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To the memory of our fathers

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The Ortho-Perio Patient
Clinical Evidence & Therapeutic Guidelines

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PREFACE

This book gathers the available evidence and offers a thorough and substantiated discussion of treatment for the ortho-perio patient. With contributions from leading scholars and clinicians all over the world, the book systematically analyzes the interaction of the two specialties from both scientific and clinical perspectives. It includes an introductory section where the fundamentals of oral physiology with relation to orthodontic-periodontic interactions are analyzed, including bone biology in adult patients and the basics of oral microbiota attachment and pellicle organization on materials. The subsequent section on periodontal considerations for the orthodontic patient covers the periodontal examination of the orthodontic patient, aspects of gingival recession and grafting, clinical attachment level, orthodontic-periodontic effects of expansion, surgical crown lengthening, and ectopic canine eruption. The last section on orthodontic considerations for the periodontic patient includes chapters on clinical attachment level, the biomechanics in compromised periodontal tissues, and principles of orthodontic treatment in periodontic patients.

The evidence provided in this book and the case series portraying the adjunct role of each specialty in the treatment planning of patients with periodontal or orthodontic needs furnish important theoretical and clinical information as well as practical guidelines to improve the treatment outcome of therapeutic protocols involving ortho-perio interventions. Thus, the book not only acts as a reference book on the topic but, more importantly, includes substantiated guidelines and validated treatment approaches, which aid the practicing clinician in individualized treatment planning. It is therefore appropriate for academics, clinicians, and postgraduate students in orthodontics and periodontology and could be used as an accompanying text for the standard seminar of specialty training in dental schools.

It may be worth noting that this book was conceived 7 years ago with an additional editor, the late Dr Vincent G. Kokich, who was instrumental in developing the scope of the text and undertook the contribution of several chapters. With his sudden and tragic passing in 2013, the project had to be re-formed, and chapters were assigned to leading clinicians and academics in the field. The editors, who were fortunate to get acquainted with his brilliant clinical expertise and visionary academic and research service, return only a fragment of the debt they owe him for the collaboration they enjoyed by acknowledging his legendary path in the field.

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SECTION I

Fundamentals of Oral Physiology

CHAPTER 1

Bone Biology and Response to Loading in Adult Orthodontic Patients

Dimitrios Konstantonis

Orthodontic movement is achieved due to the ability of alveolar bone to remodel.[1–3] The bone-remodeling process is controlled by an equilibrium between bone formation in the areas of pressure and bone resorption in the areas of tension as the teeth respond to mechanical forces during treatment. The main mediators of mechanical stress to the alveolar bone are the cells of the periodontal ligament (PDL). The PDL consists of a heterogenous cell population comprised by nondifferentiated multipotent mesenchymal cells as well as fibroblasts. The periodontal fibroblasts have the capacity to differentiate into osteoblasts in response to various external mechanical stimuli. This feature of the PDL fibroblasts plays a key role in the regeneration of the alveolar bone and the acceleration of orthodontic movement.

Current research provides scientific data that elucidates the molecular response of the human PDL fibroblasts after mechanical stimulation.[4–6] Integrins at focal adhesions function both as cell-adhesion molecules and as intracellular signal receptors. Upon stress application, a series of biochemical responses expressed via signaling pathway cascades, involving GTPases (enzymes that bind and hydrolyze guanosine triphosphate [GTP]), mitogen-activated protein kinases (MAPKs), and transcription factors like activator protein 1 (AP-1) and runt-related transcription factor 2 (Runx2), stimulate DNA binding potential to specific genes, thus leading to osteoblast differentiation. Consecutively, the activation of cytokines like receptor activator of nuclear factor κB ligand (RANKL) and osteoprotegerin (OPG) regulates osteoclast activity. Despite the importance of these biologic phenomena, the number of reports on the molecular response of human periodontal fibroblasts after mechanical stimulation and on the subsequent activation of signaling pathways is limited.

Age has a considerable impact on the composition and integrity of the periodontal tissues and, according to clinical beliefs and research studies, plays a significant role

in the rate of orthodontic tooth movement.[7–12] Apart from the observed cellular morphologic changes, the levels of proliferation and differentiation of alveolar bone and PDL cells also diminish with age. At a molecular level, aged human PDL fibroblasts show alterations in signal transduction pathways, leading to a catabolic phenotype displayed by a significantly decreased ability for osteoblastic differentiation, thus affecting tissue development and integrity.[13,14] Currently, the difference in molecular response to orthodontic load among different age groups is considered of utmost importance. Still, the clinical application of biologic modifiers to expedite or decrease the rate of orthodontic tooth movement is underway.

Biology of Tooth Movement

ALVEOLAR BONE

The alveolar bone is the thickened ridge of the jaw that contains the tooth sockets, in which the teeth are embedded. The alveolar process contains a region of compact bone adjacent to the PDL called the *lamina dura*.[15] When viewed on radiographs, it is the uniformly radiopaque part, and it is attached to the cementum of the roots by the PDL. Although the lamina dura is often described as a solid wall, it is in fact a perforated construction through which the compressed fluids of the PDL can be expressed. The permeability of the lamina dura varies depending on its position in the alveolar process and the age of the patient. Under the lamina dura lies the cancellous bone, which appears on radiographs as less bright. The tiny spicules of bone crisscrossing the cancellous bone are the trabeculae and make the bone look spongy. These trabeculae separate the cancellous bone into tiny compartments, which contain the blood-producing marrow.

The alveolar bone or process is divided into the alveolar bone proper and the supporting alveolar bone. Microscopically, both the alveolar bone proper and the supporting alveolar bone have the same components: fibers, cells, intercellular substances, nerves, blood vessels, and lymphatics. The alveolar bone is comprised of calcified organic extracellular matrix containing bone cells. The organic matrix is comprised of collagen fibers and ground substance. The collagen fibers are produced by osteoblasts and consist of 95% collagen type I and 5% collagen type III. The ground substance contains the collagen fibers, glycosaminoglycans, and other proteins. The noncalcified organic matrix is called *osteoid*. Calcification of the alveolar bone occurs by deposition of carbonated hydroxyapatite crystals around the osteoid and between the collagen fibers. Noncollagenous proteins like osteocalcin and osteonectin also participate in the calcification process.

The cells of the alveolar bone are divided into four types[16]:

- Osteoblasts: Specialized mesenchymal cells forming bone
- Osteoclasts: Multinucleated cells responsible for bone resorption
- Lining cells: Undifferentiated osteoblastic cells
- Osteocytes: Osteoblasts located within the compact bone

The alveolar bone is an extremely important part of the dentoalveolar device and is the final recipient of forces during mastication and orthodontic treatment. The reaction to these forces include bending of the alveolar socket and subsequent bone resorption and deposition, which depends on the time, magnitude, and duration of the force. Although the biologic mechanisms underlying these cellular changes are not fully known, it seems they resemble those of the body frame, where mechanical loading has osteogenic effects. Despite the similarities between the alveolar and compact bone, the different response to mechanical loading is attributed to the presence of the PDL, a tissue full of undifferentiated mesenchymal cells, which serves as the means through which the signal is transmitted to the alveolar bone.

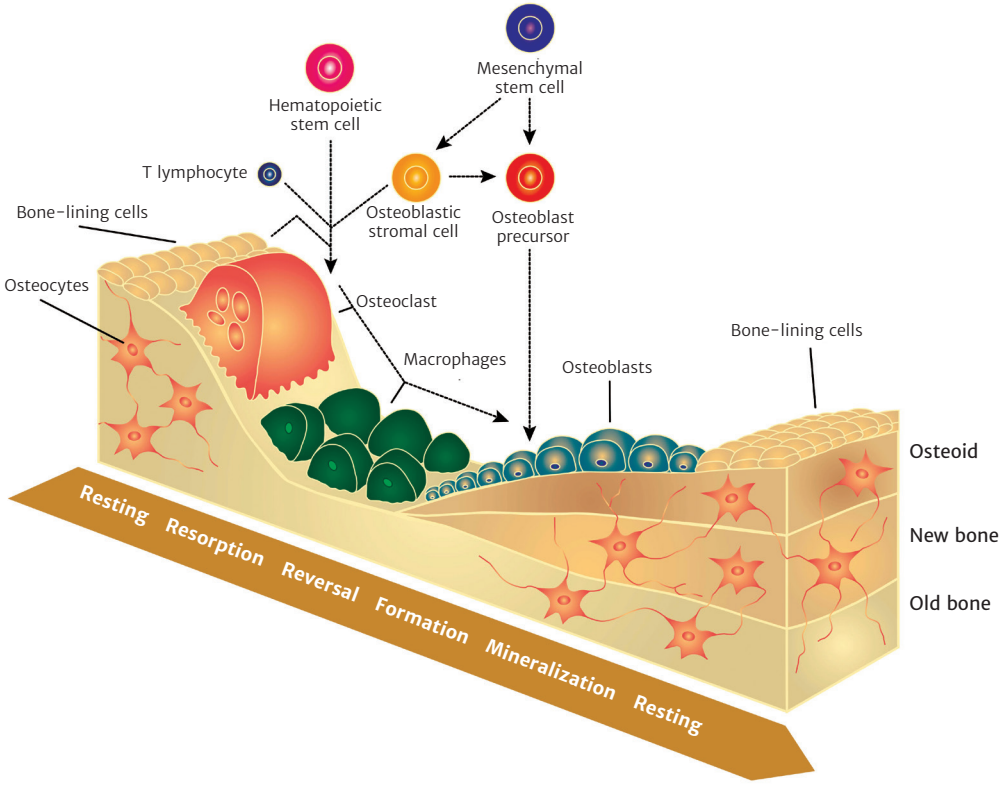


Fig 1-1 The basic multicellular unit. Cells are stimulated by a variety of signals in order to start bone remodeling. In the model suggested here, the hematopoietic precursors interact with cells of the osteoblast lineage and along with inflammatory cells (mainly T cells) trigger osteoclast activation. After osteoclast formation, a brief resorption phase followed by a reversal phase begins. In the reversal phase, the bone surface is covered by mononuclear cells. The formation phase lasts considerably longer and implicates the production of matrix by the osteoblasts. Subsequently, the osteoblasts become flat lining cells that are embedded in the bone as osteocytes or go through apoptosis. Through this mechanism, approximately 10% of the skeletal mass of an adult is remodeled each year.

CONTEMPORARY DATA ON BONE BIOLOGY

Recent studies report interesting findings on bone biology. Bone morphogenetic proteins (BMPs) are a group of growth factors, also known as *cytokines*, that act on undifferentiated mesenchymal cells to induce osteogenic cell lines and, with the mediation of growth and systemic factors, lead to cell proliferation, osteoblast and chondrocyte differentiation, and subsequently bone and cartilage production.[17] Osteoblasts derive from nonhematopoietic sites of bone marrow that contain groups of fibroblast cells, which have the potential to differentiate into bone-type cells known as *mesenchymal stem cells*, *skeletal stem cells derived from bone marrow*, *bone marrow stromal cells*, and *multipotent mesenchymal stromal cells*.[18]

Bone is constantly being created and replaced in a process known as *remodeling*. This ongoing turnover of bone is a process of resorption followed by replacement of bone that results in little change in shape. This is accomplished through osteoblasts and osteoclasts. Cells are stimulated by a variety of signals, and together they are referred to as a *remodeling unit*. Approximately 10% of the skeletal mass of an adult is remodeled each year.[19] The basic multicellular unit (BMU) is a wandering group of cells that dissolves a portion of the surface of the bone and then fills it by new bone deposition[20] (Fig 1-1).

