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Biological events related to corticotomy-facilitated orthodontics

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Abstract

Corticotomy-facilitated orthodontics is a clinical treatment modality comprising the application of conventional orthodontic forces combined with selective decortication of the alveolar process of the bone, which generates a localized process of bone remodeling (turnover) that enables accelerated orthodontic tooth movement. Compared with conventional orthodontic treatment, corticotomy-facilitated orthodontics is associated with reduced treatment time and reduces the frequency of apical external root resorption; however, this modality increases morbidity and financial costs. Although the clinical outcomes of corticotomy-facilitated orthodontics appear favorable, no results of evidence-based investigations of long-term outcomes are available in the literature, and the long-term effects of corticotomy-facilitated orthodontics on the teeth and periodontium are unclear. This narrative review discusses the biological events associated with corticotomy-facilitated orthodontics. Authoritative articles found in relevant databases were critically analyzed and the findings were integrated and incorporated in the text.

Keywords

Regional acceleratory phenomenon, corticotomy-facilitated orthodontics, bone turnover, orthodontic tooth movement, alveolar process, periodontium, accelerated bone remodeling

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Introduction

Corticotomy-facilitated orthodontics[1–4] is a surgical procedure that is used in conjunction with conventional fixed orthodontics or with clear aligners to accelerate load-induced orthodontic tooth movement.[5–8]

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The surgical procedure comprises reflecting full thickness flaps, selectively decorticating buccal and lingual bone between the teeth to be moved, placing bone allograft material, and closing and suturing the flaps (Figure 1).[1,5,9–12] This bone augmentation increases bone volume around the teeth to be moved, thereby minimizing fenestration, dehiscence, and gingival recession, ensuring long-term stability of the orthodontic outcome.[1,5,12–15]

Selective corticotomy limited to the buccal surface of the alveolar bone—with or without a mucoperiosteal flap—reduces operating time and postoperative discomfort, and can prevent damage to lingual tissues.[7,8,16] Furthermore, corticotomy by piezoelectric surgery, with or without a three-dimensional printed surgical guide based on computed tomography scan data, may increase precision while reducing bleeding and improving intraoperative visibility; this can reduce the risk of iatrogenic tissue damage.[8,16–19] Typically, active orthodontic treatment is initiated immediately after surgery, and the appliances are activated every 2 weeks thereafter.[5,9,11,12,14,20] Surgical selective decortication is followed by a physiological process that initially comprises predominantly catabolic bone remodeling (turnover), thereby supporting accelerated conventional orthodontic treatment.[1,5,21–24] Unless it causes damage to the dentition, the pattern of surgical decortication is of minimal significance; however, it must provide sufficient surgical stimulus to elicit localized intense, robust bone remodeling and healing.[2,5,25,26] The post-surgical course is typical of any extensive periodontal surgical procedure.[2,11]

