Periodontal Changes Associated with Hyrax vs Damon Expansion

by

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Abstract

Background: Orthodontic expansion is used to treat maxillary transverse deficiency (MTD). **Purpose:** Determine alveolar and soft tissue changes in adolescents undergoing expansion using Hyrax expanders followed by non self-ligating brackets or only the Damon® system. **Research Design:** MTD patients were randomly allocated to the Hyrax or Damon® group. Records (CBCT, photos) were taken at different stages of treatment. CBCTs from 45 patients were used to measure buccal alveolar bone widths (BBW) at 2 root points for 4 tooth groups before treatment, at debond and 2 years post treatment using a technique described by diGregorio et al (2019). Recession (GR) and its risk factors were ascertained using the same patients' digital photos via techniques described by Le Roch et al (2019). **Results:** At debond, the maxillary molars demonstrated a significant reduction in buccal alveolar width in response to orthodontic expansion ($p=.016$) and the Hyrax group demonstrated more BBW loss than the Damon® group ($p=.014$). There was no difference between the groups at follow up ($p=.085$). Absence of BBW measured via CBCT is not related to the presence of GR at debond ($p=0.74$) or at two year follow up ($p=0.99$). Keratinized tissue width (<2mm) had higher odds of exhibiting gingival recession than KT width ≥ 2mm [OR≥12, p<.0005]. **Conclusions:** BBW decreased with expansion. There was more BBW loss with Hyrax than Damon® at debond, though groups were similar at follow up. Absence of BBW measured via CBCT was not related to the presence of gingival recession; however keratinized tissue width <2mm was.

Preface

This thesis is an original work by Marissa Kobewka. This project was approved by the University of Alberta Ethics Board (Pro00013379) under the name "Periodontal Changes Associated with Hyrax vs Damon Expansion."

No part of this thesis has been previously published.

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Chapter 1: Introduction

1.1 Maxillary Transverse Deficiency

Common characteristics of *Maxillary Transverse Deficiency* (MTD) include narrow palate and posterior crossbite. Crossbite has been related to 7% of the American population in children and it does not self-correct over time, increasing to 9.5% of the adult population.[1,2] Differentiation between dental and skeletal etiology can be ascertained via the width of the palatal vault (i.e. narrow) and the dental compensations (i.e. buccally tipped crowns) present. Simply put, should the transverse dimension of the palate match that of the inter-tooth width of the mandible yet the posterior teeth are bucally inclined, the etiology of the crossbite is dental. Contrarily, should the transverse dimension of the palate be less than that of the mandible and the teeth are upright or buccally tipped, then the etiology of the crossbite is skeletal and a diagnosis of MTD can be ascertained (Figure 1.1).[2(p201)] Other issues that arise from MTD include distortion of the arches, tooth abrasion from anterior interferences, and functional shifts that can lead to the possibility of mandibular skeletal asymmetry during growth and development.[2(p430)]

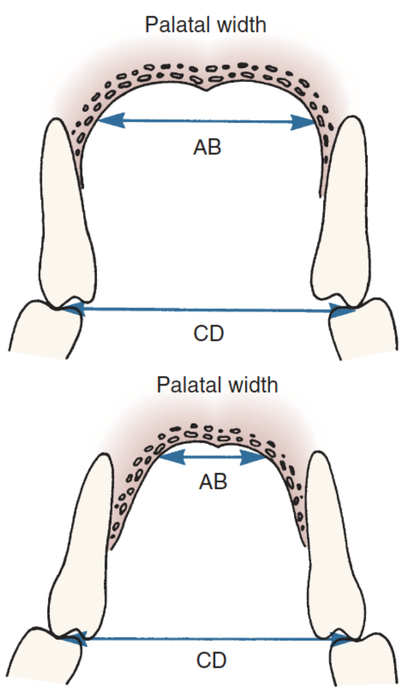


Figure 1.1 Posterior crossbite can be either dental (i.e., distance AB approximately equals distance CD), or skeletal (i.e., distance CD is considerably larger than distance AB). Adapted from Proffit, 2017 pg 201.

Orthodontic expansion is a widely accepted procedure performed by orthodontists to correct a posterior crossbite and/or transverse maxillary deficiency, depending on the aforementioned etiology of the diagnosis.[1] For example, brackets and archwires (i.e. Full Fixed Appliances (FFA)), are typically indicated to achieve dental expansion in the case of a crossbite of dental etiology. Treatment of MTD; however, depends on the skeletal maturation of the patient and the severity of crossbite. It can be treated with surgery (i.e. surgically assisted rapid palatal expansion (SARPE)), distraction of palatal halves (i.e. Mini-screw Assisted Rapid Palatal Expansion (MARPE), Rapid Palatal Expansion (RPE)), or a combination of both (i.e. Slow Maxillary Expansion (SME), semi-rapid maxillary expansion (SRME)).[2,3]

During growth and development, transverse growth is the first to cease at the end of the pubertal growth spurt. As transverse growth approaches completion, the midpalatal suture becomes more tortuous and interdigitated with age rendering transverse growth modification less predictable as the patient approaches late adolescence.[2(p430)] Prior to the adolescent growth spurt, the customary treatment is opening the midpalatal suture via palatal expansion, which widens the roof of the mouth and the floor of the nose.[2(p430)]

1.1.1 Orthodontic Tooth Movement

The popular 'tension-compression' theory explains orthodontic tooth movement via forces applied to teeth that lead to compressive and tensile stresses in the surrounding tissues followed by bone resorption and apposition, respectively.[4] While this is an oversimplification of a more complex interaction between the biomechanics of the force applied and the hosts response with respect to inflammation and bone remodeling,[2] the physiology of tooth movement will not be thoroughly explored due to the nature and scope of this study.

The PDL plays an important role in mediation of mechanical force, in alveolar bone modeling and remodeling and in maintenance of physiologic equilibrium within periodontal tissue.[5,6] For example, the definition of *optimal continuous orthodontic forces* is based on approximating orthodontic forces to the capillary pressure of the PDL in order to sufficiently compress the PDL to facilitate movement without occluding the vessels.[2,5,7,8] Mice models have shown that the application of orthodontic compression force to the PDL in genetically identical mice immediately causes a 40% decrease in bone volume, with a 30% decrease in overall bone density away from the PDL-bone interface.[4] This suggests that the inflammatory mediators generated by

PDL compression spread widely enough to start general bone remodeling in a relatively large area around the tooth socket.[4]

While the force system generated by orthodontic appliances is discussed in the orthodontic literature, the reality is that the precise relationship between force magnitude and orthodontic tooth movement is not fully understood and has been the subject of several hypotheses.[9] More importantly, even with very light force, localized stress in the PDL may be excessively high, as is the case in uncontrolled tipping. For example, numerical models have shown that uncontrolled tipping produces localized high stress and is five times greater than the uniform stress in translation produced by the same magnitude of force.[4(p184)] Thus, the concept of one appliance being able to produce a universal and optimal orthodontic force throughout the dentition without consideration of the resultant movement, is overly simplistic.[9]

1.1.1.1 Palatal Expansion and the Hyrax Appliance

Three types of palatal expansion are commonly shown in the literature: rapid maxillary expansion (RME), slow maxillary expansion (SME), and semi-rapid maxillary expansion (SRME)[2,3] RME is associated with intermittent high-force systems[10]and tooth-tissue-borne appliances (e.g. Hyrax, Haas).[11] SME is often associated with continuous low-force systems (e.g. quad-helix appliances or coil springs).[2] Brunetto et al.[3] evaluated the buccal bone after RME and compared the results to SME. Both protocols provided maxillary expansion, although SME has been related to more physiologic effects on sutural tissues, greater tooth movement, and lower orthopaedic effects compared with rapid maxillary expansion.[3] Similarly, both RME and SME cause lateral flexion of the alveolar processes and buccal displacement of the anchorage teeth with varying degrees of inclination as well as horizontal and vertical bone loss.[3] That said, more buccal inclination was observed in the RME group and increased buccal bone loss in the SME group.

The Hyrax appliance has been the customary treatment modality to achieve sutural expansion in late mixed to early permanent dentition for over 100 years.[2,8] To do so, it transmits heavy intermittent forces to anchor teeth using a screw system (see appendix C).[4(p188),12] The maxilla widens at the roof of the mouth and the floor of the nose; however, it opens more anteriorly than posteriorly and more inferiorly than superiorly.[2(p430)] As such, affirmation of a sutural split is often realized by the presence of a midline diastema, which can close spontaneously via skeletal relapse and tooth movement created by stretched gingival fibers.[2(p434)] Histologic studies in animal models show that this heavy force can collapse capillary circulation and block the blood flow in the PDL, leading to aseptic necrosis and hyalinization.[4(p188)] Capillary collapse may occur via direct crushing of cells, interruption of blood flow, or induction by inflammatory factors. Necrotic areas that need to be removed can temporarily slow down the rate of tooth movement, allowing the force to be transmitted to the mid-palatal suture, although rapid undermining resorption can follow.[4(p200)]

Since distraction of the palatal halves requires healing for 10 to 12 weeks, most providers follow RME with a quiescent period, free of posterior orthodontic forces. During this time, there is subsequent contracture of the palatal soft tissues and the maxillary halves are able to relapse while the teeth are held in situ.[2(p433)] Despite mostly skeletal expansion initially (i.e. 80%), the net skeletal / dental contribution to expansion after the suture has been allowed to heal is 50% / 50%.[2(p435),13] Additionally, the orthopedic effect of the Hyrax appliance diminishes as the suture matures. This can lead to in dental tipping, increasing the risk of positioning teeth through the envelope of the alveolar process which can result in a reduction of alveolar bone height, bone dehiscence, and gingival recession.[13–17]

1.1.1.2 Alveolar Remodeling and the Damon® System

The Damon® system advertises the use of *passive self-ligation* (PSL) and super-elastic nickel-titanium arch wires to produce biologically compatible, continuous light force that can induce bony apposition during transverse expansion.[8(p997.e96),18] According to the authors, the appliance itself functions within an *optimal force zone* (i.e. below the capillary pressure of the PDL. For more information, see Appendix C), that helps to maintain the patency of PDL blood vessels and facilitates maximal cellular maintenance during tooth movement.[8(p997.e96),8(p156),19,20] As teeth are moved, the ultra-low force system is thought to allow adaptation of the buccal alveolar bone and connective tissues until an equilibrium is attained between the appliances and the existing musculature.[2,8(p156),18,21] For example, the Damon® system claims to be able to widen the posterior arch while maintaining the canine width, without the use of high-force palatal expansion.[8(p997.e97)] The authors report CBCT evidence of increases in premolar width of up to 6 to 8 mm with little tipping and alveolar bone development coincident with such movement.[8(p997.e118)]

While laboratory evidence[22] corroborates PSL's ability to deliver lower-magnitude forces compared with elastomeric-ligated appliances applied to the same malocclusion (i.e. high buccal ectopic canine), other studies[20,23–26] suggest no difference between the forces delivered and subsequent alveolar outcomes produced in the Damon® system compared to conventional self-ligating systems. Similarly, transverse expansion was found to occur mainly as a result of buccal tipping (rather than true translation), akin to that reported in studies using RME.[13,20,21]

1.1.2 Purpose and Objectives

Displacement of the teeth outside the alveolar anatomic limits can damage the periodontium, [3,27,28] thereby compromising patient satisfaction, tooth longevity, and the esthetic result.[2,3,29–31] The purpose of this study was challenge the hypothesis that the Damon® appliance is better suited for correction of MTD compared to the Hyrax appliance due to its ability to adapt the buccal alveolar bone and connective tissues in the transverse dimension. To evaluate the periodontal changes on maxillary first molars, premolars and canines as well as mandibular first premolars in patients with MTD treated with Damon® and Hyrax appliances using CBCT measurements and photos analysis.

1.2 Review of Cone Beam Computed Tomography (CBCT)

Radiographic imaging was declared a valuable diagnostic aid to orthodontics by Price in 1900, just five years after the discovery of x-rays.[32,33] While axial and spiral CT have been available for more than 40 years their application did not extend beyond medicine due to their considerable cost and the relatively large radiation dose.[2] As such, conventional 2D imaging remained the mainstay of radiographic records until CBCT became commercially available in the mid-1990s in Europe, and FDA approved in North America in 2001.[32,34] Since it joined the marketplace CBCT is considered the most significant advancement in dental imaging since rotational panoramic radiography.[32] It has undergone a rapid and ongoing evolution with respect to imaging capabilities and radiation dose and today, there are over 50 models available worldwide.[32,33] While it has become useful in all aspects of dentistry, CBCT has been indicated for orthodontics for a variety of reasons, including: skeletal and/or dental malformations, impacted teeth, temporomandibular joint (TMJ) considerations, orthognathic surgical planning, airway analysis, placement of temporary anchorage devices (TADs), and assessment of the effects of orthopaedic appliances.[35] Additionally CBCT is the only *non-invasive* technique available to visualize the quality of the buccal alveolar bone overlying the dentition (despite its short-comings, discussed in section 1.3).

1.2.1 What is CBCT?

Cone Beam Computed Tomography (CBCT) is an imaging technique consisting of X-rays that are divergent, pyramidal or cone-shaped, whereby the source of ionizing radiation is directed through the middle of the area of interest onto an area X-ray detector on the opposite side.[32,36] The X-ray source and detector rotate around a fixed fulcrum within the region of interest (ROI).[32,36] Using CBCT, a 3D volumetric image is generated in three phases: acquisition, reconstruction and visualization (Figure 1.2).[32]

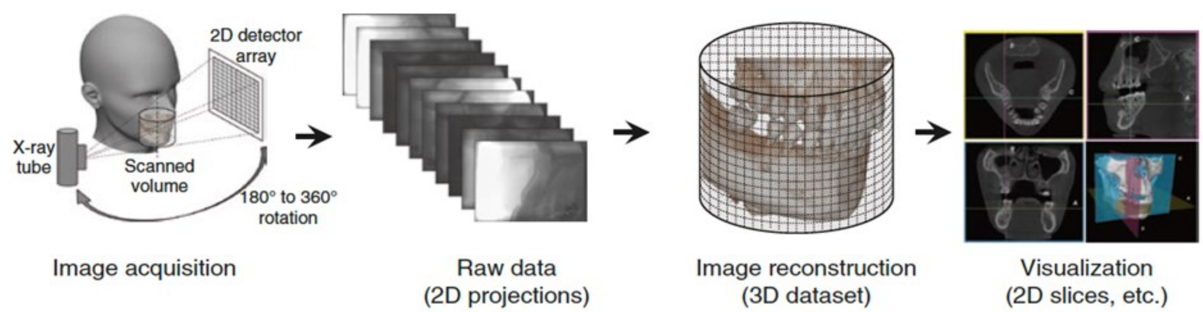


Figure 1.2 Schematic diagram showing the stages of CBCT image production. During a 180º–360º rotation of the X-ray tube and detector, multiple planar basis projections (raw data) are acquired. The raw data is then reconstructed into a volumetric dataset (primary reconstruction), which is subsequently reformatted as sequential, contiguous orthogonal slices (secondary reconstruction). The data may be further reformatted (e.g., volume rendering, curved reformatting, maximum intensity projection).[36] Partially adapted from Pauwels et al. (2015a).

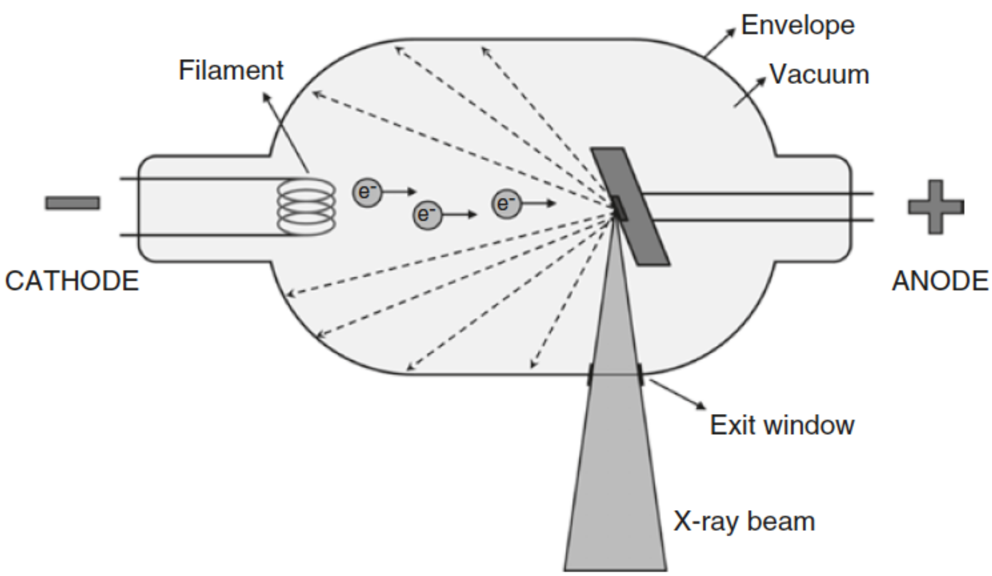
1.2.2 Image Acquisition

The x-ray tube (aka Source) of a CBCT unit is similar to that of most other 2D devices. Electrons are liberated from a heated filament via *thermionic emission*[*] on the cathode side of an electric potential in a vacuum, then accelerated onto a focal spot (0.5mm wide) made of high density material (e.g. tungsten).[32,36] Collision of the accelerated electrons results in *Bremsstrahlung*,[†]

[*] liberation of electrons from an electrode by virtue of its temperature (releasing of energy supplied by heat). This occurs because the thermal energy given to the charge carrier overcomes the work function of the material.

[†] ˈbrɛmʃtrɑːləŋ/ from bremsen "to brake" and Strahlung "radiation"; i.e., "braking radiation"

which reduces their kinetic energy and produces heat and electromagnetic radiation (EMR) in the x-ray spectrum.[32,36] These x-rays are emitted in all directions, but absorption within the anode, the tube housing and aperture-based x-ray blockers (i.e. *collimators*) results in a beam emerging from the tube perpendicular to the electron beam(Figure 1.3).[32,36] It is the applied collimation that determines the field of view (FOV) of the image.[32,36]



The difference between 3D and 2D devices is more obvious when one considers *how* the scan is taken. That is, 3D image acquisition through a partial (≥180) or full (360) rotational scan of the X-ray tube and a reciprocating 2D flat detector, both fixed to a rotating gantry.[32,36] The axis of rotation of this configuration is centered at a certain region of interest (ROI) within the patient's head (standing or sitting).[32] Throughout the rotation, a divergent pyramidal or cone-shaped beam of X-rays is directed towards the detector on the opposite side, which then converts incoming photons to an electrical signal.[32,36] During the time is takes the gantry to rotate, many sequential 2D projection images (i.e. raw data) are captured, each covering the FOV from a slightly

Figure 1.3 Schematic diagram of the components of an X-ray tube. Electrons are released from the filament at the cathode through *thermoionic emission* and accelerated to a small area on an anode (i.e. the focal spot (dark gray)) due to a high tube voltage (kV). After electrons hit the focal spot, part of the energy is released as X-rays.[3] Figure reproduced from Pauwels et al. (2015a)[3].

1.2.2.1 Pre-Processing

Most CBCT systems use indirect flat panel detectors (FPD) made up of a scintillator (typically cesium iodide [CsI]) medium, which converts X-ray photons into visible light photons.[32,36] The scintillator medium is usually layered over a thin film photodiode/transistor matrix in a 2D pixel array (i.e. a photon detector), which converts light into an electrical signal to be digitized.[32,36] An important property of FPDs is their pixel size (i.e., the physical size of individual detector elements), which is a principal determinant of resolution and therefore detail of the images. In CBCT, each projection image consists of a pixel matrix with a 12- to 16-bit value (proportionate to the detected X-ray intensity) assigned to each pixel.[32]

1.2.2.2 Reconstruction

Due to the complexities associated with reconstruction, the acquired data is often reconstructed on a separate computer by a series of algorithms.[32] The most widely used reconstruction algorithm in CBCT is the *Feldkamp –Davis–Kress* (FDK) algorithm.[32,36] Using back projection, the total X-ray attenuation measured at each pixel is equally distributed to all voxels in the FOV along the path of the X-ray, then averaged between different projection angles.[32,36] Since the back-projected image is often blurred compared to the original, the projections can be filtered to enhance the edges. [32,36] Additional parameters to enhance image visualizations are also utilized.[32] The data is transformed into a 3D volumetric dataset composed of cuboidal volume elements (i.e. voxels), which can be sectioned *orthogonally*.[‡] Each voxel is assigned a 'gray value' depending on the attenuation of the material(s) inside it.[32,36] A lower gray value corresponds to a lower attenuation, with the lowest gray values corresponding to air.[32] The range of gray values depends on the bit depth of an image, with an image of *n* bit having 2^n possible gray values (e.g. 12 bit = 2^{12} = 4096 gray values).[32,36]

1.2.2.3 Image Optimization

[‡] perpendicular images in all three planes

Image quality is a subjective descriptor of data visualization, used to determine if an image contains enough information to allow for a medical decision to be made with an acceptable degree of certainty for a specific medical task.[37] For example, the image quality parameters (i.e. spatial resolution, contrast, noise, artefacts) required for the treatment planning of an impacted cuspid may be very different than for orthognathic surgery. That said, the technical parameters by which the CBCT acquires and reconstructs a volume (i.e. kV, mAs, FOV size, voxel size) as well as the radiation dose (Table 1.1) will ultimately affect the image quality.[32,36–39] *Image Optimization* describes what the clinician determines to be an optimal exposure to achieve adequate image quality vs. radiation dose exposure for the task at hand.

Table 1.1 Effect of imaging and reconstruction parameters on image quality and radiation dose, Table reproduced from Pauwels et al. (2015a).[36]

Imaging parameter	*Spatial resolution*	*Contrast*	*Noise*	*Artefacts*	*Radiation dose*
FOV size ↑[a]	–	↓	↑	↑	↑
kV ↑	–	↓	↓	–[b]	↑
mAs ↑	–	–	↓	–	↑
Voxel size ↑	↓	–	↓	–	–

↓, decrease; ↑, increase; FOV, field of view; kV, tube voltage; mAs, tube current-exposure time product.
[a]Minor image quality effects due to factors like beam divergence and truncation of the FOV not being taken into account.
[b]Beam hardening is somewhat reduced at higher tube potential values

1.2.2.3.1 Radiation Dose

The dental community has a professional responsibility of beneficence when prescribing imaging with ionizing radiation; that is, because malignancies are the principal long-term effect of exposure to ionizing radiation, it is prudent that the best interest of the patient is upheld when CBCT is necessary to maximize diagnostic benefit.[39] Using the "as low as reasonably achievable" (ALARA) principle, justification of every radiographic exposure must be based primarily on the individual patient's presentation including: considerations of the chief complaint, medical and dental history, and assessment of the physical status (as determined with a thorough clinical examination), treatment goals, economic and societal factors.[36,39] That said, blindly decreasing the radiation dose may lead to degrading the image quality below what is necessary to allow for a medical decision to be made with an acceptable degree of certainty for a specific medical task.[37,38] Therefore knowledge of the parameters that affect radiation dose and image quality as it pertains to a specific treatment is imperative.

For most CBCT systems, the tube voltage (kVp) is fixed, and the tube current (mA) and exposure time (s) can be varied depending on the desired image quality and patient size.[36] Radiation exposure increases is based on: increasing the resolution, decreasing the voxel size, and increasing the FOV.[2] In general, exposure can be reduced by accepting a reduced resolution; that is, increasing the voxels and using the smallest FOV that would be compatible with an adequate diagnosis.[2]

1.2.2.3.2 Tube Voltage (kV)

The relationship between tube voltage (kV) and radiation dose is approximately quadratic; where, increasing kV not only increases the number of X-rays produced over a given scan time, it also increases the overall and maximum energy.[32,36,38] For example, increasing the voltage from 60 to 90 kV can effectively triple the radiation dose if all other exposure parameters remain the same.[32,36] By this account, most CBCT units operate at or below 90 kV, and only a few can operate up to 120 kV.[32]

In terms of qualitative parameters increasing kV reduces image noise and beam hardening, but affects contrast through an interplay between X-ray scatter and beam penetration.[32] That said, a recent study by Park et al.[38] found that tube voltage did not exert a significant influence on overall image quality whereas tube current (mA) did. The authors proposed reducing tube voltage to decrease the radiation dose and minimize degradation of image quality.[38]

1.2.2.3.3 Tube Current (mA)

Tube current (mA) determines the number of X-rays exiting the tube per unit time(s) and is often represented in its combined form (mAs).[32] The radiation dose increases proportionately with the mAs in a 1:1 ratio; however, these changes do *not* affect the maximum or mean energy of the X-ray beam.[36] In terms of image quality, increasing mAs results in an increased quality via increased detector signal; however, since the mA required for a diagnostically acceptable image is coupled with time and kV, the choice of the milliamperage setting depends on the FOV and voxel size chosen.[37]

The effect of increasing both mA and kV is increased radiation dose, decreased noise (due to increased total amount of emitted X-rays) and increased contrast-to-noise ratio.[36] Both levels should be selected based on the patient size and the required image quality to increase optimization.[36]

1.2.2.3.4 Field of View (FOV)

The dimensions of the FOV are primarily dependent on the detector size and the beam projection geometry (e.g., source-object and object-detector distance).[32] Since FOV is primarily determined by the applied collimation of the primary X-ray beam, it can considerably reduce patient radiation dose.[32,36] Larger FOVs produce more scatter per detector area and higher beam divergence at the edge of the volume, which ultimately results in image quality deterioration.[32,36] In terms of image quality, Elshenawy and colleagues (2019) found that CBCT scans with smaller FOVs and voxel sizes are associated with higher linear measurements accuracy than those made with larger FOVs and voxel sizes.[40] Even with voxel size held constant, the authors reported that smaller FOVs are associated with higher CBCT linear measurements accuracy than those made with larger FOVs.[40] The FOV should be minimized according to the region of interest corresponding to the diagnostic task.[2,32,36]

1.2.2.3.5 Voxel Size

When the 3D image is generated, each voxel is assigned a gray value (i.e., generic, whole numbers, with lower numbers corresponding with darker voxels) according to its estimated X-ray attenuation.[32,36] In a discrete imaging system using a voxel matrix, each voxel can only be assigned a shade of gray, which on a radiograph should ideally represent the X-ray absorption of the object-part represented in that particular pixel/voxel.[41] Voxels are normally isotropic (i.e. cuboidal), orthogonally symmetric and they vary in size between 0.07 and 0.6 mm based on the CBCT unit and exposure protocols.[32]

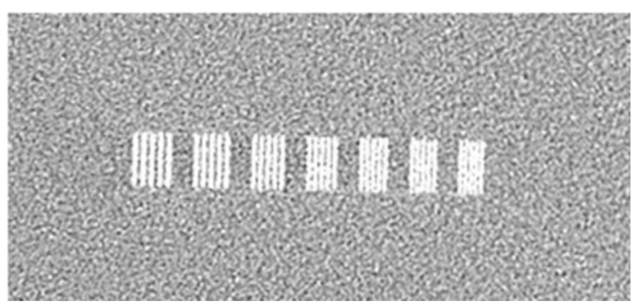
Smaller voxel size facilitates tasks requiring high detail, at the expense of increased radiation dose and noise. [32,37] For example, when voxel size is decreased, more x-rays (or photons) are required to increase the photon-per-voxel count at a constant image quality.[37] Depending on the FOV, the size of the element at the level of the detector varies, where small FOVs correspond with small detector elements (voxels).[37] For larger FOVs, the reconstructed voxel size cannot be as small as for small FOVs due to computational limitations.[32]

1.2.2.3.6 Spatial Resolution

Spatial resolution describes the ability to distinguish between separate structures that in close proximity.[2,32,36,42] Often it is used synonymously with voxel size, which makes technical sense because the voxel is the smallest unit that can be detected in an image.[43] However, while some studies have reported at least 50% of the variation in image resolution can explained by voxel size alone, the relationship between voxel and spatial resolution is not linear.[43] In fact, it is determined by many additional factors, such as focal spot size, beam projection geometry, scatter, patient motion, number of base images, partial volume averaging, reconstruction algorithms, and other parameters beyond the scope of this review.[32,36,42] These factors thus make it impossible to achieve a resolution equal to the voxel size.[42]

In the literature, spatial resolution has been measured both subjectively and objectively. Traditionally, it is assessed in line-pairs per millimetre (lpmm^{-1}) (Figure 1.4).[41]

Figure 1.4 Example of a CBCT image of a line pair phantom. Figure reproduced from Brüllmann & Schultz (2015).



In order to represent a line-pair a minimum of two voxels is necessary – one that represents the more dense line in a light colour and one that represents the space between two lines in a dark colour.[41] This suggests that the spatial resolution of a CBCT would be at least around double the voxel size. That said, the use of phantoms to determine spatial resolution will consistently overestimate the in-vivo ability of CBCT units, where a range of contrast in human patients that makes landmark identification more difficult.[43] For example, if a voxel represents an area of 75% lucent soft tissue and 25% opaque cortical bone, the voxel will appear more lucent than opaque. This *partial volume averaging* can make the boundaries between densities harder to accurately distinguish, and results in lower spatial resolution (Figure 1.5)[41,42]

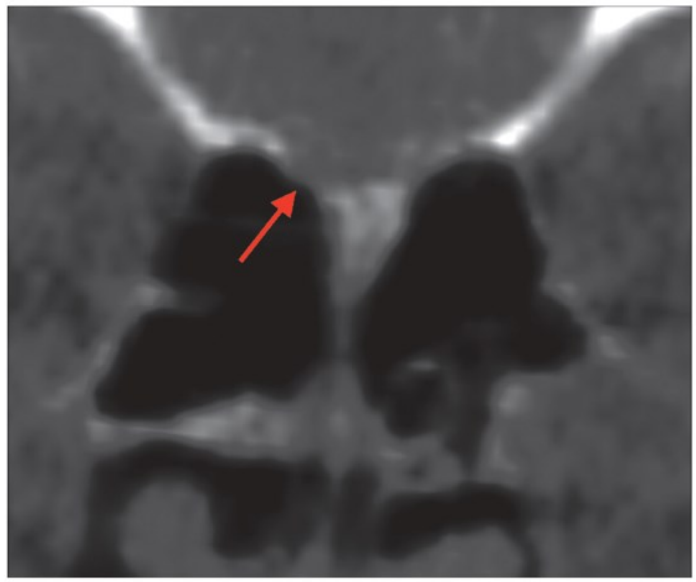


Figure 1.5 Coronal slice of sphenoid sinus. Red arrow indicates where partial volume averaging has created the false appearance of a communication between the sphenoid sinus and the anterior cranial fossa. Figure reproduced from Brüllmann & Schultz (2015).

Another measure of spatial resolution is *modulation transfer function* (MTF). [32,36,41] Though it is beyond the scope of this review, MTF is an objective measurement, whereby systems with higher spatial resolution have higher MTF (i.e. they are better able to transfer high-frequency image information).[36]

1.2.2.3.7 Contrast Resolution

Contrast resolution is the ability to discriminate objects of different density and distinguish them from noise by examining the difference in mean voxel value between two regions of an image.[32,36,38] It describe large, slowly varying characteristics of the image rather than small details (for which spatial resolution is a better measure).[36,38] That said, while CBCT is ideal for imaging relatively high attenuating structures (i.e. teeth, bone), it lacks the ability to record subtle changes in attenuation of soft tissue.[32] It is limited by scatter radiation, which is affected by the dynamic range (i.e. the detectable range of exposure values) of the detector, the exposure factors (i.e. kVp, mAs, FOV, voxel size) and the bit depth of the reconstructed image, as well as display settings (i.e. window/level).[32,36] Moreover, it is related to other quality parameters such as spatial resolution and noise. In fact, contrast is measured using the same formula as Noise – The *Contrast to Noise Ratio* (CNR) describes mean difference in gray values averaged over their variance.

1.2.2.3.8 Noise

Image noise in radiography is random the variability in voxel values in an image of uniform density.[32,36,38] It can vary by the source system (filtration, focal spot size, FOV size), technical parameters (kVp, mAs) and reconstruction parameters (voxel size) (among others).[37,38,42] Noise and spatial resolution are often managed in a trade-off, since many factors that improve one (e.g. voxel size, reconstruction filter etc.) degrades the other.[36] While noise can be reduced by adjusting the technical parameters to increase the energy at the detector, the main cause of noise is scatter radiation, which will increase as the FOV increases.[32,36,38,42] As such, the easiest way to decrease noise from scatter is to use the smallest FOV that encompasses the region of interest.[42]

1.2.2.3.9 Artifacts

Artifacts in radiography are appearances in the image that do not correspond to the physical reality, yet are deterministic (i.e. non-random) with respect to the projection data.[32,36] For example, *Metal artifacts* are the result of high absorption of X-rays by metal objects, and the inability of the reconstruction algorithm to cope with this, leading to dark and bright regions and streaks in the vicinity of the metal (i.e. *beam hardening*), which ultimately affects the visibility of other nearby structures.[32,38] Since the presence of a metal in the FOV renders the image unreadable in certain areas, metal objects should be removed prior to scanning (e.g., removable prosthesis, orthodontic brackets).[32,36,38] The adjustment of exposure parameters such as kV and mAs has little effect on metal artifacts, and the effect on radiation dose is disproportionate.[32,36]

Another relatively common type of artifact in CBCT is due to *patient motion*. If a patient moves during the scan by more than the voxel size, the result is motion blur, or a double contour.[32,36] This occurs when the projections acquired after the movement are back projected into the volume, allocating gray values from neighboring structures, instead of the correct structure, to the voxel.[41] In general, both technical parameters (i.e. FOV, voxel size and scan time) and patient factors (i.e. age, medical history) will affect the likelihood of patient motion artifacts in a 3D image. That said, studies have cited normal physiologic function (i.e. breathing, heartbeat, swallowing, etc.) as capable of producing motion artifacts up to 80 microns.[32,41] The most effective way to limit movement artifacts is to decrease the scan time; however, reducing the scan time also reduces the number of frame acquisitions (raw data) which can leads to under-sampling and reduction in image quality,[32,42]

1.2.3 Image Visualization

1.2.3.1 Reformatting

There are three types of 2D reformatting possible of the 3D volume: multiplaner, oblique and curved. In multiplaner reformatting, the 3D reconstruction can be viewed as a series of 2D cross-sectional images in axial, sagittal and coronal views which are inter-related through cross-sectional lines (crosshairs).[36] In oblique reformation the user is able to cut through the volume at any angle, either by rotating the image itself or rotating the intersection lines (as well as drawing new lines).[36] Since voxels in oblique planes are not aligned either horizontally or vertically, oblique reformation requires estimation algorithms.[36] For manually drawn curved planes (i.e. in a reformatted panoramic x-ray or cross-sectional images) it is often not possible to visualize the upper and lower dental arch in one image unless a "ray sum" of these synthetic panoramic views can be calculated that resembles an image acquired from a panoramic radiograph.[36]

1.3 The Role of CBCT in the Study of Alveolar Response

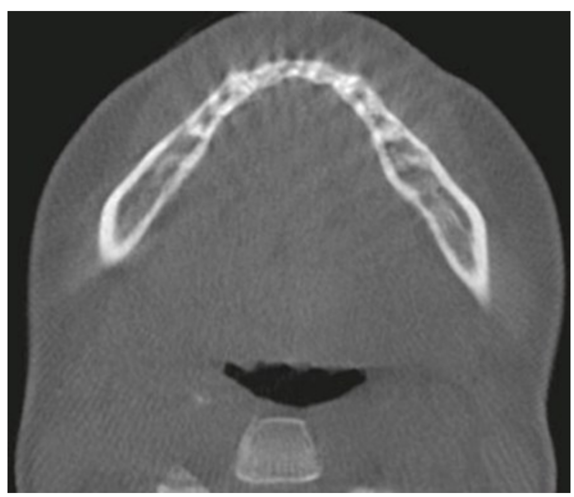
Radiographs are necessary diagnostic records required to establish a diagnosis and inform dental treatment. In the pre-treatment assessment of the orthodontic patient, radiographs supplement clinical diagnosis and cast and/or virtual models.[39] It is a widely available, technically simple, low-cost, rapid acquisition radiographic procedure.[39,44] Compared to traditional two-dimensional (2D) imaging, cone beam computed tomography (CBCT) enables the practitioner to visualize the patients head and neck anatomy in three dimensions (3D). Of utmost importance is its ability to allow for unique reconstruction and segmentation of patient anatomy – namely via software techniques – which enables 2D and 3D visualization of the maxillofacial skeleton, airway space and soft tissue boundaries.[39] An added bonus is that the linear (e.g. lateral and posteroanterior cephalometric images) and curved planar projections (e.g. simulated panoramic images) traditionally used in orthodontic diagnosis, cephalometric analysis, and treatment planning can be derived from one single scan.[39] Specific to orthodontics, the alveolar and soft tissue effects of treatment mechanics (i.e. proclination and expansion), which have been difficult to visualize using study models and traditional radiographic imaging, are easily assessed with CBCT.[45]

While there are many methods used to measure CBCTs in orthodontics (i.e. segmentation, 2D radiograph rendering, linear measurement, etc.),[4,39] this review will cover only those methods relevant to the present study.

1.3.1 Visualization of Dentoalveolar Morphology

Simply put, the dentition exists within an alveolar housing bound facially and lingually by cortical plates of bone. These hard-tissue boundaries provide the clinician with an understanding of the facio-palatal/lingual space available for dental procedures such as, dental implants, bone grafts, temporary anchorage devices (TADs), and orthodontic movement of teeth (i.e., retraction, arch expansion or labial movement of incisors)(Figure 1.6).[32] Specific to orthodontics, evaluation of the 3D limits of the alveolar housing can be an important consideration for biomechanics in certain patients (i.e. bimaxillary protrusion, compromised periodontal status, maxillary deficiency, and/or clefts of the alveolus).[13,32,42,46–49]

Figure 1.6 Thin alveolar bone width associated with roots of the mandibular incisor teeth. This restricts the range of orthodontic movement possible for these teeth, without dehiscence or fenestration of the roots through the cortical plate. Partially adapted from Scarfe et al. (2018).



CBCT is indicated for skeletal discrepancies. A review of the literature supports the use of CBCT in orthodontics for its high diagnostic value and relatively low radiation dose (Appendix A, Table A1.11).[32,36,39,42] Additionally, CBCT is the only evidence-based radiographic imaging modality used to facilitate the visualization and assessment of alveolar bone in three dimensions. It has been shown to be markedly superior to intraoral imaging and 2D radiography in the detection of bone defects such as dehiscence and/or fenestration.[32,48]

1.3.1.1 Image Reformatting

In general, CBCT images require image reconstruction, therefore error in the reconstruction process may result an inaccuracy in the linear measurements conducted using CBCT.[50] This is an important consideration in the selection of the orientation of the reconstructed image, where changes to the image slice (i.e. orientation, thickness, interslice interval) are often operator-determined and subject to inconsistency.[50] For example, Nikneshan et al. [50] found that by changing the slice orientation within the range of -12°to +12° with respect to the occlusal plane during image reconstruction, the accuracy of linear measurements using CBCT decreased. This is similar to findings in other studies, where changing the slice orientation changed the value of a linear measurement.[50,51]

Specific to buccal alveolar bone, measurements related to individual teeth on CBCT orthogonal and cross-sectional images is highly dependent on the orientation of the sectional plane.[32,42] This is especially true for teeth with tipped roots, where the entirety of the root cannot be visualized without scrolling through the volume. It is also true for rotated and/or crowded teeth where the thinnest portion of bone might not correspond to the buccal surface of the root (Figure 1.7).[32,42] Therefore, it is recommended that buccal alveolar measurement be taken only after sectioning the image according to the long axis of the tooth in order to provide accurate and reliable measurements over that specific teeth.[32]

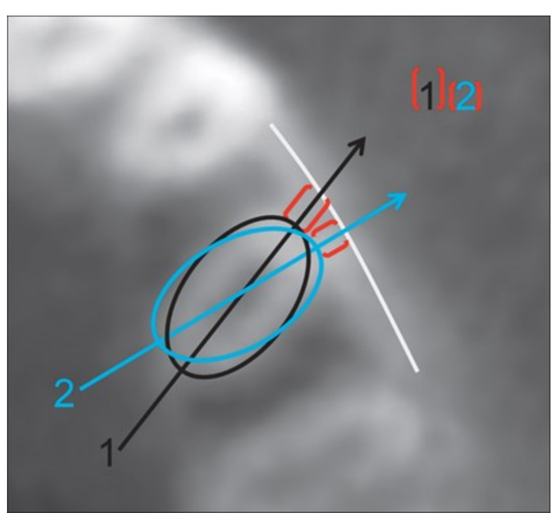


Figure 1.7 Axial slice of rotated premolar showing the effect of rotation on buccal bone thickness. The black arrow (1) indicates the rotated tooth, and the blue arrow (2) indicates the derotated tooth. Note the thinning of bone after the tooth is derotated (settings: 0.36‑mm voxel,1.08‑mm slice thickness, 12‑in FOV). Adapted from Molen, et al. (2010).

1.3.1.2 Identification of Landmarks using CBCT

To measure alveolar bone, enamel and alveolar crest landmarks need to be identified, including the *cementoenamel junction* (CEJ) and *alveolar bone margin* (ABM).[52] The CEJ is the junction between enamel and cementum.[2,32,48] Accuracy of identifying this landmark, defined by two tissues with different densities, is limited by the size of each voxel in the image with error from *partial volume averaging*.[32,48] Alternatively, the ABM is the junction between cementum and bone, two tissues with similar densities.[48] The accuracy of identifying this landmark is limited by the physical spatial resolution of the image, which can be determined by testing the CBCT unit with a resolution phantom.[32,36,48] Knowing this value, the clinician can expect areas with bone thickness less than the that of the CBCTs spatial resolution may not be fully visualized on the volume.

