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Original Article

Alveolar Bone Loss and Orthodontic Treatment Planning: Bone Morphology, Defects, and Force Application

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ABSTRACT

Alveolar bone loss represents a critical challenge in orthodontic treatment, as it affects the structural integrity of the periodontium and influences the feasibility and outcomes of tooth movement. This narrative review synthesizes evidence from peer-reviewed studies published between 2020 and 2025 to examine the interplay between alveolar bone morphology, defects, force application, and treatment planning in orthodontics. The alveolar bone's morphology, including thick and thin morphotypes, plays a pivotal role in determining susceptibility to defects such as dehiscences and fenestrations during orthodontic forces, which induce remodeling through mechanobiological pathways involving cytokines, matrix metalloproteinases, and the RANK/RANKL/OPG axis. Defects can slow tooth movement rates and increase risks like root resorption, particularly in compromised periodontal conditions. Force application must be optimized—using light, controlled magnitudes—to minimize bone loss, with finite element analyses highlighting the importance of moment-to-force ratios in edentulous areas. Treatment planning requires multidisciplinary approaches, sequencing periodontal regeneration before or during orthodontics, and incorporating AI for predictive modeling of bone changes. Key findings indicate higher incidence of defects with clear aligners in certain malocclusions, the benefits of occlusal intervention prior to orthodontics for balancing osteoblast-osteoclast activity, and innovations in regeneration using growth factors and stem cells. Objectives include providing clinicians with evidence-based strategies to mitigate risks, enhance bone preservation, and improve long-term stability. This review underscores the need for personalized, technology-aided planning to address alveolar bone loss effectively.

Keywords: Alveolar bone loss, Orthodontic treatment, Bone defects, Force application, Bone remodeling, Treatment planning

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Introduction

The alveolar bone, a specialized component of the maxilla and mandible, serves as the primary support for teeth and is integral to orthodontic tooth movement (OTM). Composed of cortical and trabecular layers, it undergoes continuous remodeling in response to mechanical stimuli, a process governed by Wolff's law, where bone adapts to applied loads [1,2]. In orthodontics, controlled forces are applied to teeth via appliances, transmitting stress through the periodontal ligament (PDL) to the alveolar bone, initiating resorption on the compression side and apposition on the tension side [3,4]. This dynamic equilibrium enables tooth repositioning but can lead to iatrogenic complications, particularly alveolar bone loss, if forces exceed physiological thresholds [5].

Alveolar bone loss, often manifesting as dehiscences (loss of cortical bone over root surfaces) or fenestrations (window-like defects), is prevalent in orthodontic patients, with incidences increasing post-treatment [6,7]. Etiologically, it stems from pre-existing periodontal disease, genetic predispositions, thin biotypes, or excessive orthodontic forces that disrupt the balance between osteoblasts and osteoclasts [8]. In patients with periodontitis, bone loss exacerbates secondary malocclusions, such as tooth migration and traumatic occlusion, necessitating integrated perio-ortho management [9]. Recent studies highlight that bone morphology—classified as thick, medium, or thin based on cortical thickness and density dictates vulnerability; thin morphotypes are prone to recession and defects during labial movements [10].

The application of orthodontic forces must consider biomechanical principles to prevent bone loss. Forces induce mechanotransduction via channels like Piezo1, activating pathways such as NF-κB and Wnt/β-catenin, which regulate remodeling [3]. Excessive or misdirected forces can cause hyalinization, root resorption, and bone atrophy, particularly in edentulous regions where resistance centers shift [11, 12]. Comorbidities like diabetes impair this process by suppressing VEGF/SP1 axes, prolonging movement and increasing risks [13].

Treatment planning in the context of alveolar bone loss requires comprehensive assessment using tools like cone-beam computed tomography (CBCT) for 3D evaluation of bone height, thickness, and volume [14-16]. Emerging technologies, including AI-driven models, predict bone responses and optimize force vectors, enhancing precision [17,18]. Sequencing with periodontal regeneration—using grafts, membranes, or growth factors—can mitigate defects, with orthodontics aiding healing by stimulating apposition [9, 19,20].

