Convergent and divergent ideas concerning the use of mini-implants

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CONVERGENT IDEAS CONCERNING MINI-IMPLANTS

Certain ideas concerning the use of mini-implants for skeletal anchorage in orthodontic practice seem to have achieved widespread consensus1-43, such as:

1. Mini-implants represent a major breakthrough in the clinical orthodontic practice of the last 10 years, arguably the most relevant for contemporary Orthodontics.

2. The anchorage afforded by mini-implants can be utilized immediately following their implantation or up to 15 days later.

   The amount of initial force must be somewhere between 150 and 200 g, preferably measured with the help of a tension gauge to avoid overload. Gradually, this force can be increased up to 350 g by taking into consideration bone quality factors, such as cortical thickness and bone density.

3. The mini-implant action mechanism results from the mechanical interlocking of its metal structure in cortical and dense bone and is not based on the concept of osseointegration. The shape and length of the cutting threads are instrumental in mini-implant placement. Resistance to fracture forces can be enhanced by means of a tapered design and self-drilling threads. These features help to dissipate compression forces from bone structures adjacent to the mini-implant during insertion.

4. Osseointegration, when it does occur, hinders mini-implant removal thereby heightening the risk of fracture. For this reason, the pureness of the titanium alloy used in its composition is degree V. In areas where bone density is low and cortical bone thin osseointegration may be necessary. In these cases the titanium alloy employed should have a degree of pureness IV while the surface is treated with double acid etching to increase the contact surface. In 2007, Vannet et al.39 (Fig. 1) placed mini-implants in dogs and were able to unequivocally determine that from a histomorphometric standpoint partial osseointegration occurred in all specimens after 6 months of skeletal anchorage.

5. Mini-implant insertion can be simple in the hands of a well-prepared, skillful professional but can involve risks, especially if poorly planned and performed. Potential complications entail contact with neighboring tooth roots, with or without drilling, mucositis, contamination and fractures. Oral hygiene is fundamental if normal standards are to be maintained.

6. Mini-implants can be classified – according to their shape and use – as: a) self-drilling, the safest to avoid root perforation, and b) self-tapping, which requires prior bone drilling since it does not have a cutting edge.

7. Mini-implant structure comprises three sections: Body, transmucosal profile and head. The

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transmucosal profile represents the intermediate section in contact with the mucous membrane. The sections can vary according to shape and size, especially in terms of thickness and length.

8. The key success factors are: Gingiva anatomy, bone quality and/or density, distance to the roots and cortical bone thickness. According to Kyung et al.25 the successful use of mini-implants depends on the following factors: Surgeon skills, patient condition, appropriate site selection, initial stability and oral hygiene.

9. Mini-implants are also called micro-implants, micro-screws and anchorage pins, which together fall under the general category of Temporary Anchorage Devices3,4,28.

**ISSUES UNDER DEBATE CONCERNING THE CLINICAL USE OF MINI-IMPLANTS:**

To develop adequately, an idea needs to be disputed on an ongoing basis. Probing is the fuel behind its permanent development. Obviously, the same applies to mini-implants. Some of the issues most commonly addressed have to do with:

**What if, during placement, the mini-implant touches or brushes against a neighboring root?**

Teeth have their roots lined with cementoblasts and are permeated by Sharpey fibers, which are the periodontal fibers attached to cementum. Cementoblasts protect roots from continuous bone resorption. This protection results from an absence of receptors - on the cementoblast membrane – for the mediators in charge of permanent bone remodeling. Thus, hormonal changes, inflammation and periradicular stress are incapable of performing tooth resorption. If tooth resorption is to occur the cementoblasts on the root surface need to be removed. Such is the case in anoxia-induced orthodontic movement, traumas caused by direct mechanical action and chronic periapical lesions due to bacterial products.

