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Bone Changes In Geriatric Patients And Its Management In Prosthodontics: A Review

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Abstract

Geriatric patients experience significant age-associated changes in bone physiology, including reduced mineral density, alveolar ridge resorption, cortical thinning, and alterations in collagen and trabecular architecture, which compromise skeletal strength and oral support for prostheses. Systemic conditions such as osteoporosis, diabetes, and long-term medication use, along with local oral factors like thinning mucosa and decreased salivary flow, further affect prosthetic stability and healing. Maxillary and mandibular bone resorption follows distinct patterns, reducing retention and support for removable and fixed prostheses, while systemic and iatrogenic influences exacerbate ridge loss. Effective prosthodontic management requires a multidisciplinary, individualized approach integrating preventive strategies, conventional and modified prostheses, surgical interventions, and implant-supported solutions. Innovations in digital dentistry, advanced biomaterials, and regenerative therapies offer promising avenues for restoring bone volume, optimizing prosthetic outcomes, and improving function, comfort, and quality of life.

Keywords: Geriatric prosthodontics; Bone resorption; Alveolar ridge; Implant rehabilitation; Age-related Bone aging; Collagen.

Introduction

Bone, a specialized and hard connective tissue that forms the vertebrate skeleton, provides essential structure and protection for vital organs, while also facilitating movement. It's crucial for housing bone marrow for blood cell production and acts as a major reservoir for calcium and other minerals.[1] Bones are classified into several types based on their shape: long bones, short bones, flat bones, irregular bones, and sesamoid bones. Geriatrics is a medical specialty dedicated to addressing the unique health concerns of older adults, particularly those associated with aging and age-related conditions. Within dentistry, this focus extends into gerodontics, which emphasizes the oral health care and prosthodontic management of elderly individuals. A geriatric patient is typically defined as an individual aged 65 years and above, often exhibiting age-related biological changes that affect the skeletal system and other organs.[2]

Based on psychological responses to aging, geriatric patients can be grouped into three categories. The realistic group consists of individuals who adapt to aging pragmatically, accepting physical changes and planning around their limitations. The resentment group includes those who struggle with aging, often feeling frustrated or resentful about physical and social changes.[3] The resigned group comprises individuals who passively accept aging, with little motivation to maintain health or engage in preventive care. [3]Functionally, Ettinger and Beck (1984) classified the elderly based on their independence levels.

The functionally independent elderly can maintain their daily activities without assistance and generally present fewer challenges in dental care, as they are able to manage oral hygiene and visit dental clinics independently. The frail elderly require some level of support due to limited mobility, chronic illnesses, or mild cognitive impairments; they may need adjustments in treatment and assistance with dental care. The functionally dependent elderly rely fully on caregivers for daily activities and dental needs, often necessitating home or institutional care. Winkler's classification further segments elderly patients into three groups.[4]

The hardy elderly are those who maintain robust physical and mental health, showing minimal impact from the aging process and often displaying resilience in facing age-related challenges. Those with senile aged syndromes experience significant physical and cognitive decline, impacting their overall health and complicating dental care. In-between groups represent a range of conditions between these extremes, with mixed health statuses that may require variable levels of support. Aging itself can be viewed across three primary aspects: physiologic, psychologic, and pathologic. [5]

The dynamic processes of bone growth and resorption (breakdown) are influenced by several factors, including genetics, hormones (like estrogen, testosterone, and parathyroid hormone), and nutrition (adequate calcium, vitamin D, and protein are vital). Physical activity, especially weight-bearing exercise, stimulates growth, but age significantly affects bone health, as formation slows and resorption increases over time. Additionally, certain medications or diseases can accelerate bone resorption. Ultimately, calcium's role is paramount, as it is essential for mineralization, providing the bone's necessary hardness and strength; insufficient calcium intake impairs formation, while regulatory mechanisms, such as increased parathyroid hormone, can lead to increased bone resorption.[6]

The global rise in the geriatric population has created a rapidly growing demand for prosthodontic care, as elderly individuals increasingly face oral health challenges such as edentulism, tooth loss, and the influence of chronic systemic conditions on prosthetic outcomes.[4] Bone, being a dynamic tissue, undergoes significant systemic and local changes with age, including osteoporosis, reduced mineral density, and progressive alveolar ridge resorption, all of which directly affect treatment planning and the long-term success of prostheses.[5]

