

Bone remodeling and oral rehabilitation plans with mini-implants and mini-plates: a few analogies to facilitate understanding

Alberto CONSOLARO

Abstract: *Our bones renew themselves constantly and bone turnover allows a great capacity of adaptation to new physical and metabolic demands. Bones renewal is complete in 4 to 5 years in a young adult's skeleton, daily behaving as the poet once said "... I prefer being a moving metamorphosis!". In the simple orthodontic movement, in the placement of an osteointegration implant or in an orthodontic appliance, in dental movement with bone augmentation or change in form by anchorage in mini-implants, be it in the remote transmission of forces based on the anchorage offered by mini-plates, the osteocytes are involved, with their three-dimensional network, in bone design control, for they coordinate the activity of their commanded ones on the cortical and trabecular surfaces: the osteoblasts and clasts. Understanding bone biology and skeleton constant remodeling allows us to safely and accurately act in our patients' rehabilitation planning and, also, increases the possibilities of types of intervention to give aesthetics and function back to them.*

Keywords: *Bone remodeling. Mini-plates. Mini-implants. Dental implants. Orthodontic movement.*

Full professor, Universidade de São Paulo (USP), School of Dentistry, Bauru, São Paulo, Brazil. Professor of the postgraduate program, Universidade de São Paulo (USP), School of Dentistry, Ribeirão Preto, São Paulo, Brazil.

How to cite: Consolaro A. Bone remodeling and oral rehabilitation plans with mini-implants and mini-plates: a few analogies to facilitate understanding. *Dental Press Implantol.* 2015 July-Sept;9(3):15-37.
DOI: <http://dx.doi.org/10.14436/2358-2553.9.3.015-037.exp>

Submitted: August 11, 2015 - **Revised and accepted:** August 25, 2015

Contact address: Alberto Consolaro
E-mail: consolaro@uoi.com.br

» The author reports no commercial, proprietary or financial interest in the products or companies described in this article.

An adult human skeleton has 206 bones that are formatted with impeccable design to meet functional demands and absorb, create or transfer forces in

the body movements. Muscles and tendons are inserted in the bones to take us everywhere (Fig 1). Let us observe, in the human skeleton, the shape of



Figure 1. Muscles and tendons exert loads over the mineralized bone structures that, in order to meet the increased functional demand, thicken their cortical trabeculae, as long as there are stimuli that characterize bone remodeling dynamism.

the bones, how they are articulated amongst themselves or related to other parts of the body: bones have no corners, cutting edges, pointed structures – everything must be regular, flat and rounded. If bones were not rounded, they would harm soft tissues, to which they relate in peripheral areas. Those who have had fractures and spurs know how much it hurts. Bones must be rounded and smooth. Surgeries that result in sharp edges or straight angles in the operated cavities and areas, will get rounded bones in a few weeks, for they bones have this natural and adaptive way of relating with tendons, muscles and other soft tissues.

What is vital is guarded by the bones: the brain, the heart and lungs, apart from the marrow, which produces the liquid that provides us life and defense – the blood. Bones, as wonderful anatomic pieces, have several variables in their functional composition, such as cartilages, tendons and articulations. By analogy or comparison, bones come gift-wrapped because there is, on their external surface, a resistant and protective membrane, which composes the periosteum, a fibrous connective tissue with abundantly cellularized and vascularized areas.

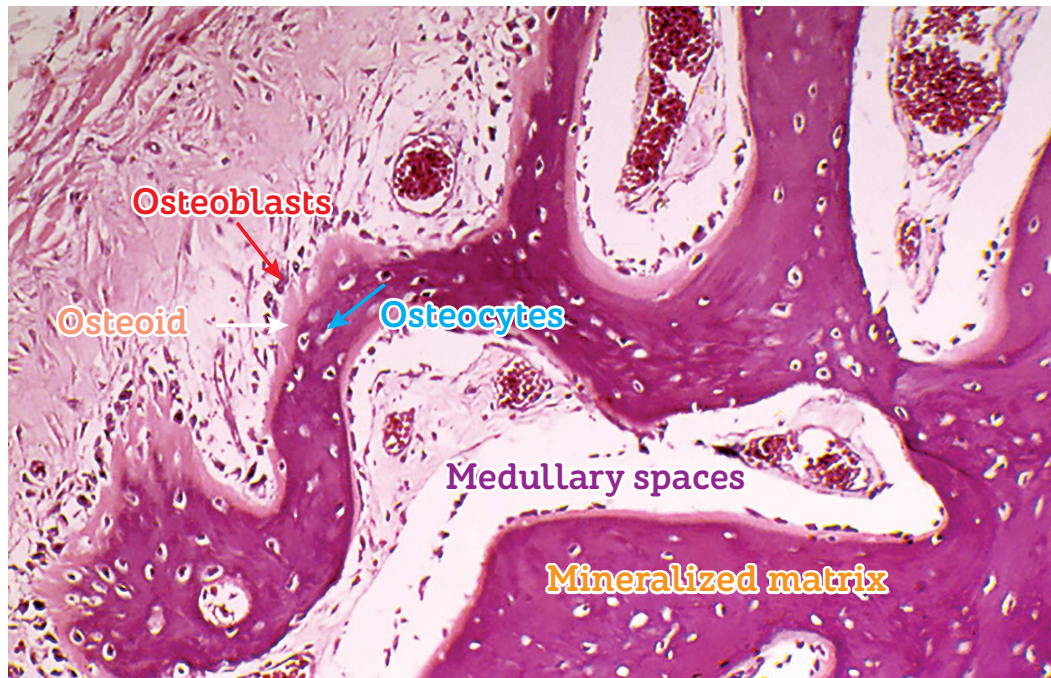
The first one to suggest that bone shape was directly related to applied forces was Galileo Galilei, in 1638. In

his turn, Julius Wolff, in 1892, was eloquent when proposing that bones adapt or respond to forces.³

Athletes' bones tend to be bigger, voluminous and more mineralized, because, in a dynamic way, their structure and design adapt to the function. As for sedentary individuals, bones get thin, less dense and mineralized. We were initially taught that bones were dry, hard and resistant pieces in the constitution of a skeleton – like those exposed at anatomy departments and museums –, but they are, in fact, malleable, moldable, adaptable and they try to meet all the functional demands we come up with.

Bones are 'humid' and they present surfaces with thin layers of matrix still not mineralized (Figs 2 and 3); they are abundant in water, minerals and many proteins and growth factors – which are pretty much 'hidden' or composed with minerals.

To sum up, our skeleton is an excellent fellow, who is understanding and malleable, who removes their marks and 'sorrows' as they remodel themselves. Brain and heart are different, for they are definitive in life. They cannot be remodeled and keep permanent marks whenever they get injured. That must be the reason why, in folk wisdom, they call stubborn people hardheaded, and say that love is forever!



18

Figure 2. Bone structures emphasizing, in the trabeculae, the presence of a thin layer of osteoid, osteoblasts and, especially, osteocytes – which, via canaliculi, with their numerous cytoplasmic extensions, constitute a cell-to-cell communication network, through mediators, and with trabecular and cortical surfaces, actively influencing bone remodeling (HE, 25X).

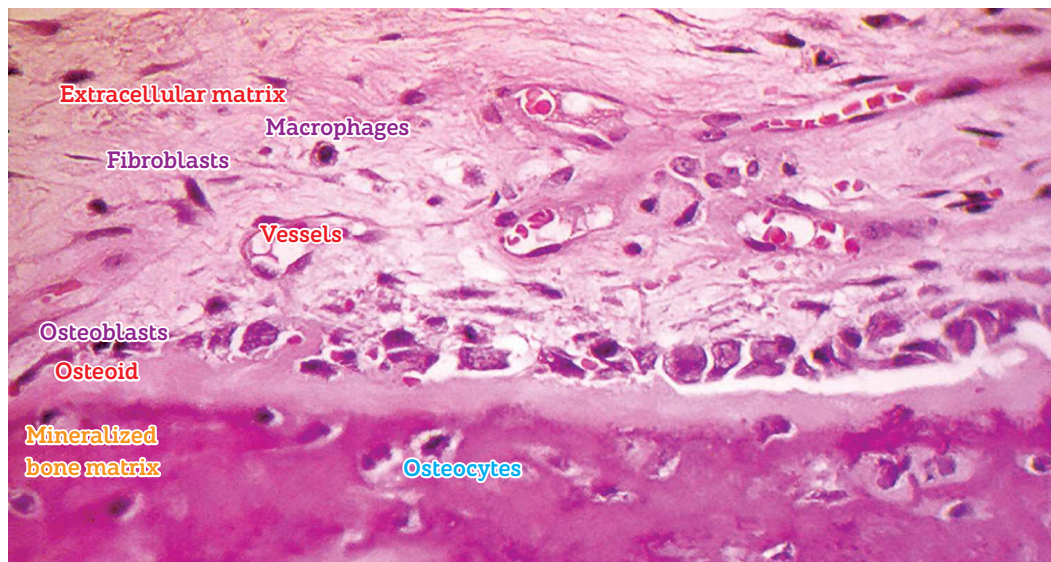


Figure 3. On the surfaces, osteoblasts deposit new bone layers as they are stimulated by the mediators released by local adjacent cells, coming through systemic via or even by osteocytes via a canaliculi networked structure (HE, 25X).

