Simplified Removal of an Osseointegrated Implant for Space Closure to Correct Anterior Crossbite

Abstract

***Diagnosis**: A 45-year-old male presented for orthodontic consultation with concerns about a concave facial profile and anterior crossbite. Clinical examination showed facial asymmetry with a cant in the occlusal plane that was associated with mandibular deviation to the right by about 2mm. Cephalometrics revealed a skeletal bimaxillary protrusion (SNA 89°, SNB 89°, ANB 0°). There were multiple missing teeth (UR8, UR7, UR4, UL2 , UL8, LR6, LL6, and LL8), and four endodontically treated teeth (UL5, UL7, LR7, LL6, and LL7). Missing lower first molars were restored with a fixed prosthesis on the right side and an implant-supported prosthesis on the left. A large area of pathology, possibly condensing osteitis, was distal and apical to the root of the LL4.*

***Treatment**: All restorations were replaced by provisional crowns except for the metal crown on the UL7, and the gold crown on the LL8. The pontic restoring the LR6 was cut out with a handpiece. An osseointegrated implant-supported prosthesis (ISP) restoring the LL6 was removed with a sustained counterclockwise torsional load (see text for details). A passive self-ligated, fixed appliance with anterior bite turbos (UR1, UL1) was used to correct the anterior crossbite by retracting the anterior segment with space closure mechanics, supplemented with light force Class III elastics. The edentulous space for the UR4 was opened with a compressed coil spring to receive an ISP. After orthodontic treatment, all provisional crowns were restored with porcelain fused to metal (PFM) prostheses.*

***Results**: After 38 months of treatment, the profile was improved, midlines were coincident, and normal overjet/overbite was achieved. The anterior crossbite was corrected and molar relationships were Class II on the left and Class I on the right. Lower incisors were tipped distally (76.5°), and upper incisors were flared labially (116°). All prostheses were restored as needed. The apparent condensing osteitis apical to the root of the LL4 decreased in size and remained asymptomatic, but endodontic evaluation is indicated. A complex malocclusion with a Discrepancy Index (DI) score of 19 was treated to a Cast Radiograph Evaluation (CRE) of 13.*

***Conclusions**: Osseointegrated implants can be easily removed with a simplified torsional overload procedure to permit optimal orthodontic management of malocclusion. (J Digital Orthod 2020;58:68-90)*

Key words:
Implant removal, anterior crossbite, minimally invasive approach, space closure

Introduction

The osseointegration concept was first described by Dr. Brånemark in 1952 and published by Albrektsson et al. in 1985.[1-3] These researchers referred to the result of their technique as "a direct functional and structural connection between living bone and the surface of a load carrying implant.' There is direct bone anchorage to an implant (*locking the implant into the jaw bone*) which can provide a foundation to support a prosthesis. Therefore, osseointegration is a perfect boundary for an implant surgeon, but a nightmare for an orthodontist. An osseointegrated implant is like an ankylosed tooth. It is almost unmovable, which obstructs tooth alignment and space redistribution. This case report presents a minimally invasive approach of an atraumatic removal of an implant and a treatment of a Class I malocclusion with anterior crossbite.

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The dental nomenclature for this report is a modified Palmer notation. Upper (*U*) and lower (*L*) arches, as well as the right (*R*) and left (*L*) sides, define four oral quadrants: UR, UL, LR and LL. Teeth are numbered 1-8 from the midline in each quadrant, e.g. a lower right first molar is LR6.

Diagnosis and Etiology

A 45-year-old male came for orthodontic consultation. He had a migraine and doubted that it was induced by his malocclusion. External examination indicated protrusive lower lip, asymmetric facial structures (*Fig. 1*) and coincident dental midlines that deviated to the right of facial midline (*Fig. 2*). The asymmetry of the mandible can be observed from the cephalometric and panoramic radiographs (*Figs. 5 and 6*).

Fig. 1: Pre-treatment facial and intraoral photographs

Fig. 2:
Coordinated dental midlines were shifted to the right of the facial midline.

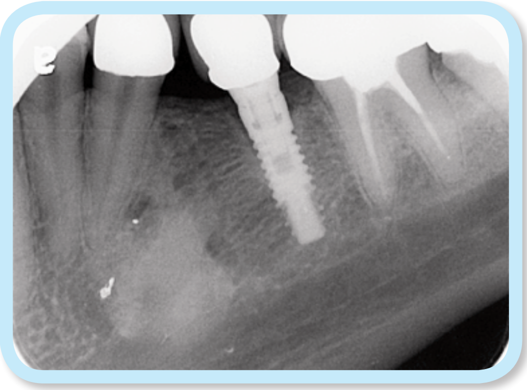


Fig. 3:
An implant was placed 5 years ago to restore a missing LL6. An asymptomatic mass of sclerotic tissue is noted distal to the root of the LL5. See text for details.

Fig. 4: *Pre-treatment study models (casts)*

Fig. 5: *Pre-treatment cephalometric radiograph*

Fig. 6:
Pre-treatment panoramic radiograph. Notice the canted occlusal plane and the asymmetry of the mandible.

Intraoral examination revealed an anterior crossbite of five mandibular teeth - from the right canine to the left lateral incisors. An edentulous space presented on the right maxilla (*Fig. 1*) and there was a canted occlusal plane (*Figs. 1 and 7*), which may have resulted from the imbalanced mandibular corpus or early loss of the mandibular permanent first molars. The pre-treatment study cast showed Class I molar relationship on the right, but end-on class III on the left (*Fig. 4*). The pre-treatment cephalometric analysis showed a 0° ANB angle and a low mandibular plane angle (*Fig. 5, Table 1*). Panorammic radiography (*Figs. 3 and 6*) revealed missing teeth (*UR8, UR7, UR4, UL8 and LR6*), teeth with crowns (*UR6, UL5, UL7, LL8, LL7 and LL5*), and an implant-supported prosthesis LL6.

Fig. 7:
When the bite is opened, no mandibular shift was noted. However, the occlusal plane is canted inferiorly on the right.

CEPHALOMETRIC SUMMARY			
SKELETAL ANALYSIS			
	PRE-Tx	POST-Tx	DIFF.
SNA° (82°)	89°	89°	0°
SNB° (80°)	89°	89°	0°
ANB° (2°)	0°	0°	0°
SN-MP° (32°)	29°	29°	0°
FMA° (25°)	22°	22°	0°
DENTAL ANALYSIS			
U1 To NA mm (4 mm)	6	6.5	0.5
U1 To SN° (104°)	113°	116°	3°
L1 To NB mm (4 mm)	8	4	4
L1 To MP° (90°)	88°	76.5°	11.5°
FACIAL ANALYSIS			
E-LINE UL (-1 mm)	-3	-2.5	0.5
E-LINE LL (0 mm)	2.5	-0.5	3
%FH: Na-ANS-Gn (53%)	56%	56%	0
Convexity: G-Sn-Pg' (13°)	-4.5°	-4°	0.5°

Table 1: Cephalometric summary

The American Board of Orthodontics (*ABO*) Discrepancy Index (*DI*) was 19 points, as shown in the supplementary Worksheet 1.

Treatment Objectives

1. Remove all prostheses and place provisional crowns as needed.
2. Use full fixed, passive self-ligating (*PSL*) appliance to level and align both arches.
3. Open edentulous space between the UR3 and UR5 with a compressed coil spring.
4. Restore the missing UR4 with an ISP.
5. Remove the LR6 pontic and the LL6 implant.
6. Correct anterior crossbite closing L6 spaces to retract the lower anterior segment.
7. Optimize occlusion with finishing bends and posterior vertical elastics.

Treatment Options

Plan A. Removal of the LL6 implant (*Fig. 8*) was the preferred approach, but the patient was concerned about a difficult surgical procedure. It was explained that bone supporting an osseointegrated implant has a relatively weak layer near the implant surface. Thus, a sustained counterclockwise torsional load (*reverse torque*) causes bone failure and the implant can be easily removed with minimal trauma.

