Osseous Remodeling Around Dental Implants

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Adequate bone remodeling may be a primary parameter for long-term successful complication-free dental implant treatment. A 1.8-mm osseous thickness around dental implants is thought to be the minimum thickness for adequate vasculature for osteocyte nutrition and function. A dental implant does not provide progenitor cells or angiogenic or osteogenic factors. Thus, the surrounding bone may need to have a 1.8-mm thickness to accommodate the vasculature necessary for nutrients for appropriate remodeling. Additionally, the 1.8-mm dimension may provide for mechanical load resistance. There is no evidence to illustrate the physiologic need for the 1.8-mm dimension. This dimension requirement is based on clinical outcome observations. Basic science research for bone survival around dental implants is needed.

Key Words: dental implant, osseous, bone, remodeling, dimension, vascular supply

INTRODUCTION

For a long-term functional and esthetic outcome of dental implant treatment, a minimum of 1.8-mm thickness of surrounding bone has been advocated.[1] The reason for this dimension is not well understood but it may be because of physiologic bone remodeling.

Bone renews throughout life. It is a remodeling process where osteoclasts and osteoblasts play critical roles. Remodeling is important for bone mass, bone strength, and mineral homeostasis.[2] There is stringent regulation of remodeling by cytokine communications among osteoclasts, osteoblasts, and osteocytes. An imbalance of this process can result in osseous resorption.[2] Bone remodeling around dental implants occurs just as in natural teeth.[2]

Vascularization is fundamental for bone formation and bone tissue homeostasis. Around natural teeth the vasculature consists of periosteal, intraosseous, and periodontal ligament arteries (Figures 1 through 4). With dental implants there are no periodontal ligamental arteries, so the blood supply is compromised. The cortices of natural teeth can be very thin and still be maintained but thin bone around dental implants can undergo resorption.[1] Many natural teeth have osseous dehiscences and maintain functional stability but may not maintain gingival height and facial gingival attachment.

Dental implants do not have a periodontal ligament (PDL). The PDL provides vascularity and progenitor stem cells.[3] Some types of stem cells originate from perivascular cells and then congregate around blood vessels for angiogenesis.[4] Thus, dental implants may not enjoy an equivalent remodeling functionality as does the bone around natural teeth.

Alveolar bone remodeling is importantly influenced by occlusal loading on teeth, implants, and dental prosthetics.[2] Dental implant success and longevity is determined by the health of the surrounding bone.[2] Bone remodeling is critically important for bone maintenance for stability and implant survival.

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This endeavor is a discussion of a hypothesis that states: "Bone thickness around dental implants needs to be at least 1.8mm for a long term functional and esthetic outcome."[1] This thickness may be a physiologic requirement for vascular supply and bone remodeling.

ANGIOGENESIS AND OSTEOGENESIS

It has been suggested that there needs to be a minimum of 1.8 mm of facial bone for a successful long-term implant functional and esthetic outcome.[1] The 1.8-mm dimension of bone thickness may allow for an adequate vascular supply. There may be a physiologic dimensional requirement that allows appropriate bone remodeling provided by a vascular supply. Bone physiology may require a minimum thickness for healthy, normal remodeling. This apparently may be 1.8-mm minimum thickness. If 1.8-mm is necessary for long-term successful outcome, then this dimension may be critical for remodeling.[1]

Osteogenesis and angiogenesis work in tandem during bone formation, homeostasis, and repair.[4] Blood vessels carry oxygen and nutrients for osteogenesis and remodeling. Tissue hypoxia is a major stimulus for angiogenesis.[5,6] Hypoxia activates factor alpha (HIF alpha) pathway, a central regulator of hypoxia adaptation. HIF alpha triggers hypoxia-responsive gene expression, vascular endothelial growth factor (VEGF). This is critical for angiogenesis and bone repair. VEGF exerts its action on bone indirectly by stimulation of angiogenesis.[5,6]

Osteoconduction, osteoinduction, and osteogenesis are basic healing processes of grafted bone sites. All these require new vascularization. Angiogenesis plays an important role in regeneration of tissues, in inflammation, degenerative disease, and the formation of neoplasms.[7] Angiogenesis is important in engineered bone regeneration for dental implants. Insufficient vascularization will result in poor healing or fibrosis if there no vascular emanations from the patient's surrounding bone.[7]

There are basically two ways for graft vascularization: (1) graft material that directs and facilitates bone and cell growth, and growth factors or (2) drugs that promote regeneration. Most graft materials provide a scaffold for tissue ingrowth and do not provide growth factors.[7] Much of implant dentistry

involves bone regeneration, via grafting, that requires angiogenesis for osteogenesis.

