Orthodontic forced eruption: Possible effects on maxillary canines and adjacent teeth

Part 3: Dentoalveolar ankylosis, replacement resorption, calcific metamorphosis of the pulp and aseptic pulp necrosis

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Canine forced eruption comprises one among a number of procedures that can be used in orthodontic treatment to ensure that cuspids are positioned in the dental arch in normal esthetic and functional conditions. Canine forced eruption should be characterized as an orthodontic movement.

Unfortunately, in discussions of clinical orthodontic practice some professionals are reluctant to indicate orthodontic forced eruption, especially of maxillary canines. These professionals believe that orthodontic forced eruption can cause many clinical problems during and after surgery. Among the most widely cited reasons for restricting the indication of orthodontic forced eruption are:

1) Lateral root resorption in lateral incisors and premolars.
2) External cervical resorption of canines due to forced eruption.
3) Dentoalveolar ankylosis of the canine involved in the process.
4) Calcific metamorphosis of the pulp and aseptic pulp necrosis.

In two previous works, we reviewed the first two topics. In this last article in the series we address the biological foundation of dentoalveolar ankylosis, replacement resorption, calcific metamorphosis of the pulp and aseptic necrosis cases either directly or indirectly related to the orthodontic forced eruption of canines.

How to distinguish orthodontic forced eruption from other procedures

There are other ways to position unerupted, or erupted but poorly positioned canines in the dental arch using surgical procedures. Surgical displacement of canines is given such names as "fast-track canine forced eruption," or rapid canine extrusion,

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but in fact involves an autogenous intra-alveolar[10] transplant and does not make use of induced tooth movements with the aid of periodontal tissues. There is no such thing as surgical canine "forced eruption" since this expression refers to a force applied to the tooth. A more appropriate denomination would be surgical displacement or intra-alveolar autogenous tooth transplant. Surgical displacement of canines can cause:

a) disruption of the periodontal ligament.

b) compromised vasculo-nervous bundle of the pulp.

c) The need to partially or fully prepare a socket to receive the canine.

The rupture of the periodontal ligament can damage cementoblasts and the epithelial rests of Malassez, structures without which inflammatory resorption, dentoalveolar ankylosis and replacement resorption tend to occur. Moreover, disruption of or damage to the vascular pulp-periodontium bundle may induce aseptic pulp necrosis or calcific metamorphosis of the pulp.

Consequences of the surgical displacement of canines are similar to the possible effects of traumatic injuries as the affected structures are the same. In surgical displacement however, unlike in dental trauma, microbial contamination can be controlled while tissue damage can be minimized through adequate planning. In dental trauma the forces are unpredictable when applied to the tissues and injuries vary in scope and intensity. In a basic analogy, one can say that the consequences of the surgical displacement of an unerupted canine can resemble dental trauma.

Genuine canine forced eruption is an orthodontic movement, not a surgical displacement. This distinction becomes crucial as soon as one begins to analyze the possible consequences of canine orthodontic forced eruption.

Surgically induced dislocation is independent of orthodontic forced eruption

Surgical approach of the canine crown involves handling the tissues of the dental follicle, exposing enamel, and sometimes improperly and inconveniently also exposing the cementoenamel junction, which may result in external cervical resorption, among other consequences. These surgical issues regarding the dental follicle, exposure of the enamel and cementoenamel junction and their impact on bracket bonding have been presented in previous papers.[6,7,8]

When requesting surgeons to bond a bracket on the crown of an unerupted tooth, orthodontists are not requesting, nor expecting surgeons to complement the surgical procedure by dislocating the canine with the purpose of facilitating orthodontic movement. Strictly speaking, surgically induced dislocation in cases of canines that require forced eruption should be undertaken at the orthodontist's request. When performed without such request, for reasons identified during the surgical period, the orthodontist must necessarily be informed by the surgeon about such decision.