HOW CAN BONES DO THAT?

A young adult's skeleton renews itself, entirely, every 4 to 5 years, but it continues in transformation until the end of life: considering a 70-year old man, there would be, on average, seven complete renewals throughout life.

The mineralized bone structure, be it the cortical or the trabecular one, has millions of minuscule gaps which are the size of a cell and, three-dimensionally, it becomes spider-shaped, with tens

of ramifications. Such empty gaps are named osteoblasts and their function is accommodating the osteocytes (Fig 4).

Osteocytes, in their turn, have from 20 to 50 cytoplasmic extensions, randomly distributed in the hard part of the bone, by means of true bone tubules or canaliculi (Fig 5). Each of these cells get connected to other 20 to 30 cells. Imagine a lot of spiders holding 'hands', or making contact, cell to cell, with other 20 to 30 spiders (Fig 6).

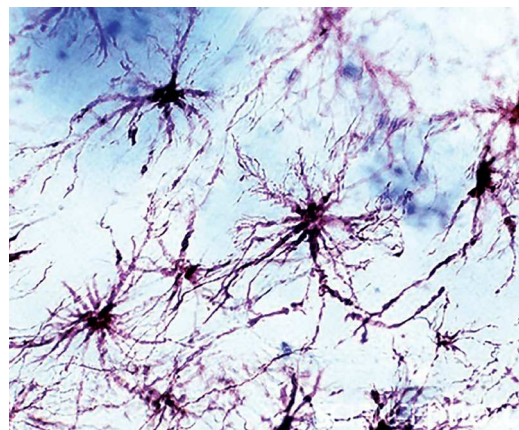
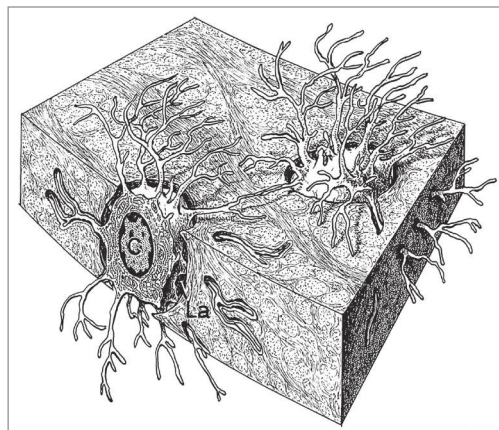
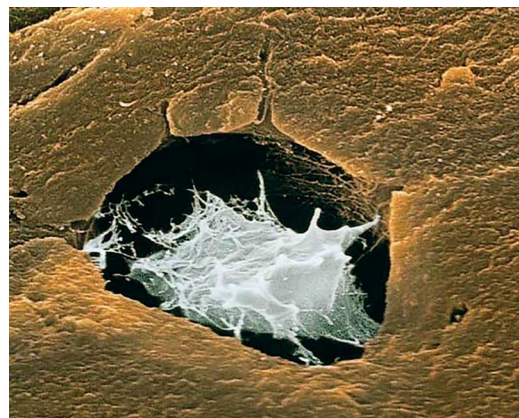
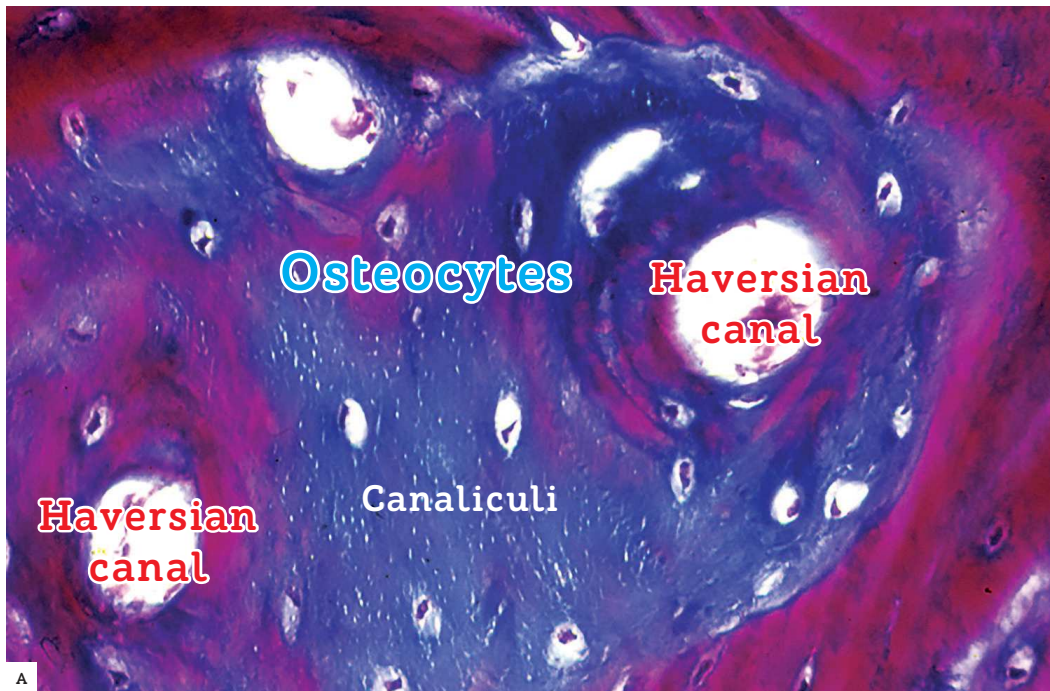


Figure 4. Osteocytes in their distinctive shape, with tens of extensions forming an efficient communication network inside the mineralized bone matrix. The lacunae where osteocytes are located are called osteoplasts.



20



Figure 5. Osteocytes in the mineralized matrix, revealing numerous cytoplasmic extensions inside the intercommunicating canaliculi (HE; **A** = 25X, **B** = 40X).