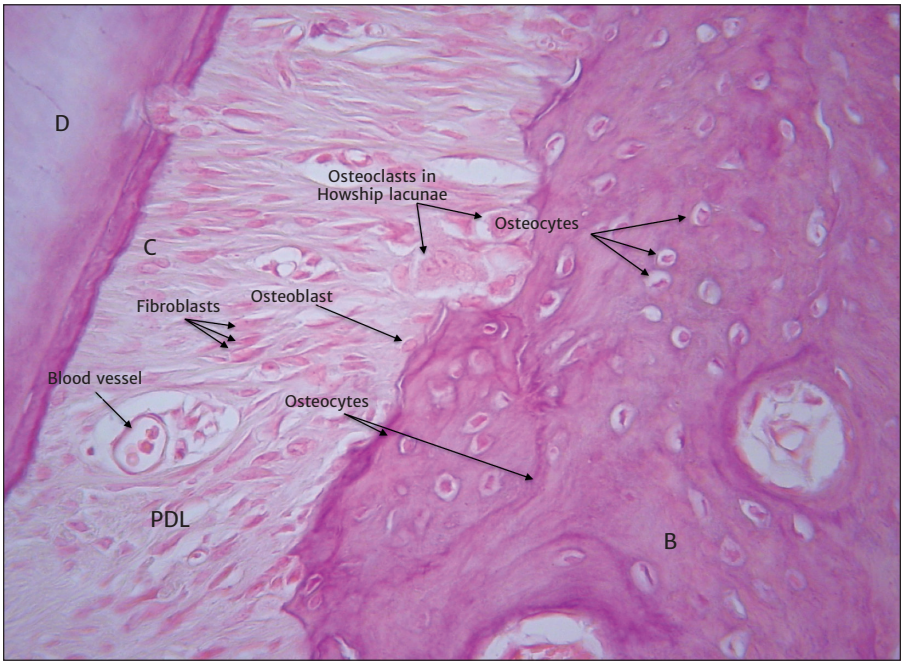


Fig 1-2 Histologic cross section through a PDL under mechanical load. D, dentin; C, cementum; B, alveolar bone. (Courtesy of Dr K. Tosios, National and Kapodistrian University of Athens, Greece.)

The osteoblasts are dominant elements of the basic skeletal anatomical structure of the BMU. The BMU consists of bone-forming cells (osteoblasts, osteocytes, and bone-lining cells), bone-resorbing cells (osteoclasts), and their precursor cells and associated cells (endothelial, nerve cells).

The bone is deposited by osteoblasts producing matrix (collagen) and two further noncollagenous proteins: osteocalcin and osteonectin. Activation of the bone resorption process is initiated by the preosteoclasts, which are induced and differentiated under the influence of cytokines and growth factors into active mature osteoclasts. Osteoclasts break down old bone and bring the end of the resorption process[21] (Fig 1-2).

The cycle of bone remodeling starts with the regulation of osteoblast growth and differentiation, which is accomplished through the osteogenic signaling pathways. A hierarchy of sequential expression of transcription factors results in the production of bone. Undifferentiated multipotent mesenchymal cells progressively differentiate into mature active osteoblasts expressing osteoblastic phenotypic genes and then transform into osteocytes within the bone matrix or undergo apoptosis.

The following three families of growth factors show a considerable impact on osteoblastic activity[22]:

- Transforming growth factor βs (TGF-βs)
- Insulinlike growth factors
- BMPs

Growth factors act primarily through specialized intracellular interactions and interactions with hormones or transcription factors. They also act in response to the activity of glucocorticoids, parathyroid hormone, prostaglandin, sex hormones, and more. The BMPs induce the production of bone in vivo by promoting the expression of Runx2 in mesenchymal osteoprogenitor and osteoblastic cells and the expression of Osterix in osteoblastic cells. The TGF-βs play a crucial role in osteoblast differentiation by promoting bone formation through the upregulation of Runx2 while simultaneously reducing the levels of transcription factors that lead the cells to adipogenesis.

Table 1-1 Clinical deformities resulting from transcription factor mutation

Transcription factor	*Deformity*
Parathyroid hormone–related protein (PTHrP)	Fatal chondroplasia
Sox5, Sox6, Sox9	Campomelic dysplasia
Fibroblast growth factor receptor 3 (FGFR3)	Achondroplasia
Runx2/3	Cleidocranial dysplasia

The absence or dysfunction of several transcription factors involved in bone metabolism leads to severe clinical deformities[23] (Table 1-1).

RUNX2 TRANSCRIPTION FACTOR

Runx2, also known as *core-binding factor subunit α1* (CBF-α1), is a protein that in humans is encoded by the *RUNX2* gene.[24] Runx2 is a key transcription factor associated with osteoblast differentiation. This protein is a member of the Runx family of transcription factors and has a Runt DNA-binding domain. It is essential for osteoblastic differentiation in both intramembranous and endochondral ossification and acts as a scaffold for nucleic acids and regulatory factors involved in skeletal gene expression. The protein can bind DNA either as a monomer or, with more affinity, as a subunit of a heterodimeric complex. Transcript variants of the gene that encode different protein isoforms result from the use of alternate promoters as well as alternate splicing. Differences in Runx2 are hypothesized to be the cause of the skeletal differences (eg, different skull shape and chest shape) between modern humans and early humans such as Neanderthals.[25]

Mutations in this gene in humans have been associated with the bone development disorder cleidocranial dysplasia[26,27] (Fig 1-3; see also Table 1-1). Other diseases associated with Runx2 include metaphyseal dysplasia with maxillary hypoplasia with or without brachydactyly. Among its related pathways are endochondral ossification and the fibroblast growth factor signaling pathway.[28] Deactivation of the gene in transgenic mice (*RUNX2-/-*) leads to complete lack of intramembranous and endochondral calcification due to lack of mature osteoblasts.[29] The mesenchymal cells in these animals retain the ability to further differentiate into adipocytes and chondrocytes.

PERIODONTAL LIGAMENT

The PDL is a dense fibrous connective tissue 0.15 to 0.40 mm thick that occupies the space between the root of the tooth and the alveolus.[16] The narrowest area of the PDL is at the midroot (fulcrum). The region at the alveolar crest is the widest area, followed by the apical region. The width is generally reduced in nonfunctional teeth and unerupted teeth, whereas it increases in teeth subjected to occlusal load within the physiologic limits and in primary teeth.

Histologically it presents a heterogenous, highly cellular structure comprised of a thick extracellular matrix with incorporated fibers arranged along the root[30] (Fig 1-4). The tooth does not come in direct contact with the alveolar bone but recedes into the alveolus, where it is retained by the PDL fibers.[31] These fibers act as shock absorbers and help the

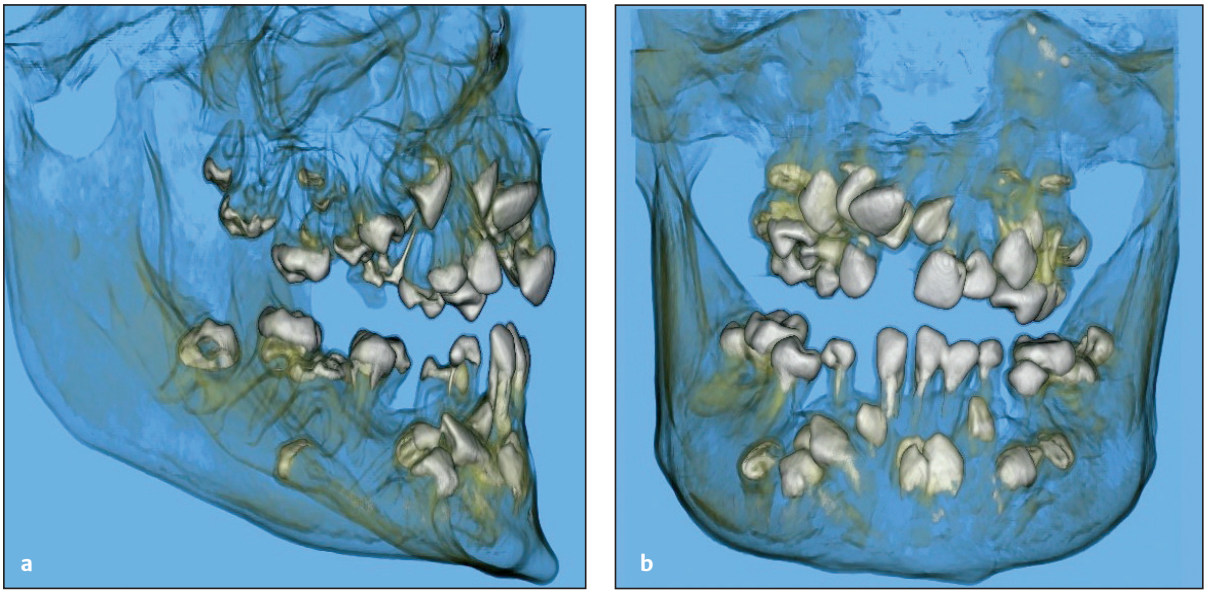


Fig 1-3 *(a and b)* Volume rendering image of cone beam computed tomography data of an adult male patient diagnosed with cleidocranial dysplasia.

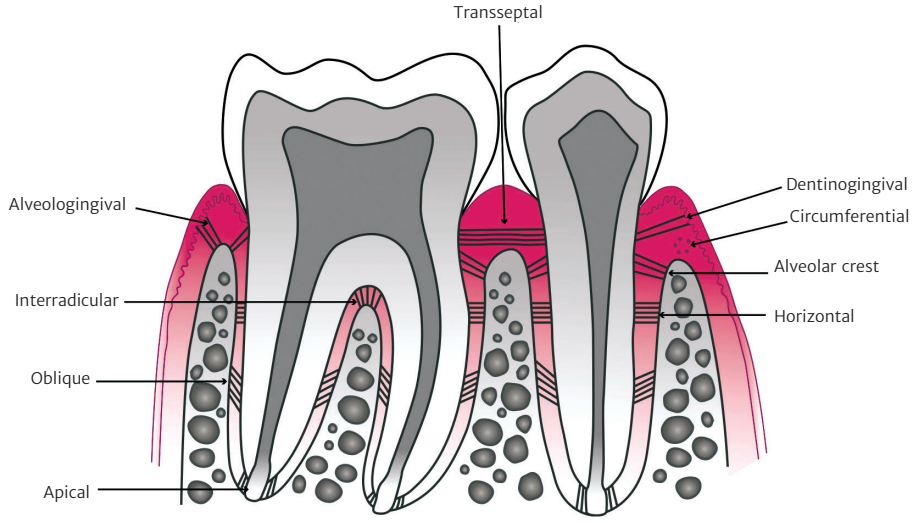


Fig 1-4 The PDL fibers are primarily composed of bundles of type I collagen fibrils. Their classification into several groups is made on the basis of their anatomical location. The principal fiber groups of the PDL are depicted here.

tooth withstand mastication forces and also respond to orthodontic load.

Like any other connective tissue, the PDL is composed of cells and extracellular components. The PDL cells comprise mainly fibroblasts (65%), which derive from undifferentiated mesenchymal cells with the ability to differentiate to preosteoblasts and cementoblasts; they produce collagen types I, II, and V. Additionally, they show similar characteristics to osteoblasts, like production of alkaline phosphatase (ALP) and osteocalcin, and response to 1,25 dihydroxyvitamin D3.