Plan B. Instead of lower space closure, the anterior crossbite can be treated with interproximal reduction (*IPR*) of the mandibular incisors and space closure to tip the lower incisors lingually. Simultaneous Class III elastics tip the maxillary incisors anteriorly to help correct the anterior crossbite (*Fig. 9*). The drawbacks for this approach are compensated (*tipped*) upper and lower incisors as well as compromised (*more protrusive*) dentofacial esthetics. The patient selected Plan A (*implant removal and space closure*) because he preferred the expected outcomes for that approach.

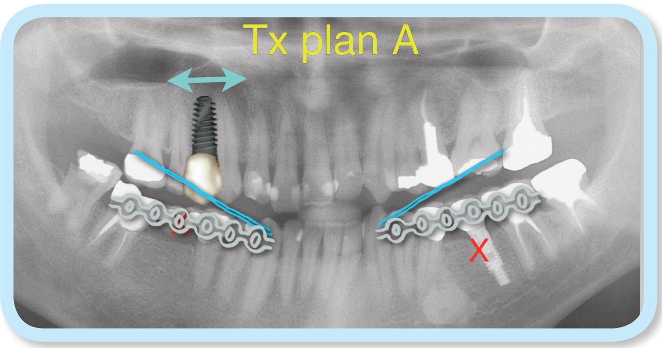


Fig. 8:
Treatment plan A: remove the LL6 osseointegrated implant and LR6 bridge pontic to create space for retraction of the mandibular anterior segment to correct the anterior crossbite. See text for details.

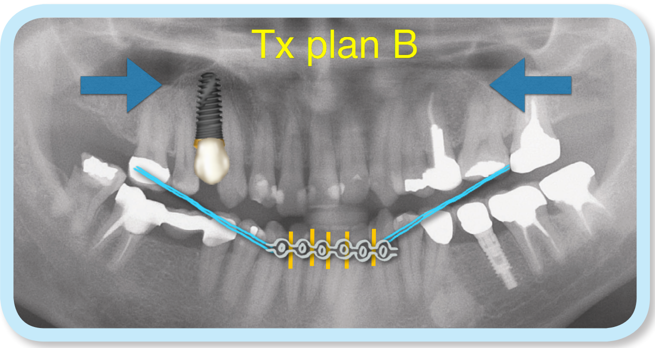


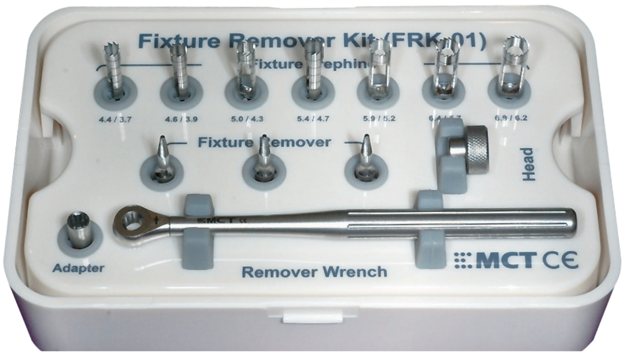
Fig. 9:
Treatment plan B: perform IPR on mandibular incisors and retract them as the maxillary incisors are tipped anteriorly with Class III elastics to correct anterior crossbite. See text for details.

Treatment Progress

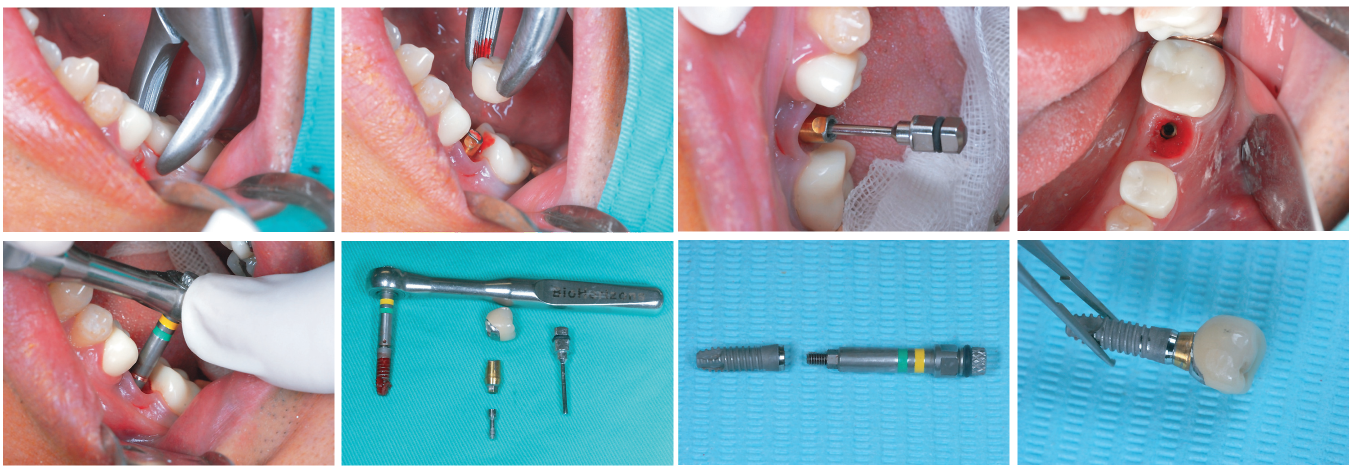
Before the start of orthodontics, all prostheses were replaced with new provisional crowns as needed. A PSL fixed appliance was selected (*Damon Q®, Ormco, Brea, CA*). The maxillary arch was bonded at the start of active orthodontic treatment (*0M*) utilizing low torque brackets on the incisors. At the same appointment, an open coil spring was inserted between the UR3 and UR5 to open space for an implant to restore the missing UR4. Bite turbos were constructed as inclined planes on the lingual of the lower central incisors to facilitate anterior crossbite correction (*Fig. 10*). Three months (*3M*) into treatment, the implant-supported prosthesis (*area LL6*) was removed. Failing implants are frequently removed for restorative purposes,[4] but a successful, well integrated 3.5x11mm fixture is a challenge (*Fig. 3*). Two sets of instruments from the original manufacturer (*BioHorizons, Birmingham AL*) were used: implant placement and removal kits (*Fig. 11*). The crown was removed with a tooth extracting forceps (*Fig. 12*), and the abutment was loosened with a screwdriver. After the implant driver was tightly secured to the fixture, the wrench was engaged and held in place with an index finger. The wrench was rotated counterclockwise with a steadily increasing pressure until the supporting bone near the implant surface failed in shear. Once the fixture was loosened, it was easily removed. The torsional load was maintained for a minute or more to allow

■ *Fig. 10: Resin bite turbos were bonded on the lower central incisors to open the bite and assist in anterior crossbite correction.*

time for a shear-type fracture in the interfacial bone. A thin layer of bone tissue was on the surface of the recovered implant (*Fig. 12*), which was consistent with an intra-osseous failure, i.e. within the primarily mineralized bone layer.[7] The implant socket (*wound*) was checked 40 minutes later to confirm that there was adequate bleeding and clot formation. Follow-up evaluations of the healing edentulous space was performed from 1-7 days after implant removal. The site was well healed at 7 days (*Fig. 13*), and space closure commenced.



