

# Occlusal bite force measurements in different malocclusions



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“In your light, I learn how to love.  
In your beauty, how to make poems.  
You dance inside my chest,  
Where no one seen you,  
But sometimes I do,  
And that sight becomes this art.”

Rumi

## Dedication

For dad.

“The father touched Siddhartha’s shoulder.

‘You will go into the forest’, he said, ‘and become a samana.

If you find bliss in the forest, come back and teach it to me.

If you find disillusionment, come back,

and we shall again offer sacrifices to the gods together.

Now go.” - from Siddhartha

by Herman Hesse.

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My mother: Ghyroonnisha Dawjee, the light that remains.

My siblings Haji, Muhammad and Zareena: for encouragement inspiration and love.

## Declaration

I, Maryam Mohamed Dawjee, declare that the dissertation I am herewith submitting for the degree MSc (Odont) at the University of Pretoria is my own work and has not previously been submitted for any other degree at any other university.

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M.M Dawjee

15-08-2014

## Executive Summary

Dento-facial anomalies have been diagnosed for centuries. The diagnosis or observation thereof is reported to date back to the Fifth Century AD where Hippocrates observed and noted numerous craniofacial deformities (Moyers, 1988)

Through a complex process of reception, selection and categorization- the human form particularly the cranium, jaws and related soft tissue structures, have been studied and categorized over the years (Moyers, 1988). Dockrell (1952) focused on the aetiology of malocclusion and through extensive work on the topic, established an equation for the improved identification of aetiology of malocclusion. This equation essentially communicates that a causative factor will act at a specified time, on an identified tissue, to produce particular results. Thus resulting in a deviation from the accepted “normal” relationship. This will result in the development of an archetypally defined, classifiable malocclusion. These so termed causative factors as elicited to in the equation play a pivotal role in the outcomes or resultant malocclusions (Dockrell, 1952).

The plasticity in aetiology and resultant Skeletal or dental positional discrepancy should not be overlooked. Multiple factors may act together stabilising each other thus lessening the severity of the expected effect. This research project aims to identify the potential of occlusal bite forces as a causative factor in the “orthodontic equation” and shed light on timing as well as resulting Skeletal and growth effects of variance in occlusal force. Occlusal force is a combined and quantifiable measurement of contributory muscle strength.

Through this study a comparative analysis of the occlusal force will be carried out amongst subjects exhibiting differences in age, gender, race, and growth pattern. This study aims to elucidate the influence of occlusal forces and masticatory muscle strength in relation to the resulting craniofacial development.

Data was collected from patients exhibiting various dental classes namely Class I, Class II and Class III dental and Skeletal patterns. Bite force strength of patients in primary, mixed and permanent dentition was measured using an occlusal force meter. The occlusal force meter was sourced from a Japanese based company, Nagano Keiki Co. LTD. The readings were done in Kilo newton, which is the standard international (SI) measurement of force.

The study population consisted of 180 male and 180 female subjects.

This group was split into Skeletal Class I, II and III subjects and then further divided into the three directions of growth (Horizontal, normal and Vertical).

The relationship of bite force to Skeletal Class and growth direction was analysed using mean readings from five points of reference on each subject. Analysis of covariance was carried out on the data set with a mean age of 15,33 years (covariant). There was a significant difference found in the bite forces between growth direction and Skeletal Class  $P < 0,05$ . There was also an interaction found between Skeletal classes and direction of growth. These findings are supportive to the hypothesis that a relationship exists between bite force and malocclusion.

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## Chapter 1: Defining the research problem

General dental practitioners and specialists diagnose various types of malocclusions daily. For the purpose of scientific study and improved methods of diagnosis and treatment, it is of paramount importance that the aetiological factors of malocclusion be further investigated and studied. Aetiological factors that are influential and pertinent to malocclusion can be both environmental and genetic entities. These factors may impact on growth and development and need to be identified and intensely studied to enrich our understanding. In the investigation of any such deviation or alteration from the normal parameters of growth and development - hereto referred to as the aetiology of malocclusion - it can be said that the ideal method of determining the aetiology, is to centre the discussion or investigation on the tissue site that is primarily involved in the resulting deviation (Dockrell, 1952).

The role of adaptability and variability amongst subjects not is overlooked as a result of a narrow focus on the primary tissues (Moyers, 1988). The adaptive ability of any bodily tissue site refers to the innate capacity to morph. This allows an improved or satisfactory function, whilst still being susceptible to any irregularities in force or environment. Variability in the human form exists on both a physiological and psychological frame.

The adaptability found in relation to the psychological frame can be explained further by exploring the idea of partiality. The concept of aesthetics is fluid in a sense that the perception of an aesthetic or un-aesthetic quality is subjective. There exists a certain degree of perceptive variability amongst individuals. This is due to psychological, cultural, evolutionary and social influence in which disparities are present. The role of subjectivity in malocclusion and aesthetic concern ipso facto warrants or justifies the capacity for alteration in the taxonomy of the aetiological factors of malocclusion. “One should always permit lateral, open-ended study in the consideration of variability and adaptability” (Moyers, 1988). It is as a result of this kind of study and report, that the identification of normal has been established and documented. This has henceforth permitted any deviation or alteration from the standards formerly identified as “Normal” to be acknowledged.

As advised by Dockrell (1952), Mayne (1969), Harvold (1979) and Moore (1969) the aetiology of malocclusion is best studied from clinical classification and then regressing to potential causes of a current situation. The consequences of malocclusion are not solely limited a poor facial appearance, as it may often result in additional and more debilitating functional obstacles. These can be expressed as poor oral hygiene leading to disease, functional alterations in speech, respiration, digestion, and temporo-mandibular joint function. (Moyers, 1988)

Various malocclusions have been defined and classified and numerous causal factors have been studied and documented. The classifications of aetiological factors have been defined by White, Gardiner and Graber (Bhalajhi, 2012).

Table 1 establishes the aetiological classification according to Moyers. There are seven categories based on the kind of causative factor. Moyers relates the causative factor to a tissue site, the consequence of this is that any tissue site affected may be susceptible to change and thus result in a malocclusion (Moyers, 1988).

Table 1: Moyers classification of aetiology (Bhalajhi, 2012)

1: Hereditary	Neuromuscular Bone Teeth Soft tissue
2: Developmental defects of unknown origin	
3: Trauma	Prenatal trauma and birth injuries Postnatal trauma
4. Physical agents	Premature extraction of primary teeth. Nature of food
5. Habits	Thumb sucking and digit sucking Tongue thrusting Lip sucking and lip biting Posture Nail biting Other habits
6. Diseases	Systemic diseases Endocrine disorders Local diseases: nasopharyngeal and respiratory. Gingival and periodontal. Tumours and caries.
7. Malnutrition	

White and Gardiner however classify aetiological factors a little differently, attention is paid to the timing of growth and development. Abnormalities are then related to various time frames, as displayed in Table 2.

**Table 2: White and Gardiner's classification of Aetiology (Bhalajhi, 2012)**

<p>White and Gardiner's Classification of aetiology of malocclusion</p> <p>A. Dental base abnormalities</p> <ol style="list-style-type: none"><li>1. Anterior-posterior mal-relationship</li><li>2. Vertical mal-relationship</li><li>3. Lateral mal-relationship</li><li>4. Disproportion of size between teeth and basal bone</li><li>5. Congenital abnormalities</li></ol> <p>B. Pre-eruption abnormalities</p> <ol style="list-style-type: none"><li>1. Abnormalities in position of developing tooth germ</li><li>2. Missing teeth</li><li>3. Supernumerary teeth and teeth abnormal in form</li><li>4. Prolonged retention of deciduous teeth</li><li>5. Large labial frenum</li><li>6. Traumatic injury</li></ol> <p>C. Post-eruption abnormalities</p> <ol style="list-style-type: none"><li>1. Muscular – active, rest, sucking habits, path of closure</li><li>2. Premature loss of deciduous teeth</li><li>3. Extraction of permanent teeth</li></ol>
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From this information it is feasible to comprehend the aetiological factors, which have thus far been identified, studied and theorised. Dockrell's malocclusion delineates a pattern of aetiology resulting in the manifestation of malocclusion. It can be briefly explained as the identification of a cause, time, tissue and the subsequent result. A cause acts at a time, on a tissue to present a result (Dockrell, 1952). However it can be alleged the influence of bite force and its impact in Dockrell's malocclusion equation necessitates added investigation.