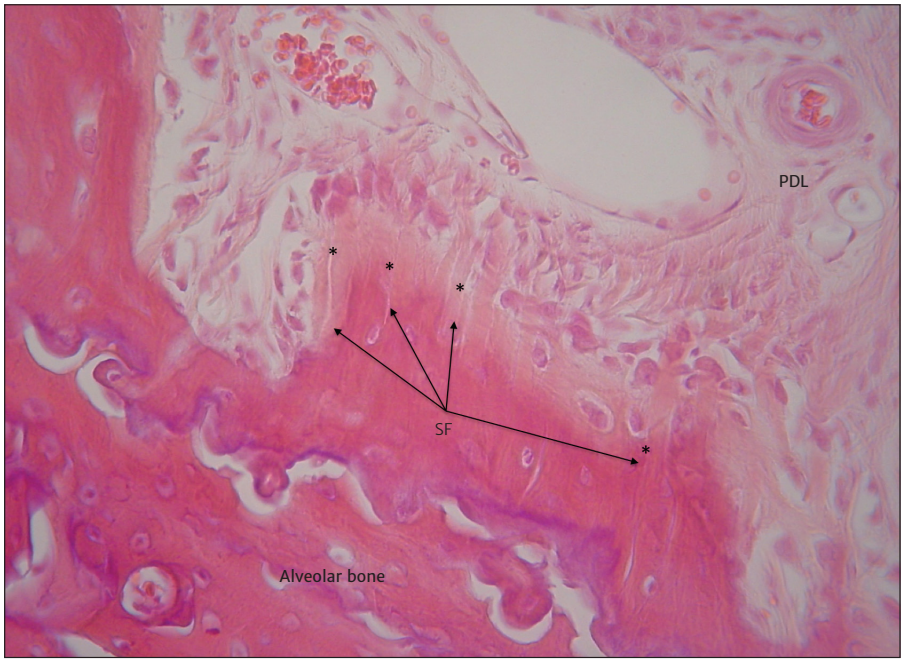


Fig 1-5 Higher magnification of the junction of the PDL with the bone. Sharpey fibers, which are the mineralized part of the thick fiber bundles (marked with an *), originate in the PDL and help anchor the tooth to the bone. In this histologic section, the mineralized bone (including the Sharpey fibers) appears magenta as compared to the purple color of the nonmineralized portions of the fibers. (Courtesy of Dr K. Tosios, National and Kapodistrian University of Athens, Greece.)

The possibility of differentiation of the PDL fibroblasts to preosteoblasts upon the application of orthodontic force plays an important role in bone remodeling.[32] Recent investigations report that the PDL is a major source of multipotent mesenchymal stromal cells that could be used for in vivo tissue regeneration such as cementum and the PDL itself.[33–37] The potential transplant of these cells, which may be detached with relative ease and then proliferate ex vivo, has significant therapeutic use on the restoration of periodontal breakdown in periodontic patients.

The rest of the PDL cells include cementoblasts, osteoblasts, osteoclasts, undifferentiated mesenchymal cells, and the epithelial rest cells of Malassez. The PDL cells play synthetic, resorptive, and defensive roles. They are also progenitor cells. The ground substance is a gel-like matrix that accounts for 65% of the PDL volume and comprises glycoproteins and proteoglycans. It contains 70% water and has a significant effect on the tooth's ability to sustain load. Cellular components like the collagen fibers are embedded within this matrix. The collagen fibers according to their location are divided into transseptal, alveolar crest, horizontal, interradicular, oblique, and apical. The PDL supports and protects the teeth within the alveolus with simultaneous sensory, nutritive, and formative functions.[31] The teeth are anchored into the alveolar process by Sharpey fibers, which are the terminal ends of the principal PDL fibers that insert into the cementum and the periosteum of the alveolar bone (Fig 1-5).

The integrity of the alveolar bone is also associated with the presence of the PDL. In extraction sites or in ankylosed teeth, the PDL is destroyed, and progressive absorption of the alveolar ridge occurs (Fig 1-6). The imbalance between osteoblasts and osteoclasts leads to degenerative bone activity. This is due to the reduction in the number of osteoblasts and the simultaneous increase in osteoclasts. In the continuous cycle of bone remodeling that takes place around the tooth alveolus, the PDL has a role of a continuous source of osteoblasts.

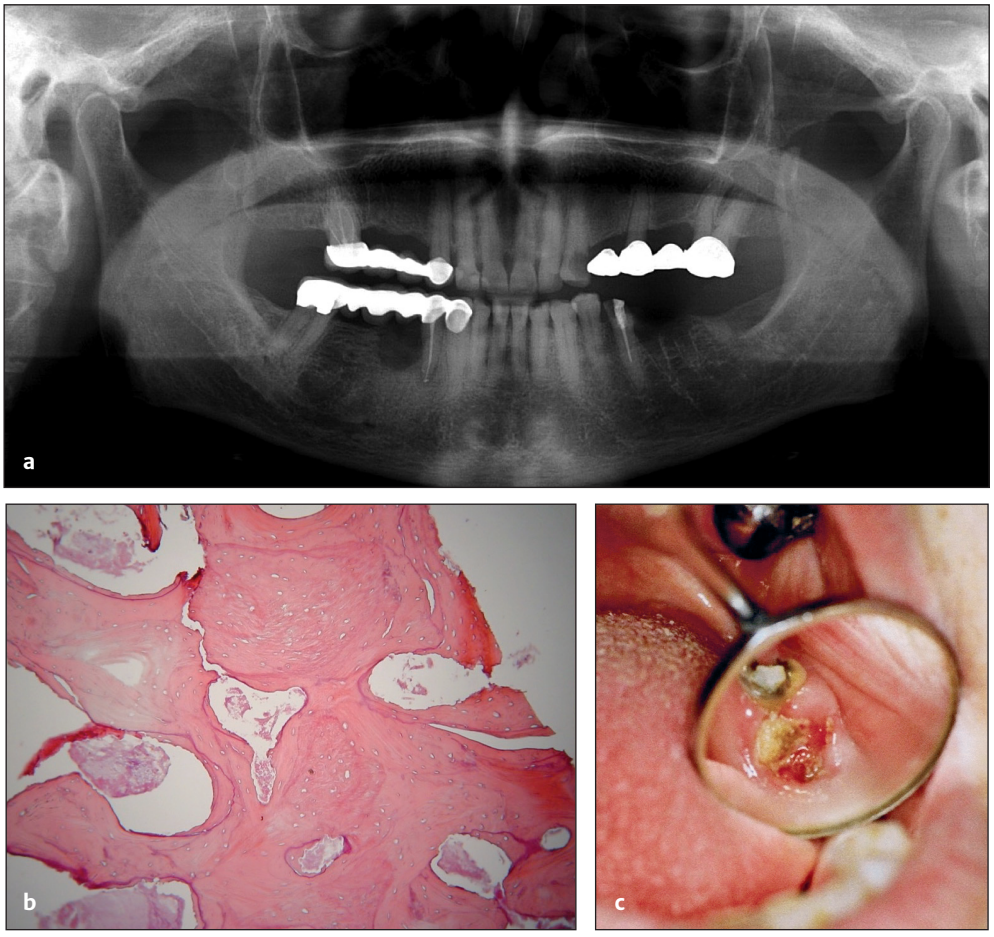


Fig 1-12 *(a to c)* Radiographic, histologic, and clinical images of a 65-year-old woman with mandibular osteonecrosis following extraction of the mandibular left first molar. The patient was undergoing treatment for osteoporosis with bisphosphonates. (Courtesy of Dr K. Tosios, National and Kapodistrian University of Athens, Greece.)

greater increase was observed in PGE2 signaling levels in mesenchymal stem cells from aged animals, most likely as a compensation for decreased ERK1/2 and NO signaling.[111]

Bisphosphonates are a synthetic class of pyrophosphate analogs that inhibit bone resorption by reducing osteoclast activity. They are commonly used as a medication for the prevention and therapy of osteoporosis and osteopenia but are also used to treat tumor diseases. Bisphosphonates have unique pharmacologic characteristics unlike those of any other drug group, and their half-life can be more than 10 years.[112] Because these drugs interfere with bone metabolism, they have a considerable impact on orthodontic treatment. The pharmacologic effects of these drugs, which can change bone physiology and interfere with osteoclastic resorption, could probably decrease the rate of orthodontic tooth movement and subsequently hinder treatment. Orthodontic treatment should begin after obtaining the patient's informed consent.[112] One important side effect is bisphosphonate-associated osteonecrosis of the jaws[113–118] (Fig 1-12). The severity of osteonecrosis is dependent on the type of bisphosphonate used as well as the dose, duration, and route of administration (intravenous or oral).

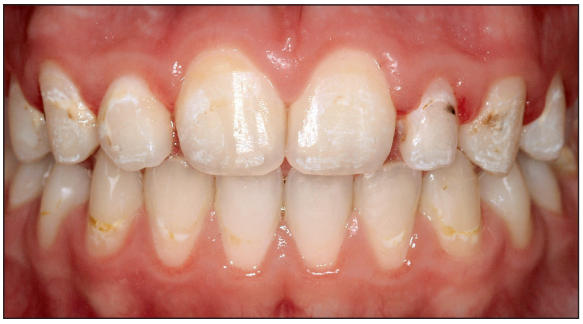


Fig 3-2 Clinical image of an orthodontic patient after therapy. Note the white spot lesions and areas of enamel demineralization in the regions around where the brackets were during orthodontic treatment.

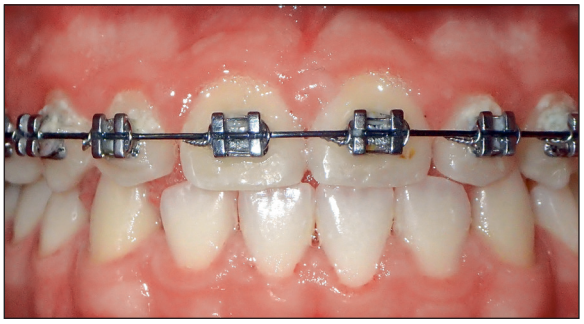


Fig 3-3 Brackets in close proximity to the gingival margin facilitate the accumulation of dental plaque that initiates changes in the health of the gingiva. Note the plaque accumulation and gingival inflammation of the maxillary lateral incisors and canines. Gingival swelling can also be noted, especially between the maxillary central incisors.

exhibit significantly higher gingival index, in tandem with an increase in plaque, when compared with a similar group without brackets.[16]

Thus, the placement of brackets on the different supragingival regions of the tooth crowns will affect the amount and balance of specific bacteria. The bacterial types adhering and forming the plaque biofilm primarily push toward a more cariogenic composition. In this way, increases in cariogenic bacteria, primarily species such as *Streptococcus mutans* and lactobacilli can be detected in patients undergoing orthodontic therapy.[13,17] These bacteria are responsible for the increase in demineralization of the enamel around the brackets and cause early caries lesions in the form of white spots (Fig 3-2). However, if the brackets are placed in close proximity to the gingival margin, then changes can also be initiated in the health of the gingiva (Fig 3-3). This is especially true when orthodontic bands are placed, which by their nature are next to the gingiva or even extend into the sulcus, especially in interdental areas. Inflammation will usually ensue as a result of the plaque-retentive properties in combination with inadequate oral hygiene and possibly the focal irritation they may cause. Corbacho de Melo et al found that adults with banded second molars presented significantly higher gingival inflammation than the control group of similar nonbanded adults.[18] These bands with the propensity for higher plaque accumulation provide the necessary conditions for the balance in the plaque composition to move toward a more complex configuration. In time, more gram-negative, anaerobic, and periodontopathic bacteria can be found within this complex biofilm bacteria, which, if allowed to overgrow, will in turn cause a further inflammatory reaction of the gingival tissues.[19,20] This has been found to be true especially in interdental regions, where the bacteria are even further protected from removal forces. In a group of adolescent orthodontic patients, Kim et al found that bands, depending on their placement in relation to the gingival margin (level with or below the margin), had a tendency for deepening the sulcus (2.9 mm when placed below the gingival margin, 2.5 mm when placed level with the gingival margin), especially compared to brackets (2.3 mm).[21] However, no important differences in the subgingival bacterial composition could be determined.