There are a variety of clinical classification schemes for optimal implant osseointegration.[8] These are usually based on the local structural characteristics of bone such as the classifications of the Misch bone type density.[8]

The biological characteristics of the local bone around an implant is important. For example, in healing Misch type 3 bone there is a positive correlation among remodeling rates, mitotic activity, osteotomy site healing, and high endogenous Wnt metabolic protein signaling pathways (the name Wnt is a portmanteau of Wg and int, and stands for "wingless-related integration site" conjured from the genetic research behind this). The healing osteoid matrix that is responsible for implant osseointegration originates from Wnt-responsive cells and their progeny.[8] The molecular role of bone biology and oral mechanics are responsible for a long-term successful dental implant outcome.[8]

Matrix metalloproteinases (MMPs) constitute a large group of extracellular matrix proteases active in many physiological and pathological processes, including angiogenesis.[9] The activity of MMPs in vessel formation is not well studied. They act to remove cells that may be in the path of vessel proliferation. Additionally, MMPs have been found to be required for angiogenesis in collagen and fibrin matrices. Stromelysin MMP-11 and membrane-type-1-MMP are highly active during collagen vessel formation. Gelatinase MMP-2 and stromelysin MMP-3 are present at high levels in fibrin formation. MMPs are thought to have a major role in angiogenesis.[9]

Impaired angiogenesis is associated with abnormalities in bone and osteoporosis. Enhancing vessel formation in bone is important for new bone formation. Type H capillary blood vessels are associated with osteogenesis and increased osseous activity.[10] This is type of capillary is associated with osteoprogenitors and couple angiogenesis with osteogenesis.[4] Type H capillaries help to regulate bone metabolism.[11] Nonetheless, a direct molecular function has not yet been found that relates angiogenesis and cytokine activity that promotes bone disease or age-related bone loss. Type H capillaries and osteoprogenitors may help prevent loss of osseous microarchitecture and osteopenia.[11]

The parenteral iron and aluminum chelating agent, desferioxamine, (Deferal, Novartis, E Hanover, NJ) is used after multiple blood transfusions.[11] It enhances angiogenesis, osteogenesis by promoting type H capillary formation, and bone mass.[12] An abundance of these H vessels is an early marker of bone remodeling. The H capillaries are a potential therapeutic target for improving bone quality by inducing their proliferation.[12] A low number of type H capillaries may indicate osteopenia and is not desirable around dental implants.[12]

Bone sialoprotein and osteopontin are small integrin binding ligand N-linked glycoproteins that are involved in bone formation, hematopoiesis, and angiogenesis.[13] Any lack bone sialoprotein and osteopontin affects the metabolism of hematopoiesis, angiogenesis, and osteogenesis.[13] Patients with poor nutrition may have no or low production of these glycoproteins.[14] Thus, poor nutrition may be a relative contraindication for dental implant treatment.

There are agents that can stimulate angiogenesis. New vasculature formation during bone graft healing can be increased by doping tricalcium phosphate graft material with silicon dioxide and zinc oxide.[15] Very small amounts of these oxides apparently stimulate angiogenesis. Additionally, systemic administration of venom from stinging nettle (*Urtica dioica*) may be effective in accelerating new bone formation and reducing inflammation in orthopedic maxillary expansion and prevent positional relapse.[16] It can induce new bone and capillary formation as well as increase osteoblasts and osteoclasts.[16] There may be additional undiscovered agents that stimulate angiogenesis.

It has been found that nerves migrate in the absence of blood vessels.[17] A low oxygen tension can cause neurons to retract from such an area and migrate to reform in an area with appropriate vasculature.