This article details on bone changes in geriatric patients and its management in prosthodontics

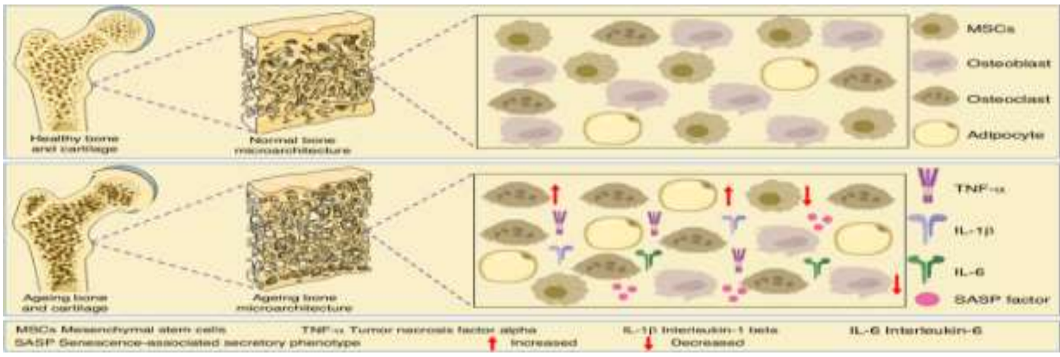

Figure 1: Physiologic bone Changes

Age-Related Changes in Bone Physiology and Their Clinical Implications

Bone is a dynamic tissue whose mechanical behavior is maintained through a delicate balance of stiffness, strength, and toughness to resist fracture while supporting skeletal function. With aging, this balance shifts due to alterations in mineral density, collagen integrity, and bone microarchitecture, resulting in stiffer yet more brittle bones that are increasingly prone to fragility and fracture.[6] Bone mineral density (BMD) naturally declines with age, particularly in conditions such as osteoporosis, although in rare cases like osteopetrosis it may increase; however, fracture risk depends not only on BMD but also on tissue-level properties, collagen cross-linking, and bone geometry.[7]

Aging is associated with cortical thinning, increased porosity, trabecular strut weakening, and disruption of collagen structure, all of which reduce resilience and toughness, while micro-damage accumulates as cracks propagate more readily due to diminished repair capacity. Morphological changes, such as periosteal expansion with endosteal resorption (cortical drift), result in wider but structurally weaker bones with thinner cortices, further compromising mechanical strength.[8] Systemic factors exacerbate these changes: hormonal imbalances (declining estrogen and androgens), vitamin D and calcium deficiency, poor nutrition, and medications such as glucocorticoids accelerate bone loss, whereas bisphosphonates and related agents may slow resorption. Additionally, chronic systemic inflammation and age-related immune activation further disrupt remodeling balance.[9,10]

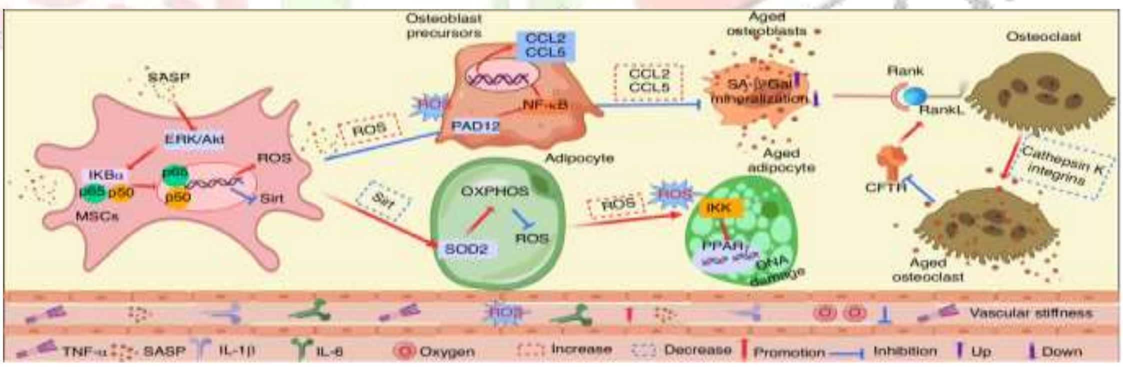

Figure 2: Bone Cell Changes

Bone Cell Aging and Structural Changes

Bone tissue is maintained and remodeled through the coordinated activity of different cell types, each of which undergoes age-related changes that impact skeletal integrity. Osteoblasts, derived from mesenchymal origin, are responsible for bone matrix synthesis and mineralization, but with age, their differentiation, lifespan, and responsiveness decline, leading to reduced bone formation.[11]