Changes toward a more periodontopathic subgingival microbiota are clearly associated with the period of orthodontic therapy that involves appliances in the oral cavity.[22–24] Very quickly after initiating the active phase of therapy (3 months after bracket placement without the use of bands), Naranjo et al,[22] using classic microbial culture techniques, found an increase in putative periodontal pathogens such as *Fusobacterium*

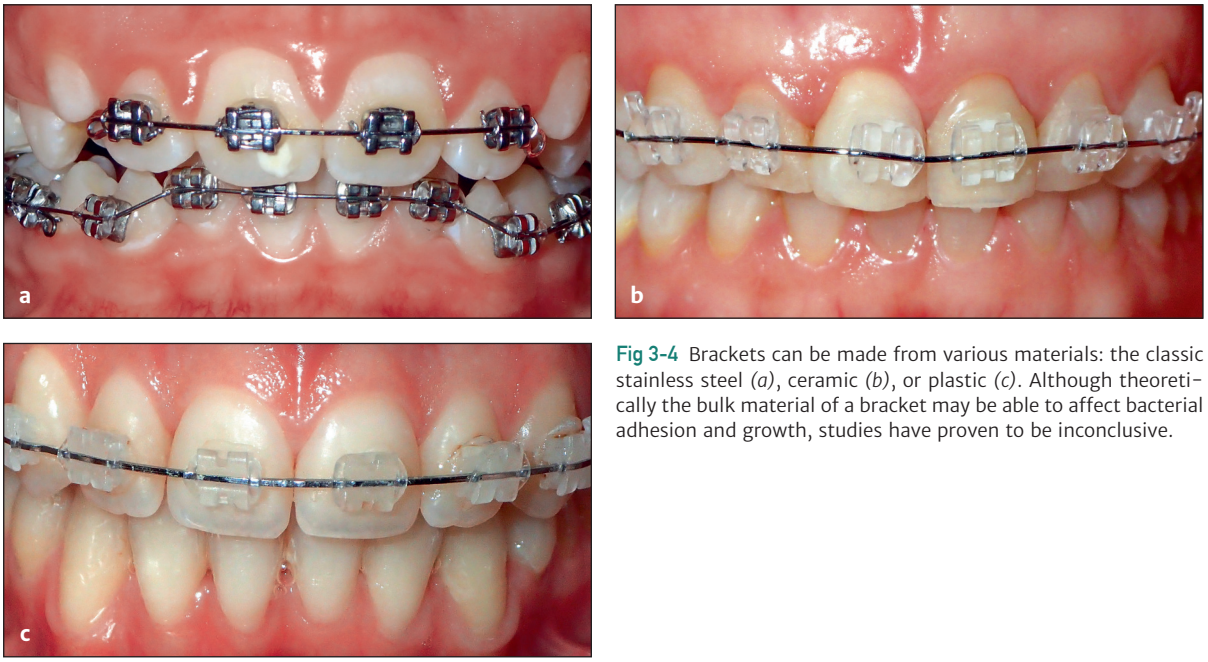


Fig 3-4 Brackets can be made from various materials: the classic stainless steel *(a)*, ceramic *(b)*, or plastic *(c)*. Although theoretically the bulk material of a bracket may be able to affect bacterial adhesion and growth, studies have proven to be inconclusive.

in intimate contact with the enamel surface of the teeth but also with the periodontal tissue. The primary factor regarding plaque accumulation concerns the bulk material of which they are made. Apart from the classic stainless steel, brackets can be made from various ceramic or plastic materials (Fig 3-4). Initial relations with bacteria in the oral cavity are through electrostatic and hydrophobic interactions, where the various characteristics of the surfaces may be a factor. However, studies in the past have shown conflicting results concerning the adherence and biofilm formation of cariogenic bacteria on plastic in comparison to stainless steel or ceramic brackets when the bacteria are allowed to interact with the clean surface.[30,31] In a more recent in vitro study concerning both cariogenic species and periodontopathogens, Papaioannou et al concluded that the bracket material alone had no significant effect on the adhesion of these bacteria.[32,33] Nonetheless, an important factor to consider is that all surfaces in the oral cavity are coated with the salivary, or acquired, pellicle, which will have a great impact on the adhesion of bacteria. This phenomenon occurs very quickly, and the resultant adsorbed film seems to further mitigate any differences in the surface characteristics while at the same time helping to promote the adhesion of bacteria, even on bracket surfaces, and the formation of *P gingivalis* biofilms.[33] This ability of periodontopathogens to form biofilms on bracket surfaces suggests that they may also act as reservoirs for pathogenic organisms.

TYPE OF LIGATION

The type of ligation used in fixed orthodontics—that is, elastomeric or stainless steel ligatures—has also been an important topic of laboratory and clinical research, as these too have been implicated in the accumulation of plaque. In an attempt to minimize the number of bacteria accumulating around brackets, researchers and clinicians have proposed the use of self-ligating brackets. These brackets were first introduced in the 1980s and are presented as having two important advantages over the classic bracket design: *(1)* They do not require the use of elastic or wire ligatures, and *(2)* they help to improve oral hygiene in that their design does not facilitate the retention of dental plaque.

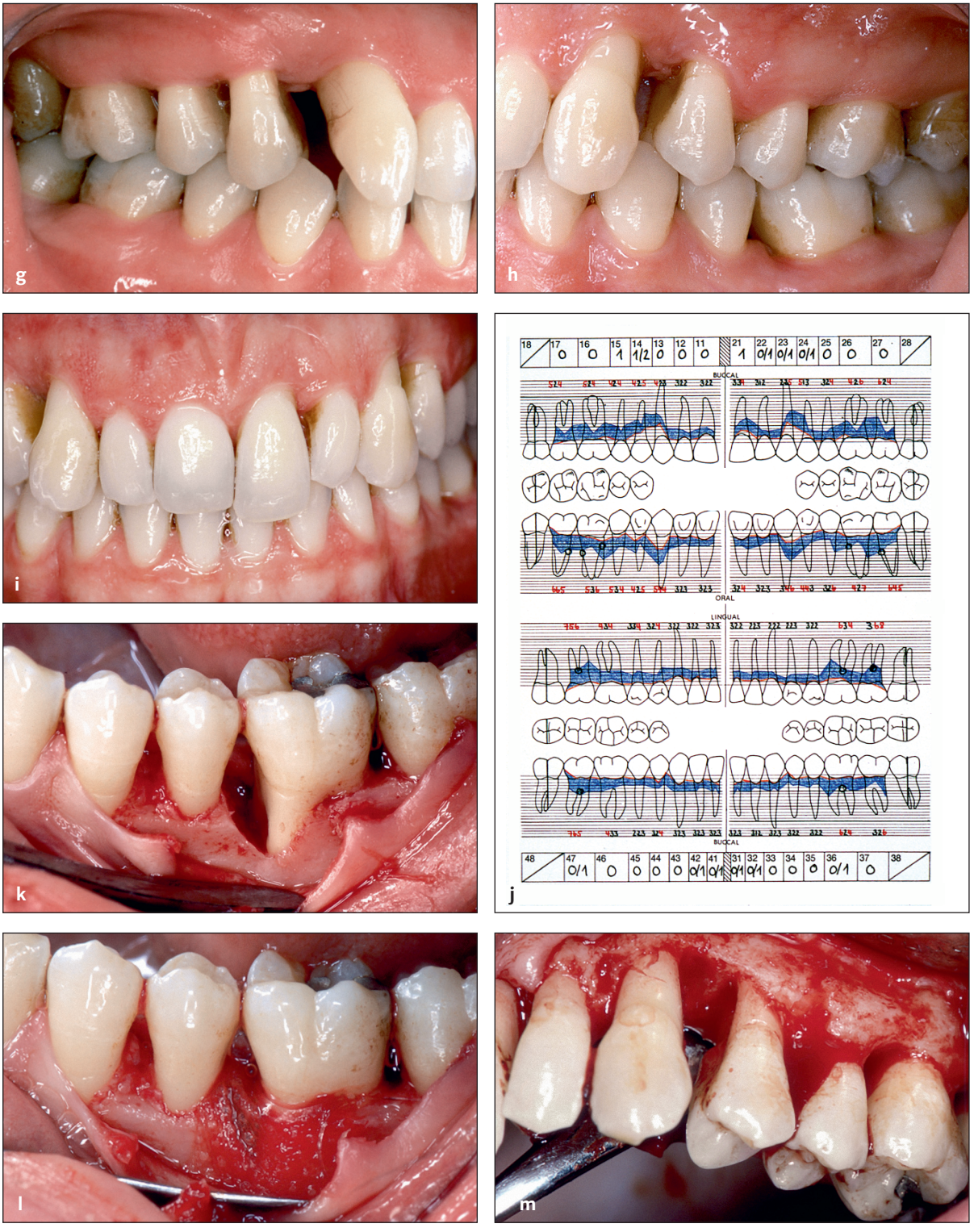


Fig 5-1 *(cont)* *(g to i)* Anterior and lateral views of the patient at re-evaluation following initial periodontal therapy. *(j)* Periodontal chart of the patient at re-evaluation after initial periodontal therapy. *(k to m)* Surgical views of the mandibular and maxillary left quadrants.

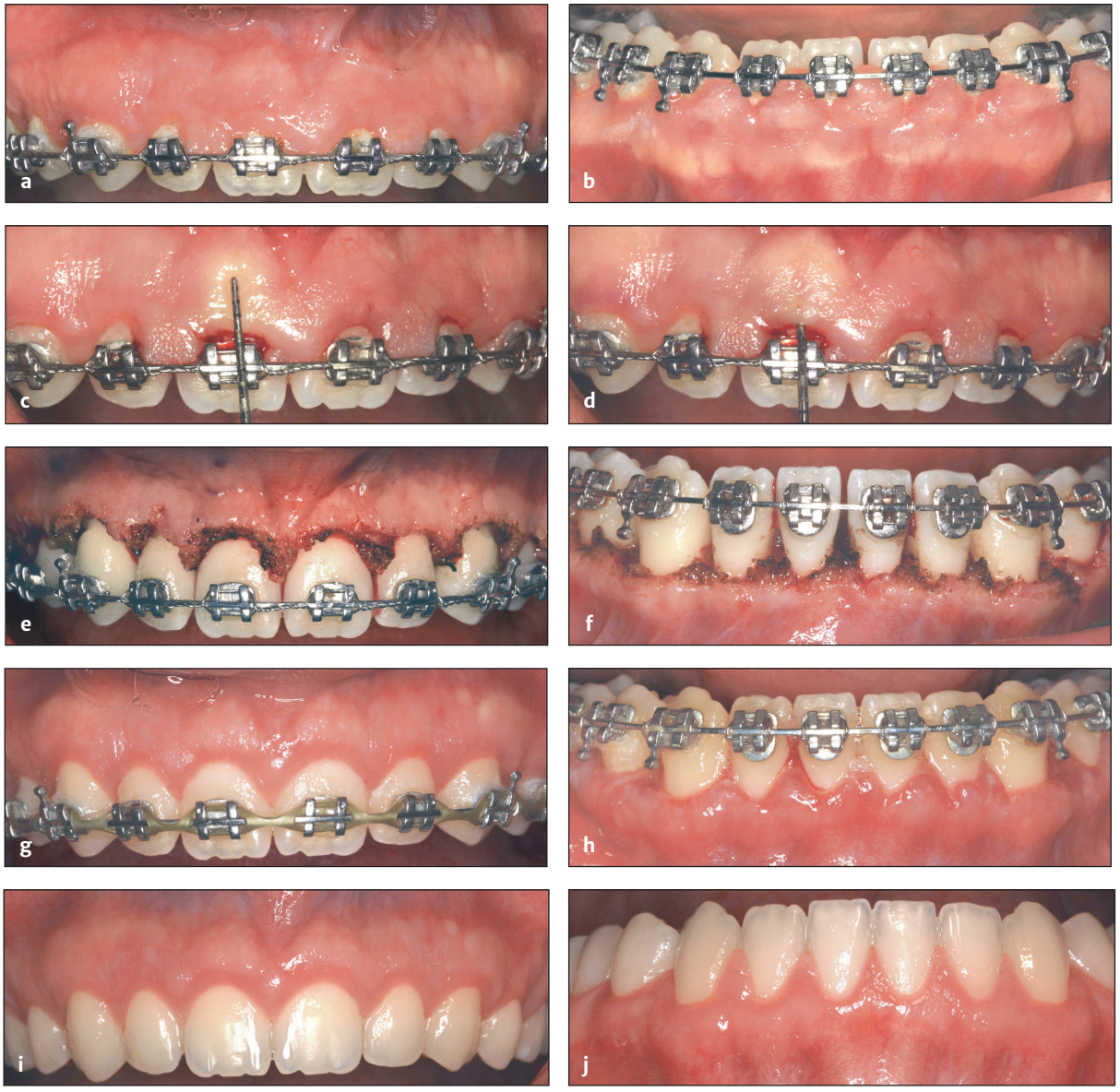


Fig 9-1 Case of gingival overgrowth treated with crown lengthening. *(a and b)* Pretreatment condition. *(c)* Much of the clinical crown is hidden beneath the overgrown gingival tissue. This will hinder proper oral hygiene around the orthodontic appliances. *(d)* Bone sounding. *(e and f)* Surgical crown lengthening has been performed to expose more of the clinical crowns utilizing a scalpel and an Nd:YAG (neodymium–doped yttrium aluminum garnet) laser. *(g and h)* After healing. *(i and j)* After removal of the orthodontic appliances.