Studies have cited much ambiguity in identifying the alveolar bone on CBCT imaging as a result of its proximity to the cementum as well as its often submillimetre buccolingual dimension (see section 1.4).[52] CBCT images have become known to have high *specificity* and a high *negative predictive value* for both dehiscence and fenestration.[48,53] This means that while CBCT can give false positives for bony defects, it is unlikely to give false positives for the presence of alveolar bone.[53] For example, Leung et al.[48] found a high false-positive rate, with three times the number of defects detected on CBCT images compared with direct examination.[47] Similarly, other studies found that when the buccal plate was evaluated with CBCT, it was consistently absent in places where the alveolar bone was known to be thinnest based on the tooth, its location in the arch and which arch it was in.[54]

Finally, the presence of a mucogingival defect always indicates the presence of a dehiscence or loss of alveolar bone; however, the presence of a dehiscence does not necessarily lead to a mucogingival defect.[28,55] Inflammatory and soft tissue alterations cannot be diagnosed with CBCT, as cone beam imaging is quantitative and not qualitative.[56,57]

1.3.2 Accuracy of Linear Measurements

The ability to make precise measurements is critical for basing clinical decisions in diagnosis, treatment planning and prognosis.[35] In orthodontics, linear measurements are routinely used to measure the distance between anatomical landmarks both clinically and radiographically.[35,50] A review of the literature suggests variable accuracy when using CBCT for the linear measurement of alveolar bone. For example, Timock et al.[47] compared CBCT with direct measurements on cadavers for buccal bone height and thickness and reported mean absolute errors between the two (0.30 and 0.13 mm, respectively) which were not statistically significant. Similarly, a systematic review by Fokas et al.[44] found that the majority of studies comparing CBCT linear measurements with the "gold standard" (usually digital calipers measuring cadaver skulls) reported submillimeter differences without a statistically significant difference. That said, the range of differences between measurements often exceeded 1mm, which can be clinically significant in dentistry depending on the proposed treatment.[44]

Morais et al.[25] suggested that the accuracy of linear measurements of thin objects in CBCT presents limitations related to the image resolution or *partial volume averaging*. This means it is difficult to detect the presence of bone on the images in sites in which the bone has the same thickness or less than the voxel size.[25,48] The relationship between voxel size and linear measurement accuracy was corroborated by Sun et al.'s[58] study which found that measurements on CBCT for 0.25 mm voxel size images were closer to direct digital caliper measurements compared to 0.4-mm images.[59] While decreasing the voxel size would can offer better image resolution, it comes at the expense of exposing patients to a higher dose of ionizing radiation and measurement accuracy is always not linearly dependent on voxel size alone.[25,48] For example, Damstra et al.[60] found no difference between anatomic and CBCT measurements for images with 0.4 mm and 0.25 mm voxel sizes on CBCT when compared to a digital caliper.[59] Based on voxel size, other studies also found no statistically significant difference between linear measurement accuracy on CBCT images with 0.4mm voxel size and those with 0.125mm,[61] 0.2mm, or 0.3mm voxel size,[62,63] compared to measurements with digital calipers.[59] As such, other parameters that affect not only partial volume averaging, but also spatial resolution (which is larger than the voxel size) should be considered in determining the accuracy of linear measurement using

CBCT. As previously mentioned, spatial resolution can be determined by testing the CBCT unit with a resolution phantom.[32,36,48]

1.4 Gingival Recession

Cortellini & Bissada define the normal mucogingival condition as the “absence of pathosis (i.e. gingival recession, gingivitis, periodontitis).” [64] A mucogingival deformity is a departure from the normal dimension and morphology of and/or interrelationship between gingiva and alveolar mucosa.[64,65] *Gingival recession* (GR) is one such mucogingival deformity and is defined as the apical shift of the marginal periodontal tissues relative to the CEJ resulting in exposure of the root surface to the oral environment (Figures 1.8 & 1.9).[64–66]

Figure 1.8 High muscle attachment of the frenum resulting in direct pull on the gingival margin. Adapted from Chatzoopoulou & Johal (2015).

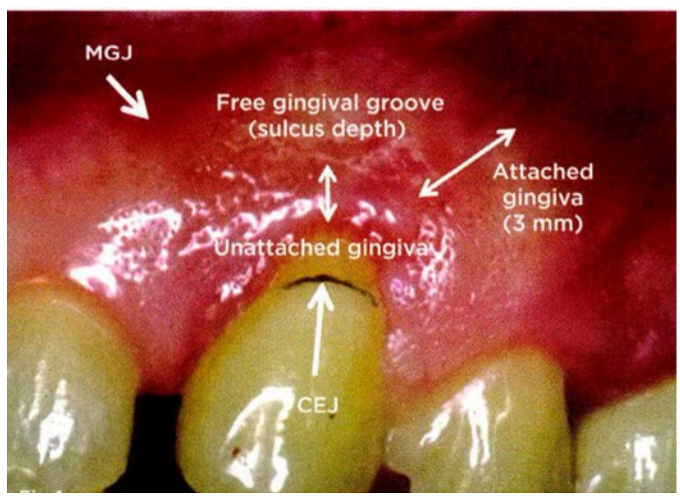


Figure 1.9 Clinical landmarks related to GR: The MGJ, the free gingival groove, the CEJ and the attached gingiva. Note that the CEJ is darkened to make it obvious on the photo. Adapted from Miller (2018).

If left untreated, GR is highly likely to progress and increase in the recession depth over time, even with good oral hygiene.[64,67] The presence of GR does not impair the long-term survival of teeth; however, it has been associated with esthetic problems, hypersensitivity, root caries, tooth abrasion and reduced *Oral Health Quality of Life* (OHQoL).[64,65,68]

1.4.1 Diagnosis and Classification

Since the diagnosis of GR is reliant on the presence of the CEJ, difficulties arise when the CEJ is not distinct or absent (i.e. due to restorations or lesions).[64,69] While this can be overcome using techniques such as anatomical CEJ reconstruction,[64] achieving an accurate diagnosis without an essential landmark may impose difficulties with diagnosis and underestimate the prevalence of GR in the population.

The 2017 World Workshop on periodontal disease marked a turning point for the diagnosis of mucogingival defects.[64] Prior to 2017, GR was classified using Miller's[70] classification which used the interproximal bone height and the *mucogingival junction* (MGJ) as primary variables for the classification.[71] While the main advantage of this method is the ability to predict outcomes based on the diagnosis and classification of the lesion,[71] it lacked in information about the amount of root prominence present, despite radiographic evidence of interproximal alveolar height. The second major drawback of Miller's classification is the absence of criteria to classify marginal tissue recession with inter-proximal bone loss, which does not extend to the MGJ.[72] To improve this classification, an updated recession classification based on the interdental *clinical attachment level* (CAL) measurement has been proposed by Cairo et al:[64,73]

- **Recession Type 1 (RT1)**: Gingival recession with no loss of interproximal attachment. Interproximal CEJ is clinically not detectable at both mesial and distal aspects of the tooth.
- **Recession Type 2 (RT2)**: Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss (measured from the interproximal CEJ to the depth of the interproximal sulcus/pocket) is less than or equal to the buccal attachment loss (measured from the buccal CEJ to the apical end of the buccal sulcus/pocket).
- **Recession Type 3 (RT3)**: Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss (measured from the interproximal CEJ to the apical end of the sulcus/pocket) is greater than the buccal

attachment loss (measured from the buccal CEJ to the apical end of the buccal sulcus/pocket).

The Cairo Classification a treatment-oriented classification to forecast the potential for root coverage through the assessment of interdental CAL.[64,73]

1.4.2 Prevalence

A frequently observed clinical condition, GR is reported to be present in the majority of the permanent dentition.[71,74] It is more prevalent and severe at buccal than at interproximal surfaces of teeth and is often associated with wedge-type defects at the cervical aspect of the affected tooth.[27,71,74,75] While it is overall more prevalent in the mandible than the maxilla;[27,76] Murray[77] found that GR is most prevalent on the following tooth groups, in decreasing order:

1. Mandibular incisors
2. Maxillary first molars
3. Mandibular first molars
4. Maxillary and mandibular premolars
5. Maxillary second molars
6. Mandibular second molars
7. Maxillary and Mandibular canines
8. Maxillary incisors

The prevalence of GR is age dependent.[27,71,74,78–84] While mucogingival problems in primary dentition are rare,[85] the prevalence ranges from 1-19% in mixed dentition (especially the mandibular incisors) [85,86] and increases in the permanent dentition over time.[85] For example, Kassab and Cohen[74] found that GR associated with labially positioned teeth occurred in 40% of patients aged 16 to 25 years and increased to 80% of patients in the 36 to 86 years age group. Other epidemiological studies suggest that 50-60% of those 18-65 have at least one site with GR.[71,74,80] This number increases to up to 90% for those over 50 years old.[27,71,74,81] While some studies have reported an increased prevalence of GR among males compared to females;[78–80,82,87] others found no difference in prevalence between genders in adolescence and young adulthood.[27,83]

Globally, GR is highly prevalent in regions with both high levels of oral hygiene as well as those with barriers to accessing care.[27,71,81,87] In low *socioeconomic status* (SES) populations it is associated with calculus accumulation and progression of destructive periodontal disease, whereas in high SES populations it is associated with traumatic toothbrush abrasion.[71,82] GR has been diagnosed more often in African American populations than other racial/ethnic groups. It is more commonly associated smoking,[82] mal-aligned teeth,[74,78] and trauma.[71,82]

1.4.3 Pathophysiology

Despite its prevalence, GR is considered to be a pathologic process. The exact mechanism of GR is not well understood; however, evidence suggests that the predisposing mucogingival problems associated with GR may originate in the primary dentition.[86] For example, the position in which the tooth erupts (i.e. labially or lingually) through the alveolar process and its final position in the arch will influence the amount of *keratinized tissue* (KT) around that particular tooth.[85]Additionally, the labio-lingual width of alveolar bone compared to the size of the tooth will affect the overlying bone and KT over the tooth.[85] Much controversy exists, however, as to how much vertical growth and development will contribute to self-correction of a high risk area.[85,86,88]

While the actual mechanism remains unclear, it is believed that GR is the result of chronic inflammation, which can be exacerbated by predisposing factors. The apical migration of gingival tissue can be understood in four stages (Figure 1.10):[88]

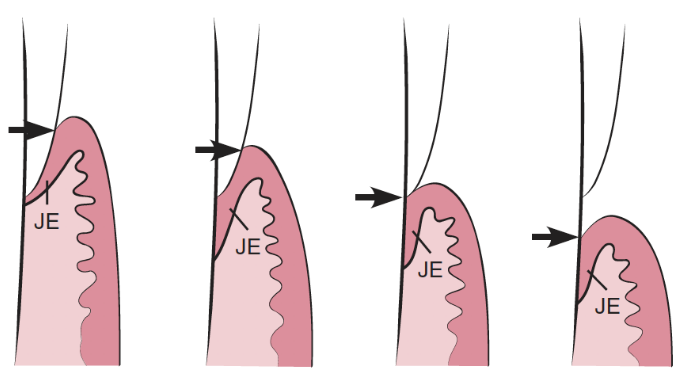


Figure 1.10 Diagrammatic representation of the four steps of passive eruption according to Gottlieb and Orban.(105) 1. The base of the gingival sulcus (arrow) and the junctional epithelium (JE) are on the enamel. 2. The base of the gingival sulcus (arrow) is on the enamel and part of the junctional epithelium is on the root. 3. The base of the gingival sulcus *(arrow)* is at the cemen-toenamel line, and the entire junctional epithelium is on the root. 4. The base of the gingival sulcus *(arrow)* and the junctional epithelium are on the root. Adapted from Carranza's Clinical Periodontology (13th ed.) – Ch. 3, p. 33.

Stage 1: The teeth reach the line of occlusion. The junctional epithelium and the base of the gingival sulcus are on the enamel.

Stage 2: The junctional epithelium proliferates so that part is on the cementum and part is on the enamel. The base of the sulcus is still on the enamel.

Stage 3: The entire junctional epithelium is on the cementum, and the base of the sulcus is at the cementoenamel junction. As the junctional epithelium proliferates from the crown onto the root, it does not remain at the cementoenamel junction any longer than at any other area of the tooth.

Stage 4: The junctional epithelium has proliferated farther on the cementum. The base of the sulcus is on the cementum, a portion of which is exposed. Proliferation of the junctional epithelium onto the root is accompanied by degeneration of the gingival and periodontal ligament fibers and their detachment from the tooth.

1.4.4 Risk Factors related to GR

While no single causative factor has been identified, and the pathogenesis of gingival recession remains unclear,[76,80] identifiable risk factors related to GR have been well studied and divided into two groups: predisposing and precipitating factors.[76,89] Predisposing factors are associated with inherent biological characteristics increasing the risk GR development, also known as anatomic factors. Patient factors, or precipitating factors can lead to an acceleration of the defect; however, they are modifiable.[71,76,89] Table 1.2 lists the risk factors according to their category. Others have identified periodontal disease,[27] anatomic variations (i.e. dehiscence and abnormal tooth position), iatrogenic factors related to the location of restoration margins,[71] previous periodontal surgery, high muscle attachment and aberrant frenum, and the use of smokeless tobacco, and gingival lesions associated with bacterial plaque.[65,74,80] For the purpose of this review, only hard and marginal soft tissue factors will be reviewed as epidemiological data was not collected for the sample.

Table 1.2 Predisposing and precipitating factors associated with GR.[68,72,85]

Predisposing/Anatomic Factors	**Precipitating/Patient Factors**
• *Alveolar bone dehiscence* • *Gingival biotype* • Skeletal pattern • Narrow symphysis • Ectopic tooth eruption or morphology	• *Plaque, biofilm* • Traumatic tooth brushing • Traumatic overbite • Age • Smoking • Parafunctional habits • Pregnancy • Piercing • Inappropriate orthodontic treatment mechanics

1.4.4.1 Predisposing Factors

1.4.4.1.1 Periodontal Biotype

Overall, the distinction among different biotypes is based upon anatomic characteristics of components of the masticatory complex, including periodontal biotype, which includes *KT thickness* (KTt) and *KT width* (KTw); bone morphotype; and tooth dimension.[64] The morphology of gingiva is associated with the underlying bone anatomy, tooth length, form, and shape.[65] When combining information from cone-beam computed tomographs, diagnostic impressions, and clinical examinations, the periodontal biotype can be related to gingival thickness, facial plate thickness, position of alveolar crest, zone of keratinized tissue and the gingival architecture.[65] The quantity of the hard and soft tissues adjacent to the dentition may play an important role in the development of recession.[84]

1.4.4.1.2 Bone Morphology

In the absence of pathology, studies have identified a *bone morphotype* for mean buccal bone thickness of 0.343mm (±0.135mm) for thin biotype and 0.754mm (±0.128mm) for thick/average biotype.[64] The alveolar bone adjacent a tooth root surface is not uniform in bucco-lingual width. For example, in CBCT studies, buccal bone has been shown to be thinner adjacent to the first premolars, which also exhibited the highest prevalence and extent of recession[83] compared to the other maxillary posterior teeth.[90] For more information on buccal alveolar bone and CBCT, see section 1.3.

Buccal alveolar bone may have a *fenestration* (i.e. isolated perforations) and/or a *dehiscence* (i.e. deficiencies in alveolar bone height), especially on the buccal/facial surface.[32,55,71] The difference between the two diagnoses lies in whether or not the marginal bone is in-tact. For example, a dehiscence is a defect in which the root surface is denuded from marginal bone, while fenestrations occur when the marginal bone is present, while the denuded root surface is covered in only periosteum and soft tissue (Figure 1.11).[88] Together these defects occur on approximately 20% of the teeth; they occur more often on the facial bone than on the lingual bone, they are more common on anterior (i.e. 9.9–51.6%) teeth than on posterior teeth (i.e. 13.9–84.5%), and they are frequently bilateral.[32,88,91] They are most commonly found over the maxillary canines and first premolars.[32,92] Both dehiscence and fenestration reduce the bony support for the teeth and, in presence of plaque-induced inflammation, can be detrimental to the health of the teeth and the periodontium.[48]

Figure 1.11 Dehiscence on the canine and fenestration of the first premolar. Adapted from Carranza's Clinical Periodontology (13th ed.) – Ch. 3, p. 46.

The cause of alveolar defects is not clear.[88] While there is microscopic evidence of lacunar resorption at the margins, the anatomical mismatch between the bucco-lingual width of the alveolus compared to the tooth size, or root contour may reduce the alveolar coverage of a root surface. [85,88] Additionally, the amount of space loss, crowding and path of eruption may cause a tooth to erupt into an area with less alveolar support.[85,88]

1.4.4.1.2.1 Dehiscence

While the presence of a dehiscence is a prerequisite for GR,[28,45,55,76,84] it is not pathognomonic with its presentation.[27,46,55,92,93] Since the crest of bone normally exists within 2-3mm of the *gingival margin*,[55,71] there is much variation in how a dehiscence is diagnosed with respect to the CEJ. For example, some authors consider a bone dehiscence to be when the CEJ to crestal bone distance is greater than 2mm,[46,92] 3mm,[48] and even 4mm.[94] While the prevalence of dehiscence is highly variable most authors agree that its prevalence increases with crowding, age, orthodontic treatment.[3,28,45,85,92,95] For example, Jäger et al.[95] observed that, before orthodontic treatment, 20% of patients exhibited dehiscence defects. In these cases, tooth position can primarily affect the bone morphology around a tooth, such that a buccally orientated course of eruption may therefore be prone to development of dehiscence sand recession.[71] That said, it's been observed that dehiscence increases to up 90% of patients with at least one tooth with dehiscence after orthodontic intervention.[95,96]

Alveolar bone width has been radiographically measured with *cone-beam computed tomography* (CBCT) which has high diagnostic accuracy at the expense of exposure to radiation (see chapter 1.3 and 1.4 for more information).[64,97]

1.4.4.1.3 KT Thickness

There is a body of evidence to support that the *quality* of attached gingiva is a predictive factor of GR.[27,64,71,76,98–100] However, the definition of the condition is not universal and the parameters and measurement methods vary among studies.[56] For example, Seibert and Lindhe[101]introduced the term *periodontal biotype* to describe the morphological characteristics of the periodontium; whereby a thin biotype is characterized by high-scalloped thin soft tissue, slender teeth, and long interproximal gingival embrasure spaces with small contact points located at the incisal third of teeth, and flat gingivae were combined with square tooth form and thick bone.[56,65] Some years later, Müller and Eger[102] preferred the term periodontal *phenotype* for describing periodontal tissues' morphology as either thin-scalloped and/or thick-flat.[56] While it is found in the literature as "gingival" or "periodontal" *biotype*, *morphotype* or *phenotype*,[64] the consensus is that thin (vs. thick) KT is more at risk for the development of GR.[71]

KTt ranges from 0.63 (±0.11) mm to 1.79 (±0.31) mm.[64] Some authors have suggested KTt varies by gender.[88,103] For example, De Rouck et al[103] found that females with slender teeth most often had thin and clear gingiva whereas a clear, thick gingiva was found primarily in males.[88] However, the consensus is that overall, a thick biotype (51.9%) is more frequently observed than a thin biotype (42.3%).[64] KTt is difficult to assess qualitatively as it can be confounded by root prominence.[104] For example, thicker gingival tissue has been reported buccal to the maxillary second premolars than the first premolars and molars, which typically have less root prominence.[102]

Recently, Nikiforidou et al.[56] found that KTt at the level of the CEJ was highly related to tooth and alveolar factors. For example, KTt was found to be positively correlated to thickness of the labial plate, crown form and the distance between bone crest and gingival margin midbuccally and negatively correlated with the distance between bone crest and CEJ midbuccally. Should GR develop, its progression depends on tissue type. For example, Baker and Seymour[105] found that after the onset of GR, it progresses rapidly in thin tissue type, but is more prone to pocketing in thick tissue types.[71,76,98]

It is assessed most commonly by *probe visibility* while in the facial sulcus (Figure 1.12).[64] Gingiva is defined as thin (≤1.0 mm) or thick (>1 mm) upon the observation of the periodontal probe visibility through the gingiva.[64] This method was found to have a high reproducibility by De Rouck et al,[103] showing 85% inter-examiner repeatability.[64] Other common methods include: *transgingival probing* (accuracy to the nearest 0.5 mm) under local anesthesia, which could induce a local volume increase and patient discomfort, and *ultrasonic measurement* (accuracy within 0.5 to 0.6 mm range) which is not as accurate in the second and third molar areas.[64,106]

Figure 1.12 Periodontal probing on the maxillary central incisors. Adapted from Vassilopoulos et al. (2013).

Finally, KT biotype can be evaluated by *visual assessment* and, more recently, with *CBCT*.

In particular, visual assessment relies on the identification of specific features associated with each biotype, as described previously, and it is subject to personal perception.[65] When tested against expert diagnosis using the probe visibility test (gold standard), Cuny-Houchmand et al.[107] found that visual assessment was not an accurate method for gingival biotype diagnosis. Similarly, the precision of visual assessment for KTt amongst practitioners with different levels of training was assessed by Eghbali et al.[108] The authors concluded that visual inspection may not be considered a valuable method identify KTt since the biotype was only accurately identified in approximately half of the cases, irrespective of the clinician's experience.[108] Others have identified the need for visual assessment of KTt when probing is not possible, as is often the case in children. For example, Dridi et al.[104] tested the diagnostic accuracy of the *gingival whitening test* (i.e. GW test) and the *blood supply test* (i.e. BS test) against the probe visibility test in mixed dentate children. The GW test ascertains a thin tissue diagnosis with whitening of the attached gingiva as the lips are retracted, due to its low resistance under tension, while the BS test uses the visibility of capillaries located in the chorion to determine tissue thickness (Figure 1.13).[104] That said, compared to the probe visibility test, both the GW and the BS test had low sensitivity to thick tissue type; however, the GW test had high specificity for thin tissue type.[104]

Figure 1.13 Gingival whitening and blood supply tests for thin (a) vs. thick (b) keratinized tissue biotype. Adapted from Dridi et al. (2018).

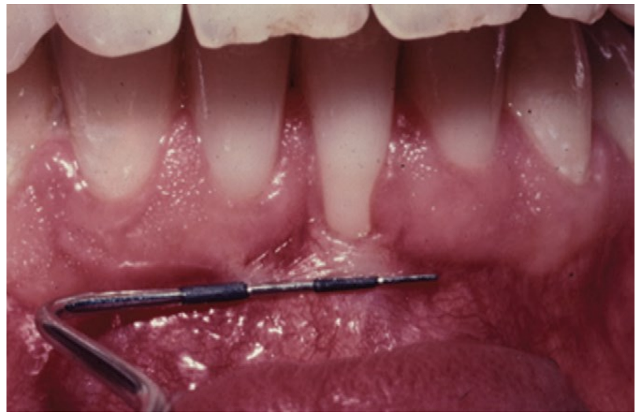
1.4.4.1.4 KT Width

KT width (KTw) is a measurement of the vertical dimension of KT, measured from the *free gingival margin* (FGM) to the MGJ (Figure 1.14).[88,109] Combined with the *probing depth* (PD), it contributes to the measurement of *attached tissue* (AT) around a tooth (i.e. KTw – PD = AT).

KTw <2mm has been identified in the literature as a predisposing risk factor to developing GR.[64,76,98,110,111] Lang and Löe[111] suggested that a minimum width of attached tissue was protective against periodontal breakdown. They found that even in the absence of clinically detectable plaque or other risk factors (i.e. aberrant frenal attachment), areas with <2 mm of KTw (i.e. <1 mm of attached tissue) remain inflamed and at risk of attachment loss.[111] This definition has stood the test of time such that the 2015 *American Academy of Periodontology* (AAP) Consensus Statement on Mucogingival Conditions defined an inadequate amount of KTw as < 2mm, of which < 1mm is attached.[71,100] Today, the general consensus is that a minimum amount of KTw is not needed to prevent attachment loss when optimal plaque control is present;

Figure 1.14 Keratinized gingiva and oral mucosa form the mucogingival junction (MGJ) as denoted by the blue dashed lines. Adapted from Carranza's Clinical Periodontology (13th ed.) – Ch. 3, p. 20.

Figure 1.15 Mucogingival defect extending into the alveolar mucosa with lack of adequate KTw and AT. Adapted from Carranza's Clinical Periodontology (13th ed.) – Ch. 5, p. 77.



however, approximately 2 mm of KT and 1 mm of attached tissue are desirable around teeth to maintain periodontal health since a movable gingival margin would facilitate the introduction of microorganisms into the gingival crevice (Figure 1.15).[64,111]

KTw varies by location, progression in time and plaque control. With respect to regional variation, there is an overall decrease in KTw from anterior (3.5 to 4.5mm in the maxilla, 3.3 to 3.9mm in the mandible) to posterior (i.e., 1.9 mm in the maxillary first premolars and 1.8 mm in the mandibular first premolars).[88,112,113] The regions which consistently showed the narrowest KTw were the lingual surface of the mandibular anterior teeth and the buccal surface of the mandibular canines and first premolars.[111] In split mouth studies, teeth in a labio-or buccoversion had narrower zones of KTw than their aligned counterpart.[112] According to Bowers[112] areas with narrow KTw are often associated with a high frenal muscle attachment. Moreover, when GR was associated with a high frenal attachment, it usually more extensive than elsewhere in the mouth.[112] Allen et al.[114] suggest the high frenal attachment can predispose a site to GR in 2 possible ways: first, attachment close to the gingival margin compromises the plaque removal from the area, and secondly, by direct pull on the frenum on the margin.[71] Diagnosis of an aberrant frenal attachment can be done using a *tension test,* whereby gingival margin blanching due to tension on the associated part of the lip or frenum itself may warrant close observation or complete removal of the frenal attachment as a preventive or corrective measure.[71]

With respect to temporal variations, Bowers[112] found that there was an increase in the mean width of AT from deciduous to adult dentition and there was little change after maturity.

Maynard and Ochsenbien[85] found that in a sample of children ages 4-16yo, AT ≤1mm was only found in 12 – 19% of the permanent dentition. Since tooth eruption follows vertical growth, KTw <2mm is often of little consequence in growing patients.[86,88,113] However, knowing that the MGJ remains stationary throughout adult life, any changes in KTw can be attributed to coronal modifications (i.e. GR).[88,115]

1.4.4.1.5 Interdental Papilla

The interdental gingiva that occupies the interproximal space apical to the contact (i.e. the *gingival embrasure*) is called the *interdental papilla* (IP).[88] It is pyramidal with a tip at the contact point (Figure 1.16).[88] The amount of papilla fill is determined by several factors, including tooth shape, the location of the contact point and the distance of the osseous crest to the contact point (i.e. within 5mm).[116]

The IP is important in smile esthetics such that its absence results in a dark region known as a *black triangle* (BT).[65,116] The apical migration of interproximal crest >5mm from the contact point will lead to the development of interproximal tissue recession appearing as BTs. Clinically it's assessed with respect to its fill in relation to the CEJ and the contact point of the teeth.[106]

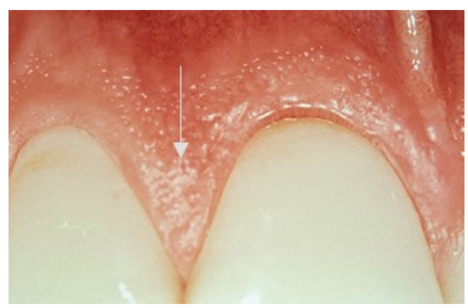


Figure 1.16 Interdental papillae (arrow) with a central portion formed by the attached gingiva. The shape of the papillae varies according to the dimension of the gingival embrasure. (Courtesy Dr. Osvaldo Costa.) Adapted from Carranza's Clinical Periodontology (13th ed.) – Ch. 3, p. 21

1.4.4.2 Precipitating Factors

1.4.4.2.1 Plaque Biofilm

GR has been related to inflammation in periodontal connective tissue, known as *gingivitis*.[14,27,105,117] It is characterized by a dense infiltrate of lymphocytes and other mononuclear cells, fibroblast alterations, leading to increased vascular permeability, continuing loss of collagen and proliferation of the junctional epithelium in response to the microbial challenge.[117,118] Clinically this presents as swelling, edema, redness and/or tenderness, and bleeding on probing that is reversible if the bacterial challenge is substantially reduced by improved oral hygiene.[64] The relationship between microbial plaque and developing gingivitis is well established. In fact, Löe et al.[120] demonstrated the relationship between microbial plaque and gingivitis whereby the cessation of oral hygiene consistently lead to the manifestation of gingivitis within 2 to 3 weeks in healthy adults.[118] With respect to GR, Baker & Seymour[105] described a process whereby the presence of microbial plaque around teeth provokes an inflammatory host response in the connective tissue, through the activation of innate and immune responses, which can result in attachment loss. Depending on the patient-specific predisposing and precipitating factors, this attachment loss can manifest as GR, pocket formation, or a combination of the two.[71,105]

While the primary etiological factor is microbial plaque, the tissue response can modified by systemic factors that contribute to host's inflammatory response.[118] This includes the endocrine changes associated with puberty, the menstrual cycle, pregnancy, and diabetes.[118,121–123] It should be noted that exacerbations of a gingival inflammatory response (i.e. due to pathogenic biofilms or modified by fluctuations in sex steroid hormone secretions) may be protective responses to an invading organism (Figure 1.17).[123] For example, gingival inflammation is normally fully reversible in otherwise healthy persons within weeks following the removal of local factors and reduction of the microbial load around the teeth.[118,119] Additionally, longitudinal studies[124] examining the natural history of attachment loss show incomplete conversion of long-term, chronic gingival inflammation to attachment loss. This brings to question the predilection for attachment loss at particular inflamed gingival sites and not others.[123] Due to the multifactorial nature of attachment loss, the take home is that while inflammation itself has been associated with mucogingival disease and periodontitis,[81] the presence of gingival inflammation is not necessarily pathognomonic with tissue breakdown and attachment loss.[123]

Figure 1.17 13yo female with hormonal-exaggeration of marginal and papillary inflammation in the absence of attachment loss. Adapted from Carranza's Clinical Periodontology (13th ed.) – Ch. 5, p. 58.

1.4.4.2.2 Toothbrush Trauma

The *toothbrushing* (TB) method has been proposed as the most important mechanical factor contributing to the development of GR.[27,64,71,74,77,78,81,87,105,125] For example, Khocht et al.[125] found that an aggressive cleaning technique (i.e. horizontal scrubbing with excessive force) and the use of hard tooth brushes may lead to the mechanical destruction of gingival tissues.[27] Addy et al.[126] found that traumatic TB may be associated with GR, with left-side buccal recession noted more frequently (likely due to right hand dominance in the population).[74] Additionally, in a sample of patients from Ohio State University College of Dentistry, Gorman et al.[78] found GR to occur more frequently in those with good rather than poor oral hygiene. This implies that TB technique, including duration, frequency of brushing, brushing force, frequency of changing toothbrushes, and hardness of bristles must also be considered when evaluating the role TB plays in a particular patient's etiology of GR.[64,71,78]

1.4.4.2.3 Orthodontic Treatment

Numerous sources have cited orthodontics as a precipitating factor in the development of GR, where the possibility of initiation or progression increases during or after orthodontic treatment.[27,28,64,83,85,87,127] Slutzkey & Levin[87] found a correlation was between severity and extent of recession to past orthodontic treatment, where 8.4% who reported past orthodontic treatment showed recessions of 3 mm or more compared with only 0.9% with no past orthodontic treatment. Additionally, 14.5% who reported past orthodontic treatment had three or more teeth with gingival recession compared with only 2.7% with no past orthodontic treatment.[87] It has been reported that the prevalence of GR related to orthodontic treatment is 5% to 12% at the end of treatment and up to 47% in long term follow up (5 years).[64] Susin et al.[82] found that the proportion of orthodontically treated patients in Brazil with severe GR (i.e. >3mm) increased almost 10-fold (6% to 54%) from the late teenage years (14-19yo) to adulthood (30-39yo).[83]

The mechanism by which GR is associated with orthodontic treatment is not well understood. Animal studies have demonstrated that the periodontium is somewhat resilient to iatrogenic dehiscence formation, depending on the presence of the other aforementioned risk factors and whether or not the tooth is retained.[16,17,47,71] For example, after an eight month retention period of buccally displaced lower incisors in Rhesus monkeys, Batenhorst et al.[17] found dehiscence formation, approximately 3mm of GR, elongation of the epithelium attachment and reorientation of the supra-crestal fibers. After three weeks of retention in a monkey model, Steiner et al.[16] also reported GR and reduction of connective tissue width in buccally displaced lower incisors. Conversely, after bodily advancement of teeth through the buccal plate in Monkeys, Wennström et al.[14] demonstrated that bodily movement of maxillary incisors resulted in deep and wide dehiscences not necessarily linearly related to the apical migration of the gingival margin. It is interesting to note that in the absence of retention, both Karring et al.[128] and Thilander et al.[15] found that it was possible to move teeth through the buccal alveolar bone and then back to their original position in the dental arch without loss of connective tissue attachment in Beagle dogs. However, when Engelking & Zachrisson[129] attempted to relocate buccally displaced incisors in monkeys after eight months of retention, the overall bone gain was approximately 50% with negligible soft tissue changes due to presence of inflammation and diseased root surface.

Many authors have stressed the importance of the presence of an alveolar bone dehiscence as a prerequisite for the development of GR,[15,16,28,71,109(p975),128,130] such that the *direction* of tooth movement is understood to be the critical link between orthodontics and GR. If buccal tooth movement results in reduced buccal dimensions of the periodontal biotype and lingual tooth movement results in the opposite,[109(p976)] orthodontically positioning teeth within the alveolus should help to maintain adequate bone volume and density and reduce the risk of GR.[14,28,71,85,89,109] Studies show that undiagnosed dehiscence can lead to greater potential for treatment relapse and/or mucogingival defects in expansion cases.[14,28,48,71,76,98,109] Buccal dehiscences in the maxillary arch have been demonstrated after arch wire expansion,[25,90,132] as well as rapid and slow maxillary expansion.[3,26,133] Within a year after treatment, small but significant losses of attachment on the maxillary posterior teeth have been reported for patients who underwent rapid palatal expansion compared with those who did not.[29,84] However, the literature demonstrates a range of responses to labial tooth movement in humans. For example, compared to untreated controls, the prevalence of individuals with GR in the lower anterior region was significant higher in treated cases,[27,99] which steadily increased after orthodontic treatment.[83] Additionally, the literature suggests that more proclined teeth had generally more apical migration of the FGM relative to the non-proclined or less proclined teeth[134–137] Contrary to these findings, many authors[76,99] found only a small number of patients (i.e. 15% of the teeth) experienced development or aggravation of GR with labial tooth movement.[76] These contradictory findings might be explained by the presence of the aforementioned factors related to GR. For example, Wennström et al.[14] observed that, in the presence of plaque induced gingivitis, bodily movement of teeth *within the alveolar housing* did not produce attachment loss. However, many authors[76,98,105] have observed that a thin periodontal biotype is more susceptible to complete breakdown than a thick one during facial movements. An additional consideration may be the amount of time allowed to lapse between debond and follow up. This may be due to 'stretching' of the buccal tissue that favors the destructive effect of the plaque associated inflammatory lesion in a region without adequate boney housing.[109(p978)] Moreover, if the tooth movement is expected to result in the establishment of an alveolar bone dehiscence, a soft tissue evaluation should be considered as a factor that may influence the development of soft tissue recession.[109(p978)]

Various periodontal challenges have been considered in the pathophysiology of GR associated with orthodontic treatment, including: site specific challenges (i.e. molar bands or extractions) or host-specific challenges (i.e. alteration of oral hygiene habits or plaque retentive nature of orthodontic appliances during treatment).[87,117,119,127,137] For example, placing bands in recently erupted teeth as the junctional epithelium is still adherent to the enamel surface can traumatize the attachment and cause the gingiva to can detach from the tooth.[119] This can result in the apical proliferation of the junctional epithelium with an increased incidence of attachment loss.[119] With respect to the inadvertent increase in plaque-related gingivitis, research has identified that a spectrum of responses may be observed, with some individuals developing a more pronounced inflammatory response for a given plaque challenge than others, thereby underscoring the importance of the host response.[87,117,119] To further this, it is interesting to note that even in the presence of excellent plaque removal, Zachrisson & Zachrisson[138] found evidence of gingival hyperplasia in the presence of orthodontic appliances.

Presence of gingival inflammation and baseline recession,[76] a thin gingival biotype,[76,98] a narrow width of keratinized gingiva[76,98] or a thin alveolar bone with respect to the direction of tooth movement[84] were found to correlate significantly with the development or increase in gingival recession in orthodontic patients.[27]

Inter-Rater Agreement	RES[139]		BASS[140]		PES[141]	
	Students	Teachers	Students	Teachers	Students	Teachers
High (>60%)	• Degree of GR wrt CEJ • lack of scar • FGM follows CEJ	• Degree of GR wrt CEJ • lack of scar • FGM follows CEJ • Color	• Degree of GR wrt CEJ • Color • Texture • Lack of scar	• Degree of GR wrt CEJ • Color • Texture • Lack of scar	• ST level (GR) • Texture • Color	• ST level (GR) • Volume • Shape - mesial papilla • Shape - distal papilla
Moderate (60%-40%)	• MGJ aligned • Color	• MGJ aligned	• Volume • Contour	• Volume • Contour	• Contour • Shape - mesial papilla • Shape - distal papilla	• Contour • Texture
Low (<40%)			• KTw	• KTw	Volume	• Color

Table 1.3 Visual assessment of the periodontium.

1.4.5 Visual Assessment of the Periodontium

Intra-oral photography is often used in dentistry, though it's often not standardized with respect to patient positioning, camera and settings, mirror positioning and software reformattting.[106] In a recent multi-center study, however, Le Roch et al.[142] demonstrated that the *Root Coverage Esthetic Score* (RES) system developed by Cairo et al.,[139] the *Before-After Scoring System* (BASS) developed by Kerner et al.,[140] and the *Pink Esthetic Score* (PES) system by Fürhauser et al.[141] were high to moderately reproducible tools for the evaluation of variables related to gingival soft tissue esthetics using standardized intraoral photography among students and teachers. The RES system had the highest reproducibility, followed by the BASS system, then the PES system.[142] The RFs evaluated and their reproducibility are summarized in Table 1.3. Similar to the aforementioned literature regarding visual assessment of KTt,[104,107,108] soft tissue volume had moderate to low agreement for the BASS and the PES systems.[142] Other parameters with low (<40%) reproducibility were KTw using the BASS system, and color match amongst teachers using the PES system.[142] That said, there was high agreement with respect to GR using all tools amongst both teachers and students and almost perfect agreement was found for papilla shape in the PES system amongst instructors.[142] Details with respect to the scoring system can be found in Chapter 2.

1.5 Systematic Review of Alveolar Changes for Maxillary Expansion using Damon® or Hyrax

1.5.1 Introduction

Mucogingival defects mostly consist both of periodontal recession and more importantly lack of adequate keratinized gingiva. It is defined as the displacement of the tissue apical to the cementoenamel junction and can affect any root surface.[69,89] They are a concern for the orthodontist since the presence of *gingival recession* (GR) can lead to reduction in keratinized tissue support/barrier around the tooth, poor aesthetics,[30,31] tooth hypersensitivity,[68,126] loss of periodontal support, difficulties in maintenance of oral hygiene,[67,111] and increased susceptibility to root caries.[84] Though gingival recession is reported to increase in both severity and prevalence with age (with greater than 90% of adults aged 50 years and above demonstrating its presence[143]), its aetiology remains unclear. Risk factors related to mucogingival defects include anatomic and morphological characteristics[64] (i.e. alveolar bone dehiscence, thin periodontal biotype, crowding, presence of aberrant fraenula and ectopic tooth eruption) whereas accelerated defect progression is often seen in traumatic tooth brushing and in relation to intra-oral piercings.[87]

It has been widely postulated that certain orthodontic treatment may also facilitate the apical migration of gingival tissue.[64,127] Orthodontic forces may contribute to periodontal recession by moving roots close to or through the buccal alveolar cortical plates leading to bone dehiscences,[16,76] cortical plate resorption, loss of periodontal attachment and unstable tipping of teeth.[84,131] Since lack of alveolar bone reduces the ability of gingival soft tissue to resist recessives forces (i.e. toothbrush abrasion, friction from piercings, etc.), it is postulated that a lack of proper alveolar bone support may be a risk factor to the apical migration of marginal gingiva leading to root exposure.[28] An association between orthodontic treatment and GR is of great interest to the specialty; especially in light of the shift toward a non-extraction, dental arch expansion approach to treatment.[144]

Maxillary dental arch expansion can be accomplished non-surgically with intra-oral appliances that facilitate opening the mid-palatal suture, buccal displacement of teeth or both, or surgically. Of particular interest for this review are non-surgical palatal expansion and the buccal displacement of teeth via intra-oral appliances. Three types of palatal expansion are commonly shown in the literature: *rapid maxillary expansion* (RME), *slow maxillary expansion* (SME), and *semi-rapid maxillary expansion* (SRME)[2,3] RME is associated with intermittent high-force systems[10]and tooth-tissue-borne appliances (e.g. Hyrax, Haas).[11] SME is often associated with continuous low-force systems (e.g. quad-helix appliances or coil springs).[2] Brunetto et al.[3] evaluated the buccal bone after RME and compared the results to SME. Both RME and SME cause lateral flexion of the alveolar processes and buccal displacement of the anchorage teeth with varying degrees of inclination as well as horizontal and vertical bone loss.[3] That said, more buccal inclination was observed in the RME group and increased buccal bone loss in the SME group.

Buccal displacement of teeth to expand the dental arch within the physiological confines of the alveolus has been common-place in orthodontic practice. However, the concept of tooth movement facilitating apposition of the dental alveolus is relatively new. After clinical observation of bone remodeling via slow growing cysts, changes in occlusion due to muscular pressure and alveolar regeneration by way of moving a tooth into edentulous sites, a new paradigm of dental-alveolar expansion was developed, whereby *ultra-low forces* are thought to be the key to facilitate expansion via alveolar apposition.[8(p997.e96)] The Damon® system is a low-friction/low-force system using *passive self-ligating* (PSL) brackets and continuous arch mechanics.[8(p997.e96),9,145] Badawi et al.[22] reported evidence with an *orthodontic force simulation apparatus* (OSIM) that seems to support the ability of PSL brackets to deliver lower-magnitude forces compared with elastomeric-ligated appliances applied to the same malocclusion (i.e. high buccal ectopic canine) in an in vitro model.[18] As such, clinical evidence has been reported suggesting significant widening of the dental arches following treatment with this system.[146]

The existing literature has shown that many studies utilizing Damon® brackets have provided inconclusive evidence with respect to the effect that the appliance might have on the alveolus and its contribution to mucogingival defects.[27,84,89] Systematic reviews examining periodontal defects associated with orthodontic movements are largely composed of analyses of retrospective studies with high potential for selection bias and variable diagnostic reliability, and animal models with low generalizability.[23,27,127,144] In human studies, the existing literature measures periodontal defects using diagnostics using clinical data such as intra-oral photography and/or clinical cast models. [2,127] More recently, however, cone-beam computerized tomography (CBCT) has been used for the evaluation of maxillofacial hard-tissue, enabling practitioners to visualize the cortical bone in three-dimensions.[2] Evaluation with CBCT is of particular interest due to its ability to generate a three-dimensional image with tissue contrast, elimination of blurring and overlapping, and projection effects,[37] as well as acceptable inter-evaluator accuracy.[147] Issues with CBCT include inherent limited spatial resolution,[3,23] lack of validity testing (i.e. compared to a periodontal examination using a periodontal probe), as well as variations in diagnostic quality (based on exposure and field of view setting).[37] Among those review articles published, there is a generalized lack of matching for risk factors (i.e. periodontal biotype) for cases and controls and there is a need for stratification by orthodontic mechanics.