Periosteum is responsible for apposition and resorption during osseous growth.[18,19] Periosteum is critical for bone regeneration via its stem/osteoprogenitor cell content. Normal healing after invasive dental procedures is importantly linked to the reestablishment of the periosteal microcirculation.[20] The reconstruction and repair of lost tissues is also performed by periosteum. Periosteum initiates cell differentiation during bone repair and remodeling.[20]

The maxilla and mandible have differences in their blood supply and have fracture healing characteristics similar to, but not exactly the same as the long bones.[20] These bones also display differences in osteoporosis and reactions to bisphosphonates. These reactions are coupled to differences in periosteal microcirculatory reactions. Thus, formation of periosteum over grafted bone may enhance osteogenesis in the grafted site.[20] Flapless implant placement, where the periosteum is left intact, has faster healing with less resorption than flapped procedures where the periosteum is lifted away from the bone.[21]

The vasculature of the skeletal system controls osteogenesis and hematopoiesis.[4] The vasculature in bone is also involved with tumor cell metastasis. Matrix and growth factors in the vascular system can promote metastatic growth and, conversely, promote dormancy or reactivate dormant tumor cells.[4]

Calcification is a normal physiological process in bone and, incidentally, also is a primary pathophysiological process in cardiac vasculature disease. There are expressions of calcification regulators during vascular calcification in bone and vasculature. Levels of gene expression of osteoprotegerin, receptor activator of NF-κB ligand (RANKL), osteopontin, matrix gla protein, bone sialoprotein, mothers against decapentaplegic homolog 6, and runt-related transcription factor 2 have been found in bone, aorta, and external iliac artery.[21,22] Expression of these cytokine regulators of calcification in bone occur early in the calcification and calcium/phosphate deposition process.[22] Water in bone is essential for structuring apatite crystals. Water pores in bone provide a conduit for nutrients and waste removal in bone metabolism and bone remodeling.[23] Bone has three types of porosity, the vasculature, the lacunocanalicular system and voids that occur in the collagen-apatite matrix.[23] There is interstitial nanoscopic flow in these porosities. Free water occurs inside hydroxyapatite pores that

are only few nanometers in diameter.[23] The mechanical load properties of bone matrix are related to nanoscopic water diffusion and osteocytic activity.[23] Nutrition and hydration may influence the ability of bone to resist loads.[14]

The outcomes of dental implants with thick keratinized facial tissue have equivocal results.[24] Nonetheless, a band of attached gingiva of immobile mucosa is important for facial bone retention.[24] An appropriate soft tissue covering may promote vascular maintenance.[24] Thin mucosa may not protect against facial marginal bone and soft tissue loss.[24] If gingival thickness does not influence marginal bone loss, which is apparently true for platform switched implants, the osseous thickness issue remains as a factor.[25] Thin osseous facial cortices around dental implants are prone to resorption, so a thick facial cortex may better resist mechanical loads and support bone remodeling.[26]

Bone graft material primarily maintains space for new bone formation.[27] After a tooth extraction, if the facial cortical plate is missing, the socket will undergo a significant volumetric reduction compared to when the facial plate is intact.[27] To minimize this shrinkage, the prudent clinician, to preserve the osseous ridge, would place a slow resorbing particulate graft material with a barrier membrane.[27] This may reduce the later osseous regenerative requirements if an implant is to be placed in the site.[27]

The socket shield technique is where a facial sliver of tooth root is allowed to remain on the facial aspect of a fresh extraction socket of an extraction site and immediate placement of an implant. The segment of tooth root apparently aids in maintaining facial cortical bone. Nonetheless how the long term osseous remodeling of this technique would be affected is yet to be seen.[28,29]

There are two types of osteogenesis.[30] First is static osteogenesis is characterized by stationary osteoblasts that need to be located about 28 microns from a capillary. Second is dynamic osteogenesis by monostratified laminae of unattached osteoblasts. There are no significant structural and ultrastructural differences between stationary and dynamic osteoblasts.[30] These are all secretory cells but unlike dynamic osteoblasts, static osteoblasts transform into osteocytes in the same place where they differentiate. Static osteogenesis builds the first trabecular bony framework. Then there is subsequent bone apposition by dynamic osteoblasts for support function in calcified trabeculae. Thus, static osteogenesis increases the bone external size, while dynamic osteogenesis performs bone densification by in-filling canal (haversian) spaces with osteons.[30]

Bone Remodeling

After a dental implant is placed there is an osseo-integrative healing process, similar to an osseous fracture. The relevance of blood supply for bone fracture healing has been published in the medical literature, but there is no information about the required vasculature for bone fracture healing.[31] Although osseous fracture treatment is a different procedure than dental implant surgery, the healing mechanism is the same, inflammation, angiogenesis, and osteogenesis.[31] Additionally, there is