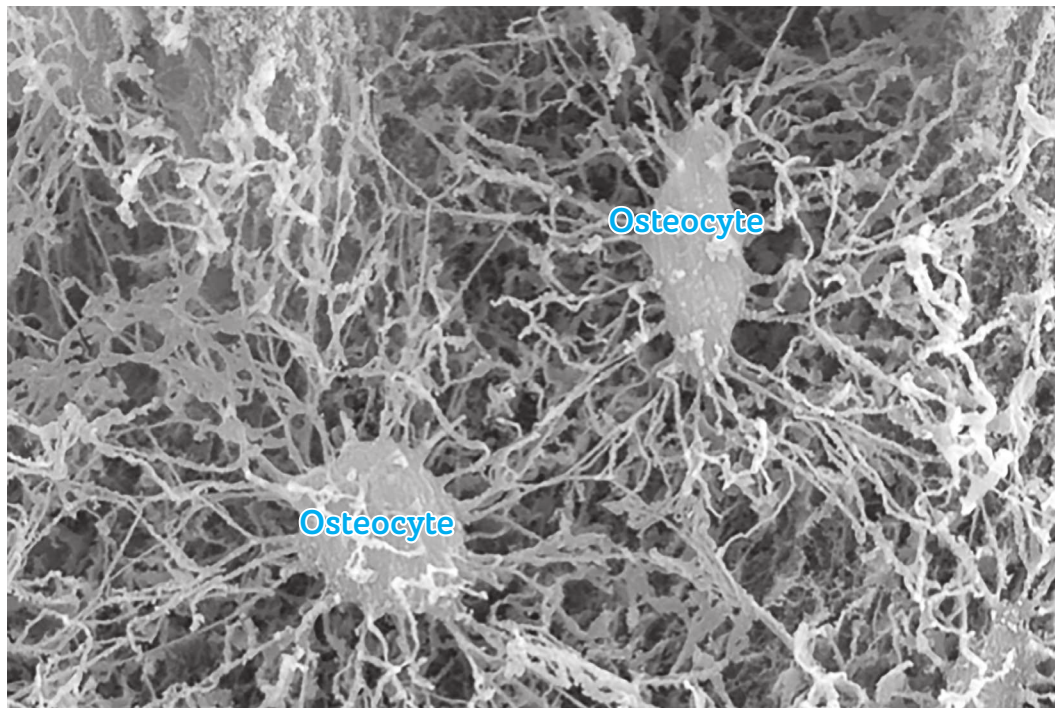


Figure 6. Resin was placed at the osteocytes' place and, when polymerized, took the form of their lacunae and canaliculi - visible under a scanning electronic microscope, after the acids totally removed the mineralized part. The mentioned network helps to understand the sensitivity of the osteocytes to capture bone deformations, even the most discreet ones.

Osteocytes form an intercommunicating network inside the bone mineralized structure. Whenever force is applied during movements, this network is deformed, for it stretches or compresses, in order to capture the shape alteration, and this new situation is immediately communicated or transferred to this osteocytes network.

One of the most efficient ways of message transmission, besides the cell-to-cell contact, is the release of substances (chemical products or mediators) to

the internal or external surfaces of the cells with, for example, the following message, biochemically modified:

“ – Change your current shape; it is necessary or required to adapt to the situation; improve your design.”

Bone design adapts to use every day. It is a real show or demonstration of flexibility, adaptability and willingness to serve a collective body; an exemplary predominance of collectivity over individuality.

THE COMPONENTS

The cells on the bone surface are like workers: the osteoclasts 'bring down' or unmake walls or their covering (Figs 7 and 8), the osteoblasts erect and reinforce pillars and structures (Figs 3 and 9). Bone surface cells seem more and more commanded by the osteocytes. A short time ago, researchers believed that osteocytes were isolated cells on the corner of the mineralized matrix: they were wrong!

If one wishes to reinforce their skeleton, muscles and tendons must be stretched and deformed, in order to deform and stimulate the osteocytes

network, making them adapt to the design expected by whoever planned this functional demand!

If discouraged or demotivated, osteocytes will not have stimuli to renew their skeleton and adapt it to a certain life style. Bones will get increasingly fragile, less hard and not sufficiently resistant! Like it or not, at least our skeleton is a moving metamorphosis and this constant process is referred to as remodeling or bone turnover. Whereas in children and adolescents bones are modeled and remodeled at the same time in order to assist development and growth, in adults bones keep remodeling continuously. In an adult's skeleton,

22



Figure 7. Mononuclear cells flow towards an area of bone surface exposed by osteoblasts' local movement from the action of mediators and changes in the local conditions - such as pH reduction - due to cellular stress. It is possible to notice discreet Howship lacunae, with emphasis being placed on the osteocytes in the mineralized matrix (HE, 25X).



Figure 8. Two Bone Multicellular Units (BMUs) in Howship lacunae, with emphasis on the osteocytes, osteoblasts and macrophages as important components.

there are 1 to 2 million microscopic bone resorption points.

Bone remodeling consists of four stages:

1. Activation: with precursor cells responding to physical and hormonal signs in a certain area of the bone surface, organizing as clasts.
2. Resorption: the activated clasts open small and shallow cavities or Howship lacunae of 60µm, between 7 and 15 days.
3. Reversal: once resorption is complete, the clasts cease by stimuli removal. In 7 to 14 days, a new fine matrix line is deposited, with collagen fibers in contrast to the surface of well-organized and lamellar collagen. Such line demarcates the edge of the stopped resorption with the one of the newly formed bone.
4. Formation: newly required pre-osteoblasts start the deposition of the new matrix. The period between the deposit of the not mineralized immature bone and of the mature bone with the previous one is 21 days. The time it takes for filling and normalization is, in general, 8 to 12 weeks.

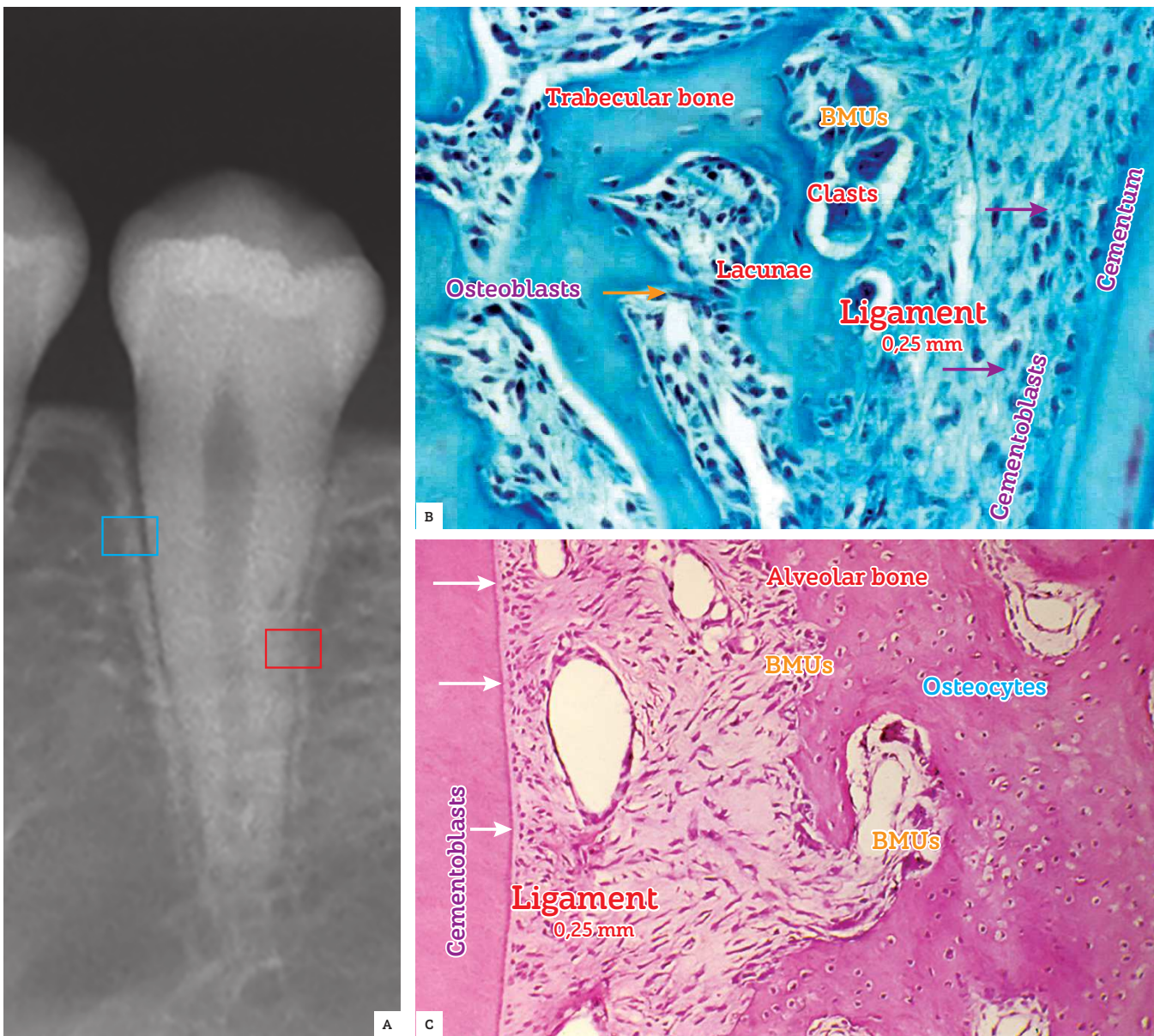


Figure 9. On the bone surface of the periodontal ligament, Bone Multicellular Units (BMUs), as well as in the entire organism, constantly renew bone structures, and that provides a minimal natural dental movement throughout life. The cementoblasts on the root surface, without membrane receptors for the bone mediators, do not take part in bone remodeling (**B** = Mallory, 25X, **C** = HE, 25X).

