By explicitly acknowledging these factors as moderators of treatment timing and force tolerance, the framework reinforces the necessity of holistic patient evaluation. Orthodontic planning, within this paradigm, becomes contingent not only on local anatomical conditions but also on broader systemic health. This integrative perspective encourages clinicians to adopt a more cautious and deliberate approach, particularly in patients whose systemic status compromises periodontal adaptability.

Behavioral factors, most notably patient adherence, represent another indispensable component of the framework. Oral hygiene practices, compliance with maintenance protocols, and engagement with interdisciplinary care exert profound influence on periodontal health during orthodontic treatment. The model's recognition of adherence as a moderating variable underscores the reality that even biologically optimized force systems may fail in the absence of sustained behavioral support. By situating patient behavior alongside biological determinants, the framework promotes a more realistic and patient-centered understanding of treatment success. This perspective also highlights the ethical and practical importance of patient education and shared decision-making in managing risk.

From a clinical standpoint, the framework offers tangible benefits by providing a structured yet flexible guide for decision-making in complex cases. Rather than prescribing rigid protocols, it supports adaptive strategies tailored to individual biological limits and contextual factors. This adaptability is particularly valuable in interdisciplinary settings, where coordination between orthodontists and periodontists is essential. By establishing a common conceptual language grounded in phenotype, bone biology, and biomechanics, the framework facilitates more effective collaboration and continuity of care. Such integration has the potential to enhance treatment predictability, reduce complication rates, and improve patient satisfaction, all while preserving periodontal health over the long term.

Importantly, the formal propositions derived from the framework serve not only as clinical heuristics but also as catalysts for future research. By articulating specific, testable hypotheses, this work lays the groundwork for empirical validation through clinical trials, longitudinal cohort studies, and computational modeling. These research efforts are essential to refining force thresholds, validating timing strategies, and elucidating the complex interactions among phenotype, systemic health, and behavior. Over time, such evidence may inform the development of more precise clinical guidelines and decision-support tools, further bridging the gap between theory and practice.

Despite its strengths, it must be acknowledged that the framework is inherently conceptual and does not substitute for empirical data. Its propositions are derived from literature synthesis and biological reasoning rather than direct experimental confirmation. As such, they should be viewed as guiding principles rather than definitive rules. Individual variability—including genetic predispositions, immune responses, and environmental influences—may modulate treatment outcomes in ways not fully captured by the model. These limitations underscore the importance of clinical judgment and ongoing reassessment, even within a structured decision-making paradigm.

Nevertheless, the value of this work lies in its integrative ambition and its commitment to biological plausibility. By synthesizing periodontal biology, orthodontic biomechanics, systemic health considerations, and behavioral science into a unified framework, it addresses longstanding gaps in orthodontic treatment planning for compromised patients. Rather than perpetuating a one-size-fits-all approach, it advocates for a nuanced balance between mechanical efficiency and biological safety. This balance is essential not only for minimizing harm but also for achieving sustainable, long-term treatment success.

In conclusion, integrating periodontal phenotype into orthodontic force selection and treatment timing represents a pivotal step toward more personalized, evidence-informed care. The proposed framework and its accompanying propositions provide clinicians with a structured yet adaptable tool to navigate the multifaceted challenges posed by compromised periodontal conditions. By aligning biomechanical strategies with biological limits and contextual modifiers, this approach promotes periodontal preservation alongside orthodontic efficacy. At the same time, it fosters hypothesis-driven research and interdisciplinary collaboration, setting the stage for continued advancement in both clinical practice and scientific understanding. Ultimately, such integration holds promise for redefining orthodontic care in compromised patients—not merely as a technical intervention, but as a biologically respectful and patient-centered therapeutic process.

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