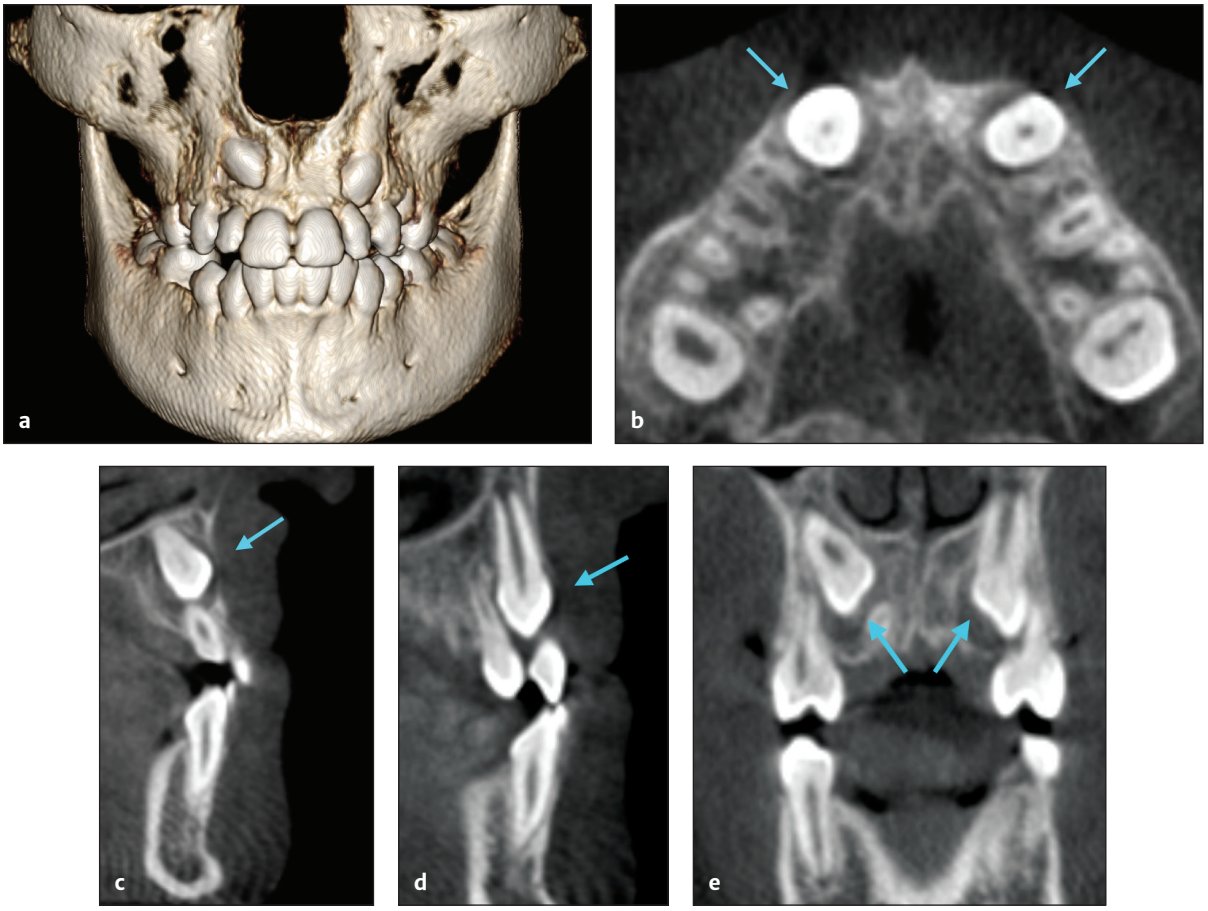


Fig 10-10 CBCT images of a 14-year-old boy with bilateral buccally impacted maxillary canines *(arrows)*. *(a)* 3D reconstructed view. *(b)* An axial slice through the cementoenamel junctions of the impacted maxillary canines. *(c)* A sagittal slice through the maxillary right canine. *(d)* A sagittal slice through the maxillary left canine. *(e)* A coronal slice through the mesial aspects of the maxillary first premolars shows a false palatal position of the buccally impacted maxillary canines.

multiplane examination of the impactions in axial, sagittal, coronal, and 3D reconstructed views should be applied. Locating impacted maxillary canines utilizing only one plane of space may present incorrect identification of the canine position. For example, the buccolingual position of the impacted maxillary canines cannot be determined from coronal slices alone. As shown in Fig 10-10, the coronal view (see Fig 10-10e) shows a palatal position of the bilaterally impacted maxillary canines. On the other hand, axial, sagittal, and 3D reconstructed views (see Figs 10-10a to 10-10d) confirm the buccal position of the same canine impactions.

CBCT images are also helpful to differentiate vertical versus horizontal position of the ectopic maxillary canines in both sagittal and transverse planes (Figs 10-11 and 10-12). This is critical for planning surgical access and biomechanics and estimating treatment duration.

DEPTH OF IMPACTION

It is essential for both the orthodontist and the surgeon to know the depth of impaction of maxillary canines prior to treatment. Impacted maxillary canines may present as superficial soft tissue impactions, partial intraosseous impactions, or deep full bony impactions (Fig 10-13). Deeper impactions will often take a longer time to be translated into the arch and may require bonding of the attachment

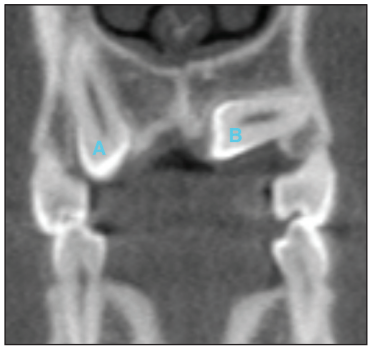


Fig 10-11 CBCT image of the coronal slice of a 14-year-old patient with a vertically impacted maxillary right canine (A) and a horizontally impacted maxillary left canine (B).

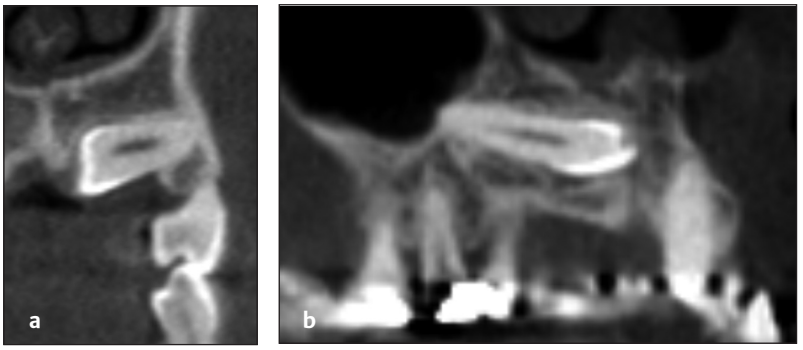


Fig 10-12 CBCT representation of horizontal maxillary canine impaction. *(a)* Coronal slice at the level of the first premolar showing a horizontal maxillary canine impaction in a buccolingual direction. *(b)* Sagittal slice representing horizontal maxillary canine impaction in the anteroposterior direction.

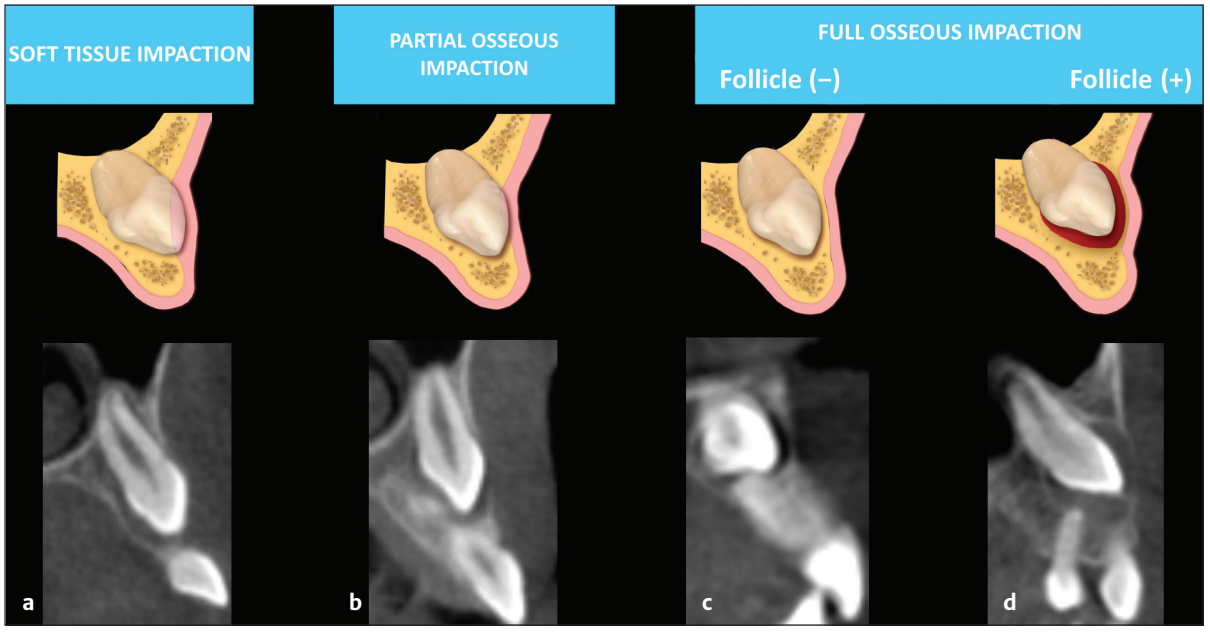


Fig 10-13 Depth of impaction. *(a)* Soft tissue impaction. *(b)* Partial osseous impaction requiring osseous recontouring during surgical exposure. *(c)* Deep intraosseous impaction without enlarged follicle (F–) requiring bone removal above the crown of the impacted tooth. *(d)* Deep osseous impaction with enlarged follicle (F+) requiring curettage of the follicular tissue and removal of the overhanging bone in the path of movement of the exposed tooth.

during surgical uncovering due to a risk of soft tissue overgrowth. Having this information prior to treatment will aid in the selection of an appropriate exposure technique and the mechanics necessary to bring an impacted tooth into the arch.