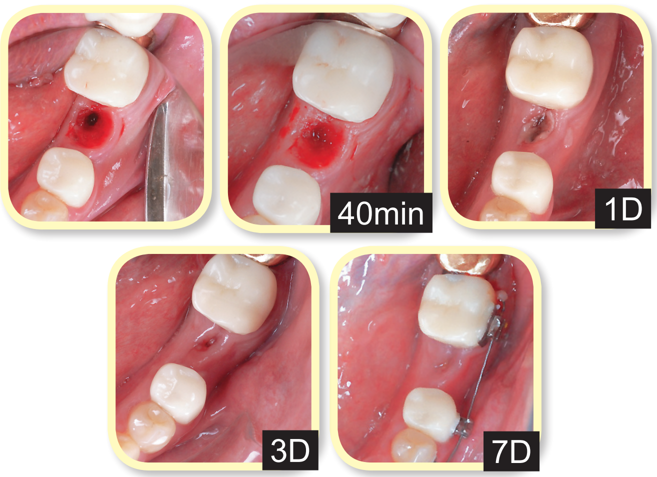
■ *Fig. 11: Implant fixture removal kit*



■ *Fig. 12:*

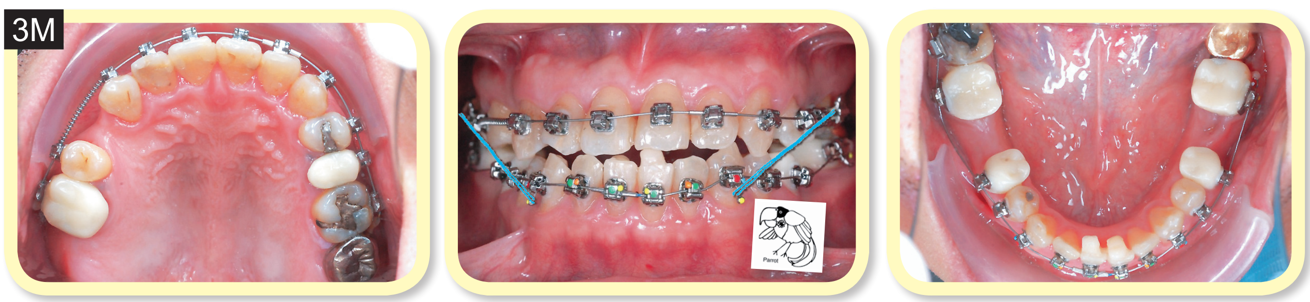
Implant removal (extraction) required only 10 minutes, but a sustained mechanical overload in torsion (reverse torque) was required to fracture interfacial bone rather than the implant. See text for details.

One month after the start of upper arch treatment (*3M treatment time*), a full fixed appliance was installed on the lower arch. Low torque brackets were bonded upside down to deliver positive torque to the incisors, high torque brackets were placed on the canines (*Fig. 14*), and the initial wire that was inserted was a 0.014 NiTi. Early light Class III elastics (*Parrot 2 oz.*) from the U6s to the L3s were used for anterior crossbite correction. Three months later (*6M treatment time*), the anterior crossbite was improved to an edge-to-edge relationship (*Fig. 15*), and the anterior bite turbos were removed. In the 10th month (*10M*), lingual buttons were bonded on the lower first premolars, second molars and third molars, respectively. Power chains were applied on both the labial and lingual surfaces of the buccal segments to close space (*Fig. 16*), and reactivated once a month. Class III elastics were changed to Fox (*3.5 oz.*). One month later (*11M treatment time*), a reverse curve of Spee was bent into the lower arch.



■ *Fig. 13:*
Occlusal intraoral photographs show healing of the alveolus at 40 minutes, as well as 1-7 days (D). See text for details.

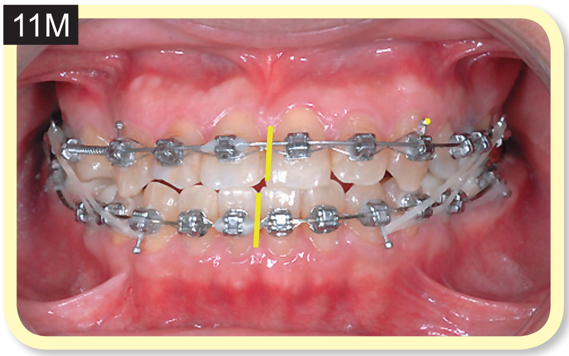
The upper dental midline was moved left by the open coil spring (*Fig. 17*). In the 15th month (*15M*), the anterior crossbite was corrected. Class III elastics were changed to Class II elastics on the right side for midline correction. In the next month (*16M into treatment*), the upper archwire was expanded, and a Class II elastic was added from the LR5 via the LR3, and up to the UL1 to reinforce dental midline correction. The bracket that was bonded on the gold crown of the LL8 failed (*Fig. 18*). A pre-operative cone-beam computed tomography (*CBCT*) scan was taken to evaluate the bone volume of the implant site (*Fig. 19*). The bone volume was sufficient to place a 4x9mm implant.



■ *Fig. 14:*
In the third month (3M), low torque brackets were bonded upside down on the lower incisors, and high torque brackets were placed on the canines. Early light Class III elastics (blue lines) were applied. See text for details.

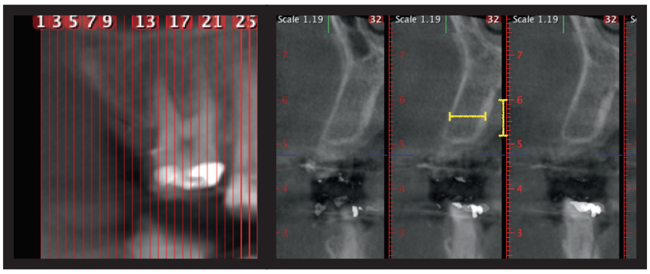
■ *Fig. 15:*
By the 6^{th} month (6M) of active treatment, the incisors were edge-to-edge. See text for details.

■ *Fig. 16:*
In the 10^{th} month, buttons were bonded on the lingual surfaces of lower first premolars as well as the third and second molars, respectively. Power chains were applied on both buccal and lingual sides to close space. See text for details.



■ *Fig. 17:*
At eleven months (11M), the upper dental midline was moved left by the open coil spring. See text for details.

■ *Fig. 18:*
Bonding a bracket on the LL8 gold crown failed, which compromised the space closure mechanics. See text for details.



■ *Fig. 19:*
A pre-operative cone-beam computed tomography (CBCT) scan shows the bone morphology of the UR4 implant site.

Implant Placement Procedure

After 25 months of orthodontic treatment, the UR4 implant was placed. Flap reflection was achieved with crestal and sulcular incisions on the buccal and palatal sides of adjacent teeth. After the first lancer drill, a periapical film was taken, with a surgical guide pin to check the long axis of the osteotomy and its proximity to the adjacent teeth. Following the manufacturer's recommended drilling and expansion procedures, the implant site was surgically developed, step by step according to the 2B-3D rule: 2-mm of buccal bone and 3-mm apical to desired margin of the future crown.[14,15] A 4S x 9mm Astra OsseoSpeed™ (*Dentsply Implants, Mannheim, Germany*) implant fixture was installed. A flared healing abutment (*5.5mm x H4mm*) was screwed into the implant to form the peri-implant mucosal contour. A post-operative periapical radiograph documented the final position of the implant with its healing abutment (*Fig. 20*).

Orthodontic Finishing

Correction of anterior crossbite usually requires a detailed finishing approach.[13,16] Brackets are rebonded as needed to correct dental axis inclinations. IPR and space closure may be required in either anterior segment, particularly if black triangles are a problem.[16] The current patient required IPR for the upper central incisors to improve crown form, interproximal contacts with adjacent teeth, and overjet (*Fig. 21*).

Implant Prosthesis Fabrication

Eight months after the implant was placed, the healing abutment was removed. A direct abutment width at >2mm of occlusal clearance for PFM crown construction was installed in preparation for the prosthesis fabrication. Before the impression was made, the abutment was torqued twice to 25-30 N-cm with a torque wrench. After the pick-up impression, the abutment was covered with the Tony cap[15] to prevent soft tissue overgrowth.