Despite advances, challenges persist, including asymmetric movement in defective bone [21, 22] and greater resorption with fixed appliances versus aligners [23-26]. This review aims to: (1) elucidate alveolar bone morphology and its orthodontic relevance; (2) describe bone defects and their etiology; (3) explore force application mechanisms and remodeling; and (4) discuss planning strategies to minimize loss and optimize outcomes. By integrating recent evidence, it provides clinicians with tools for evidence-based practice in this evolving field.

Alveolar Bone Morphology and Its Relevance to Orthodontics

Alveolar bone morphology encompasses the structural characteristics of the cortical and trabecular components, which vary by individual biotype and influence orthodontic outcomes. Classified into thick (robust cortical plates), medium, and thin (delicate, prone to fenestration) morphotypes, these variations affect bone density, measured in Hounsfield units via CBCT, and susceptibility to remodeling [10, 27, 28]. Thin morphotypes, common in inflammatory periodontal disease, exhibit lower optical density and increased risk of recession during tooth movement, necessitating careful planning [10].

In orthodontic context, morphology dictates the center of resistance, shifting apically with bone loss and altering moment-to-force ratios [11, 29]. For instance, in edentulous areas, the resistance center is 0.43 root lengths from the crest, requiring optimized forces to avoid tipping and stress concentrations [11, 30]. Vertical skeletal patterns further modify this; hyperdivergent patterns show greater labial height loss in incisors during decompensation [14,31]. AI integration enhances assessment by fusing CBCT and scans for precise morphology tracking, achieving high accuracy in jawbone segmentation [17, 32].

Morphological variations also impact hygiene and stability. Thin bone correlates with higher complication rates (up to 55% with lingual braces), while overlaying CT on digital models improves planning efficiency by 40% [10, 33]. In diabetes, impaired SP1/VEGF axes exacerbate remodeling suppression, highlighting morphology's role in comorbid planning [13, 34].