Some osteoblasts transform into osteocytes long-lived cells embedded in mineralized bone that regulate mechanotransduction and remodeling through canalicular networks. However, osteocyte apoptosis increases with age, disrupting microcrack repair and contributing to bone fragility. Osteoclasts, multinucleated cells of hematopoietic origin, mediate bone resorption, but their differentiation is impaired in older individuals, and paradoxically, aged bone is resorbed preferentially, worsening the imbalance between resorption and formation.[12]

At the molecular level, telomere shortening, oxidative stress, and reduced telomerase activity accelerate cellular senescence, while signaling pathways such as Wnt/β-catenin, which are crucial for osteoblastogenesis, decline with age. Protein and matrix-level changes further weaken bone: collagen fibrils accumulate non-enzymatic cross-links (AGEs) that stiffen the matrix and reduce toughness, while enzymatic cross-linking efficiency declines.[13] This accumulation of AGEs promotes osteoclast activity and inhibits osteoblast function, aggravating fragility.

Additionally, age alters collagen orientation and reduces the production and quality of non-collagenous proteins that regulate mineralization and signaling.[14] Mineral content also changes with age bone density initially increases but later declines, crystals enlarge and carbonate substitution rises, while hydroxyl and calcium deficiencies decrease, resulting in stiffer yet more brittle bone. [15]

Figure 3: Age Related Changes in Maxilla & Mandible

Hormonal and Systemic Influences on Bone Remodeling and Ridge Resorption

Hormones play a pivotal role in maintaining the balance of bone remodeling, while chronic diseases can significantly disrupt this equilibrium, leading to alveolar bone loss and ridge resorption, particularly following tooth extraction or periodontal disease. Estrogen acts as a protective factor by inhibiting osteoclast-mediated bone resorption and stimulating osteoblast activity through modulation of RANKL signaling; thus, its deficiency during menopause accelerates bone loss and predisposes to ridge resorption.[7] Parathyroid hormone (PTH) regulates calcium homeostasis and exerts dual effects on bone promoting either formation or resorption depending on its secretion pattern whereas growth hormone primarily enhances osteoblastic activity, supporting bone formation. In contrast, thyroid hormones, glucocorticoids, and other endocrine regulators can elevate bone turnover and, when imbalanced, lead to progressive bone loss.[13] Chronic systemic conditions such as diabetes, osteoporosis, and inflammatory diseases further aggravate resorption by inducing prolonged inflammation, altering hormonal pathways, and impairing the functional balance between osteoblasts and osteoclasts. Elevated cytokines like IL-1 and IL-6 in chronic inflammation enhance osteoclastic activity and hinder osteoblastic regeneration, resulting in compromised healing and progressive ridge atrophy. Consequently, hormonal deficiencies, dysregulated PTH levels, and metabolic or inflammatory disorders synergistically intensify ridge resorption by favoring bone loss over regeneration. Comprehensive management of hormonal health and underlying chronic conditions is therefore essential to mitigate alveolar bone loss and preserve ridge integrity in prosthodontic and periodontal patients.[17]

Age-Related Maxillary and Mandibular Changes and Their Prosthodontic Implications

With advancing age, the maxilla and mandible undergo significant anatomical and physiological changes that directly influence bone density, morphology, and the support available for prosthodontic rehabilitation. In the maxilla, progressive pneumatization of the sinus reduces the bone available for anchorage, while cortical thinning and centripetal alveolar ridge resorption lead to upward and inward bone loss, compromising denture stability and altering facial contours.[16]

In the mandible, resorption follows a centrifugal pattern,producing downward and outward bone loss that progressively decreases ridge height and width, particularly after tooth loss, which accelerates the process. These changes are compounded by reduced trabecular density and thinning cortical plates, leaving the jaw structurally weaker and more prone to fracture.[17] Clinically, such alterations reduce the retention and stability of both removable and fixed prostheses, often necessitating modifications in design and the use of specialized impression techniques or materials.