WHY IS IT LIKE THAT?

In the harmonious organization and functioning of the organism, at the several levels of magnitude, calcium ion is essential for life: it takes part, directly or indirectly, in basically all the biological processes inherent to human life. In general terms, 99% of the calcium in our body are in the bones, and only 1% is used in general metabolism. Calcium, in our organism, is linked to proteins, and it easily disconnects from them whenever it is induced or necessary.

Calcium availability to cells and tissues must be assured no matter what happens, for it is related to life maintenance. In time intervals between meals, calcium for the serum level comes mainly from the skeleton, which acts as an active mineral supply. Matrix and minerals apposition by the odontoblasts and osteocytes, and bone resorption — for focal disassembling and for ions release to the blood by the clasts — assist, primarily, the maintenance of the serum levels of ions, especially calcium.

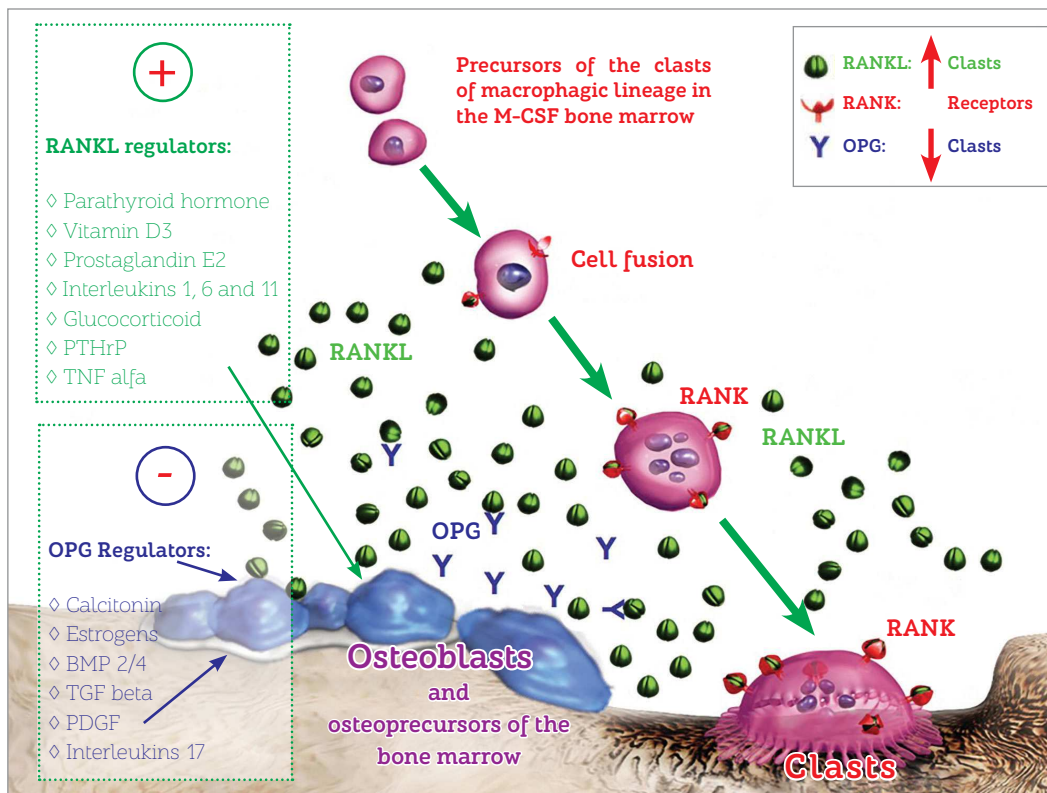
The cells of four parathyroids, placed beside the thyroid, in the trachea, capture the reduction in level of calcium in the blood and release, directly to the blood, parathormone, the main inductor mediator of clastic activity. By distributing and quickly interacting with the other body cells, via membrane receptors — especially in the neoblasts that command the clasts —, parathormone

determines resorption activities for the removal of ions from the bones and for reestablishing normality of serum levels (Figs 10 and 11).

Vitamin D3 plays a very important role in this process, for it increases the amount of calcium resorption in the intestinal mucosa, while working as an important stimulator of the osteoclastogenesis, that is, new clasts maturation.

This resorption activity may, in a few minutes or hours, considerably raise calcium serum level, and that can be detected by C cells or parafollicular cells of the thyroid, which are calcitonin producers. The release of calcitonin into the bloodstream inhibits bone resorption in the other cells of the skeleton and contributes to the prevalence of bone apposition phenomena, placing mineral ions in the bone matrix. Estrogens also contribute to this reversal from resorption to predominant bone apposition (Figs 10 and 11).

It is due to the dynamics of constant apposition and resorption for maintenance and ionic balance of the body that bones have this great adaptive capacity to functional demands. These are the reasons why the analysis of bone structure and composition makes it possible to determine the life style and the functional muscular and skeletal patterns of an individual in recent times, since bones renew themselves and adapt to each stage of life.



26

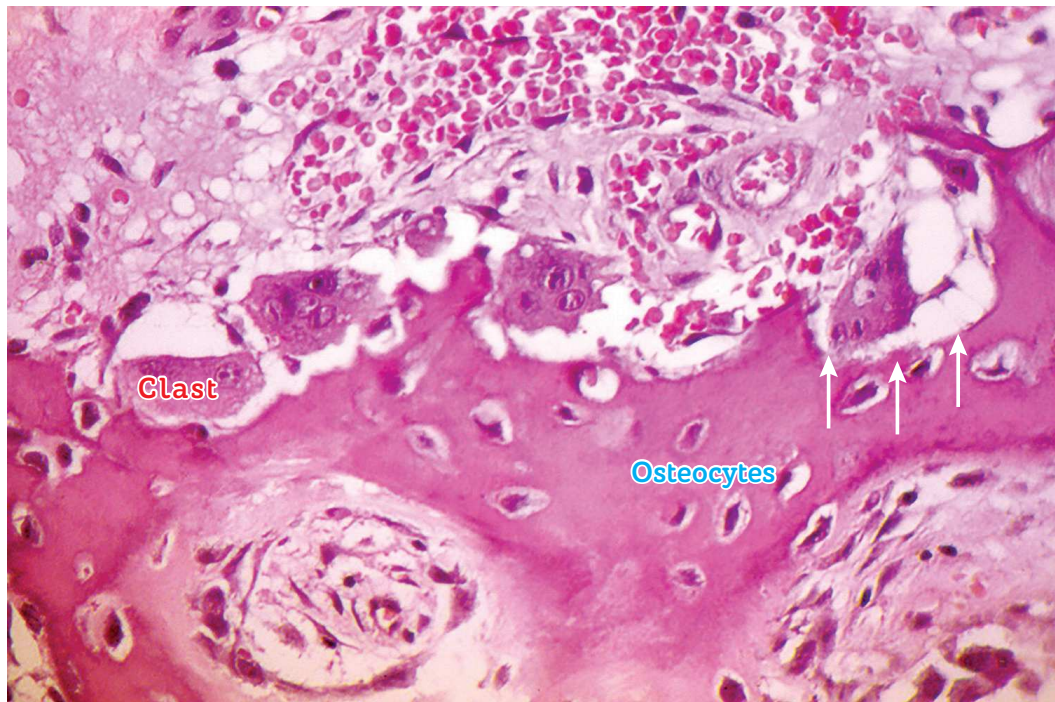
Figure 10. In bone environment, there are intrinsic mediators of BMUs, which accelerate or inhibit bone remodeling according to stimuli that are extrinsic to them, represented by local and systemic bone remodeling mediators. Some of these mediators stimulate osteoblasts, osteocytes and other local cells to release RANKL, which stimulates clastic activity; while others stimulate the production of osteoprotegerin, or OPG, which reduces RANKL effect, coupling with their molecules and hindering their interaction in the membrane clastic receptors, or RANKL.

**ANCHORAGE:
LIMITS AND POSSIBILITIES**

In the maxilla and in the mandible (Fig 9), as in any bone, the process and the general design of the anatomic piece (such as volume, shape and minor details) are determined by a functional demand. The alveolar process is used to maintain and support the teeth; when they are missing, there is no reason for this bone protuberance to exist, and it tends to atrophy.