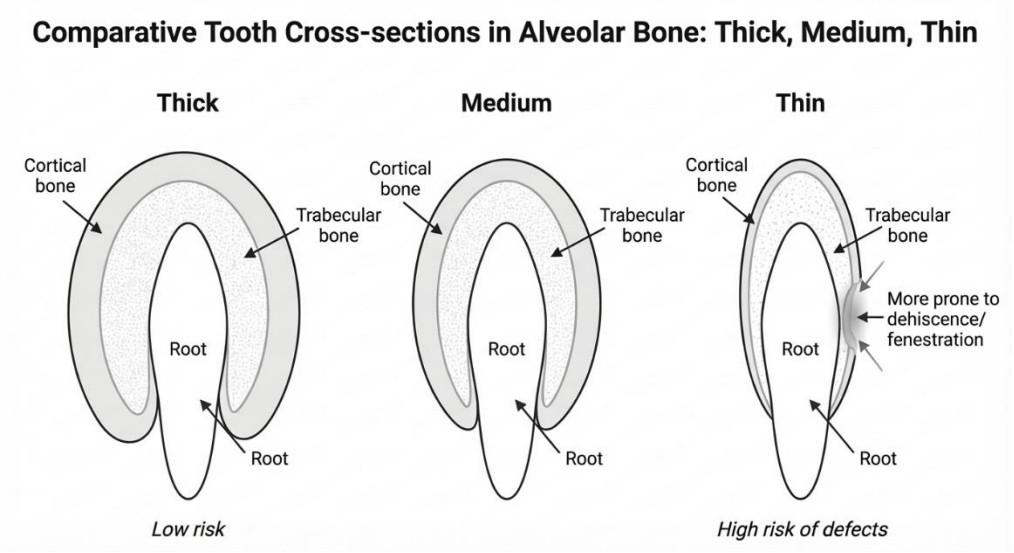

Figure 1. Alveolar bone morphotypes and their orthodontic relevance

Types and Etiology of Alveolar Bone Defects

Alveolar bone defects manifest primarily as dehiscences and fenestrations, each with distinct morphological characteristics and clinical implications. Dehiscences are characterized by loss of cortical bone overlying the root surface, resulting in partial exposure of the root without affecting the marginal bone crest. Fenestrations, in contrast, represent isolated perforations of cortical bone, typically leaving the marginal crest intact [6, 35-37]. Both types of defects may pre-exist prior to orthodontic intervention but can be exacerbated by tooth movement, particularly when forces are applied to thin alveolar cortices or in the presence of compromised periodontal support. Epidemiological data demonstrate that orthodontic therapy can increase the prevalence of these defects. For instance, in patients treated with clear aligners combined with intermaxillary elastics, the incidence of dehiscences increased from 28.1% to 47.4%, whereas fenestrations rose from 9.3% to 12.8%, highlighting the cumulative effect of mechanical forces and anatomical predisposition [6, 38].

Etiology is multifactorial, encompassing pre-treatment anatomical, demographic, and periodontal factors. Crowding significantly contributes, with odds ratios indicating a 1.318-fold increased risk of defect formation [2, 6, 39]. Tooth position and morphology also influence vulnerability, with canines and incisors particularly susceptible due to their prominent labial root contours and thinner overlying cortical bone. Age is another determinant, as alveolar bone density and remodeling capacity decline over time, rendering older patients more prone to dehiscences and fenestrations [6].

In the context of periodontitis, alveolar bone loss follows a progressive pattern, often culminating in intrabony defects of ≥3 mm and furcation involvement in molars, which compromise tooth stability and contribute to secondary malocclusions [8]. Orthodontic forces applied to teeth with thin gingival biotypes (<2 mm keratinized tissue) further exacerbate defect formation, necessitating pre-orthodontic soft tissue augmentation or bone grafting to mitigate risks [8, 40]. Biomechanically, these defects impede efficient tooth movement; fenestrations can reduce movement rates to approximately 0.87 mm/month, and quasi-defects slow it to 0.62 mm/month, compared with 1.17 mm/month in teeth with intact alveolar bone, emphasizing the functional impact of pre-existing or induced bone deficiencies [21, 41].

The pattern of bone loss is also influenced by malocclusion type and appliance selection. In Class III malocclusions, labial resorption predominates with aligner therapy, whereas fixed appliances may produce palatal cortical defects due to force vectors and archwire mechanics [23, 42, 43]. At the molecular level, inflammation plays a pivotal role in defect progression. Elevated cytokines, including interleukin-1β (IL-1β) and tumor necrosis factor-α (TNF-α), stimulate matrix metalloproteinases (MMPs) and the RANKL signaling pathway, promoting extracellular matrix degradation and osteoclastic resorption [1, 43]. Genetic predispositions, such as IL-1β polymorphisms, further amplify susceptibility, illustrating the interplay between environmental, mechanical, and hereditary factors in alveolar bone defect etiology [1, 44].

Mechanisms of Orthodontic Force-Induced Bone Remodeling

Orthodontic tooth movement relies on the principle that controlled mechanical forces elicit a cascade of cellular and molecular events within the periodontal ligament (PDL) and alveolar bone, leading to site-specific bone remodeling. On the compression side, applied forces deform the PDL, triggering mechanosensitive ion channels such as Piezo1, which facilitate calcium influx into osteocytes and PDL cells. This calcium signaling initiates downstream activation of the NF-κB pathway, resulting in upregulation of receptor activator of nuclear factor kappa-B ligand (RANKL) and subsequent differentiation of osteoclast precursors into mature, resorptive osteoclasts [3, 45]. Simultaneously, tension areas experience fibroblast and osteocyte mechanotransduction that activates Wnt/β-catenin signaling, stimulating osteoblastic activity and osteoid deposition to reinforce alveolar bone [3].