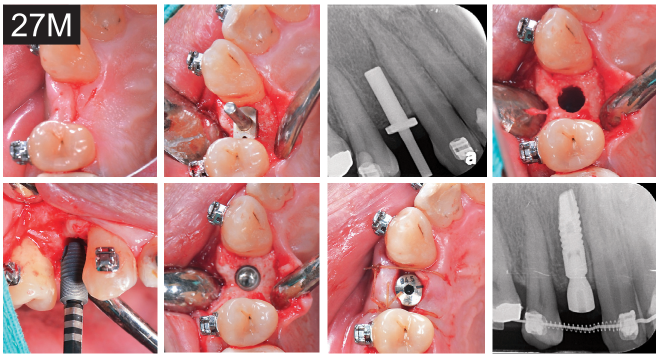


Fig. 20:
The UR4 implants was placed at twenty-seven months (27M) into treatment. A series of intraoral photographs and radiographs document the procedure.

Fig. 22:
The UR4 implant prosthesis fabrication procedure is illustrated with a series of photographs. See text for details.

Fig. 21:
At thirty months (30M) into treatment, space closure is in progress for the lower arch (left), and the UR4 implant is healing (center). The occluded relationship of the jaws is show on the right. See text for details.

All permanent crowns were delivered in the 38th month. Marginal integrity was verified with a dental explorer (*Figs. 22-24*). Clear overlay retainers were delivered for both arches. The patient was instructed to wear the overlays full time for the first month and nights only thereafter.

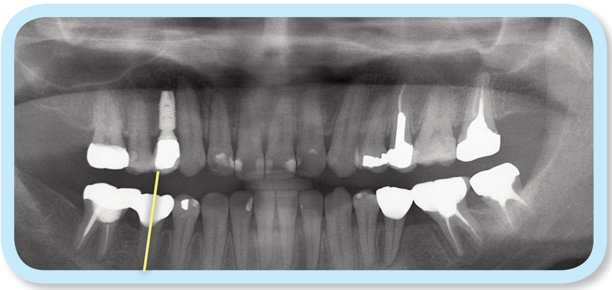
■ *Fig. 23:*
All final prostheses were secured after appropriate tightness of the contact area was confirmed with dental floss.

Treatment Results

The patient was treated to the desired dentofacial result as documented in Figs. 23-28. Negative overjet was corrected and the concave facial profile was improved to an acceptable, straight relationship. The ABO Cast-Radiograph Evaluation (*CRE*) score was 13 points (*Worksheet 2*). The major residual problems were in the occlusal relationships (*7 points*) and overjet (*3 points*). The post-treatment panoramic radiograph revealed the root of the LR5 was mesially inclined (*Fig. 26*). Superimposed tracings (*Fig. 27*) showed that the mandibular incisors were extruded and excessively tipped to the lingual (*from 88° to*

■ *Fig. 24: Post-treatment facial and intraoral photographs*

■ *Fig. 25: Post-treatment cephalometric radiograph*

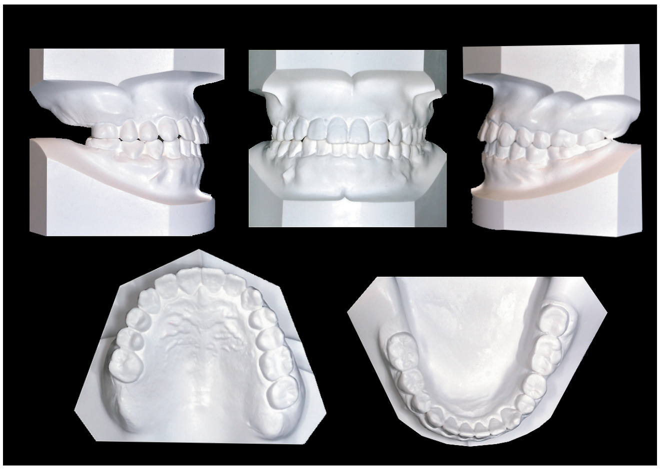


■ *Fig. 26:*
Post-treatment panoramic radiograph. The root of tooth LR5 was tilted mesially (yellow line).

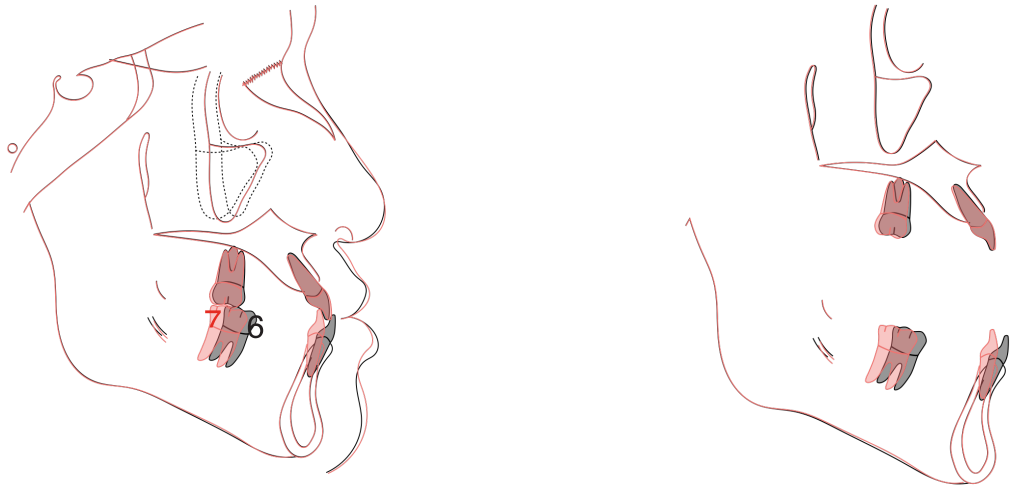
76.5°), molars were substituted with the protracted second molars, and the lower lip was retracted. There was little change in the maxilla. The patient was satisfied with the result.

Discussion

There are no reports in the literature for extracting a successful osseointegrated implant for orthodontic purposes. Osseointegration is a term coined by Per-



■ *Fig. 28: Post-treatment study models (casts)*



■ *Fig. 27:*
Cephalometric tracings before (black) and after (red) treatment are superimposed on the anterior cranial base (left), maxilla (upper right) and mandible (lower right). The lower first molar (6) are substituted by the lower second molar (7) after space closure. See text for details.

Ingvar Brånemark[1-3] that defines the direct structural and functional connection between ordered living bone and the surface of a load-bearing implant, i.e. ankylosis.[7] A well integrated implant can support a prosthesis and/or serve as orthodontic anchorage.[5,6] An osseointegrated implant cannot be moved with an orthodontic load,[5,6] so a well integrated fixture can interfere with tooth movement.[7] The current case report presents a minimally invasive approach for atraumatic removal of an implant as part of a comprehensive treatment plan to correct a severe asymmetric malocclusion with anterior crossbite (*Figs. 1-7*). Careful consideration of the physiology and biomechanics of osseointegration[5-7,11] provided the rationale for a relatively atraumatic extraction procedure (*Fig. 12*) that healed well (*Fig. 13*). The bone healing process, prosthetic procedures, biomechanics, implant failure mechanisms, and soft tissue considerations are relevant to understanding the achievement and maintenance of osseointegration.[17-22]

Bone Healing Process

After implant insertion, the gap between adjacent bone and the implant surface fills with a blood clot and healing begins with intramembranous[18] or de novo[17] bone formation. The healing process is a sequence of platelet activation, blood clot formation, angiogenesis, osteoconduction (*osteogenic cell recruitment and migration*), woven bone formation, compaction of woven bone by lamellar bone, and eventually secondary remodeling of the primary osteons.[7,17-22] The expected sequence is:

1. Wounded bone is covered with a blood clot after implantation. Leukocytes and macrophages are engaged in the wound-cleansing process. Macrophages secrete angiogenic and fibrogenic growth factors.[17]

2. High concentration of fibronectin allows attachment of fibroblasts. Cells migrate into the wound. The coagulum starts to be replaced by granulation tissue, and new angiogenesis is observed.[18-21]