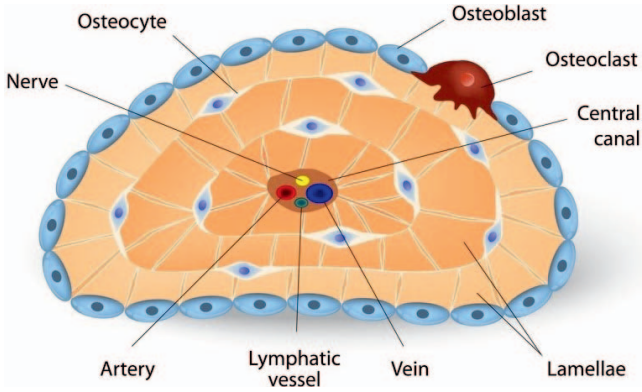

Figure 1. Schematic of bone haversian system.

no research that has established a physiologic basis for bone remodeling around dental implants.

Osteocytes are relatively inert cells that reside in mature bone and have a half-life of about 25 years.[32,33] They do not undergo mitosis and develop from osteoprogenitors in mesenchyme.[32,33] The osteocytes dwell in lacuna or bone pits (Figure 1). Osteocytes generally are stellate shaped and are about 5 microns by 20 microns in size.[33,34] The cellular cytoplasmic processes extend into canaliculi that network with other osteocytes. There are typically 40–60 of these cellular cytoplasmic dendritic processes emanating from each cell. These course through the canaliculi. Osteocyte are located 20–30 microns apart.[33,34]

There are gap junctions that allow an exchange of nutrients and discharge of metabolic waste products.[32,33] Osteocytes regulate bone mass and conduct remodeling of bone matrix. This is stimulated by a variety of mechanosensory chemical mechanisms.[32,33] Increased load causes increased metabolic activity. Old bone is eradicated by osteoclasts in a process called osteocytic osteolysis. New matrix is formed around the osteocyte with hydroxyapatite, calcium carbonate, and calcium phosphate thus increasing density.[32,33] The bone around dental implants under occlusal load undergoes apposition.[35]

Bone remodeling is lifelong. About 10% of bone is replaced annually.[36] Osteoclasts remove mature bone and new bone is deposited and ossified by osteoblasts. These processes are stimulated by the cytokine tissue growth factor beta and insulin growth factor.[36] Bone responds to the oral function of occlusal loading by increasing in density.[36] There are multiple cellular and molecular actions involved.[36] There is intimate cooperation between the two cell groups and other ancillary cell groups.[36] There is a multitude of cytokine signaling among the cell groups, such as immune cells.[36] Bone resorption and formation involve an intercellular signaling from a multitude of pathways and control mechanisms. Several hormones are involved, parathyroid, vitamin D, growth hormone, steroids, calcitonin, and various medullary cytokines.[36,37] Growth factors such as M-CSF, RANKL, VEGF, and the interleukin 6 family are importantly involved as well.[37] The osteoclasts-osteoblast-immune cell-cytokine complex is known as the basic multicellular unit (BMU). The duration for complete remodeling of the BMU is the bone remodeling period.[38]

Osteoblasts secrete osteoid, the unmineralized organic

precursor to mature bone.[38] Osteoid is made of collagen fibers and ground substance and calcifies into mature bone.[38,39] It comprises about 50% of bone volume and 40% of bone weight. The predominant fiber is type I collagen and comprises about 90% of osteoid. The ground substance is comprised of chondroitin sulfate and osteocalcin.[39]

A system of receptors, ligands, and metabolic enzymes called the endocannabinoid system is important in bone physiology. It stimulates bone formation by feedback circuit from sympathetic nerve terminals to osteoblasts. It enhances bone formation and decreases osteoclastic activity.[40]

An implant surface with an acid-etched surface embedded with calcium ions can reduce marginal bone remodeling around the dental implants.[41]

Capillary propagation encourages bone remodeling.[42] During bone formation the capillaries anastomose in the mineralized matrix canaliculi and enable remodeling and mineralization homeostasis.[43] The capillary network and the collagen matrix influences bone remodeling and the arrangement of collagen fibers during osteoid formation.[43] The collagen fibers around dental implants are transvers and not connected to the fixture itself.[44] In osteoporosis, there is a cellular change in the osteoblasts and osteoclasts during remodeling that occurs in intracortical and endosseous bone.[45] This results in loss of cortical thickness and increased porosity.