The RANK/RANKL/osteoprotegerin (OPG) axis serves as a central regulator of this remodeling process. RANKL expression on PDL and osteocyte surfaces binds to RANK on osteoclast progenitors, inducing NFATc1 transcription for osteoclast maturation and bone resorption. This axis is finely modulated by microRNAs, which can either promote or inhibit osteoclastogenesis depending on the molecular context [1]. In parallel, macrophage polarization contributes to spatially distinct remodeling: M1 macrophages dominate compression sites, secreting pro-inflammatory cytokines such as IL-1β and TNF-α to enhance resorption, whereas M2 macrophages on tension sides secrete anti-inflammatory signals that support bone formation [1].

Excessive or improperly directed forces can disrupt this balance, leading to PDL hyalinization, localized hypoxia, and apoptosis mediated by HIF-1α and Bax pathways, thereby impairing remodeling and increasing the risk of root resorption [1]. In cases of hypofunctional occlusion, pre-orthodontic occlusal adjustments can restore mechanical equilibrium, enhancing remodeling through the SIRT1/β-catenin axis. This promotes the expression of osteogenic genes such as RUNX2 while downregulating osteoclastogenic genes like NFATC1, effectively optimizing bone turnover before active tooth movement [5].

Finite element analyses (FEA) provide quantitative insights into force distribution and tissue stress. Simulations indicate that optimized forces of 15–50 g maintain alveolar stress at approximately 0.0255 MPa in edentulous or thin cortical bone regions, minimizing the risk of iatrogenic damage [11]. Interestingly, local forces applied to specific teeth can propagate remodeling signals throughout the maxilla, influencing adjacent bone and PDL adaptation [46]. Systemic conditions, such as diabetes mellitus, attenuate these responses by impairing SP1/VEGF signaling, which reduces osteoclast recruitment and vascular support for remodeling [13]. Prolonged orthodontic treatment also induces changes in tissue architecture, including increased PDL volume and enhanced bone porosity, which may affect subsequent tooth movement and stability [47].

In summary, orthodontic force-induced remodeling is a tightly regulated interplay of mechanical, cellular, and molecular factors. Effective treatment requires calibrated force magnitudes and directions to harness mechanotransduction pathways while minimizing pathological outcomes such as root resorption or alveolar defects. Understanding these mechanisms enables clinicians to personalize orthodontic strategies, particularly in patients with compromised periodontal or systemic conditions.

Impact of Orthodontic Treatment on Alveolar Bone

Orthodontic interventions inevitably alter alveolar bone morphology through remodeling processes that balance resorption and formation. In skeletal Class II cases undergoing decompensation, lingual alveolar bone resorption is particularly pronounced, with hypodivergent craniofacial patterns showing greater susceptibility due to denser cortical bone and altered stress distribution [14]. For Class I malocclusions, fixed appliances induce more extensive alveolar remodeling and resorption than clear aligners, especially in extraction cases where space closure generates concentrated forces on cortical plates [24]. In Class III patients, the pattern of alveolar change varies with appliance type: fixed appliances tend to produce palatal cortical thinning, whereas aligners lead to labial cortical reduction, reflecting differences in force vectors and torque control [23].

Post-periodontitis orthodontic tooth movement (OTM) appears safe when periodontal inflammation is resolved prior to treatment. Studies indicate comparable RANKL expression in previously compromised and healthy tissues, suggesting that controlled orthodontic forces do not exacerbate alveolar bone loss once periodontal stability is achieved [48]. Specific movements, such as incisor retraction, reduce crestal bone thickness, with stress concentration at the alveolar crest leading to localized remodeling and cortical deformation [49]. Importantly, bone resorption often exceeds root resorption in magnitude, indicating that alveolar adaptation, rather

than root damage, primarily governs displacement during treatment [15]. Cone-beam computed tomography (CBCT) studies corroborate these findings, revealing reductions in alveolar height post-treatment, frequently accompanied by measurable incisor root shortening, underscoring the need for precise force control and monitoring [15].