The significance of the concept of growth and the need to persistently define, clarify and establish parameters of evaluation forms the base of any desired treatment outcome. "Growth is the raw material of orthodontic treatments" (Moyers, 1988) and the understanding of growth needs to be sought across short-term (selective individual) alterations to a more extensive period of alteration impacting genetic and evolutionary change.

An integral component of the assessment and comprehension of bite forces, malocclusion and the associated relationship between growth and force is to understand and address the stages of growth and development that propagate the establishment of the occlusion.

The development of the occlusion involves the eruption of teeth into the oral cavity and the formation of inter and intra arch relationships between the teeth. However an important aspect of development that should not be overlooked is the process involved during the development of the dentition.

Occlusion is defined as, “any contact between the incising or masticating surfaces of the maxillary and mandibular teeth”. Simply put, it is the way the teeth of the maxilla and the mandible come together or meet each other when a patient closes their mouth (Bhalajhi, 2012).

The eruption of teeth into the oral cavity and the development of inter and intra-arch relationships between the teeth, is a process that is propagated by an individuals’ inimitable genetic make-up and is therefore very idiosyncratic. Furthermore, the eruptive process is affected and normalized by continuously changing and dynamic environmental factors. There are several anatomical components that contribute to the development of teeth and the formation of the occlusion. The teeth, periodontal ligament, maxillary and mandibular processes, temporo-mandibular joint (TMJ), muscles and lastly the nervous system all have a purpose and a function (Moyers, 1988).

Occlusion comprises of two vital components. Firstly the static component which relates to the establishment of the form and alignment. The word static gives inference to an element being still i.e.

without movement or change. Thus the inter-arch and intra-arch relationship of the teeth, as well as the relationship between the teeth and the supporting bone can be classified as the static component of occlusion (English et al., 2009).

Secondly there is the dynamic component. The narrative term dynamic gives reference to the idea that an element is in relentless change. Dynamic occlusion refers to the practical elements involved in occlusion, such as the teeth, TMJ as well as the neuromuscular and nutritive components. Peltomäki and co-workers (2009) are of the opinion that occlusion is exclusively a dynamic structure, as the dental arch dimensions undergo diverse alterations and there is nothing that remains static within this developmental process.

Ideal occlusion is said to be the position of teeth within the specific arches as well as the association between teeth in opposing arches. This ideal provides for the finest function and harmony between soft and hard tissue components and aesthetic appearance (Knösel & Jung, 2011).

When assessing the occlusal forces of an individual, the teeth are not the only contributors to possible alteration or deviation in the normal accepted development.

The muscles of mastication play an integrated role in the forces produce, which may bring about developmental change or departure from the growth form studied and accepted as “normal”.

Mastication is a complex and integrated process involving numerous motor functions. It ensues through a series of actions, all of which are co-ordinated by complex neural structures and carried out by several anatomic edifices and physiologic procedures (Yamamura et al., 2002). The act of mastication has been defined as the inaugural stage in the digestive process (Ganong & Barrett, 2005). Through mastication the food is fragmented and moistened with saliva.

Saliva provides moisture not only for the registration of taste by the taste buds in the oral cavity, but also for the formation of a slippery bolus or ball, which in turn facilitates the deglutination process (Pedersen et al., 2002). The swallowed food undergoes additional breakdown and absorption as it passes throughout the digestive tract.

The physical act of mastication is multifarious in its nature. Mastication transpires through an interface between the facial, elevator, and supra-hyoid muscles and of course the tongue (Van der Bilt et al., 2006). These muscles work hand in hand with other accessory elements such as saliva and the teeth to produce rhythmic movements of the jaw.

The significance of the teeth within the masticatory process is that they offer the surface area for the crushing or break down of food.

The quantity of teeth present affects the ability of surface area available and will be influential in the extent of the chewing process (Hannam et al., 1977). The volume of surface area present is also instrumental to the quantity of activity necessary by the muscles and other related components in order to accomplish effective bolus materialisation (Olthoff & van der Bilt, 2012). A diminution in surface area as a consequence of missing teeth or poor occlusion may force the muscle activity to be increased.

The amount of masticatory cycles in total may also be increased to ensure that there is satisfactory destruction of the food particles to expedite swallowing (Bradley, 1981). The intensity of the bite force, which is established by various muscles and structures in the oro-facial area, also contributes to archetype mastication. These masticatory forces are ordered and governed by the motor neural control of the muscles around the mandible. This control is essential to bring about the rhythmic jaw movements that are indispensable to the masticatory process.

An equally important neuronal contributor is the sensory component of neuronal activity. The sensory reaction is stimulated by the insertion of food particles into the mouth. It is dependent on the individual characteristics and type of food particles such as fat and water content as well as the texture.

These characteristics contribute to the nature of the rhythmic movements (Van der Bilt et al., 1995). The tongue, cheeks and saliva are all supplementary to the masticatory procedure. The tongue and cheeks enable the movement of food particles around the mouth and the aforementioned saliva forms an integral part of taste, containing imperative enzymes whilst also providing moisture for bolus formation (Van der Bilt et al., 1995).

The anatomical structures involved in the masticatory process, the physiological and neuronal control and other contributory factors will all be dissected and discussed further.

Through the comprehension of aetiological factors, occlusal development, mastication and influence of oro-facial musculature and skeletal growth, clarity in understanding the complex inter-connected system is possible. With this knowledge as a foundation it is possible to predict the following: A Class II malocclusion with a deep bite, may exhibit an increased occlusal bite force, conversely an open bite malocclusion, exhibiting excessive Vertical growth, may have a

decrease in the strength of the occlusal bite force. One would expect males to have a greater occlusal bite force than females. This notion is an extraction from suppositions founded on logical cognition of the relationship within the musculoskeletal system of the oro-facial region.

The investigation of these relationships and identification of occlusal forces in various malocclusions done in this study will enable further understanding of aetiology, manifestation and treatment of malocclusion. Furthermore it could propagate investigation into the influence of occlusal force on development of malocclusion and thus allow further study relative to early detection and alteration of deviation in the magnitude of occlusal force.

## Chapter 2: Literature review and motivation

In order to identify and establish the aetiology of malocclusions and the relationship of bite forces in the manifestation of different growth patterns and skeletal classification, it is important to understand the basic of occlusal development.

### Embryological development of teeth

The course of tooth development is a process regulated by genetics, involving collaboration between the oral epithelium and the underlying mesenchymal tissues (Ohshima, 2008).

Teeth develop from a horseshoe band of tissue, which has been assigned the term: dental lamina. The permanent teeth that will supersede primary predecessors are derived from the lingual annexe of this dental lamina. Distal proliferation of this dental lamina expedites the development of the permanent posterior teeth i.e. permanent molars. The permanent molars (first, second and third) have no primary or deciduous precursors (Bhalajhi, 1998).

