TITLE: PHYSIOLOGY AND METABOLISM OF ALVEOLAR BONE

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ABSTRACT

The portion of the maxillary and mandibular bones that contains and supports the tooth sockets, or alveoli, is called the alveolar bone. It is a very active tissue that reacts to external influences to keep the balance between the resorption and production of new bone. Osteoblasts, or cells that produce bone, and osteoclasts, or cells that resorb bone, control this balance. A variety of systemic and local variables can affect this process. To successfully install dental and implant prostheses and control the alveolar bone's reaction to prosthetic loads, it is essential to comprehend the physiology and metabolism of the bone. Though this topic is basic knowledge for the dentists, but one of the less spoken also. Hence this review aim to discuss the alveolar bone properties, the physiology and metabolism of alveolar bone and its response to prosthetic restoration loads.

Keyword *: Alveolar Bone[1]; Osteoclastic Activity[2]; Osteoblastic Activity[3]; Bone Resorption[4].*

1. INTRODUCTION

The alveolar bone is formed during tooth development, and the presence of teeth is essential for the bone's growth. The alveolar bone changes as the teeth emerge and enter a functional occlusion to handle the occlusal load and mastication pressures.[1]

The balance between osteoclastic resorption and osteoblastic bone creation, which is especially active during childhood and adolescence when teeth are erupting, maintains the bone.

1. Bone Structure and Composition

One particular kind of cortical and trabecular bone is the alveolar bone. Its composition is made up of:

The outer cortical layer is compact and dense, giving it structural strength. The interior trabecular bone is a high-surface-area, pliable bone that helps with metabolic processes including the exchange of calcium and phosphate.

The connective tissue that holds teeth to the alveolar bone is called the periodontal ligament (PDL). Mechanotransduction, the process by which forces applied to teeth are transferred to the alveolar bone, involves the PDL. [2]

Ten Cate AR, et al. claim that a more prominent remodelling activity results from the mastication's continuous forces. [3]

2. Mechanotransduction and Bone Remodelling

Alveolar bone is constantly remodelling as a result of osteoclasts resorbing old bone and osteoblasts forming new bone. Biochemical cues and mechanical loading control this. Under Wolff's Law, bone tissue adjusts to the quantity and direction of functional forces, the bone responds to masticatory forces, tooth movement, and prosthetic loads.

Loading: Osteoblast activity is stimulated by the physiological pressures of mastication and occlusion, which promotes the production of new bone. Bone health and density are dependent on controlled and uniformly distributed stresses.

Emptying or Overloading: A lack of loading (as in tooth loss) causes disuse atrophy and alveolar bone resorption, whereas excessive or inappropriate loading (as in poorly constructed prostheses) can cause micro-damage and hasten bone resorption.

According to Frost HM, alveolar bone cells react to signals of strain; sufficient loading encourages the production of new bone, whereas insufficient loading results in the resorption of existing bone. On the other hand, Duyck J. and associates showed that implant-supported prostheses had a major impact on the alveolar bone's biomechanics, with the amount of stress influencing the remodelling of the bone surrounding the implants.[5]

3. Activity of the Osteoblast and Osteoclast

The metabolic status of the alveolar bone is determined by the dynamic balance between osteoblast activity (bone creation) and osteoclast activity (bone resorption). During resorption, osteoclasts disintegrate the bone matrix, releasing phosphate and calcium into the blood. Next, new bone matrix (osteoid) is created by osteoblasts and later mineralises to produce new bone.

Resorption of Bone: The process of bone resorption is mediated by osteoclasts and is controlled by substances such as RANKL (Receptor Activator of Nuclear Factor Kappa-B Ligand) and its receptor RANK, which promote the development of osteoclasts. Pathological bone loss may result from overactivation of this system, which may occur in response to high prosthesis loading.

Bone Formation: Growth factors, mechanical stimulation, and hormones including vitamin D and parathyroid hormone (PTH) control osteoblast activity. Osteogenic signals, such as Bone Morphogenetic Proteins (BMPs), are stimulated by mechanical loading and lead to increased osteoblast activity and differentiation.

According to Teitelbaum SL, the molecular pathways RANK/RANKL/OPG signalling are important for bone metabolism, and osteoclasts play a role in bone resorption.[6] Klein-Nulend J and colleagues demonstrated that mechanical stress causes osteoblasts to release prostaglandins and nitric oxide, which in turn promotes the creation of new bone and controls the activity of osteoclasts.[7]

4. Loss of Alveolar Bone Following Teeth Extraction

Alveolar bone resorption is the consequence of a major decrease in mechanical stimulation brought on by tooth loss. This process is more noticeable in the vertical and horizontal dimensions of the alveolar ridge and is particularly quick in the initial months following extraction. Disuse atrophy of the alveolar ridge is caused by the absence of stimulation from the periodontal ligament, which typically transfers occlusal stresses to the bone.