Impacted maxillary canines often present with enlarged follicles. Their size and shape can be easily determined from 3D images (Fig 10-14). Ericson and Bjerklin[30] in a CT study reported that a circular follicular shape found on conventional 2D radiographs does not always correspond to the one on 3D images. They described symmetric (see Figs 10-14a and 10-14b) and asymmetric (see Figs 10-14c and 10-14d) shapes of the dental follicle. It has been

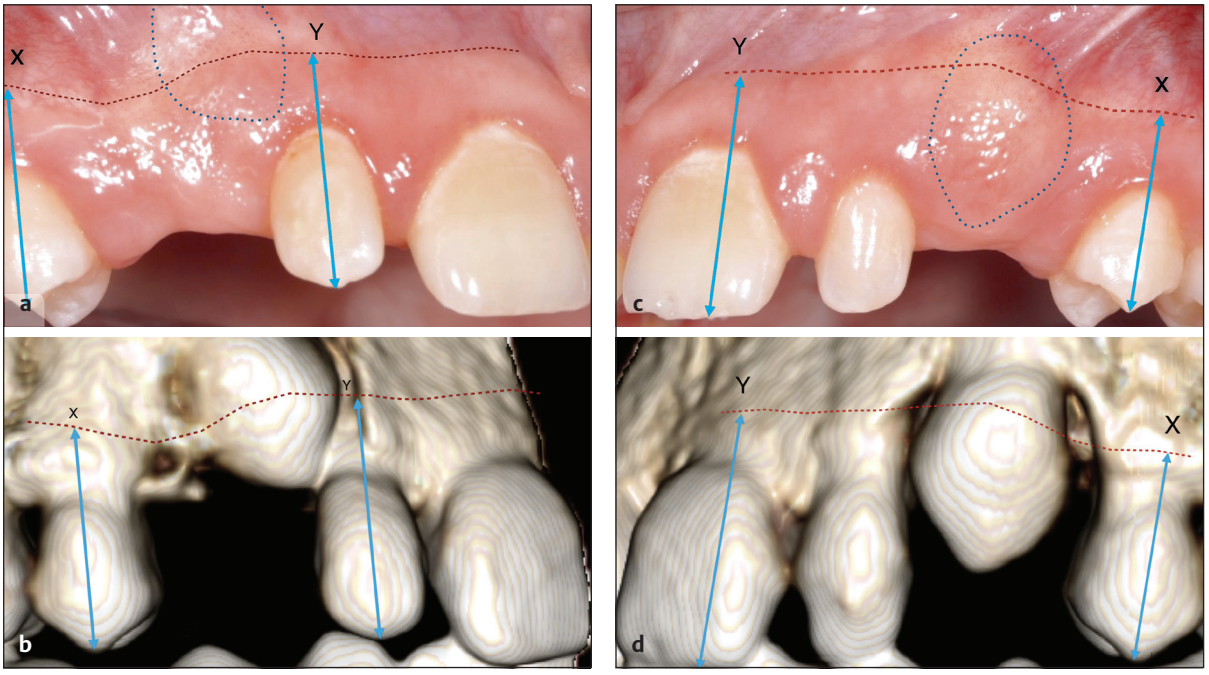


Fig 10-26 Projection of the MGJ in the CBCT 3D reconstructed images utilizing linear clinical measurements. *(a and b)* Buccally impacted maxillary right canine is identified to be positioned halfway below the MGJ. *(c and d)* Buccally impacted maxillary left canine is identified to be positioned two-thirds below the MGJ.

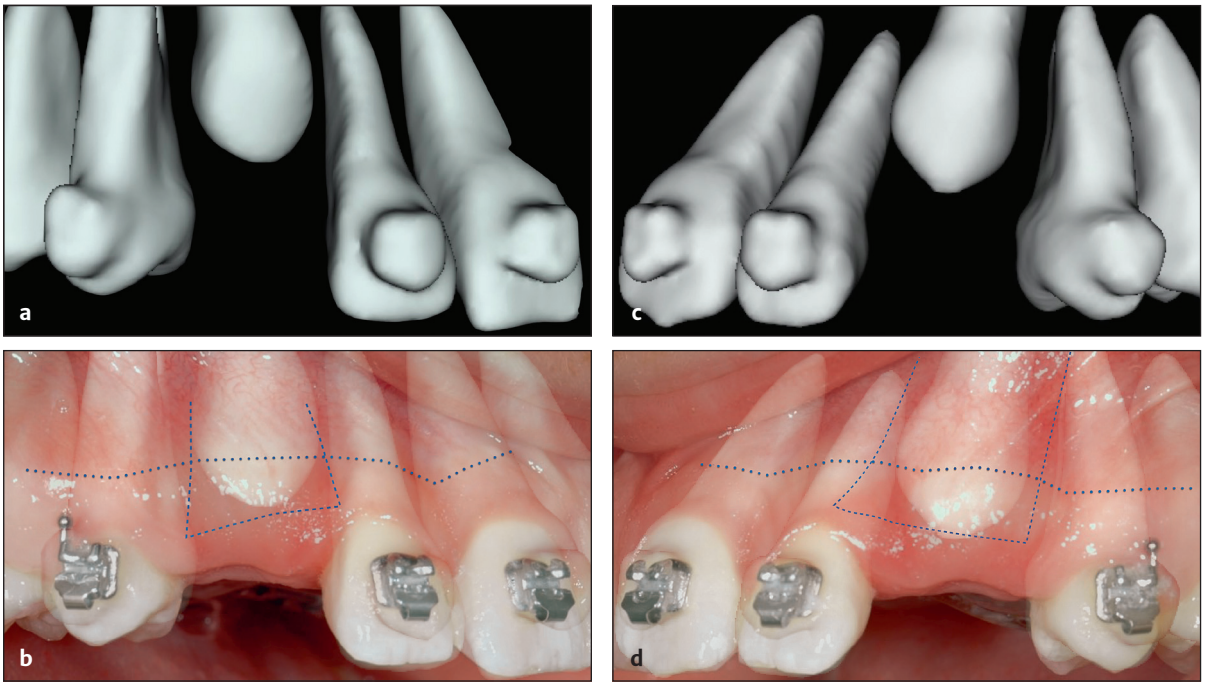


Fig 10-27 *(a to d)* 3D superimposition of the 3D CBCT model over the clinical photographs with outlined MGJ used in planning the apically positioned flap.

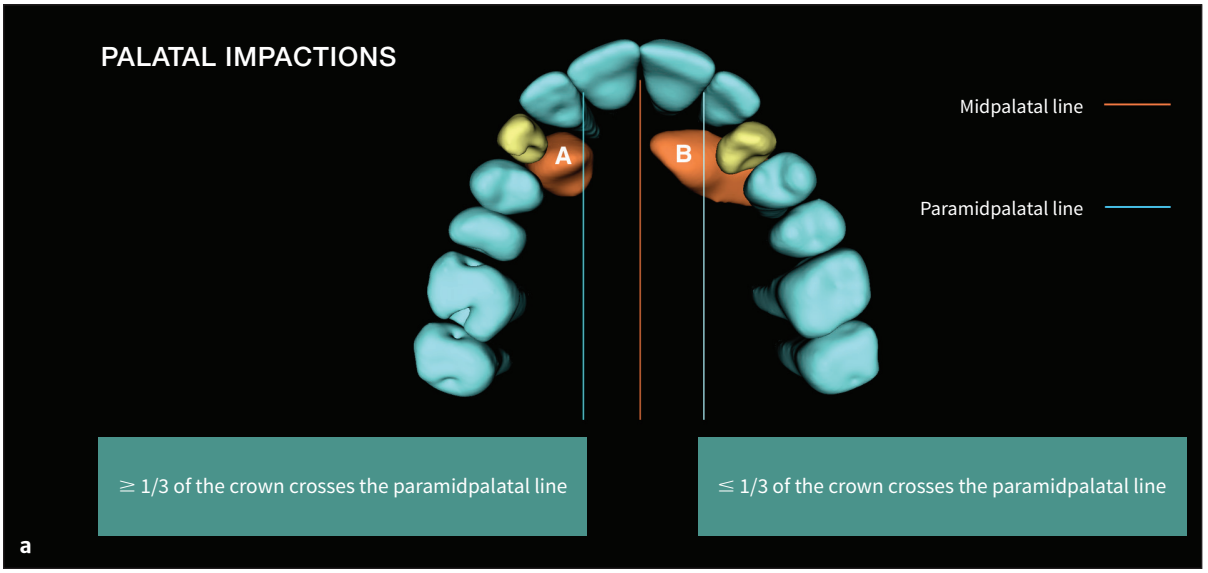
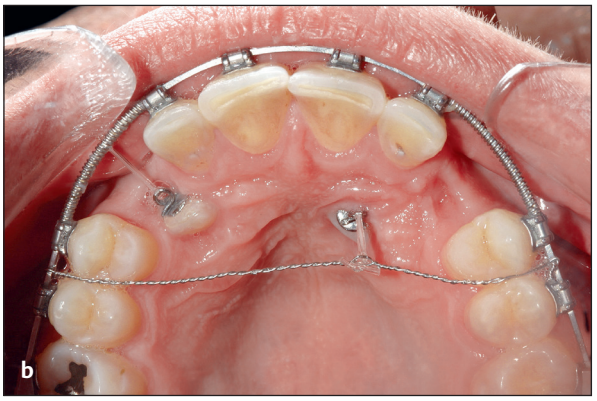
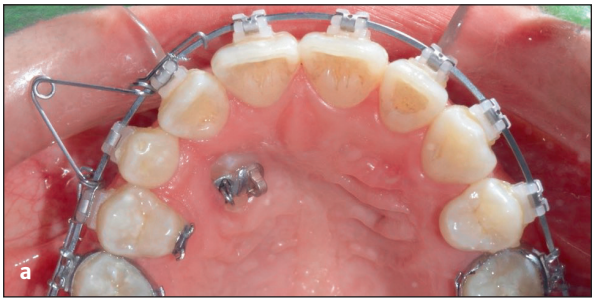
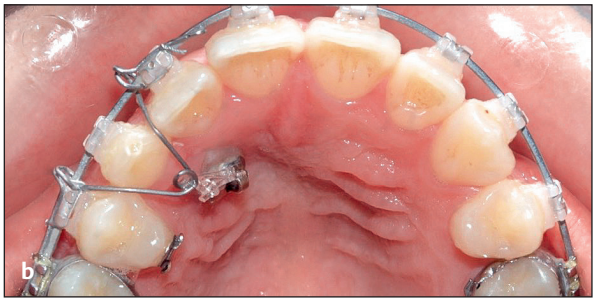


Fig 10-46 Relationship of the palatally impacted canine to the paramidpalatal line. *(a)* 3D reconstructed image of the bilaterally impacted canines positioned distal (A) and mesial (B) to the paramidpalatal line. *(b)* Palatal view of the same patient with activation directly to the archwire (right canine) and activation to the transpalatal ligature wire in the posterior-occlusal direction (left canine).







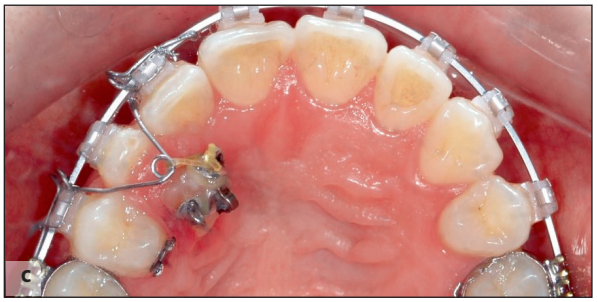


Fig 10-47 *(a)* Vertical eruption of the palatally positioned maxillary right canine with a ballista spring. *(b)* One-month follow-up. *(c)* Two-month follow-up.