The proliferation of the dental lamina proceeds through three progressions to facilitate the formation of the teeth. These stages are termed, in order of manifestation as the Bud stage, the Cap stage and the Bell stage as illustrated in Figure 1 (Moyers, 1988):

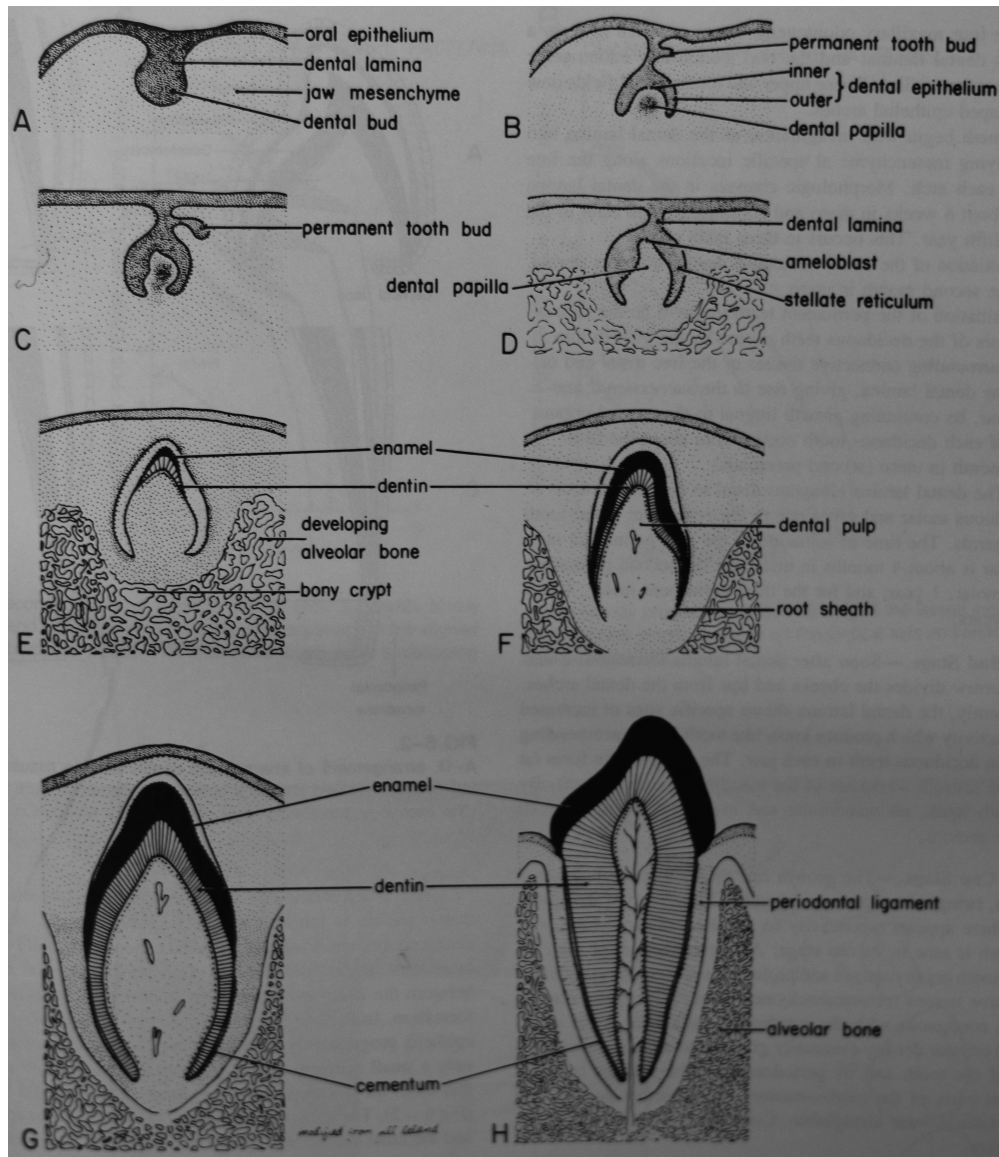


Figure 1: Early stages of development (Moyers, 1988)

- **The Bud Stage:** The proliferation and condensation of the mesenchymal cells that occurs as a consequence of amplified mitotic activity will culminate in the formation of ten tooth buds (Bhalajhi, 1998). Every tooth bud links to a deciduous tooth. The oral epithelium grows inwards and forms bud shaped tooth germs (Moyers, 1988).
- **The Cap Stage:** arises after the initial formation of the buds or bud shaped tooth germs. Mesenchymal tissue promptly starts to condense around the bud. The dental papilla and dental sac become more distinctive. Cellular differentiation transpiring during this stage results in the formation of the enamel organ, dental papilla and dental follicles.
- **The Bell Stage:** the tooth germ proceeds to enlarge and commences the development a concavity and the tooth germ now resembles a bell. Enlargement transpires as an outcome of central cells secreting an acidic muco-polysaccharide, this secretion instigates the upsurge of water in the extra-cellular spaces. These overextended cells at the centre of the bell form the stellate reticulum and supplementary differentiation of cells spearheads the formation of tooth specific cells.

- Cells lining the dental papilla form the inner dental epithelium; cells lining the edge of the tooth germ form the outer dental epithelium (Moyers, 1988).

Odontoblasts are the cells that are accountable for the development of dentine, the differentiation of these cells originate from the cells of the inner dental epithelium. The start of enamel development, from the differentiation of cells into ameloblasts, is reliant on the magnitude of dentine deposition (Zheng *et al*, 2013). Only once an appropriate quantity of dentine has been positioned can the ameloblasts commence to form the enamel. The Cervical loop area is the region where the outer enamel epithelia and the inner enamel epithelia progressively shroud the dental papilla leaving a slight foramen at the base. The dental sac will form the periodontal fibres. These fibres bridge the gap between the root surface of the tooth and the alveolar bone (Friel, 1954).

## Stages of development

### Pre dental period

The pre dental period ranges from birth to six months. There are no teeth existing in the mouth through this stage. The alveolar processes are referred to as “Gum Pads” (Sillman, 1938). These horseshoe shaped gum pads are firm, pink in colour and enclosed by a fibrous periosteum, the dimension of which can be influenced by a variety of factors.

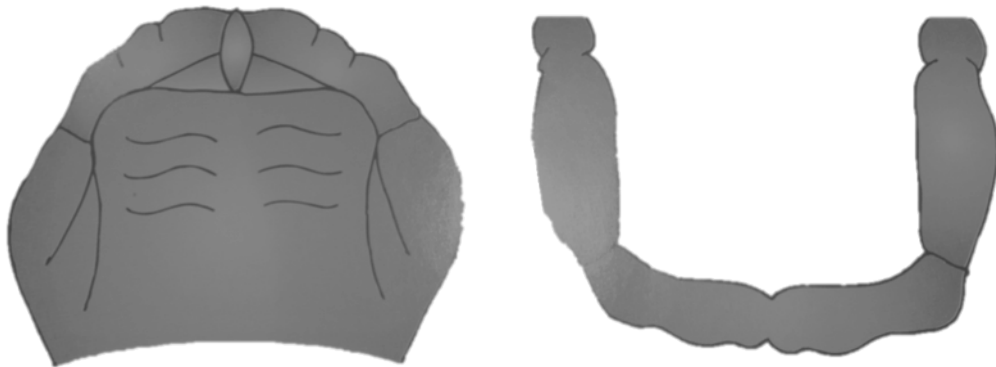


Figure 2: A) maxillary gum pad and B) mandibular gum pad (Bhalajhi 2012)

The inter-arch relationship i.e. the relationship between the maxillary and mandibular gum pad **Figure 2**, can be established at an early age. This can be done by the assessment of the lateral sulci.

The lateral sulci, is a groove that exists between the canine segment and first molar segment on the pre-dental gum pad. The mandibular sulci should be distal to the maxillary sulci (Friel, 1954).