Bone cytosol, the liquid portion of cytoplasm, consists mostly of water. A 20% reduction of the intracellular aqueous portion can inhibit cellular activity whereas a 70% reduction causes a cessation of cellular activity. Thus, nutrition and hydration are important for bone remodeling and implant maintenance.[14]

Hydrogen sulfide (HS) is a metabolic signaling molecule that has multiple physiological and pathological functions.[46] These include vasodilation, neurotransmission, inflammation, hypoxia sensing, and bone remodeling. HS may also be involved in periodontal tissue remodeling during the orthodontic tooth movement by signaling increased periodontal ligament cell differentiation, tissue mineralization, bone formation, and collagen synthesis.[46] HS increases orthodontic tooth movement and decreases bone mineral density of alveolar bone. Importantly, HS facilitates alveolar bone remodeling by increasing osteoclast and osteogenesis activity.[46]

Physical energy influences bone metabolism. Low-intensity pulsed ultrasound has been found to promote the mandibular bone remodeling; however, the mechanism of action is not clear.[47]

There are a variety of mechanisms in cortical bone that preserve its integrity: extrinsic effects from mechanical loading and intrinsically through remodeling and renewal. In an osseous overload a crack will enlarge explosively unless the cortical bone is tough enough to resist the load. Toughness may allow a bone crack, but a fracture will not occur if there is a crack deflection and bridging, as in flapless implant placement where the overlying periosteum is intact and functional. During a greenstick fracture collagen interpolymeric fibers break but the main polymer chains do not break.[48] The main collagen polymers remain intact giving bone a certain toughness.[48]

There needs to be metabolic regulation of both mineral crystal size and the heterogeneity of the bone mineral and matrix phases. Additionally, adequate nutrition factors into osseous remodeling.[49] Thus, a patient with poor nutrition may experience inadequate bone remodeling for toughness and subsequent marginal bone loss under load.[49] This may be an important factor in late implant failures.

Bone remodeling may be influenced by medications such as bisphosphonates.[50] Bisphosphonates are known to inhibit osteoclast activity.[50] Bone can undergo necrosis caused by the inhibition of bone removal by osteoclastic activity. Implants placed in the posterior mandible may have a higher risk osteonecrosis of the jaws. The implant placement itself creates a risk but since the remodeling process is altered by the bisphosphonate there is a continuous ongoing risk for implant failure.[51]

Bevacizumab (Avastin, Genentech, South San Francisco, Calif) is a medication used for the treatment of colorectal, lung, kidney, cervical, and ovarian cancers.[52] Bevacizumab inhibits proliferation of new vasculature which may affect bone remodeling (Figure 5). The effects of these and other medications on peri-implant bone is not known. Nonetheless, the prudent clinician should be informed of the risk for bone loss and implant failure in those patients taking any medication with known physiologic effects on bone or vasculature metabolism.

Interleukin IL-1 polymorphism can cause an overproduction of collagenase and this may influence bone metabolism.[49] About 10% of patients can have this polymorphism.[53] It is responsible for at least 1 form of periodontitis.[53] There is a higher implant failure rate for patients with a history of periodontitis.[53] Most of these patients may have this polymorphism and these patients have a higher incidence of peri-implantitis.[53] Osseous cytokines, such as tissue necrosis factor α, are significantly increased in these patients with bone loss.[53] The peri-implant mechanism relative to this type of implant failure is not well understood, but the patient should be informed as to this increased failure rate.

There is no information as to any critical implant diameter that may inhibit or block remodeling by virtue of the implant's actual displacement. Nonetheless, there may be an actual physical blocking of cellular activity because of large implant size.

1.8-mm Thickness Issue

A dental implant does not provide progenitor cells, or a vascular supply as does a tooth periodontal ligament.[3] Thus peri-implant bone remodeling depends on vascular access for physiologic remodeling. This may mean an adequate bone thickness is needed to contain an adequate number of capillaries for transmission of cytokines and nutrition.

If a bone cell needs to be at most 28 microns from the nearest capillary for nutrients and an osteocyte is maximally 20 microns wide, then from the center of the osteocyte to the capillary is about 38 microns.[30] If the optimal minimal dimension for bone around dental implants is 1.8 mm then there are about 47 capillaries in a volume of bone section 1.8 mm, assuming dense cortical bone and a horizontally abreast cellular alignment.