When it comes to toothless people, it may end up disappearing.

The use of osseointegrated implants right after tooth loss contributes for at least mitigating this alveolar bone loss. Masticatory load in osseointegrated implants is transmitted to adjacent bone tissues and, thus, their structure tends to remain, since there is a constant functional demand.



27

Figure 11. Several BMUs with their clasts in the respective Howship lacunas, and many networked osteocytes related to the resorption phenomena - probably releasing mediators and directly interfering in local bone remodeling (HE, 25X).

Teeth move normally throughout life and follow the vectors that are initially provided by modeling and, in adult life, by remodeling. The balance of muscular forces and other soft tissues over the teeth, plus masticatory load associated to alveolar bone support, determine greater or milder stability of the tooth in the dental arch.

Induced dental movement initially promotes directions to the natural movements, and very often induces intentionally directed forces to make bone modeling happen more quickly on one of the radicular faces, so that a new dental position is reached.

On the compression side, the cells of the periodontal ligament release many cellular stress mediators, which stimulate bone cells to reabsorb faster on the periodontal face of the alveolar bone (Fig 13 and 14). In the extended or stretched areas of the periodontal ligament, bone apposition phenomena prevail, for the stress mediators appear in lower concentration and, in such conditions, they induce apposition phenomena.

Induced dental movement represents a biological phenomenon directed to therapeutic and esthetic purposes, but it has limits. Such limits are related to the

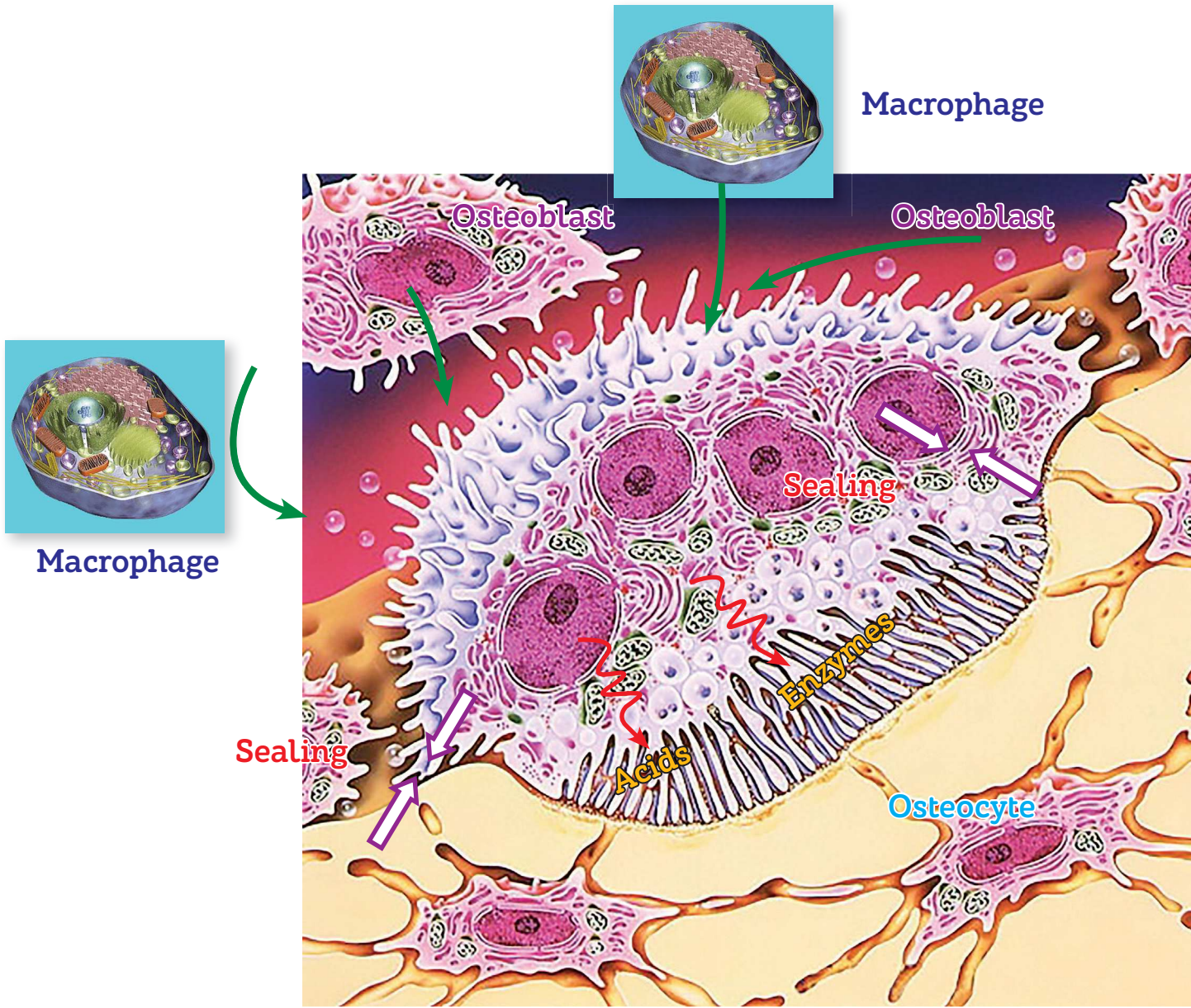


Figure 12. A demonstrative scheme of the function and organization of a BMU. BMU managing is done by osteoblasts and, in second place, by macrophages through RANKL-type mediators. Acids and enzymes are released on the active edge or on the brush-edge. They are contained at the same place by an efficient sealing zone formed by the molecular fusion between proteins and bone membranes. It is worth highlighting the relation with osteocytes.

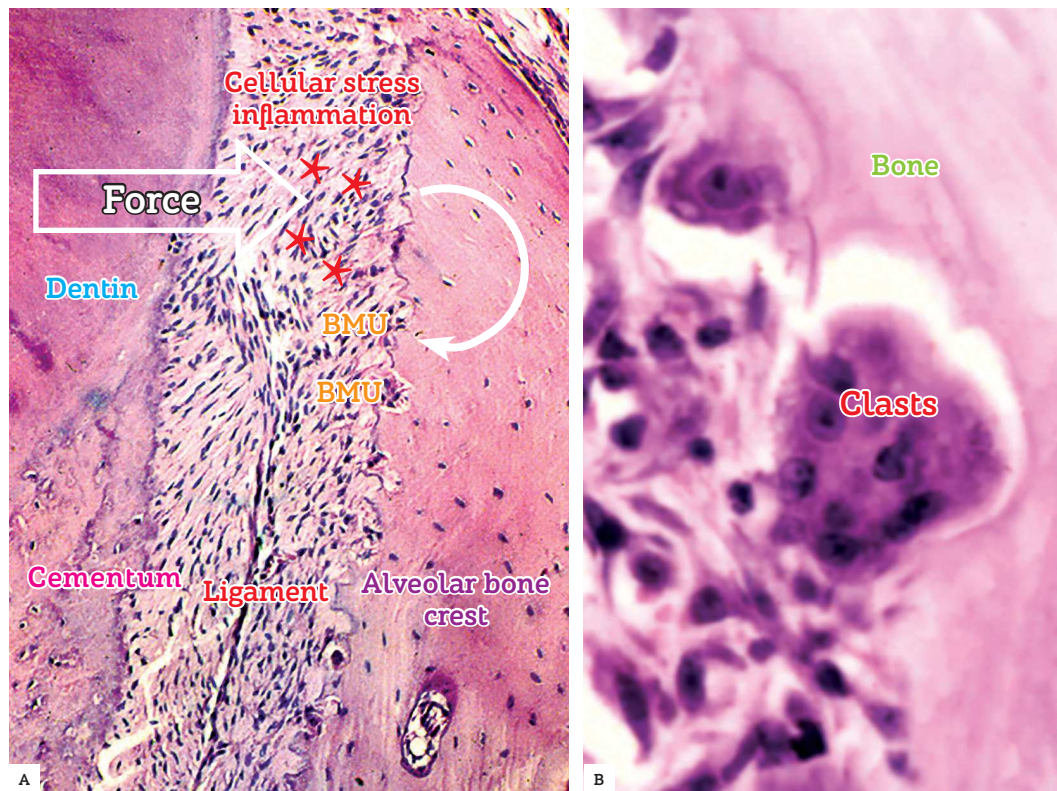


Figure 13. On the periodontal surface of the bone crest, in a tooth subjected to induced dental movement, it is possible to observe areas without osteoblasts and, peripherally, with stress and ligament inflammation (stars). After that, BMUs settle and start the bone resorption process. In **B**, we can notice BMU components working with peripherally located clasts and mononuclear cells representing osteoblasts and macrophages (**A** and **B** = HE, 10 and 40X, respectively).

speed at which it would happen and to its extreme and harmonious extension in the set of teeth. In those patients, which these time and extension limits exist for the induced dental movement, orthodontists in general choose the orthognathic surgery.