3. Hypoxia attracts macrophages and the stimulated vascular endothelial growth factor (*VEGF*) induces detachment of pericytes from the outer walls of the vessel (*Fig. 29*). The pericytes give rise to the new endothelial progenitor cells.[7-9] These cells then proliferate to form hollow capillary buds (*Fig. 30*) and they arrange themselves to form tubes which are connected to an existing blood vessel. Under VEGF influence, a new vascular loop is created so blood can flow through. The detached pericytes (*osteoprogenitor cells*) migrate forward along the fibrin networks until they reach the bone or implant surface, then differentiate into osteoblasts (*Fig. 31*). The initiation

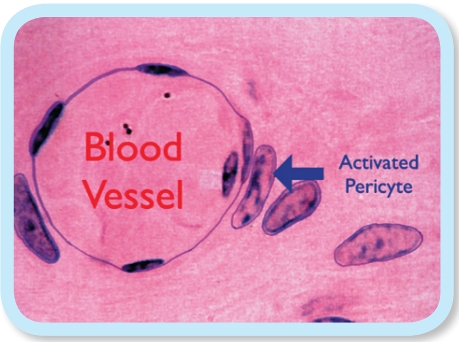


Fig. 29:

Activated pericytes are precursors for osteoblasts. (From Chang et al.[8,9])

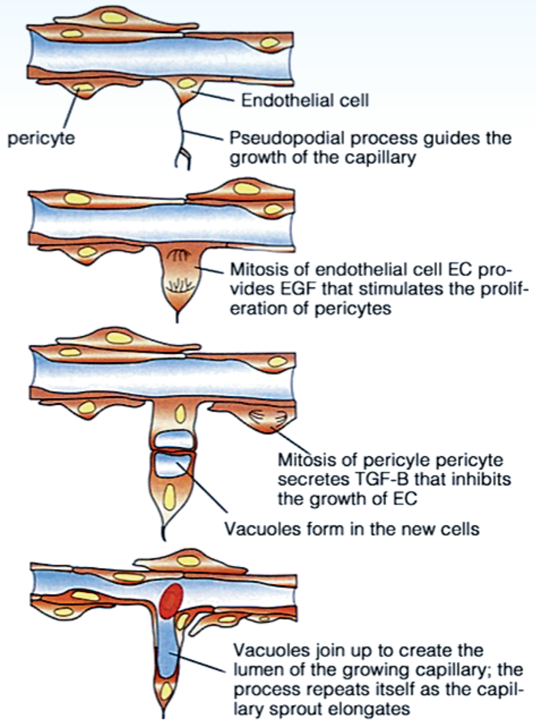


Fig. 30:

Pericytes propagate along the surface of enlarged capillary sprout. (From Chang et al.[8,9])

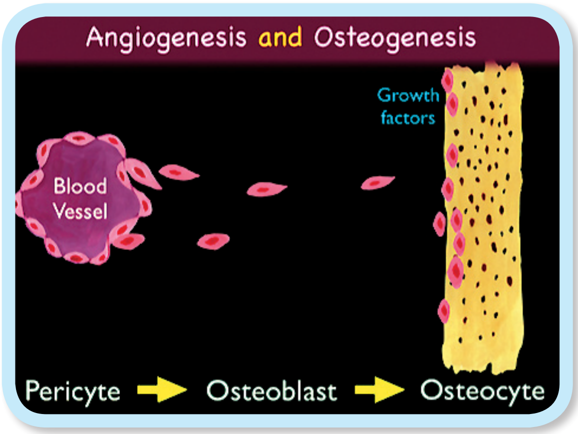


Fig. 31:

Angiogenesis and Osteogenesis: pericytes are stimulated by growth factors to migrate away from blood vessels and differentiate into osteoblasts. Some of the osteoblasts become osteocytes. See text for details.

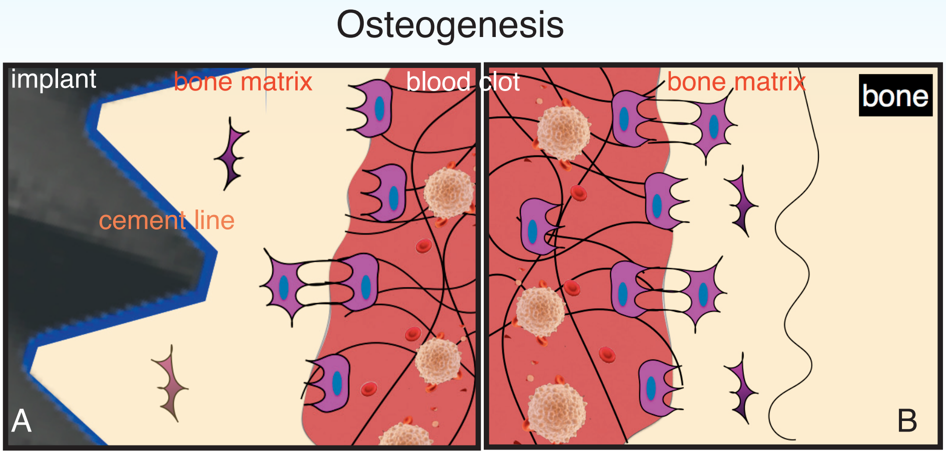
of platelet activation results in osteogenic cell recruitment and migration to the implant surface (*osteoconduction*).[8,9] Woven (*immature*) bone appears in the mesenchymal tissues.[7]

4. New bone formation begins with the secretion of a collagen matrix by osteoblasts. This matrix is subsequently mineralized by hydroxyapatite.[7] Then nanometer-sized uniaxially oriented hydroxyapatite crystal plates (*foot plates*) are formed within the collagen fibers. Woven bone formation increases, surrounding the implant.[17-21]

5. The immature woven bone is replaced with mature bone via a remodeling process that produces only lamellar bone.[7] The initial woven bone is oriented parallel to the titanium surface in the grooves of the threads. The subsequent lamellar bone forms on the macro-threads, except at the tip of each thread which is a stress riser.[11]

Bone-Implant Interface

The cement line (*Fig. 32*) along the bone interface of an endosseous implant is required to attach new bone. In effect, the surface of a titanium implant is viewed as old bone from a physiologic perspective.[7] Cement lines separate old from new bone at all remodeling sites.[7,24] Osborn and Newesley[20] describe two distinct phenomena for bone formation at the bone/implant interface: distance and contact osteogenesis. In distance osteogenesis, new bone is formed on the surfaces of old bone in the peri-implant site, not directly on the implant itself but on the surface approaching it. In contact



■ *Fig. 32*:
Contact osteogenesis (A) and distance osteogenesis (B) are related to implant healing. The drawing is after Davies.[17] *See text for details.*

osteogenesis, new bone forms first on the implant surface where no bone is present. The implant surface must be colonized by bone cells before bone matrix formation can begin. De novo bone formation, as described by Davies,[17] begins with bone matrix secreted by osteoblasts differentiated from local osteogenic cells.[7-9] The osteogenic cells reach the implant surface via fibrin before initiating extracellular matrix synthesis. The osteoblasts secrete a thin layer of collagen-free organic matrix, cement substance, directly on the implant surface before new bone is attached.[7,19] Two non-collagenous bone proteins, osteopontin and sialoprotein, are present in the initial organic phase. Calcium phosphate nucleation is followed by crystal growth and the initiation of collagen fiber assembly. This collagen compartment of bone will be separated from the underlying substratum by the collagen-free calcified tissue layer (*cement line*).[19] Distance osteogenesis is defined as bone formation approximating the implant surface while contact osteogenesis is bone apposition along the implant surface.[18]

The matrix secreted by osteoblasts is mineralized and becomes bone tissue. The embedded osteoblasts turn into osteocytes or die (*Fig. 32*). Bone matrix mineralizes so bone tissue has no capacity for inherent expansion (*"grow"*).[7] The continued growth of bone away from the implant surface is due to continued recruitment and migration of osteogenic cells,[8,9] processes which are deemed "*osteoconduction*." The combination of osteoconduction and de novo bone formation results in contact osteogenesis.[12]