An anterior open bite **Figure 3** exists throughout the pre-dental phase the manifestation of this open bite assists the suckling practise of an infant. There is nevertheless still contact between the maxillary and mandibular gum pads in the molar region. An over-jet relationship can also be acknowledged. It is uniform and spreads from anterior to posterior. This indicates that the maxillary gum pad is larger than the mandibular gum pad, both in width and in length. This is in harmony with the cranio-caudal gradient of growth. (Sillman, 1938)

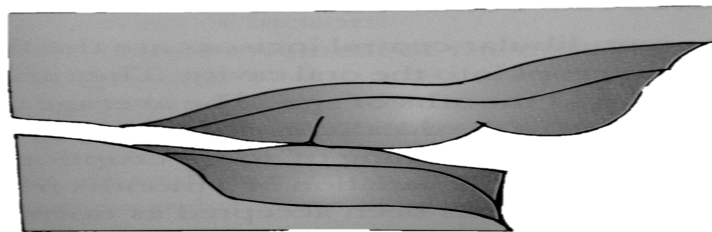


Figure 3: Gum pad relationship (Bhalajhi, 2012)

In rare instances teeth may appear during this stage of growth. Teeth, which are present in the oral cavity at the time of parturition, are referred to as natal teeth. Neo-natal teeth however erupt during the first post-partum month and are found in the anterior mandible. The natal or neo-natal teeth are almost always in the mandible and Moyers (1988)

advises that while the manifestation of these teeth may cause trauma and pain during breastfeeding, it is crucial that they are established as supernumerary teeth before they are removed.

During the first year post-partum, these gum pads endure substantial growth and development. This transformation in size permits for the conception of space indispensable to the accommodation of the deciduous incisors (Friel, 1954).

### Pre-emergent eruption

Pre-emergent eruption concerns the movement of teeth within the alveolar bone. This process cannot be perceived clinically. The process arises when the roots of the teeth begin forming. Pre-emergent eruption occurs in both the primary and secondary tooth germs, within their respective skeletal components i.e. in both the maxilla and the mandible. During this stage of the formation process, the buds remain static within the alveolar bone (Proffit & Frazier-Bowers, 2009).

Movement of the developing bud is initiated simultaneously with the initiation of root formation (Proffit & Frazier-Bowers, 2009). The eruptive process cannot occur without the establishment of an eruptive pathway. The formation of this pathway exhibits differences during the primary dentition and the permanent dentition (English et al., 2009).

Resorption of the alveolar bone is required in order for the eruptive process to occur in the primary dentition, whilst in the permanent dentition, the resorption of the roots of the deciduous teeth will be observed (Friel, 1954). During a study conducted, in which the primary aim was to assess the treatment modalities of jaw fractures, the ligation of the mandible resulted in ligation of developing tooth buds. As a result of the tooth bud being ligated during fracture treatment, the force caused by the formation of the roots was not present. However a clear eruption pathway in the alveolar bone was still observed (Proffit & Frazier-Bowers, 2009). From this as well as similar more pre-meditated studies conducted on dogs, Proffit and Frazier-Bowers (2009) deduced that, “the rate and direction of resorption to clear the eruption path, and not the force developed to move the tooth, controls the pre-emergent eruption” (Proffit & Frazier-Bowers, 2009).

Crowding in the maxillary and mandibular teeth is often seen during the pre-eruptive stage (El-Nofely et al., 1989). The Skeletal growth of both the maxilla and mandible will increase the space available, allowing for the correct positioning of the tooth germs. This permits the ideal eruption and positioning to occur (El-Nofely et al., 1989).

### Emergence

The term 'emergence' is used when the cusps of the teeth begin to penetrate the gingiva into the oral cavity. Emergence occurs when 75% of the root has been developed (English et al., 2009). The eruption process is believed to follow the circadian rhythm of the human body, in a similar pattern as that of the secretion of both growth and thyroid hormones (English et al., 2009). There have been recent reports that an alteration in the parathyroid receptor 1 gene results in an alteration in the eruptive process (Frazier-Bowers & Rhoads, 2012). The role that hormones play in the eruption of teeth can also be noted in various other genetic and medical conditions, which may exhibit vicissitudes in the timing or sequence of tooth eruption (Proffit & Frazier-Bowers, 2009). Conditions that may influence the eruption pattern, due to alterations in the rate of bone metabolism thus affecting the formation of an eruption pathway are Osteopetrosis (Frazier-Bowers & Rhoads, 2012), Cleidocranial Dysostosis, Hypothyroidism, and Hypopituitarism (Proffit & Frazier-Bowers, 2009).

There are numerous theories surrounding normal eruption of teeth however the precise mechanism of eruption is still debatable (Frazier-Bowers & Rhoads, 2012). The following theories concerning eruptive forces have been proposed.

**Root growth:** There is an increase in the length of the roots in an apical direction, this sends a force directed axially that causes the eruption of teeth. This theory postulates that the increase in the length of the root causes pressure in the alveolar bone surrounding the apices of the teeth. This pressure produces osteogenesis (bone deposition) around the apices and osteoclast activity around the crowns of the teeth (Frazier-Bowers & Rhoads, 2012). This theory is not supported, as it has been noted that the timing of growth and development of the root does in fact not coincide with the eruption of the tooth. The eruption process happens at a more rapid pace.

**Hydrostatic pressure:** An increase in the blood supply and water retention within the periodontal ligament is believed to occur during the eruptive process. The extra cellular matrix around the apex of the developing tooth bud has the capacity to increase in volume by 30%-50%. This study is supported by human studies, whereby vasodilators were injected locally which precipitated tooth eruption (Frazier-Bowers & Rhoads, 2012).

**Bone remodelling:** The dental follicle that surrounds the tooth plays a crucial role in the eruption of teeth consequently this theory has been referred to as the “Dental follicle theory” (Frazier-Bowers & Rhoads, 2012). The dental follicle provides the “environment and chemo-attractants” (Frazier-Bowers & Rhoads, 2012) for odontoclastic differentiation from monocytes. This theory is strongly supported by experiments in which the dental follicle was removed and teeth failed to erupt as a consequence (Frazier-Bowers & Rhoads, 2012).

**Periodontal ligament traction:** experiments have also been carried out to establish the contributory role of the fibroblasts (found in the periodontal ligament). It is believed, that the capacity of the fibroblasts to contract precedes the eruption of teeth (Bhalajhi, 1998).

### Post-emergent eruption

This is the eruption process that occurs when the teeth emerge into the oral cavity and continues until the point where teeth come into contact with the teeth in the opposing arches. Teeth do not however stop erupting when contacting an antagonist within the opposing arch. The teeth continue to erupt, in a manner that collaborates with the Vertical growth of the face. As teeth erupt they stimulate additional growth of the alveolar bone wherein they are embedded (Moorrees, 1959). This occurs concurrently with eruption. If teeth are extracted, resorption of

the alveolar bone is observed. If teeth are absent, then the alveolar bone will not develop in that area (English et al., 2009).

The post emergent eruptive movements have been divided in to four stages (Proffit & Frazier-Bowers, 2009)

**Pre-functional spurt:** Movement of the tooth from its initial emergence up till the point of contact within the occlusal plane.

**Juvenile equilibrium:** During this stage both jaw growth and eruptive movements are quite slow.

**Adolescent eruptive spurt:** An increase in overall growth is coupled by further eruption of the teeth in order to maintain the established occlusal contacts.

**Adult equilibrium:** Further growth of the jaws makes it imperative for the first permanent molars to erupt approximately one centimetre more than when they first emerged. Adult eruptive movements are also observed at the loss of a tooth, where the over-eruption of its corresponding antagonist in the opposite arch can be seen.

Post eruptive movements are the movements of a tooth that transpire to accommodate the growth and to compensate for the occlusal wear that may result from functional and para-functional movements (Bhalajhi, 1998). The loss of teeth may also result in a post eruptive movement ensuing in the neighbouring teeth as well as the correlating antagonist in the opposing arch. As the jaws grow, the sockets of the teeth undergo a remodelling process, which allows the position of the teeth to

be readapted. This is critical to note as the functional position of the teeth are maintained in this manner (Friel, 1954).