Corticotomy-facilitated orthodontics is recommended for adults who prefer a

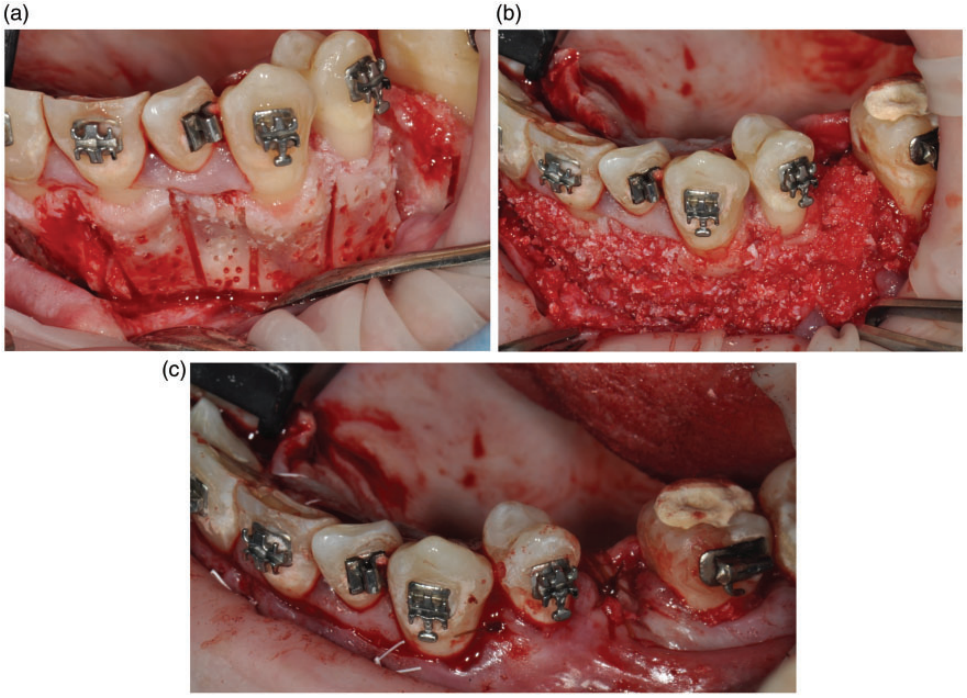

Figure 1. Surgical procedure for corticotomy-facilitated orthodontics. (a) Interdental slots and buccal cortical perforations, (b) Augmentation with bone allograft and (c) Repositioned and secured labial flap.

shorter course of orthodontic treatment and can afford the substantial costs; for the treatment of class I malocclusions with moderate or severe crowding in which extractions can be avoided because of the increased bone volume; for class II malocclusions in which required expansion or extraction can be avoided because of the increased bone volume; and for mild class III malocclusions.[5,11,12,14,27,28] Corticotomy-facilitated orthodontics should not be attempted in patients with metabolic bone disease, in those taking bisphosphonates, and those on long-term corticosteroid treatment.[4,28] Existing publications regarding corticotomy-facilitated orthodontics comprise case reports and low-to-moderate-quality evidence-based studies;[3,11,23,27] reportedly, there is no loss of periodontal bone volume, no increase in apical external root resorption, and no loss of tooth vitality in association with carefully performed corticotomy-facilitated orthodontics.[11,14,20,24,29,30] However, a recent animal study indicated that corticotomy-facilitated orthodontics may cause alveolar bone loss after tooth movement.[31] This review discusses the biological rationale of corticotomy-facilitated orthodontics.

Physiological bone remodeling

Bone remodeling or bone turnover is a physiological process comprising osteoclast-mediated bone resorption coupled with osteoblast-mediated bone formation. The balance between bone resorption and bone formation determines the ultimate bone mass at the site of bone remodeling.[32,33] Bone resorbing osteoclasts originate from the monocyte/macrophage lineage of hematopoietic stem cells in the bone marrow, whereas bone-depositing osteoblasts originate from multi-potential mesenchymal cells within the bone marrow stroma.[32,33] The differentiation, maturation, and functional activity of osteoclasts are mediated and regulated by several biological agents including the receptor activator of nuclear factor κB (RANK)/RANK ligand/osteoprotegerin signaling pathways, macrophage colony-stimulating factor, parathyroid hormone (PTH), estrogen, and various cytokines.[34,35] In contrast, the differentiation and maturation of osteoblasts are driven by osteogenic transcription factors (e.g., RUNX2 and osterix), which are activated by bone morphogenic proteins (BMPs) and the Wnt/β-catenin signaling pathways. The Wnt/β-catenin signaling pathways may participate in crosstalk with other intracellular signaling pathways (e.g., pathways activated by BMPs, nitrous oxide, or prostaglandins) to drive the process of osteogenesis.[32,34]

Bone sialoprotein, osteocalcin, alkaline phosphatase, and type one collagen are proteins essential for bone formation; they are synthesized by osteoblasts in the local microenvironment. The functional activity of osteoblasts is regulated by PTH, 1,25-dihydroxyvitamin D, and growth factors that include platelet-derived growth factor (PDGF), transforming growth factor β (TGF-β), and fibroblast growth factors (FGFs).[34] Within the three-dimensional lacunocanalicular network, osteocytes communicate with osteoblasts and other osteocytes via gap junctions involving the ends of their dendritic processes.[32] A gap junction is a channel that connects the cytoplasms of two adjacent cells, which allows the passage of ions, metabolites, and small signaling molecules. The functional activity of a gap junction is regulated by mechanical, chemical and electrical factors.[32]