The facial plate of bone abutting a dental implant would

have facial and implant-side cortices. Trabecular bone would be present if the facial and implant-side cortices of the facial plate of the implant site were thin, about 0.5 mm (totaling 1.0 mm with 0.8 mm of medullary cancellous bone interceding). However, generally, facial maxillary edentulous cortical thickness is about 1.7mm and mandibular edentulous facial bone thickness is about 1.8 mm.[54] Thus, 1.8 mm facial bone would most likely consist of completely cortical bone and no trabecular bone, except at the apical level where the bone would likely be thicker. Lingual edentulous cortical bone is generally thicker, about 2.3 mm and thus may contain trabecular bone especially at the apical level.[54]

The ability to resist off axial loads is also important for support of the implant under load. An overload would cause an osseous microfracture and potentially resorption. Any bone loss from resorption could manifest as marginal bone loss or fibrous formation around the implant fixture and subsequent exfoliation. In thick dense cortical bone an implant may be well able to resist up to 150 Newtons of direct horizontal off axial loading.[54,55] Bone repair and apposition would occur if the loads were in a range of 1500–6000 microstrain and any microstrain over 6000 would result in resorption.[56] A mechanical overload may result in resorption and implant failure.

Thus, it may be that the 1.8 mm dimension for long-term implant treatment success is physiologically based on load resistance requirements and subsequent remodeling of an adequate layer of cortical bone with a vascular supply for remodeling.

Teeth move in the bone up to 250 microns. There can be implant movement in bone up to 8 microns. The stress of loading creates pressure on the bone to stimulate osseous remodeling.[54] Remodeling requires angiogenesis and osteogenesis. Remodeling will occur if the bone is not stressed to fracture.

After osseous loading for about 8 weeks, trabecular and cortical bone density increases.[57] Nonetheless, since dental implant movement under load may be at most 8 microns, significant increased density may not be seen. Thus, the density and load capability at time of placement may not increase after prosthetic function.[57]

Age and Bone Remodeling

There is an increased remodeling rate in midlife in women and late in life in both sexes that results in a decline of periosteal apposition with a net loss of bone and increased fragility.[45,58]

Aging is associated with a functional attenuation of homeostasis, repair, osteopenia, and osteoporosis. Bone healing can be affected in alveolar bone. Aging can result in an overt osteoporotic phenotype in long bones, but only a subtle phenotype in alveolar bone. There is slower alveolar bone healing in extraction sites and in osteotomies. In aged patients, osteotomy sites have a wider layer of dying and dead osteocytes and there may be delayed osteoblast differentiation from osteoporotic phenotype.[7]

Exosomes are extracellular vesicles on a variety of cells that secret regulatory proteins into the blood for osteoblastic or osteoclastic regulation. These are activated during bone loss and age-related bone loss and remodeling failure.[59] The molecular function of these proteins in aged patients with low bone density is not well understood. Nonetheless, they suppress the integrin-mediated mechanosensation, activate osteoblastic cells, and initiate differentiation osteoclasts. They may be protective by facilitating adhesion of bone cells and suppressing oxidative stress.[59]

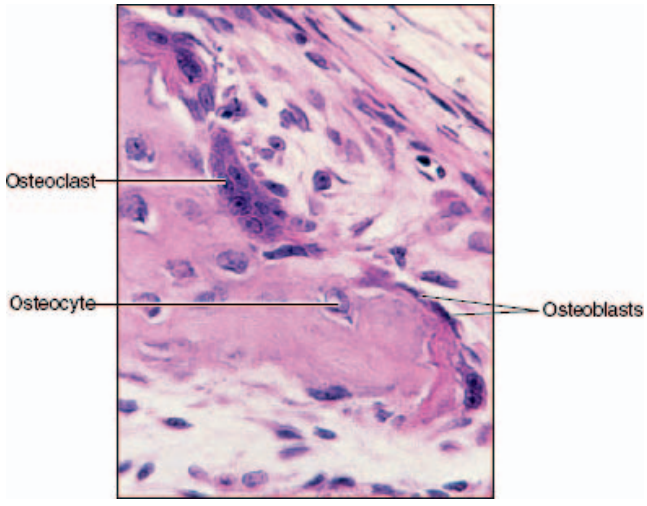

Figure 2. Photomicrograph of osseous cells.