Interproximal wear can lead to a mesial drift of the teeth. Teeth will drift mesially to sustain adjacent contact position. The reason teeth have a tendency to drift in a mesial direction is twofold. Firstly, it is due to the natural mesial inclination of the teeth and secondly, the contraction of the transeptal fibres will pull teeth together and keeping them in contact with each other. (Bhalajhi, 1998)

#### Deciduous dentition period

The first teeth to erupt into the oral cavity are the mandibular incisors. They can be seen erupting at approximately six to seven months of age. The sequence of eruption (Table 3) is central incisors, lateral incisors, first molars, canines and second molars. A variation of about 3 months from the expected age of eruption is considered within normal range, as the eruption sequence is a highly unique and capricious process (Moyers, 1988):

**Table 3: Eruption timing (Moyers, 1988)**

Lower central incisors	7months
Upper central incisors	10 months
Lateral incisors	12 months
1 <sup>st</sup> molars	16 months
Canines	20 months
2 <sup>nd</sup> molars	28 months

Spacing is a typical feature of the primary dentition. The physiological or developmental spacing, are spaces that can be observed both mesial and distal to the primary incisors. These will allow for the accommodation of much larger permanent incisors upon eruption. A consequence of the absence of the physiological spaces in the primary dentition is that crowding may result in the permanent anterior segments (El-Nofely et al., 1989). Primate spaces are the largest spaces that can be distinguished.

### Mixed dentition period

The mixed dentition period begins with the eruption of the first permanent molar, which approximately occurs at the age of 6 years. There are three phases of development:

### The first transitional period:

The eruption of the first permanent molars and the exfoliation of the primary incisors followed by eruption of permanent incisors occur during this period. The shedding of primary teeth occurs due to the resorption of roots by the erupting permanent successors.

Odontoclasts resorb the deciduous dental hard tissues, namely the cementum, dentine and enamel and can be found on the outside or external surfaces of the roots (Kondo et al., 2009).

### The first permanent molars

The mandibular first molars begin to erupt at approximately six years. The eruption and positioning of the first permanent molars are guided by the relationship of the distal surfaces of the maxillary and mandibular primary second molars. This has been termed the terminal plane. The terminal plane of the deciduous second molars will therefore play a vital role in the guidance of the erupting first permanent molars (English et al., 2009).

There are three kinds of terminal plane relations that exist between the maxillary and mandibular second primary molars.

*Flush terminal plane:*

The distal surfaces of the upper and lower deciduous second molars lie in a Vertical line and this is a normal standard feature of the primary dentition (**Figure 4**). A flush terminal plane permits the permanent first molars to lie, flush or cusp to cusp with each other upon their eruption.

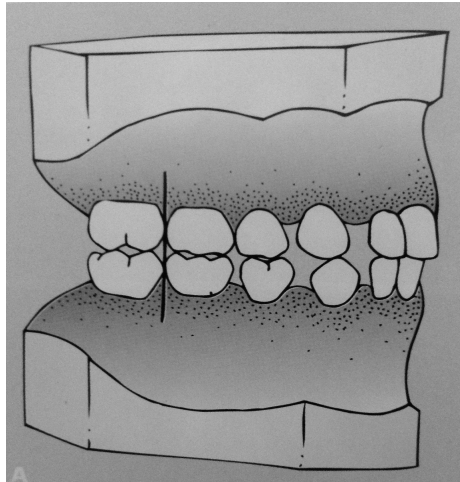


Figure 4: Flush terminal plane (Bhalajhi, 2012)

*Mesial step terminal plane:*

The primary second molars exhibit an Angle's Class I relationship. Thus the permanent first molars or 6's will erupt directly in the desired Angle Class I or Class III position (**Figure 5 and 7**). The downfall of this is that due to the continued growth of the mandible and the utilisation of spaces i.e. leeway and primate could result in an unfavourable Class III positioning of the first permanent molars.

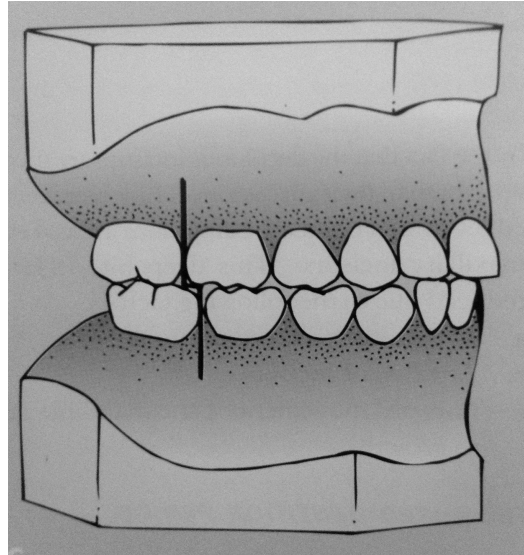


Figure 5: Mesial step terminal plane (Bhalajhi, 2012)

*Distal step terminal plane:*

In contrast to the mesial step, the distal surface of the mandibular second primary molar occludes distally to the maxillary second primary molar (Figure 6 and 7), thus the permanent molars will erupt in an Angles Class II position.

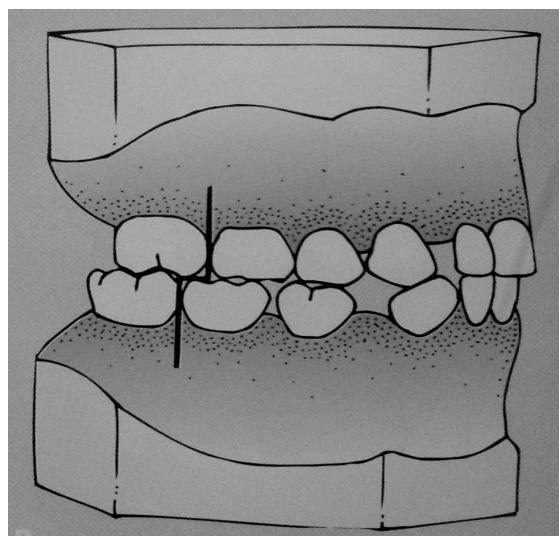


Figure 6: Distal step terminal plane (Bhalajhi, 2012)

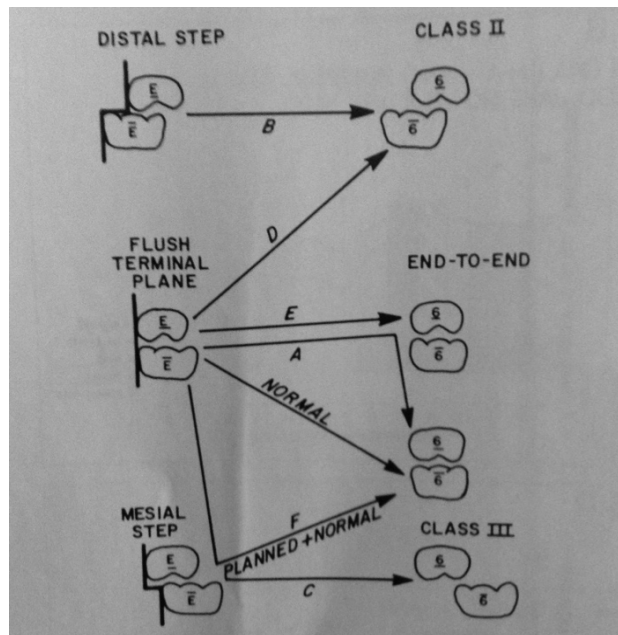


Figure 7: Patterns of transitional occlusion (Moyers, 1988)

Leeway space (Clinch, 1951) is the mesio-distal difference in size, between the primary first and second molar and the permanent first and second premolar. This space can be approximated to 0.9mm of excess per arch in the maxilla and 1.7mm of excess per arch in the mandible.

Upon exfoliation of the deciduous molars and the eruption of the permanent premolars the leeway space plays is then taken up by the mesial drift of the permanent molars, thus enabling the establishment of a Class I molar relationship (Clinch, 1951).