In the bone microenvironment, cells conduct crosstalk with the extracellular matrix (ECM) through focal adhesion domains on their plasma membranes.[36] A focal adhesion is a multifunctional cellular structure comprising a complex network of transplasma membrane integrins and cytoplasmic proteins. Through focal adhesions, cells regulate the assembly of ECM proteins and ECM remodeling; importantly,

the ECM can regulate cell adhesion, migration, proliferation, differentiation, apoptosis, and biochemical cellular responses.[36] A stiff ECM induces multiple strains at the cellular focal adhesion domain, thus resulting in strong cell adhesion to the ECM; undifferentiated mesenchymal progenitor cells may differentiate into distinct lineages based on the stiffness characteristics of their microenvironmental ECM.[36] Physiological bone remodeling begins with osteoclastic bone resorption, characterized by the dissolution of inorganic crystalline apatite, followed by enzymatic degradation of the organic component with the release of biologically active agents (e.g., BMPs, FGFs, and TGF-β) from the organic matrix of the resorbed bone into the local microenvironment.[32,37] Subsequently, these biological agents mediate the proliferation and differentiation of osteoblast precursors, as well as the activation of osteoblasts that secrete non-collagenous proteins and collagen fibers, to form an organized matrix that subsequently undergoes mineralization, thereby forming new bone.[32,37]

Tooth movement relative to applied orthodontic forces

The movement of teeth in response to applied orthodontic force occurs in three overlapping stages:[36] the initial stage is characterized by tooth displacement in the periodontal ligament space within the bony socket. After 24 to 48 hours, a lag stage occurs, characterized by necrosis and hyalinization in the periodontal ligament and neighboring alveolar bone in response to the compressive stresses ahead of the moving tooth; this stage lasts 20 to 30 days. The hyalinized and necrotic tissues prevent further tooth movement; after the hyalinized tissue has been removed by macrophages and multi-nucleated giant cells, and the necrotic bone has been removed by undermining resorption, the postlag stage of orthodontic tooth movement can begin. In this last stage, bone remodeling occurs, comprising osteoclastic bone resorption in the compression zone ahead of the moving tooth, coupled with osteoblastic bone formation in the tension zone behind the moving tooth, supported by a vigorous process of angiogenesis; this constitutes the mechanism of continuous orthodontic tooth movement.[36]

Orthodontic forces applied to teeth generate complex mechanical loading patterns of compressive and tensile strains in the periodontium immediately surrounding the loaded teeth. These strains induce resident cells to release numerous active biological agents into the local microenvironment; the agents are differentially expressed around the mechanically loaded teeth, triggering an aseptic inflammatory response and local periodontal tissue remodeling.[2,36] Compressive strains in the periodontal ligament and alveolar bone stimulate the release of biological agents that induce osteoclastogenesis, thereby initiating osteoclast-induced bone resorption. In addition, tensile strains in the periodontal ligament and alveolar bone stimulate the release of osteogenic factors that increase the rate of differentiation of osteogenic progenitor cells into mature osteoblasts depositing osteoid that subsequently undergoes mineralization.[32] Thus, the process of continuous bone remodeling comprises osteoclast-mediated bone resorption in the compressive zone, coupled with osteoblast-mediated bone formation in the tension zone, enabling progressive tooth movement in response to mechanical loading.[32,36] Other factors that influence tooth movement in response to orthodontic forces include the magnitude, type (continuous or intermittent), direction, and duration of the applied force; the nature of the tooth movement (intrusion, extrusion, tipping, or bodily movement); the overall duration

of orthodontic treatment; the number, shape, length, and angulation of the tooth roots; and the structure, density, and rate of turnover of the bone supporting the teeth.[22,36,38]