Implant placement also becomes more challenging due to limited bone volume and altered load distribution, sometimes requiring augmentation procedures for long-term success. [18]

Multifactorial Influences on Jaw Bone Resorption and Prosthodontic Implications

Bone resorption in the jaws results from a complex interplay of local, systemic, iatrogenic, and genetic factors, each of which significantly affects prosthodontic rehabilitation and patient comfort. Locally, tooth loss initiates rapid alveolar ridge resorption, particularly in the first year following extraction, while periodontal disease, trauma, parafunctional habits such as bruxism, and poor oral hygiene exacerbate the process. Ill-fitting dentures add to the problem by exerting uneven pressure, accelerating atrophy and compromising the ridge foundation.[19] Systemic influences such as osteoporosis, metabolic bone disorders, and hormonal changes including estrogen deficiency and endocrine imbalances further hasten bone loss, with nutritional deficiencies of calcium and vitamin D compounding the decline in bone quality. Iatrogenic factors also play a role, as poorly executed extractions, surgical trauma, or inadequately designed prostheses can trigger or worsen resorption by causing instability and excessive occlusal loading.[20] Additionally, genetic predisposition contributes to individual variability, with inherited traits affecting osteoblast activity, bone density, and susceptibility to conditions like osteoporosis; anatomical features such as thin or narrow ridges make certain patients more prone to accelerated resorption.[21]

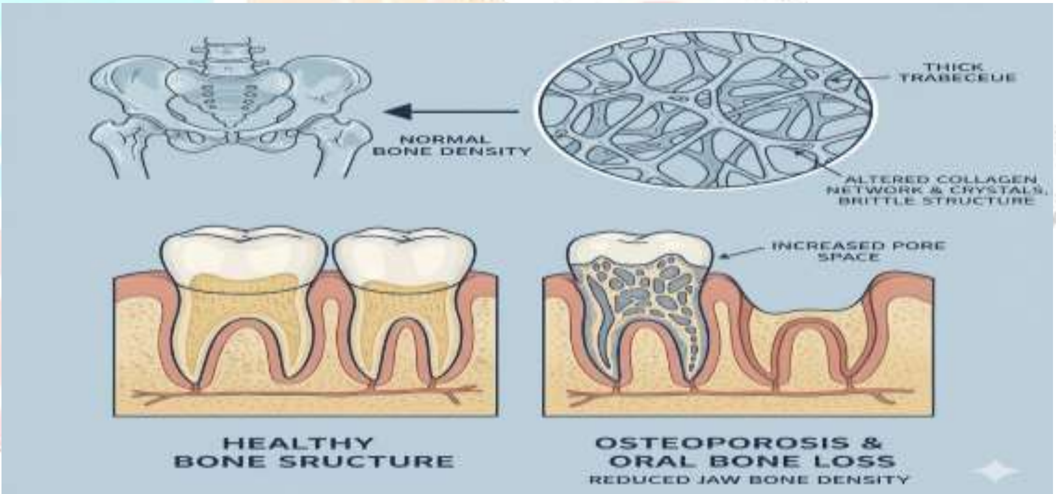

Figure 4: Osteoporosis

Osteoporosis and Oral Bone Loss: An Age-Associated Condition

Osteoporosis, often termed the “silent disease,” is marked by reduced bone mass and increased fracture susceptibility, but it should not be regarded strictly as a disease of aging, rather as one that is age-associated. While bone strength and mass decline with age, not all elderly individuals display skeletal fragility or fractures, and osteoporosis can also occur in the very young, as seen in idiopathic juvenile osteoporosis.[22] Characterized by thinner trabeculae and cortices, altered collagen maturity, and increased mineral crystal size, osteoporotic bone fractures more easily than normal bone. Although fragility fractures rise sharply with age, genetic factors, systemic influences, and individual variability highlight that osteoporosis is not exclusively age-related. In the oral cavity, alveolar bone formed through intramembranous ossification also undergoes age-associated changes, with mandibular bone being more severely affected than the maxilla.[23] Clinical studies reveal moderate correlations between systemic bone mineral density and alveolar bone quality, though findings remain inconsistent due to methodological differences, small samples, and confounding factors such as tooth loss, mechanical stress, and oral hygiene status. Similarly, animal models, including ovariectomized rats and monkeys, demonstrate mixed associations between systemic osteoporosis and jawbone changes, further complicated by the role of periodontal disease, which itself drives alveolar bone resorption through chronic inflammation.[24]