This study aimed to perform a systematic review to determine the buccal alveolar effects of orthodontic maxillary expansion using the Hyrax appliance (standard of care) compared to a PSL system (new proposed approach) in adolescents with permanent dentition using CBCT analysis.

1.5.2 Methods

1.5.2.1 Protocol and Registration

The study protocol was not registered in advance.

1.5.2.2 Eligibility Criteria

The following selection criteria were applied for the review:

- Study design: clinical trials, cohort, case-control, and cross-sectional studies
  - Human-based studies for growing children in permanent dentition without any developmental abnormalities or syndromes. Studies with a larger age range were considered, only if data was stratified by age or tooth-development groups
  - Examination of changes to the alveolar bone (including the presence of fenestrations, dehiscences and/or recessions) before and after maxillary expansion, with follow-up by CBCT measurement with or without clinical tooth / pocket measurements

    - Use of RME with a four banded Hyrax appliance (maxillary first premolars and first molars). Studies with other expansion methods (i.e. bone-anchored, Haas-type expander) were considered, only if the results were stratified by expansion mechanics
    - Passive Self-Ligating or Damon® bracket treatment. Studies with other bracket systems methods (i.e. twin, self-ligating) were considered, only if the results were stratified by expansion mechanics

- There was no restriction on language (pending an English translation was available) or place of publication

- Exclusion criteria: opinion papers and research proposals were excluded, as well as studies with imaging that did not disclose voxel size

1.5.2.3 Information Sources and Search Strategy

A literature search was performed independently by two reviewers (MK, DC) inclusive to February 2021. With the help of a specialized health sciences librarian at the John W. Scott Health Sciences Library, University of Alberta, Edmonton, Alberta, Canada, search terms were established using the OVID (includes MEDLINE, EMBASE) database and then adjusted, as required, for the following electronic databases (Table 1.4):

- SCOPUS (from 1970 to week 2 of Feb 2021)
- CINHAL (from1937 to week 2 of Feb 2021)

To complete the search, a review of grey literature was performed (i.e. Google Scholar, product websites, Health Technology Assessments, Medical Device Listings). References of each selected publication were hand searched.

Table 1.4 Database search strategy.

MEDLINE via OVID (1946 to week 2 of Feb 2021)	{[(exp Tooth Movement Techniques/ or exp Orthodontic Appliances/ or exp Orthodontics/ or orthodont*.mp. or exp Orthodontic Brackets/) AND (exp Palatal Expansion Technique/ or expansion.mp.)] OR [(Hyrax.mp.) or (damon.mp.)]} AND {[(exp Gingival Recession/)] OR (exp Gingiva/ or exp Periodontium/ or periodont*.mp. or exp Alveolar Bone Loss/)]}
SCOPUS (from 1970 to week 2 of Feb 2021)	(((TITLE-ABS-KEY ((tooth AND movement AND techniques) OR (orthodontic AND appliances) OR (orthodontics) OR (orthodont*) OR (orthodontic AND brackets))) AND (TITLE-ABS-KEY ((palatal AND expansion AND technique) OR (expansion)))) OR (TITLE-ABS-KEY (Hyrax OR damon))) AND ((TITLE-ABS-KEY (gingival AND recession)) OR (TITLE-ABS-KEY ((gingiva) OR (periodontium) OR (periodont*) OR (alveolar AND bone AND loss))))
CINHAL (from1937 to week 2 of Feb 2021)	((((tooth AND movement AND techniques) OR (orthodontic AND appliances) OR (orthodontics) OR (orthodont*) OR (orthodontic AND brackets)) AND ((palatal AND expansion AND technique) OR (expansion))) OR ((Hyrax OR damon) AND ((gingival AND recession) OR (gingiva) OR (periodontium) OR (periodont*) OR (alveolar AND bone AND loss))))

1.5.2.4 Study Selection

Using RefWorks (Legacy ed.), the search results were exported and duplicates removed. Selection of relevant articles was carried out in two stages. In the first stage, each reviewer (MK and DC) independently scanned titles and the abstracts to select potentially relevant papers according to the inclusion criteria. In phase two, the full texts of the included articles were retrieved and reviewed by the same reviewers for relevant content. Any disagreement was resolved during a discussion and consensus meeting. It is important to note that it was not possible to review all of the potentially relevant articles due to access restrictions at the University of Alberta Libraries. The flow diagram (Figure 1.18) illustrates the results of the search, summarizing the process of identifying, including, and excluding studies.

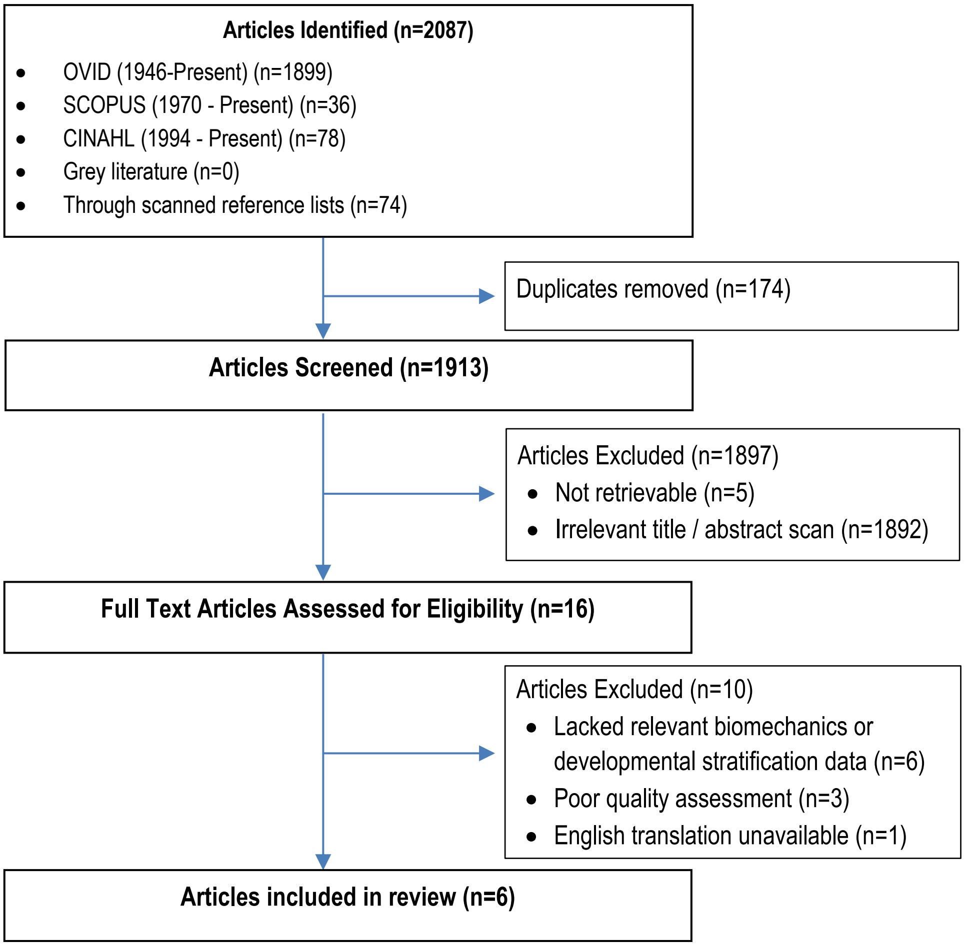


Figure 1.18 Literature selection (as of February, 2021).

1.5.2.5 Data Collection Process and Data Items

Using a developed standardized data collection form based on the Cochrane Consumers and Communication Review,[148] one reviewer (MK) gathered relevant data (details of the participants, intervention, evaluation, and authors' conclusion) were extracted from each of the selected studies. The second reviewer (DC) cross-referenced the data and confirmed its accuracy. Any disagreement was resolved during a discussion and consensus meeting.

1.5.2.6 Risk of Bias in Individual Studies

Studies were selected for inclusion using *the Joanna Briggs Institute* (JBI) Critical Appraisal Tool.[149] For each bias domain, a judgment score was given following their recommendations.

The judgment involved recording:

- ‘‘yes’’ for if the study adequately answered the question, rendering it low risk of bias
- ‘‘no’’ for if the study inadequately answered the question, rendering it high risk of bias
- ‘‘unclear’’ in the case of no, insufficient, or uncertain information with respect to the question
- “not applicable” for if the study, by nature of its design, would inherently have that bias

If there was lack of information in a key area of the study, it was judged as ‘‘unclear,’’ and the reviewers tried to contact field experts and/or the paper’s authors to obtain more information and ascertain a definitive ‘‘yes’’ or ‘’no’ judgement. As indicated in the JBI critical appraisal tool, a response of ‘no’ to any of the questions negatively impacts the quality of the study.

1.5.2.7 Summary Measures

Measurements of continuous data were in *millimetres* (mm), and categorical data and scores were collected for some selected clinical indices.

1.5.2.8 Synthesis of Results

Included studies assessed periodontal and outcomes related to each expansion treatment. A meta-analysis was planned pending relative homogeneity of the data and the methods for obtaining it, for each selected article.

1.5.2.9 Evaluation of the Level of Evidence

The level of evidence was calculated using the Grading of Recommendations, Assessment, Development and Evaluation Pro software (GRADEpro Guideline Development Tool).[147] It grades the quality of evidence in four levels: very low, low, moderate, and high. ‘’High quality” suggests that the true effect lies close to the estimate of the effect. ‘‘Very low quality’’ suggests that there is very little confidence in the effect estimate and the estimate reported can be substantially different from what was measured.

Table 1.5 JBI Critical appraisal checklist for case series.

Reliability Qualifier	Atik & Ciger, 2014	**Baysal et al., 2018**	**Cattaneo et al., 2011**	Domann et al., 2011	**Garib et al, 2006**	**Lin, et al., 2015**	**Morais et al., 2018**	**Pangrazio-Kulbersh et al., 2013**	Vi & Lagrave re, 2017
Were there criteria for inclusion in the case series?	Yes	Yes	Unclear	Unclear	Yes	Yes	Yes	Unclear	Unclear
Was the condition measured in a standard, reliable way for all participants included in the case series?	No	Yes	Unclear	Yes	Yes	Yes	Unclear	Yes	No
Were valid methods used for identification of the condition for all participants included in the case series?	No	Yes	Yes	No	Yes	Yes	Unclear	No	Unclear
Did the case series have consecutive inclusion of participants?	No	No	Yes	No	No	No	No	No	Yes
Did the case series have complete inclusion of participants? [Not Applicable]	Unclear	Unclear	No	Unclear	Yes	Un-clear	No	Unclear	No
Was there clear reporting of the demographics of the participants in the study?	Yes	Yes	Unclear	Yes	Yes	Yes	Yes	Yes	No
Was there clear reporting of clinical information of the participants?	Yes	Yes	Unclear	No	Yes	Un-clear	Yes	Yes	No
Were the outcomes or follow-up results of cases clearly reported?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Was there clear reporting of the presenting site(s)/clinic(s) demographic information?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Was statistical analysis appropriate?	No	Yes	Yes	No	Un-clear	Un-clear	Yes	Yes	No
Included in the study	No	Yes	Yes	No	Yes	Yes	Yes	Yes	No

Outcome Measures: Yes, No, Unclear, Not Applicable
A response of 'no' to any of the questions negatively impacts the quality of the case series; therefore, those with more than two no response was excluded from this review

1.5.2.10 Risk of Bias within Studies

Of the sixteen articles selected for full-text review, nine were subject to critical appraisal using the JBI Critical Appraisal Tool.[149] Since stratified data from each study was to be extracted; the methodological quality was evaluated as *case series* research, irrespective of the overall study design. For each bias domain, a judgment score was given following JBI's recommendations. The authors of this review have accepted the definition of case series as a study in which only patients with the treatment are sampled (i.e. those who have a diagnosis that warrants the treatment) and does not permit calculation of an absolute risk. That said, JBI's ten bias domains, nine were considered key, excepting *consecutive inclusion of participants* because this is an inherent bias of case series design. Since all of the selected studies suffered from small sample sizes, rendering their generalizability and statistical outcomes questionable (even with power analyses), studies with more than two "no" responses outside of the aforementioned limitations were excluded from this review, rendering six studies suitable for inclusion (Table 1.5).

1.5.3 Results

1.5.3.1 Study Selection

A total of 2033 manuscripts were selected for preliminary assessment (Figure 1.18). After removing duplicates, 1860 studies were screened and 1845 excluded following a title/abstract/title assessment. Fifteen articles were selected to receive a full-text reading (phase two). Of the full-text articles retrieved and reviewed, nine studies were later excluded due to lack of relevant measurement,[150] biomechanics[3,127,151], stratification data,[150] poor quality assessment[24,152,153] or unavailability of English translation.[154] Therefore only six studies fulfilled the criteria to be included in this review. Since studies directly comparing periodontal outcomes between Hyrax and Damon® expansion were not accessible, results from direct comparisons were not possible. Nevertheless, relevant data for each method were still extracted individually from the selected studies.

1.5.3.2 Synthesis of Results

As a result of the nature of the question and the available data (i.e. heterogeneity of the evaluation methods employed, different measurements employed - continuous and categorical data) a meta-analysis was not possible and would not have allowed meaningful comparisons. Only simple and descriptive comparisons are reported.

1.5.3.3 Study Characteristics

The characteristics of the studies selected are listed in Table 1.6. Among them, there were five case series (two retrospective[155,156] and three prospective[25,26,133]) and one RCT.[90] Sample size ranged from four[133] to twenty-two[90] with a mean age of 12.6years[133] to 15.5years[90] at the initiation of the study. The sample consisted of adolescents in permanent dentition who were diagnosed with maxillary deficiency[25,26,90,155,156] for treatment with a Hyrax appliance or class I/II malocclusion with moderate crowding,[44] in conformity with the guidelines for Damon 3MX® brackets system.[90] Two of the six studies chosen[26,90] had unclear inclusion criteria and one[26] was not clear with their definition of the condition being treated. Only Cattaneo et al.[90] clearly ascertained the consecutive inclusion of participants and, along with Morais et al.,[25] reported on loss to follow up. All of the chosen articles clearly reported the location of the sample (Turkey,[155] Denmark,[90] Brazil,[25,133] Korea[156] and USA[26]); however, with the exception of one article,[90] specific participant demographics were only minimally reported (i.e. age and gender). While relevant clinical information was clearly outlined, authors of two studies required the reviewer to further investigate their measurement protocol[156] and treatment application[90] beyond what was included in their study.

All chosen studies used 3-D imaging (CBCT,[25,26,90,155,156] spiral CT[133]) to evaluate pre-defined landmarks at (minimum) two time points - before and after treatment. Of the six selected studies, only Baysal, et al.[155] included an evaluation of some follow-up patients. A large field of view was used in all of the studies, and voxel size ranged from 0.2mm[156] to 0.36mm.[90] One study measured the condition using two different exposure time parameters and voxel sizes (.25mm and 0.30mm),[25] whereas one seemed to arbitrarily set an isoline to discern bone apposition.[90]While three of the six studies disclosed patient scan orientation to minimize angulation error,[133,155,156] none of the authors used the same overall coordinate system. A range of software (i.e. Dolphin Imaging software,[26,155] Mimics Software,[25] ITK-SNAP open-source software,[90] InVivo Dental by Anatomage[156] and Alatoview software[133]) were used for data analysis and interpretation of the digital files. Finally, only one study included an additional model analysis which was used to evaluate bone apposition with use of isolines.[90]

In all cases, data was obtained using digitally rendered 2D cross-sections in order to facilitate landmark placement over maxillary first premolars and/or first molars. While all studies confirmed landmark placement on the buccal alveolus, authors employed different reference points and cross-section methods to obtain alveolar bone height and width measurements. Four of the studies employed their own method,[25,90,133,156] of which only one[25] piloted. Two of the studies used methods outlined by other authors: one[26] used a method outlined by Rungcharassaeng, et al.,[13] and one[155] by Evangelista, et al.[92] More variation was observed in measurement of alveolar width with studies evaluating alveolar bone thickness adjacent and apical to the right maxillary permanent first molar furcation,[133,155] CEJ,[156] and an area apical to the CEJ, in the cervical 1/3 of the root of the tooth in question.[25,26,90] Vertical bone height was measured using either the incisal tip[26,133,155] or the CEJ[25,90,156] as a reference point.

Table 1.6 Study characteristics

Study	Participants (Hyrax, Damon®)				Intervention				
Location	*N, age, sex*	*Group Allocation*	*Condition*	*Orthodontic Device*	*Activation Protocol*	*Time Points*	*Data Collection method*	*Interpretation*	*Analysis Technique*
Baysal et al., 2018 - Case Series (Retrospective)									
Oral and Maxillofacial Radiology Department, Dicle University (Diyarbakır, Turkey).	N= 20 (9♂, mean age: 13.97 ± 1.17 years) 11♀, mean age: 13.53 ± 2.12 years)	Patient Criteria: - no history of previous orthodontic treatment or a systemic disease; - all maxillary teeth present and fully erupted, (not third molars)	Bilateral cross-bite related to maxillary transverse deficiency	Hyrax-type expander: banded first molar with palatal extension to first premolar	2 turns/ day (morning & evening) until the palatal cusps of the upper posterior teeth are in contact with the buccal cusps of the lower posterior teeth. 3mo after last activation: transition to TPA	T1: before appliance placement T2:directly after the end of the activation. T3: N=10; 6-month retention records	i-CAT® (Model 17-19) 120 kVp 5.0 mAs 9.6- sec axial slice thickness: 0.3mm *Measurement error disclosed*	Dicom analyzed in Dolphin Imaging 11.0 Orientation: x - Frankfort horizontal line Y-transporionic line Z- midsagittal line	Method by *Evangelista et al* Measured bone thickness at three different levels Cross-sections parallel to Frankfort horizontal at M1 trifurcation, middle-DB root, apex DB root
Cattaneo, et al., 2011 - Randomized Control Trial									
School of Dentistry, Aarhus University, Denmark	N=21 (mean age: 16.1 ± 5.7 years)	Follows Damon 3MX® brackets system guidelines (Ormco Corporation, Orange, CA, USA) and In-OvationR (GAC International Inc., Bohemia, NY, USA). Randomization sequence - 1:1 allocation using random block size of 4.	Exclusion: Patients with severe Class III, obvious need for extraction, with periodontal problems, and major skeletal discrepancies	0.022-inch Damon 3MX® appliance system (Ormco/A Company, San Diego, Calif).	Tx delivered: December 2004 - November 2009. Damon 3 MX® passive SLBs and Damon® arch wires, treatment protocol according to the Damon Workbook	T0: pre treatment T1: after treatment completion	Digital study models (O3DM; Ortolab, Czestochowa, Poland) CBCT (NewTom 3G; QR, Verona, Italy) 0.36-mm isotropic voxel dimension. *Measurement error disclosed*	DICOM analyzed in ITK-SNAP open-source software (http://www.itksnap.org) isoline: 0.7 mm *No CBCT orientation listed*	*Used own method* Measured bone area Cross section: centre of root canal and CEJ of premolar (buccal root of 2 rooted teeth) and the alveolar point closest to the root, and 9mm apical
Garib, et al., 2006 - Case Series (prospective)									
Dept of Orthodontics, Bauru Dental School, University of São Paulo. Brazil	n= 4 ♀ mean age:12.6 years (11.5-13.9 years).	From N=87 subjects excluding: Persistence of any deciduous teeth Absence of maxillary posterior permanent teeth Metallic restorations on	angle Class I/II malocclusion uni-/bi-lateral posterior crossbite	Hyrax-type expander; banded first bicuspid and first molar	Two turns / day - 7mm expansion After the active expansion phase, the screw was fixed with acrylic resin, and the appliance was kept as a retainer for 3	T1: before expansion T2: after the 3-month retention period when the expander was	Spiral CT machine (model Xvision EX, Toshiba Corporation Medical Systems Company, Otawara-Shi, Japan)	network computer workstation (Silicon Graphics, Toshiba Corporation Medical Systems Company) with Alatoview software (Toshiba Corporation Medical Systems	*Used own method* Measurements taken at axial section parallel to the palatal plane, at the level of the right maxillary permanent first molar furcation Tooth rotations

		maxillary posterior teeth Previous periodontal disease Previous orthodontic treatment Male gender.			months.	removed.	120 kV 100 mA scanning time: 1s/ sect w/ FC30 scanning filter FOV:12.6x12.6 cm, matrix: 512 x 512 pixels Window width: 2400HU with a center of 1300 Hounsfield unit (HU). *Measurement error disclosed*	Company) generated 2-D reformatted images Orientation: X- glabella, filtrum Y- Camper's plane Z- lateral eye canthus	present: bone plate was measured where root was closer to external contour of alveolar ridge.
Lin, et al., 2015 - Case Series (Retrospective)								
Kyung Hee University Dental Hospital, Seoul, South Korea	N= 13 ♀ Age= 17.4 ± 3.4 years	Initial CBCT: suture area <2 mm^2), > 7 mm of activation, and no surgical or other treatment that might influence the RME outcome during the expansion period.	Transverse maxillary deficiency with unilateral or bilateral posterior crossbite	Hyrax-type expander: Bands on first molars and first premolars	7-mm screw (Dentaurum, Ispringen, Germany); activated with a complete turn after placement, then 1/4 turns in the morning & evening up to locking, on the 16th day. - expansion screw activated 7 mm in all patients. - Retention: screw fixed with acrylic resin (3 mos)	T1: before treatment T2: 3mo after activation	Alphad vega, Asahi Roentgen, Kyoto, Japan: analyzed: 80 kVp 10 mA, 30- second scan time 0.2-mm voxel size *Measurement error disclosed*	InVivo Dental (Anatomage, San Jose, Calif). Orientation: x - Palatal Suture y - parallel to palatal plane z - tangent to nasal floor	*Used own method* Alveolar bone dehiscence measured from CEJ to the alveolar crest on buccal side.
Morais, et al., 2018 - Case Series (prospective)								
Dept of Orthodontics, Bauru Dental School, University of São Paulo. Brazil	N=22 (mean age: 14.7 ± 1.2 years) 13♂, 9♀ 20 class I 2 class II	N/A - only one group	(1) Class I or Class II molar relationship (2) >4mm maxillary crowding (3) full permanent dentition	Damon 3MX® (Ormco, Glendora, Ca) standard torque brackets No additional interventions, such as	Archwire sequence: (1) 0.014-inch Damon® copper-nickel-titanium (CuNiTi) - 10 weeks or until the teeth were passively engaged in all bracket slots	T0: pre treatment T1: < 4 weeks after insertion of the 0.019x 0.025-inch SS archwire	i-CAT scanner, (Imaging Sciences International, Hatfield, USA) 120 kV 5 mA field of view (FOV) of 13 cm	DICOM analyzed in Mimics software (version 14.01, Materialise, Leuven, Belgium) cross-section images were imported into the software, ImageJ (NIH, Bethesda, Md) *No CBCT orientation*	*Used own method: pilot study completed* Cross sections: pulp apex - crossing the center of the root, perpendicular to the alveolar contour at the level of the root cervical third.

			anterior to the first molars (4) age 11–17 years; (5) healthy periodontium (6) no previous orthodontic treatment.	Inter-proximal reduction, inter-maxillary elastics, or any orthopedic mechanics, were used	(2) 0.014 x 0.025-inch Damon® CuNiTi archwires kept until tooth alignment and passive fit in slot (3) 0.019 x 0.025-inch stainless-steel (SS) archwire contoured for maintenance of the arch form developed in the first two phases. Oral hygiene: monitored during this period. Appointment intervals were approximately q5weeks.		height x 16 cm diameter 20-second exposure time — isotropic voxel size of 0.3 mm (9 patients) or 40-second exposure time — isotropic voxel size 0.25 mm (13 patients) *Measurement error disclosed*	*listed*	This can be done regardless of the angulation/rotation of the tooth relative to the alveolar process or the presence of crowding.
Pangrazio-Kulbersh et al., 2013 - Case Series (Prospective)									
Detroit Mercy Dental School, USA	N=12 (6 ♂, 6♀) mean age = 12.5 ± 1.6 years	Dolichocephalic growth pattern: bonded expanders Mesocephalic / brachycephalic growth pattern: banded expanders	Maxillary palatal constriction	Banded Hyrax expander: Bands on molars and first premolars	The expanders were activated one turn a day for 4–6 weeks, with total expansion ranging from 6 to 10 mm	T1: before expansion T2: 6 months after the last activation of the expander, which coincided with appliance removal	I-CAT imaging scanner (Imaging Sciences International, Hatfield, Penn). 120 kVp 18.54 mAs 8.9-second scan time 0.3-mm voxel size. Each *Measurement error disclosed*	DICOM (digital imaging and communications in medicine) data files were assessed using Dolphin3D (version 11.5; Dolphin Imaging and Management Solutions, Chatsworth, Calif). *No CBCT orientation listed*	Method by *Rungcharassaeng et al.* BT & MBL measures: Axial view: open polygon cut made buccal-lingual to bisect the roots bilaterally @ PM1 & M1 root levels Coronal image from open-polygon cut, reference lines were constructed from the buccal cusp tips to the buccal root tips, bilaterally. Perpendicular line (PL1) from reference line at most coronal

								point where bone meets tooth Perpendicular line (PL2) made at level of buccal bone deflection
Summary - RCT: 1; Case Series: 5								
Turkey:1 Denmark: 1 Brazil: 2 Korea: 1 USA: 1	N=92 Damon®: n=42 Hyrax: n=50	Hyrax: Maxillary Deficiency Damon®: Class I/II Crowded	Hyrax: - Banded Molars & Premolars - 3 - Bonded Molars, premolar extensions arms - 1 Damon®3MX brackets - 2	Hyrax: 2 turns/day - 1 1 turn/day - 3 Hyrax - Total Expansion: 7mm expansion - 2 6mm-10mm expansion - 1 Palatal cusps of mx M1 in contact with buccal cusps of Md M1 - 1 Damon: Damon®brackets and AWs, as per their treatment guidelines	T1: before treatment T2: at the end of active treatment - 3 T2: 3mo after the end of active treatment - 2 T2: 6mo after the end of active treatment - 2	Voxel size: 0.2mm - 1 0.25mm - 1 0.3mm - 3 0.36mm - 1 Unknown - 1	Software: - Dolphin - 2 Mimics - 1 Anatomage - 1 ITK-SNAP - 1 Alatoview software - 1	Method: Evangelista et al. - 1 Rungcharassaeng et al. - 1 Own method - 4

1.5.3.4 Results of Individual Studies

A summary of the results can be found in Table 1.7. Overall, the authors found a reduction in buccal alveolar bone height and width in both the Hyrax and Damon® groups. With respect to Hyrax RME, all studies found an increase in dehiscence formation and a decrease in buccal bone width, especially at banded abutment teeth.[26,133,155,156] More specifically, Baysal et al.[155] found a decrease in mean buccal bone width ranging from .24mm (± .43mm) over the maxillary canines to 1.11mm (±1.42mm) over the apical region of the first premolars. The mean distance from the crest to the CEJ ranged from .40mm (±.83mm) over the distobuccal root of the maxillary first molars to 1.32mm (±1.64mm) over the maxillary first premolars. Garib et al.[133] found a reduction in mean buccal alveolar bone from the maxillary first premolar (.7mm±.1mm) to the distal of the maxillary first molar (.8mm±.3mm). The banded teeth had a greater presence of dehiscence with the largest mean reduction in bone height being at the maxillary first premolars (7.1mm±4.6mm) followed by maxillary first molars (3.8mm±4.4mm).[133] Lin et al.[156] found that the maxillary first premolars showed 5.05mm more mean bone loss in the Hyrax group with the maxillary first premolars showing more bone loss compared to other teeth ($p<.001$). Pangrazio-Kulbersh et al.[26] found a mean reduction in bone thickness over the maxillary right and left first molars of .59mm and .50mm, respectively, and .72mm and .57mm over the right and left first premolars, respectively. Similar to the other studies,[133] dehiscence formation was more common over banded first molars (.63mm) and first premolars (.37mm).[26]

Studies examining the periodontal effects associated with Damon® expansion found a decrease in buccal alveolar thickness and height[25] as well as volume.[90] For example, Cattaneo et al.[90] found a decrease in mean buccal alveolar bone area for maxillary first premolars from 18 – 23% with Damon® brackets without alveolar bone deposition in most cases. Similarly, Morais et al.[25] found that following treatment with Damon® brackets, bone thickness on the mesiobuccal root of the maxillary first molar showed an overall significant reduction in buccal bone width 3mm and 6mm from the CEJ (36% and 45%, respectively). They also found significant apical migration of marginal bone in relation to the maxillary first molars (0.3 mm), with high variability.[25]

Table 1.7 Results of Individual Studies

Location	*Study Results*	*Authors' Conclusions*
Baysal et al., 2018 - Case Series (Retrospective)		
Oral and Maxillofacial Radiology Department, Dicle University (Diyarbakır, Turkey).	BCBT decreased for all investigated teeth at most coronal aspect. Averaged statistically significant results at furcation: ~ 50% increase BAH increased for all teeth. Averaged statistically significant results at furcation: ~ 90% increase	- RME may have detrimental effects on the supporting alveolar bone, since the thickness and height of the buccal alveolar bone were decreased - the increased dehiscence formation may support these findings.
Cattaneo, et al., 2011 - Randomized Control Trial		
School of Dentistry, Aarhus University, Denmark	decrease in buccal alveolar bone from 18 – 23% with Damon® brackets with no alveolar bone deposition in most cases.	The anticipated translation and buccal bone modeling could not be confirmed in the majority of the cases. Individual pre-treatment factors: initial teeth inclination and occlusion = important in determining the final outcome of the individual treatment. CBCT-technology combined with digital casts is all important to analyze 3D treatment outcomes both at dental and bone level in large study groups.
Garib, et al., 2006 - Case Series (prospective)		
Department of Orthodontics, Bauru Dental School, University of São Paulo. Brazil	- Reduction in mean BBPT* for PM1 (0.7±0.1mm) to the M1-D (0.8±0.3mm). - Banded teeth had a greater presence of dehiscence with BACL** being at the PM1 (7.1±4.6mm) followed by M1 (3.8±4.4mm). *Buccal bone ** buccal alveolar crown length	- RME orthodontic effect reduced the BBPT of maxillary posterior teeth and increased the LBPT. - RME induced bone dehiscences on the anchorage teeth's buccal aspect, especially in subjects with thinner buccal bone plates. The tooth-borne expander produced more reduction of first premolar BACL than did the tooth-tissue borne expander. 0.6–0.9 mm reduction in buccal bone plate thickness of the banded teeth
Lin, et al., 2015 - Case Series (Retrospective)		
Kyung Hee University Dental Hospital, Seoul, South Korea	Max PM1 - 5.05mm more bone loss for Hyrax (p<.01) Max PM1 had more bone loss than other teeth (p<.001)	Late-adolescent patients, tooth-borne expanders produced less transverse skeletal expansion More alveolar bending, more dental tipping, and more vertical alveolar bone loss at the Max PM1.
Morais, et al., 2018 - Case Series (prospective)		
Department of Orthodontics, Bauru Dental School, University of São Paulo. Brazil	- BT 3mm and 6mm from CEJ showed a significant reduction (36% and 45%, respectively), - BA decreased 40% - significant apical migration of BH @ M1 (0.3 mm), with high variability	Significant bone loss (in terms of both thickness and height) was observed at the maxillary central incisors and the mesiobuccal root of the first molars. Initial bone thickness, crowding severity, and the amount of expansion during treatment had a weak, though significant, impact on the buccal bone reduction.
Pangrazio-Kulbersh et al., 2013 - Case Series (Prospective)		
Detroit Mercy Dental School, USA	Banded M1 BBT: −0.50/−0.53 P1 BBT: −0.73/−0.46 M1 BMBL: 0.63/0.30 P1 BMBL: 0.16/0.37 Bonded M1 BBT: −0.55/−0.36 P1 BBT: −0.49/−0.55 M1 BMBL:0.20/−0.66 P1 BMBL: −0.67/−0.44	Horizontal buccal bone (BT) was reduced on MRt, MLft, PMRt, and PMLFt following RME. Banded RME had a tendency for vertical buccal bone loss (MBL) on the MRt and PMLft The CBCT is a reliable method for assessing changes in the buccal bone following RME
Summary - RCT: 1; Case Series: 5		
Turkey: 1 Denmark: 1 Brazil: 2 Korea: 1 USA: 1		Hyrax and Damon® brackets are associated with a decrease in buccal alveolar bone height and thickness The anticipated translation and buccal bone modeling could not be confirmed in the majority of the cases.

1.5.3.5 Evaluation of the Level of Evidence

The level of evidence was assessed using the Grading of Recommendations, Assessment, Development and Evaluation (GRADE) tool.[157] The GRADE quality of evidence was judged as very low due to the impossibility of performing a meta-analysis and subsequent use of narrative synthesis resulting in ‘‘serious’’ limitations in imprecision. Additionally, articles with unique periodontal outcomes were judged to be unclear according to JBI analyses (Table 1.8).

Table 1.8 GRADE risk of bias across studies

Certainty assessment									
No. of Studies	Study Design	Risk of Bias	Inconsistency	Indirectness	Imprecision	Other Considerations	Impact	Certainty	Importance
Maxillary Buccal Alveolar Bone Thickness (assessed with: (Cone Beam) Computed Tomography)									
6	Observational studies[a]	Not serious	Not serious	Not serious	Serious[b]	None	Both Hyrax and Damon® expansion was associated with a reduction of maxillary buccal alveolar bone thickness	⊕○○○ VERY LOW	Important
Maxillary Buccal Alveolar Bone Height (assessed with: (Cone Beam) Computed Tomography)									
6	Observational studies[a]	Not serious	Not serious	Not serious	Serious[b]	None	Both Hyrax and Damon® expansion were associated with a reduction of maxillary buccal alveolar bone height	⊕○○○ VERY LOW	Critical
Maxillary Buccal Alveolar Bone Apposition (assessed with: digital scan isolines of 0.7mm)									
1	Observational studies[a]	Serious[c]	Not Serious	Not Serious	Serious[b]	None	The anticipated translation and buccal bone modeling in the Damon® system could not be confirmed in the majority of the cases.	⊕○○○ VERY LOW	Important

[a] Data is not pooled; [b] Narrative synthesis was conducted and the estimates are not precise.
[c] The information on the bias in the measurement of outcomes was not clear according to JBI

1.5.4 Discussion

1.5.4.1 Summary of Evidence

This review examined the effects of orthodontic expansion using the Damon® system and Hyrax RME on the buccal alveolus. While significant heterogeneity among the studies existed, all of the articles selected for review found a reduction in maxillary buccal alveolar width and height immediately after expansion treatment with both Hyrax[26,133,155,156] and Damon®[25,90] appliances. This is in line with the findings of another systematic review examining the periodontal effects of RME whereby significant loss of buccal bone thickness and marginal bone level were observed in anchored teeth in all of the selected studies.[158] Additionally, similar outcomes were found in clinical studies evaluating resultant buccal bone thickness using Hyrax appliances for orthodontic expansion in subjects with permanent dentition.[24,150,153,159] For example, in the present review, the maxillary first premolars decreased in bone width ranging from 0.57mm[26] to 1.11 (±1.42)mm[155] and height ranging from .37mm[26] to 7.1(±4.6)mm.[133] This is similar to other studies reporting decrease in bone width over maxillary first premolars of 0.19mm[153] to 1.23mm[13] and mean apical migration of crestal bone more than 4.75mm.[13] The present review showed the maxillary first molars decreased in bone width ranging from 0.50mm[26] to 0.8mm[133] and height ranging from 0.63mm[26] to 3.8mm.[133] Again, this is similar to Rungcharrassaeng et al.[13] who suggested a reduction in mean buccal bone width of up to 1.27mm[13] and height of up to 3.27mm in their sample.

While this review was limited to studies examining children in permanent dentition, it is interesting to note that the literature evaluating alveolar bone response to Hyrax RME in the mixed dentition suggests that the deleterious buccal alveolar effects of the appliance were reduced when anchored to deciduous molars.[133,160] This is likely a result of the growth potential of children in mixed vs. permanent dentition, where more favorable and predictable outcomes usually found in younger, growing patients.[52] As the child progresses beyond puberty, the circum-maxillary sutures become more interdigitated and continue to increase in complexity throughout growth and development. This makes skeletal expansion increasingly difficult and increases the likelihood of dental expansion, tipping, and mucogingival defects as the patient matures.[8(p156)]

The body of literature examining Damon® appliances and passive self-ligation is limited. Findings in this review identified dental tipping beyond the cortical plate as a major contributor to resultant expansion with Damon® appliances rather than translation and bone apposition (as suggested by company's marketing).[18] This is similar to other studies who found that he majority of the expansion seen in Damon® appliances was due to dental tipping.[9,24] Previous studies examining the effect of dental tipping on the buccal alveolus in animal models similarly found alveolar bony dehiscence and apical migration of the tissue attachment.[16,17] Despite following the Damon® expansion guidelines, Cattaneo et al.[90] reported bone loss at the maxillary first premolars from 18 – 23% an inability to observe buccal remodeling and apposition after expansion in the majority of the cases. Similarly, Morais et al.[25] found that following treatment with Damon® brackets, bone thickness on the mesiobuccal root of the maxillary first molar showed an overall significant reduction in buccal bone width 3mm and 6mm from the CEJ (36% and 45%, respectively). They also found significant apical migration of marginal bone in relation to the maxillary first molars (0.3 mm), with high variability.[25] Contrary to these findings, Kraus et al.[159] was able to histologically identify the formation of bone on periosteal surfaces of cortical bone with buccal expansion using the Damon® system in a canine model, indicating that apposition is possible on the leading edge of tooth movements. That said, the same study also showed uncontrolled tipping, and bone dehiscence on other teeth treated with the same protocol.[159]

1.5.4.2 Limitations

1.5.4.2.1 Study Level

Major limitations of the studies selected for this review include small sample sizes (i.e. n<30) and subsequent lack of generalizability, a vague diagnosis to justify the treatment type and extent of expansion, flaws in the study design related to obtaining and analyzing data as well as inadequate temporal data and inadequate reporting on etiology and severity of cases. The cases selected for each study were based on treatment-types, and limited by inclusion and exclusion criteria. As outlined in several of the selected articles, patients with thinner initial buccal bone plates had a larger reduction of alveolar bone level after expansion, regardless of the treatment type.[26,133] Of the selected studies, only three expanded based on the clinical presentation and etiology,[25,90,155] as would be done in clinical scenarios. Future studies should identify the etiology and severity of the diagnosis and prescribe expansion accordingly so as to avoid over expansion and allow for stratification of clinically meaningful outcomes.

Methodologically, the selected studies differed in determining changes in alveolar width and height. Most obviously, each study used a different 3D imaging unit with different analysis software and rendered full *field of view* (FOV) volumes with voxel-sizes ranging from 0.2mm - 0.36mm. Scatter noise associated with the large FOVs frequently used in orthodontics decreases spatial resolution.[42] Inherently these devices elicit false negative measurements of approximately 0.3-0.6mm (depending on the voxel size and exposure parameters)[37,48,147] in addition to the possible measurement error of the examiner. That said, since the buccal cortical plate often exists in a thickness of around 0.3mm, the imaging device itself poses a significant limitation to determining true alveolar changes. Future studies may consider reducing the FOV and voxel size to more closely ascertain the overlying width and height of alveolar bone.

When different time points are analyzed, the impact of cumulative landmark location errors should also be considered.[161] That said, all of the six articles reported measurement error, reporting either inter-examiner reliability testing or a calculation of error using *Dalhberg's Formula,* yet none reported the cumulative potential error in measurement.

Similarly, the angulation of the teeth and the angulation of the volume clipping poses another methodological limitation such that every landmark can have non-uniform error for all three axes.[161] The line perpendicular (x-axis; transverse) to the pre-defined y-axis (vertical) will determine the extent of alveolus available to measure.[37,48,147] In the case of expansion, if the tooth is buccally tipped (as is the case in most of the studies selected) and the investigator is measuring alveolar thickness or height, the line bisecting a vertical line through a cusp tip compared to the central fossa and roots may elicit different results. Similarly, increased buccal crown tipping can reduce the vertical distance between a cusp tip and the buccal alveolus when a line, tangent to the buccal alveolus and the incisal edge creates said vertical distance. Future studies should work to validate methods to measure hard tissues using 3D imaging and create a standard for patient positioning and DICOM analysis that accurately and consistently the alveolus.

Finally, all of the included studies evaluated patients at maximum 6 months post-treatment, with both Damon® studies[25,90] evaluating patients at debond only. It is well known in the literature that during orthodontic tooth movement, the alveolar bone in the direction of the applied force undergoes resorption, which decreases its density both *in vivo* and on a CBCT volume.[42] Since it may take approximately six to 24 months to heal, future studies should consider including long-term follow up of patients treated with expansion since the present studies do not account for any long-term changes that may occur as a result of bone remodeling or environmental risk factors to bone loss. Future studies should attempt to triangulate 3D data with intra-oral imaging, cast analysis and periodontal charting to improve the validity of their outcomes and allow for a more complete long term follow up.

1.5.4.2.2 Review Level

Due to the lack accessible studies in the literature that compare the periodontal outcomes of the Damon® system to Hyrax RME, this review selected studies that satisfied the inclusion criteria and stratified results based on treatment type. Since each study was evaluated as a case-series and a meta-analysis was not justifiable, the certainty of this study' findings are automatically *very low* (Table 1.5.4). To minimize biases, this review followed the PRISMA guidelines for systematic reviews,[162] critically appraised articles that met the inclusion criteria using a peer reviewed and validated appraisal system[149] and followed the recommendations in the Cochrane Handbook for Systematic Reviews of Interventions.[157] To minimize selector and retrieval biases the principle reviewer involved a second and third, blinded reviewer to resolve discrepancies with the review process. To minimize expectation bias, the second reviewer (DC) is a specialist in periodontics, with different exception biases than the primary reviewer. That said, no consideration to editorial policies was given during journal selection, which could potentially be a source of publication bias. Finally, to minimize language and location biases, no limitation on location or language was placed; pending a translator at the University of Alberta was available to translate a non-English article. However, the single article that examines the periodontal effects of expansion with the Damon® system compared to Hyrax RME is published in Chinese.[154] Though every attempt to minimize language bias was made, translation was not possible within the time constraints of this review's due date.

1.5.4.3 Clinical Relevance

The studies included in this review reported changes in alveolar bone width less than 0.5mm, which is clinically relevant. Therefore, the statistically significant changes in bone less than 0.5mm should be regarded carefully as these results may not be clinically meaningful due to the methodological error.