Regional acceleration of bone remodeling in corticotomy-facilitated orthodontics

The literature suggests that regional acceleration of bone remodeling following deliberate corticotomy of the alveolar process, with the intent of accelerating orthodontic tooth movement, is a special phenomenon.[39–41] Indeed, the underlying biological process is similar to that demonstrated during the healing of damaged bone. The "phenomenon" of local surgical bone remodeling begins with the release by resident cells of a burst of biological mediators into the microenvironment, thus initiating and promoting sequential bone remodeling and tissue healing.[39–41] In the context of selective decortication, this burst of biological mediators induces local bone remodeling, as in typical bone injuries; this comprises an acute inflammatory phase and intense osteoclastic bone resorption, which manifests as transient local osteopenia[2,20,21,42] combined with decreased osteoblastic bone formation.[43] The osteopenic bone is more susceptible to orthodontic tooth movement than normal bone.[28] Selective decorticotomy-induced transient osteopenia is followed by a process of osteoblastic bone formation.[2,44] During this induced sequential osteoclastic/osteoblastic process, orthodontic tooth movement can occur more rapidly than it would under standard treatment conditions.[5,21] Thus, the moderate orthodontic load-induced sterile inflammatory process ordinarily associated with orthodontic tooth movement is superimposed on the selective decorticotomy-induced sequence of bone remodeling, resulting in accelerated tooth movement.[14,20,36] Following the above-described surgically induced insult to cortical bone, bone turnover begins within a few days, peaks at 1 to 2 months after surgery, and subsides within 6 months when healing is complete.[1,9,41] In the context of corticotomy-facilitated orthodontics, the process of bone healing and remodeling lasts approximately 4 to 6 months;[3,12,25,45] accelerated orthodontic tooth movement can only occur during this period. Therefore, the activation of orthodontic appliances should commence within 2 weeks after selective decortication, and the orthodontic appliances should be reactivated every 2 weeks, enabling full utilization of the relatively short period of bone remodeling and healing.[11] If the orthodontic appliances are not activated during this period, the benefit of the corticotomy procedure will be lost.

To the best of our knowledge, there are no reports in the literature documenting the early biological events that occur in response to the traumatic stimulation of bone remodeling caused by selective decortication. Early biological events are likely to include the formation of a transient fibrin-based blood clot; organization and stabilization of the clot; release of vasoactive, vasoregenerative, and immuno-inflammatory agents; formation of an angiogenic connective tissue; and bone resorption and formation.[37,46–48] The structural matrix of the blood clot serves as an osteoconductive medium for mesenchymal progenitor cells from the bone marrow and for pluripotent pericytes from adjacent small blood vessels. These progenitor cells eventually differentiate and mature into endothelial cells and bone-forming osteoblasts, thus gradually replacing the blood clot with osteoid that is subsequently mineralized.[37,46,47] These well-known early healing events are universal to all bone injuries and have been described in the context of periodontal osteotomies and crown lengthening procedures that are relatively similar to selective decortication.[37,46,47]

Following damage to bone, platelets, macrophages, and monocytes in the altered local microenvironment produce and release growth factors and cytokines (e.g., PDGF, FGF, insulin-like growth factor, vascular endothelial growth factor, BMPs, interleukin-1β, and tumor necrosis factor-α), which recruit and stimulate the differentiation of osteoblastic, osteoclastic, and endothelial progenitor cells that subsequently carry out bone remodeling and angiogenesis.[37,46,49] Furthermore, growth factors and other biologically active agents are released from injured blood vessels and demineralized bone matrix; together, these accelerate the recruitment and differentiation of osteoclast and osteoblast precursors, thereby intensifying the process of bone remodeling.[32,46,48] Thus, the "phenomenon" of regional acceleration of bone remodeling following selective decortication is likely to be characterized by robust release of biologically active agents and recruitment of osteoclastogenic and osteogenic cells; these factors induce increased bone turnover, which allows more rapid orthodontic tooth movement.[20,21] In response to selective decortication, there is also likely to be an immediate reduction in local blood flow in the bone due to damage to the blood vessels, as well as disruption of the three-dimensional lacunocanalicular system within which osteocytes and their dendritic processes are bathed in extracellular fluid.[32,48] Damage to this lacunocanalicular network, including altered fluid flow, may activate intracellular molecular signaling pathways within osteocytes, thus triggering the secretion of biological mediators (e.g., nitric oxide, prostaglandin E_2, and TGF-β) that can mediate bone remodeling and contributing to the "phenomenon" of regional acceleration of bone remodeling.[32]