The cement line was first described for Haverian systems by von Ebner in 1875.[7,19] It demarcates old from new bone (*Fig. 33*). Cement lines are composed of an afibrillar, collagen-free, but mineralized interfacial matrix is laid down between secondary

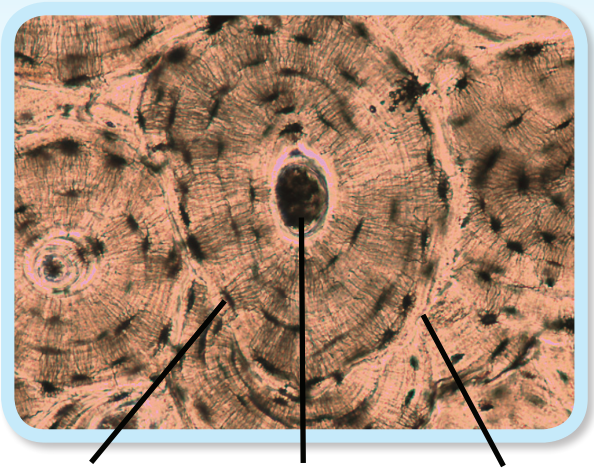


Fig. 33:
Haversian system: an osteon in cortical bone has lacuna that contain osteocytes and a peripheral cement line. See text for details.

osteons and pre-existing bone.[7-24] Although its thickness and appearance vary, this zone forms on the implant surface. More recent high resolution immunocytochemical studies[25] demonstrate that the electron-dense interfacial layer is rich in noncollagenous proteins, such as osteopontin (*OPN*) and bone sialoprotein (*BSP*) which are believed to play roles in cell adhesion and binding of minerals.[22] As reported by Carter and Hayes,[24] mechanical failure of normal bone frequently occurs at cement lines, so they are generally considered a relatively weak area within cortical bone.[25] Assuming the afibrillar mineralized cement line is similar to cortical bone, the strength is about 7.31 MPa for a small hole in the supporting plate. However, a test more relevant to osseointegration failure is a large hole in the supporting plate. Under the latter conditions, the strength of the cement line is about 74 MPa, which approximates the strength of bone lamellae.[25] Mechanical testing of a variety of implanted biomaterials confirms that the toughness of the bone-implant interface is significantly inferior to the intrinsic strength of supporting bone.[29] For natural bone, the shear strength is about 68 MPa and the tensile strength is about 100-105 MPa according to Cowin et al. (*1983*).[26] In 1997, Edwards et al.[27] reported tensile strengths for a bone formed on a smooth hydroxyapatite interface as 0.15 ± 0.11 MPa at 55 days and 0.85 ± 0.55 MPa at 88 days in a rabbit tibial model. These relatively low strengths for bone attachment to a smooth surface suggest that internal strength of a bone and implant interface strength primarily reflects a 3D mechanical interlocking of living and dead materials. In all of the studies reviewed, the strength of interfacial bone is less than the strength of the fully mineralized supporting bone. When an implant is loaded in torsion in the direction to unscrew the fixture (*counterclockwise*), the cement line attaching the bone to the implant may fail. However, it is a very thin (*1-5μm*) layer,[25] so a cement line failure may not result in loosening of an implant because of the overall irregularity of a screw-form implant surface. Furthermore, the thin layer of bone on the recovered implant (*Fig. 12*) suggests the failure was within interfacial bone (*intra-osseous fracture*), not at the cement line. Fragility of the cement line is only part of the unique physiology for bone support of implants. The strength of lamellar bone within 1mm of the implant surface must also be considered.[28-34]

Interfacial Layer of Partially Mineralized Bone

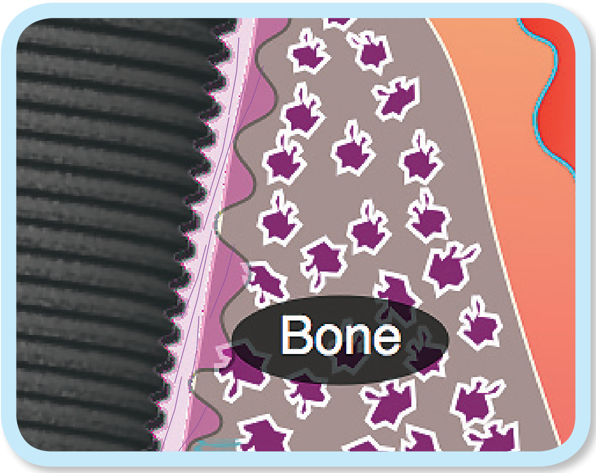
An osseointegrated bone-implant interface has a layer of rapidly remodeling bone within about 1mm of the fixture surface.[28-30] This partially mineralized bone layer at the interface has less strength compared to the supporting fully mineralized bone.[35] A sustained torsional load in a counterclockwise direction is expected to result in shear failure of interfacial bone, and loosening of the implant with no damage to surrounding tissue. There is a mismatch in the modulus of elasticity at the titanium-bone interface because titanium is about 10x stiffer than cortical bone.[7,30-34] Bending and flexure of dissimilar structures, such as the implant and supporting bone, creates surface shear that drives a high rate of bone remodeling within about 1mm of the implant interface.[7,33,34] The interfacial bone turns over completely several times per year so there is inadequate time for it to undergo secondary mineralization.[7,29-31] Bone strength is directly related to the mineral content.[35] Thus the 1mm layer of primarily mineralized bone at the implant interface has less strength than the metal implant or the fully mineralized bone supporting it.[33,34] In effect, the primarily mineralized bone is a compliant layer between to rigid materials (*implant, fully mineralized bone*) which is analogous to the periodontal ligament.[31] The cushioning effect of an intermediate relatively compliant layer may be a requirement for anchoring a rigid material like titanium in living bone.

Fibrous Capsule

When an implant remains stable in relation to supporting bone, osseous integration occurs along the implant surface. Unfavorable mechanical conditions such as micromotion, premature loading, and trauma cause motion between the implant and supporting bone that disrupts the osteogenic reaction, which in turn results in a fibrous encapsulation of scar-like fibrous connective tissue (*Fig. 34*). Szmukler-Moncler[36] concluded that about 100 µm of micromotion disrupts the fibrin network and new vasculature that is critical for a normal bone healing process. The disturbed mesenchymal stem cells divert from the bone pathway and differentiate into fibroblasts that produce a fibrous capsule (*scar tissue*). Prior to osseointegration, fibrous encapsulation of an implant was considered a "*pseudo-periodontium*," but the biomechanics and physiologic integrity of the supporting tissues was never established. Brånemark et al.[1-3] clearly confirmed that fibrous encapsulation of an implant is an integration failure: subsequently, the entire dental implant field has accepted that standard.

Inflammation

Inflammatory destruction of soft tissues supporting dental implants is termed mucositis and peri-implantitis.[37] Mucositis is a bacteria-induced,



■ *Fig. 34:*
Implant Failure: micromotion disrupt the newly formed healing bone tissue and results in fibrous encapsulation. See text for details.

reversible inflammatory process affecting peri-implant soft tissue. The symptoms are reddening, swelling, and bleeding on periodontal probing, that occurs prior to radiographic bone loss. In contrast, peri-implantitis is a progressive, irreversible disease of supporting tissues that is manifested as increased bone resorption, decreased osseointegration, periodontal pockets >5mm, and purulence.[38]

None of the implant failure criteria reviewed (*bone loss, fibrous encapsulation, mucositis or peri-implantitis*) applied to the ISP restoring the LL6 that was removed for orthodontic purposes. The periodontal probing depth was <4mm (*Fig. 35*) and there was no radiographic evidence of bone loss (*Fig. 36*). A radiolucency around the neck of the implant is consistent with a modest cratering of supporting bone, which is normal for functionally loaded implants. Neither crestal bone loss nor implant mobility was evident. The relatively simple torsional overload method for atraumatic implant removal exploits the principles of bone physiology and biomechanics associated with osseointegration.