The clinical relevance of the response of the buccal alveolus to orthodontic expansion is questionable. The periodontal literature demonstrates the significant contribution of the periosteum to the circulation of the gingival unit, where its loss reduces perfusion to the soft tissues thereby reducing its healing and regenerative capacity.[88,109] Since the results show an overall reduction in alveolar width and height with use of both Hyrax and Damon® expansion appliances, one might assume that the resultant, alveolar dehiscences may pose an additional risk factor to developing mucogingival defects.[16,17,76,90,127] However, it is well known in the orthodontic and periodontal literature that gingival sites with underlying alveolar dehiscences may or may not display mucogingival defects depending on the tissue biotype and local inflammatory processes.[163] Additionally, it has been widely reported that mucogingival changes are usually not immediately present post-orthodontic treatment and these changes are more likely to arise from exposure to environmental factors such as toothbrush abrasion or periodontal pathogens over time.[84] Since none of the studies included the resultant soft tissue outcomes in relation to their hard tissue findings, the relevance of the reduction of buccal alveolar bone is questionable.

1.5.5 Conclusions

- Based on evaluation of the studies that met the inclusion criteria, there were no significant or clinical differences to permit a sound conclusion about what type of maxillary expansion, Hyrax or Damon®, is more appropriate regarding a more favorable periodontal response. The identified level of uncertainty was very low.
- Both expansion modalities demonstrated a reduction in buccal bone height though the reader is cautioned to interpret this based on a very low certainty level.
- Both expansion modalities demonstrated a reduction in buccal bone width though the reader is cautioned to interpret this based on a very low certainty level.

1.5.6 Acknowledgements

The authors acknowledge the assistance of health sciences librarians at the University of Alberta. The authors declare no conflicts of interest and no funding.

Chapter 2: Methods

2.1 Introduction

A summary of the clinical trial can be found in Appendix C. The framework for method development was as follows:

1. Sample selection and retrieval of the DICOM images and intra-oral photographs for analysis.
2. Measure buccal alveolus using method similar to the protocol described by DiGregorio et al. (2019)
3. Determine presence of gingival recession (GR) and associated risk factors using a photo analysis protocol similar to that described by Cairo et al (2009)
4. Hypothesis development
5. Validation of the method using statistical analysis

2.2 Retrospective Study

2.2.1 Objectives

1. Evaluate periodontal (buccal alveolar and soft tissue) changes associated with orthodontic expansion using both a tooth borne rapid maxillary expander (Hyrax) and Damon® appliances.
2. Compare alveolar changes between tooth borne RME vs Damon® system groups
3. Correlate soft tissue and alveolar risk factors with GR after treatment

2.2.2 Registration and Ethical Approval

This retrospective study was completed at the Orthodontic Clinic in the University of Alberta (Alberta, Canada) as part of the thesis fulfillment requirement for the MSc. Orthodontics program. Ethics approval was obtained from the University of Alberta Research Ethics Board (Pro00013379).

2.2.3 Participant Inclusion

Records were assessed for eligibility if both pre- and post-treatment CBCTs and intra-oral photos were present. Of the 95 subjects included in the study, 94 had pre-treatment CBCTs taken. There were 19 who did not have debond imaging taken and only 34 who returned for their two-year follow-up appointment by the time the data was collected.

Of the 76 patients for whom a debond CBCT was taken, 31 were removed from the study. Exclusion was based on:

- CBCT unit mismatch between time points (14). During the course of the treatment period, the imaging device was upgraded (2012). As such, those patients who had initial records taken earlier than 2012 were excluded from the study.
- Obstruction of landmarks on initial scan (15) due to unerupted teeth in question (10), patient movement (4), bonded appliances (1)
- Obstruction of landmarks on debond scan due to first premolar extraction (1).
- Missing intraoral photos (1).

A total of 45 patient records were included to compare baseline and debond (Twenty-one in the Hyrax group and 24 in the Damon® group). Eligible follow-up (retention) records were assessed from the pool of accepted baseline and debond records. Of the 21 follow-up records, two were excluded based on obstruction of landmarks (i.e. bonded appliances (1) and patient movement (1)). A total of 19 subjects (eight in the Damon® group and 11 patients in the Hyrax group) were included in the follow-up portion of this study. Figure 2.1 represents loss to follow-up and participant inclusion criteria. Demographic characteristics of study subjects are outlined in Table 2.1.

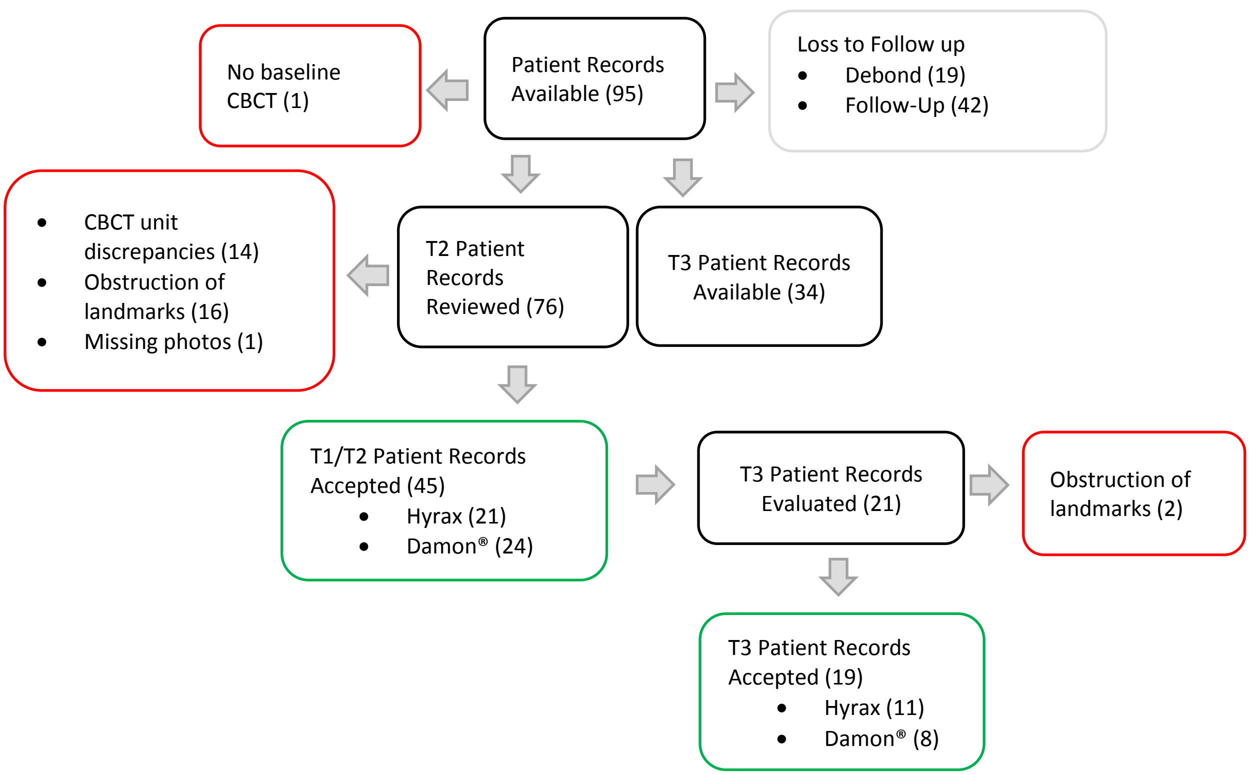


Figure 2.1 Patient records inclusion.

Table 2.1 Patient demographics

Patient Demographics			
Demographic	Group A (Damon®) *Mean (SD) or n (%)*	Group B (Hyrax) *Mean (SD) or n (%)*	*P value*
Age (y)	14.3 (1.9)	13.9 (1.6)	0.47
Sex			0.004
Male	5 (20.8%)	13 (61.9%)	
Female	19 (79.2%)	8 (38.1%)	
Total	24	21	

2.2.4 Randomization and Blinding

Records were evaluated at random. A third party, external to the study, was responsible for reorganizing the list of patient files to ensure temporal randomization of the data and each subject was assigned a unique code to conceal their demographic information. Since imaging was examined without orthodontic appliances, the investigator (MK) was blinded to patient assignment

2.2.5 Linear Measurements of Buccal Alveolar Width using CBCT

CBCT volumes were measured using a previously validated method described by Digregorio, et al. in 2019.[160] DICOM files were visualized and measured using Mimics software (version 19.0; Materialise, Leuven, Belgium), which was used with the ISO-surface and exposure of 300-1000 and 1mm slice thickness on the same laptop (Lenovo). Eight teeth were examined: the right and left maxillary permanent first molars, first premolars, canines and mandibular first premolars. The teeth were selected based on the appliance design and the reported prevalence of recession in the literature. That is, the Hyrax appliance includes the maxillary first molars and first premolars in the appliance design. The literature suggests that after the mandibular incisors, the mesiobuccal root of the maxillary first molars are most commonly associated with GR,[77] and the maxillary first premolars are often associated with thinner buccal alveolar bone.[83] Maxillary canines with buccal eruption, as is the case in most patients with maxillary transverse deficiency, are often also cited as sites with minimal hard tissue support.[32,92] The mandibular first premolars were chosen as they are easily visualized in intra-oral photographs and subject to less angular distortion in buccal views. Since the maxillary arch is expanded to fit the mandibular arch, the mandibular first premolar was chosen as a control tooth, with respect to both BBW and periodontal risk factors. Since the maxillary first premolars are included in the design of the Hyrax appliance, they're literature cites them as being frequently associated with GR associated with orthodontic treatment.[90] Axial, coronal and sagittal views were used to create an orientation plane passing through the long axis of each tooth (Figure 2.2). The plane was defined with three landmarks based on each tooth's dental anatomy (i.e. the root apex, the tooth mid-point and a coronal point). For each root, three dental landmarks were placed, based on the following method (Figure 2.3):

1. Cementoenamel junction (CEJ): created using the orientation plane
2. Root Point 3 (RP3): intersection of a circle with a 3-mm radius on the buccal root profile, with the centre corresponding to the CEJ landmark
3. Root Point 6 (RP6): intersection of a circle with a 6-mm radius on the buccal root profile, with the centre corresponding to the CEJ landmark

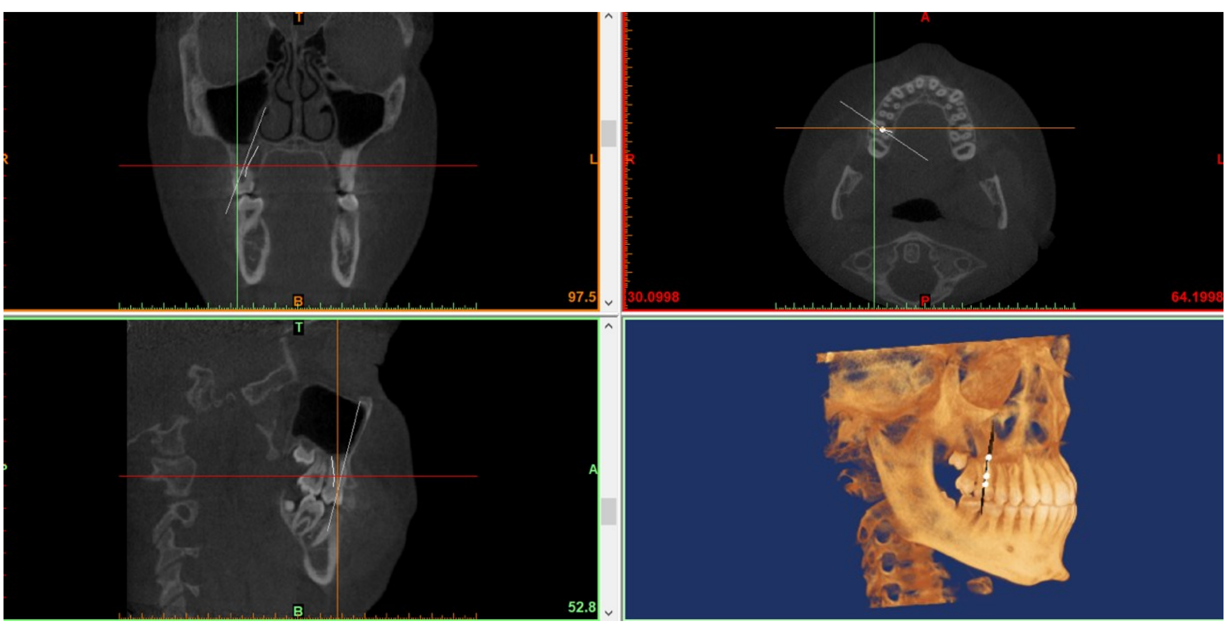


Figure 2.2 Creation of an orientation plane using axial, coronal and sagittal views.

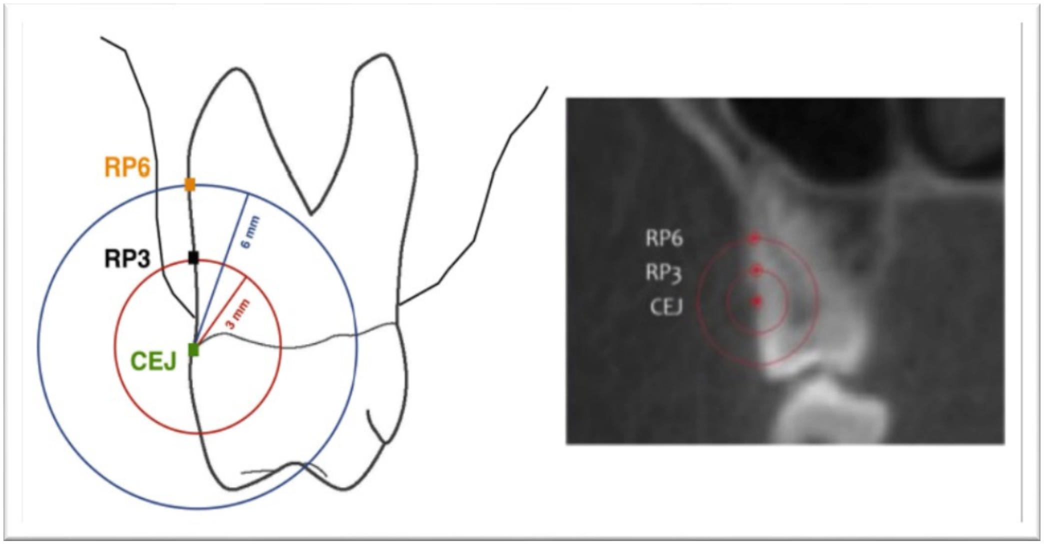


Figure 2.3 Definition of the landmarks for measurements of buccal bone plate thickness. Adapted from DiGregorio et al. (2019).

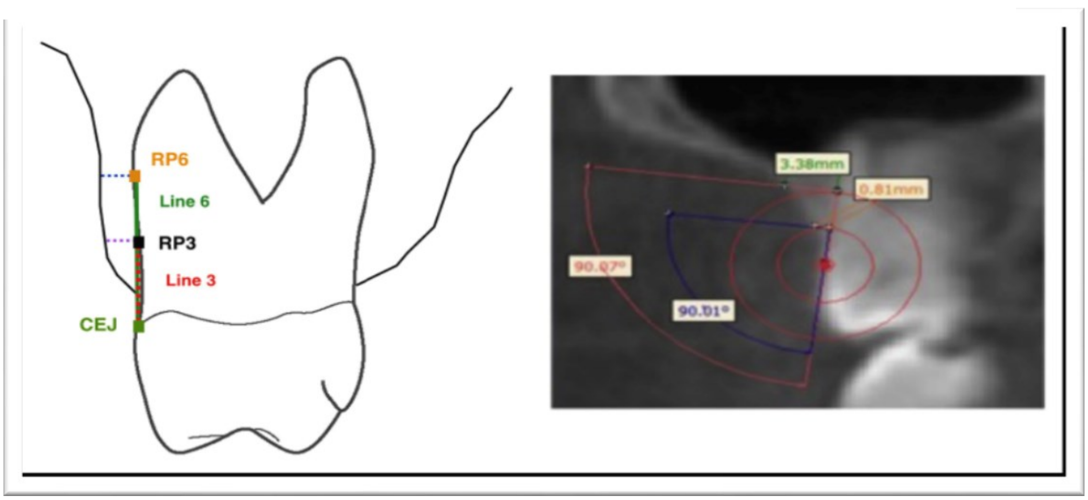


Figure 2.4 Definition of buccal bone plate thickness measurements. Adapted from DiGreorio et al. (2019).

Buccal bone plate thickness was measured on the orientation plane using lines perpendicular to the height of root points 3 (RP3) and 6 (RP6) (Figure 2.4). This step was repeated for each time point: before treatment, at debond and at follow up. See Appendix A, Tables A1.12 and A1.13, for a summary of the orientation plane and landmarks.

2.2.5.1 Reliability Testing

Ten patients were randomly selected from the pool of 45 participants in order to determine both measurement error and method reliability for alveolar bone width. Measurement error was calculated using average differences between repeated measurements. Consistency (intra-rater) was measured using *intra-class correlation coefficient* (ICC) under a single measures two-way mixed model. Measurements were repeated in a blinded fashion 1 week apart by the same examiner (MK). They were repeated three times for measurement error and five times for ICC. The reliability of the measured outcomes, buccal alveolar bone width, was used as an indicator for the reliability of each individual step whereby adequate reliability of the measured alveolus would imply that anatomical landmarking and defining the reference planes were also reliable.

2.2.5.2. Results

The reported average error was 0.21 mm, with a minimum error of 0.09mm for the canines at RP3 and a maximum error of 0.31mm for the canines at RP6 (Appendix A, Table A1.1). Intraclass Correlation Coefficient (ICC) ranged from 0.82 [95%CI, 0.68 to 0.92] to 0.98 [95%CI, 0.95 to 0.99]. A complete table of reliability results can be found in Appendix A, Table A1.2. In general, values above 0.75 are indicative of good reliability.[164]

2.2.6 Photo Analysis

The second part of the study included a photo analysis to determine the prevalence of GR and soft tissue risk factors at debond and two-year follow up. To achieve this, we adapted the *Root coverage esthetic Score* (RES) system described by Cairo et al.,[139] the *Before and After Scoring System* (BASS) described by Kerner et al.,[140] and the *Pink Esthetic Score* (PES) system described by Fürhauser et al.[141] which were validated via a multi-centre evaluation by Le Roch, et al.[142] In this method, each case is assessed using matched clinical views of identical teeth before and after treatment (Figure 2.5). The quality of the tissue was graded based on a visual inspection of soft tissue factors, irrespective of the type of procedure used, the probing depth, and without magnification.

The RES,[139] BASS[140] and PES[141] methods were adapted to analyze factors related to GR before, after and two-years post - orthodontic expansion treatment. For each subject, frontal and lateral intraoral photographs were taken at each time point by two of six calibrated dental assistants using an Olympus TG-6 Camera and intra-oral mirrors, and uploaded into Dolphin Imaging Software (v. 11.95). These photos were used to evaluate the same eight sites as in the CBCT portion of the analysis (i.e. maxillary first molars, first premolars, canines and mandibular first premolars). The factors under investigation were:

1. Gingival Recession (GR)
2. Plaque
3. Keratinized tissue thickness / root prominence (KTt)
4. Keratinized tissue width / height (KTw)
5. Inflammation
6. Presence of black triangles or blunted papilla (BT)

Figure 2.5 Clinical example: before and after comparison of soft tissue T1 vs T2.

GR, KTw, Inflammation and BTs were analyzed by comparing before to after treatment while plaque and KTt were evaluated before treatment. Plaque was evaluated before treatment or at follow up since the teeth are usually polished and plaque free at debond. Similarly, KTt was evaluated before treatment or at follow up since the tissue can be evaluated free from treatment related gingival hypertrophy. All factors were evaluated from the buccal and frontal intra-oral views. If a landmark was missing from both viewpoints, the case was disregarded (n=3). A score of 0 or 1 point was used to denote the presence (1) or absence (0) of each factor. If the RF presented on one site, a score of 1 was assigned to the entire case (Table 2.2). For T2 vs. T3, a score of 1 was given to the GR parameter should the free gingival margin (FGM) be located more apical in T3 than in T2.

Table 2.2 Scoring of risk factors associated with gingival recession (GR).

		Score	
		0	**1**
Parameters	GR	FGM Follows CEJ	FGM is apical to CEJ
	Plaque	no plaque visible	Presence of visible plaque
	KTw	FGM - MGJ ≥ 2mm	FGM - MGJ < 2mm
	KTt	Thick: Roots/ alveolar prominences *not* visible through tissue	Thin: Roots/ alveolar prominences visible through tissue
	Inflammation	Blanching of the MGJ color of marginal tissue is uniform from marginal tissue to vestibule, lack of swelling	No blanching of the MGJ color of marginal tissue varies from color more apical (red), swollen, oedematous
	BTs	complete papilla fill in gingival embrasure	partial (blunted) or total loss of interproximal papilla occurs following treatment

Le Roch et al.[142] found high agreement (κ>.60) for the diagnosis of GR (using the RES,[139] BASS[140] and PES[141] systems). GR was given a score of 1 if the FGM was apical to the CEJ by visual assessment of the lateral and frontal shots.[139,141,165] The CEJ was defined as the junction of the enamel and the root surface of the tooth.[88,109(p29)] Should no color change be visible between the enamel and root surface, the visibility of an anatomical depression between the enamel and cementum was accepted as the CEJ.[88]

Soft tissue aspects of the periodontal biotype were assessed at various time points, depending on the factor under investigation and possible confounders. For example, KTt was assessed on the pre-treatment photographs since debond photos typically showed high levels of hypertrophy and evidence of soft tissue abrasion. It was defined as thin (1) on the basis of visual inspection of the gingival texture, color change with respect to root visibility, and the presence of vasculature and/or whitening of retracted tissues.[76,104] Conversely, KTw was evaluated in the post-treatment photos and was defined as the most apical point of the FGM to its related mucogingival junction. A score of 1 was given when the visual assessment was determined to be <2mm. The mucogingival junction was identified by the color and texture differences between the alveolar mucosa and the keratinized gingiva. If the lip covered the gingiva in the lateral view, the frontal view was used and vice versa. While KTw had low (κ<.40) reproducibility in the BASS[140] system of photo-analysis, it was included as a risk factor since other studies[76,98] found KTw to be a correlate of GR.

Gingival inflammation was assessed in the 'after' photos (i.e. debond and follow up). Gingival inflammation was diagnosed (score of 1) when the visual hallmarks of inflammation (i.e., swelling, edema, redness)[64] were present on visual inspection of the study teeth from either frontal or buccal views. Plaque, on the other hand, was assessed in the before photos via visual appearance of color or texture changes along the tooth surface.

Finally, the changes in papilla height were evaluated by visual inspection of matched intra-oral photos. A score of 1 was assigned to papillae mesial or distal to the study teeth that appeared to have partial or blunted interproximal papilla after treatment.

2.2.6.1 Reliability Testing

Internal consistency with respect to the photo analysis was measured using Cronbach's alpha (α). Assessments of all of the subjects were repeated three times in a blinded fashion one week apart by the same examiner (MK). To test consistency of the method between examiners, assessments were repeated once by a principal investigator periodontist (MPG). Despite the high internal consistency and high reproducibility noted by the aforementioned authors,[139–142] inter-rater correlation was low to moderate ($\kappa = .11\ to\ \kappa = .51$). A third periodontist (DC) was brought in and, after calibration, similar low to moderate results were obtained (Appendix A, Table A1.3). As such, a consensus between MK, MPG and DC was obtained for each parameter by case and time point. A total of 83 cases (i.e. T1vs.T2, T1vs.T3 and T2vs.T3) were reviewed and risk factors were decided upon as a group over the span of one week's time.

2.2.6.2. Results

In general, values above 0.75 are indicative of good reliability.[164] Internal consistency showed good agreement from 0.86 for KTt to 0.99 for GR (Appendix A, Table A1.4). Inter-rater reliability between MPG and MK ranged from low to moderate ($\kappa = .11\ to\ \kappa = .51$). That said, even with a third evaluator (DC), the overall reliability was low to moderate ($\kappa = .11\ to\ \kappa = .51$). As such, a group consensus was determined by two practicing periodontists (MPG and DC) and the principal investigator (MK).

2.3 Collection of Data

2.3.1 Collection of Demographic Data

Demographic information, including age and gender, of the selected (n=45) subjects was gathered to evaluate similarities between treatment groups.

2.3.2 Collection of Data through CBCT Analysis

For each subject who participated and completed the study, the measured buccal bone width (BBW) was organized based on the following parameters:

1. Appliance type (Damon®, Hyrax)
2. Tooth
   a. Maxillary first molars (#1.6, #2.6)
   b. Maxillary first premolars (#1.4, #2.4)
   c. Maxillary canines (#1.3, #2.3)
   d. Mandibular first premolars (#3.4, #4.4)
3. Buccal Alveolar bone width
   a. Buccal alveolar bone width at root point 3 (RP3)
   b. Buccal alveolar bone width at root point 6 (RP6)
4. Time point
   a. Before treatment (T1)
   b. Debond (T2)
   c. Two-year (retention) follow-up (T3)

Continuous measurements were input into an Excel spreadsheet for statistical analysis.

2.3.3 Collection of Data through Photo Analysis

The photos from each of the subjects were analyzed using the methods described by Le Roch et al.[142] at the aforementioned sites for RFs and soft tissue changes with orthodontic expansion treatment.

1. Recession (GR) - progression

2. Plaque – before
3. Keratinized tissue thickness / root prominence (KTt) - before
4. Keratinized tissue width / height (KTw) - after
5. Inflammation - after
6. Presence of black triangles (BT) - after

A score of 0 or 1 point was used to denote the presence (1) or absence (0) of each factor (Table 2.3). If the RF presented on one site, a score of 1 was assigned to the entire case. Categorical measurements were input into an Excel spreadsheet for later statistical analysis.

2.4 Statistical Methods

2.4.1 Part 1 - Analysis of BBW changes assessed through CBCT analysis

2.4.1.1 Data Organization

The dependent variable, buccal alveolar bone width, was measured at the continuous level, whereas the independent factors (teeth, radius, time, and treatment) were measured at the categorical level. There were three within subject factors (teeth, radius, and time) and one between-subject factor (treatment) (Appendix A, Table A1.5).

2.4.1.2 Statistical Test Selection

The decision to use a specific statistical test was dependent on the question to be answered and the assumptions required to satisfy the test's requirements. The number of independent and dependent variables influenced the type of analysis chosen. The significance level for all tests were set to $\propto = 0.05$ for all statistical analyses, which were performed using the IBM SPSS version 27.

A mixed analysis of variance (ANOVA) was chosen to determine whether there were any differences in BBW, for different teeth (i.e. maxillary first molar, first premolars, canines and mandibular first premolars), and root points (i.e. *RP3, RP6*) between baseline and debond. The same test was used to determine whether differences in the change in BBW were related to the type of appliance used for orthodontic expansion (i.e. Damon® or Hyrax) for the same time-frame. A Friedman's test was used to compare the distribution of BBW at baseline, debond and follow up, for 19 patients (38 teeth). Similarly, a Kruskal Wallice H test (KW) was used to compare the distribution of BBW between treatment groups at baseline, debond and follow up, for each *tooth-root-point* (TRP).

2.4.1.3 Data Modification and Calculation of Variables

ANOVA was used to determine whether there were any differences between tooth groups for the BBW prior to treatment (i.e. T1) in order to ensure homogeneity between intervention groups. A summary table of the descriptive statistics for BBW_{T1} can be found in Appendix A, Table A1.6. The data violated the assumption of normality for all TRP groups according to a Shapiro-Wilkes test of normality (Appendix A, Table A1.7). The histogram distribution of each within-subject parameter shows an overall left skewed distribution whereby a large frequency of the measured BBW was zero (Appendix A, Figure A1.1).

To meet the assumptions of ANOVA (i.e. normal distribution, equal variance, sphericity) and reduce the number of within-subjects factors, the data was transformed. That is, teeth were grouped by type (i.e. Maxillary first molars and premolars, canines and mandibular first premolars) and the change (Δ) in BBW between time points was calculated for each tooth-root-point.

The change in buccal alveolar bone width was calculated using the following formulae:

$$\Delta BBW_{T1-T2} = BBW_{T1} - BBW_{T2}$$

$$\Delta BBW_{T1-T3} = BBW_{T1} - BBW_{T3}$$

$$\Delta BBW_{T2-T3} = BBW_{T2} - BBW_{T3}$$

Where *BBW* is the buccal alveolar bone width at *T1, T2* and *T3* are time points before treatment, at debond and two-year follow-up, respectively. The ΔBBW was calculated for each root point (i.e. *RP3, RP6*) separately.

Mixed ANOVA was used to determine mean differences for ΔBBW_{T1-T2}. Descriptive statistics for the ΔBBW_{T1-T2} at each TRP are summarized in Appendix A, Table A1.8. Although violations of normality were observed, especially in the maxillary canines and mandibular first premolars, ANOVA tests are robust against departures from normality provided that the sample sizes are large (n>30) and approximately equal. Since the sample size was sufficiently large (i.e. 45 patients and 90 teeth) and the data satisfied the assumptions of normality and sphericity, no further transformation was performed.

That said, the assumptions for ANOVA (i.e. equal variance, normality and minimum sample size were) violated for ΔBBW_{T1-T3} and ΔBBW_{T2-T3}, despite data transformation. A Friedman's test was used to compare the distribution of BBW at baseline, debond and follow up, for 19 patients (38 teeth). Similarly, a Kruskal Wallice H test (KW) was used to compare the distribution of BBW between treatment groups at baseline, debond and follow up, for each TRP. Refer to Appendix 2B for complete data tables and specifics associated with hypothesis testing.

2.4.2 Part 2 - Analysis of soft tissue changes assessed through photo analysis

2.4.2.1 Data Organization

All of the dependent (i.e. GR) and independent (i.e. Plaque, KTt, KTw, Inflammation, BT) variables were measured at the categorical (dichotomous) level (Appendix A, Table A1.9).

2.4.2.2 Statistical test selection

To evaluate the risk factors associated with GR after orthodontic treatment, a binomial logistic regression (multiple discrete variables vs. one discrete variable) was performed to determine the effects of the risk factors on the likelihood that participants have GR at debond or two year follow up. This was performed for T1vs.T2 (n=45), T1vs.T3 (n=19) and T2vs.T3 (n=19).

2.4.3 Part 3 - Relationship of BBW to GR

2.4.3.1 Data Organization

The dependent variable, GR, was measured at the discrete, dichotomous level (i.e. 0,1), whereas the independent factor, BBW, was measured at the continuous level at each time point. Raw data BBW measurements were used at the later time points (i.e. T2, T3) to evaluate the relationship between dehiscence and recession defects after orthodontic treatment.

2.4.3.2 Data Modification and Statistical Test Selection

A binomial logistic regression was conducted to determine the relationship of BBW on the likelihood that participants have GR at debond or two year follow up.

Since soft-tissue scores were assigned to a case, and continuous BBW measurements were assigned to tooth root points, the raw data required transformation. $BBW_{T2,\ T3}$ measurements were re-coded into categorical data ($BBW'_{\ T2,T3}$), as follows:

- 0 – RP3 & RP6 > 0mm
- 1 – RP3 = 0mm; RP6 > 0mm
- 2 – RP3 & RP6 = 0mm

Since the presence of a dehiscence is a prerequisite for GR ,[28,45,55,76,84] only dehiscences (i.e. not fenestrations) were considered in the recoding. Should buccal alveolar bone be present at RP3 and RP6 for all tooth-root points, the teeth under investigation would be within the alveolar housing, and therefore protected against GR formation.[14,28,71,85,89,109] If BBW at RP3 and RP6 was greater than zero, a score of *zero* was assigned. If BBW was zero on one tooth such that a dehiscence was present, (i.e. RP3=0), a score of *one* was assigned to the participant. Similarly, if BBW was zero at both RP3 and RP6 on one tooth, then a score of two was assigned to the participant (Appendix A, Table A1.9)

Appendix A: Tables & Figures

Table A1.1 Measurement Error of CBCT Method.

Tooth Group	Tooth	Measurement Error		Measurement Error by Tooth Group	
		RP3	*RP6*	*RP3*	*RP6*
Max	#16	0.18	0.37	0.14	0.30
Molars	#26	0.11	0.23		
Max	#14	0.17	0.31	0.18	0.26
PM1	#24	0.19	0.21		
Max	#13	0.10	0.24	0.09	0.31
Canines	#23	0.07	0.37		
Mand	#44	0.19	0.19	0.18	0.21
PM1	#34	0.18	0.24		
average		**0.21**		**0.21**	

Table A1.2 Intraclass Correlation Coefficient for BBW (mm) on maxillary first molars, first premolars, canines and mandibular first premolars at RP3 and RP6.

Patient	Intraclass Correlation	95% Confidence Interval	
		Lower Bound	*Upper Bound*
1	.814	.669	.919
2	.976	.952	.990
3	.881	.777	.950
4	.904	.817	.960
5	.949	.900	.979
6	.824	.684	.924
7	.910	.828	.963
8	.825	.687	.924
9	.897	.805	.957

Two-way mixed effects model where people effects are random and measures effects are fixed.
The estimator is the same, whether the interaction effect is present or not
Type 3 intraclass correlation coefficients using a consistency definition. The between-measure variance is excluded from the denominator variance[164]

Table A1.3 Internal reliability for photo-analysis of gingival recession (GR), keratinized tissue width (KTw), keratinized tissue thickness (KTt), Plaque, Inflammation and Black Triangles (BTs) using Cronbach's alpha (α).

Risk Factor	**α**	**Statistics**		
		First *mean (SD)*	Second *Mean (SD)*	Third *Mean (SD)*
GR	.814	.44 (.50)	.42 (.50)	.44 (.50)
KTw	.964	.56 (.50)	.58 (.50)	.51(.51)
KTt	.855	.67 (.48)	.67 (.48)	.69 (.47)
Plaque	.985	.78 (.42)	.80 (.40)	.80 (.40)
Inflammation	.899	.96 (.21)	.98 (.15)	.98 (.15)
BTs	.968	.44 (.50)	.42 (.50)	.40 (.50)

Table A1.4 Intra-rater reliability for photo-analysis of gingival recession (GR), keratinized tissue width (KTw), keratinized tissue thickness (KTt), plaque, inflammation and black triangles (BTs) using Fleiss' Kappa and weighted Kappa (κ).

Risk Factor	**Raters**	**Weighted κ**	**95% confidence interval**		**p-value**
			Lower bound	*Upper bound*	
GR	***Fleiss' κ***	***.35***	***.18***	***.52***	***.000***
	MPG - MK	**.51**	**.26**	**.76**	**.001**
	MPG - DC	**.33**	**.05**	**.60**	**.027**
	MK - DC	.20	-.08	.49	.175
KTw	***Fleiss' κ***	***.18***	***.01***	***.35***	***.035***
	MPG - MK	**.34**	**.08**	**.61**	**.010**
	MPG - DC	.09	-.19	.37	.533
	MK - DC	.15	-.14	.44	.309
KTt	***Fleiss' κ***	***.39***	***.21***	***.55***	***.000***
	MPG - MK	**.42**	**.16**	**.69**	**.005**
	MPG - DC	**.42**	**.16**	**.69**	**.005**
	MK - DC	.29	.01	.57	.053
Plaque	***Fleiss' κ***	***.25***	***.08***	***.42***	***.003***
	MPG - MK	.24	-.07	.55	.105
	MPG - DC	.28	-.04	.59	.062
	MK - DC	.24	-.07	.55	.105
Inflammation	***Fleiss' κ***	*.05*	*-.12*	*.22*	*.58*
	MPG - MK	.16	-.19	.52	.169
	MPG - DC	.00	.00	.00	.
	MK - DC	.00	.00	.00	.
BTs	***Fleiss' κ***	*.11*	*-.06*	*.28*	*.197*
	MPG - MK	.11	-.18	.39	.465
	MPG - DC	.20	-.07	.48	.155
	MK - DC	.07	-.19	.33	.616

Table A1.5 CBCT Study Variables.

Factors		Name	Measurement	Levels
Dependent		Buccal alveolar bone thickness	Continuous	
Independent	Between Subjects	Treatment	Categorical	1. Hyrax 2. Damon®
	Within Subjects	Tooth	Categorical	1. R & L Maxillary first molar (MxM1) 2. R & L Maxillary first molar (MXPM1) 3. R & L Maxillary first premolar (MXC) 4. R & L mandibular first premolar (MdPM1)
		Root Point	Categorical	1. Root Point 3 (RP3) 2. Root Point 6 (RP6)
		Time	Categorical	1. Before Treatment 2. After Treatment

Table A1.6 Descriptive Statistics for BBW (mm) at Baseline (T1), Debond (T2) and Follow Up (T3).Descriptive Statistics for BBW (mm) at Baseline (T1), Debond (T2) and Follow Up (T3).

		T1 (n_T=90) (n_D=48; n_H=42)					T2 (n_T=90) (n_D=48; n_H=42)					T3 (n_T=38) (n_D=16; n_H=22)				
		Min (mm)	*Max (mm)*	*Mean (mm)*	*Std. Dev*	*Variance*	*Min (mm)*	*Max (mm)*	*Mean (mm)*	*Std. Dev*	*Variance*	*Min (mm)*	*Max (mm)*	*Mean (mm)*	*Std. Dev*	*Variance*
Molars RP3		.00	3.08	1.08	.70	.48	.00	2.48	0.95	0.65	0.42	.00	1.57	.58	.55	.31
	Damon®	.00	3.08	1.17	.76	.57	.00	2.48	0.95	0.65	0.42	.00	1.57	.61	.55	.30
	Hyrax	.00	1.83	.97	.61	.37	.00	1.73	0.59	0.62	0.38	.00	1.55	.56	.57	.32
Molars RP6		.00	3.53	1.25	.81	.65	.00	3.09	0.74	0.72	0.51	.00	3.50	.59	.79	.63
	Damon®	.00	3.53	1.38	.92	.85	.00	3.09	0.89	0.82	0.67	.00	3.50	.86	1.08	1.17
	Hyrax	.00	2.04	1.11	.63	.39	.00	1.75	0.56	0.54	0.29	.00	1.28	.40	.42	.18
MxPM RP3		.00	1.65	.80	.51	.26	.00	2	0.6	0.55	0.3	.00	1.74	.46	.52	.27
	Damon®	.00	1.63	.80	.48	.23	.00	2	0.68	0.56	0.32	.00	1.74	.61	.57	.33
	Hyrax	.00	1.65	.80	.55	.31	.00	1.82	0.52	0.52	0.27	.00	1.23	.34	.45	.20
MxPM RP6		.00	2.43	.89	.56	.31	.00	2.19	0.73	0.61	0.38	.00	1.35	.41	.38	.14
	Damon®	.00	1.64	.81	.52	.27	.00	2.06	0.75	0.59	0.35	.00	1.11	.37	.33	.11
	Hyrax	.00	2.43	.98	.60	.36	.00	2.19	0.71	0.65	0.42	.00	1.35	.43	.41	.17
Canines RP3		.00	1.36	.34	.43	.19	.00	1.95	0.19	0.4	0.16	.00	.94	.30	.35	.13
	Damon®	.00	1.29	.26	.39	.15	.00	1.95	0.21	0.44	0.2	.00	.94	.29	.39	.15
	Hyrax	.00	1.36	.44	.46	.21	.00	1.08	0.16	0.35	0.12	.00	.85	.31	.33	.11
Canines RP6		.00	1.34	.58	.41	.16	.00	2.45	0.52	0.47	0.22	.00	1.06	.48	.38	.15
	Damon®	.00	1.21	.59	.37	.13	.00	2.45	0.52	0.49	0.24	.00	1.06	.42	.42	.17
	Hyrax	.00	1.34	.56	.45	.20	.00	1.78	0.52	0.45	0.2	.00	1.02	.52	.36	.13
MdPM RP3		.00	1.38	.31	.35	.13	.00	1.18	0.2	0.31	0.1	.00	.95	.15	.29	.08
	Damon®	.00	.98	.24	.32	.10	.00	0.96	0.15	0.29	0.08	.00	.90	.20	.32	.10
	Hyrax	.00	1.38	.39	.38	.14	.00	1.18	0.26	0.33	0.11	.00	.95	.11	.26	.07
MdPM RP6		.00	1.71	.49	.48	.23	.00	1.9	0.32	0.42	0.17	.00	.72	.12	.24	.06
	Damon®	.00	1.57	.44	.45	.21	.00	1.45	0.35	0.4	0.16	.00	.72	.12	.26	.07
	Hyrax	.00	1.71	.55	.50	.25	.00	1.9	0.29	0.43	0.19	.00	.60	.12	.23	.05

Where n_T= number total teeth measured, n_D=number of teeth measured for the Damon® group and n_H= number of teeth measured for the Hyrax group

Table A1.7 Shapiro-Wilk Tests of Normality for BBW at T1.