In relation to corticotomy-facilitated orthodontics, the generation of mucoperiosteal flaps[41] and the selective decortication of alveolar bone processes can together initiate bone remodeling in the immediate vicinity of the corticotomy.[20,21,47,50] Following surgical damage to the cortical bone and the adjacent spongiosa of the alveolar process, the periodontal ligament becomes hyperemic, edematous, infiltrated with acute inflammatory cells, and physically widened. The resulting modulation of the viscoelastic properties of both bone and periodontal ligament, as well as the release of matrix metalloproteinases and other catabolic agents, disrupts the integrity of the extracellular matrix and increases the elasticity of the bone, thus favoring orthodontic tooth movement.[20,21,47,50] Because of the localized osteopenia and increased bone remodeling caused by selective decortication of the alveolar process, the formation of hyalinized tissue is minimized, which may shorten the lag stage and permit more rapid orthodontic tooth movement.[2,3,21,22,24] Compared with conventional orthodontic treatment, corticotomy-facilitated orthodontics is reportedly associated with both diminished frequency and magnitude of apical external root resorption.[9,23] This may be related to the reduced formation and more rapid removal of necrotic and hyalinized tissues ahead of the moving tooth, and may contribute to the overall decrease in the length of active orthodontic treatment.[27]

Conclusion

In corticotomy-facilitated orthodontics, the reflection of mucoperiosteal flaps and selective decortication of alveolar bone processes results in localized bone remodeling with inflammation-induced widening of the periodontal ligament space and transient local osteopenia. In addition, there is reduced formation and more rapid removal of necrotic and hyalinized tissues in the periodontal ligament and adjacent alveolar bone ahead of the rapidly moving tooth. However, prospective randomized controlled studies have not been performed to

compare conventional orthodontic treatment and corticotomy-facilitated orthodontics with respect to treatment time and the quality of orthodontic treatment outcome. Thus far, all available information is based on case reports and low-to-moderate evidence-based studies; studies with more robust evidence are needed to more conclusively evaluate the effects of this approach.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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References

1. Wilcko WM, Wilcko T, Bouquot JE, et al. Rapid orthodontics with alveolar reshaping: two case reports of decrowding. *Int J Periodontics Restorative Dent* 2001; 21: 9–19.
2. Baloul SS, Gerstenfeld LC, Morgan EF, et al. Mechanism of action and morphologic changes in the alveolar bone in response to selective alveolar decortication-facilitated tooth movement. *Am J Orthod Dentofacial Orthop* 2011; 139: S83–S101.
3. Mathews DP and Kokich VG. Accelerating tooth movement: the case against corticotomy-induced orthodontics. *Am J Orthod Dentofacial Orthop* 2013; 144: 5–13.
4. Zimmo N, Saleh MH, Mandelaris GA, et al. Corticotomy-accelerated orthodontics: a comprehensive review and update. *Compend Contin Educ Dent* 2017; 38: 17–26.
5. Wilcko W and Wilcko MT. Accelerating tooth movement: the case for corticotomy-induced orthodontics. *Am J Orthod Dentofacial Orthop* 2013; 144: 4–12.
6. Kalemaj Z, Debernard IC and Buti J. Efficacy of surgical and non-surgical interventions on accelerating orthodontic tooth movement: a systematic review. *Eur J Oral Implantol* 2015; 8: 9–24.
7. Cassetta M, Altieri F and Barbato E. The combined use of corticotomy and clear aligners: a case report. *Angle Orthod* 2016; 86: 862–870.
8. Cassetta M, Altieri F, Pandolfi S, et al. The combined use of computer-guided, minimally invasive, flapless corticotomy and clear aligners as a novel approach to moderate crowding: a case report. *Korean J Orthod* 2017; 47: 130–141.
9. Alghamdi AS. Corticotomy facilitated orthodontics: review of a technique. *Saudi Dent J* 2010; 22: 1–5.
10. Mulchin BJ, Newton CG, Baty JW, et al. The anti-cancer, anti-inflammatory and tuberculostatic activities of a series of 6,7-substituted-5,8-quinolinequinones. *Bioorg Med Chem* 2010; 18: 3238–3251.
11. Murphy KG, Wilcko MT, Wilcko WM, et al. Periodontal accelerated osteogenic orthodontics: a description of the surgical technique. *J Oral Maxillofac Surg* 2009; 67: 2160–2166.
12. Einy S, Horwitz J and Aizenbud D. Wilckodontics - An alternative adult orthodontic treatment method: rational and application. *Alpha Omegan* 2011; 104: 102–111.
13. Wilcko MT, Wilcko WM, Pulver JJ, et al. Accelerated osteogenic orthodontics technique: a 1-stage surgically facilitated rapid orthodontic technique with alveolar augmentation. *J Oral Maxillofac Surg* 2009; 67: 2149–2159.
14. Wilcko MT, Wilcko WM and Bissada NF. An evidence-based analysis of periodontally accelerated orthodontic and osteogenic techniques: a synthesis of scientific perspectives. *Semin Orthod* 2008; 14: 305–316.
15. Bhattacharya P, Bhattacharya H, Anjum A, et al. Assessment of corticotomy facilitated tooth movement and changes in alveolar bone thickness – a CT scan study. *J Clin Diagn Res* 2014; 8: ZC26–ZC30.
16. Cassetta M, Giansanti M, Di Mambro A, et al. Minimally invasive corticotomy in orthodontics using a three-dimensional printed CAD/CAM surgical guide. *Int J Oral Maxillofac Surg* 2016; 45: 1059–1064.
17. Dibart S, Sebaoun JD and Surmenian J. Piezocision: a minimally invasive, periodontally accelerated orthodontic tooth