Osseointegrated implants, due to the absence of the periodontal ligament

and without natural dental movement show much greater stability. The use of osseointegrated implants in rehabilitation planning, as an anchorage point in mechanics with mini-plates, represents a perfectly feasible option through a biological and physical point of view. Anchorage forces, even the greatest ones, are much lower than those generated during daily masticatory loads.

Cellular hemostasia

Basic functions:

- ◇ Breathing
- ◇ Protein synthesis
- ◇ Ionic balance
- ◇ Cell proliferation

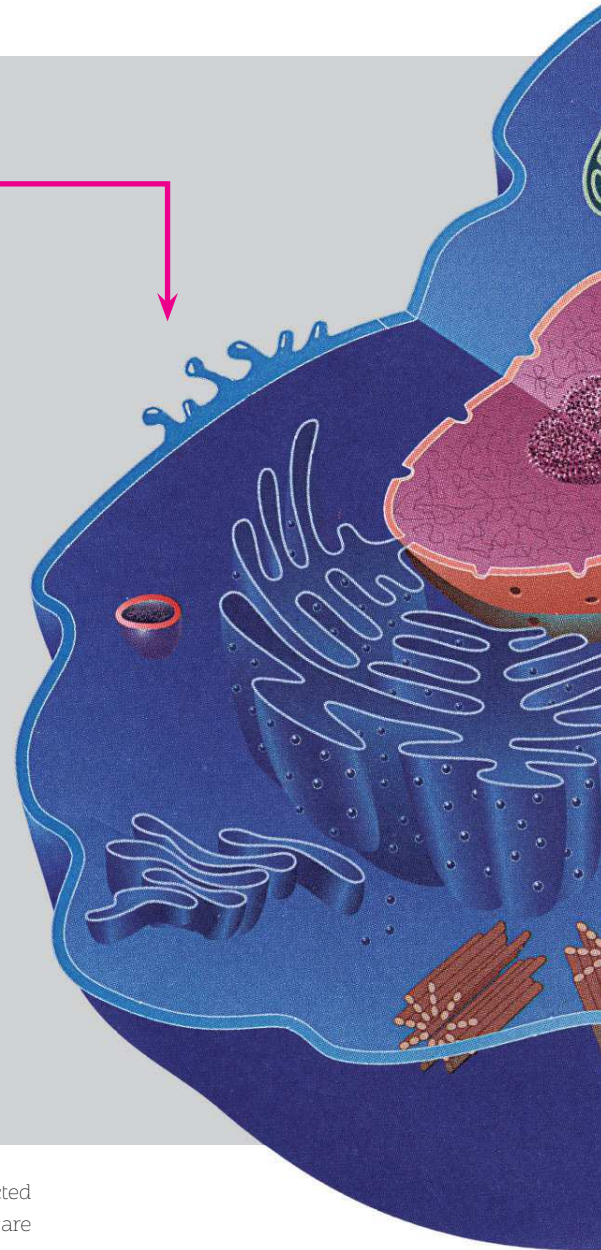


Figure 14. More common stimuli and aggressors in bones and teeth subjected to forces action, which will be responsible for releasing the mediators that are fundamental to the remodeling biological process.

Stimuli / Aggressions

- ◇ Hypoxia
- ◇ Compression / mechanic deformation
- ◇ Hiperactivity

Cellular stress

Greater release of:

- ◇ Kitocins
- ◇ Growth factors
- ◇ AA products
- ◇ Other mediators

**ANCORAGES
HAVE BROADENED THE POSSIBILITIES OF REHABILITATION TREATMENTS
or
Anchorage have broadened Orthodontics' role in oral rehabilitation**

One of the factors that used to increase orthodontic treatment time and reduce the possibilities of certain types and extensions of dental movement was lack of anchorage. Mini-implants, at first, were incorporated as a proposal of absolute anchorage; however, later on, it was verified that they had limits when supporting very heavy loads, that is, they were not that absolute. They help solving many cases, mostly cases in which additional anchorage is required apart from the one offered by the teeth, but they also have limits.

Some cases of extensive movements, almost unthinkable for many decades, required a real reanatomization of the maxilla and the mandible. There were cases that could not be solved even with the anchorage provided by mini-implants. There were also some cases in which, quite often, not even an orthognathic surgery was the ideal solution, in all aspects.

More complex cases, regarding esthetics and function, require a joint action by Orthodontics with many other clinical specialties for the patient's complete rehabilitation. In those cases, the risk of leaving a lot to be desired in the final results is high and technical, physiological and anatomic limitations may be pointed as the reason for that.

The use of mini-plates has come to offer a greater opportunity of anchorage and new solutions of orthodontic-mechanical and maxillary-orthopedic nature, for they allow higher intensity and greater reach of these forces in almost all the extension of the maxilla and the mandible.

Under no perspective does the orthodontic or orthopedic use of mini-plates replace or involve planning changes in orthognathic surgeries. It solely involves enhancing the chances of success in the most complex cases, in which all therapeutic resources must be synergistically used. Not in all cases are mini-plates indicated, nor would they solve the problem; the same applies to orthognathic surgery.

A sculptor, just like a surgeon, in front of ivory or wood,sculpts the way it was determined by his planning, and makes his art come true from something that appeared to him! Moving the teeth and modifying the bone shape based on anchorages and active forces, thank to bone remodeling dynamics may be compared to the art of a potter. By making the most of the malleability of the material, fingers, hands and instruments —just like the forces in the bone— lead to new forms and details to build the final design. There are two

ways of achieving the final goal: beauty and function of art in human imagination. To a surgeon and an orthodontist, what is beautiful is human kind!

Understanding how mini-plates act in orthodontic and orthopedic planning requires mastering bone biology concepts in the context of bone remodeling, previously mentioned in this paper. In order to make that understanding faster and more diligent, applying analogies may be of great help.

BONE SHAPE: OSTEOCYTES AND ANALOGIES ABOUT MINI-IMPLANTS AND MINI-PLATES

A bone has more than 50% of organic components, including water. That allows us to say that bones have great deformability, and that they are malleable, able to deform in face of compression or stretching. The osteocytes intercellular network immediately alters (Fig 4 to 6) and changes the three-dimensional pattern of the cytoskeleton (Fig 15) of each osteocyte.