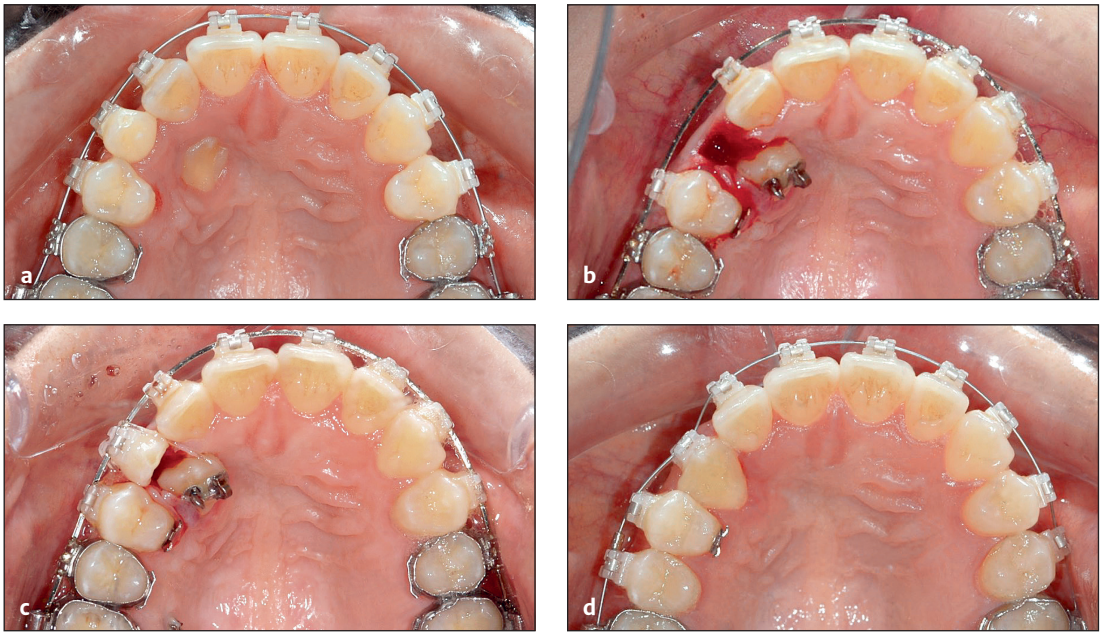


Fig 10-48 A 25-year-old woman presented with a palatally impacted maxillary right canine and an overretained primary right canine. *(a)* The primary canine was kept for esthetic reasons and was engaged in the initial archwire. *(b)* The primary canine was extracted when the permanent canine was moved closer to the optimal position in the arch. *(c)* The crown of the primary canine was preserved and remained engaged in the archwire until the permanent canine was moved into the arch *(d)*.

Conclusion

Traditional 2D images have been greatly enhanced with advanced 3D CBCT technology. The latest computer-guided software and CBCT imaging have opened the doors to accurate diagnosis and treatment planning. Virtual planning after a thorough and accurate diagnosis has become a reality and allows for improved treatment among clinicians in an interdisciplinary approach. This allows us to further fine-tune our parameters so that we may continue to improve treatment outcomes for the benefit of patients.

References

1. Stones HH. Oral and Dental Diseases: Aetiology, Histopathology, Clinical Features and Treatment, ed 4. Edinburgh: E & S Livingstone, 1962.
2. Bass TB. Observations on the misplaced upper canine tooth. Dent Pract Dent Rec 1967;18:25–33.
3. Becker A. The Orthodontic Treatment of Impacted Teeth, ed 2. Oxford: Informa Healthcare, 2007.
4. Kokich V, Mathews D. Surgical and orthodontic management of impacted teeth. Dent Clin North Am 1993;37:181–204.
5. Peck S, Peck L, Kataja M. The palatally displaced canine as a dental anomaly of genetic origin. Angle Orthod 1994;64:249–256.
6. Bishara SE. Impacted maxillary canines: A review. Am J Orthod Dentofacial Orthop 1992;101:159–171.
7. Cooke J, Wang HL. Canine impactions: Incidence and management. Int J Periodontics Restorative Dent 2006;26:483–491.
8. Proffitt WR, Fields HW Jr, Sarver DM. Contemporary Orthodontics, ed 4. St. Louis: Mosby, 2007.
9. Bedoya MM, Park JH. Diagnosis and management of impacted canines. J Am Dent Assoc 2009;140:1485–1493.
10. Ericson S, Kurol J. Radiographic examination of ectopically erupting maxillary canines. Am J Orthod Dentofacial Orthop 1987;9:483–492.
11. Kim Y, Hyun HK, Jang KT. The position of maxillary canine impactions and the influenced factors to adjacent root resorption in the Korean population. Eur J Orthod 2012;34:302–306.
12. Kokich V. Surgical and orthodontic management of impacted maxillary canines. Am J Orthod Dentofacial Orthop 2004;126:278–283.
13. Dewel BF. The upper cuspid: Its development and impaction. Angle Orthod 1949;19:79–90.
14. McConnell TL, Hoffman DL, Forbes DP, Janzen EK, Weintraub NH. Maxillary canine impaction in patients with transverse maxillary deficiency. ASDC J Dent Child 1996;63:190–195.

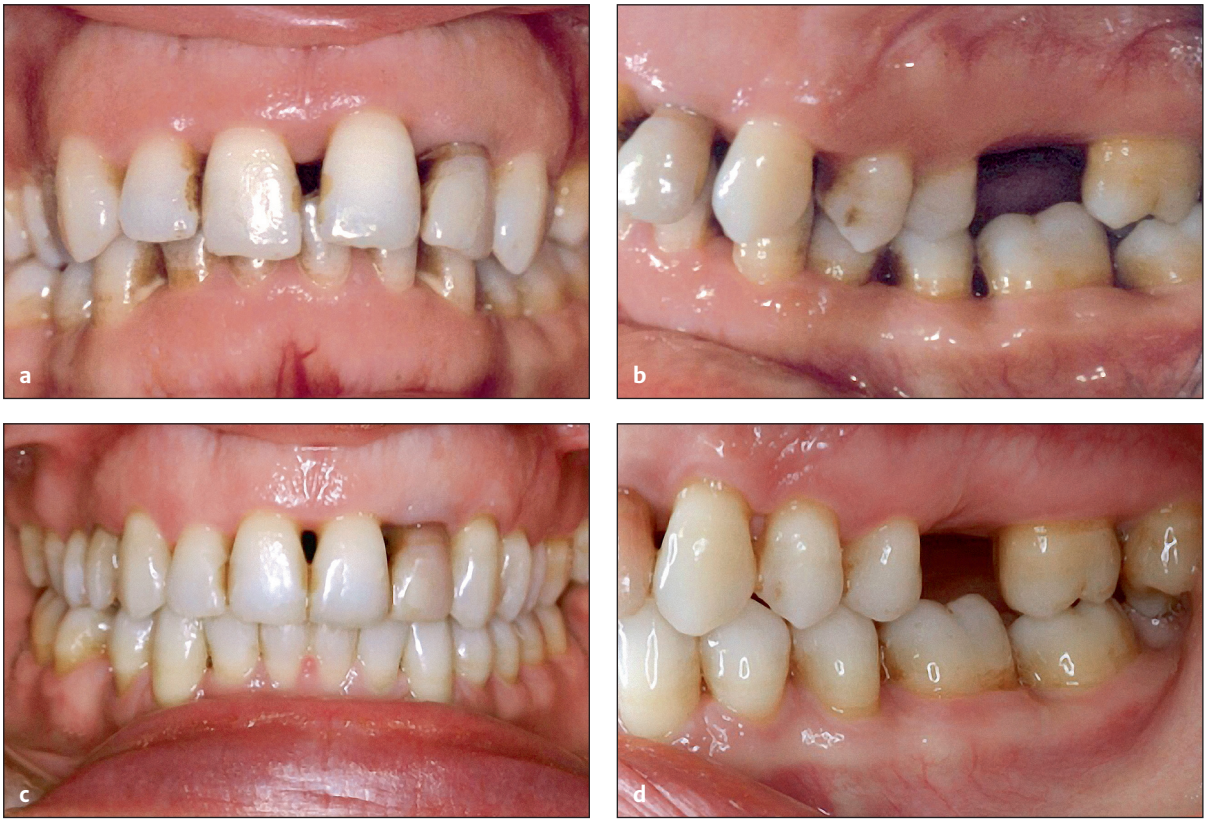


Fig 12-1 *(a and b)* Frontal and lateral intraoral views of a 48-year-old woman affected by horizontal bone loss and vertical collapse. *(c and d)* Same views 7 years after combined periodontal and orthodontic treatment. Of utmost importance is the establishment of proper occlusal contact that allows maintenance of the results by the patient.

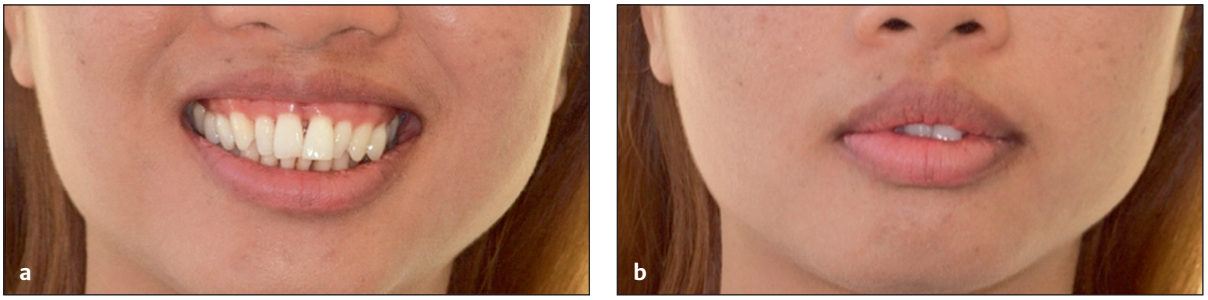


Fig 12-2 *(a and b)* The loss of periodontal support leads to tooth migration, which is further worsened by a functional interference, in this case a lip catch.

where bone support is reduced, the stresses and strains are distributed over a smaller surface area, so the resistance offered by the alveolar crest is less and the extrusive component is more pronounced.[3,4]

In patients with periodontal disease whose teeth have already extruded as a result of the condition, maximum control of the vertical movement is mandatory. From a biomechanical point of view, the use of any continuous arch technique, in which action and reaction forces are not clearly defined, must be limited. Therefore segmented arches, in which active and reactive units are clearly defined, are indicated.[5] Figure 12-4 illustrates a segmented arch approach to retract and intrude the maxillary incisors. The activation of PDL cells should be minimized in order to avoid movement in the posterior anchorage unit. To