Diagnostic Tools for Assessing Alveolar Bone in Orthodontic Planning

Accurate pre-treatment assessment is critical to minimize iatrogenic bone loss. CBCT enables three-dimensional evaluation of alveolar height, cortical thickness, and the presence of fenestrations or dehiscences, providing a comprehensive view unattainable with traditional two-dimensional imaging [14,15]. The integration of artificial intelligence (AI) enhances predictive capability, allowing for the detection of potential resorption areas with reported accuracies up to 95%, and facilitating individualized force planning [17]. Finite element analysis (FEA) models simulate stress distribution in compromised alveolar bone, guiding clinicians on optimal force magnitudes and vectors for safe tooth movement [11,15]. Additionally, overlay techniques enable dynamic morphotype assessment, comparing pre- and post-treatment scans to evaluate changes in bone architecture and adapt ongoing treatment plans [10].

Strategies for Treatment Planning in Patients with Alveolar Bone Loss

Consensus guidelines emphasize achieving periodontal stability before initiating orthodontic therapy, particularly in patients with thin cortical plates or prior bone loss [8]. Light forces (5–15 g per tooth) are recommended to minimize ischemia and prevent excessive cortical stress, while fixed appliances are preferred in cases requiring precise control, such as severe crowding or complex root movements [8]. Sequencing strategies should be individualized: regenerative procedures are advised prior to orthodontics in narrow or vertical defects, whereas specific movements, including extrusion or molar uprighting, may precede regenerative therapy to optimize alveolar morphology and enhance clinical outcomes [9]. AI-assisted predictive modeling further refines treatment by simulating potential bone responses and adjusting force delivery in real time [18]. Emerging regenerative adjuncts, such as bone morphogenetic proteins (BMPs) and stem cell–enhanced grafts, offer promising avenues to restore alveolar defects pre- or mid-treatment, potentially accelerating safe tooth movement while preserving bone integrity [19]. Retention strategies should be lifelong in high-risk cases, combining fixed lingual wires with removable overlays to maintain alveolar and periodontal stability, particularly in patients with thin phenotypes or prior periodontitis [8]. Overall, a multidisciplinary approach integrating precise diagnostics, force optimization, regenerative support, and individualized retention protocols ensures both functional and aesthetic success in patients with compromised alveolar bone.

Discussion

The interplay between alveolar bone loss and orthodontic treatment planning is multifaceted, encompassing bone morphology, defect etiology, force-induced remodeling, and strategic interventions. As highlighted in the review, alveolar bone morphology significantly influences orthodontic susceptibility to defects. Thin morphotypes, characterized by reduced cortical thickness, are particularly vulnerable to dehiscences and fenestrations during tooth movement, especially in adult patients where age-related maladaptation exacerbates remodeling challenges [1]. Studies utilizing CBCT have consistently demonstrated that pre-treatment assessment of morphotypes—such as in patients with skeletal Class III malocclusion—reveals poorer initial bone conditions, with further resorption occurring during treatment [9, 10]. This underscores the necessity for personalized morphometric evaluations to predict risks, aligning with systematic reviews indicating vertical and horizontal bone loss in anterior regions post-treatment [23]. However, while thin biotypes predispose to complications, evidence suggests that appropriate force control can mitigate these effects, as seen in comparisons between surgery-first and conventional approaches where alveolar changes were similar but emphasized the role of initial bone status [10].