### The permanent incisors

The mandibular primary incisors are the first to exfoliate. A difference in the size exists between the primary incisors and the permanent incisors. This is referred to as the incisor liability. Incisor liability is estimated at around 7mm in the maxillary arch and 5mm in the mandibular arch. The erupting incisors are contained in their respective arches by utilisation of space. Space is created in one or several of the following manners: an increase in the arch width, inter-dental spaces, labial tipping of the erupting permanent incisors, and lastly through distal shifting of the primary canines (Moyers, 1988).

### Inter-transitional period:

This is a stable period during which there is a combination of primary and permanent teeth in the mouth. This phase is accompanied by a negligible amount of alteration.

### The second transitional period:

This is the phase in which the primary molars are replaced by the permanent premolars and all leeway space is utilized. The primary canines are also replaced by the permanent successors. This completes the transition from primary to permanent dentition. Due to the uptake of leeway spaces that occurs during this transitional period, a decrease in the arch length can be expected (English et al., 2009).

A larger leeway space in the mandible exists, which allows for a larger decrease in the mandibular arch length as opposed to that of the maxilla.

According to Moorrees as noted by English, Peltomäki and Pham-Litschel (2009) approximately 2-3mm of shortening in the mandibular arch length is observed in the transition from full primary dentition to permanent dentition.

According to Moyers (1988) the development of a favourable relationship in the canine to premolar region is dependent on the following factors:

- A complementary eruption sequence.
- The size of the tooth has to be relative to the space available for it to be positioned ideally within the arch.
- Ideal molar relationship with minimal loss of space caused by mesial drift of molars.
- The bucco-lingual relationship of the maxillary to mandibular alveolar process has to be in an advantageous position.

## Permanent dentition period

The permanent dentition starts to develop in the jaws soon after birth with an exception of the first permanent molars, which develop before birth. The permanent incisors follow a lingual to labial path of eruption due to their formative position (Moyers, 1988). Secondary or late crowding can be detected in the late teens or early twenties subsequent to the establishment of an occlusion. Secondary crowding is often observed in the mandibular arches. The cause of this crowding is said to be the mesial force of the erupting wisdom teeth, in addition to the maturation of the soft tissues that may trigger an increase in the pressure of the lips against the dentition (English et al., 2009).

The discrepancy in maxillary and mandibular growth may also have an effect on the presence of this type of crowding. The maxillary growth comes to cessation ahead of the mandible consequently trapping the mandibular incisors by the overlapping maxillary incisors. The mandible however continues to grow, resulting in a lingual inclination of the mandibular incisors, thus furthering the crowding observed in this area (English et al., 2009).

There are several factors contributing to abnormal eruption patterns, resulting in a disturbance in the development of an ideal occlusion

Delayed eruption being more frequently noted (English et al., 2009). Systemic factors that may contribute to the delay of tooth eruption often involve a disease process thus affecting an entire dentition as opposed to a single tooth. Bone metabolism, a fundamental process that is essential to the resorption process may be disturbed, accordingly affecting the eruption of the permanent or primary teeth (Moyers, 1988). “Primary Failure of tooth eruption” (English et al., 2009), occurs when a permanent tooth fails to move from its crypt in the alveolar process into its position in the oral cavity. Local factors associated with primary failure can be mechanical obstructions such as supernumerary teeth, fibrous tissue that doesn’t allow the erupting tooth to penetrate easily, ankylosed primary teeth, and sclerotic bone (Carpenter, 1978).

An array of forces contributes to the positioning of teeth within the oral cavity, the obvious of which are the eruptive and occlusal forces (Proffit & Frazier-Bowers, 2009). The force exerted by the cheeks, lips and tongue will affect the bucco-lingual positioning of the teeth. The fibres of the periodontal ligament stabilize teeth. These fibres prevent movement of the teeth by the forces exerted in the bucco-lingual direction (English et al., 2009). Light prolonged force, exerted by resting soft tissues, periodontal ligament and gingival fibres, as well as heavy, shorter acting masticatory forces also play a role in the movement and stability of teeth.

The anterior component of force must be differentiated from the mesial drift tendency of teeth. The anterior component force occurs as a result of muscle and inter-cuspatation of teeth, this force is counteracted by the mesial and distal contact points of the teeth in addition to the forces exerted by the lips and cheeks. Mesial drift is a display of a natural characteristic that teeth exhibit. (Van Beek, 1978)

### Static Occlusion

The relationship of the teeth within and between maxillary and mandibular arches whilst in contact and sedentary is referred to as the static occlusion. Andrews (1972), has listed six keys dictating what an ideal occlusion should comprise of. In his opinion, the manifestations of these elements were essential for normal occlusion.

### Ideal Occlusion

The concept of an ideal occlusion should be referred to as the unicorn of orthodontics-virtually unattainable. It should perhaps be used as the precedent, a blue print to treatment planning. As a result of the nature of human development and the ability to be influenced by nature, genetics and even economics each case may not be treatable to the point of achieving this “ideal” occlusion. Therefore there should be additional measurable components in place to establish the benchmark of treatment objectives. Function and aesthetics would be of primary importance when treating a patient (Knösel & Jung, 2011).

Functional stability is of fundamental importance to nutrition, and thus forms part of a basic physiological need. Canine guidance can be assessed during the slow lateral movements of the mandible. Whilst maintaining contact of canines on the working side i.e. the side the mandible moves towards, all posterior teeth and non-working canines will disclude. This too, is not a mutually exclusive concept of ideal function, as Sadowsky and Begole (1980) found 91% of a sample population to have non-working contacts.

Functional stability is also of importance to maintain equilibrium between the Skeletal and muscular components that are affected by the occlusal relationships of teeth. The temporomandibular joint and muscles of the face and mouth should be in equilibrium with each other, neither component taking on too much strain. This sheds light on the concept of maximum intercuspation. When the teeth are in this ideal position, what is the appropriate position of the mandibular condyle within the glenoid fossa during maximum intercuspation? Rinchuse (1995) reported that there was general consensus regarding these two components, intercuspal position (ICP) should coincide with retruded contact position (RCP) also referred to as centric relation. However further studies bare testament to the fact that ICP-RCP discrepancies of greater than 2mm, with no prior orthodontic treatment, have shown no evidence of harm (Milosevic & Samuels, 2000).

Aesthetics should be the second measurable outcome of any treatment, but is it really measurable? The influence of aesthetic desires in a patient comes from a variety of sources. The foundations stem from evolutionary traits, ideas that are inherent in human nature without necessitating conscious recognition. Psychological and social factors, as well as media and culture that prescribe what is acceptable and what is not influence aesthetic desires. In keeping a balance between the desirable “ideal” outcomes of occlusion, any treatment aimed at aiding the establishment of occlusion has to be allied with the aesthetic component. There should be a fine balance in the needs and desires of an individual, and outcomes should not solely be based on achieving the ideal angles and ratios that have been proposed (Kiekens et al., 2008).

The second component that is integral in establishing the force of a bite are the muscles of mastication. As hypothesised earlier, the expectation that males would have larger forces than female based on the premise that they possess stronger muscles or larger muscles for that matter needs clarification. This allows investigation into the muscles of mastication and the masticatory complex as a whole facilitating an improved comprehension.

## Anatomical Components of masticatory muscles

### Embryological development

The muscles of mastication develop in the mesenchyme of the mandibular arch. The differentiation of these muscles begins in the seventh week of intra-uterine development. Nerve fibres, only become apparent in the eighth week. Muscles of mastication develop in close relation to the Meckels cartilage as well as other cartilage of the cranial base (Norton, 2011). It is important to note that Meckels cartilage and other cartilage of the cranial base are primarily independent of attachment and only later do they begin to attach to the skeleton. Meckels cartilage also degenerates in adulthood (Bradley, 1981).

The first pharyngeal arch develops into the maxillary and mandibular process. The muscles of mastication which originate from their mesoderm, are the masseter, temporalis, lateral and medial pterygoid, mylohyoid, anterior digastrics, tenso tympani and tenso veli palatine (Bradley, 1981).