As the mini-implant touches or brushes against the root surface cementoblasts and Sharpey fibers are eliminated and a resorption process begins at the site where trauma was induced. Although root resorption can be triggered by cementoblast removal, the process will not remain active for weeks, months or indefinitely unless local mediators, such as those causing cellular stress and inflammation, are present. When mediators disappear from the site and the cause of inflammation is removed the neighboring cementoblasts proliferate and once again cover the injured surface generating cementum deposition and the reattachment of periodontal fibers. Therefore, root resorption, if it does occur, will be limited, superficial and short-lived.

This phenomenon occurs because the area is
now free from bacteria which, if present, would likely prolong inflammation indefinitely. In 2005 Asscherickx et al.⁶, in a seminal study, experimentally induced (Fig. 2) contact with the root surfaces of dog teeth. The researchers found microscopically that after 12 weeks a new cementum had been formed and covered the entire region, as shown in figure 2.

This evidence supports the recommendation that during the placement of self-drilling mini-implants, in the event that the root surface is touched or brushed against, the best alternative lies in removing the mini-implants, repositioning them or even replanning the surgery from scratch. There is no need for any direct intervention. It should suffice to follow up on the case for 12 weeks using monthly periapical radiographs (Fig. 2). Inflammatory root resorption generates radiographic images after three weeks. Substitution tooth resorptions caused by alveolodental ankylosis take up to three months to generate radiographic images.

A mini-implant should never be allowed to come into direct and continued contact with the tooth root and, should contact take place, it should be removed. Tooth movement in the alveolus, when caused by chewing, permanently induces local damage by destroying cementoblasts and fostering micro areas of inflammation owing to a continual production of mediators. Left unchecked, such tooth movement is likely to bring about severe resorption of the mini-implant/root interface.

**What to do when the root is perforated?**

Self-drilling mini-implants have a tapered lower medial third and a cutting-edge. Their sur-
gical protocol is simpler, reducing the possibility of injury to the roots and providing greater primary stability compared with self-tapping mini-implants. Should they touch the roots, the likelihood of detouring or brushing against the roots is very high since they do not require prior bone drilling. In the case of self-tapping mini-implants, prior bone drilling can perforate the root.

It is important that contamination be prevented whenever a tooth root is accidentally perforated during mini-implant placement. In this situation, one should ask whether the perforation has reached as far as the pulp or root canal. If the cementum and dentin have been perforated but the pulp remains unscathed, the same behavior should be adopted as if the mini-implant has been touched or brushed against. It should either be removed and repositioned or a new placement be planned from scratch. Should a tooth be perforated without impairing the pulp, root resorption is likely to occur for a few weeks while traumatic and surgical inflammation will gradually disappear along with the resorption process mediators, since no bacterial contamination has taken place. Within a period of 3 to 6 months, periodontal tissues are likely to go back to normal with the area having been covered with new cementum and periodontal fibers reattached. Periapical radiographs should be taken on a monthly basis until the periodontal space has totally returned to a normal condition.

Should the dentin be perforated and the pulp and root canal be damaged, it should be noted that pulp and periodontal tissues have a remarkable healing capacity. In the event of horizontal root fractures, the literature is rich in reports describing the way professionals immediately bring together – as closely as possible – both root fragments, immobilize the crowns by means of splinting and, after a few months have elapsed, the fracture line is largely consolidated. Externally, cementum deposition will occur while internally the reactional and/or reparative dentin will form. The pulp will retain its vitality and may, on occasion, undergo premature ageing or evolve into calcific metamorphosis. When perforation is caused by the mini-implant, pulp trauma and injury are considerably less severe and circumscribed to a given area. The pulp may self-heal internally through the deposition of reactional or reparative dentin and undergo focal aging. The periodontal tissues will form new cementum and ligament.

The possibility of pulp necrosis cannot be ruled out, but only in severe pulp lesion accompanied by ruptured or crushed blood vessels. This is not a common condition since the symptoms are usually observed during the drilling stage, prior to the placement of a self-tapping mini-implant. If the self-tapping mini-implant is inserted, the likelihood of pulp necrosis developing in the affected tooth root is very substantial.