Bone defects, including dehiscences and fenestrations, arise from a combination of pre-existing periodontal conditions, genetic factors, and iatrogenic forces. Recent multicenter studies report increased incidence post-clear aligner therapy, with factors like crowding, intrusion magnitude, and sagittal patterns contributing significantly [3]. For instance, fenestrations and dehiscences were more prevalent on buccal surfaces in anterior teeth, with odds ratios linking them to pre-treatment alveolar thickness and gingival biotype [13, 24]. In orthodontic contexts, defects not only compromise aesthetics and stability but also alter movement kinetics; alveolar defects reduce tooth

displacement rates, with fenestrations slowing movement to approximately 0.87 mm/month compared to intact bone [48]. This is mechanistically tied to disrupted osteoblast-osteoclast balance, where inflammation-driven cytokines like IL-1β and TNF-α amplify RANKL-mediated resorption [5]. Moreover, in periodontally compromised teeth, defects persist or worsen without prior regeneration, as evidenced by case reports showing attachment loss despite laser therapy integration [6]. These findings highlight a critical gap: while CBCT enables precise defect localization, longitudinal data on defect progression during OTM remain limited, often confounded by variable appliance types (fixed vs. aligners) and patient comorbidities like diabetes, which impair VEGF pathways and prolong remodeling [1,13].

Force application emerges as a pivotal modulator of bone remodeling, governed by mechanobiological principles. Orthodontic forces trigger Piezo1-mediated Ca^{2+} signaling, polarizing macrophages (M1 on compression, M2 on tension) and activating pathways like NF-κB and Wnt/β-catenin [15,50]. Recent investigations elucidate STAT3's role in osteoblasts as a mechanosensitive regulator, where its deletion hampers osteoclastogenesis and slows OTM [15]. Similarly, YAP mechanosensors mediate NF-κB-induced osteoclast activity, emphasizing force magnitude's impact on remodeling efficiency [51]. Optimal forces (light, continuous) minimize hyalinization and resorption, as finite element models confirm reduced stress in edentulous areas with adjusted moment-to-force ratios [47]. However, excessive forces in defective bone amplify asymmetry, with studies showing greater labial loss in aligner-treated Class III cases versus palatal in fixed appliances [3, 23]. Innovations like augmented corticotomy demonstrate preventive efficacy, preserving or increasing bone thickness during decompensation without added root resorption [17]. Yet, challenges persist in comorbid scenarios; for example, estrogen modulates OTM, and parathyroid hormone enhances homeostasis in periodontitis models via STAT3/β-catenin crosstalk [52,53]. These insights suggest that force optimization must integrate patient-specific factors, but clinical trials often overlook long-term remodeling, limiting generalizability.

Treatment planning strategies for alveolar bone loss advocate multidisciplinary sequencing, prioritizing periodontal stabilization before OTM. Expert consensus supports light forces (5-15 g) and regeneration using biomaterials like autologous dentin matrix or deproteinized bovine bone, which yield comparable augmentation in deficient sites [18]. AI-driven tools enhance precision, fusing CBCT for segmentation and predictive modeling of bone responses, achieving 95% accuracy in defect detection [3, 17]. For instance, automated 3D cephalometry systems facilitate landmark identification, aiding in boundary assessment and surgical guide design [21]. In extraction cases, planning must account for post-retraction remodeling, where maxillary alveolar process changes are pronounced but potentially recoverable long-term [14]. However, limitations abound: most studies are retrospective or narrative, with small samples and heterogeneous methodologies, risking bias [5, 23]. CBCT's radiation exposure necessitates justification, particularly in pediatrics, where evidence supports its use only for complex cases like impacted canines [49]. Moreover, while innovations like nanotechnology and photobiomodulation show promise in accelerating remodeling, their integration into routine planning lacks robust RCTs [15, 24].

Clinically, these elements inform risk mitigation; for example, in high-angle Class III patients, limiting anterior proclination prevents fenestration escalation [9,19]. Multidisciplinary approaches, combining perio-ortho interventions, yield superior outcomes in compromised periodontium, as seen in laser-assisted cases [6]. Yet, socioeconomic barriers to advanced diagnostics like CBCT and AI may limit accessibility, emphasizing the need for cost-effective alternatives. Overall, while evidence supports cautious OTM in bone loss scenarios, prospective studies with standardized protocols are essential to refine guidelines and address gaps in long-term stability.