Collagen is major component of the extracellular matrix. There is an equilibrium with degradation and formation of collagenous proteins for homeostasis. During tissue turnover, fragments of collagen polymer are released into the vascular system and may act as cytokines to stimulate remodeling. A lowered collagen turnover is affected by age.[60]

Aging is associated with decreased bone quality and altered mineral metabolism. During life, osteocytes undergo perilacunar maintenance and remodeling but changes in osteocyte lacunar morphology may affect bone structural integrity and mechanosensitivity. Osteocyte lacunae become smaller, more spherical, more spatially disorganized, and more sparsely populated with increased age which may result in bone strength.[61] Bone remodeling in older patients results in net bone loss.[62]

Osteoporosis of aging does not significantly cause adverse dental implant outcomes.[63,64]

Clinical Implications

If there needs to be a minimum of 1.8-mm bone thickness surrounding an implant for long-term success, then preoperative assessment is critical to meet this need. Bone grafting, either before or during implant placement, may be needed to insure this dimension is satisfied. Bone augmentation grafting, ridge-splitting, and osteo-distraction can be used to increase bone volume to attain the required circumferential bone thickness. Additionally, to maximize facial bone thickness, implants may be placed at an angle or parallel to the lingual bone contour (Figure 2).

There seems to be a natural propensity for the 2 mm dimension in oral anatomy in epithelial attachment (biologic width), osseous cortical thickness, and gingival thickness over

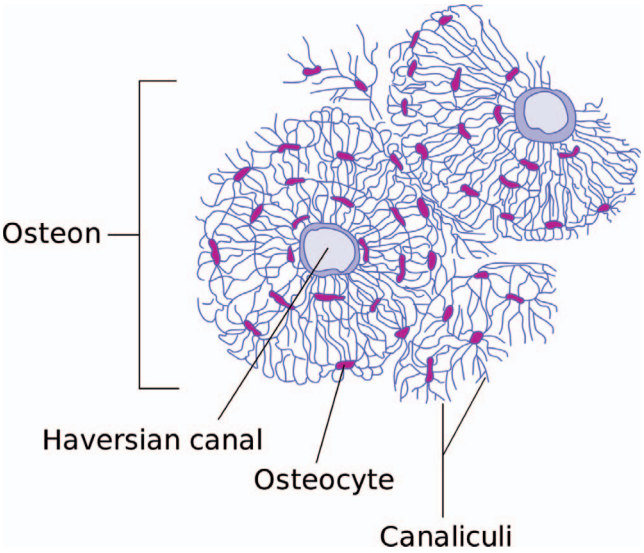

Figure 3. Schematic of an osteon.

edentulous ridges.[1,65–67] This may be due to a physiologic vascular requirement for maintaining cellular metabolism that needs to be encased in 2 mm of tissue volume.[67]

There may be a positive relationship between facial bone thickness and soft tissue thickness.[68] It remains to be seen if thick bone encourages thick soft tissue or thick soft tissue encourages bone thickness. Particulate allografting with barrier membrane coverage to augment facial bone defects may encourage osseous thickness.[69] However, grafted bone may not be adequate for a long term functional and esthetic outcome.

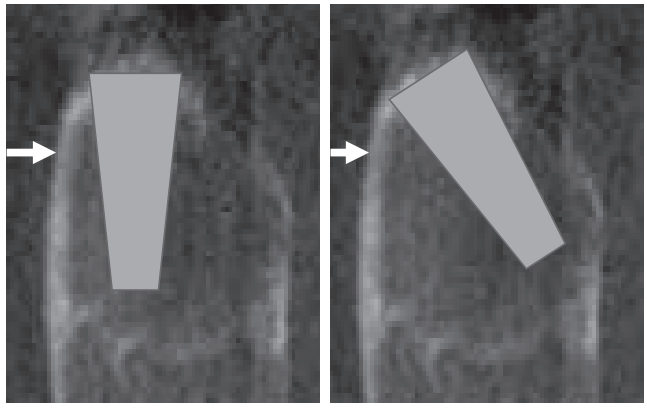

Figure 5. Implant placement parallel to the facial bone may not provide adequate bone thickness for long term success. An angled position parallel to the lingual or palatal bone my allow for a thicker facial bone.