Fig. 35:
The probing depth was 4mm. There was no soft tissue reddening or swelling.

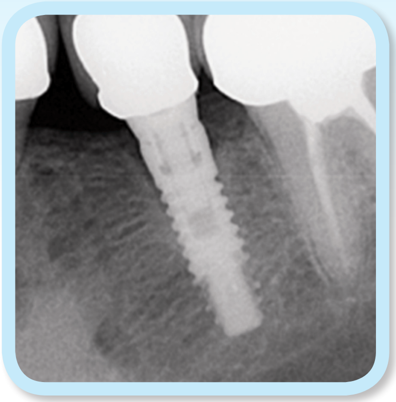


Fig. 36:
There was a radiolucent shadow (crevice) around the neck of the implant, which is consistent with the slight cratering morphology that is typical of successful ISP in occlusal function. There was no crestal bone loss nor mobility. The LL6 ISP was a healthy implant. See text for details.

Conclusions

There are three features at bone to implant interface: 1) cement line (*osseointegration*), 2) fibrous capsule (*fibrointegration*), and 3) inflammation tissue (*peri-implantitis*). Each of them is an intrinsically weak area. An implant that can be screwed in can therefore be screwed out, as long as it can be firmly secured to the removal instrument. The three keys to the successful outcome of this treatment are: 1) a correct diagnosis and treatment plan, 2) an atraumatic removal of the implant, and 3) an accurate mechanism to retract the mandibular dentition and close space.

Fig. 37 documents the current condition of the patient around 3 years and 7 months post-treatment.

Acknowledgment

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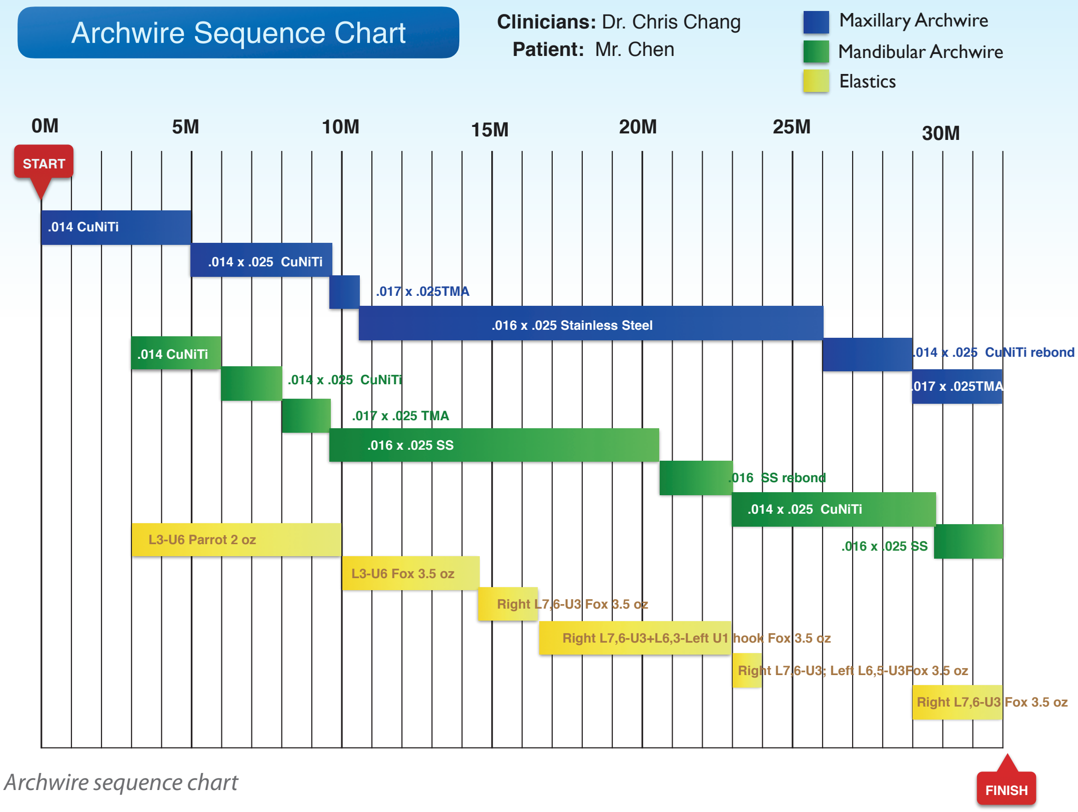

Table 2: Archwire sequence chart

Fig. 37: Facial and intraoral photographs at 3Y7M follow-up

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Discrepancy Index Worksheet

TOTAL D.I. SCORE 19

OVERJET

0 mm. (edge-to-edge)	=	
1 – 3 mm.	=	0 pts.
3.1 – 5 mm.	=	2 pts.
5.1 – 7 mm.	=	3 pts.
7.1 – 9 mm.	=	4 pts.
> 9 mm.	=	5 pts.

Negative OJ (x-bite) 1 pt. per mm. per tooth = 1+2+2+1+1

Total = 7

OVERBITE

0 – 3 mm.	=	0 pts.
3.1 – 5 mm.	=	2 pts.
5.1 – 7 mm.	=	3 pts.
Impinging (100%)	=	5 pts.

Total = 0

ANTERIOR OPEN BITE

0 mm. (edge-to-edge), 1 pt. per tooth
then 1 pt. per additional full mm. per tooth

Total = 0

LATERAL OPEN BITE

2 pts. per mm. per tooth

Total = 0

CROWDING (only one arch)

1 – 3 mm.	=	1 pt.
3.1 – 5 mm.	=	2 pts.
5.1 – 7 mm.	=	4 pts.
> 7 mm.	=	7 pts.

Total = 0

OCCLUSION

Class I to end on	=	0 pts.	
End on Class II or III	=	2 pts. per side	4 pts.
Full Class II or III	=	4 pts. per side	___ pts.
Beyond Class II or III	=	1 pt. per mm. additional	___ pts.

Total = 4

LINGUAL POSTERIOR X-BITE

1 pt. per tooth Total = 0

BUCCAL POSTERIOR X-BITE

2 pts. per tooth Total = 0

CEPHALOMETRICS (See Instructions)

ANB ≥ 6° or ≤ -2° = 4 pts.

Each degree < -2° ________x 1 pt. = ________

Each degree > 6° ________x 1 pt. = ________

SN-MP

≥ 38° = 2 pts.

Each degree > 38° ________x 2 pts. = ________

≤ 26° = 1 pt.

Each degree < 26° ________x 1 pt. = ________

1 to MP ≥ 99° = 1 pt.

Each degree > 99° ________x 1 pt. = ________

Total = 0

OTHER (See Instructions)

Supernumerary teeth	________	x 1 pt. =	________
Ankylosis of perm. teeth	________	x 2 pts. =	________
Anomalous morphology	________	x 2 pts. =	________
Impaction (except 3rd molars)	________	x 2 pts. =	________
Midline discrepancy (≥3mm)		@ 2 pts. =	________
Missing teeth (except 3rd molars)	3	x 1 pts. =	3
Missing teeth, congenital	________	x 2 pts. =	________
Spacing (4 or more, per arch)	________	x 2 pts. =	________
Spacing (Mx cent. diastema ≥ 2mm)		@ 2 pts. =	________
Tooth transposition	________	x 2 pts. =	________
Skeletal asymmetry (nonsurgical tx)		@ 3 pts. =	3
Addl. treatment complexities	1	x 2 pts. =	2

Identify:
5-year-old implant removal

Total = 8

IMPLANT SITE

Lip line : Low (0 pt), Medium (1 pt), High (2 pts) =_

Gingival biotype : Low-scalloped, thick (0 pt), Medium-scalloped, medium-thick (1 pt), High-scalloped, thin (2 pts) =_