	Statistic	df	Sig.
MolarsRP3PRE	.92	90.00	.00
MolarsRP6PRE	.94	90.00	.00
MxPMRP3PRE	.90	90.00	.00
MxPMRP6PRE	.94	90.00	.00
CaninesRP3PRE	.76	90.00	.00
CaninesRP6PRE	.90	90.00	.00
MdPMRP3PRE	.80	90.00	.00
MdPMRP6PRE	.87	90.00	.00

Figure A1.1 Histograms of BBW distribution at T1

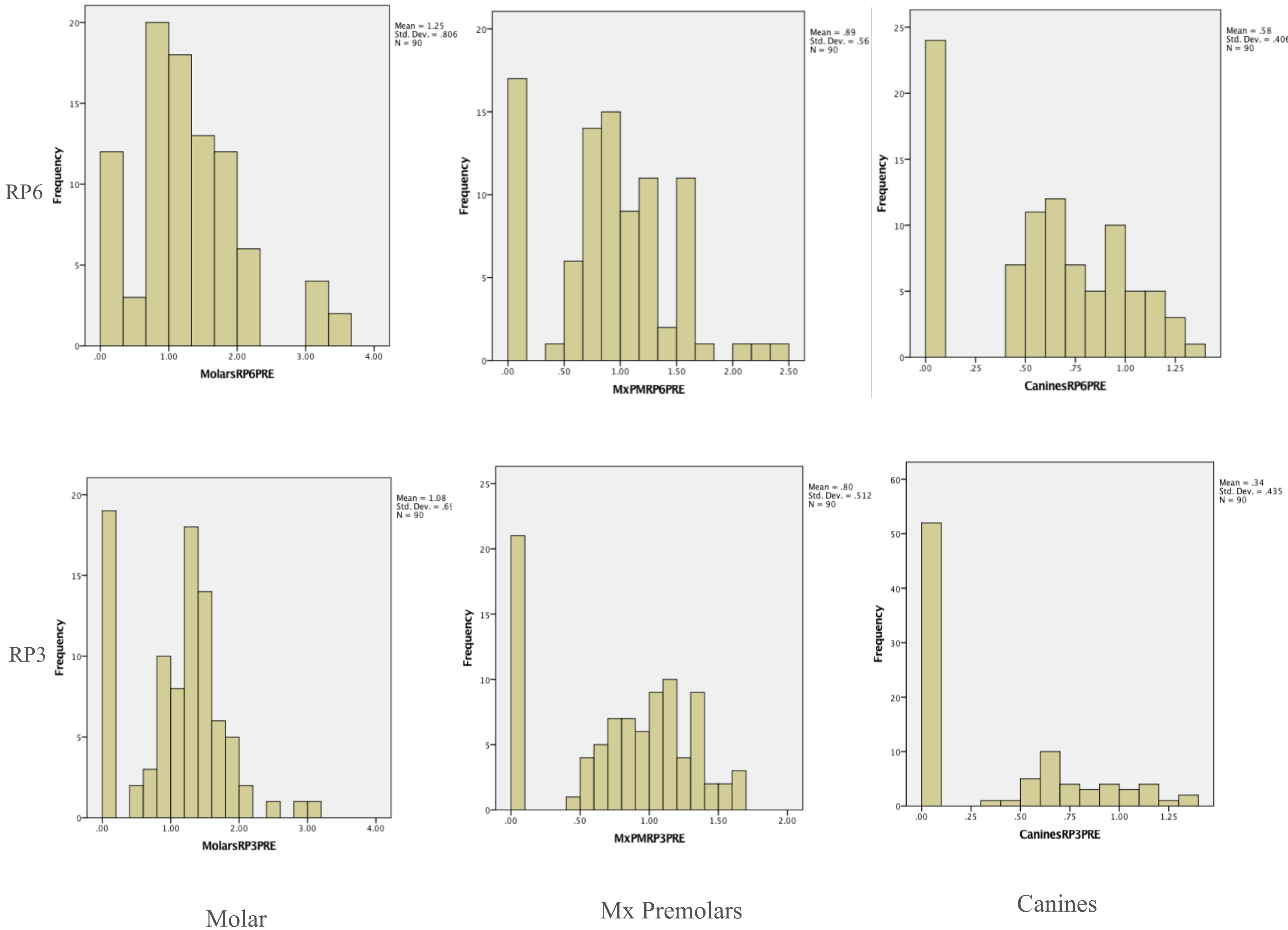


Table A1.8 Descriptive statistics for ΔBBW_{T1-T2}

		N	Range	Minimum (mm)	Maximum (mm)	Mean (mm)	Std. Error	Std. Deviation	Variance
MolarsRP3		90	3.23	-1.26	1.97	.29	.07	.68	.46
	Damon®	48	3.23	-1.26	1.97	0.22	0.09	0.62	0.39
	Hyrax	42	2.65	-1.14	1.51	0.38	0.11	0.74	0.54
MolarsRP6		90	3.26	-1.03	2.23	.52	.07	.67	.45
	Damon®	48	3.26	-1.03	2.23	0.49	0.1	0.71	0.51
	Hyrax	42	2.71	-0.69	2.02	0.55	0.1	0.63	0.39
MxPMRP3		90	2.81	-1.20	1.61	.19	.06	.61	.37
	Damon®	48	2.59	-1.2	1.39	0.12	0.09	0.63	0.4
	Hyrax	42	2.57	-0.96	1.61	0.27	0.09	0.58	0.34
MxPMRP6		90	3.71	-1.28	2.43	.16	.06	.60	.36
	Damon®	48	2.46	-0.95	1.51	0.06	0.08	0.54	0.29
	Hyrax	42	3.71	-1.28	2.43	0.26	0.1	0.66	0.43
CaninesRP3		90	3.27	-1.95	1.32	.15	.05	.52	.27
	Damon®	48	3.24	-1.95	1.29	0.04	0.07	0.51	0.26
	Hyrax	42	2.35	-1.03	1.32	0.28	0.08	0.5	0.25
CaninesRP6		90	2.84	-1.70	1.14	.06	.05	.51	.26
	Damon®	48	2.83	-1.7	1.13	0.07	0.07	0.51	0.26
	Hyrax	42	2.32	-1.18	1.14	0.04	0.08	0.53	0.28
MdPMRP3		90	1.80	-.80	1.00	.11	.04	.39	.15
	Damon®	48	1.69	-0.72	0.97	0.09	0.05	0.35	0.12
	Hyrax	42	1.8	-0.8	1	0.13	0.07	0.43	0.19
MdPMRP6		90	2.87	-1.24	1.63	.17	.06	.56	.31
	Damon®	48	2.73	-1.16	1.57	0.09	0.08	0.55	0.3
	Hyrax	42	2.87	-1.24	1.63	0.26	0.09	0.56	0.32

Table A1.9 Risk Factors for Recession.

Variable Type	Name	Measurement	Levels
Dependent	Gingival Recession	Categorical, Dichotomous	0 – Follows CEJ 1 – Apical to CEJ
Independent	Plaque	Categorical, Dichotomous	0 – no plaque visible at T1 1 - plaque visible at T1
	KG thickness	Categorical, Dichotomous	0 - FGM - MGJ > 2mm 1 - FGM - MGJ < 2mm
	KG width	Categorical, Dichotomous	0 – Thick: Roots/ alveolar prominences not visible through tissue 1 - Thin: Roots/ alveolar prominences visible through tissue
	Inflammation	Categorical, Dichotomous	0 - color of marginal tissue is uniform from marginal tissue to vestibule, lack of swelling 1 - varies from color more apical (red), swollen, oedematous
	Black Triangles	Categorical, Dichotomous	0 complete papilla fill in gingival embrasure 1- partial (blunted) or total loss of interproximal papilla occurs following treatment
	BBW	Categorical	0 – RP3 & RP6 > 0mm 1 – RP3 = 0mm; RP6 > 0mm 2 – RP3 & RP6 = 0mm

Table A1.10 Crosstabulation for BBW=0 by TRP at T1, T2 and T3.

	T1								
	Mx M1		Mx PM1		Mx C		Md PM1		
Tx	Root Point								**Totals**
	3	**6**	**3**	**6**	**3**	**6**	**3**	**6**	
Hyrax	10	7	12	7	20	14	18	14	**102**
Damon®	9	5	9	10	32	10	29	20	**124**
Totals	**19**	**12**	**21**	**17**	**52**	**24**	**47**	**34**	**226**
	T2								
	Mx M1		Mx PM1		Mx C		Md PM1		
Tx	Root Point								**Totals**
	3	**6**	**3**	**6**	**3**	**6**	**3**	**6**	
Hyrax	20	17	18	13	34	15	25	26	**168**
Damon®	11	15	16	12	37	17	36	24	**168**
Totals	**31**	**32**	**34**	**25**	**71**	**32**	**61**	**50**	**226**
	T3								
	Mx M1		Mx PM1		Mx C		Md PM1		
Tx	Root Point								**Totals**
	3	**6**	**3**	**6**	**3**	**6**	**3**	**6**	
Hyrax	10	10	13	9	11	6	18	17	**94**
Damon®	6	6	6	6	10	7	11	13	**65**
Totals	**16**	**16**	**19**	**15**	**21**	**13**	**30**	**30**	**160**

Table A1.11 Diagnostic uses of CBCT in orthodontics. Adapted from the American Academy of Oral and Maxillofacial Radiology (2013)[39]

Dental structural anomalies	These comprise assessments of variations in tooth morphology, hypodontia, retained primary teeth, supernumeraries/gemination/fusion, root abnormalities, and external and internal resorption.
Anomalies in dental position	These include dental impactions, presence of unerupted and impacted supernumeraries, locations of molars in relation to the inferior alveolar canals, anomalies in eruption sequences, and ectopic eruptions (including teeth in clefts).
Compromised dento-alveolar boundaries	The assessment of dento-alveolar volume (in addition to that which can be determined by clinical examination and study models) is needed when there is reduced buccal/lingual alveolar width, bimaxillary protrusion, compromised periodontal status, and/or clefts of the alveolus.
Asymmetry	Clinically, asymmetry presents as chin or mandibular deviation, dental midline deviation, and/or occlusal cant discrepancies as well as other dental and craniofacial asymmetries.
Anteroposterior discrepancies	These are skeletally based Class II and Class III malocclusions.
Vertical discrepancies	Initial facial patterns assessed clinically or radiographically may suggest skeletal discrepancies related to vertical maxillary deficiency or excess and may present as anterior open bite or deep overbite.
Transverse discrepancies	These anomalies may be present as either skeletal lingual or buccal crossbites or discrepancies without the presence of crossbites in which there is excessive dental compensation of the bucco-lingual inclination of posterior teeth.
Temporomandibular joint (TMJ) signs and/or symptoms	TMJ pathoses that result in alterations in the size, form, quality and spatial relationships of the osseous joint components may lead to skeletal and dental discrepancies in the three planes of space. In affected condyles, perturbed resorption and/or apposition can lead to progressive bite changes and compensations in the maxilla. In addition, tooth position, occlusion and the articular fossa of the non-affected side of the mandible can become involved. The sequelae of these changes are unpredictable orthodontic outcomes. Such TMJ conditions include developmental disorders such as condylar hyperplasia, hypoplasia, or aplasia, arthritic degeneration, persistently symptomatic joints, and bite changes including progressive bite opening and limitation or deviation upon opening or closing.
Dentofacial deformities and craniofacial anomalies	CBCT imaging can facilitate analysis of these conditions and be used to simulate virtual treatments and plan orthopedic corrections and orthognathic surgeries. Computer-aided jaw surgery is increasing in use clinically because virtual plans accurately represent surgical procedures in the operating room.
Conditions that affect airway morphology	A number of authors have used CBCTimaging to measure airway dimensions and reported changes over time with specific therapies including orthognathic surgery and particularly obstructive sleep apnea. There are challenges in the use of CBCT clinically as the validity of such measurements may vary. The boundaries of the nasopharynx with the maxillary/paranasal sinuses and of the oropharynx with the oral cavity are often not consistent among subjects and image acquisitions, and airway shapes and volumes vary markedly with dynamic processes such as breathing and head postures. In addition, CBCT has been reported useful in preoperative assessment and/or postoperative evaluation of treatment outcomes for specific research applications including:
Specific surgical procedures	Research in the areas of craniofacial growth and development as well as assessments of the short- and long-term outcomes of various treatment regimens has the potential to benefit from CBCT assessments of longitudinal changes and diagnostic characterization of tooth and facial morphology of hard and soft tissues. Studies on the morphological basis for craniofacial growth and response to treatment can help elucidate clinical questions on variability of outcomes of treatment, as well as clarify treatment effects and areas of bone remodeling and displacement
Orthodontic mini-implants used as temporary anchorage devices	Numerous authors have identified CBCT imaging as being clinically useful in identifying optimal site location for placement of orthodontic mini-implants.
Maxillary expanders	CBCT imaging of maxillary transverse deficiencies treated with fixed and removable expanders has been reported of benefit in characterizing appliance specific skeletal displacement, associated dental effects and quantifying changes in skeletal dimensions of the nasal cavity and maxillary sinus volume.

Table A1.12 Table of Orientation planes

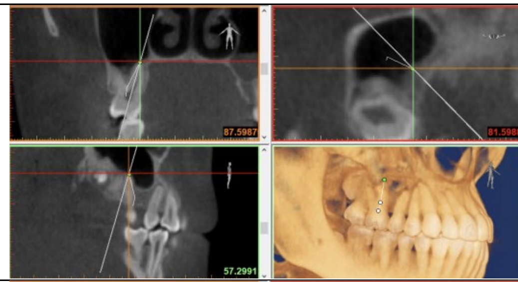
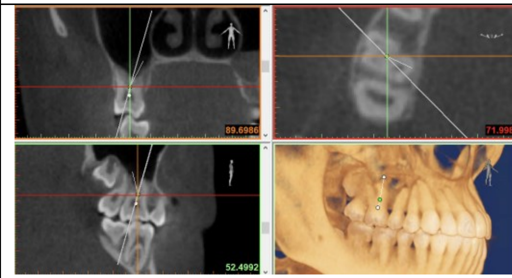
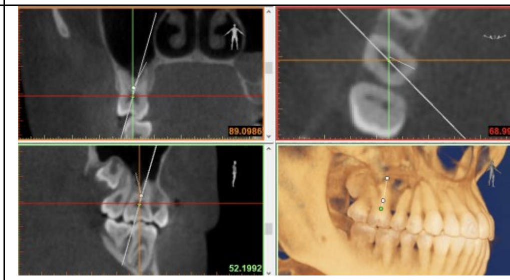
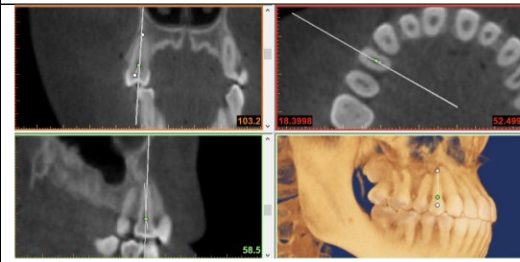
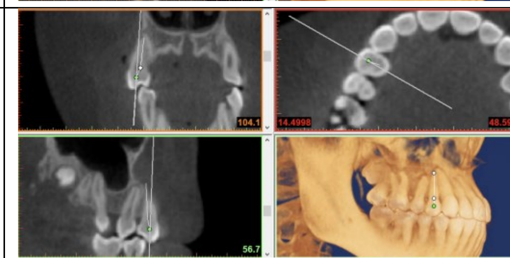
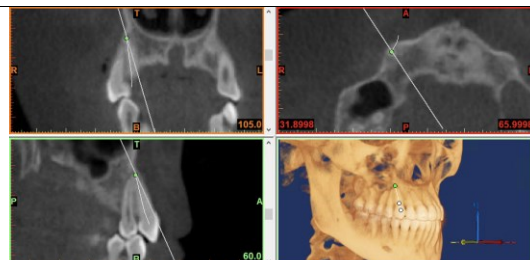
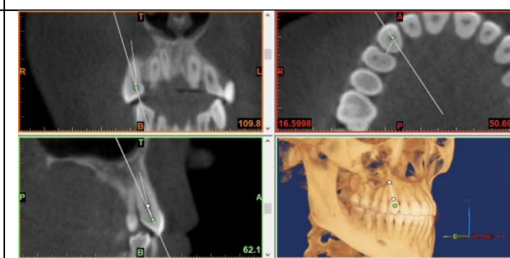
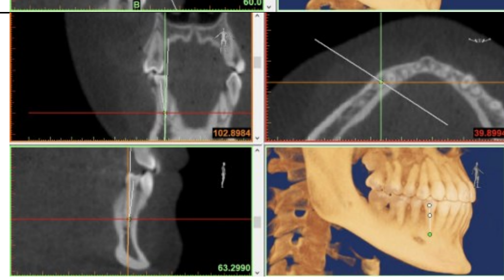
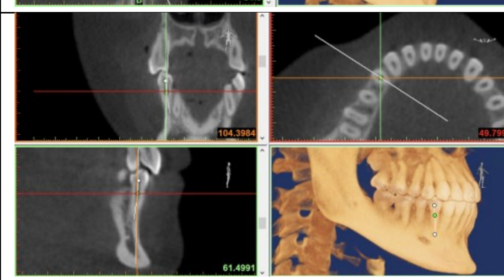
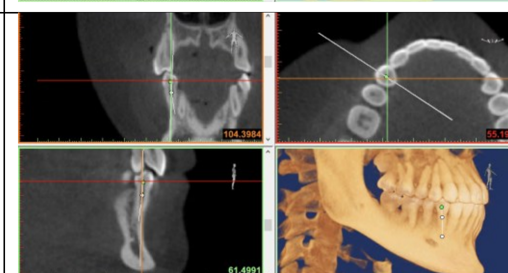
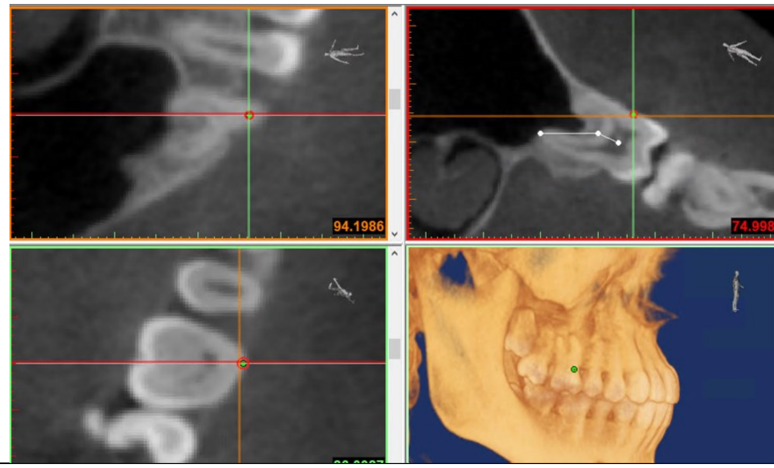
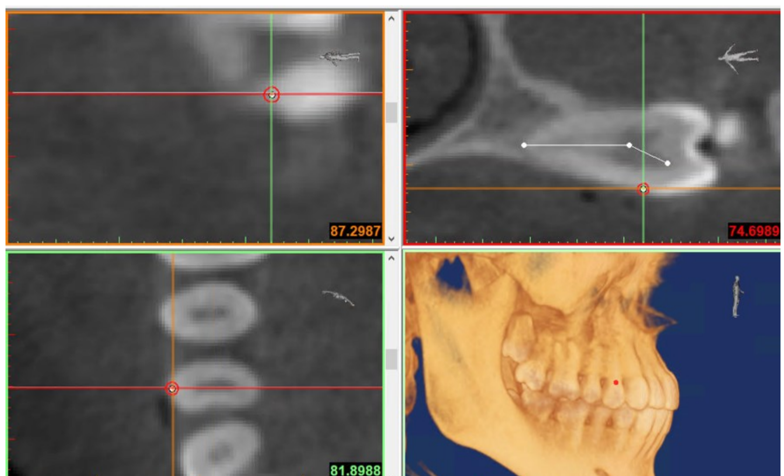
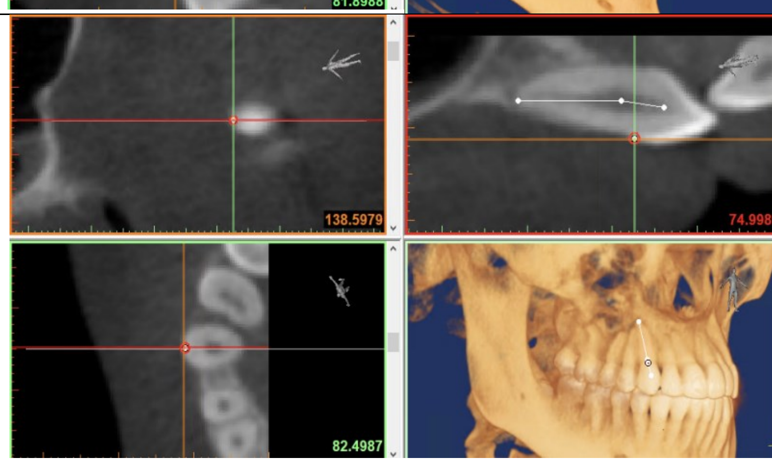
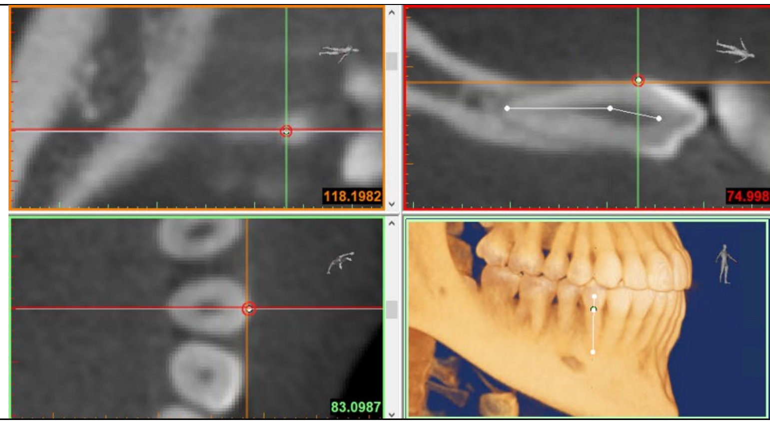
	Description	Apical Point (a)	Middle point (b)	Coronal Point (c)
Maxillary Molars (#1.6, #2.6)	a) Root apex of palatal root b) Centre of buccal furcation c) Centre of pulp chamber			
Maxillary Premolars (#1.4, #2.4)	a) Root apex of buccal root b) Centre of pulpal canal at level of interproximal crestal bone c) buccal pulp horn			
Maxillary Canines (#1.3, #2.3)	a) Root apex b) Middle of pulpal canal at level of interproximal crestal bone c) coronal most aspect of pulp horn			
Mandibular Premolars (#3.4, #4.4)	a) Root apex of buccal root b) Middle of pulpal canal at level of interproximal crestal bone c) buccal pulp horn			

Table A1.13 Location of the CEJ using the orientation plane		
	Description	CEJ Location
Maxillary First Molars (#1.6, #2.6)	Mesiobuccal aspect of facial surface	
Maxillary First Premolars (#1.4, #2.4)	Midfacial aspect	
Maxillary Canines (#1.3, #2.3)	Midfacial aspect	

Mandibular First Premolars (#3.4, #4.4)	Midfacial aspect	

Appendix B: Hypothesis Testing

The decision to use a specific statistical test was dependent on the question to be answered. This study is organized in two parts, based on the specific questions asked and the parameters evaluated. The number of independent and dependent variables influenced the type of analysis chosen.

For the first part of the study, the authors were interested in determining the response of the buccal alveolar bone width (continuous within-subjects, dependent variable) at baseline, debond and two year follow up (categorical, within-subjects factor) at the at the two root points on each tooth (categorical, within-subjects factors) to orthodontic treatment overall and between Damon® and Hyrax treatment groups (categorical, between-subjects factor)(Table B1.1).

For the second part of the study, the authors were interested in the relationship between risk factors of gingival recession and the presence of gingival recession with orthodontic expansion treatment. The dependent variable, gingival recession (categorical) was compared to independent risk factors (categorical), including:

- Keratinized Tissue thickness / Root Prominence (KTt)
- Keratinized Tissue vertical width / height (KTw)
- Initial Plaque
- Inflammation - after
- Black triangles – after (BT)
- Buccal alveolar bone thickness - after (BBW')

The risk factors and their codes are summarized in Table B1.2. Since the presence of dehiscence (i.e. absence of buccal alveolar bone) is a prerequisite for gingival recession formation, the measured BBW at debond and follow-up were re-coded to account for severity of dehiscence (Section 2.4.3.2).

Table B1.1 CBCT study variables.

Factors		Name	Measurement	Levels
Dependent		Buccal alveolar bone thickness	Continuous	
Independent	Between Subjects	Treatment	Categorical	1. Hyrax 2. Damon®
	Within Subjects	Tooth	Categorical	5. R & L Maxillary first molar (MxM1) 6. R & L Maxillary first molar (MXPM1) 7. R & L Maxillary first premolar (MXC) 8. R & L mandibular first premolar (MdPM1)
		Root Point	Categorical	3. Root Point 3 (RP3) 4. Root Point 6 (RP6)
		Time	Categorical	3. Before Treatment 4. After Treatment

Table B1.2 Risk factors for recession.

Variable Type	Name	Measurement	Levels
Dependent	Gingival Recession	Categorical, Dichotomous	0 – Follows CEJ 1 – Apical to CEJ
Independent	Plaque	Categorical, Dichotomous	0 – no plaque visible at T1 1 - plaque visible at T1
	KG thickness	Categorical, Dichotomous	0 - FGM - MGJ > 2mm 1 - FGM - MGJ < 2mm
	KG width	Categorical, Dichotomous	0 - Thick: Roots/ alveolar prominences not visible through tissue 1 - Thin: Roots/ alveolar prominences visible through tissue
	Inflammation	Categorical, Dichotomous	0 - color of marginal tissue is uniform from marginal tissue to vestibule, lack of swelling 1 - varies from color more apical (red), swollen, oedematous
	Black Triangles	Categorical, Dichotomous	0 complete papilla fill in gingival embrasure 1- partial (blunted) or total loss of interproximal papilla occurs following treatment
	BBW	Categorical	0 – RP3 & RP6 > 0mm 1 – RP3 = 0mm; RP6 > 0mm 2 – RP3 & RP6 = 0mm

B1. Hypothesis Testing for ∆BBW

To investigate the change (∆) in buccal alveolar width (BBW) from before to after and two-years post-expansion treatment, regardless of the appliance type and between treatment (Damon® and Hyrax) groups, the following hypotheses was tested:

Ho_1: There is no change in mean buccal alveolar bone widths measured at two root points on four teeth before and after treatment, regardless of appliance type

Ha_1: There is a change in mean buccal alveolar bone widths measured at two root points on four teeth before and after treatment, regardless of appliance type

Ho_2: There is no change in mean buccal alveolar bone widths measured at two root points on four teeth before and after treatment with either Damon® or Hyrax appliances

Ha_2: There is a change in mean buccal alveolar bone widths measured at two root points on four teeth before and after treatment with either Damon® or Hyrax appliances

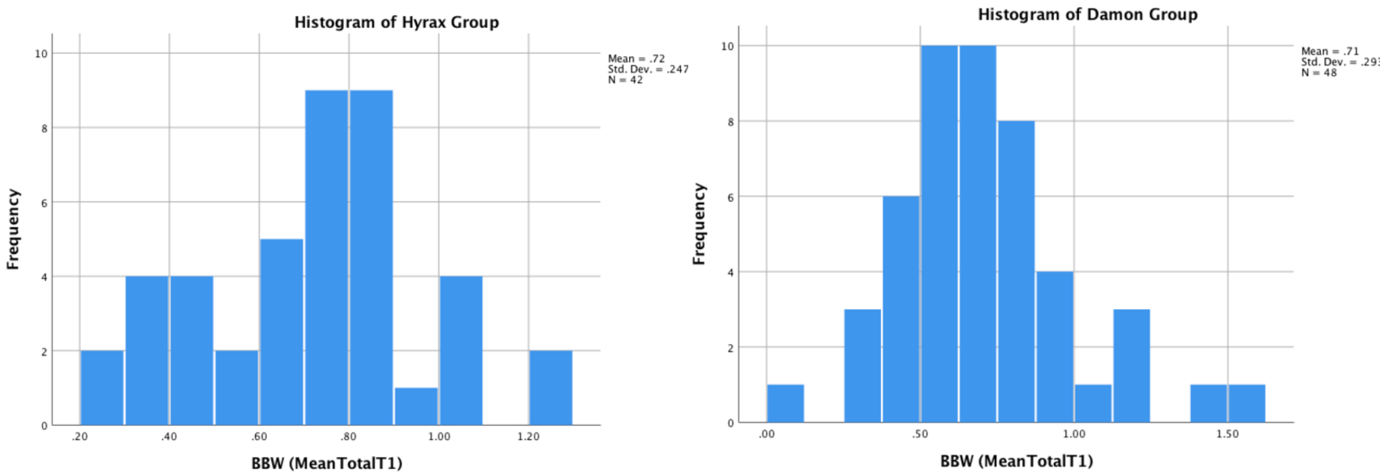
B2. Baseline Comparisons

B2.1 Comparisons of Pre-Treatment Factors

In order to ensure that the treatment groups, Hyrax and Damon®, were homogenous at T1 an ANOVA omnibus test was used. A summary table of the descriptive statistics for BBW_{T1} can be found in Appendix A, Table A1.6. The collected data violated the assumption of normality for all tooth-root-point (TRP) groups according to a Shapiro-Wilk's test of normality (Appendix A, Table A1.7). The histogram distribution of each within-subject parameter shows an overall left skewed distribution whereby a large frequency of the measured BBW was zero (Appendix A, Figure A1.1). Data transformation was required to correct for the skew.

Checking the assumptions for ANOVA, the independent sampling requirement was fulfilled as the sampling of one participant was not influenced by the selection of another. The normal distribution assumption was violated for the Damon® group according to Shapiro-Wilk's assessment of Normality ($p=0.043$); however, a visual inspection the histograms shows relative symmetry with only mild left skewedness (Figure B1.1). The equal variance assumption was met as assessed by the Levene's Test of Equal variance for the mean total BBW ($F(3, 86) = .828$, $p = 0.482$).

Figure B1.1Histograms of baseline BBW (mean total) by treatment group (mm).



B2.2 Comparisons of Transformed Factors

The data was transformed in order to meet the assumptions of parametric statistics (i.e. normal distribution, equal variance, sphericity) and reduce the number of within-subject factors. Observations were grouped into their respective Tooth-Root Point (TRP) combinations, and the ΔBBW between time points was calculated, were grouped by type (i.e. Maxillary first molars and premolars, maxillary canines and mandibular first premolars) and the ΔBBW between TRPs (i.e. RP3 and RP6) was calculated for each time point.

The change in buccal alveolar bone width was calculated using the following formulae:

$$\Delta BBW_{T1-T2} = BBW_{T1} - BBW_{T2}$$

$$\Delta BBW_{T1-T3} = BBW_{T1} - BBW_{T3}$$

$$\Delta BBW_{T2-T3} = BBW_{T2} - BBW_{T3}$$

Where *BBW* is the buccal alveolar bone width and *T1, T2* and *T3* are time points before treatment, at debond, and at two-year follow-up (retention), respectively. Descriptive statistics for the ΔBBW at each TRP for each time pair (i.e. T1-T2, T1-T3 and T2-T3) are summarized in Table B1.3.

Table B1.3 Descriptive statistics for ΔBBW at each TRP for each time comparison (CBCT analysis).

	ΔBBW_{T1-T2} N=90		ΔBBW_{T1-T3} N=38		ΔBBW_{T2-T3} N=38	
	Mean (mm)	SD	Mean (mm)	SD	Mean (mm)	SD
Max M1 RP3	0.29	0.07	0.41	0.09	0.03	0.07
Max M1 RP6	0.52	0.07	0.42	0.11	0.04	0.06
Max PM1 RP3	0.19	0.06	0.38	0.09	0.07	0.09
Max PM1 RP6	0.16	0.06	0.36	0.10	0.02	0.06
Max C RP3	0.15	0.05	0.15	0.08	-0.08	0.05
Max C RP6	0.06	0.05	0.06	0.09	-0.02	0.05
Md PM1 RP3	0.12	0.04	0.19	0.07	0.12	0.05
Md PM1 RP6	0.17	0.06	0.30	0.07	0.07	0.04

B2.2.1 Assumptions for repeated measures mixed ANOVA

A summary of the tests for assumptions can be found in Table B1.4. There is homogeneity of variances, as assessed by Levene's test for equality of variances ($p > 0.05$). Sphericity was tested using Mauchly's test for ΔBBW_{T1-T2} where $\chi 2(5) = 6.203$, $p = 0.287$. The ΔBBW_{T1-T2} for each TRP were normally distributed as assessed by a Shapiro-Wilk's test ($p > 0.05$) with the exception of the maxillary first premolars at RP6, maxillary canines at RP3 and the mandibular first premolars at both root points. Visual inspection of histograms reveals mostly symmetrical distribution with the large number of measurements at $\Delta BBW_{T1-T2} = 0$ and mild left skewedness of maxillary first premolars at RP6 and maxillary canines at RP3. An inspection of the boxplots showed non-normal distributions for the maxillary canines at RP3 and the mandibular premolars at both root points, indicating that the normality assumption is overly violated. Outliers were maintained in the analysis due to the use of human data and natural variability in the anatomy (Figure B1.2a).

Table B1.4 Normality and equal variance assumptions by time point (CBCT Analysis).

	ΔBBW_{T1-T2} N=90		ΔBBW_{T1-T3} N=38		ΔBBW_{T2-T3} N=38	
	Shapiro Wilk's	Levene's Test	Shapiro Wilk's	Levene's Test	Shapiro Wilk's	Levene's Test
	p-value		p-value		p-value	
Max M1 RP3	*0.43*	*0.50*	0.02	*0.75*	0.02	*0.41*
Max M1 RP6	*0.19*	*0.45*	*0.21*	*0.38*	*0.06*	*0.76*
Max PM1 RP3	*0.50*	*0.39*	*0.33*	*0.04*	0.01	*0.88*
Max PM1 RP6	0.04	*0.48*	0.03	*0.57*	0.01	*0.75*
Max C RP3	0.00	*0.11*	0.01	*0.27*	0.00	0.03
Max C RP6	*0.14*	*0.75*	*0.20*	*0.21*	0.01	*0.36*
Md PM1 RP3	0.00	*0.15*	0.01	*0.09*	0.00	*0.59*
Md PM1 RP6	0.01	*0.27*	0.00	*0.33*	0.00	*0.30*

For ΔBBW_{T1-T3}, there was homogeneity of variances as assessed by Levene's test (p > .05) with the exception of maxillary first premolars at RP3 (p=0.038); however, Mauchly's test of sphericity indicated that the assumption of sphericity was met , $\chi 2(5) = 7.91$, p = .161. The normality assumption was assessed by a Shapiro-Wilk's test (p > .05) and the following groups violated normality: the maxillary molars at RP3 (p=.018), maxillary first premolars at RP6 (p=.027), maxillary canines at RP3 (p=.012), and the mandibular first premolars at RP3 and RP6 (p=.010 and p=.001, respectively). A visual inspection of histograms reveals mostly symmetrical distribution with the majority of measurements at $\Delta BBW_{T1-T3} = 0$ and mild left skewedness of maxillary first premolars at RP6, mild right skewedness of the maxillary canines at RP6 and mandibular premolars at RP6. An inspection of the boxplots showed non normal distributions for the maxillary canines at RP3 and the mandibular premolars at both root points (Figure B1.2b). Outliers were maintained in the analysis due to natural variability in patient's anatomy.

Finally, for ΔBBW_{T2-T3}, the normality assumption was found to be violated when assessed by a Shapiro-Wilk's test (p < .05), with the exception of the maxillary molar group at RP6 (p=.064). That said, visual inspection of histograms reveals mostly symmetrical distribution with the majority of measurements at $\Delta BBW_{T2-T3} = 0$ and mild left skewedness of mandibular first premolars at both root points, mild right skewedness of the maxillary first premolars at RP6 maxillary canines at RP3. An inspection of the boxplots showed non-normal distributions for all of the TRP groups (Figures B1.2c). There was homogeneity of variances as assessed by Levene's test ($p > .05$) with the exception of the maxillary canines at RP3 ($p=0.028$); however, sphericity was met as per Mauchly's test, where $\chi2(5) = 7.91$, $p = .161$.

A mixed ANOVA was used to determine mean differences across tooth, root point and treatment type for ΔBBW_{T1-T2}. Although violations of normality were observed, especially in the maxillary canines and mandibular first premolars, ANOVA tests are somewhat robust against departures from normality provided that the sample sizes are large (n>30) and approximately equal. Since the data satisfied the assumption of sphericity and there were 45 patients (90 teeth), no further transformation was performed and the authors proceeded with a repeated measures mixed ANOVA to test the hypotheses for ΔBBW_{T1-T2}.
Despite the transformation, ΔBBW_{T1-T3} and ΔBBW_{T2-T3} violated the assumptions of ANOVAs (i.e. equal variance, normality and minimum sample size). A Friedman’s test was used to compare the distribution of BBW at baseline, debond and follow up, for 19 patients (38 teeth). Similarly, a Kruskal Wallice H test (KW) was used to compare the distribution of BBW between treatment groups at baseline, debond and follow up, for each TRP.

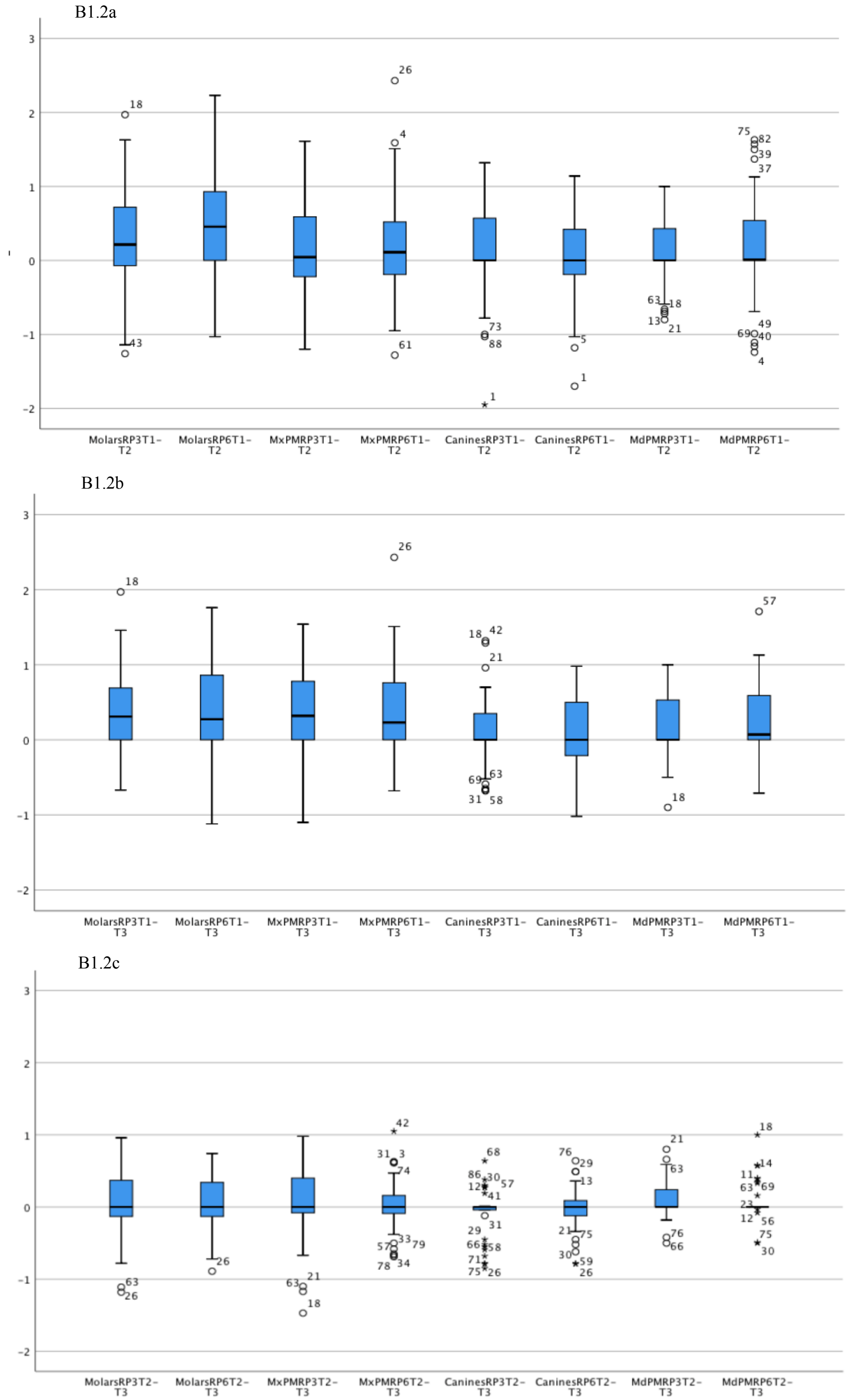


Figure B1.2a-c Boxplots of the change in buccal bone width, between baseline and after treatment, baselines at follow up and after treatment and at follow up.

B3. Response: Change in Bone Width

B3.1 ΔBBW_{T1-T2}

A mixed ANOVA was run to determine the effect of different treatments over time on buccal alveolar bone width (BBW). Data are mean (±SD) unless otherwise stated. There was a statistically significant two-way interaction between tooth and root point $F(3, 264) = .31$, $p = .013$, partial $\eta^2 = .04$. Therefore, simple main effects were analysed using mixed ANOVA for each significant interaction parameter (i.e. root point and tooth)

Independent of the expansion appliance, there was evidence to suggest that the maxillary first molars experienced a significant reduction in BBW at RP6 in response to orthodontic expansion treatment (p=.016). This was an average reduction in width of 0.22mm (±.09) [95% CI, .04 to .40] compared to RP3. Similarly, at RP6, the maxillary molars demonstrated a significant reduction in buccal alveolar width in response to orthodontic expansion, 0.35mm (±0.09) [95%CI, .10 to .61], .462mm (±.083) [95%CI, .24 to .69] and .34mm (±.088) [95% CI, .10 to .58] more than the maxillary first premolars, canines and mandibular premolars, respectively, at the same root point. Therefore, at a 5% significance level, the data provides sufficient evidence that there is a decrease in mean BBW measured at two root points on four teeth before and after treatment, regardless of appliance type. A summary of the results, independent of appliance type can be found in Table B1.5.

Table B1.5 Results in ΔBBW_{T1-T2} by tooth

Tooth$_a$	*Tooth$_b$*	*Mean Difference (a-b) (mm)*	*Std. Error*	*p-value*	95% Confidence Interval for Difference (Bonferroni adjustment) *Lower Bound*	*Upper Bound*
Mx M1	Mx PM1	.23	.07	.01	.04	.41
	Mx C	.30	.07	.00	.12	.48
	Md PM1	.26	.07	.00	.09	.44
Mx PM1	Mx M1	-.23	.07	.01	-.41	-.04
	Mx C	.07	.07	1.00	-.11	.25
	Md PM1	.04	.06	1.00	-.14	.21
Mx C	Mx M1	-.30	.07	.00	-.48	-.12
	Mx PM1	-.07	.07	1.00	-.25	.11
	Md PM1	-.04	.05	1.00	-.18	.11
Md PM1	Mx M1	-.26	.07	.00	-.44	-.09
	Mx PM1	-.04	.06	1.00	-.21	.14
	Mx C	.04	.05	1.00	-.11	.18

With respect to the differences between treatment groups, there is evidence to suggest that the mean change in alveolar bone thickness did differ by appliance type (p=.014), where the Hyrax group demonstrated .12mm ($\pm$.05) [95% CI, .03 to .22] more buccal alveolar loss than the Damon® group, overall. At the 5% significance level, the data provides sufficient evidence that there is a difference between Damon® or Hyrax appliances. That said, the change was only observed on the maxillary canines at RP3 (p=.032) where the distribution of ΔBBW between treatment groups is different (Chapter 3, Figure 3.1). For maxillary canines at RP3, those treated with the Hyrax appliance demonstrated a significant mean reduction in buccal alveolar bone width of .23mm ($\pm$.11) [95% CI, .02 to .45] more than those treated with Damon® appliances. A summary of the main effects of treatment is listed in table B1.6

Table B1.6 Treatment effects of ΔBBW_{T1-T2} by site.