movement procedure. *Compend Contin Educ Dent* 2009; 30: 342–350.
18. Dibart S, Surmenian J, Sebaoun JD, et al. Rapid treatment of Class II malocclusion with piezocision: two case reports. *Int J Periodontics Restorative Dent* 2010; 30: 487–493.
19. Milano F, Dibart S, Montesani L, et al. Computer-guided surgery using the piezocision technique. *Int J Periodontics Restorative Dent* 2014; 34: 523–529.
20. Sebaoun JD, Kantarci A, Turner JW, et al. Modeling of trabecular bone and lamina dura following selective alveolar decortication in rats. *J Periodontol* 2008; 79: 1679–1688.
21. Kim SJ, Park YG and Kang SG. Effects of corticision on paradental remodeling in orthodontic tooth movement. *Angle Orthod* 2009; 79: 284–291.
22. Iino S, Sakoda S, Ito G, et al. Acceleration of orthodontic tooth movement by alveolar corticotomy in the dog. *Am J Orthod Dentofacial Orthop* 2007; 131: 448.e1–448.e8.
23. Patterson BM, Dalci O, Darendeliler MA, et al. Corticotomies and orthodontic tooth movement: a systematic review. *J Oral Maxillofac Surg* 2016; 74: 453–473.
24. Liem AM, Hoogeveen EJ, Jansma J, et al. Surgically facilitated experimental movement of teeth: systematic review. *Br J Oral Maxillofac Surg* 2015; 53: 491–506.
25. Amit G, Jps K, Pankaj B, et al. Periodontally accelerated osteogenic orthodontics (PAOO) - a review. *J Clin Exp Dent* 2012; 4: e292–e296.
26. Shih MS and Norrdin RW. Regional acceleration of remodeling during healing of bone defects in beagles of various ages. *Bone* 1985; 6: 377–379.
27. Hoogeveen EJ, Jansma J and Ren Y. Surgically facilitated orthodontic treatment: a systematic review. *Am J Orthod Dentofacial Orthop* 2014; 145: S51–S64.
28. Gil APS, Haas OL Jr, Mendez-Manjon I, et al. Alveolar corticotomies for accelerated orthodontics: a systematic review. *J Craniomaxillofac Surg* 2018; 46: 438–445.
29. Long H, Pyakurel U, Wang Y, et al. Interventions for accelerating orthodontic tooth movement: a systematic review. *Angle Orthod* 2013; 83: 164–171.
30. Medeiros RB, Pires FR, Kantarci A, et al. Tissue repair after selective alveolar corticotomy in orthodontic patients: a preliminary study. *Angle Orthod* 2018; 88: 179–186.
31. Kurohama T, Hotokezaka H, Hashimoto M, et al. Increasing the amount of corticotomy does not affect orthodontic tooth movement or root resorption, but accelerates alveolar bone resorption in rats. *Eur J Orthod* 2017; 39: 277–286.
32. Feller L, Khammissa RA, Schechter I, et al. Biological events in periodontal ligament and alveolar bone associated with application of orthodontic forces. *Scientific World J* 2015; 2015: 876509.
33. Dibart S, Alasmari A, Zanni O, et al. Effect of corticotomies with different instruments on cranial bone biology using an ex vivo calvarial bone organ culture model system. *Int J Periodontics Restorative Dent* 2016; 36: s123–s136.
34. Feller L, Kramer B and Lemmer J. A short account of metastatic bone disease. *Cancer Cell Int* 2011; 11: 24.
35. Feller L, Khammissa RA, Thomadakis G, et al. Apical external root resorption and repair in orthodontic tooth movement: biological events. *Biomed Res Int* 2016; 2016: 4864195.
36. Feller L, Khammissa RA, Schechter I, et al. Periodontal biological events associated with orthodontic tooth movement: the biomechanics of the cytoskeleton and the extracellular matrix. *Scientific World J* 2015; 2015: 894123.
37. Feller L, Jadwat Y, Khammissa RA, et al. Cellular responses evoked by different surface characteristics of intraosseous titanium implants. *Biomed Res Int* 2015; 2015: 171945.
38. Verna C, Dalstra M and Melsen B. The rate and the type of orthodontic tooth movement is influenced by bone turnover in a rat model. *Eur J Orthod* 2000; 22: 343–352.
39. Frost MH. The biology of fracture healing: an overview for clinicians. Part 1. *Clin Orthodont* 1989; 248: 283–293.
40. Frost MH. The biology of fracture healing: an overview for clinicians. Part II. *Clin Orthodont* 1989; 249: 294–309.