All these considerations regarding root perforation and pulp reaction have arisen from analogy and were inferred from a knowledgebase on pulp biology caused by tooth trauma, root fractures, accidental pulp exposure and pulpotomies. The literature has hitherto not yet produced experimental evidence or case studies of this particular subject in humans.

**Why do mucosites and perimini-implant growth tissue hyperplasia occur?**

From a biological standpoint, the most fragile part of an inserted mini-implant used for temporary anchorage is the area where it interfaces with the mucous membrane’s epithelial tissues. The epithelium binds to the mini-implant transmucosal profile by means of hemidesmosomes and in other alternative ways, including through the secretion of cementing substances into the interface of both structures. The epithelium in this interface proliferates as it seeks to simulate junctional epithelium, just as is the case with conventional dental implants.

Microbial biofilms grow on natural and artificial oral surfaces as a result of inadequate hygiene.
Biofilms can comprise vast populations of microbes, which gradually grow and ultimately settle in the epithelium/mini-implant interface where they induce an inflammatory process akin to gingivitis. Periodontitis may result if the underlying bone tissues are to any extent impaired.

In the case of mini-implants, mucositis is likely to arise and, if the process is allowed to evolve, may cause a perimini-implantitis, which can compromise mini-implant stability and eventually result in mini-implant failure.

Some mini-implants feature a design with a small circular winglet or metal ledge above the transmucosal profile, in the section right next to the head. Apparently, this metal ledge protects the mini-implant/mucous membrane interface on the outer surface but this protection is probably more physical than microbial since it is likely to enable the formation and maintenance of microbial film and can hamper access by tooth brushes and antiseptics to the region. In this respect, further research would be necessary. Mucosites and perimini-implantites occur even with well-inserted mini-implants, but only as a result of microbial biofilm formation on the parts which are exposed to the oral environment and in the absence of adequate hygiene.

**What about perimini-implant growth tissue hyperplasia?**

Skin and mucosa repair is accomplished through the development of granulation tissue, which fills lost spaces and gives rise to new connective tissue, thereby recovering the area affected by a given lesion. The key function of epithelial lining tissue is to isolate the internal milieu from the external milieu. When minor epithelial ruptures occur, such as chapped lips due to dryness, small periungual lesions, provisional crown and orthodontic band barbs, these are usually associated with the presence of low-virulence microbiota. Under these circumstances, the connective tissue - as a defense measure and to promptly reestablish the normal state of affairs - encourages the formation of granulation tissue in the region along with the proliferation of epithelial lining.

However, in children, adolescents, young adults, pregnant women or women using contra-
ceptive medication (Fig. 3), this reactionary capacity can be significantly exacerbated. The granulation tissue, in its initial and intermediate phases, is characterized by angiogenesis and the aforementioned individuals will suffer an increase in levels of serum and tissue angiogenesis stimulating factors.

In the exposed micro-areas, these individuals’ granulation tissue, once it is formed in an exacerbated manner, can generate an increased volume characteristic of pyogenic granuloma and pulp polyp, for example. These lesions indicate angiomatous hyperplasia of the granulation tissue. A reddish volume increase accompanied by bleeding can occur around the mini-implants (Fig. 3), especially in the epithelium/mini-implant interface covered with microbial biofilm.

The perimini-implant inflammation can no longer be characterized only by a reddish, peripheral area. This condition is replaced by a festooned volume increase, regular or irregular, presenting with bleeding and rather fragile to the touch (Fig. 3). The neighboring epithelium is stimulated towards a hyperplastic condition in order to cover this granulation tissue volume increase. Some of these perimini-implant hyperplasias are more reddish, but some feature pink, firm areas where the hyperplastic epithelium plays the part of lining (Fig. 3).