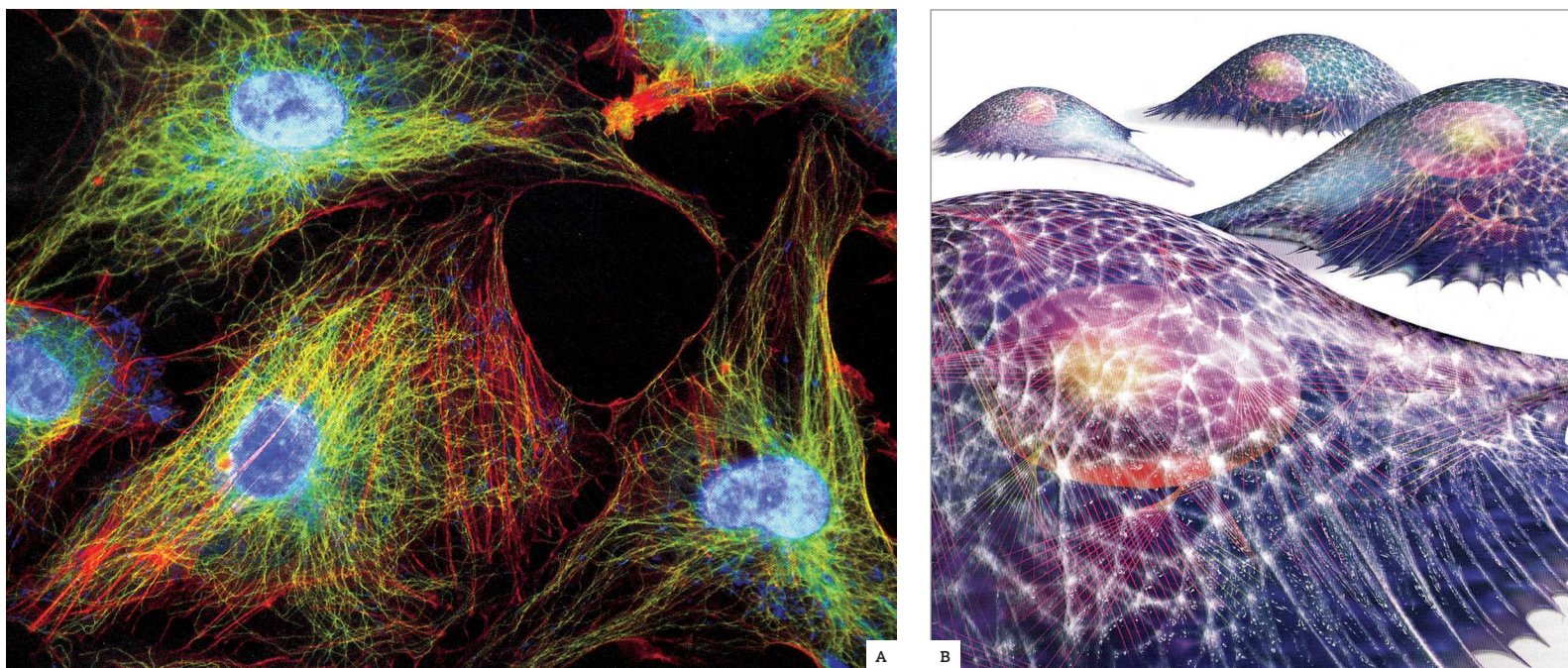


Figure 15. Cells with cytoskeleton proteins evidenced by immunofluorescence (A) and schematically, in B. The proteins marked in red and green reveal the structural support that keeps the cell shape stable and provides it with mobility, whenever it is necessary. Cytoskeleton proteins internally connect with integrins in the cellular membrane and with the nuclear membrane in the most central portion, marked in blue.

Just like a three-dimensional network of osteocytes, inside each of them their shape is determined by another network, now of interlinked proteins, known as cytoskeleton (Fig 15). Balance loss in the stable shape of this network leads to cellular stress, characterized by an increase in metabolism and chemical mediators' secretion, and the surrounding environment will induce phenomena, no matter which ones, to reestablish such physical and metabolic stability (Fig 14).

In other words, bone deformation increases metabolism and message exchange among osteocytes by the release of mediators, and between them and the osteoblasts and clasts on the trabecular and cortical surfaces (Figs 11, 12 and 13).

Bone deformation by compression and stress raises the number of Rankl mediators and sclerotin by the osteocytes, increasing resorption mechanisms by the clasts (Fig 16). On the other hand, reduced functional demand with light stimuli causes reduction in the release of these mediators in the region by the osteocytes, and bone deformation phenomena will prevail by the osteoblasts (Fig 16).

Osteocytes biochemically command the activity of bone cells on structural surfaces, indicating where to reabsorb and place bone layers to adequate the shape, design or anatomic profile of the bone, adjusting it to the functional demands that the captured deformity represents (Fig 17).

34

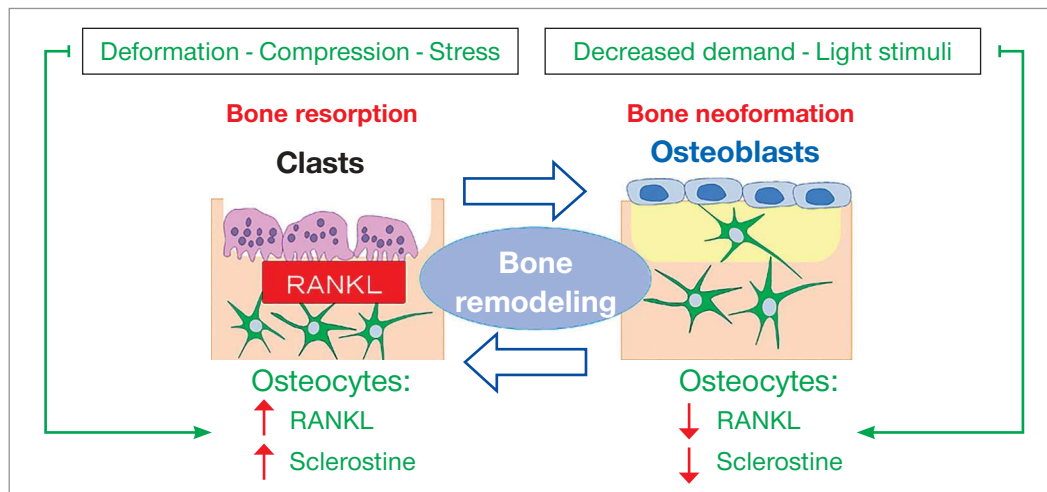


Figure 16. Demonstrative scheme on how osteocytes actively participate in bone remodeling as mechanotransducers. Deformation of the osteocytes network induces mechanical stress, with increase in the local RANKL level, with greater number of active clasts and greater sclerotin release on the part of osteocytes. Once the response has been adjusted to the stimulus, there is a reduction in both number and activity of the clasts, and also a reduction in sclerotin by the osteocytes.

The role of the osteocytes in bone biology was ignored for decades —it was even despised. In forefront analysis of studies on bone homeostasis, it is possible to notice that the role played by osteocytes became a key element for understanding metabolic bone diseases and in the processes of repairing and bone remodeling. Osteocytes represent 90 to 95% of bone cells.

In **induced dental movement**, the applied forces minimally deform the teeth themselves, but especially, and a little

more, the alveolar processes externally (Fig 17) and their maxillary bone basis. In the orthodontic movement, not only is the internal part of the alveolus of the involved teeth modified, but also its size, thickness and shape of the trabeculae of the adjacent bone and the external surface of the corresponding cortical, being established or modified in bone layers juxtaposed by the subjacent periosteum. The architect osteocytes, from their osteoblasts, command the workers that are juxtaposed on the internal and external bone surfaces (Fig 17).

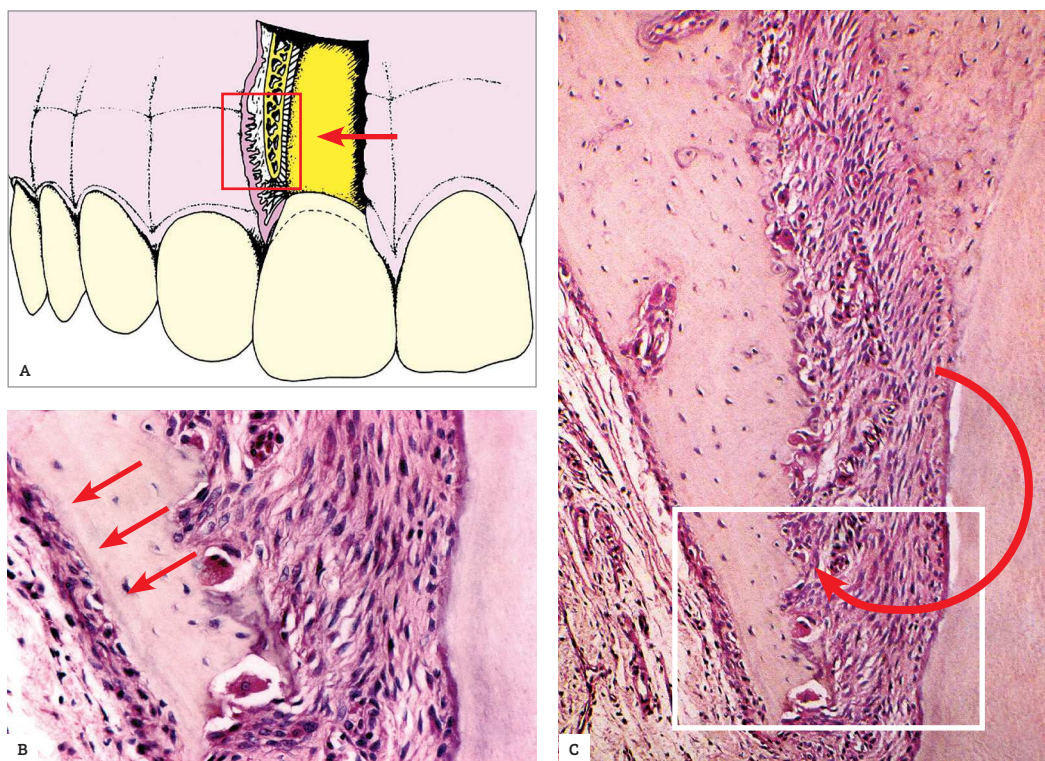


Figure 17. The periosteum, in the area that corresponds to the induced dental movement (larger arrow), receives mechanical stimuli, and also stimuli by the mediators released by the osteocytes, which are deformed and act as mechanotransducers, reacting with the deposition of new bone layers in the cortical surface (smaller arrows), changing shape, volume and size of the maxillaries, besides teeth position (HE; 25X).