This idea of "facilitating" forced eruption through dislocation can only be understood in the world of physics without considering that tooth movement—of which orthodontic forced eruption is but one example—comprises a set of biological events. Forces delivered through orthodontic movement induce biological events, determine the intensity and sites where such forces should be applied, but do not replace these events.

Surgically induced dislocation of teeth necessarily involves the rupture of the periodontal ligament, rupture of periodontal vessels, nerves and fibers, and cellular fragmentation and disorganization of epithelial rests of Malassez. Three-dimensionally, the epithelial rests of Malassez appear as a network of well-organized basketball hoops around the tooth in the context of the periodontal ligament. These changes induced by periodontal dislocation can be controlled if surgical procedures are well planned

and accurately performed without overdoing forces and repetitive handling of instruments.

Surgically induced dislocation is a risky procedure to which teeth should be subjected only when potential benefits are significant, as in cases of well-established and accurately diagnosed dentoalveolar ankylosis. Among the risks of induced dislocation is dentoalveolar ankylosis. Should such condition not be present, consequences may involve replacement resorption, calcific metamorphosis of the pulp and aseptic pulp necrosis.

The procedure of surgically induced dislocation refers to increased tooth mobility in the alveoli attained through the agency of surgical instruments. Such mobility is higher than the one commonly observed as a function of the periodontal ligament. In ankylosed teeth, tooth mobility, even such as results from the presence of periodontal ligament, is not observed. Lever movements performed with surgical instruments can accomplish dislocation and this is perceived as discrete forces applied to the tooth with the instrument heads. However, professionals, in their eagerness to verify that mobility has occurred during dislocation, can—with their instruments or fingers—induce considerable movements in the alveoli. If the dislocation itself had not produced major periodontal injuries, these verification or checking maneuvers can now cause such injuries or even enlarge them.

Indications for surgically induced dislocation during orthodontic forced eruption

Surgically induced dislocation for therapeutic purposes is a valid alternative but only when clearly indicated after a clinical and/or definitive imaging diagnosis of dentoalveolar ankylosis, and not performed preemptively to mechanically "facilitate" orthodontic forced eruption. In the presence of an unerupted canine, an indication for induced dislocation can be reached by following this diagnostic path or protocol to decide on the therapeutic approaches to be undertaken. This protocol can be divided into three necessarily sequential different times:

1. First step of diagnosis and therapeutic decision: evaluate and create adequate space for the canine in the dental arch.

When an unerupted canine is present, the first evaluation should ascertain space availability in the dental arch as well as normal dental follicle tissues.[2,5,11] Should eruption be mostly attributed to the dental follicle, space availability in the dental arch should disclose not only the mesiodistal width of the crown but also the presence of follicular tissue in the follicular space.[1,2,5]

The measurement to be added to the canine mesiodistal width, which must be considered to accommodate the uncompressed dental follicle in the eruptive path, with or without orthodontic forced eruption, can use as reference half of that width (1.5 times the mesiodistal canine width) although this is not always applicable in all clinical cases. In many cases, the potential space is much smaller and the canine erupts, but this increases the risk of resorption in neighboring teeth[6,7]—although sometimes such risk is inevitable. It must be assumed that the dental follicle of maxillary canines,given their unique anatomy, tend to bulge and broaden laterally more than any other teeth.

In some cases, space is sufficient and natural eruption is just a matter of time. But depending on patient age, orthodontic assessment and clinical need, there is no time or reason to wait.

2. Second step of diagnosis and therapeutic decision: orthodontic forced eruption.

Even when the available and required space is orthodontically provided for natural eruption of the canine, the tooth does not move toward the arch. It may be impacted in an area of denser bone, hindered by a more pronounced root curvature, intercepted by the root of a neighboring tooth,

or else it just may not display eruptive force. After a two month period with no sign of the eruption, even with sufficient space available, one can opt for orthodontic forced eruption—which requires the bonding of a bracket, some specific orthodontic device, or even perforation of the enamel for anchoring the orthodontic wire and applying the necessary force in terms of intensity in the appropriate direction.

