compression exerted by the action of orthodontic and orthopedic appliances, similarly to what occurs with natural body movements.

**Osteocytes as mechanotransductors: more details**

Osteocytes individually pick up signals by mechanical deformation of their cytoskeleton. At the same time, the network in which each osteocyte participates, distributed throughout the bone structure, picks up deformations, overloads, deflections and limitations of nutrients. Deformation of the cytoskeleton as well as restriction of oxygen and nutrients stress the osteocytes. As a result, osteocytes release mediators to communicate with each other, as well as with osteoblasts and clasts on the bone surface, inducing them to reactive or adaptive phenomena.

During orthodontic movement, osteocytes undergo mechanical stress, which increases the production of mediators that circulate through the fluid in the canaliculi. Mediators reach the respective periodontal and bone surfaces and stimulate or inhibit bone formation and/or bone resorption in the “distant” cortical bone surface. In the bone marrow inside the bone, these mediators can influence higher or lower production of clastic cells and osteoclastogenesis.

Therefore, osteocytes strongly influence the function of bone to adapt its shape according to the determination of functional demands, thereby changing mechanical stimuli into biochemical events, a phenomenon also known as mechanotransduction.2,7,13 Osteocytes also play a major role in regulating mineral metabolism,9 in addition to inducing changes in the properties of bone matrix around it.12 However, these functions were already well known.

The skeleton is able to continuously adapt to mechanical loads by adding new bone so as to increase the ability to resist or remove bone in response to a lighter load or lack of use.6,8 Osteocytes have high interconnectivity and are considered as bone mechanotransductors. Osteocytes increase glucose-6-dehydrogenase phosphatase after a few minutes of load.19 This enzyme is a marker of increased metabolism which occurs in cells associated with bone surface. Seconds after load is applied on the osteocytes; nitric oxide, prostaglandins and other molecules, such as ATP, increase.1

Therefore, when facing induced loads, osteocytes have the ability to release mediators that stimulate the precursors of clasts or osteoclastogenesis to differentiate into new clasts, increasing the rate of resorption. Among these mediators, the M-CSF, or stimulating factor of colonies for macrophages, and the RANKL are the most significant ones.14 It can be argued that osteocytes can command the activities of the clasts on bone surfaces according to functional demand. The set of osteocytes or the lacunocanalicular osteocyte system can be considered as a real endocrine body.4

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**Figure 2** - In the mineralized bone matrix, osteocytes have many cytoplasmic extensions that interconnect with other 40 to 50 cells and, three-dimensionally, capture minor structural deformations. They fill the lacunae known as osteoplasts and their extensions are distributed in canaliculi filled with a tissue fluid that carries mediators (Mallory, 100X).
Correction of maxillary and skeletal alterations; miniplates and mini-implants

Tooth movement associated with changes in bone position, volume and shape continuously changes, for weeks or months, the tridimensional shape of maxillary bone. Absolute anchorage is required for these deformations to be efficient.

The use of miniplates provides enough anchorage to change the osteocyte network, causing it to release mediators that induce osteoblasts and clasts to directly reshape and restore bone volume and structure. Bone shape responds to functional demand and is able to correct major skeletal alterations, which not long ago was only possible through surgery.

The use of mini-implants with a view to causing major changes in shape, volume and dentoskeletal relationship is limited. Mini-implants are usually placed in the alveolar process of the maxilla and/or mandible.

"absolute" anchorage mini-implants provide within a limited system of force.

If mini-implants require greater force for correction of skeletal alterations, we can say that they offer subabsolute anchorage. As for miniplates, they offer real absolute anchorage as a result of being fixed in upper areas such as thicker cortical bone and denser trabeculae. Proper anchorage and thicker bone structure hardly allow deflection and deformation of the osteocyte network, thus providing absolute anchorage.

Final Considerations

This Insight recommends some studies that can be used to examine the degree of changes in shape, volume and structure in the areas where mini-implants and miniplates are placed for anchorage necessary for tooth movement and associated skeletal correction. Such studies allow us to classify mini-implants as devices of subabsolute anchorage and miniplates as devices of absolute anchorage.

REFERENCES