Tooth	**Root Point**	**Mean Difference** (Hyrax – Damon) *(mm)*	**Std. Error**	**p-value**	**95% Confidence Interval for Difference**	
					Lower Bound	*Upper Bound*
Max M1	RP3	.16	.14	.26	-.12	.45
	RP6	.06	.14	.69	-.23	.34
Max PM1	RP3	.15	.13	.25	-.11	.41
	RP6	.21	.13	.11	-.05	.46
Max C	RP3	**.23**	**.11**	**.03**	**.02**	**.45**
	RP6	-.03	.11	.76	-.25	.18
Md PM1	RP3	.04	.08	.65	-.13	.20
	RP6	.17	.12	.16	-.07	.40

B3.2 $\Delta BBW_{T1\ vs\ T2\ vs\ T3}$ *– Baseline vs. Debond vs. Follow up*

A Friedman's two-way analysis of variance test was used to assess the ΔBBW distrtibution for each TRP at baseline, debond and follow up. Pairwise comparisons were run using SPSS version 27. Statistical significance was accepted at the $p < .002$ level correcting for an inflated alpha for post hoc analysis. Descriptive statistics and pairwise results are summarized in Table B1.7. Descriptive statistics and pairwise comparisons of BBW distributions by TRP over time can be found in Figure B1.3.

The distributions differed by TRP and was statistically significantly different during the course of treatment and follow up, $\chi^2(23) = 187.80$, $p < .0005$. Post hoc analysis revealed statistically significant differences in BBW distribution for maxillary first molars at RP3 and RP6, and maxillary first premolars at RP3. For maxillary molars at RP3, there was a statistically significant difference in BBW distribution from pre- (*Mdn* = 1.24) to post-treatment (*Mdn* = .80) (*p* =.001) and pre-treatment to follow up (*Mdn* = .69) (*p* < .0005), but not post-treatment and follow up.

Table B1.7 Descriptive statistics and p-values for pairwise comparisons for BBW (mm) by time point.

			Baseline (T1)				**Debond (T2)**				**Follow Up (T3)**				**P-Value <.002** *(PW comparisons)*		
Arch	**Tooth**	**Root Point**	*Median*	*IQR*	*Min*	*Max*	*Median*	*IQR*	*Min*	*Max*	*Median*	*IQR*	*Min*	*Max*	*T1 / T2*	*T1 / T3*	*T2 / T3*
Maxilla (n=19)	**First Molars** (n=38)	*RP3*	1.24	1.47	.00	1.97	.80	1.13	.00	1.50	.69	1.06	.00	1.57	***.001***	***<.0005***	.646
		RP6	.95	.98	.00	3.27	.58	.96	.00	3.09	.52	.81	.00	3.50	.022	***.001***	.276
	First Premolars (n=38)	*RP3*	.98	.73	.00	1.65	.61	.95	.00	1.82	.87	.87	.00	1.74	.022	***.000***	.207
		RP6	.83	.65	.00	2.43	.48	.66	.00	1.05	.51	.65	.00	1.35	.012	*.002*	.528
	Canines (n=38)	*RP3*	.45	.88	.00	1.32	.00	.49	.00	1.22	.00	.66	.00	.94	.088		
		RP6	.66	.92	.00	1.26	.58	.76	.00	1.34	.58	.78	.00	1.06	.796		
Mandible (n=19)	**First Premolars** (n=38)	*RP3*	.45	.61	.00	1.00	.00	.58	.00	1.18	.00	.10	.00	.95	.047		
		RP6	.41	.67	.00	1.71	.00	.39	.00	1.11	.00	.00	.00	.72	.034	.004	.456

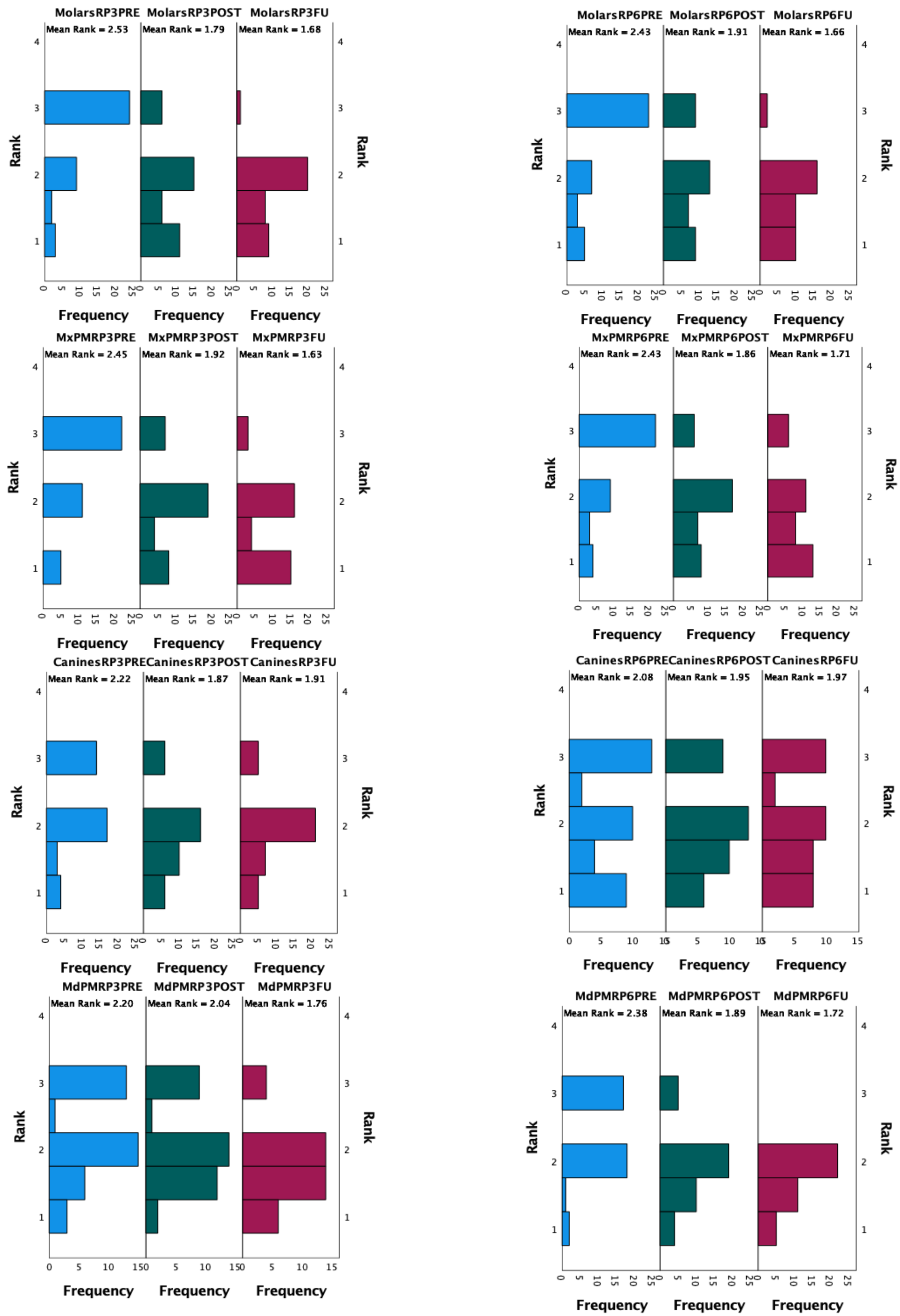


Figure B1.3 Related Samples Friedman's Two-Way Analysis of Variance by Ranks for each TRP, at baseline (PRE), debond (POST) and follow up (FU) for maxillary first molars and premolars.

At RP6, there was a statistically significant difference in BBW distribution from baseline (*Mdn = .95*) to follow up (*Mdn* = .52) (P=.001); however, the distribution was not significantly different between the other time points. Similarly, maxillary premolars at RP3 also showed a statistically significant decrease in BBW distribution from baseline (*Mdn* = .98) to follow up (*Mdn* = .61) (P<.0005) but not between these and the debond time point. While there was no significant difference in BBW for any time point for maxillary first premolars at RP6, it should be noted that the p-value (p=.002) was exactly equal to the significance level, which is the cut off for the accepted significance. There was no significant difference in BBW distribution for neither maxillary canines nor mandibular first premolars at either root point. Therefore, at the 0.2% significance level, there is sufficient evidence to suggest that there is a difference in BBW distribution measured at two root points on four teeth before and after treatment, regardless of appliance type. That said, there was no significant difference observed between BBW distributions at debond and at follow up.

A KW test was used to evaluate the BBW at each TRP between Hyrax (n=11) and Damon® (n=9) treatment groups. Distributions of BBW measurements were not similar for all groups, as assessed by visual inspection of the box plots (Figures B1.4, B1.5, B1.6 & B1.7).

BBW distributions were statistically significantly different between the Hyrax and Damon® groups. Pairwise comparisons were performed using a Bonferroni correction for multiple comparisons. This post hoc analysis revealed statistically significant differences in BBW distributions between Hyrax and Damon® treatment groups for the maxillary first molars and canines at RP3.

At baseline, there was a statistically significant difference in BBW between treatment groups for maxillary canines at RP3 ($\chi^2(1) = 3.99$, p=.046). The distributions of BBW were not similar as assessed by visual inspection of the boxplots, where the Hyrax group had a median BBW of .49mm more than the Damon® group (*Mdn*= .00mm). The BBW for the maxillary canines at RP3 did not have a significant difference at debond (p=.667) or follow up (p=.871) between treatment groups.

While the BBW for maxillary first molars at RP3 did not have a difference in the distributions between treatment groups at baseline (p=.269), there was a statistically significant difference at debond ($\chi^2(1) = 6.56$, p=.01). The distributions of the BBW for the maxillary first molars at RP3 were similar as assessed by visual inspection of the boxplots, where the Damon® group (*Mdn*=.81mm) had a median BBW of .015mm more than the Hyrax group (*Mdn*=.80mm). That said, there was no difference in BBW distributions between Hyrax and Damon® at follow up (p=.83). Therefore, at the 5% significance level, there is there is sufficient evidence to suggest a difference in the distribution of BBW measured at two root points on four teeth before and after treatment with either Damon® or Hyrax appliances; however, this was not the case at follow up. Findings are summarized in Table B1.8.

Table B1.8 Descriptive statistics and p-values for pairwise comparisons for BBW (mm) by treatment and time point.

				Baseline (T1)					**Debond (T2)**					**Follow Up (T3)**				
Arch	**Tooth**	**Root Point**	*Treatment*	*Median*	*IQR*	*Min*	*Max*	*p-value (<.05)*	*Median*	*IQR*	*Min*	*Max*	*p-value (<.05)*	*Median*	*IQR*	*Min*	*Max*	*p-value (<.05)*
Max (n=19)	**M1** (n=38)	*RP3*	Damon®	1.13	1.30	.00	1.97	**.269**	.81	1.15	.00	1.19	**.010**	.69	1.11	.00	1.57	**.830**
			Hyrax	1.33	1.47	.00	1.83		.80	1.03	.00	1.50		.67	.1.06	.00	1.55	
		RP6	Damon®	.88	1.40	.00	3.27	.476	.77	1.08	.00	3.09	.057	.57	1.13	.00	3.50	.275
			Hyrax	1.01	.95	.00	2.04		.53	.74	.00	1.27		.49	.68	.00	1.28	
	PM1 (n=38)	*RP3*	Damon®	.82	.68	.00	1.63	.611	.73	1.82	.00	1.82	.188	.64	1.00	.00	1.74	.160
			Hyrax	1.14	1.27	.00	1.65		.55	.80	.00	1.15		.00	.68	.00	1.23	
		RP6	Damon®	.75	1.10	.00	1.52	.147	.55	.62	.00	1.02	.725	.49	.58	.00	1.11	.384
			Hyrax	.85	.34	.00	2.43		.47	.85	.00	1.05		..52	.68	.00	1.35	
	Canines (n=38)	*RP3*	Damon®	.45	.78	.00	1.29	**.046**	.00	.66	.00	1.22	**.667**	.00	.67	.00	.94	**.871**
			Hyrax	.29	.94	.00	1.32		.00	.00	.00	.87		.23	.60	.00	.85	
		RP6	Damon®	.65	.36	.00	1.14	.954	.53	.70	.00	1.34	.694	.47	.83	.00	1.06	.451
			Hyrax	.70	.99	.00	1.26		.60	.81	.00	1.03		.64	.78	.00	1.02	
Mand (n=19)	**PM1** (n=38)	*RP3*	Damon®	.00	.56	.00	.81	.075	.00	.62	.00	.96	.099	.00	.48	.00	.90	.321
			Hyrax	.48	.67	.00	1.0		.00	.58	.00	1.18		.00	.00	.00	.95	
		RP6	Damon®	.00	.59	.00	.71	.332	.00	.56	.00	1.11	.442	.00	.00	.00	.72	.885
			Hyrax	.48	.89	.00	1.71		.00	.13	.00	.76		.00	.11	.00	.60	

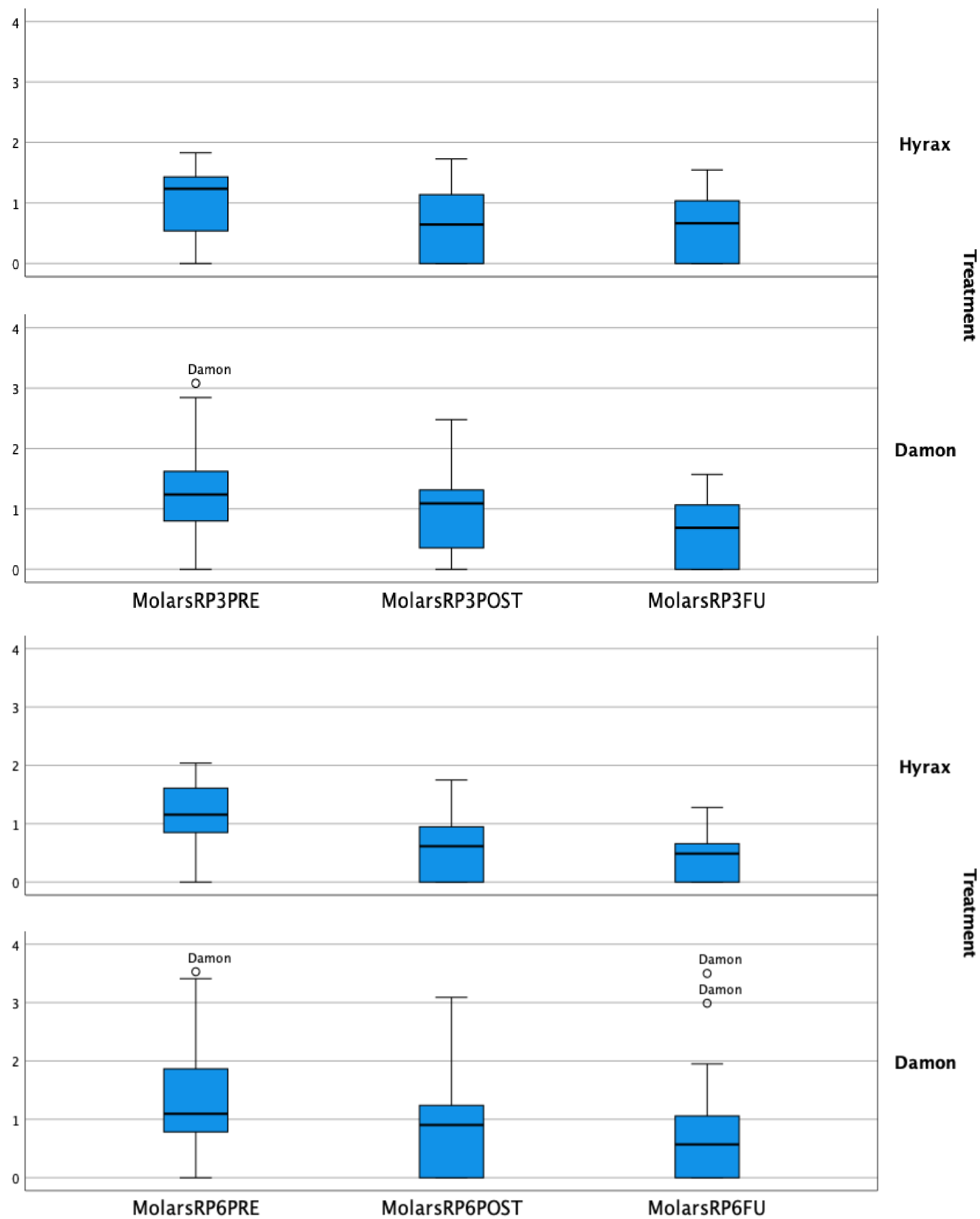


Figure B1.4 Boxplots of the BBW over the maxillary first molars at baseline (PRE), debond (POST), and follow up (FU) by treatment group (Damon® and Hyrax).

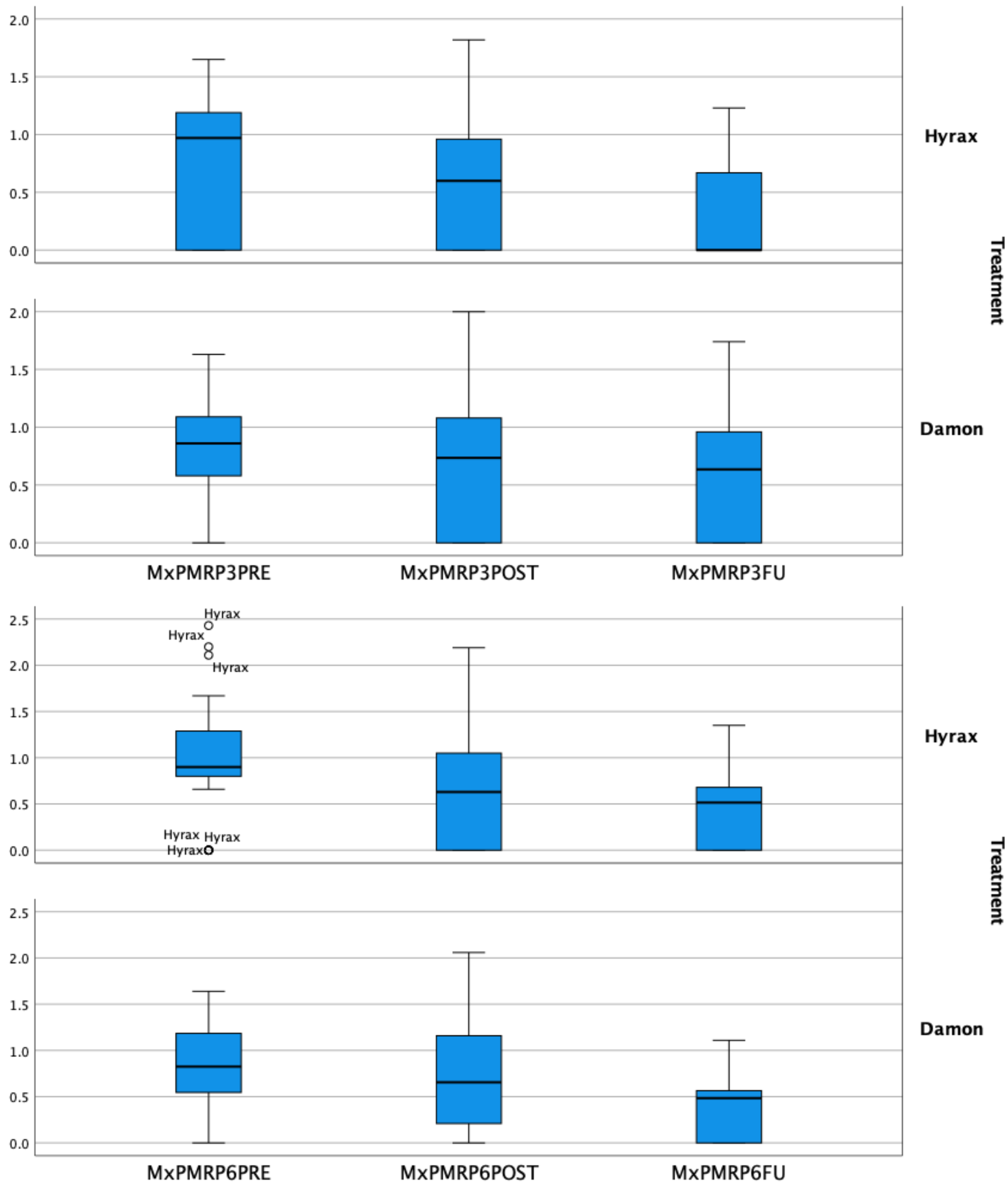
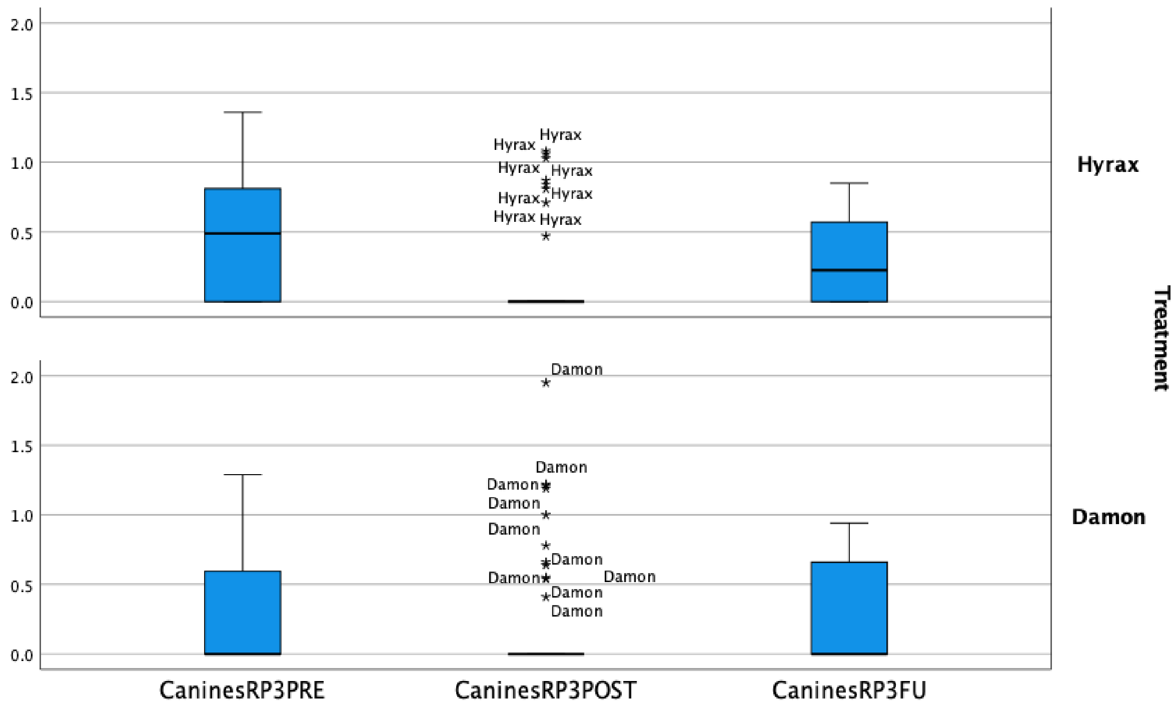


Figure B1.5 Boxplots of the BBW over the maxillary first premolars at baseline (PRE), debond (POST), and follow up (FU) by treatment group (Damon® and Hyrax.



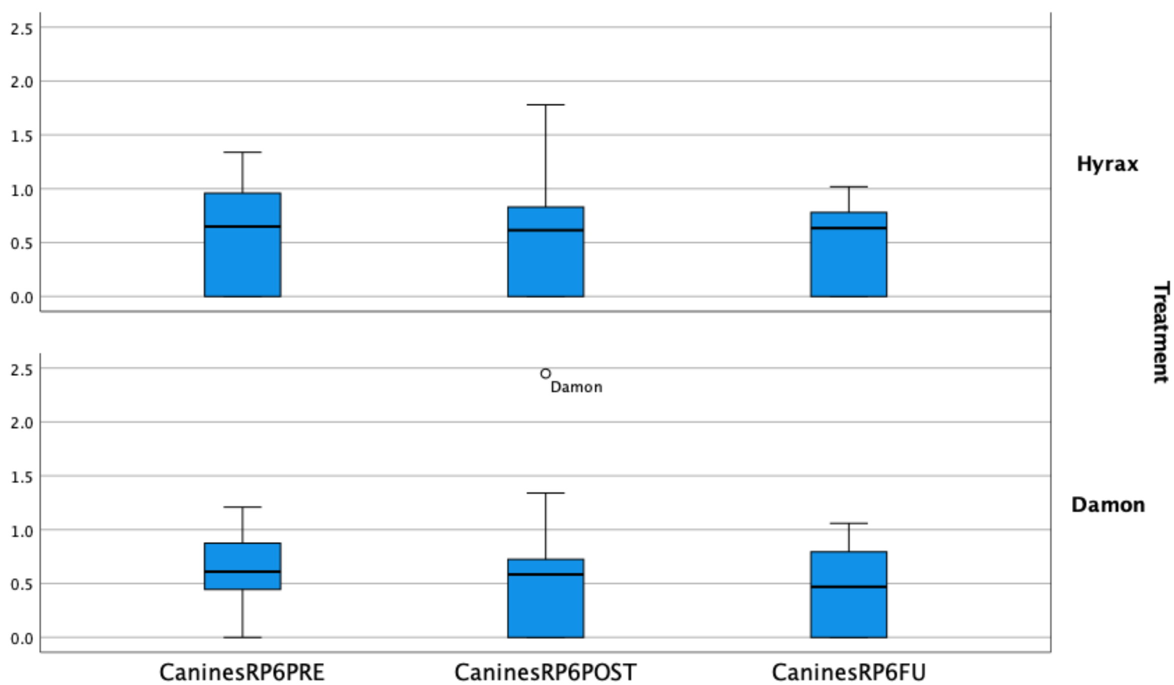


Figure B1.6 Boxplots of the BBW over the maxillary first canines at baseline (PRE), debond (POST), and follow up (FU) by treatment group (Damon® and Hyrax).

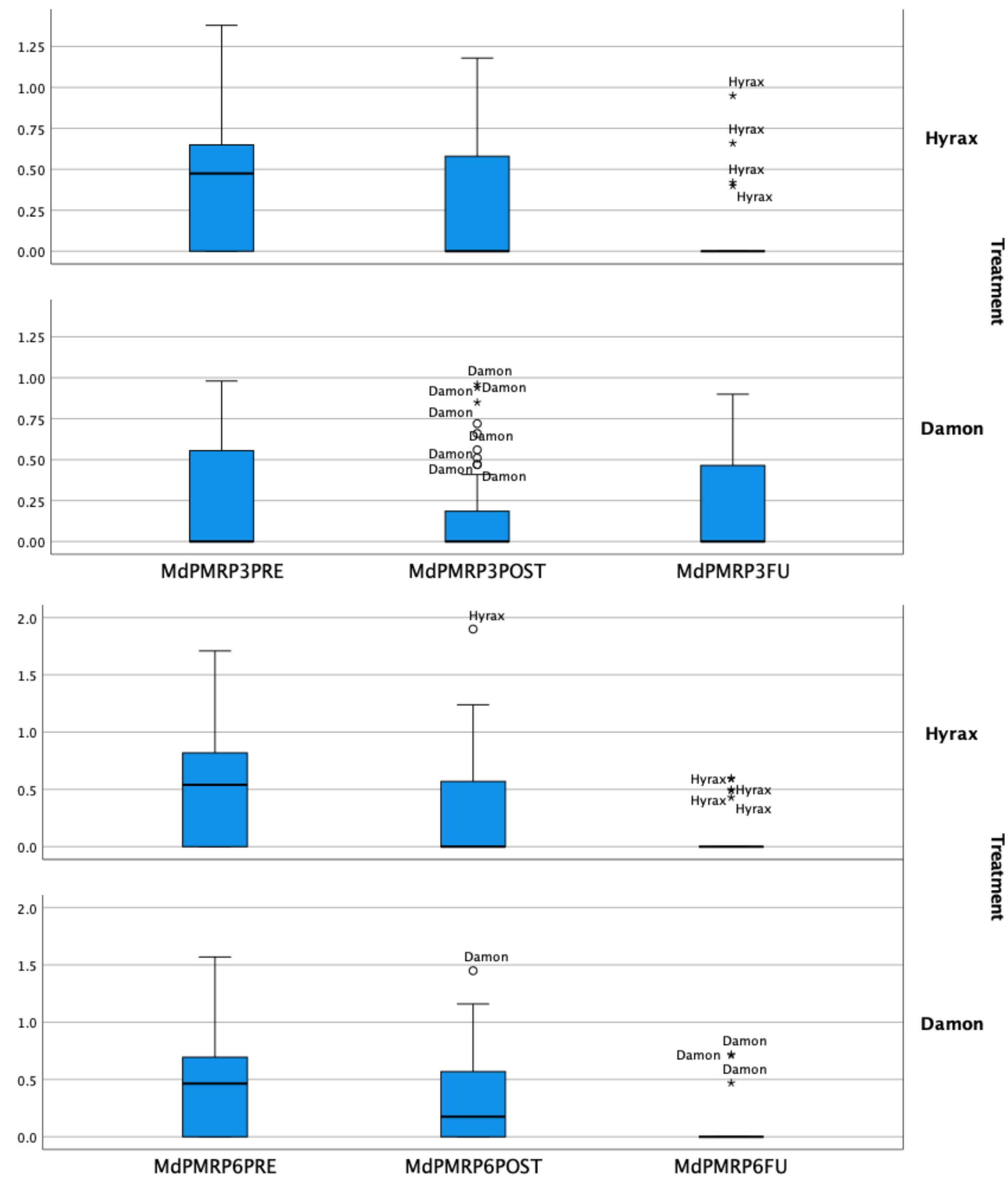


Figure B1.7 Boxplots of the BBW over the mandibular first premolars at baseline (PRE), debond (POST), and follow up (FU) by treatment group (Damon® and Hyrax).

B4. Hypothesis Testing for Recession Risk Factors

To investigate the risk factors and combination of risk factors associated with recession defects before, after and two-years post- expansion treatment, the following hypotheses were tested:

H_0: $\beta_1 = 0$, $\beta_2 = 0$, … $\beta_{13} = 0$ (the slope/coefficients of all independent variables are equal to zero)

H_a: $\beta_x \neq 0$, where x = 1, 2, …, 13 (at least one slope/coefficient is ***not*** equal to zero)

B5. Baseline Comparisons

Overall, there were approximately 1.5 times more females enrolled than males in the sample. The average age at the beginning of the study was 14.13 ± 0.26 years. For descriptive statistics of the collected data, refer to Chapter 3, Table 3.1. Unless otherwise stated, the significance level for all tests were set to $\alpha = 0.05$.

B5.1 Transformed Data

A binomial logistic regression was conducted to determine the relationship of BBW on the likelihood that participants have gingival recession at debond or two year follow up.

Since soft-tissue scores were assigned to a case, and continuous BBW measurements are assigned to tooth root points, the raw required transformation. $BBW_{T1,\ T2,\ T3}$ measurements were re-coded into categorical data ($BBW'_{T1,T2,T3}$), as follows:

- 0 – RP3 & RP6 > 0mm
- 1 – RP3 = 0mm; RP6 > 0mm
- 2 – RP3 & RP6 = 0mm

Should buccal alveolar bone be present at RP3 and RP6 for all tooth-root points, the patient should be protected for recession formation and a score of *zero* was assigned. If BBW was zero on one tooth at the more coronal root point (i.e. RP3), a score of *one* was assigned to the participant. Similarly, if BBW was zero at both RP3 and RP6 on one tooth, then a score of two was assigned to the participant.

B5.2 Assumptions of Linearity

Linearity of the continuous variables with respect to the logit of the dependent variable was assessed via the Box-Tidwell (1962) procedure. A Bonferrroni correction was applied using all 13 terms in the model, resulting in a significance being accepted when p<.004. Based on this assessment, the age of the patient at baseline (i.e. the continuous independent variable) was found to be linearly related to recession the logit of recession (i.e. the dependent variable) (p=.044). Moreover, gender (p=.820) and treatment type (p=.370) were not related to recession outcomes. There was one standardized residual with a value of -2.67 standard deviations, which was kept in the analysis.

B6. Results

The frequencies of observations are summarized in Table B1.9.

B1.4 Boxplots of the BBW over the maxillary first molars at baseline (PRE), debond (POST), and follow up (FU) by treatment group (Damon® and Hyrax).

B 1.5 Boxplots of the BBW over the maxillary first premolars at baseline (PRE), debond (POST), and follow up (FU) by treatment group (Damon® and Hyrax).

B 1.6 Boxplots of the BBW over the maxillary first canines at baseline (PRE), debond (POST), and follow up (FU) by treatment group (Damon® and Hyrax).

B 1.7 Boxplots of the BBW over the mandibular first premolars at baseline (PRE), debond (POST), and follow up (FU) by treatment group (Damon® and Hyrax).

Table B1.9 Frequency of variables.

Variables	**Description**	**Frequency**		
		T1vs.T2 (n=45)	*T1vs.T3 (n=19)*	*T2vs.T3 (n=19)*
Recession	≥CEJ	13	4	4
	Apical to CEJ	32	15	15
Risk Factors				
Age	Mean	16.36	18.25	
Gender	Male	17	6	
	Female	28	13	
Treatment	Hyrax	21	11	
	Damon®	24	8	
KTt	Thick	21	12	12
	Thin	24	7	7
KTw	≥ 2mm	28	11	11
	<2mm	17	8	8
Plaque	Absent	11	1	8
	Present	34	18	11
Inflammation	Absent	1	6	5
	Present	44	13	14
BT	Absent	24	10	10
	Present	21	9	9
BBW'(0)	RP3 & RP6>0	1	-	-
BBW'(1)	RP3=0;RP6>0	8	2	2
BBW(2)	RP3&RP6<0	36	17	17

B6.1 RF for Recession (T1 vs. T2)

A binomial logistic regression was performed to ascertain the effects of age at debond, gender, treatment type, KTt, KTw, plaque, inflammation, BTs and BBW' on the likelihood that participants have recession at debond. The logistic regression model was statistically significant according to the Hosmer and Lemeshow test, $\chi^2(7) = 3.88$, $p = .793$ The model explained 37.8% (Nagelkerke R^2) of the variance in recession and correctly classified 73.3% of cases. Sensitivity was 81.3%, specificity was 53.8%, positive predictive value (PPV) was 81.3% and negative predictive value (NPV) was 53.9%. Of the eight predictor variables only KTw was statistically significant (as shown in Table B1.10). KTw< 2mm had 18 times higher odds to exhibit recession than KTw≥ 2mm [95% CI, 1.28 to 254.66, p=.032]. When compared to KTw alone (Table B1.11), the model for recession was statistically significant, $\chi^2(1) = 8.26$, $p < 0.004$. The model explained 24% (Nagelkerke R^2) of the variance in recession and correctly classified 71.1% of cases. Sensitivity was 100%, specificity was 0%, PPV was 71.1% and NPV was 0%. KTw< 2mm had 12 times higher odds to exhibit recession than KT width ≥ 2mm [95% CI, 1.39 to 103.48, p=.024].

Table B1.10 Risk factors for recession between baseline and debond (T1 vs. T2).

	B	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**95% C.I.for EXP(B)**	
							Upper	*Lower*
KTt	.24	.92	.07	1	.794	1.27	.21	7.63
KTw	**2.89**	**1.35**	**4.60**	**1**	**.032**	**18.08**	**1.28**	**254.66**
Plaque	-1.06	1.06	1.00	1	.318	.35	.04	2.76
Inflammation	-17.51	40193.02	.00	1	1.000	.00	.00	.
BTs	.75	.87	.76	1	.383	2.13	.39	11.60
BBW'(0)			.30	2	.859			
BBW'(1)	-20.69	40193.02	.00	1	1.000	.00	.00	.
BBW'(2)	-21.31	40193.02	.00	1	1.000	.00	.00	.
Gender	-.20	.86	.05	1	.820	.82	.15	4.45
Treatment	-.88	.98	.80	1	.370	.41	.06	2.85
Age(T1)	.01	.24	.00	1	.958	1.01	.63	1.62
Constant	39.62	56841.54	.00	1	.999	1.61E+17		

Table B1.11 Risk factors of KTw <2 mm for recession between baseline and debond (T1 vs. T2).

	B	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**95% C.I.for EXP(B)**	
							Upper	*Lower*
KTw	**2.48**	**1.10**	**5.11**	**1**	**.024**	**12.00**	**1.39**	**103.48**
Constant	.29	.38	.57	1	.451	1.33		

B6.1.1 Recession T1 vs. T3

A binomial logistic regression was performed to ascertain the effects of age, gender, treatment type, KTt, KTw, plaque, inflammation, BT and BBW' on the likelihood that participants have recession at follow up. The logistic regression model was statistically significant, $\chi^2(6) = 16.78$, $p = .010$. The model explained 91.3% (Nagelkerke R^2) of the variance in recession and correctly classified 94.7% of cases. Sensitivity was 93.3%, specificity was 100%, PPV was 100% and NPV was 80%. None of the five predictor variables were statistically significant (as shown in Table B1.12).

B6.1.2 Recession T2 vs. T3

A binomial logistic regression was performed to ascertain the effects of KTt, KTw, plaque, inflammation, BTs and BBW' on the likelihood that participants have increased recession at follow up compared to at debond. The logistic regression model was not statistically significant, $\chi^2(6) = 16.78$, $p = .010$ The model explained 91.3% (Nagelkerke R^2) of the variance in recession and correctly classified 94.7% of cases. Sensitivity was 93.3%, specificity was 100%, PPV was 100% and NPV was 80%. None of the five predictor variables were statistically significant (as shown in Table B1.13).

Table B1.12 Risk factors for recession between baseline and follow up (T1 vs. T3).

	B	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**95% C.I.for EXP(B)**	
							Upper	*Lower*
KTt	56.20	16330.45	.00	1.00	1.00	2.54E+24	.00	.
KTw	-74.68	19321.50	.00	1.00	1.00	.00	.00	.
Plaque	21.20	40192.97	.00	1.00	1.00	1.62E+9	.00	.
Inflammation	18.84	8720.00	.00	1.00	1.00	1.52E+8	.00	.
BT	-18.74	8276.36	.00	1.00	1.00	.00	.00	.
BBW' (T3)	-8.06	7678403519.89	.00	1.00	1.00	.00	.00	.
Constant	37.22	13233.85	.00	1.00	1.00	1.46E+16		

Table B1.13 Risk factors for recession between debond and follow up (T2 vs. T3).

	B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I.for EXP(B)	
							Upper	*Lower*
KTt	57.66	18095.19	.00	1.00	1.00	1.10E+25	.00	.
KTw	-57.99	18260.08	.00	1.00	1.00	.00	.00	.
Plaque	19.48	13480.69	.00	1.00	1.00	2.90	.00	.
Inflammation	-.28	15090.14	.00	1.00	1.00	.75	.00	.
BT	-19.94	12686.92	.00	1.00	1.00	.00	.00	.
BBW' (T3)	-8.09	8.86E+9	.00	1.00	1.00	.00	.00	.
Constant	20.27	12605.95	.00	1.00	1.00	6.33E+8		

B6.1.3 Role of BBW' in Recession

The bone width after treatment was not a statistically significant predictor variable of recession before treatment compared to debond, before treatment compared to follow up nor between debond and follow up ($p=0.74$, $p=0.99$, $p=0.083$, respectively).

Appendix C: Clinical Trial

C1. Introduction

The clinical trial was conceived by Dr. Manuel Lagravere-Vich (MLV), in partnership with Dr. Paul Major (PM) and Dr. Hisham Badawi (HB). It involved adolescent patients between the ages of 11 and 17 years old with maxillary transverse deficiency (MTD) treated with either Damon® or Hyrax™ appliances.

C1.1 Initial Objectives

1. Develop and validate a measurement instrument applied to digital volumetric images to accurately determine changes in the craniofacial structures related to maxillary expansion treatments.
2. Evaluate skeletal (maxilla, nasal, and zygomatic) changes associated with tooth borne rapid maxillary expander
3. Evaluate dental (maxillary first molar, first premolar, cuspid and central incisor) changes (related to crown, root and angulations) associated with tooth borne rapid maxillary expander
4. Evaluate skeletal (maxilla, nasal, zygomatic) changes associated with Damon® system
5. Evaluate dental (maxillary first molar, first premolar, cuspid and central incisor) changes (related to crown, root and angulations) associated with Damon® system
6. Compare skeletal and dental changes between tooth borne Hyrax vs Damon® system groups

C1.2 Registration and Ethical Approval

This randomized clinical trial was completed at the Orthodontic Clinic in the University of Alberta (Alberta, Canada) with the ethics approval from the University of Alberta Research Ethics Board (Pro00013379). Radiation approval was obtained by the University of Alberta's Radiation Committee. Informed consent was obtained by each participant and no external funding was accepted.

C1.3 Sample Size

A minimum sample size of 36 patients per group (i.e. N=72) was calculated to be needed based on the previous research using a statistical power of 0.90 considering an α=0.5.[166] The final number registered in the trial was 95, which decreased to 90 due to unforeseen drop out and changes to the treatment plan (e.g. premolar extractions).

C1.4 Participants

Each patient had an orthodontic clinical examination to determine their eligibility, conducted by one of the clinical investigators (MLV, PM, HB). Inclusion and exclusion criteria are outlined in Table 2.1. Once the participants met the inclusion criteria and provided informed consent to treatment, a Cone Beam Computed Tomography (CBCT) digital volumetric image (iCAT, Imaging Science International, Hatfield, PA, USA) was taken prior to treatment (a large field of view 16 x 13.3 cm, voxel size 0.30 mm, 120 kVp, 18.54 mAS, and 8.9 seconds). Demographic characteristics of study subjects are outlined in Table 2.2.

C1.5 Randomization and Blinding

None of the investigators (MLV, PM, HB) were involved in the randomization process. Once the participants met the inclusion criteria and provided informed consent to treatment, a third party randomly allocated patients to one of the two treatment groups using Microsoft Excel (Microsoft, Redmond, WA). A block randomization method was used to ensure equal sample size in the two groups. Once the patient satisfied inclusion criteria and consent and assent forms were signed, designation to group was done by contacting person with randomized table to get the group following the randomized list.

C1.6 Intervention and Protocols

Patients in Group A received orthodontic treatment using the Damon® system (passive, self-ligating brackets). Initial alignment was done sequentially with Insignia prefabricated 0.014 NiTi, 0.016 NiTi, and 0.014x0.025 NiTi archwires in Damon® Arch Form. In addition, as per recommendation by the company's treatment consultant, buttons were bonded on palatal cusp surface of maxillary first molar and first premolar and bite ramps were placed on palatal cusps of maxillary first molars to facilitate the full-time use of crossbite elastics (3/16 inch, 2 ounce force) until 6 months into treatment.