3. Third step of diagnosis and therapeutic decision: surgically induced dislocation, followed by orthodontic forced eruption.

Even when sufficient available space is orthodontically provided, sometimes the unerupted tooth will not move, and in some cases, even through forced eruption one fails to direct or "pull" the tooth into that arch space. In radiographic and/or CT images, dentoalveolar ankylosis may not appear owing to the early stage of the process or to image superimposition. Dentoalveolar ankylosis only appears in imaging diagnostic tools when over 20% of its root surface area has been affected.[3] Prior to this degree of impairment, the images obtained will be normal and this can give rise to uncertainty in exclusively clinical diagnoses, where the support of diagnostic images is not available. However, one should not wait for biological phenomena to develop before generating diagnostic images. If a tooth had adequate space available and was subsequently subjected to orthodontic forced eruption and even so failed to move occlusally, the only remaining option is surgically induced dislocation.

Possible consequences of surgically induced dental dislocation

The consequences of surgically induced dislocations are directly related to the degree of injury sustained by the periodontal ligament, especially in the cementoblast layer and epithelial rests of Malassez. In the pulp, induced injury and its consequences are also dependent on the shape, intensity and direction of forces delivered during the surgical procedures of surgically induced dislocation.

Inflammatory resorption would only indicate injury to the layer of cementoblasts and maintenance of the epithelial rests of Malassez and the periodontal space, but it is not usually observed in teeth subjected to forced eruption and surgical dislocation. If periodontal damage occurs due to surgically induced dislocation, typically this will also affect the epithelial rests of Malassez, induce dentoalveolar ankylosis and subsequent replacement resorption.

Ankylosis and replacement resorption after forced eruption usually manifest themselves months or years after the procedure has been performed when the tooth is in its appropriate position in the dental arch. In most cases they are detected by chance during routine examinations. The processes of ankylosis and replacement resorption are asymptomatic, with no evident clinical signs. Tooth darkening may be associated, but when this occurs it is not due to ankylosis or resorption but rather results from injuries to the pulp, such as ***calcific metamorphosis of the pulp and/or aseptic pulp necrosis***,[4,9] which may also have been induced by maneuvers during dislocation surgery, i.e., tooth darkening represents only a simultaneous occurrence.

If during dislocation there is partial damage to the neurovascular bundle and partial and/or temporary restriction of pulp oxygenation and nutrition, the cells undergo metaplasia and settle randomly and diffusely into a dysplastic dentin, i.e., poorly formed and deposited with the purpose of filling and reducing cellular metabolism at the site to ensure survival. This dentin partially or totally obliterates the pulp chamber (Figs 1 and 2) over a period of 3 months to 1 year after procedure.[4,9] Consequently over time, the tooth will darken slowly, affecting the patient's aesthetics.