Two thirds of the ridge was lost during the first three months following tooth extraction, making it difficult to place dental implants or support conventional prostheses. Alveolar bone loss in the first twelve months following tooth extraction ranges from 11% to 22% of alveolar bone height and 29%–63% of the width.[8]

Alveolar bone loss may be made worse by prosthetic devices, particularly detachable dentures that do not transfer occlusal forces to the underlying bone.

Atwood DA (1971): Preliminary research revealed that alveolar ridge resorption, which can jeopardise the insertion of a prosthesis or implant, is caused by the absence of functional loading after tooth removal.[9]

5. Hormonal Regulation and Bone Metabolism

Like all bone tissue, alveolar bone is metabolically active and regulated by a number of systemic hormones, including:

In low calcium environments, parathyroid hormone (PTH) promotes bone resorption and controls calcium levels.

Calcitonin: Lowers bone resorption by inhibiting osteoclast activity.

Vitamin D: Promotes osteoblast function and the creation of new bone, and is necessary for the absorption of calcium and bone mineralisation.

Sex Hormones: The maintenance of bone mass is significantly influenced by both testosterone and oestrogen. Compston JE (2015), Highlighted the function of oestrogen in limiting bone loss, explaining the rise in alveolar

bone resorption seen in postmenopausal women. A reduction in oestrogen, such as after menopause, accelerates bone loss, including in the alveolar bone.[10]

Holick MF (2008), discussed how important vitamin D is for bone health and calcium metabolism, especially in relation to how it affects osteoblast function and bone production.[11]

6. Systemic Factors Influencing the Metabolism of Alveolar Bone

Hormonal Effects: The metabolism of alveolar bone is strongly influenced by hormones such growth hormone, testosterone, and oestrogen. In postmenopausal women, a decrease in oestrogen can hasten bone resorption, increasing alveolar bone loss and increasing the risk of dental implant failure.

Osteoporosis: This systemic disease causes changed bone quality and decreased bone mass, which increases the susceptibility of alveolar bone to resorption. Following tooth extraction or implant implantation, patients with osteoporosis may experience quicker rates of alveolar ridge loss, necessitating bone augmentation operations to assure implant success. [12]

7. Molecular and Cellular Processes

Osteoclasts: Made up of both osteoblasts and other cells, these bone-resorbing cells are triggered by signalling molecules like RANKL. In osteoclast precursors, RANKL binds to RANK to promote osteoclast activation and differentiation. The resorption of old bone and the generation of space for the growth of new bone depend on osteoclasts.

Osteoblasts: These are the cells that produce bone; they are in charge of creating new osteoid bone matrix, which subsequently mineralises into mature bone. Numerous local and systemic variables, including as growth factors (such BMPs) and hormones, as well as mechanical loads, control osteoblasts.

Osteocytes: Osteocytes, which are derived from osteoblasts, are embedded in the bone matrix and function as mechanosensors. They sense variations in mechanical strain and coordinate bone remodelling by communicating with osteoblasts and osteoclasts.[7]

8. Impact of Implant and Prosthetic Loading on Bone Physiology

Alveolar bone metabolism can be greatly impacted by prosthetic rehabilitation using implants, such as All-on-4 or All-on-6 systems. An implant-supported prosthesis with a well-designed distribution of occlusal stresses promotes bone preservation by reducing osteoclastic resorption and promoting osteoblastic activity. Conversely, overloading, micromovement, and accelerated bone resorption can result from inappropriate load distribution, high occlusal forces, or cantilever designs.

According to Misch CE, preserving the physiology and integrity of alveolar bone depends on effective load management.[13]

Well-distributed loads encourage bone remodelling, whereas excessive or poorly controlled loads cause peri-implant bone loss, according to research by Duyck J. and colleagues.

9. The Periodontal Ligament's (PDL) Function in Bone Metabolization

The physiological adaptation of the alveolar bone to functional loads is largely dependent on the PDL. By serving as a cushion, it transfers the occlusal stresses from the tooth to the surrounding bone and shields it from undue strain.

Mechanical stresses applied to teeth, such as those from orthodontic braces or chewing, cause the PDL to initiate a series of cellular signals that promote bone remodelling. Different biological reactions occur on the tension and compression sides of the PDL, resulting in the promotion of bone growth on the tension side and bone resorption on the compression side. [14]

10. Bone Remodelling in Reaction to Implant and Prosthetic Loads

The way forces are conveyed to the alveolar bone in the setting of dental implants is changed when the PDL is absent. Since implants are osseointegrated into the bone directly, there is no longer a PDL to cushion shock, which alters load distribution and highlights the significance of appropriate occlusal force management.

Properly positioned implants that are exposed to physiological stresses encourage the peri-implant bone to reorganise and preserve its structure. However, high stresses can result in marginal bone loss and implant failure (e.g., occlusal loading, bruxism, or poorly constructed prostheses).[5]

11. The Effect of Stress and Occlusal Forces on Alveolar Bone

Alveolar bone health is greatly influenced by the kind, strength, and direction of occlusal pressures. The long axis of the tooth or implant experiences axial forces, which are usually well tolerated and encourage bone remodelling. On the other hand, non-axial forces like lateral or oblique forces can cause microfractures and stress concentrations in the bone, especially in the vicinity of implants.