Category	Factor	Description / Impact on Ridge Resorption
Systemic Factors	**Osteoporosis**	Reduces bone mineral density, making jawbone more prone to rapid resorption after tooth loss, especially in postmenopausal women.
	Endocrine Disorders	Conditions such as hyperparathyroidism, hyperthyroidism, and estrogen deficiency disrupt bone metabolism, increase turnover, and accelerate ridge loss.
	Malnutrition	Deficiency of protein, calcium, or vitamin D impairs bone regeneration and healing, amplifying post-extraction bone loss.
Local Factors	**Trauma**	Repeated mechanical injury or excessive occlusal forces damage alveolar bone, hastening localized resorption.
	Periodontal Disease	Chronic inflammation leads to destruction of periodontal support and bone before tooth loss, predisposing to faster post-extraction resorption.
	Ill-Fitting Dentures	Uneven pressure and chronic irritation from poorly fitting dentures reduce blood supply, causing localized bone loss; frequent relining indicates progressive resorption.
Clinical Implication		Both systemic and local factors must be identified and managed to minimize alveolar ridge resorption and improve long-term prosthetic and implant outcomes.

Management of Bone Resorption and Compromised Ridges in Prosthodontics

The management of bone resorption and compromised ridges in prosthodontics requires a comprehensive and individualized approach that integrates preventive strategies, conventional prosthodontics, surgical adjuncts, and implant-based solutions to achieve optimal rehabilitation.[25] Preventive measures such as ridge preservation following extractions, retention of natural dentition whenever possible, and systemic optimization through adequate nutrition, vitamin D, calcium intake, and management of endocrine disorders help minimize bone loss and preserve alveolar integrity.[16,26] Conventional prosthodontic techniques, including modified denture designs like hollow maxillary dentures and cheek plumpers, the use of tissue conditioners and resilient liners, and functional impression methods such as the neutral zone technique and differential pressure impressions, enhance retention, stability, and patient comfort in atrophic ridges. When anatomical deficiencies severely limit prosthesis support, surgical interventions such as ridge augmentation, vestibuloplasty, sinus lift procedures, bone grafting, or removal of flabby tissues may be employed to restore foundation and improve prosthetic outcomes.[27] Implant-based rehabilitation plays a vital role, with options ranging from mini-implants and short implants to overdentures that provide enhanced stability and retention, especially in elderly and osteoporotic patients. [28]