The **application of an osseointegrated implant** does not mean just a motionless and inactive threaded pillar in the bone structure. Its function, via received load, influences its entire periphery and, from afar, influences the adjacent trabecular bone and the corresponding adjacent cortical. Constant remodeling occurs directly – and indirectly – around it and the bone is readjusted at every moment to its necessary functions and demands.

Orthopedic appliances, its plates, shields and other pieces of equipment base the change under this comprehension point of bone physiopathology: the three-dimensional osteocytes network that commands the bone. Altering this network involves changing bone shape and repositioning the concerned structures, such as teeth, muscles and tendons. The dynamism of remodeling involves constant corporal adaptation to functional demands.

Mini-implants usage has broadened the possibility of planning with appliances, for a more efficient biomechanics in teeth repositioning, with awesome results – however, they are usually restricted to a small maxillary region, where they could work as anchorage points.

Classic **dental movement with orthodontic appliance**, regardless of the type of bracket, can be compared to the transmission of forces from the reins by the rider on his horse, directly changing the command of what is right ahead. Head and body move immediately around. The rider on the horse can be compared to the bracket of the anchorage tooth.

Mini-implants usage can be compared to a horse-drawn cart or a small carriage on which, with the reins attached to the animal, or two animals (side by side), the operator commands the forces applied to the animals' mouths, leading them to make some movements to the right, left or ahead. The reins are the wires; the brakes and accessories connected to the animal's mouth correspond to the brackets. The rider, in this analogy, corresponds to the mini-implant. Thus, the horse-drawn cart changes directions and goes ahead guided by the forces, adapting to the locomotion needs of the operator.

The **use of mini-plates** corresponds to big carriages, with two or three pairs of animals, used by big and fancy events and journeys in ancient English Imperial periods. The reins, or the wires, must be well calibrated and attached to the animals' mouth; the operator has been trained to the point of reaching perfection; and the carriage, is so well stabilized to the extent of providing its passenger safety in the route that was chosen to be traveled along. The operator knows what he wants from each pair of animals: the first one, in the front, leads, with its pioneer movements, the other ones, in harmony with the force lines of the reins and rods of the beautiful carriage.

Just like a big carriage, mini-plates can from afar command the shape of the cellular osteocytes network of the bone that supports the incisive and canine teeth in the anterior region. It is, thus, possible to influence the position and relation of the maxillary and mandibular median line. In other words: the anchorage offered by mini-plates allows a force

of such intensity, that it may be transmitted to remote regions. Such property may optimize orthodontic, orthopedic and rehabilitation results, with implications to esthetics and functions restored to the patient. Mini-plates can enable a wider remodeling of the maxilla and the mandible than the one obtained in a more limited way, with brackets and mini-implants.

FINAL CONSIDERATION

Picture the following situations:

- 1) A simple orthodontic movement.
- 2) The placement of an osseointegrated implant.
- 3) The implementation of the orthopedic appliance.
- 4) Dental movement with bone gain or change in form, by the anchorage in mini-implants.

- 5) Remote transmission of forces based on the anchorage offered by mini-plates, changing the position, shape and relation among the bones.

... in all of them, osteocytes are involved with their three-dimensional network in bone design controlling for coordinating the activity of their commanded ones on the trabecular and cortical surfaces: the osteoblasts and clasts.

May we know how understand bone biology and constant remodeling of the skeleton in order to act with safety and accuracy in patients' rehabilitation planning, increasing the possibilities of intervention to give them back esthetics and function.

REFERENCES

1. Bakker AD, Soejima K, Klein-Nulend J, Burger EH. The production of nitric oxide and prostaglandin E(2) by primary bone cells is shear stress dependent. *J Biomech.* 2001 May;34(5):671-7.
2. Baron R, Hesse E. Update on bone anabolics in osteoporosis treatment: rationale, current status, and perspectives. *J Clin Endocrinol Metab.* 2012 Feb;97(2):311-25.
3. Bonewald LF. Mechanosensation and transduction in osteocytes. *Bonekey Osteovision.* 2006;3(10):7-15.
4. Bonewald LF. Osteocytes as multifunctional cells. *J Musculoskelet Neuronal Interact.* 2006;6(4):331-3.
5. Bonewald LF. The amazing osteocyte. *J Bone Miner Res.* 2011 Feb;26(2):229-38.
6. Burr DB, Robling AG, Turner CH. Effects of biomechanical stress on bones in animals. *Bone.* 2002 May;30(5):781-6.
7. Crockett JC, Rogers MJ, Coxon FP, Hocking LJ, Helfrich MH. Bone remodelling at a glance. *J Cell Sci.* 2011 Apr 1;124(Pt 7):991-8.
8. Ehrlich PJ, Noble BS, Jessop HL, Steven HY, Mosley JR, Lanyon LE. The effect of in vivo mechanical loading on estrogen receptor alpha expression in rat ulnar osteocytes. *J Bone Miner Res.* 2002 Sep;17(9):1646-55.
9. Feng JQ, Ward LM, Liu S, Lu Y, Xie Y, Yuan B, et al. Loss of DMP1 causes rickets and osteomalacia and identifies a role for osteocytes in mineral metabolism. *Nat Genet.* 2006 Nov;38(11):1310-5. Epub 2006 Oct 8.
10. Kamioka H, Horjo T, Takano-Yamamoto T. A three-dimensional distribution of osteocyte processes revealed by the combination of confocal laser scanning microscopy and differential interference contrast microscopy. *Bone.* 2001 Feb;28(2):145-9.
11. Krstic RV. *Human microscopic anatomy.* Berlin: Springer-Verlag; 1994.
12. Lane NE, Yao W, Balooch M, Nalla RK, Balooch G, Habelitz S, et al. Glucocorticoid-treated mice have localized changes in trabecular bone material properties and osteocyte lacunar size that are not observed in placebo-treated or estrogen-deficient mice. *J Bone Miner Res.* 2006 Mar;21(3):466-76.
13. Lanyon LE. Osteocytes, strain detection, bone modeling and remodeling. *Calcif Tissue Int.* 1993;53 Suppl. 1:S102-6; discussion S106-7.
14. Nakashima T, Hayashi M, Fukunaga T, Kurata K, Oh-Hora M, Feng JQ, et al. Evidence for osteocyte regulation of bone homeostasis through RANKL expression. *Nat Med.* 2011 Sep 11;17(10):1231-4.
15. Parfitt AM. The cellular basis of bone turnover and bone loss: a rebuttal of the osteocytic resorption: bone flow theory. *Clin Orthop Relat Res.* 1977;(127):256-47.
16. Poole KE, van Bezooijen RL, Loveridge N, Hamersma H, Papapoulos SE, Löwik CW, et al. Sclerostin is a delayed secreted product of osteocytes that inhibits bone formation. *FASEB J.* 2005 Nov;19(13):1842-4.
17. Raab-Cullen DM, Thied MA, Petersen DN, Kimmel DB, Recker RR. Mechanical loading stimulates rapid changes in periosteal gene expression. *Calcif Tissue Int.* 1994 Dec;55(6):473-8.
18. Skerry TM, Bitensky L, Chayen J, Lanyon LE. Early strain-related changes in enzyme activity in osteocytes following bone loading in vivo. *J Bone Miner Res.* 1989 Oct;4(5):783-8.