Skeletal components involved in the masticatory structure consist of the maxilla, temporal plate, zygoma and mandible.

The temporalis muscle begins to develop laterally in the eighth week of intra-uterine development, in the space anterior to the otic capsule.

During the thirteenth week, ossification of the temporal bone ensues as the muscles begin to align and attach along the anterior portion of the temporal bone (Bradley, 1981). The anterior portion of the temporal bone is proportionately broader than the posterior component (Norton, 2011).

The masseter muscle begins to develop at the beginning of the thirteenth week intra-uterine. The muscle attaches itself to the anterior portion of the zygomatic arch and then proceeds to grow laterally (Norton, 2011). This direction of growth creates the space required during the muscles further growth and development (Bradley, 1981).

Pterygoid muscle differentiation occurs prior to the differentiation of the masseter and temporalis, the differentiation of the Pterygoid muscle can be observed during the seventh week intra-uterine (Norton, 2011). There is an early relation of the muscles to the cartilages of the cranial base as well as to the condyles of the mandible. As the cranium develops, the increase in width and length causes the muscles to expand. Histologic differentiation of the muscles of mastication is evident by week 22 of intra-uterine development (Moyers, 1973).

The muscles of mastication originate on the skull and insert into the mandible and they are all derivatives of the first pharyngeal arch. The innervation of the masticatory muscles is derived from the mandibular

division of the trigeminal nerve (Bradley, 1981). Movements caused by the muscles are illustrated in Table 6.

**Table 4: Masticatory muscles and function**

Muscle	Function
Lateral Pterygoid	Medial pull
Temporalis	Superior pull
Posterior temporalis	Superior posterior
Medial Pterygoid	Superior medial
Masseter	Superior
Buccinators, tongue, orbicularis oris	Position food
Geniohyoid, mylohyoid, anterior belly of the digastrics	Depresses jaw (Opens mouth)

## Anatomy of the muscles

### Masseter

The point of origin of the superficial head is at the inferior border of the anterior two thirds or medial surface of the zygomatic arch. The muscle then inserts at the lateral surface of the mandibular ramus and its coronoid process. The primary action of the masseter muscle is to elevate or lift the mandible (Moore & Dalley, 1999). The muscle receives its innervation from the masseteric nerve branch, which stems from the mandibular division of the trigeminal nerve.

The deep head of the masseter muscle is the smaller section. It originates at the medial border of the zygomatic arch as well as the inferior border of the posterior third of the zygomatic arch (Boyd et al., 1966). This smaller segment of the masseter muscle inserts into the superior-lateral area of the mandibular ramus and coronoid process collectively. The innervation and action of the smaller deep head of this muscle matches that of the larger superficial head (Norton, 2011).

### Temporalis

The temporalis muscle is fan shaped and originates on the temporal fossa of the cranium and inserts itself on the mandible covering a substantial amount of bone in this area. Insertion begins along the apex of the coronoid process, continues along the anterior and posterior

borders as well as the medial surface (Boyd et al., 1966). The muscle then extends inferiorly to the anterior border of the mandibular ramus also known as the temporal crest, coming to an end at the region of the third molar tooth. The temporalis muscle action elevates the mandible whilst the posterior fibres of the temporalis muscle cause retrusive mandibular movements (Moore & Dalley, 1999). The large area of insertion of this muscle on the coronoid process and ramus of the mandible helps maintain the posture of the mandible. It maintains the mandible in a rest position. The innervation of the muscle is derived from the trigeminal nerve, specifically the anterior and posterior deep temporal branches of the mandibular division (Norton, 2011).

#### Medial Pterygoid: Deep head

The deep head of the medial pterygoid muscle originates on the medial surface of both the ramus and the angle of the mandible, with components arising from the pterygoid tubercles (Moore & Dalley, 1999). The function of this deeper pterygoid muscle on the mandible is in its ability to elevate, protrude as well as carry out lateral excursive movements. The nerve supply stems from the mandibular division of the trigeminal nerve, via the pterygoid branch (Norton, 2011).

### Medial Pterygoid: Superficial head

The superficial head of the medial pterygoid muscle originates on the maxillary tuberosity. The point of insertion of this muscle is on the medial surface of the angle of the mandible and the mandibular ramus, inferior to the mandibular foramen (Moore & Dalley, 1999).

### Lateral Pterygoid: Superior head

This muscle originates at the greater wing of the sphenoid infra-temporal crest to terminate at its points of insertion on the articular disc and capsule of the temporo-mandibular joint (Moore & Dalley, 1999). The primary and focal action is to depress the mandible downwards causing opening and to protrude the mandible in a forward direction; the lateral pterygoid muscles also produce a lateral excursive movement in the mandible (Boyd et al., 1966).

### Lateral Pterygoid: Inferior head

The inferior head of the lateral pterygoid muscle originates at the lateral surface of the lateral pterygoid plate and the point of insertion is on the pterygoid fovea, which can be found on the neck of the condyle of the mandible (Moore & Dalley, 1999).

The innervation of the lateral pterygoid muscles originates from the trigeminal nerve; the direct nerve supply into the muscle is a branch of the mandibular division of the trigeminal nerve, namely the lateral

pterygoid branch. The lateral pterygoid branch lies medial to the lateral pterygoid muscle after exiting the foramen ovale (Moore & Dalley, 1999).

The combined action of these muscles is to cause movement in the mandible in a depressive or downwards opening, protrusive and laterally excursive direction (Boyd et al., 1966).

The masticatory muscles are antagonistic in their activity and they all act on the mandible in some capacity. The mandibular closing muscles are the masseter, temporalis and the medial pterygoid. The digastric, mylohyoid and lateral pterygoid muscles can be classified as jaw openers (Boyd et al., 1966). Attention must be brought to the fact that contraction of the lateral pterygoid muscle does not solely produce opening of the jaw, as it is active during jaw protrusive movements as well as jaw closure (Bradley, 1981). This muscle provides stability to the head of the condyle, moving it both forward and lateral. This twofold range of motion is brought about by the independent action of the two individual heads i.e. the superior and inferior heads of the lateral pterygoid muscles (Boyd et al., 1966).

Other minor muscles that partake in the masticatory process are the supra-hyoid and infra-hyoid muscles. The supra hyoid activates during the jaw opening and the infra hyoid provides stability to the hyoid bone upon the contraction of the supra-hyoid muscle (Moore & Dalley, 1999).

Mastication involves using the muscles to move the mandible in one of three planes, in an antagonistic fashion:

- Elevation/depression
- Protrusion/retrusion
- Side to side excursion

### Anatomy of the skeletal structure

The Skeletal component of the masticatory process is that of the cranium, the mandible as well as the teeth. The base of the skull and mandible articulate with each other at the temporo-mandibular joint, more specifically between the squamous portion of the temporal bone and the condyle of the mandible (Norton, 2011).

In order to maximise the surface area available for chewing, the teeth should be positioned in an ideal relation to each other. Maximum intercuspation should involve maximum contact between the functional cusps of the maxillary and mandibular teeth (Gibbs et al., 1981).

## Physiological component of mastication

### Mandibular movements

The movement of the mandible is a rhythmic motion; it causes the separation and meeting of the maxilla and mandible. Within this rhythmic motion there exists protrusive, retrusive as well as laterally excursive movements (Anderson, 1956). The movements of the condylar heads within their respective fossa are antagonistic on either side. The movement is dependent on the direction of the movement. It is only when the mandible is moved uniformly downward, that both condyles act in the same manner, translating downwards and forwards (Boyd et al., 1966, Bradley, 1981).

The rate and force of the masticatory process is dependent on the consistency of the food. The initial forces of mastication and muscle activity often differ from the final or later stages when the food particle has already been broken down (Horio & Kawamura, 1989). This change is a result of the sensory feedback response system that is controlled and regulated by the central nervous system. Thus changes in the motion of mastication, forces and activity of the muscles and the movement of the jaws can be observed within a single “session” or cycle of mastication (Lavigne et al., 1987).