41. Yaffe A, Fine N and Binderman I. Regional accelerated phenomenon in the mandible following mucoperiosteal flap surgery. *J Periodontol* 1994; 65: 79–83.
42. McBride MD, Campbell PM, Opperman LA, et al. How does the amount of surgical insult affect bone around moving teeth? *Am J Orthod Dentofacial Orthop* 2014; 145: S92–S99.
43. Pfeilschifter J, Wuster C, Vogel M, et al. Inflammation-mediated osteopenia (IMO) during acute inflammation in rats is due to a transient inhibition of bone formation. *Calcif Tissue Int* 1987; 41: 321–325.
44. Wang L, Lee W, Lei DL, et al. Tissue responses in corticotomy- and osteotomy-assisted tooth movements in rats: histology and immunostaining. *Am J Orthod Dentofacial Orthop* 2009; 136: 770. e1–770.e11.
45. Kaipatur N, Major P, Stevenson T, et al. Impact of selective alveolar decortication on bisphosphonate burdened alveolar bone during orthodontic tooth movement. *Arch Oral Biol* 2015; 60: 1681–1689.
46. Feller L, Chandran R, Khammissa RA, et al. Osseointegration: biological events in relation to characteristics of the implant surface. *SADJ* 2014; 69: 112–117.
47. Feller L and Lemmer J. Tooth mobility after periodontal surgery. *SADJ* 2004; 59: 407–411.
48. Feller L, Jadwat Y, Chandran R, et al. Radiolucent inflammatory implant periapical lesions: a review of the literature. *Implant Dent* 2014; 23: 745–752.
49. Kanno T, Takahashi T, Tsujisawa T, et al. Platelet-rich plasma enhances human osteoblast-like cell proliferation and differentiation. *J Oral Maxillofac Surg* 2005; 63: 362–369.
50. Mostafa YA, Mohamed Salah Fayed M, Mehanni S, et al. Comparison of corticotomy-facilitated vs standard tooth-movement techniques in dogs with miniscrews as anchor units. *Am J Orthod Dentofacial Orthop* 2009; 136: 570–577.