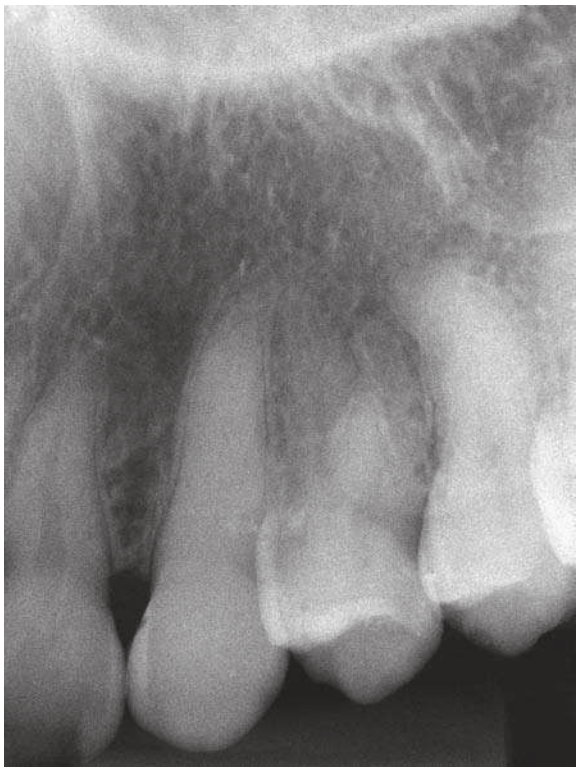

FIGURE 1 - Maxillary canine subjected to orthodontic forced eruption which after many months showed gradual crown darkening and radiographs showed obliteration of pulp chamber by calcific metamorphosis of the pulp. The most likely causes were surgically induced dislocation performed simultaneously with placement of bracket/orthodontic device, showing injury to neurovascular bundle of pulp and/or "fast-forced eruption."

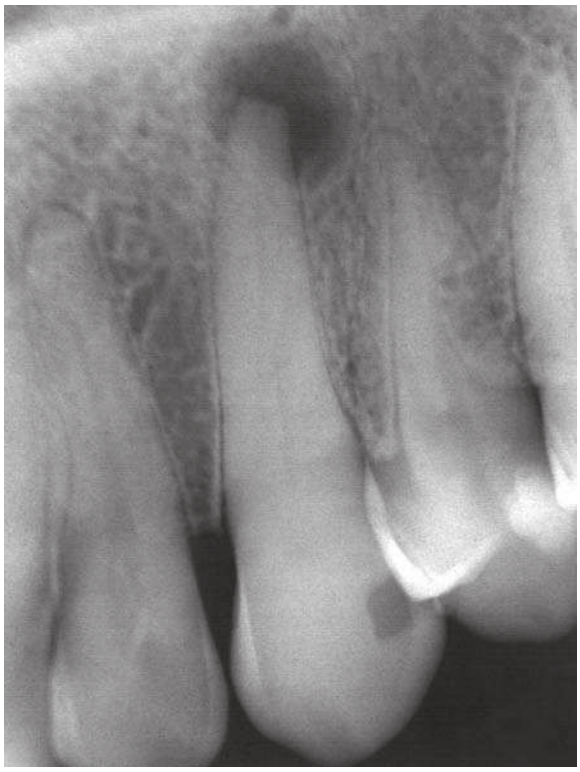

FIGURE 2 - Maxillary canine with obliteration of pulp chamber by calcific metamorphosis of the pulp. It is noteworthy that after a few years chronic periapical lesion was detected. It is found in approximately a quarter of cases between 2 and 22 years of monitoring.

Although it is asymptomatic, within periods of up to 22 years later calcific metamorphosis of the pulp can produce chronic periapical lesions in 24% of affected teeth[4,9] (Figs 1 and 2). Root canal therapy may be rendered impracticable due to canal obliteration, making it necessary to use a paraendodontic approach. In cases where endodontic treatment is no longer possible and chronic periapical lesions are not yet manifest, yearly external dental bleaching can improve esthetics, although not as a definitive solution because the deposit of dysplastic dentin in the pulp chamber cannot be resolved. More lasting and satisfactory esthetic and functional results may be attained through facet installation.

In cases of aseptic pulp necrosis there was complete disruption of the pulpal neurovascular bundle during surgically induced dislocation. Pulp cells contain few lysosomes with their proteolytic enzymes and thus, when they undergo necrosis their proteins tend to coagulate, remaining in the site indefinitely. In other words, without vascularization the pulp undergoes anemic infarct, a necrosis due

to protein coagulation. Thus, one can spend months or years with no symptoms as one's interface and relationship with the rest of the body is conducted exclusively through the minute apical foramen. In general, the most common clinical consequence for the patient manifests as gradual darkening of the tooth depending on the gradual and slow decomposition of dead tissues and incorporation of pigments derived from the inner wall of the dentin. The pulp chamber is maintained and over the years one can detect the presence of chronic periapical lesions. Endodontic treatment is indicated as well as external and/or internal dental bleaching.