Conclusions

This narrative review consolidates contemporary evidence regarding alveolar bone dynamics in orthodontic treatment, emphasizing the interplay between morphology, mechanical forces, and treatment planning in determining susceptibility to iatrogenic defects. The morphology of alveolar bone and gingival phenotype emerges as a critical determinant of vulnerability, with thin biotypes and patients exhibiting skeletal Class III patterns showing higher predisposition to dehiscences, fenestrations, and localized bone loss during orthodontic tooth movement. Such defects not only compromise periodontal support but can also impede the rate and predictability of tooth movement, necessitating preemptive regenerative or augmentation procedures to establish a stable foundation for orthodontic interventions.

Mechanistic studies highlight the role of cellular and molecular pathways in force-induced remodeling. Mechanosensitive signaling cascades, including STAT3, YAP/TAZ, and Wnt/β-catenin, coordinate osteoblast and osteoclast activity, modulating bone deposition and resorption in response to orthodontic forces. Understanding these pathways provides opportunities to tailor force application, predict remodeling outcomes, and explore pharmacologic or biologic adjuncts to enhance alveolar preservation. Furthermore, individualized force management—favoring light, continuous loads—and strategic application of pre-orthodontic grafting or soft tissue augmentation have been shown to mitigate iatrogenic bone loss, particularly in high-risk phenotypes.

Advances in diagnostic technology, especially the integration of high-resolution CBCT imaging with artificial intelligence (AI), have transformed treatment planning. CBCT allows volumetric assessment of alveolar bone height, thickness, and defect morphology, while AI algorithms support predictive modeling of tooth movement trajectories, force distribution, and potential dehiscence risk. Such innovations enable personalized sequencing of periodontal and orthodontic interventions, aligning regenerative procedures with mechanical treatment phases to maximize stability, minimize complications, and improve long-term outcomes. Collectively, these evidence-based strategies underscore the importance of integrating morphological assessment, biomechanical planning, and advanced diagnostics in contemporary orthodontic practice to preserve alveolar integrity across diverse patient populations.

Future Directions

Future research must address several critical gaps to advance clinical practice. Prospective, multicenter randomized controlled trials (RCTs) are essential to quantify long-term alveolar changes following orthodontic tooth movement, particularly in adult populations with comorbidities such as diabetes, osteoporosis, or age-related bone density reductions.

Longitudinal data will clarify the relationship between pre-existing bone morphology, force magnitude, and defect formation, informing guidelines for safe force application in compromised periodontia.

The development of AI-driven predictive models offers transformative potential. Machine learning algorithms could simulate individualized tooth movement, force distribution, and defect risk in real time, reducing reliance on invasive diagnostics while optimizing orthodontic planning. These models could integrate CBCT data, periodontal phenotype, and skeletal patterns to provide clinicians with evidence-based force recommendations and highlight areas requiring prophylactic augmentation.

Regenerative strategies represent another promising frontier. Biomaterials, including stem cell-enhanced grafts, growth factor-laden scaffolds, and bioactive matrices, warrant rigorous investigation for their capacity to enhance alveolar regeneration in thin or deficient bone. Adjunctive modalities such as low-level laser therapy, photobiomodulation, or pulsed electromagnetic fields could synergistically promote osteogenesis and soft tissue healing, potentially reducing treatment duration and improving stability.

Genetic and molecular investigations also hold promise for personalized care. Polymorphisms affecting key regulators of bone remodeling, such as RANKL/OPG, may stratify patients by susceptibility to orthodontically induced alveolar defects, informing individualized risk mitigation strategies. Concurrently, optimizing CBCT protocols to balance radiation exposure with diagnostic utility—particularly in pediatric and young adult populations—is essential for safe, widespread adoption .

Collectively, these advancements promise to refine risk assessment, enhance regenerative outcomes, and enable precise, individualized orthodontic care. Integrating mechanobiology, advanced imaging, AI, and regenerative adjuncts will be central to minimizing iatrogenic alveolar loss, improving treatment efficiency, and meeting the growing demand for safe orthodontic therapy among adult patients with variable periodontal profiles.

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