Recent evidence suggests that gallium-aluminum-arsenide laser with a wavelength of 830 nm applied to bone graft sites may accelerate bone remodeling.[70]

Conclusions

Adequate bone remodeling may be the primary parameter for long term successful complication-free dental implant treatment. The remodeling ability of the implant-encasing bone to physiologically maintain itself may be of primary importance. A 1.8-mm osseous thickness around dental implants may be the

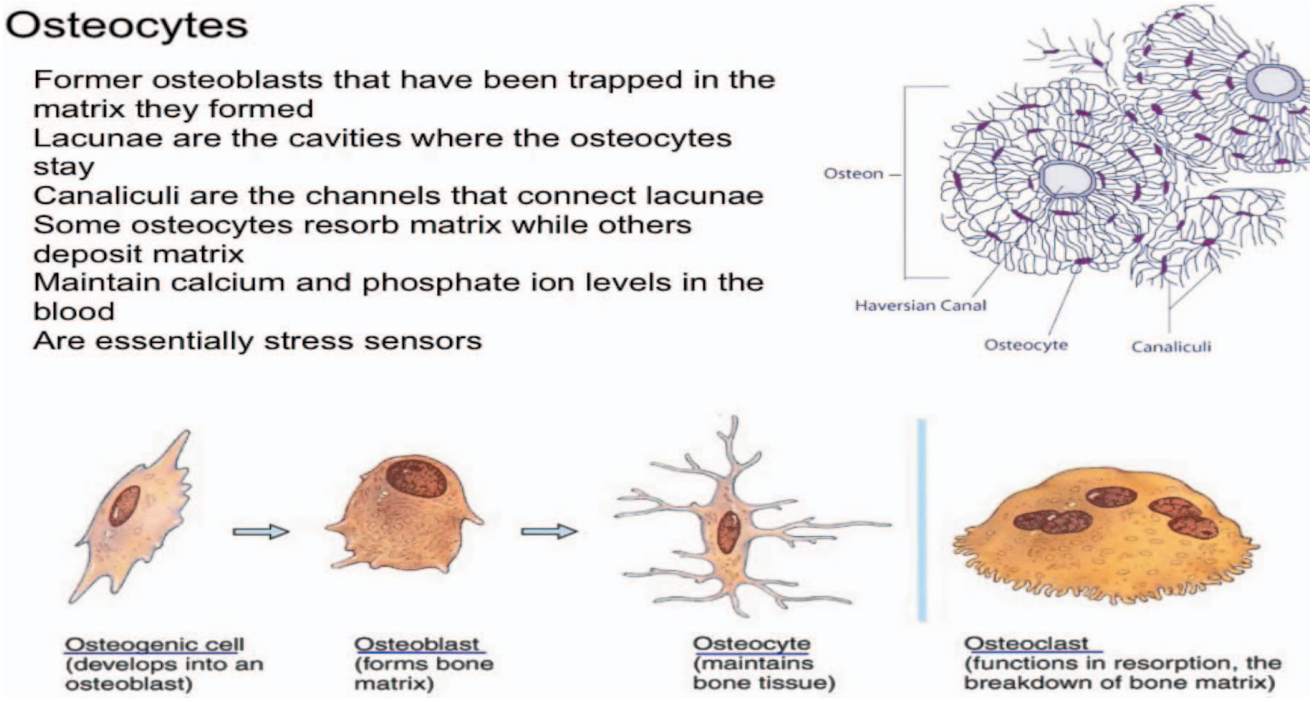

Figure 4. Lifestyle of osseous cells. Osteoblasts become trapped in the osseous matrix. Osteocytes maintain calcium and phosphorus levels for bone stability and strength. Osteocytes are stress sensors.

minimum thickness to enable an adequate vasculature for osteocyte nutrition and function.

Since a dental implant does not provide any progenitor cells or angiogenic or osteogenic factors, the surrounding bone may need to have this 1.8-mm thickness to accommodate a vasculature necessary for nutrients and cytokine delivery for appropriate remodeling. The 1.8-mm dimension may provide for a physiologically adequate mechanical load resistance and bone volume for an adequate vascular supply that enables remodeling.

However, there is no scientific evidence to illustrate the physiologic need for the 1.8-mm dimension. The dimension requirement is based on clinical outcome observations. Basic science research of bone survival around dental implants is needed to understand the osseous physiologic requirements for appropriate remodeling for successful dental implant function, survivability, and prosthetic outcomes.

Abbreviations

BMU: basic multicellular unit, an osteoclasts-osteoblast-immune cell-cytokine complex

HIF alpha: hypoxia factor alpha

HS: hydrogen sulfide

MMP: matrix metalloproteinases

PDL: periodontal ligament

RANKL: receptor activator of NF-κB ligand

VEGF: vascular endothelial growth factor

Wnt: a portmanteau of Wg and int, and stands for "wingless-related integration site"

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Note

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