The distal implants or teeth are subjected to excessive forces by cantilever designs (prostheses with unsupported extensions), which raises the possibility of bone resorption in such regions. To prevent bone loss, prostheses must be designed properly, with the smallest possible cantilever lengths and a uniform distribution of occlusal forces.

Misch CE (2005), Provided comprehensive guidance for prosthetic design to reduce unfavourable stress and maintain bone health, as well as highlighting the effect of occlusal forces on alveolar bone and implant success.[13]

12. Implant periprosthetic Loss of Bone and Stability of Implants

Increased occlusal stress, micro-movements, or biological problems like infection or peri-implantitis are frequently indicated by peri-implant bone loss. During the first year after implant placement, when bone remodelling is at its peak, this loss is very frequent.

To guarantee a successful osseointegration, primary stability the implant's early mechanical stability is crucial. Excessive functional loading of implants during the healing phase can interfere with bone healing and cause early implant failure.

Esposito M et al., (1998) reviewed the effects of loading regimens on preserving implant stability and peri-implant bone loss, coming to the conclusion that carefully controlled occlusal forces greatly extend the life of implants. [15]

13. Bone Augmentation and Grafting

In situations where there has been a loss of alveolar bone as a result of trauma, periodontal disease, or tooth extraction, bone grafting and other augmentation methods (such guided bone regeneration and sinus lifts) could be required to restore enough bone volume for dental implants.

Bone biology and the graft material's capacity to fuse with the surrounding bone tissue are key factors in these treatments' success. In reaction to mechanical loading, grafted bone experiences a remodelling process like to that of native bone, wherein it is eventually replaced by new bone tissue. [16]

CONCLUSIONS

Alveolar bone physiology and metabolism are controlled by a complex interplay of biochemical signals, mechanical loads, and systemic variables. Osteoclast-mediated resorption and osteoblast-mediated creation are two remodelling mechanisms that depend on mechanical stimuli, principally from mastication and occlusal stresses, to sustain bone mass. Hormone modulation and prosthetic load distribution further impact bone metabolism. It is crucial to comprehend these elements in order to preserve the health of the alveolar bone in patients receiving prosthetic or implant rehabilitation and to stop bone loss following tooth extraction.

REFERENCES

1.Nanci A, Ten Cate's Oral Histology. 2012. 9th Edi, Elsevier, Canada.
2.Maiko Omi, Yuji Mishina. Roles of osteoclasts in alveolar bone remodeling. Genesis 2022 ; 60(8-9): e23490
3.Ten Cate, A. R. , & Mills, C. (1972). The development of the periodontium: The origin of alveolar bone. The Anatomical Record, 173(1), 69–77.
4.Frost HM. Bone's mechanostat: a 2003 update. Anat Rec A Discov Mol Cell Evol Biol. 2003;275:1081–1101.
5.Duyck J, et al. 2010. Histological, histomorphometrical, and radiological evaluation of an experimental implant design with a high insertion torque. Clin. Oral Implants Res. 21, 877–884.
6.Teitelbaum SL. Osteoclasts and integrins. Ann N Y Acad Sci. 2006;1068:95–99.

7.Klein-Nulend J, R.G. Bacabac RG Bakker AD. Mechanical loading and how it affects bone cells: the role of the osteocyte cytoskeleton in maintaining our skeleton. Euro Cells Mater 2012;24 : 278-291.
8.Cohen, M. S., Aizenbud, D. The effect of tooth loss on the alveolar ridge: A systematic review of the literature. Journal of Oral Rehabilitation 2018; 45(1), 43-54.
9.Atwood DA. Reduction of residual ridges: a major oral disease entity. Journal of Prosthetic Dentistry 1971;26(3):266-79.
10.Compston J. Obesity and fractures in postmenopausal women. Curr Opin Rheumatol 27: 414–419, 2015.
11.Holick MF, Biancuzzo RM, Chen TC, et al. Vitamin D2 is as effective as vitamin D3 in maintaining circulating concentrations of 25-hydroxyvitamin D. J Clin Endocrinol Metab. 2008;93:677–81.
12.Wang C-W, McCauley LK. Osteoporosis and Periodontitis. Curr Osteoporos Rep. 2016 Dec; 14(6): 284–291.
13.Misch CE, Suzuki JB, Misch-Dietsh FM, Bidez MW. A positive correlation between occlusal trauma and peri-implant bone loss: Literature support. Implant Dent. 2005;14(2):108–16.
14. Bartold PM, Shi S, Gronthos S. Stem cells and periodontal regeneration. Periodontology 2000. 2006;40:164–72.
15. Esposito M., Hirsch J.M., Lekholm U., Thomsen P. Biological factors contributing to failures of osseointegrated oral implants. (I). Success criteria and epidemiology. Eur. J. Oral Sci. 1998;106:527–551.
16. Donos N, Akcali A, Padhye N, Sculean A, Calciolari E. Bone regeneration in implant dentistry: Which are the factorsaffecting the clinical outcome?. Periodontology 2000. 2023;93:26–55.