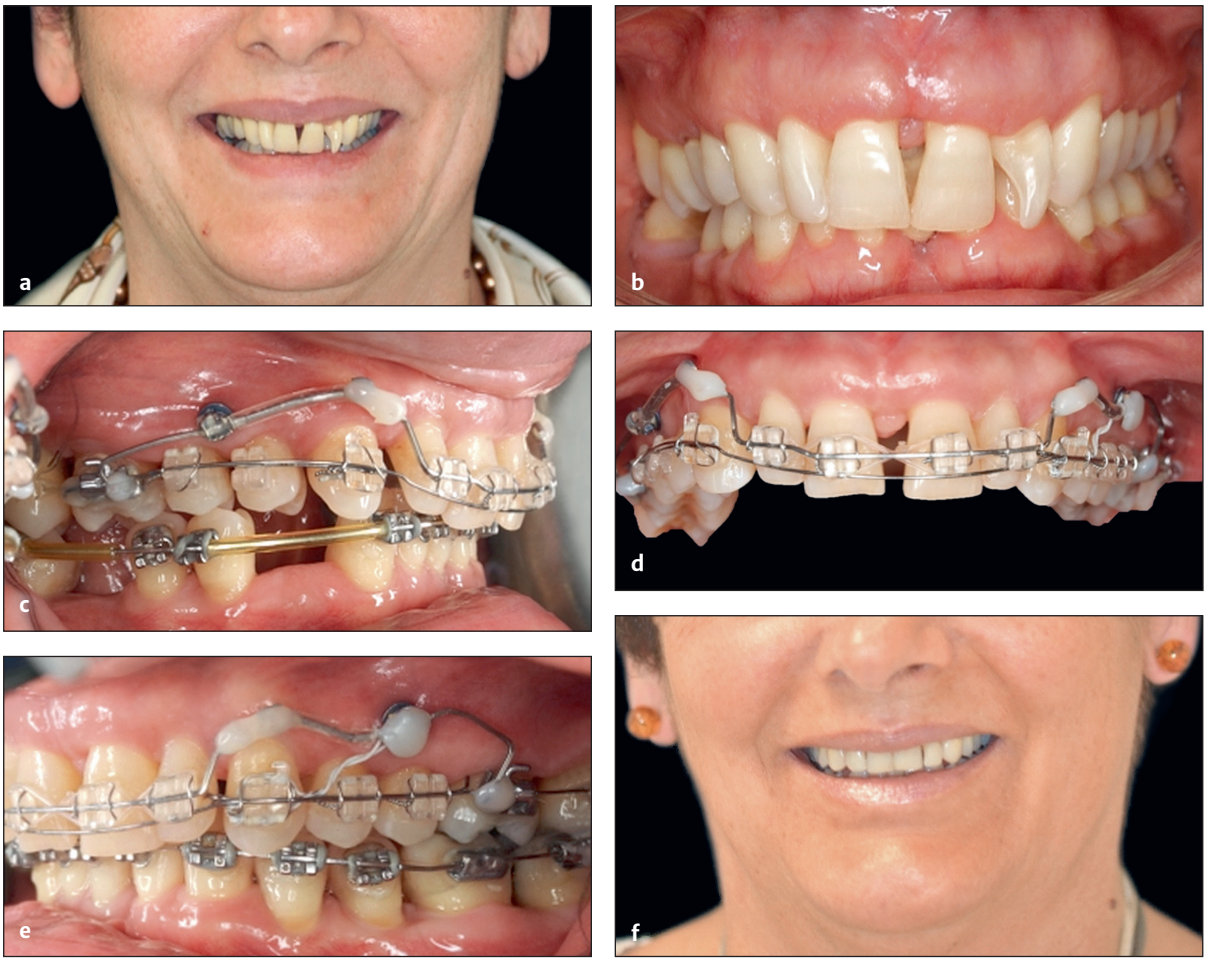


Fig 12-10 *(a and b)* Asymmetric overeruption of the maxillary anterior teeth. The posterior occlusal plane is also canted, due to differential extrusion of the molars in response to loss of posterior vertical support. *(c to e)* Two mini-implants support a cantilever that is activated both anteriorly and posteriorly to facilitate intrusion. Activation has been performed to obtain the same moment anteriorly and posteriorly in order to avoid any rotation on the mini-implants. The leveling of the lateral segments was achieved by a continuous nickel-titanium wire that bypassed the anterior segment but maintained the arch form. Any extrusive component was controlled by the mini-implants. *(f and g)* Final results. With the help of skeletal anchorage, better smile esthetics have been achieved.

and palatal impingement that required correction (Figs 12-11a to 12-11d). The correct line of action of the forces needed for intrusion and retroclination was one passing behind the center of resistance of the maxillary anterior incisors (Figs 12-11e and 12-11f).

This biomechanical system consisted of an anterior unit, or active unit, where the maxillary central incisors were connected through a palatal bonded mesh to a soldered power arm, and a posterior or passive unit (anchorage) consisting of the maxillary

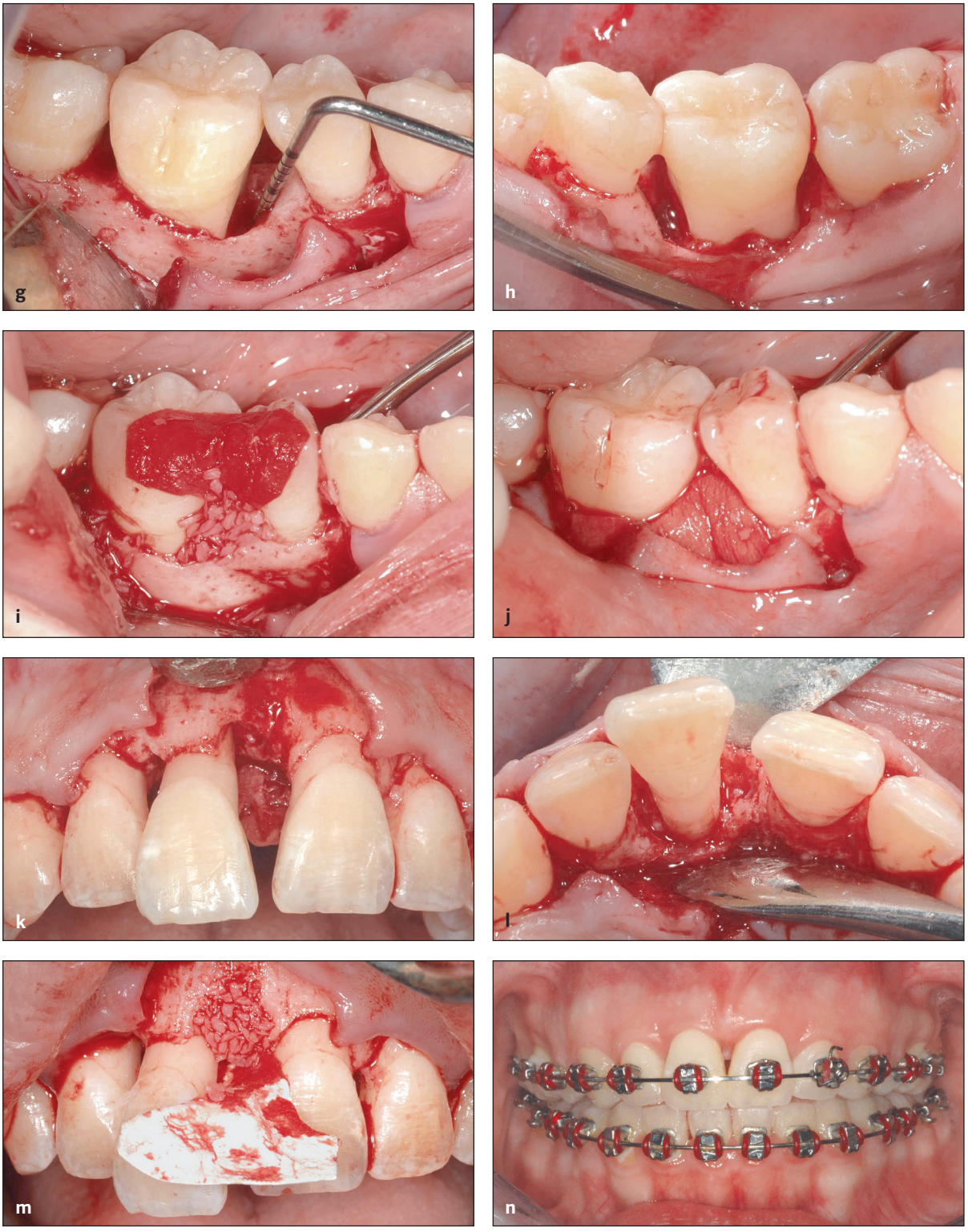


Fig 13-3 *(cont)* *(g to m)* Surgical treatment included regeneration of intrabony lesions by particulate bone allograft and resorbable collagen membrane. *(n)* Orthodontic tooth movement was initiated 8 months after the completion of periodontal surgeries. The maintenance program included supragingival professional tooth cleaning every month.

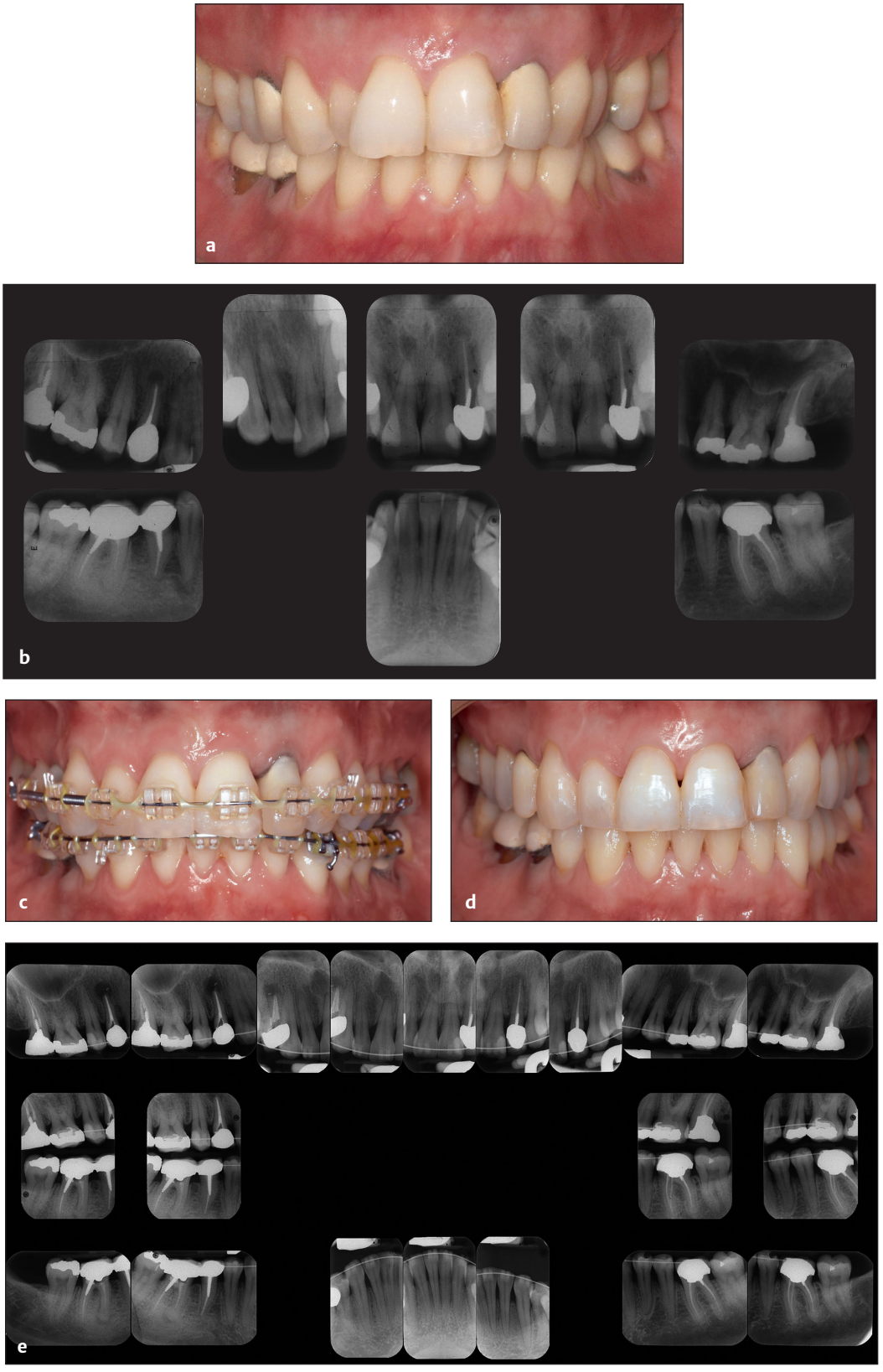


Fig 13-5 A 43-year-old woman with chronic periodontitis wanted orthodontic treatment to improve her facial esthetics. *(a)* Initial clinical view showing multiple restorations. *(b)* Initial radiographs. *(c)* Orthodontic tooth movement was initiated only after controlling the periodontal inflammation. *(d)* Final clinical view. *(e)* Final radiographs. (Case treated by Drs Tali Chackartchi and Stella Chaushu.)