Group B treatment consisted of maxillary expansion using the Hyrax appliance attached to the maxillary first premolars and first permanent molars. This treatment lasted approximately 16 days with a daily activation of the appliance with one turn of the screw, twice per day (0.25 mm per turn, 0.5 mm daily) until 6-10 mm of expansion (depending on the patient's need) was achieved. On the same day of the Hyrax insertion, non-self-ligating brackets (Insignia, Mini Diamond Ormco) were bonded from maxillary right canine to left canine and mandibular right to left first molars. The bonding was done following the Insignia (Ormco, Orange, California) protocol of indirect bonding set-up and placement. After completion of the active expansion treatment, the screw was tied with a ligature, and the Hyrax stayed passive for a five-month retention period prior to removal. The Hyrax appliance was removed and non-self-ligating bracket appliances (Insignia, Mini Diamond Ormco brackets) were placed to complete the treatment. Esthetic and occlusal objectives for all patients followed care standards that a reasonable clinician would achieve.

In the fixed banding stage of treatment, both groups used the Damon® system wires to the study's completion (approximately two years). Upon completing treatment, a CBCT and intra-oral photos were taken. Each patient was followed for two years while in retention at which point another CBCT and intraoral photos were taken.

C1.7 Study Settings

All interventions were completed at the University of Alberta's Graduate Orthodontic Clinic in the Kaye Edmonton Clinic. Data collection began in October 2011 and orthodontic treatment was completed for the last patient by June 2018. Presently, about one-fifth of the total number of patients are still within 2 years of debond (retention phase).

Chapter 3: Results

3.1 Introduction

The primary objective of this study was to determine the response of the buccal alveolar bone overlying the maxillary first molars, first premolars, canines and mandibular first premolars after orthodontic expansion treatment of maxillary transverse deficiency. The secondary objective was to determine if the type of orthodontic appliance (i.e. Damon® or Hyrax) affected the response of the buccal alveolar bone after expansion treatment. Next, the risk factors that are associated with mucogingival defects after orthodontic expansion were examined, including the presence of buccal alveolar bone. The third objective of this study was to find which risk factors were associated with the development of GR at debond and at two-year follow up. As such, this chapter will be separated into two parts: the first examining the change in buccal alveolar width with orthodontic expansion, and the second examining the risk factors associated with mucogingival defects.

3.1.1 Descriptive statistics

Overall, the mean age at baseline was 14.20 $\pm$ 1.75 years. The mean age at debond was 16.39 $\pm$ 1.70 years and 18.25 $\pm$ 1.93 years at follow up. There were approximately 1.5 times more females enrolled than males in the sample. For descriptive statistics of the collected data, refer to Table 3.1.

Table 3. 1 Sample sizes and ages (y) at pretreatment (T1), posttreatment (T2), and follow-up (T3) for the overall sample and subgroups.

			T1 *(n=45)*		***T2*** *(n=45)*		***T3*** *(n=19)*	
Group	*Subgroup*	*n*	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*
Overall	-	45	14.20	1.75	16.39	1.70	18.25	1.93
Sex	*Male*	17	14.08	1.59	16.43	1.54	17.55	1.78
	Female	28	14.27	1.87	16.36	1.78	18.58	1.98
Treatment	*Hyrax*	21	14.06	1.59	16.26	1.47	17.86	1.77
	Damon®	24	14.32	1.91	16.50	1.86	18.80	2.12

Part I: Results of CBCT Analysis

3.2 Introduction to CBCT analysis

CBCT offers an advantage over traditional imaging modalities because it allows for visualization of the buccal alveolus over each tooth, at their unique position in space. The method used to measure the alveolar bone for each individual tooth in 3-dimensions was presented in Chapter 2. A review of the literature revealed no study which examined the resultant width of buccal alveolar bone comparing specifically Damon® and Hyrax appliances with the exception of one only available in Chinese.[154] As such, a direct comparison of each treatment modality is valuable to the orthodontic profession in that it provides information with respect to alveolar remodeling both immediately after treatment and at two-years post treatment. A complete literature review can be found in section 1.5.

The goals of this retrospective study were to apply the method described by Digregorio et al.[160] to a clinical sample in order to determine the changes in alveolar width, buccal to the maxillary first molar, first premolars, canines and mandibular first premolars using Damon® and Hyrax expansion appliances.

3.3 Methods

Methods developed in Chapter 2 were applied to extract data from the pre- and post-treatment as well as follow-up CBCT records.

3.3.1 Collection of Data

For each participant who completed the study, the following parameters were collected for the right and left maxillary first permanent molars, first premolar, canines and mandibular first premolars, at root points three and six millimeters apical to the CEJ (i.e. RP3 and RP6):

1. Buccal Alveolar Bone Width (mm) before treatment (BBW_{T1})
2. Buccal Alveolar Bone Width (mm) at debond (BBW_{T2})
3. Buccal Alveolar Bone Width (mm) at follow-up (BBW_{T3})
4. Appliance Type
5. Gender
6. Age at T1

3.3.2 Calculation of Variables

The ΔBBW was calculated using the following general formula:

$$\Delta BBW \quad = BBW_{Ta} - BBW_{Tb}$$

Where,

- BBW is the buccal alveolar bone width at a given tooth-root-point
- Ta is the earlier time point (i.e. T1 or T2)
- Tb is the later time point (i.e. T2 or T3)

Refer to section 2.2.2.5 for the technique used to orient, define and measure BBW and section 2.4.1.3 for a complete breakdown of data modifications.

3.4 Hypothesis Testing

The decision to use a specific statistical test was dependent on the question to be answered and the satisfaction of the assumptions of the test. Moreover, the number of independent and dependent variables influenced the type of analysis chosen. The significance level for all tests were set to α = 0.05. Refer to Appendix B for complete data tables and specifics associated with data set up and hypothesis testing.

3.4.1 Assumptions for repeated measures mixed ANOVA

The distribution of the data for ΔBBW_{T1-T2}, was approximately normally distributed, relatively symmetric and it satisfied the assumptions of sphericity. No further transformation was performed and the authors proceeded with a repeated measures mixed ANOVA.

That said, ΔBBW_{T1-T3}, ΔBBW_{T2-T3} did not satisfy the assumptions for ANOVA (i.e. equal variance, normality and minimum sample size). A Friedman's test was used to compare the distribution of BBW at baseline, debond and follow up, for 19 patients (38 teeth). Similarly, a Kruskal Wallice H test (KW) was used to compare the distribution of BBW between treatment groups at baseline, debond and follow up, for each TRP.

3.5 Results

The results were organized into four sections for convenience. The first section examines the pre-treatment group differences. Sections two to four report differences in buccal alveolar bone width measured between each of the time points – Before and after treatment (ΔBBW_{T1-T2}), before treatment and at follow-up (ΔBBW_{T1-T3}) and post-treatment and at follow up (ΔBBW_{T2-T3}) – as the outcome measure. For descriptive statistics of the collected data, refer to Table 3.2.

Table 3.2 Descriptive statistics for baseline BBW* measurements (CBCT analysis).

	Damon® (N=24) Male=5 Female=19		**Hyrax (N=21)** Male=12 Female=9		**Total (N=45)** Male = 17 Female = 28	
	Mean (mm)	SD	Mean (mm)	SD	Mean (mm)	SD
Age (yrs @ T1)	*14.32*	*1.91*	*14.06*	*1.59*	*14.20*	*1.75*
Max M1 RP3	1.17	0.76	0.97	0.61	1.07	0.70
Max M1 RP6	1.38	0.92	1.11	0.63	1.25	0.81
Max PM1 RP3	0.80	0.48	0.80	0.55	0.80	0.51
Max PM1 RP6	0.81	0.52	0.98	0.60	0.89	0.56
Max C RP3	0.26	0.39	0.44	0.46	0.34	0.43
Max C RP6	0.59	0.37	0.56	0.45	0.58	0.41
Md PM1 RP3	0.24	0.32	0.39	0.38	0.31	0.13
Md PM1 RP6	0.44	0.45	0.55	0.50	0.49	0.48
Mean BBW	0.71	0.29	0.72	0.25	0.72	0.27

**Mean buccal bone width (BBW) for maxillary (max) first molars (M1), first premolars (PM1), canines (C), and mandibular (md) first premolars (PM1)*

3.5.1 Pre-Treatment Comparisons

Overall, there were approximately 1.5 times more females enrolled than males in the sample There were no differences between groups in terms of age or treatment for the overall mean BBW $F(3, 86) = .83$, $p = 0.48 > 0.05$. The average age at baseline was 14.20 ± 1.74 years.

3.5.2 General Comparisons

A summary of the raw data can be found in Appendix A, Table A1.6. Data are mean ($\pm$SD) unless otherwise stated. Overall, there was a decrease in mean BBW of 0.20mm after expansion treatment (T2) of with further decrease of 0.13mm by follow up (T3). While not statistically significant, the raw data demonstrate an apparent increase in mean BBW between debond and follow up for the maxillary canines at RP3 (0.11mm). Both the Hyrax and Damon® groups followed the same overall pattern.

At baseline, 24.72% of the sample teeth presented with a dehiscence 3mm apical to the CEJ. This increased to 30.83% at debond and remained relatively constant (28.95%) by follow up. The percentage of maxillary first molars with a dehiscence 3mm apical to the CEJ (i.e. $BBW_{RP3}=0$)

was similar to the first premolars before treatment (i.e. 14.44% and 17.78% respectively). Immediately after treatment this increased to 25.56% and 51.11 % of the molars and premolars, respectively. At follow up, however, the presence of a dehiscence 3mm apical to the CEJ increased marginally for the molars (26.32%), yet it decreased for the first premolars (34%). Almost half (40%) of the canines and 26.67% of the premolars presented with dehiscence at baseline. This increased to 51.11% for the canines and remained relatively constant for the premolars (22.22%). By follow up; however, both groups decreased with only 36.8% and 18.42% of the canines presenting with dehiscence, respectively.

Overall, there was a progression in the prevalence of dehiscence 6mm apical to the CEJ, where 13.89% of cases presented with 6mm dehiscence at baseline, which increased to 24.17% by debond and 27% at follow up. More severe dehiscence (i.e. $BBW_{RP3\&RP6}=0$), were present in only 6.67% the maxillary first molars and 5.56% of the first premolars at baseline. This increased to 11.11% and 12.22% by debond, and increased further to 15.79% for both groups by follow up. For the canines and mandibular premolars, severe dehiscences were identified in 17.78% and 25.56% of cases at baseline, respectively. This increased to 27.78% and 45.56% of cases at debond, respectively, and returned to approximately baseline-levels for the canines (18.42%) yet further increased for the mandibular first premolars (57.89%) by follow up.

Group differences were calculated using a Pearson Chi-square test or a Fisher's Exact test, when the sample size was small (i.e. T3). Between groups, those treated with either Hyrax or Damon® appliances had a similar overall prevalence of dehiscences at baseline (16.67% and 21.94%, respectively), with the exception of the maxillary canines at RP3 where 63.89% of canine dehiscences were found in the Damon® group (p=.031). At debond those treated with Hyrax or Damon® appliances exhibited dehiscences in 26.94% and 28.06% of the sample, respectively. Of the severe dehiscences found in the maxillary first molars at debond, 80% were in the Hyrax group (8/10) compared to two in the Damon® group (20%, p=.044). On the contrary, 17 patients in the Damon® group had dehiscences to 3mm apical to the CEJ in the mandibular first premolars (85%), compared to three in the Hyrax group (15%, p<.0001). At follow up, there appears to be marginally more dehiscence formation in the Hyrax group compared to the Damon® group 34.21% and 21.71%, respectively; however, the group differences were not significant (Table 3.3).

Table 3.3 Crosstabulation for dehiscence (3mm) and severe dehiscence (6mm) by Tooth at T1, T2, T3.

	Baseline (T1)								
	MxM1		MxPM1		MxC		MdPM1		
Treatment	3mm	6mm	3mm	6mm	3mm	6mm	3mm	6mm	Total
Hyrax	7	3	10	2	13	7	10	8	60
Damon®	6	3	6	3	23	9	14	15	79
Total Dehiscence	13	6	16	5	36	16	24	23	139
P-value	.764	1.00	.270	.645	**.031**	.581	.340	.091	
Total teeth	90		90		90		90		360
	Debond (T2)								
	MxM1		MxPM1		MxC		MdPM1		
Treatment	3mm	6mm	3mm	6mm	3mm	6mm	3mm	6mm	Total
Hyrax	12	8	10	8	22	12	3	22	97
Damon®	10	2	13	3	24	13	17	19	101
Total Dehiscence	22	10	23	11	46	25	20	41	198
P-value	.624	**.044**	.468	.108	.673	.634	**<.0001**	.525	
Total teeth	90		90		90		90		360
	Follow up (T3)								
	MxM1		MxPM1		MxC		MdPM1		
Treatment	3mm	6mm	3mm	6mm	3mm	6mm	3mm	6mm	Total
Hyrax	7	3	8	5	9	2	5	13	52
Damon®	3	3	5	1	5	5	2	9	33
Total Dehiscence	10	6	13	6	14	7	7	22	85
P-value	.143	1.00	.155	.075	.079	.428	.214	.059	
Total teeth	38		38		38		38		152

3.5.3 Response ΔBBW_{T1-T2}

A summary of results for the ΔBBW_{T1-T2} can be found in Table 3.4. Data are mean ($\pm$SD) unless otherwise stated. Independent of expansion appliance, the maxillary first molars experienced more reduction in BBW compared to the other tooth groups in response to orthodontic expansion (p=.016). This was more pronounced at RP6 than RP3 on maxillary molars, where RP6 demonstrated an average reduction in width of .22mm ($\pm$.09) [95% CI, .04 to .40] more than RP3. Similarly, at RP6, the maxillary molars demonstrated more reduction in buccal alveolar width in response to orthodontic expansion, compared to the maxillary first premolars, canines and mandibular premolars at the same root point (.36mm (±.09) [95% CI, .10 to .61], .46mm (±.08) [95%CI, .24 to .69] and .34mm (±.09) [95% CI, .10 to .58], respectively).

With respect to the differences between treatment groups, there is evidence to suggest that the mean change in alveolar bone thickness did differ by appliance type (p=.014), where the Hyrax group demonstrated .12mm (±.05) [95% CI, .03 to .22] more buccal alveolar bone loss than the Damon® group. The difference in means at had a large effect size (Cohen's d=1.25).

Table 3.4 Summary* of Results for ΔBBW_{T1-T2}

$Tooth_a$	$Tooth_b$	Mean Difference (a-b) (mm)	Std. Error	p-value	95% Confidence Interval for Difference (Bonferroni adjustment) Lower Bound (mm)	Upper Bound (mm)
Mx M1	Mx PM1	.23	.07	.01	.04	.41
	Mx C	.30	.07	.00	.12	.48
	Md PM1	.26	.07	.00	.09	.44
Mx PM1	Mx M1	-.23	.07	.01	-.41	-.04
	Mx C	.07	.07	1.00	-.11	.25
	Md PM1	.04	.06	1.00	-.14	.21
Mx C	Mx M1	-.30	.07	.00	-.48	-.12
	Mx PM1	-.07	.07	1.00	-.25	.11
	Md PM1	-.04	.05	1.00	-.18	.11
Md PM1	Mx M1	-.26	.07	.00	-.44	-.09
	Mx PM1	-.04	.06	1.00	-.21	.14
	Mx C	.04	.05	1.00	-.11	.18

*main effects of the teeth for T1-T2, irrespective of interaction

That said, this result should be interpreted with caution since the change was only observed on the maxillary canines at RP3 (p=.032) whereby those treated with the Hyrax appliance demonstrated .23mm (±.11) [95% CI, .02 to .45] more reduction in BBW than those treated with Damon® appliances. Visual inspection of the boxplots of maxillary canines at RP3 between appliance types (Figure 3.1) shows a number of outliers above and below a very narrow IQR for the Damon® group. It is also indicative of the presence of a *floor effect* with the majority of the ΔBBW measurements equal to zero. Evaluation of the raw data (see Appendix A, Tables A1.8 & A1.9) corroborates these findings as the difference in means at T2 was trivial (Cohen's d=.08).

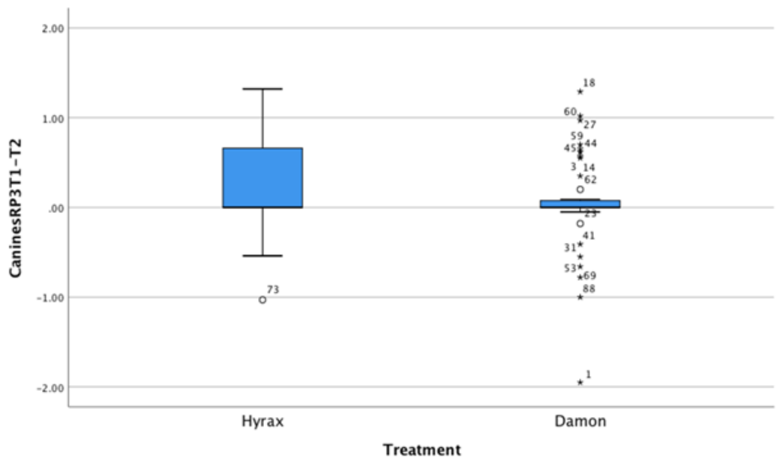


Figure 3.1 Box plot for ΔBBW maxillary canines at RP3 between T1 and T2 by treatment group.

3.5.4 $\Delta BBW_{T1\ vs\ T2\ vs\ T3}$ – Baseline vs. Debond vs. Follow up

A Friedman's two-way analysis of variance test was used to assess the BBW distribution for each TRP at baseline, debond and follow up. Statistical significance was accepted at the $p < .002$ level for post hoc analysis. Descriptive statistics and pairwise comparisons of BBW distributions by TRP over time are summarized in Table 3.5.

The BBW distributions differed by TRP and were significantly different during the course of treatment and follow up ($\chi^2(23) = 187.80$, $p < .0005$); specifically, the maxillary first molars at RP3 and RP6, and maxillary first premolars at RP3. For maxillary first molars at RP3, BBW distribution differed from pre- (*Mdn* = 1.24) to post-treatment (*Mdn* = .80) (p =.001) and pre-treatment to follow up (*Mdn* = .69) ($p < .0005$), but not post-treatment and follow up. At RP6, there was a significant difference in BBW distribution from baseline (*Mdn = .95*) to follow up (*Mdn* = .52) (P=.001); however, the distribution was not significantly different between the other time points. Similarly, the maxillary premolars at RP3 also demonstrated a significant decrease in BBW distribution from baseline (*Mdn* = .98) to follow up (*Mdn* = .61) (P<.0005) but not between these and debond. While there was no significant difference in BBW distribution for any time point for maxillary first premolars at RP6, it should be noted that the p-value (p=.002) was exactly equal to the significance level, which is the cut off for the accepted significance when baseline was compared to follow-up. The distribution of BBW for maxillary canines and mandibular first premolars at either root point was not different.

Table 3.5 Descriptive statistics and p-values for pairwise comparisons for BBW (mm) by time point.

Arch	Tooth	Root Point	Baseline (T1) Median	IQR	Min	Max	Debond (T2) Median	IQR	Min	Max	Follow Up (T3) Median	IQR	Min	Max	P-Value* (PW comparisons) T1 / T2	T1 / T3	T2 / T3
Maxilla (n=19)	**First Molars** (n=38)	*RP3*	1.24	1.47	.00	1.97	.80	1.13	.00	1.50	.69	1.06	.00	1.57	***.001***	***<.0005***	.646
		RP6	.95	.98	.00	3.27	.58	.96	.00	3.09	.52	.81	.00	3.50	.022	***.001***	.276
	First Premolars (n=38)	*RP3*	.98	.73	.00	1.65	.61	.95	.00	1.82	.87	.87	.00	1.74	.022	***.000***	.207
		RP6	.83	.65	.00	2.43	.48	.66	.00	1.05	.51	.65	.00	1.35	.012	*.002*	.528
	Canines (n=38)	*RP3*	.45	.88	.00	1.32	.00	.49	.00	1.22	.00	.66	.00	.94	.088		
		RP6	.66	.92	.00	1.26	.58	.76	.00	1.34	.58	.78	.00	1.06	.796		
Mandible (n=19)	**First Premolars** (n=38)	*RP3*	.45	.61	.00	1.00	.00	.58	.00	1.18	.00	.10	.00	.95	.047		
		RP6	.41	.67	.00	1.71	.00	.39	.00	1.11	.00	.00	.00	.72	.034	.004	.456

* significance set at p<.002

Table 3.6 Descriptive statistics and p-values for pairwise comparisons for BBW (mm) by treatment and time point.

Arch	Tooth	Root Point	*Treatment*	**Baseline (T1)*** *Median*	*IQR*	*Min*	*Max*	*p-value (<.05)*	**Debond (T2)*** *Median*	*IQR*	*Min*	*Max*	*p-value (<.05)*	**Follow Up (T3)$^{\varphi}$** *Median*	*IQR*	*Min*	*Max*	*p-value (<.05)*
Max	**M1**	*RP3*	Damon®	1.13	1.30	.00	1.97	**.269**	.81	1.15	.00	1.19	**.010**	.69	1.11	.00	1.57	**.830**
			Hyrax	1.33	1.47	.00	1.83		.80	1.03	.00	1.50		.67	.1.06	.00	1.55	
		RP6	Damon®	.88	1.40	.00	3.27	.476	.77	1.08	.00	3.09	.057	.57	1.13	.00	3.50	.275
			Hyrax	1.01	.95	.00	2.04		.53	.74	.00	1.27		.49	.68	.00	1.28	
	PM1	*RP3*	Damon®	.82	.68	.00	1.63	.611	.73	1.82	.00	1.82	.188	.64	1.00	.00	1.74	.160
			Hyrax	1.14	1.27	.00	1.65		.55	.80	.00	1.15		.00	.68	.00	1.23	
		RP6	Damon®	.75	1.10	.00	1.52	.147	.55	.62	.00	1.02	.725	.49	.58	.00	1.11	.384
			Hyrax	.85	.34	.00	2.43		.47	.85	.00	1.05		..52	.68	.00	1.35	
	Canines	*RP3*	Damon®	.45	.78	.00	1.29	**.046**	.00	.66	.00	1.22	**.667**	.00	.67	.00	.94	**.871**
			Hyrax	.29	.94	.00	1.32		.00	.00	.00	.87		.23	.60	.00	.85	
		RP6	Damon®	.65	.36	.00	1.14	.954	.53	.70	.00	1.34	.694	.47	.83	.00	1.06	.451
			Hyrax	.70	.99	.00	1.26		.60	.81	.00	1.03		.64	.78	.00	1.02	
Mand	**PM1**	*RP3*	Damon®	.00	.56	.00	.81	.075	.00	.62	.00	.96	.099	.00	.48	.00	.90	.321
			Hyrax	.48	.67	.00	1.0		.00	.58	.00	1.18		.00	.00	.00	.95	
		RP6	Damon®	.00	.59	.00	.71	.332	.00	.56	.00	1.11	.442	.00	.00	.00	.72	.885
			Hyrax	.48	.89	.00	1.71		.00	.13	.00	.76		.00	.11	.00	.60	

* n=45 subjects, 90/TRP
φ n= 19 subjects, 38/TRP

A KW test was used to evaluate the distribution of $BBWs$ at each TRP between Hyrax and Damon® treatment groups. Results are summarized in Table 3.6. BBW distributions were different between the Hyrax and Damon® groups between the maxillary first molars and canines at RP3 (Figure 3.2). The maxillary canines at RP3 differed in BBW distribution by expansion appliance at baseline ($\chi^2(1) = 3.99$, p=.046), but there was no difference at debond (p=.667) or follow up (p=.871). The distributions of BBW for the maxillary first molars at RP3 did not show a difference between expansion appliances at baseline (p=.269) or follow up (p=.830); however, there was a difference at debond ($\chi^2(1) = 6.56$, p=.010). The Damon® group (*Mdn*=.81mm) had a median BBW of .015mm more than the Hyrax group (*Mdn*=.80mm).

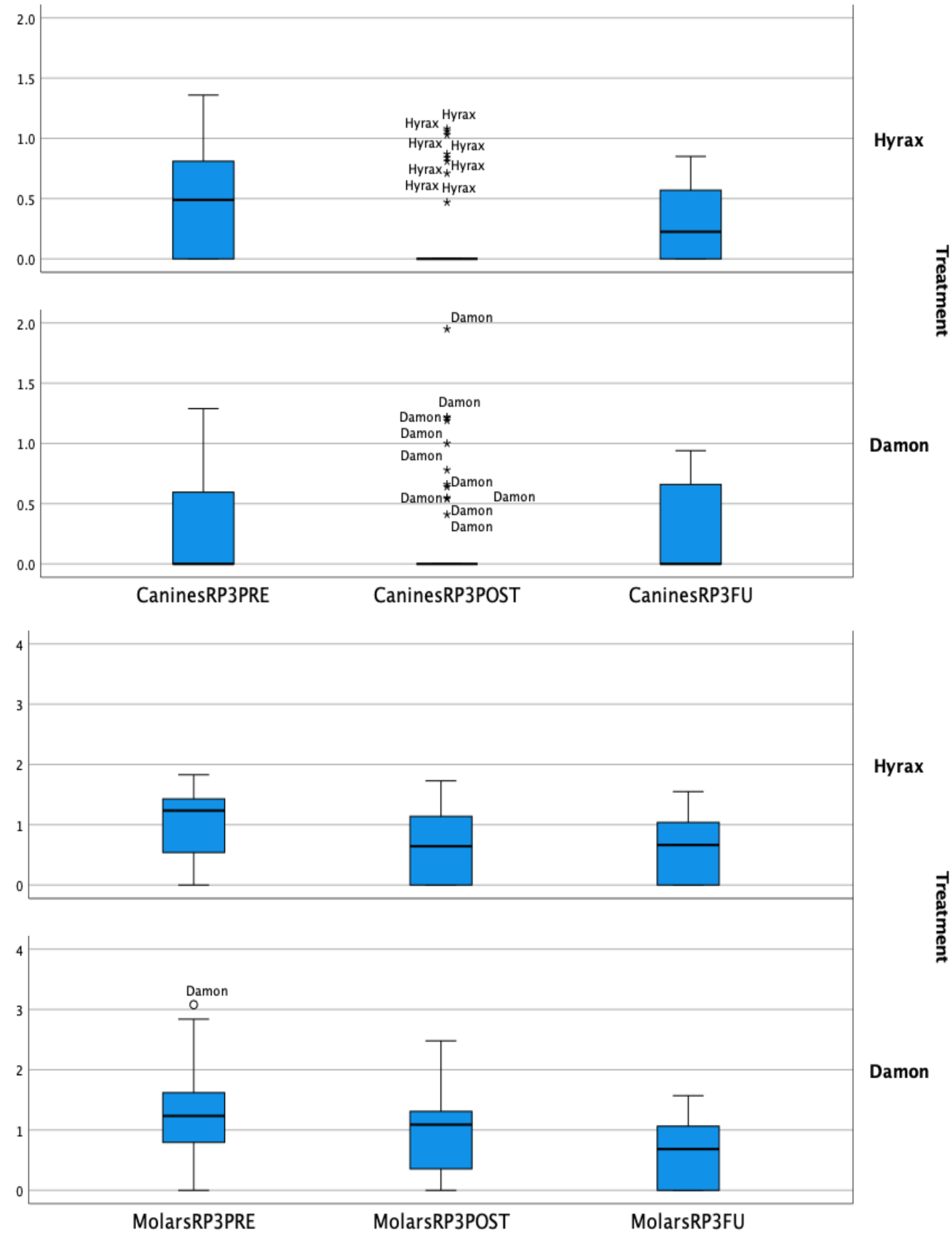


Figure 3.2 Box plots of the BBW over maxillary canines and first molars at Baseline (PRE, n=90), debond (POST, n=90), and follow up (FU, n=38) by treatment group (Damon® and Hyrax).

Part II: Results of Photo Analysis

3.6 Introduction to Photo-analysis

The discussion around orthodontic expansion and its relationship with gingival recession, has been controversial in the orthodontic literature.[14,27,28,64,83,84,87,110,131] Earlier studies have suggested an association between the two,[14] however this has been questioned by more recent studies.[131] Nevertheless, the most recent consensus statement on mucogingival conditions acknowledged that the direction of orthodontic movement is largely associated with the possibility of gingival recession initiation or the progression of recession during or after orthodontic treatment.[27,64,127] The same statement identified the possible risk factors (RFs) that can increase the probability of developing mucogingival defects, including plaque control, keratinized tissue height, keratinized tissue width / thickness and the presence of inflammation.[64] A review of the literature can be found in section 1.4.

The goal of this retrospective study was to apply the methods[139–141] validated by Le Roch et al.[142] to a clinical sample in order to determine the soft tissue changes over time in the presence of periodontal risk factors, following orthodontic expansion treatment,. The photos from each of the subjects were analyzed using the method presented in Chapter 2.

3.7 Methods

Methods developed in Chapter 2 were applied to extract data from the pre- and post-treatment as well as follow-up intraoral photos.

3.7.1 Collection of Data

For each patient that participated and completed the study, the following parameters were collected for the right and left maxillary first permanent molars, first premolar, canines and mandibular first premolars:

- Recession (GR)
- Keratinized Tissue Thickness / Root Prominence (KTt)
- Keratinized Tissue vertical width / height (KTw)
- Plaque
- Inflammation
- Black triangles / Blunted Tissue (after) - BT

A score of 0 or 1 point was used to denote the presence (1) or absence (0) of each factor (Appendix A, Table A1.9). If the RF presented on one site, a score of 1 was assigned to the entire case. Additionally parameters, such as gender, age at T1 and treatment group were included to assess the relationship between baseline patient factors and grouping with the presence of recession defects.

3.7.2 Calculation of Variables for BBW'

$BBW_{T2,\ T3}$ measurements were re-coded into categorical data ($BBW'_{T2,T3}$), as follows:

- 0 – RP3 > 0mm
- 1 – RP3 = 0mm; RP6 > 0mm
- 2 – RP3 & RP6 = 0mm

Table 3.2 shows descriptive statistics for collected data. Refer to section 2.4.2 and 2.4.3 for a complete breakdown of data organization and modifications.

3.8 Hypothesis Testing

Refer to Appendix B for specifics associated with data set up and hypothesis testing. The significance level for all tests were set to $\alpha = 0.05$.

3.8.1 Assumptions for Linear Regression

Linearity of the continuous variables with respect to the logit of the dependent variable was assessed via the Box-Tidwell (1962) procedure. Based on this assessment, the continuous independent variable (i.e. age) was found to be linearly related to the logit of the dependent variable (i.e. recession) (p=.044). There was one standardized residual with a value of -2.67 standard deviations, which was kept in the analysis.

3.9 Results

Refer to Appendix B4-6 for complete hypothesis testing and statistical analysis. GR was evaluated with the sexes combined because there was no statistically significant relationship between gender and the presence of GR in the present sample (p = .820). Similarly, no positive correlation was found between GR and age (p=.958), nor between GR and treatment type (p=.370). In fact, 53.33% of the sample was treated with Damon® appliances and at debond, and 70.83% of them showed evidence of GR. The Hyrax group showed similar evidence of recession at debond 71.43% (Table 3.7).

Table 3.7 Crosstabulation of GR by treatment at T2.

		GR		Total
Count		@CEJ	Apical to CEJ	
Treatment	Hyrax	6	15	21
	Damon®	7	17	24
Total		13	32	45

Overall, 71.11% of patients demonstrated GR on at least one of the study teeth after expansion treatment, and 78.95% exhibited GR at follow-up. Most of the patients presented with a thin tissue type (53.33%); however, tissue width ≥2mm was present on the study teeth for 62.22% of the sample. Plaque was present for 75.56% of the patients prior to bonding and 57.89% of the patients studied at debond. Similarly, 97.78% of patients displayed inflammation at debond; however, this reduced to 73.68% of patients displayed inflammation follow up. Black triangles or blunted tissue were present for 46.67% of patients at debond, which stayed relatively constant by follow up (47.37%). Finally, BBW' was absent on at least one of the study teeth at both RP3 and RP6 for 80% of patients at debond. This increased to 89.47% of patients at follow up. A complete summary of GR and the associated RFs can be found in Table 3.8.

Table 3.8 Percentages of teeth with increased GR at T1vsT2, T1vsT3 and T2vsT3.

		T1vs.T2 *(n=45)*		**T1vs.T3** *(n=19)*		**T2vs.T3** *(n=19)*	
		No.	*%*	No.	*%*	No	*%*
GR							
	≥CEJ	13	*28.89*	4	*21.05*	4	*21.05*
	Apical to CEJ	32	*71.11*	15	*78.95*	15	*78.95*
Risk Factors							
Age	Mean	16.36		*18.25*			
Gender							
	Male	17	*37.78*	6	*31.57*	6	*31.57*
	Female	28	*62.22*	13	*68.42*	13	*68.42*
Treatment							
	Hyrax	21	*46.67*	11	*57.89*	11	*57.89*
	Damon®	24	*53.33*	8	*42.11*	8	*42.11*
KTt							
	Thick	21	*46.67*	12	*63.16*	12	*63.16*
	Thin	24	*53.33*	7	*36.84*	7	*36.84*
KTw							
	≥2mm	28	*62.22*	11	*57.89*	11	*57.89*
	<2mm	17	*37.78*	8	*42.11*	8	*42.11*
Plaque – before (T1)							
	Absent	11	*24.44*	1	*5.26*	8	*42.11*
	Present	34	*75.56*	18	*94.74*	11	*57.89*
Inflammation – after (T2 or T3)							
	Absent	1	*2.22*	6	*31.58*	5	*26.32*
	Present	44	*97.78*	13	*68.42*	14	*73.68*
BTs – after (T2 or T3)							
	Absent	24	*53.33*	10	*52.63*	10	*52.63*
	Present	21	*46.67*	9	*47.37*	9	*47.37*
BBW' – after (T2 or T3)							
	Present (RP3>0)	1	*2.22*	0	*0.00*	0	*0.00*
	Loss (RP3=0; RP6>0)	8	*17.78*	2	*10.53*	2	*10.53*
	Absent (RP3&RP6=0)	36	*80.00*	17	*89.47*	17	*89.47*

3.9.1 Recession T1 vs. T2

Of the eight predictor variables only KTw was statistically significant (as shown in Table 3.9). KTw< 2mm had 18 times higher odds to exhibit GR compared to KTw≥ 2mm [95% CI, 1.28 to 254.66, p=.032]. When compared to KTw alone, there was 12 times higher odds to exhibit GR when KTw is less than 2mm compared to when KT width ≥ 2mm [95% CI, 1.39 to 103.48, p=.024] (Table 3.10)

Table 3.9 Risk factors for recession between baseline and debond (T1 vs. T2).

	B	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**95% C.I.for EXP(B)**	
							Upper	*Lower*
KTt	.24	.92	.07	1	.794	1.27	.21	7.63
KTw	**2.89**	**1.35**	**4.60**	**1**	**.032**	**18.08**	**1.28**	**254.66**
Plaque	-1.06	1.06	1.00	1	.318	.35	.04	2.76
Inflammation	-17.51	40193.02	.00	1	1.000	.00	.00	.
BTs	.75	.87	.76	1	.383	2.13	.39	11.60
BBW'(0)			.30	2	.859			
BBW'(1)	-20.69	40193.02	.00	1	1.000	.00	.00	.
BBW'(2)	-21.31	40193.02	.00	1	1.000	.00	.00	.
Gender	-.20	.86	.05	1	.820	.82	.15	4.45
Treatment	-.88	.98	.80	1	.370	.41	.06	2.85
Age(T1)	.01	.24	.00	1	.958	1.01	.63	1.62
Constant	39.62	56841.54	.00	1	.999	1.61E+17		

Table 3.10 Effect of KTw on GR (T1 vs T2).

	B	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**95% C.I.for EXP(B)**	
							Lower	*Upper*
KTw	**2.48**	**1.10**	**5.11**	**1**	**.024**	**12.00**	**1.39**	**103.48**
Constant	.29	.38	.57	1	.451	1.33		

3.9.2 Recession T1 vs. T3

Between baseline and debond, none of the five risk factors, nor three correlates were related to the presence of GR.

3.9.3 Recession T2 vs. T3

Between debond and follow up, none of the five risk factors, nor three correlates were related to the presence of GR.

3.9.4 Role of BBW' in Recession

The bone width after treatment was not a statistically significant predictor variable of recession before treatment compared to debond, before treatment compared to follow up nor between debond and follow up (p=1.00 for all time points.).

Chapter 4 – Discussion & Conclusions

4.1 Introduction

Both the direction of tooth movement[27,64,83,85,100,127] and the periodontal phenotype[64,100] are among several factors that have been related to the initiation and/or progression of GR in the literature. According to Wennström et al.,[28] however, as long as the tooth is moved within the envelope of alveolar bone, GR will not occur, irrespective of the quality (volume) and quantity (width) of keratinized gingiva. Since Damon® suggests that their appliances are able to produce orthodontic forces within an optimal zone of force that is capable of maintaining baseline buccal bone dimensions with expansion mechanics,[8(p997.e118),167] the goals of this thesis were to compare its treatment outcomes with that of Hyrax expansion (i.e. gold standard) followed by FFA. Specifically, this study aimed to study the periodontal changes (i.e. BBW and soft tissue changes) for the maxillary first molars, first premolars, canines and mandibular first premolars in response to different orthodontic expansion modalities (i.e. Damon® vs. Hyrax) for children, aged 11 to 15 years in permanent dentition using CBCT and photo-analysis.

4.2 General Discussion

The teeth under investigation were the maxillary first molar, first premolars, canines and mandibular first premolars. The maxillary teeth were chosen based on their association with GR in the literature,[77] which often cites thinner buccal alveolar bone,[83] and minimal tissue support.[32,92] The mandibular first premolars were chosen since they are easily visualized in intra-oral photographs and subject to less angular distortion in buccal views. Additionally, since the maxillary arch is expanded to fit the mandibular arch, the mandibular first premolar was chosen as a control tooth, with respect to both BBW and periodontal risk factors. This is similar to other studies investigating the periodontal effects of orthodontic expansion, which most often examined maxillary first molars,[3,13,24–26,53,133,155,156,160] first premolars[13,24,53,90,133,155,156] and canines[133,155] as well.

4.2.1 Demographics

The mean pre-treatment age of patients in this study was 14.20 years (range from 11 to 15 years). For the Damon® group, the mean age was 14.32 years and for the Hyrax group the mean age was 14.06 years. This was similar to other soft tissue studies[25,155] comparing the periodontal outcomes of orthodontic expansion treatment. While the literature suggests that the prevalence of GR is greater in males than females, [78–80,82,87] others found no difference in prevalence between genders in adolescence and young adulthood.[27,83] Since this sample had 1.5 times more females than males, BBW differences related to gender were not explored.

Finally, this retrospective study did not gather any additional information with respect to the patient's SES,[27,71,81,82,87] anatomic predispositions to GR (i.e. high muscle attachment and aberrant frenum, enamel defects), compliance-related factors (i.e. home care, smoking[82] or retainer wear) or trauma (i.e. intra-oral piercings[27,71,87] or toothbrushing technique).[65,74,80] Future studies may opt to include these confounding variables due to their high correlation to the presence of GR.[71,82]

4.2.2 Method: CBCT to measure BBW

Studies have shown that alveolar bony dehiscence reduces the foundational support of teeth.[53,58] In the presence of plaque-induced gingival inflammation, the lack of bony support during orthodontic movement can increase the potential for treatment relapse, be detrimental to the health of the teeth, and the periodontium in the form of GR and in some cases even result in periodontal disease.[28,48,53,58,76,98] As previously mentioned, GR can lead to esthetic or psychological concerns and hypersensitivity.[53,58,64,65,68,89,168] Since the presence of a dehiscence is a prerequisite for the development of GR,[28,45,55,76,109(p975)] the natural first step of this study was to evaluate the change in BBW with expansion treatment (i.e. with Damon® or Hyrax appliances) to correct MTD.

CBCT is an appropriate imaging modality for visualizing skeletal discrepancies, as may be the case for patients with MTD.[39,53] The literature supports the use of CBCT in orthodontics for its high diagnostic value and relatively low radiation dose.[32,36,39,42] It is the only evidence-based imagining modality capable of three dimensional evaluation of alveolar bone in the posterior dentition.[32,48] Linear measurements of alveolar bone were chosen as a proxy over volumetric measurements to describe the alveolar response since the method had already been defined and validated by DiGregorio et al.[160] Moreover, linear measurements may be more clinically relevant than other options such as segmentation or color mapping.[2(p194)] For example, this technique is routinely used to determine the thickness and the height of the alveolar ridge as part of the presurgical assessment for implant therapy, measure the distance between anatomical landmarks, and estimate the size of pathologic lesions of the jaws.[32,50] Should the results of this study have clinical applicability, linear measurement is meaningful and transferrable to a clinical setting.

4.2.2.1 Image Reformatting: Orientation Plane

Measuring buccal alveolar bone around individual teeth using CBCT orthogonal and cross-sectional slices is contingent on the orientation of the sectional plane relative to the tooth in question.[32,42,50] This is especially true for teeth with tipped roots (i.e. where the entirety of the root cannot be visualized without scrolling through the volume), as well as rotated and/or crowded teeth (i.e. where the thinnest portion of bone might not correspond to the buccal surface of the root).[32,42] Analyses have shown differences between linear measurements made at a standard position and upon the changing the orientation plane.[50] For example, should an orthogonal slice be rendered using a standard reference plane, the angulation of the teeth compared to that of the volume clipping may create a line bisecting a non-reproducible portion of the tooth. That is, the cross section bisecting a pre-defined vertical line (y-axis) through a cusp tip compared to a central fossa and roots may determine the extent of the alveolus available to measure (x-axis) and elicit different results.[37,48,147] Furthermore, as a rotated tooth is derotated, the landmarks relative to the standard reference plane can also change, creating non-uniform error for each landmark in all three axes.[161] Other limitations related to the orientation plane is subjectivity, which limits its reproducibility both within and between investigators.

To increase accuracy and reliability of BBW measurements, axial slices were made using tooth-specific landmarks perpendicular to the long axis of each individual tooth in the buccolingual direction.[25,32(p930),42,50,160] The landmarks required to create the orthogonal slices were the root apex (i.e. palatal root first molars and buccal root for first premolars), a tooth mid-point (i.e. the middle of the molar furcation and the midpoint between the apex and the coronal-most aspect of the pulp chamber for premolars and canines) as well as the center of the pulp chamber or coronal-most pulphorn. This is the same method used by DiGregorio et al,[160] and is also similar to Morais et al.[25] and Cattaneo et al.[90] who generated cross sections passing through the pulp apex and the center of the root of each study tooth, irrespective of contralateral teeth,[13,26] skeletal reference planes[133,155,156] or tomographic vertical planes.[3]

4.2.2.2 Identification of Landmarks: CEJ, ABM

The accuracy of identifying anatomic landmarks using various 3D software programs for CBCT has been verified in the literature, especially if an identification protocol is established and landmark identification is practiced by the operator.[32(pp18, 652),161,169] Of the tooth-borne landmarks, both the CEJ[3,90,156,160,170–172] and the incisal edge.[13,26,155] are commonly used reference points from which to establish vertical measurements. Since both landmarks are identified as regions of brightness, relative to surrounding darker densities,[48] identifying the structures is limited the scan's voxel size, or the *partial volume averaging*.[48] That said, the incisal edge is also subject to change based on occlusal wear, fractures limited to enamel, and restorative or esthetic recontouring. As such, both a 3D rendering and three planes of space were used to establish the apical-most extent of the buccal CEJ (i.e. the junction between enamel and cementum) was located for each study tooth. The buccal CEJ was used as a reference point to establish two root points (i.e. RP3 and RP6) that would act as markers for each horizontal alveolar thickness (i.e. BBW) measurement. This study used a 0.3mm voxel size, which implies that the location of the CEJ should be accurate within the same measurement.