Shape of tooth crowns : Rectangular (0 pt), Triangular (2 pts) =_

Bone level at adjacent teeth : ≤ 5 mm to contact point (0 pt), 5.5 to 6.5 mm to contact point (1 pt), ≥ 7mm to contact point (2 pts) =_

Bone anatomy of alveolar crest : H&V sufficient (0 pt), Deficient H, allow simultaneous augment (1 pt), Deficient H, require prior grafting (2 pts), Deficient V or Both H&V (3 pts) =_

Soft tissue anatomy : Intact (0 pt), Defective (2 pts) =_

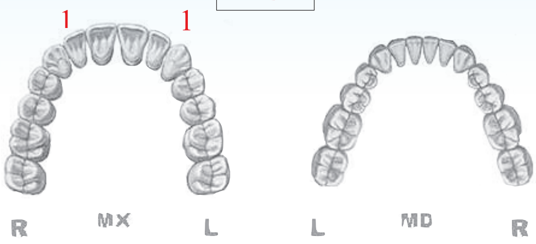
Infection at implant site : None (0 pt), Chronic (1 pt), Acute(2 pts) =_

Cast-Radiograph Evaluation

Total Score: 13

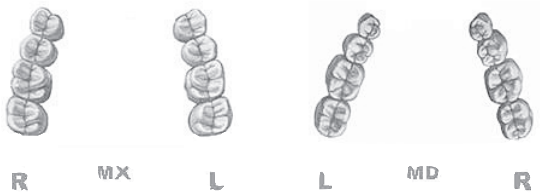
Alignment/Rotations

2



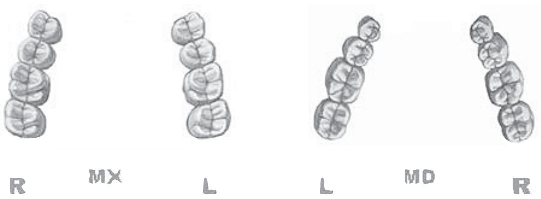
Marginal Ridges

0



Buccolingual Inclination

0

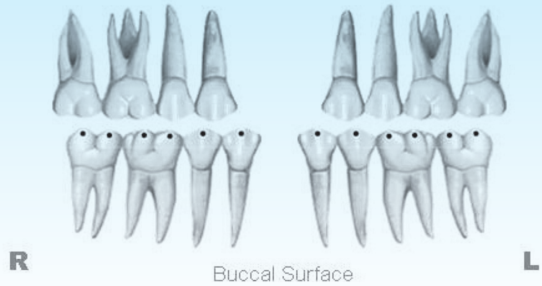


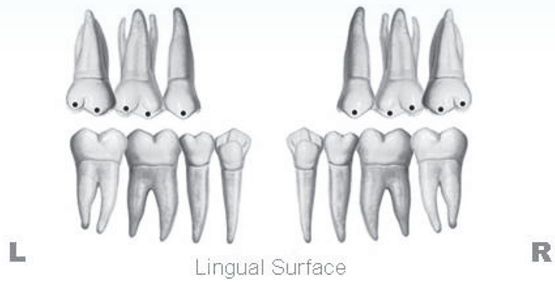
Overjet

3

Occlusal Contacts

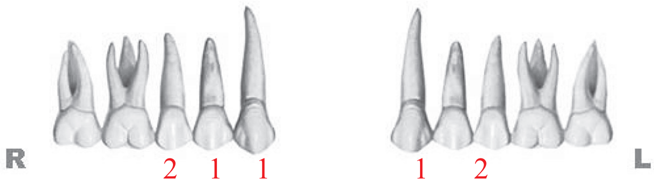
0





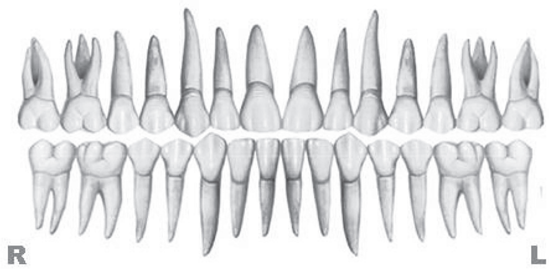
Occlusal Relationships

7



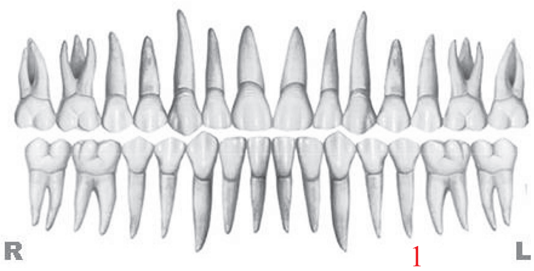
Interproximal Contacts

0



Root Angulation

1



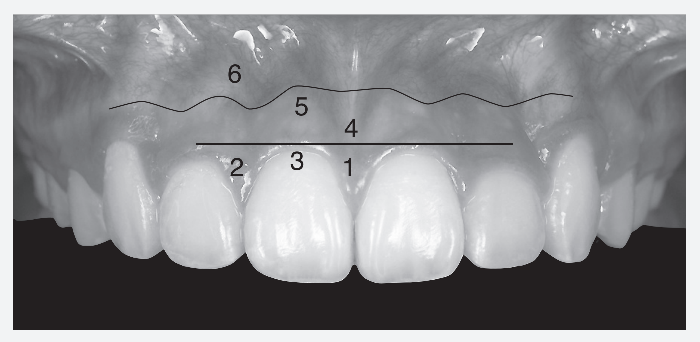
INSTRUCTIONS: Place score beside each deficient tooth and enter total score for each parameter in the white box. Mark extracted teeth with "X". Second molars should be in occlusion.

IBOI Pink & White Esthetic Score

Total Score: = 5

1. Pink Esthetic Score

Total = 2

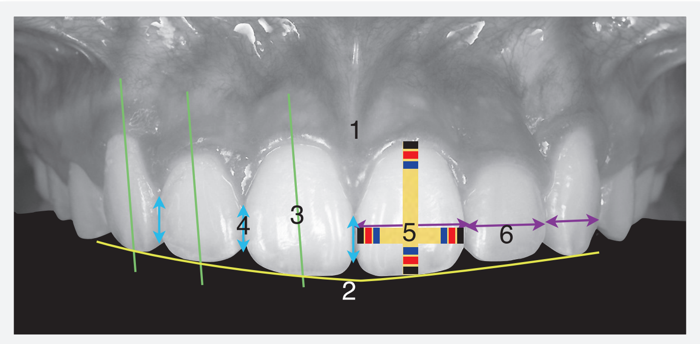


1. M & D Papillae	0	1	2
2. Keratinized Gingiva	0	1	2
3. Curvature of Gingival Margin	0	1	2
4. Level of Gingival Margin	0	1	2
5. Root Convexity (Torque)	0	1	2
6. Scar Formation	0	1	2

1. M & D Papilla	0	**(1)**	2
2. Keratinized Gingiva	**(0)**	1	2
3. Curvature of Gingival Margin	0	**(1)**	2
4. Level of Gingival Margin	**(0)**	1	2
5. Root Convexity (Torque)	**(0)**	1	2
6. Scar Formation	**(0)**	1	2

2. White Esthetic Score (for Micro-esthetics)

Total = 3



1. Midline	0	1	2
2. Incisor Curve	0	1	2
3. Axial Inclination (5°, 8°, 10°)	0	1	2
4. Contact Area (50%, 40%, 30%)	0	1	2
5. Tooth Proportion (1:0.8)	0	1	2
6. Tooth to Tooth Proportion	0	1	2

1. Midline	**(0)**	1	2
2. Incisor Curve	0	**(1)**	2
3. Axial Inclination (5°, 8°, 10°)	0	**(1)**	2
4. Contact Area (50%, 40%, 30%)	**(0)**	1	2
5. Tooth Proportion (1:0.8)	0	**(1)**	2
6. Tooth to Tooth Proportion	**(0)**	1	2