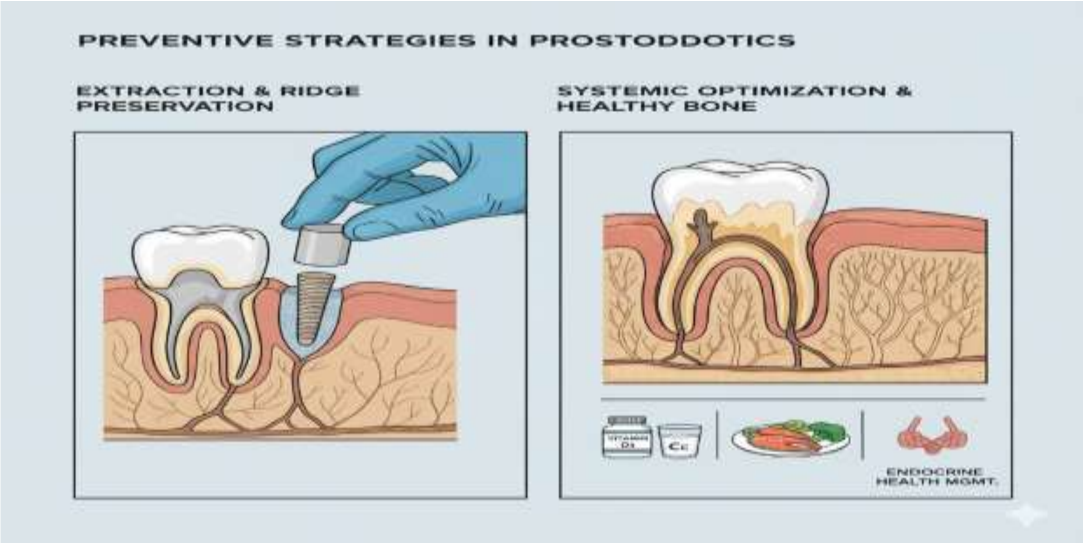

Figure 5: Management Strategies

Management of Bone Resorption and Compromised Ridges in Prosthodontics

Approach	Techniques / Methods	Description / Clinical Significance	References
Preventive Strategies	**Ridge Preservation after Extraction**	Early intervention with bone grafts or biomaterials immediately after extraction helps maintain alveolar ridge height and width for future prosthetic or implant use.	25
	Preservation of Natural Dentition	Retaining healthy teeth prevents further bone resorption and maintains alveolar structure, reducing the need for extensive prosthetic rehabilitation.	16
	Nutritional and Systemic Optimization	Adequate calcium, vitamin D, and protein intake, along with management of systemic and endocrine disorders (like diabetes and thyroid dysfunction), enhances bone metabolism and limits ridge loss.	26
Conventional Prosthodontic Management	**Modified Denture Designs**	Hollow maxillary dentures reduce prosthesis weight, and cheek plumpers improve facial esthetics in atrophic cases without compromising retention.	25
	Tissue Conditioners and Resilient Liners	Provide cushioning effect, adapt to mucosal changes, and improve retention and patient comfort in resorbed ridges.	26
	Neutral Zone Technique	Places teeth and denture base in harmony with surrounding musculature, improving mandibular denture stability and retention in severely resorbed ridges.	25

Approach	Techniques / Methods	Description / Clinical Significance	References
	Functional Impression Techniques	Includes admixed, differential pressure, and window tray methods; ensures minimal tissue displacement, accurate recording of flabby areas, and better prosthesis adaptation.	27
Surgical / Adjunctive Interventions	**Ridge Augmentation**	Uses bone grafts or biomaterials to increase ridge volume and provide a stable foundation for prostheses or implants.	27
	Vestibuloplasty	Deepens the vestibule to improve denture flange extension, retention, and stability.	27
	Sinus Lift Procedure	Elevation of the maxillary sinus floor allows placement of implants in posterior maxilla with reduced bone height.	27
	Bone Grafting and Removal of Flabby Tissue	Reconstructs severely atrophic ridges and eliminates mobile tissue to improve denture adaptation and long-term support.	27
Implant-Based Prosthodontics	**Mini-Implants / Short Implants**	Provide reliable anchorage in limited bone volume without extensive grafting; suitable for geriatric and osteoporotic patients.	28
	Overdentures	Implant-retained overdentures improve stability, retention, and comfort compared to conventional dentures, with reduced loading on residual ridges.	28
	Loading Protocols	Immediate vs. delayed loading must be selected based on bone quality and healing capacity; delayed loading preferred in elderly or high-risk cases.	28

Future Directions

Digital dentistry, particularly CAD-CAM technology, has transformed denture fabrication through the use of digital scans and computer-aided design, allowing virtual articulation, precise tooth arrangement, and highly customized prostheses with superior fit, strength, and reduced porosity compared to conventional acrylic dentures. These digitally milled or 3D-printed prostheses not only minimize chairside visits but also improve patient safety, comfort, and longevity, while the integration of artificial intelligence, advanced imaging, and teledentistry further enhances diagnostic accuracy, treatment planning, and accessibility for elderly populations. Simultaneously, novel biomaterials such as bioactive ceramics, composite scaffolds, and growth factor-enriched resorbable membranes are advancing ridge preservation techniques, facilitating bone regeneration and creating stronger foundations for future prosthetic and implant rehabilitation. Parallel to this, stem cell–based regenerative strategies are emerging as a groundbreaking approach, with mesenchymal stem cells combined with scaffolds and bioactive molecules showing potential to rebuild atrophic ridges, restore bone volume, and improve implant integration in aging jaws.[29,30]