Surgically induced dental dislocation: When should it be indicated?

In the third step of diagnosis and therapeutic decision making, dislocation is an option. If the canine remains unerupted, and remains in place with ankylosis it will evolve over time towards replacement resorption and loss. If dislocation is well planned with precise and delicate maneuvers without aggressive verification chances are that it will get back to normal if it is followed by extrusion when the subsequent orthodontic forced eruption is performed. In cases where this procedure still results in ankylosis and replacement resorption after the canine tooth is properly positioned in the dental arch, planning may involve its replacement by an osseointegrated implant, or orthodontic space closure followed by re-anatomization of the premolars.

In cases of darkening by calcific metamorphosis of the pulp and aseptic necrosis endodontic procedures lead to esthetically and functionally adequate results with preservation of the natural canine tooth.

However, surgically induced dislocation should not be indicated without restrictions in all cases of forced eruption of unerupted canines, but only when dentoalveolar ankylosis is accurately diagnosed. The risks involving ankylosis, replacement resorption, calcific metamorphosis of the pulp and pulp necrosis not only exist but are of considerable prevalence.

If orthodontic forced eruption is well planned and performed it is an orthodontic movement and as such is a safe procedure whose consequences are minor and clinically manageable. Even when conducted in association with surgically induced dislocation, also well planned and consciously performed, orthodontic forced eruption remains a safe procedure.

In short, orthodontic forced eruption, if performed as a tooth movement, does not promote ankylosis, replacement resorption, calcific metamorphosis of the pulp or aseptic pulp necrosis. These problems stem from technical procedures during surgically induced dislocation.

Speed of movement during orthodontic forced eruption

During surgically induced dislocation in cases where it was adopted as a therapy prior to orthodontic forced eruption, small movements induced during operative procedures, although intense, should not cause large displacements of the tooth in the socket as partial or total lesion of the neurovascular bundle may develop.

However, special care should also involve the intensity of the forces and the speed of tooth movement during orthodontic extrusion induced in canines whose forced eruption resulted from dislocation. Dislocation "loosens" the tooth, even when well accommodated in the tooth socket. Injuries to the neurovascular pulp bundle are commonly associated with cases of "fast-track orthodontic forced eruption," which actually consists of a therapeutically adopted tooth avulsion that causes surgically induced dislocation and tooth displacement to inflict a severe dental injury to the neurovascular bundle in addition to the other aforementioned periodontal damage. Orthodontic

forced eruption is a tooth movement and, as such, has its speed limits because movement is effected by the periodontal ligament cells.

Final considerations

Orthodontic forced eruption should be considered an induced tooth movement just like any other orthodontic movement. Its forces and direction induce tooth extrusion and are responsible for the specific features of this orthodontic procedure. In planning and implementing orthodontic forced eruption of canines, the anatomical and functional characteristics of the periodontal ligament should be considered.

The unintended consequences most often cited to restrict the indication of forced eruption are of a technical and procedural nature and can be explained biologically. They are: a) Lateral root resorption in the lateral incisors and premolars, b) External cervical resorption in the canine involved in the process, c) Dentoalveolar ankylosis of the canine involved in the process, d) Calcific metamorphosis of the pulp, and aseptic pulp necrosis.

These possible outcomes do not arise primarily and specifically from orthodontic forced eruption. They can be avoided if certain technical precautions are adopted, especially the "four cardinal points for the prevention of problems during orthodontic forced eruption,"[6] namely:

Assess the dental follicle and its relations with neighboring teeth.

Value the cervical region of the unerupted tooth to avoid exposure and surgical manipulation of the cementoenamel junction.

Ensure that the dislocation performed prior to forced eruption does not become severe dental trauma caused by unnecessary surgical procedures.

Preserve the apical neurovascular bundle that enters the root canal during the procedure of verifying that dislocation has been attained, or by increasing the speed of forced eruption in the occlusal direction.

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