The treatment of mucositis and perimini-implant hyperplasias should start as soon as the main cause – microbial biofilms - is removed. Metal barbs, entrapped food particles and other low intensity, long-lasting local irritants should also be detected. Regression takes place between 24 and 48 hours. Should the condition persist, local causes should once again be sought. Whenever tissue growth is too prominent and the chance of spontaneous regression unlikely, surgical removal of the affected tissues would be recommended.

**Can mini-implants give rise to osteomyelites?**

Osteomyelites are inflammatory lesions characterized by extensive areas of compromised bone with disorderly resorption, purulent exudates and even multiple fistulas. Some of the signs and symptoms can be systemic such as fever, prostration and asthenia. Bone inflammation circumscribed to a specific area where bone neo-formation and sclerosis predominate and no systemic repercussions are detected, are identified as osteites.

Osteomyelites only affect patients suffering from a basic disease that leads to organic debility or in patients presenting with sclerotic bone diseases at the osteomyelitis site. Among the basic systemic diseases which can be associated with osteomyelitis are decompensated diabetes mellitus, immunosuppression, leukemic conditions, anemias, ethylism, senility, etc. Among the sclerotic bone lesions which can – when contaminated – give rise to osteomyelites are florid cemento-osseous dysplasia, Paget disease, among others.

Osteomyelitis hardly ever affects systemically healthy individuals with no sclerotic bone diseases. Should this be the case, the patients should be meticulously assessed to determine whether they suffer from any of these systemic, debilitating diseases. This fact helps to explain why – despite a wide array of clinical-surgical situations involving oral contamination - maxillary osteomyelites are rather rare.

Likewise, in the case of mini-implants, the possibility of inducing osteomyelites is minimal since prior to inserting the mini-implants, a comprehensive anamnesis, clinical exam and local systemic and bone evaluation should be conducted. Patients who present with a debilitating systemic disease, once the condition subdues to medical treatment, will return to normality.

In short, although the insertion of mini-implants constitutes a straightforward clinical-surgical procedure, it exposes and mingles the internal milieu with the external milieu inside the oral cavity, which is a highly contaminated environment. It is very important to conduct a systemic and bone assessment of the patient, as well as raise
the patient’s awareness to proper hygiene, a crucial ingredient in ensuring the success of the entire procedure. For many days the internal milieu will be separated from the external, contaminated milieu by a thin, albeit efficient epithelial barrier, namely, the mini-implant/mucosa interface.

**FINAL CONSIDERATIONS**

The use of mini-implants has broadened the horizons of Orthodontics and widened Implantology’s interface. Many aspects of the mini-implant still await clarification, but a statement made by Bezerra at a symposium on orthodontic anchorage was particularly noteworthy. After a lengthy description of literature surveys, he remarked: “The fact that scientific evidence is not yet available should not deter our attempts. If it works well and is clinically applicable it is important that professors, educational institutions, universities and other research centers do their very best to find effective solutions. I’d like to unpretentiously introduce some suggestions for future studies on the use of mini-implants in orthodontic practice:

1. Pulp and periodontal reactions after mini-implants are touched or brushed against, laying foundations for prevention, conduct and treatment.
2. Pulp and periodontal reactions after root perforation with mini-implants.
3. Degree of influence exerted by mini-implants on the growth and distribution of microbial biofilms when mini-implants are exposed to the oral milieu.
4. Morphology of the oral mucous membrane tissues and their interface with mini-implants, particularly the epithelium, and interaction mechanisms between the different mucous membrane areas.
5. A comparative study between the microscopic characteristics of mucosites and perimini-implantites and those of gingivitis and periodontitis.
6. A comparative study between the clinical and microscopic characteristics of perimini-implant hyperplasias and those of pyogenic granuloma and inflammatory fibrous hyperplasias in the oral mucosa.
7. Case reports involving accidents and lesions associated with the use of mini-implants as a contribution to their prevention and treatment.

**REFERENCES**

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