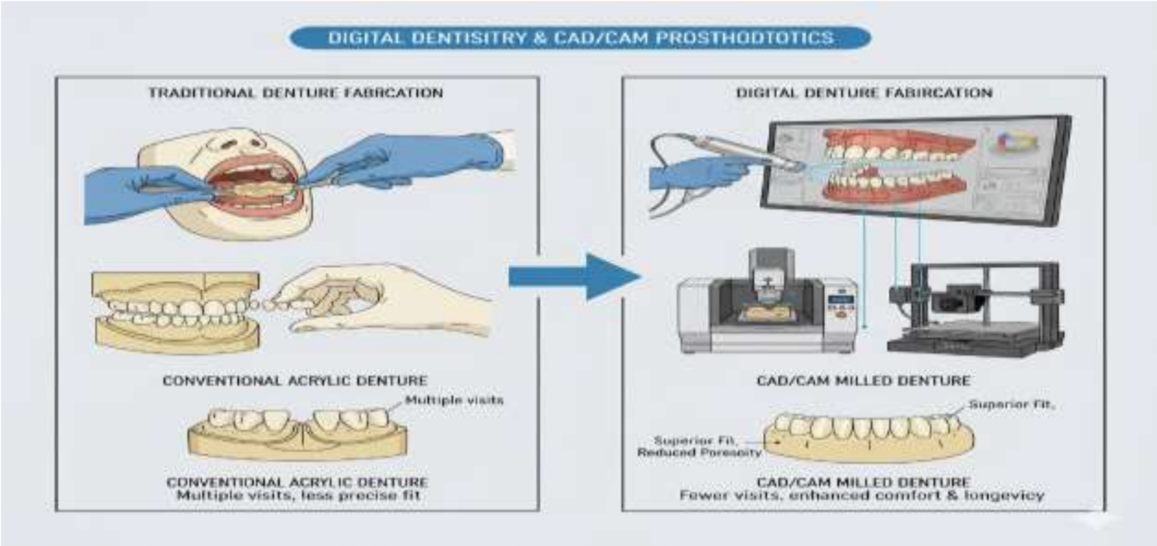

Figure 6: CAD/CAM

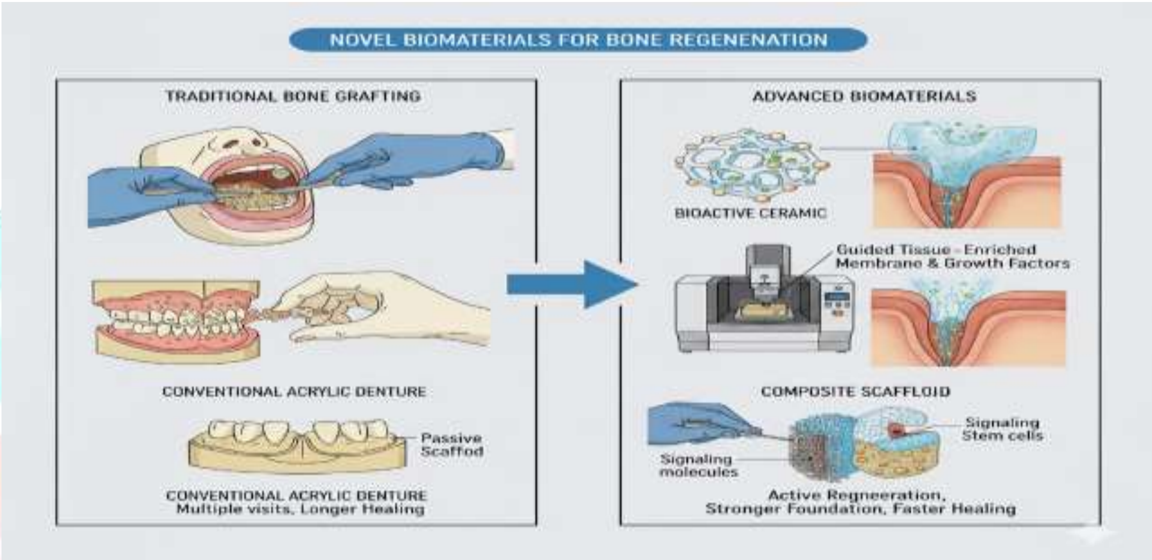

Figure 7: Bio Materials

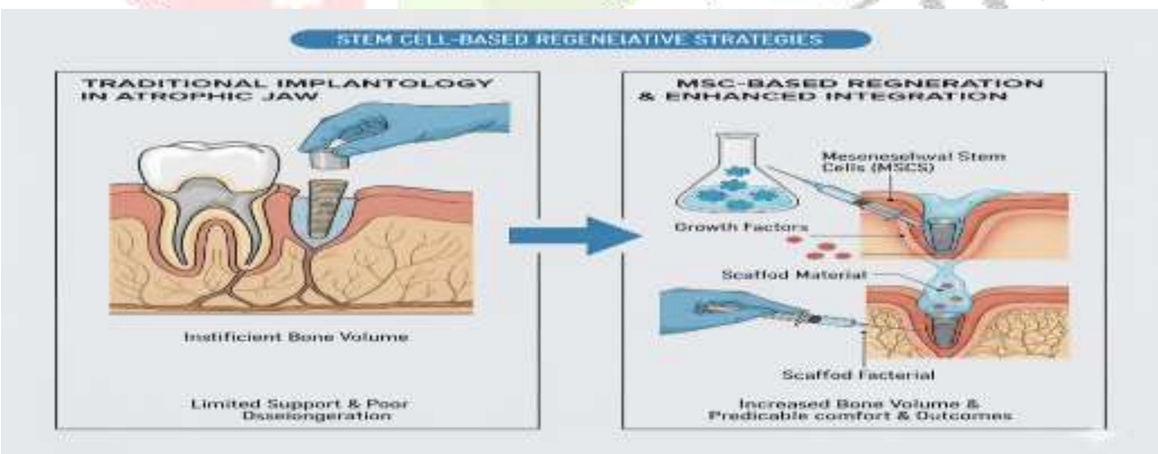

Figure 8: Stem Cell-Based Regenerative Strategies

Digital Dentures	Digital dentures, which utilize CAD/CAM design and milling or 3D printing, offer improved adaptation, better occlusal force distribution, and enhanced fit compared to conventional dentures. Recent studies show that milled implant-assisted overdentures may be especially favorable for posterior ridge preservation compared to 3D-printed versions, though both are superior to traditional fabrication techniques in force distribution and tissue adaptation. These technologies can minimize chronic trauma from ill-fitting dentures, a key contributor to alveolar bone loss.
Biometric Prosthetic Materials	Biometric prosthetic materials, including bone grafts (alloplasts, xenografts) and growth factor-laden scaffolds, are increasingly incorporated into socket preservation and ridge grafting. These materials promote natural bone regeneration, limit horizontal and vertical ridge loss after extractions, and help maintain bone volume for implants or prosthetic support. Advances in biomaterials also improve integration and longevity, enhancing clinical outcomes.
Stem Cells and Tissue Engineering	Stem cell-based approaches and tissue engineering methods hold great promise for alveolar ridge preservation. Mesenchymal stem cells can be seeded onto bioactive scaffolds to promote rapid and physiological bone healing, potentially reversing bone loss and regenerating alveolar bone structure after tooth extractions. Tissue-engineered constructs combine cells, growth factors, and biomechanically supportive frameworks to facilitate bone and soft tissue repair, emerging as adjuncts to current regenerative procedures in dentistry.