Another possible source of landmark error lies in the identification of the root surface compared to the alveolar bone margin (ABM).[52] The reason for this is two-fold. First, these tissues are often similar in brightness on a CBCT due to their similar hydroxyapatite content.[48,88,109] Secondly, the buccal bone overlying the dentition often presents in submillimetre buccolingual dimension.[25,52] Ballrick et al.[43] found that the interline distance required to visually separate four lines of identical density can be up to 0.86 mm for large FOV, large voxel-size CBCT images (i.e. the *spatial resolution*). Since this is larger than the average PDL thickness (0.5 mm), identification of the ABM with respect to the cemental surface may be obscured in the cross-section, based on the spatial resolution of the CBCT unit used. For example, Leung et al.[48] found the location of the ABM to be accurate within the same spatial resolution of the CBCT (i.e. 0.6mm) compared to direct caliper measurement. The spatial resolution of the CBCT unit used in this study is between 0.6mm and 0.8mm.[41]

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4.2.2.3 Other Factors

Reliability can be affected by image slice thickness and interslice interval which are often determined by the operator (task-specific imaging).[50] To manage this and increase the diagnostic value of the present study, a standard image reconstruction orientation protocol previously validated by DiGregorio et al.[160] was utilizes with 1mm slice thickness. The literature cites the operator-related factors such as experience with CBCT, and their ability to visualize a grayscale, which is influenced by lighting, fatigue, and visual acuity.[52] These factors can substantially increase inter-rater variability of alveolar bone height measurements from CBCT images.[52,58] The primary investigator (M.K.) of this study had three years of CBCT experience and the conditions under which the volumes were evaluated were constant (i.e. Mimics software, same laptop, same lighting conditions).

Finally, it is important to consider the technical parameters of the CBCT that limit methodological reliability (i.e. FOV, noise, scatter, focal point, number of basis images and the reconstruction algorithm).[32,36,42,173] To manage this, the present study evaluated only those subjects who were scanned with the same parameters on the same CBCT unit (i.e. those who started treatment after 2012) and the authors removed scans thereafter with evidence of artefacts (i.e. movement, beam hardening, ring).

4.2.2.4 Accuracy of linear measurements

Despite the methodological shortcomings, CBCT has been known to have small mean error and good to excellent correlation coefficients reported. Even in 2005, Ballrick et al.[43] acknowledged that even though an image " is not perfectly clear, it can still adequately represent the structures and be of diagnostic value." [43,47] More recently studies have shown that the linear measurements derived from CBCT are reliable, with submillimeter accuracy compared to digital caliper measurements.[35,44,47,49,147] For this study, the intra-rater reliability (ICC) of the BBW measurements was high, ranging from 0.82 [95%CI, 0.68 to 0.92] to 0.98 [95%CI, 0.95 to 0.99]. A low average measurement error of 0.21 mm was identified, with a minimum error of 0.09mm for the maxillary canines at RP3 and a maximum error of 0.31mm for the maxillary canines at RP6. This is similar to DiGregorio et al.[160] who reported an average measurement error of 0.14 mm, with a minimum error of 0.02 mm and a maximum error of 0.34 mm for the first molars.

Other studies evaluating BBW using CBCT reported error as either measurement error using *Dalhberg's Formula*[§164] with reported insignificant values[25,90,133,156] or intra-examiner reliability testing with ICC ≥ 0.878.[3,13,26,53,155,172] Similarly, Timock et al.[47] and Tomasi et al.[51] reported excellent inter-rater reliability with ICC > 0.98.

4.2.3 Results of CBCT analysis

4.2.3.1 Overall results

This study found that irrespective of treatment type, all study teeth had an average decrease in BBW in the transverse dimension after orthodontic expansion. This is corroborated by other studies that identified a reduction in maxillary buccal alveolar width and height immediately after expansion treatment[14–17,28,128] with both Hyrax[26,133,155,156,174] and Damon®[25,90] appliances. Our study corresponds to the above findings with a decrease in mean alveolar bone thickness from 0.72mm to 0.51mm at debond and 0.39 mm at follow up.

With orthodontic expansion, the reduction in alveolar bone overlying the maxillary first molars 3mm apical to the CEJ was less than at 6mm (34% and 52%, respectively) at debond. This is similar to the findings of Morais et al.[25] who reported for 3 and 6mm apical to the CEJ, the mesiobuccal root of the molars had a significant reduction in BBW (36% and 45%, respectively) after expansion with Damon® appliances. Additionally, 6mm apical to the CEJ on the maxillary first molars showed more of a decrease in BBW at debond than the maxillary first premolars (0.36mm [$\pm$0.09]), canines (0.46mm [$\pm$.08]) and mandibular first premolars (0.34mm [$\pm$0.09]). While the difference in the change between the each TRP is more than the average measurement error (0.21mm), none of the results are more than the spatial resolution of the scan (0.6-0.9mm) indicating possible clinical significance. Rungcharassaeng et al.[13] found significant dental expansion, buccal crown tipping, and loss in bone thickness and height of maxillary posterior teeth in the short term (within 3 months) after RME. This implies the following possibilities:

1) Since patients with MTD have characteristic buccally tipped maxillary posterior segments, it is possible that the BBW overlying the molars was thinner in the coronal 1/3 and thicker in the apical 1/3, such that there was more potential for lateral movement through bone in the apical 1/3. Since the lowest measured BBW possible was zero, the

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§ $S^2 = \frac{\Sigma d^2}{2n}$

maximum ΔBBW can only be the initial measured thickness (i.e. $\Delta BBW = BBW_b - BBW_a$; where a=after and b=before).

2) The maxillary molars had the most lateral movement at RP6, compared to the other teeth of interest at the same root point. While this is suggestive of either increased bodily movement or increased tipping of the maxillary molars compared to the other teeth under investigation, neither tooth position nor the angulation were measured in this study. Inferences regarding the type of tooth movement related to the change in BBW are presumptuous and cannot be ascertained.

Among the nineteen patients who were evaluated at follow up, there were significant changes in BBW overlying the maxillary molars at RP3 between baseline and debond (p=.001), but not between debond and follow up (p=.646). Similarly, the maxillary first molars and premolars at 3 and 6mm from the CEJ showed significant changes in BBW between baseline and follow up (p<.001, p=.001, p<.0001, p=.002). This is similar to the findings of other studies evaluating Hyrax RME,[26,133,155,156] whereby there was a significant increase in dehiscence formation and a decrease in buccal bone width, especially over banded abutment teeth.

4.2.3.2 Between Treatment Results

While our results demonstrated that those treated with Hyrax expanders had more buccal alveolar bone loss than those treated with Damon® appliances at debond (0.12mm ($\pm$0.05) [95% CI, 0.03 to 0.22]), this result should be interpreted with caution. First, the difference in the change between the two treatment groups is less than the measurement error (0.21mm), and the spatial resolution of the scan (0.6-0.8mm), indicating lack of clinical significance. Moreover, follow-up statistics revealed that the difference between treatment outcomes was observed only for the maxillary canines, at least 3mm apical to the CEJ. This is due to the high prevalence of alveolar dehiscences prior to treatment with Damon® appliances, at baseline (63.89%), compared to 36.11% of the Hyrax group (13/36, p=.031). Possible explanations for this difference could be random error during treatment group allocation at baseline during the RCT, the methodology for exclusion in this retrospective study and/or the transformation of the data to carry out statistical analysis.

A common finding in MTD is buccally displaced canines,[2(p447)] wherein the facial surface of the roots, especially 3mm apical to the CEJ, was either covered by very thin alveolar bone, or just soft tissue alone. Alveolar dehiscence may be present where the buccolingual dimension of a root is similar to or exceeds the crestal bone thickness.[74,85,175] In this case, lingualization (not expansion) is often the resultant movement to bring the crown lingual, towards the arch. It is well known in the literature that redirection of a root into the alveolar process can be complemented by improved marginal bone level and width.[28,128,129] Since it can be inferred that the canine lingualization would improve or at least maintain the alveolar bone position, the direction of movement is such that it is not relevant to this study.

The maxillary first molars demonstrated overall differences between treatment groups at RP3 where less bone loss at debond was observed in the Damon® group (p=.010). That said, there was no difference in BBW between Damon® and Hyrax treatment groups at follow up (p=.830). This implies that, despite differences in mean BBW at debond, both Damon® and Hyrax groups had similar mean BBW after two year follow up. With respect to dehiscence formation, 80% of severe dehiscences (i.e. $BBW_{RP3\&RP6}$=0mm) were observed at debond in patients treated with Hyrax appliances (8/10) compared to those treated with Damon® appliances (20%, $p<0.044$). At follow-up; there was no difference observed between the two groups. A similar result was observed for the mandibular premolars, where 85% of all dehiscences 3mm apical to the CEJ were found in the Damon® group, compared to 15% in the Hyrax group ($p<0.0001$). There was no appreciable difference observed between the groups at two-year follow up.

One possible explanation for the differences between treatment groups at debond may be related to when the CBCT was taken. Timing of image acquisition is an important consideration due to the likelihood of active alveolar remodeling around recently moved teeth. Should remodeling be ongoing, the osteoclastic activity may decrease the density of the active bone, aka *regional acceleratory phenomenon* (RAP).[42] For example, buccal bone undergoing RAP would appear less dense on a CBCT scan, giving the impression that the ABM is along the less-active bone.[42] Since RAP takes six to 24 months to fully subside after the end of tooth movement,[37,42] simultaneous debond and CBCT acquisition may be confounded by alveolar remodeling. Therefore, measurements taken at two-year follow up may be more representative of treatment related changes.

CBCT has a high specificity and a high negative predictive value for alveolar bone dehiscence.[48,53] As discussed, the identification of submillimeter alveolar bone is difficult to detect since the structures measured are often less than less than spatial resolution and the size of the voxel.[25,32,48] Since the buccal cortical plate often exists in a thickness of around 0.3mm,[64] this study's scanning parameters (i.e. full FOV CBCT at 0.3mm voxel size) may not be appropriate to identify thin cortical bone since the full FOV scans that are frequently used in orthodontics are associated with increased scatter and noise, which decreases the spatial resolution.[42] This, combined with the RAP effect at debond could very well have contributed to the observed floor effect at TRPs with particularly thin alveolar bone. While it is also possible that some relapse may have occurred between debond and follow up allowing buccal bone deposition,[129] the limitations of CBCT imaging of biologically active sites should not be overlooked.

4.2.4 Method: Photo-analysis to identify soft tissue risk factors associated with GR

In the presence of plaque-induced gingival inflammation, the lack of bony support during orthodontic movement can increase the potential for treatment relapse, be detrimental to the health of the teeth, and the periodontium in the form of GR and, in some cases, even result in periodontal disease.[28,48,53,58,76,98] As such, developing GR during or after orthodontic treatment can pose a significant clinical problem.[89] This not only highlights the need to undertake a risk assessment before treatment is commenced, but also to re-evaluate for signs of GR or associated risk factors (RFs) after treatment and at subsequent follow up visits.[32,89] For example, the current reported prevalence spans 5% to 12% at the end of treatment,[64] however, this increases to up to 47% in longer-term observation (5 years).[64] That said, the task remains to identify contributing RFs.[87]

This study was particularly interested in the onset and/or advancement of GR concomitant with expansion treatment and the associated periodontal RFs. Presence of gingival inflammation and baseline recession,[76] a thin gingival biotype,[76,98] a narrow width of keratinized gingiva[76,98] or a thin alveolar bone with respect to the direction of tooth movement[84] were found to correlate significantly with the development or increase in gingival recession in orthodontic patients.[27]

Methods described to evaluate soft tissue retrospectively in the literature were most often photo[76,110,176] and/or cast[76,110] analysis. For example, Melsen and Allais[76] projected intraoral photographic slides onto a 0.8 x 1m screen from 2m away for analysis in a dark room. The slides were calibrated using the measured width of a central incisor from a study cast. This method was repeated in the evaluation of soft tissue RFs associated with GR by Ashfaq et al.[176] Similarly, Sawan et al.[110] used a combination of photo and cast analysis to evaluate RFs that might contribute with GR during orthodontic treatment; however, they used direct measurement on the cast to create a scale and allow for measurement on the photo.

The methodology for photo-analysis for this study was adapted from a multi-center study by Le Roch et al.[142] whereby the authors validated the methods in Kerner et al.'s Before and After Scoring (BASS) system[140], Cairo, et al.'s Root Esthetic Score (RES) system[139] and Fürhauser et al.'s Pink Esthetic Score (PES) system.[141] Using matched clinical views (i.e. frontal and lateral intraoral photographs) of identical teeth at two time points (i.e. baseline, debond, or follow-up), the tissue was graded (0,1) based on a visual inspection of soft tissue factors, irrespective of the type of procedure used, the probing depth, and without magnification. Prior to treatment, the RFs evaluated included the keratinized tissue thickness (KTt) and the visual presence of plaque. The RFs examined after treatment included the keratinized tissue width (KTw), the visual presence of inflammation, and signs of papilla blunting or black triangles (BT).

4.2.4.1 Reliability of the Method

Le Roch et al.[142] found the RES,[139] BASS[140] and PES[141] systems were high to moderately reproducible tools for the evaluation of variables related to gingival soft tissue esthetics using standardized intraoral photography among students and teachers. The RES system had the highest reproducibility, followed by the BASS system, then the PES system.[142] While internal consistency for this study showed excellent agreement from 0.86 for KTt to 0.99 for GR, the results of the multi-center study[142] differed from our study where the reliability between examiners was low to moderate for all parameters. For example, inter-rater reliability between MPG and MK ranged from low to moderate ($\kappa = 0.11\ to\ \kappa = 0.51$); and, even with a third evaluator (DC), the overall reliability was moderate ($\kappa = 0.05\ to\ \kappa = 0.39$). As such, the identification of risk factors was determined by a group consensus of two practicing periodontists (MPG and DC) and the principal investigator (MK).

In general, reliability is influenced by the number of categories used.[164(p496)] While this study used a dichotomous scale to determine the presence of soft tissue risk factors, it is important to note that Le Roch et al.'s[142] multi-centre study compared each variable across six categories for each of the RES, BASS and PES systems. Due to different scoring methods, we cannot directly compare the measures of agreement directly to our current study; however, they have been included for discussion purposes knowing that increasing the number of categories and the number of raters will decrease the extent of agreement.[164(p496)] The overall and weighted reliability of the parameters investigated in this study can be found in Appendix A, Table A1.4.

Overall, the lower reliability observed in this study can be attributed to several factors. Most obviously was the inclusion of multiple posterior teeth, which may have been more difficult to take quality photographs of in children due to the stretching of the cheeks that is required for a high resolution, perpendicular image. There was a lack of standardization of the intra-oral photos, wherein structures may have been out of focus, cropped out of the view-finder or obscured (i.e. by saliva or debris). Where the RES system, for example, limited the photo being evaluated to a single tooth, taken with a ring-flash system, with a parallel, eye-level orientation to the camera, the present study utilized an Olympus TG-6 camera with mounted flash and sextant photography (i.e. right and left lateral/buccal shots and a frontal shot). Photos were angled from 90 to up to 45 degrees to the buccal surface. Another possibility is that the present study evaluated the RFs using a global evaluation method, where the entire case was scored as 0 or 1 for each RF, based on the presence of a RF on one of the sites of interest. In the evaluation of esthetics, the BASS[177] system also suggested using a global evaluation of aesthetics and found similar, low to moderate reproducibility (≤ 31%), whatever the examiner's training.[142]

4.2.4.2 Reliability of the Individual RFs

At the item level, GR is defined as the apical shift of the marginal periodontal tissues relative to the CEJ resulting in exposure of the root surface to the oral environment.[64–66] It was identified when the FGM was apical to the CEJ. Should no color change be visible between the enamel and root surface, the visibility of an anatomical depression between the enamel and cementum was accepted as the CEJ.[88] While the inter-rater reliability for GR for this study was moderate ($\kappa = 0.35$), Le Roch et al.[142] found high agreement (κ>0.60) for the diagnosis of GR (using the RES,[139] BASS[140] and PES[141] systems) independent of the level of the participants' training. This is similar to the findings of Ashfaq et al.[176] where intra-panel reliability was assessed for photographic measurement for GR using the ICC and found it to be highly correlated (0.884). A possible explanation for the lower reliability in this study may be due to the acceptance of the CEJ as an anatomical depression between the enamel and cementum, as this was only visible in the frontal shot and may have been affected by posterior shadows. Other studies evaluated the presence of GR from a position perpendicular to the facial surface of the tooth of interest only.[76,99,107,139,141,142,177]

The vertical width (KTw) of the attached tissue was defined as the most apical point of the FGM to its related MGJ, assessed on the 'after' photos. The MGJ was identified by the color differences between the alveolar mucosa and the keratinized gingiva.[76] When the vertical height of tissue apical to a study tooth was <2mm, KTw was considered to be a RF. Visual evaluation of this quantitative variable likely affected this study's reproducibility (κ<0.18). To overcome this, other studies used cast measurements to create a digital scale on the imaging software and directly measured the height of tissue on intraoral images.[76,99,176] That said, using the same method as the present study, the evaluation of keratinized tissue was at least moderately reproducible (κ<0.40) with the BASS[140] system. Therefore, it is also possible that factors such as the presence of inflammation and/or tissue hypertrophy at debond may have obstructed the evaluators' estimate of the keratinized tissue.

Black Triangles (BTs) or tissue blunting was assessed with respect to its fill in relation to the CEJ and the contact point of the teeth.[106] Le Roch et al.[142] found that identification of the shape of the papilla to have high reproducibility ($\kappa > 0.80$) among instructors using the PES[141] system. That said, the reproducibility in this study was poor ($\kappa > 0.11$). Again, the quality of the photos as well as the global evaluation of a tooth-by-tooth RF most likely contributed to the variation observed.

Keratinized tissue thickness (KTt) was evaluated as thick or thin, based on the pre-treatment photographs since debond photos typically showed high levels of hypertrophy and evidence of soft tissue abrasion. It was defined as thin (1) on the basis of visual inspection of the gingival texture, color change with respect to root visibility, and the presence of vasculature and/or whitening of retracted tissues.[76,104] Melsen and Allais[76] also evaluated gingival biotype on the pre-treatment intraoral slides as thin or thick on the basis of visual inspection of the gingival texture and capillary transparency.[104,109] While other studies used the shape and scallop of the gingival margin to establish tissue type,[104,109,175,176] De Rouk et al.[103] included a crown width/crown length ratio, gingival height, and papilla height to establish gingival thickness. In the present study, the reproducibility of KTt was fair (κ<0.39), and similar to that reported in the literature. For example, Le Roch et al.[142] found that soft tissue volume had poor to moderate agreement for the PES and BASS systems. It was also similar to Eghbali et al.[108] who identified poor to moderate (k: 0.127–0.547) reproducibility for identification of tissue biotype.

Gingival inflammation was ascertained in the 'after' photos as swelling, edema, and/or erythema/redness[64] on the study teeth from either frontal or buccal views. Plaque, on the other hand, was assessed in the 'before' photos via visual appearance of color or texture changes along the tooth surface. The visual presence of plaque and gingival inflammation was also identified by Allais and Melsen[76,99] on intraoral slides. Le Roch et al.[142] found high agreement ($\kappa > .60$) for color (using the RES[139] and BASS[140] systems) and texture (using the BASS[140] system) changes among specialists; however, the reproducibility for inflammation and plaque for this study was fair to poor ($\kappa = 0.25\ and\ \kappa = 0.05$, respectively). An explanation for this is likely the over-representation of these RFs at the chosen time points for evaluation. That is, of the matched baseline and debond cases, 75% and 98% presented with signs of plaque and inflammation, respectively. When examining homogeneous characteristics, the expected agreement by chance alone will be high, rendering little opportunity for non-chance agreement and deflating kappa.[164(p496)]

4.2.5 Results of the Photoanalysis of GR and Soft Tissue RFs

4.2.5.1 Development of GR at Debond and Two-Year Follow up

Overall, 71% of patients demonstrated evidence of GR on at least one of the study teeth at debond, after expansion treatment. While a strong correlation exists between the extent and severity of GR to a past history of orthodontics,[87] the current consensus on the reported prevalence spans only 5% to 12% by the end of treatment.[64] In this study, the presence of GR at two year follow up was 79%. While this is more similar to the reported prevalence of 47% at follow up (5 years),[64] it is still considerably higher. The most obvious explanation of the high prevalence GR observed in this study, compared to the literature may be related to the extent and direction of tooth movement given the physiologic limitations of the alveolar housing in patients with MTD. That is, when the transverse dimension of the palate is less than that of the mandibular intermolar transverse width,[2(p201)] the extent of transverse movement required for correction will be higher than for those with a palatal width that is the same as the mandibular intermolar width. Therefore, the difference between the population-level data with respect to orthodontics and GR and our sample of patients with MTD can be explained by the amount of orthodontic expansion required to treat MTD non-surgically.

The concept of multiple etiologies of GR has been supported by parallel longitudinal studies in Norwegian and Sri Lankan populations.[74,81] Between the study populations, GR was more widespread among those with better oral hygiene.[81] This implies that factors related to particular populations, such as the aforementioned predisposing and precipitating factors, may exacerbate the effects of age on GR.[27,71,74,78–84] That is, differences with other studies may be explained by sample related differences such as factors related to KTw, [64,76,98,110,111] KTt, [27,64,71,76,98–100] inflammation, [14,27,105,117] and past orthodontic history[27,28,64,83,85,87,127] and factors not controlled for, such as oral health habits (i.e. toothbrushing), [27,64,71,74,77,78,81,87,105,125] access to oral care,[78] hygiene, diet and/or smoking.[83]

In a US-based study, Morris et al.[84] identified GR (of at least 1 tooth) in 18% of children treated orthodontically at debond. This increased to 56% at fifteen year follow up. In the Netherlands, Renkema et al[83] found that only 6.6% of treated children had GR at debond; however, this increased to 38% at five year follow up. While the present study did not include oral hygiene practices, the presence of plaque was identified on 76% of the patients at baseline and 58% of the patients at debond (since the teeth were polished). Similarly, 98% of patients displayed signs of inflammation at debond, which reduced to 68% at follow up. In our sample, approximately 24.72% of the teeth evaluated at baseline presented with radiographic evidence of dehiscence 3mm from the CEJ. More severe dehiscences 6mm from the CEJ were noted in 13.89% of the teeth at baseline. After expansion, this increased to 30.83% and 24.17%, respectively. This is important since site specific challenges (i.e. molar bands or dental tipping) or host-specific challenges (i.e. alteration of oral hygiene habits or plaque retentive nature of orthodontic appliances during treatment) can initiate or exacerbate GR.[48,87,117,119,127,137] That is, the high prevalence of plaque and inflammation observed in the current sample may have contributed to the results.

It is also possible that the method used for identification of GR affects the reported prevalence, whereby visual inspection of photographs or model analysis alone, or the use of photographs and model analysis in concert may render a different estimation of GR in the sample. For this study, the anatomical CEJ depression was analyzed from the frontal view as well as the presence of both the CEJ depression and/or a color change indicating cemental surface exposure from the lateral view. Other studies with high reliability utilized color change and/or a color change indicating cemental surface exposure from the perpendicular view only, as was described my many other authors.[76,99,107,139,141,142,177] It is, therefore, possible that this study may have overestimated GR in the sample compared to other studies, due to the use of the CEJ depression[88] from the frontal photo that were not otherwise visualized in the lateral photo. Contrarily, the other possibility is that photo analysis using only the perpendicular view may actually underestimate GR. This would be due to the variation in clinical presentation of the CEJ, (i.e. the cementum can either overlap the enamel [60-65%], create an edge-to-edge butt joint with the enamel [30%], or exist separately from the enamel, exposing dentin [5-10%]),[88] and the FGM which can obstruct the CEJ due to hypertrophy, for example.

Other considerations for the differences in the literature can be attributed to the cut off points for the diagnosis of GR. For example, while Susin et al.[82] noted recession only if it was 1 mm or greater, this study included any evidence of apical migration from the CEJ as GR.

4.2.5.2 Age, Gender and Treatment Method

Despite suggestions in the literature that GR is more prevalent in males compared to females,[78–80,82,87] the present study found no relationship with gender. This is comparable to others that found no difference in prevalence of GR between genders in adolescence and young adulthood.[27,83] This study also found no relationship between GR and the baseline age of the patient. While this differs from the findings of Renkema et al.,[83] where age at the end of treatment was associated with the increased numbers of recessions, where those who finished under the age of 16 years were less likely to develop GR, the patients of this study were all treated for two years, so the observed differences in age at the end of treatment would be the same as at baseline. Epidemiological studies examining the untreated, natural history of GR where there is an increase in prevalence with age. For example, Albander and Kingman[80] demonstrated that the prevalence and extent of recession among untreated subjects increases with age using data representative of the US population (NHANES III). Similarly, in an untreated representative sample from Brazil, Susin et al.[82] also demonstrated significant increases in the prevalence and extent of recession with age where approximately 96% of the subjects in their 30 to 39-year-old cohort demonstrated GR on at least 1 tooth. This is interesting since the Hyrax expansion appliance works optimally prior to suture fusion, and decreases in orthopaedic efficacy based on development thereafter.[178] It is logical to assume this sample would be subject to age-related differences based on developmental differences during puberty. That said, our results are in line with other studies examining the relationship between GR and orthodontic treatment where most found no age-related differences within a particular age group.[76,87,99,135]

Finally, this study found no relationship between the development of GR and the use of Damon® or Hyrax appliances for expansion. This is in contrast to the Damon® hypothesis that moving teeth within an optimal force zone throughout treatment allows physiological adaptation to take place and allows the alveolar bone and associated periodontal soft tissues to move with the teeth.[167] In fact, of the 53% of the sample treated with Damon® appliances, 70.83% of them showed evidence of GR at debond; this is almost identical to the Hyrax group where 71.43% showed evidence of recession at debond.

4.2.5.3 Buccal Bone Width and GR

The BBW measurements were categorized based on the definition of dehiscence being bone loss 3mm or more from the CEJ.[48] Three dehiscence categories were developed based on apical extent of bone loss from the CEJ:

1) If the measured buccal bone width is greater than zero at both 3mm and 6mm from the CEJ, there was no dehiscence (i.e. BBW'=0)
2) If the measured buccal bone width is zero 3mm from the CEJ, but greater than zero at 6mm from the CEJ, there is a dehiscence up to at least 3mm from the CEJ. (i.e. BBW'=1)
3) If the measured buccal bone width is less than zero at both 3mm and 6mm from the CEJ, there is a dehiscence up to at least 6mm from the CEJ (BBW'=2)

Overall, baseline dehiscences were found in 38.61% of the sample teeth, with 3 and 6mm vertical bone loss found in 24.72% and 13.89% of the sample teeth, respectively. Patients were assigned a score of 0-2, based on the presence of at least one of the aforementioned categories being found at either debond or follow up. That said, most patients (93%) exhibited at least one dehiscence (3mm or 6mm) prior to treatment on at least one of the study teeth, with 53.33% being severe. At debond, 97% of patients exhibited at least one dehiscence with 80% being severe. At follow up, 100% of the patients exhibited at least one dehiscence with 89.47% being severe. Due to the ubiquity of dehiscence in the sample, there was no relationship between the dehiscence and the presence of recession. This is similar to the findings of Greenbaum and Zacchrisson,[29] who noted that regardless of the movement pattern of the dentition involved, the post-treatment response of the buccal tissues appears to have been minimal when compared with similar tissues not exposed to the forces of palatal expansion therapy.

It was intersting to note that, even though the RAP period had subsided two-years after the end of tooth movement,[37,42] both the prevalence and severity of dehiscence increased in the follow up groups (though this was no statististically significant). From an occlusal perspective, this may be due to the traumatic effect of tipping teeth into position, resulting in non-axial loading and micro-trauma to the alveolus.[71] That said, neither tooth position nor the angulation were measured in this study, and the participants' occlusion was not evaluated so the role of occlusion is purely speculative. Another possibility is that the cohort presenting for follow up may be inherently more compliant and more likely to wear their retainers, which would hold their teeth in position and reduce potential for the teeth to settle.

Additionally, the severity and prevalence of dehiscence in this sample was increased compared to other studies examining general orthodontic patients. For example, Jäger et al.[95] observed that, before orthodontic treatment, 20% of patients exhibited dehiscence defects – 73% lower than what we observed in this study. Due to the differences, it is plausible that patients with MTD may have dental and skeletal patterns that increase their risk for periodontal attachment loss. For example, Choi et al.[53] found that adult subjects with posterior crossbite had a higher prevalence of total bony defects, especially buccal dehiscence in the posterior region, than subjects with no posterior crossbite. However, the differences in prevalence was approximately 10%, which may not be clinically meaningful.[53] While patients with posterior crossbites may prefer to have correction with orthodontic expansion (i.e. instead of more invasive surgical methods), the patient's specific anatomy and the etiology of the crossbite (i.e. dental vs skeletal) may result in tooth movement through a thin osseous plate and immediate reduction of alveolar support.[32,53] Especially since the deterioration of underlying periodontal structures may not be immediately clinically evident, orthodontists may inadvertently underestimate the creation of irreversible hard-tissue changes that could accompany such tooth movements.[47] As such, it is recommended that orthodontists understand the anatomical limits of tooth movement and be aware of potential periodontal impact of expansion orthodontics.[53]

4.2.5.4 Risk Factors for GR

Of the predictor variables evaluated, only KTw demonstrated a significant relationship to GR, where KTw less than 2mm had 18 times higher odds to exhibit GR compared to when KTw was greater than or equal to 2mm. When compared to KTw alone, there was 12 times higher odds to exhibit GR when KTw is less than 2mm compared to greater than or equal to 2mm. Our findings are similar to other photo-analyses that also identified KTw as an important correlate to GR. In a study examining the soft tissue effects of orthodontic treatment, Sawan et al.[110] found that both pre- and post-treatment keratinized gingival height were significantly related to the prevalence of GR, where each 1 mm increase in pre-treatment keratinized gingival height, was related to 0.77 times lower odds of gingival recession. For each 1 mm increase in post-treatment keratinized gingival height, there was 0.51 times lower odds of gingival recession.[110] Melsen and Alais[179] found that with facial movement of the mandibular incisors, the onset or exacerbation of GR was related to the KTw at baseline. For this study, tissue width less than 2mm was present on 37.78% of the study teeth. Maynard and Ochsenbien[85] found that, in children, KTw less than 2mm was only found in 12 – 19% of the permanent dentition; however, for those who are treated with orthodontics, the width of the attached tissue has shown variability based on the direction of tooth movement. Moreover, classic studies like that by Lang and Löe[111] demonstrated that while gingival health is compatible with a very narrow gingiva, in areas with less than 2mm keratinized gingiva inflammation and varying amounts of gingival exudate persisted in spite of effective oral hygiene. Since then, the quantity of attached gingiva, as per the AAP consensus statement on mucogingival conditions,[64] suggests that keratinized tissue <2mm in height (i.e. <1mm attached gingiva) is more susceptible to the development of GR due to the absence of an adequate band of gingiva.[71] The literature suggests that this would reduce the tissue resistance to plaque accumulation and aberrant muscle attachment and lead to GR.[71,111]

It is important to remember that the region under examination is composed of distinct tissues – the gingiva, periodontal ligament, cementum and alveolar bone - that function together as a single unit.[88] That is, the vertical height and bucco-lingual thickness of the alveolar bone overlying the teeth, and the vertical height and thickness of the attached / keratinized gingival tissue act in concert to orthodontic forces and changes in tooth position. It is well known that the tissue boundaries (i.e. both facial and FGM) are susceptible to change with the apical migration of the alveolus, in both vertical and bucco-lingual dimensions.[28,98] Therefore, expansion of teeth into this boundary can induce free gingival-margin tension, which would become narrower with apico-coronal height and thinner with buccolingual dimension and should make labial gingival tissue more vulnerable and less resistant to plaque and toothbrush trauma.[28,98] That said, the consensus statement discusses *periodontal phenotype* in relation to both KTw and KTt, concurrently.[64]

Most of the patients presented with a thin tissue type (53%). Numerous sources cite the importance of gingival biotype (i.e. thick / thin) as the critical link between the onset and/or progression of GR with orthodontic expansion treatment.[27,64,71,76,98–100] A recent study by Ashfaq et al.[176] found that the odds ratio of GR in thin gingival biotype increases by 10.2 times more than those in the thick gingival biotype. Studies reporting on the development of GR after labial movement of lower incisors have also demonstrated that a thin gingival margin is a significant predisposing factor for the development of GR with gingival thickness of less than 0.5mm rendering the site more susceptible.[71,98] That said, this study found no correlation between tissue type and GR. This may be explained, at least in part, by the limitations of this study's photo-analysis method. For example, in their attempt to evaluate the accuracy of visual inspection of tissue biotype for calibrated dental professionals, Cuny-Houchmand et al.[107] concluded that simple visual inspection was not effective for identifying tissue type and that there may be differences by location in the mouth. The authors found that thick-flat biotype is most frequently identified in the maxilla (i.e. 44.87%) while it is less frequently identified in the mandible (i.e. 32.65%).[107] While this study isolated risk assessment to the study teeth, a 0/1 score was given for the patient, not by site. Coding the entire mouth as thick or thin may generalize jaw-related nuances related to skeletal hypoplasias and the site-specific nuances related to dental crowding.[107] Similarly, Eghbali et al.[108] found that, regardless of experience, gingival biotype was accurately identified only in about half of the cases under review. In both of the aforementioned studies, visual inspection of biotype fell short for those cases most at risk of GR, where thin-scalloped biotype was the most difficult to identify.[107,108]

4.3 Limitations

4.3.1 Study Level

The most obvious limitation of this study is the retrospective study design, which introduces information bias in the form of incomplete or inaccurate records, and unexplained loss to follow up.[164] Another major limitation of this study was the small sample size (n=19) for two-year follow up and the subsequent lack of generalizability of the results beyond the sample characteristics. For example, a minimum sample size of 30 patients per group (i.e. N=72) was calculated to be needed based on the previous research using a statistical power of 0.90 considering an α=0.5.[166] The follow up group consisted of 19 subjects, 11 who underwent expansion with a Hyrax expander, and eight who underwent Damon expansion. Since the model with the most amount of data points (i.e. T1 vs. T2) was found to be the most useful, inclusion of more data at follow-up would likely render more significant results. Future studies should aim to include larger samples (i.e. n>30) with longer term follow up.

While the CBCT analysis compared the alveolar effects of Damon® to Hyrax as the gold standard, neither leg of this study compared results to a control group. While this was not possible for CBCT analysis due to the justification for radiation exposure,[32,42] the results of the soft tissue analysis would be more meaningful with an untreated sample of aged matched patients with MTD as a control group. According to the literature, confounding variables such as SES,[27,71,81,82,87] anatomic predispositions to GR (i.e. high muscle attachment and aberrant frenum, enamel defects), compliance-related factors (i.e. home care, smoking[82] or retainer wear) or trauma (i.e. or intra-oral piercings[27,71,87]toothbrushing technique)[65,74,80] are related to GR. It would be prudent for future studies to include these confounding factors in the study design.

4.3.2 Methodological Limitations

4.3.2.1 Methodological Limitations of CBCT

The knowledge that there is close to 100% probability of opening the midpalatal suture with a banded or bonded expansion device for children in the early permanent dentition[2(p433)] helped to guide the inclusion criteria for this study. That said, as the adolescent growth spurt ends, interdigitation of the suture reaches the point that opening it may no longer be possible. Since Hyrax expansion is more effective at sutural expansion prior to the adolescent growth spurt, more dental tipping vs. sutural expansion may be seen based on the patients growth status. As such, developmental stage (instead of patient age), may be an important confounder in this study since the amount of expansion attributed to skeletal and dental movements was not investigated. Future studies may consider inclusion of one of several developmental staging methods, including *cervical vertebra maturation staging* (CVMS), which is used to estimate mandibular growth potential,[2(p434),180] to evaluate mid-palatal suture maturation using readily available from any cephalometric radiograph. Other options include evaluation of a hand-wrist radiograph for calcification of the sesamoid bone, or the *mid-palatal suture density ratio* (MDSR). [2(p434),181] In the latter method, Grünheid et al.[182] developed a ratio of CBCT derived gray levels in a defined palatal region, where 0 indicates less calcification and gray levels closer to soft tissue and 1 indicating a more calcified suture with gray levels closer to palatal bone.[2(p434)] As such, future CBCT studies may choose to correlate the MSDR method to determine the orthopedic potential of an expansion appliance.

The presence of a floor effect significantly affected the statistical analysis of the BBW, which the authors chose to deal with via transformation and non-parametric testing. A possible alternative option may have been re-coding the raw measurements into categorical groups based on voxel size (i.e. 0.3mm); however, this was not possible due to time constraints. Methodological alternatives may have been segmental analysis or superimposition of the volume rendering with color mapping.[2(p194)]

CBCT offers distinct advantages over 2-dimensional imaging methods because it allows for study and analysis of 3-dimensional realities. That said, the creation of a 2D image from a 3D volume seems paradoxical, especially knowing that volumetric segmentation is available as method. Linear measurement reduces the ROI to a representative slice and it can be argued that linear measurements do not make use of the full potential of the CBCT like a volumetric measurement would. Moreover, the cumulative error associated with the use of landmarks to create orientation planes and establish reference points over time was not considered.[161]

Due to the *as low as reasonably achievable* (ALARA) protocol for radiographs,[35,39] many CBCTs are taken with a FOV and exposure parameters that allow the clinician to use a single volume to create a panorex, ceph, and generally assess the baseline skeletal and dental presentation of the patient. That said, the use of a full FOV CBCT to assess buccal alveolar bone has been highly criticized since the spatial resolution of the scan often exceeds the dimension of the cortical bone.[32,36,42] Recently Park et al. discussed the concept of *as low as diagnostically acceptable* (ALADA), where the region of interest is imaged without loss of adequate image quality. Inherently these devices elicit false negative measurements of approximately 0.3-0.6mm (depending on the voxel size and exposure parameters)[37,48,147] in addition to the measurement error of the examiner. This indicates a need to reduce the FOV to obtain diagnostically relevant imaging. Future studies may consider reducing the FOV and voxel size to ascertain the overlying width and height of alveolar bone more closely.

4.3.2.2 Methodological Limitations of Photo Analysis

The soft tissue evaluation was not related to a clinical examination and was reliant on visual assessment alone. Since many of the parameters evaluated were highly prevalent in the sample, and inter-rater reliability was low to moderate for all parameters, a precise and careful examination of the gingival soft tissues using periodontal probing[103] is necessary to ascertain a diagnosis, guide treatment, monitor patients during treatment and re-evaluate during retention.[107] Within the limits of the present study, it can be concluded that a visual inspection is not a reproducible method for soft tissue diagnosis. Moreover, the evaluation of soft tissue risk factors should not be generalized to the entire mouth, but individualized to each tooth, or group of teeth. Future studies should consider the use to a clinical periodontal evaluation in concert with imaging to truly correlate soft tissue and radiographic findings.

4.4 Conclusions

The first objective of this study was to evaluate periodontal (buccal alveolar and soft tissue) changes associated with orthodontic expansion using both a tooth borne rapid maxillary expander (Hyrax) and Damon® appliances. Overall, the teeth undergoing expansion demonstrated a decrease in BBW and an increase in dehiscence formation over time. Because such deterioration of underlying periodontal structures might not be reflected in a 1:1 relationship with the clinical presentation of the soft tissues, orthodontists may underestimate the creation of irreversible hard-tissue changes that accompany expansion treatment.[47] Since patients with posterior crossbites have characteristic hypoplastic maxillae with a higher prevalence of dehiscence and fenestration than patients without crossbites[53] and thin alveolar bone has been found to be more susceptible to bone dehiscence (which increases their risk of GR),[26,76,133,134] the physiologic boundary of the alveolus should be considered prior to prescribing orthopedic or dental expansion. This is in addition to the limitations of the appliance chosen and the soft tissue factors related to GR.

This study found that as teeth are moved in a buccal direction, the BBW decreases overlying the expanded teeth, irrespective of appliance type. This was especially true 6mm apical to the CEJ in maxillary molars.

The second objective of this study was to compare alveolar changes between patients treated with Hyrax and Damon® appliances. Contrary to the Damon® hypothesis that orthodontic forces within an optimal zone are able to mediate bone development,[8(p997.e118),167] no appreciable difference was observed between patients treated with Damon® compared to Hyrax appliances, with the exception of the maxillary first molars at debond. The presence of bone loss 3mm apical to the maxillary first molar CEJ and the onset of severe dehiscence 6mm apical to the CEJ was significantly higher in the Hyrax group, albeit not clinically significant at 3mm. It is important to note that while there were significant differences at debond, the groups were not significantly different at follow up.

Lastly, we correlated soft tissue and alveolar changes with the onset or worsening of GR after orthodontic expansion treatment in patients with MTD. This study found that when the width of keratinized tissue is <2mm, the patient is at least 12 times more likely to show at least one site with GR, than those with KTw ≥2mm. Periodontal risk factors related to the development of

GR, including the width of the keratinized tissue should be assessed to determine the risk of developing GR with expansion treatment. Therefore, taking this into consideration in a comprehensive treatment plan may reduce the risk of future attachment loss, especially when teeth are moved in a labiolingual/buccolingual direction.

Since the literature highlights the importance of other RFs, such as the presence of dehiscence, tissue biotype and the presence of bacteria-induced inflammation, future clinical trials should include periodontal charting in their records to avoid the methodological limitations associated with photo-analysis. Additionally, since there is a known relationship between age and onset of GR, future studies should examine the relationship between expansion treatment and periodontal changes over a longer-term span, as well as with larger sample sizes to reduce the probability of type II error. Other known risk factors such as tooth brushing, diet, smoking, etc. may also be important in determining the differences in GR. With respect to the CBCT measurements, a color overlay may be useful to determine the tooth angulation and assess both alveolar and root angulation changes with respect to possible relapse along with reducing the floor effect observed in this study. Finally, consideration for a smaller field of view CBCT to reduce the radiation exposure, noise, scatter and ultimately spatial resolution would be helpful in the examination of periodontal changes associated with expansion.

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