Conclusion

Aging profoundly influences the structure and physiology of bone, resulting in progressive alveolar ridge resorption, reduced bone mineral density, and compromised structural integrity that collectively challenge prosthodontic rehabilitation. These changes stem from cellular and molecular alterations in osteoblasts, osteocytes, and osteoclasts, which regulate the delicate balance between bone formation and resorption. The decline in osteoblastic activity, reduced vascularity, hormonal fluctuations, and systemic conditions such as osteoporosis or endocrine imbalances further accelerate bone loss in geriatric individuals.

Given the complex interplay of local, systemic, and iatrogenic factors in bone resorption, successful prosthodontic management must adopt a personalized and comprehensive approach. This includes early preventive interventions such as ridge preservation and maintenance of natural dentition, precise prosthodontic techniques tailored to residual ridge morphology, and the use of advanced materials and digital workflows to enhance precision and comfort. Surgical adjuncts and implant-based solutions, including mini-implants and overdentures, offer valuable options for restoring stability and function even in cases of severe atrophy.

Ultimately, individualized treatment planning that integrates systemic health optimization, bone biology understanding, and patient-centered considerations is critical for achieving long-term prosthetic success. The future of geriatric prosthodontics lies in the seamless combination of regenerative, digital, and evidence-based innovations to restore not only oral function but also the confidence, esthetics, and overall quality of life of aging patients.

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