### Frontal plane movements

The movements of the mandible in a frontal plane vary according to the type and texture of the food particles. During the cycles the mandible does not maintain a constant velocity as the sensory feedback mechanism is undergoing constant change, as the food particles are being broken down (Bradley, 1981).

### Sagittal movements

There is less variation in this type of movement, as it is independent of food type and not subjected to human individualism. Opening begins with a protrusive movement, this movement is synchronised with the movement of the mandible seen in the previously mentioned frontal plane (Bradley, 1981).

### Border movements

The following border movements have been recognised to allow for the manual positioning of the mandible in a safe and comfortable modus. These are the centric occlusion position - where the teeth of the maxilla and mandible are in maximum inter-cuspatation and the centric relation jaw position, which is the most retruded position of the mandible to the maxilla in which lateral movements are still possible. It is important to note that the movements of the jaw during the masticatory process are not confined to these defined border movements.

Border movements should not be misconstrued for the limits in which mandibular motion is contained (Bradley, 1981).

## Muscle Physiology

The muscles of mastication are striated muscles much like the Skeletal muscle in the body. In response to a brief stimulation the muscle will rapidly shorten and then proceed to return to its rest length at a relatively slow pace. Passive forces within the masticatory muscles are imperative in the establishment of the rest position of the mandible. In the absence of any contractile stimuli, the passive components maintain balance. The tonic activities of the muscles fundamentally contribute to the position of the jaw. This phenomena can be observed by the dropping of the jaw and opening of the mouth during a semi-conscious state or whilst sleeping (Ganong & Barrett, 2005). The muscles of mastication are made up of two different types of extra-fusal fibres. The first being the small type which produces a slower rate of contraction, and then there is the larger fibres which are capable of a faster contraction (Bradley, 1981). Initial muscle activity takes place in the medial pterygoid muscle (Figure 8) this occurs during closure of the mandible (Bradley, 1981). Then the temporalis muscle on the working side comes into action. The next two muscles that begin to contract are the contralateral temporalis and the masseter muscles bilaterally. After this contraction sequence there is a period of inactivity that occurs just

as the teeth come into contact with each other in maximum intercuspation. Figure 8 depicts the relative activity of the various muscles as recorded on an electromyography (Bradley, 1981).

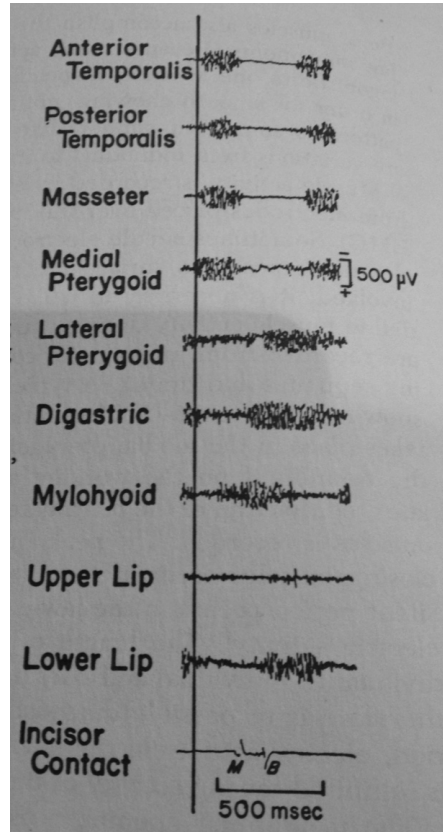


Figure 8: M=tooth contact B=break of contact (Bradley, 1981)

The muscles that are responsible for the opening of the mandible follow the following pattern (Bradley, 1981).

1. The mylohyoid muscles
2. The digastric muscles
3. The lateral pterygoid muscles

These muscles do not become active simultaneously.

For the three aforementioned muscles to be effective in function, it is imperative that the hyoid bone be stabilised in relation to the base of the skull. The hyoid bone stabilisation is brought about by the contraction of the infra-hyoid and stylo-hyoid muscles (Boyd et al., 1966). Although the buccinator muscles, orbicularis oris and the muscles of the tongue are not true muscles of mastication in that they do not supply a masticatory force, they aid in the manipulation of the food bolus against the teeth thus facilitating the masticatory process (Bradley, 1981). In this way these muscles form accessories to the masticatory process.

## The chewing process

As solid foods are placed into the mouth a variation in the force, pattern of mandibular movements and cycle speeds comes into play, although the general pattern of processing maintains uniformity amongst subjects (Matsuo & Palmer, 2009). Food is transported from the front of the mouth towards the occlusal surfaces of the posterior teeth. It is here that the food is processed by a series of masticatory cycles. This allows the breakdown of the ingested food particles and when the food is ready for swallowing it is passed posteriorly into the oropharynx (Matsuo & Palmer, 2009). In the oropharynx the food particles accumulate into a bolus, the formation of this bolus is aided by the mucins that are found in the saliva. Food is then swallowed (Bradley, 1981).

## Effect of chewing on digestion and the role of food texture

The breakdown of food particles by the masticatory system is a systematic process. Mastication occurs to produce food particles that are of a uniform size, to allow for the formation of a bolus and ease of deglutination. Studies have shown that an excessive amount of chewing may not be necessary for digestion to take place, however an increased amount of mastication does in fact aid the process (Van der Bilt et al., 1995). Food that is broken up into smaller particles allow for a greater surface area to come into contact with the various digestive enzymes, in comparison to larger food particles that will present with a smaller surface area and a decreased exposure to digestive enzymes (Van der Bilt et al., 2006, Pedersen et al., 2002).

Mastication aids digestion additionally by the production of saliva, which in turn stimulates the flow of the gastric juices involved in the digestive process. Finally the sizes of food particles are influential in the timing of the digestive process within the gastric cavity, larger particles take longer to be broken down (Van der Bilt et al., 2006).

The taste and the texture of food particles influence the masticatory process. Characteristics that have an influence on the process are water, fat content, and hardness (Pedersen et al., 2002). Hardness in particular has been found to have an impact on the force of mastication, resulting in a proliferation of activity in the muscles, as well as an escalation in the movement of the mandible (Van der Bilt et al., 1995).

Hardness and volume of food has been allied to changes in the quantity of masticatory cycles that can be detected. Softer foods require fewer cycles in turn to be adequately broken down. Harder foods such as carrots for example would necessitate an escalation in the quantity of masticatory cycles so as to attain the equivalent acceptable consistency as that of the softer foods (Van der Bilt et al., 1995). Moisture has also been found to have an impact on the duration of swallowing. Dry foods may compel more salivary mucins to permit the formation of a satisfactory bolus, adding masticatory time needed to permit the secretion of this supplementary saliva. “This is the reasons to why it would be quicker to eat a slice of Melba toast with butter on it, than it is to eat a dry slice of Melba toast” (Pedersen et al., 2002).

A study conducted by Mioche and co-workers established that a difference in the texture of food ingested elicits diverse activity between the temporalis and masseter muscles (Mioche et al., 1999). This study ascertained that the masticatory process is influenced by and adjusted to accommodate the fluctuations in the textures of food particles. Texture of food has been categorised by the way it undergoes deformation when subjected to the forces of mastication. However in most scenarios texture denotes the physical characteristics of the food item (Mioche et al., 1999). Mioche and co-workers also stated that the temporalis muscle activity was influenced in a greater capacity by the texture of the food particles than the masseter muscle function (Mioche et al., 1999).

The identification and subsequent muscular response to the textures of various food items is dependent on the sensory feedback control mechanism, which regulates the cycle of mastication as well as the Skeletal and muscle movements. Röhrlé and Pullan (2007) have referred to this neural circuitry as the central pattern generator from here on referred to as the CPG (Röhrlé & Pullan, 2007).

### Neuronal component of mastication

The mastication cycles do not occur at random. They are variable and not purely mechanical. Cycles, forces and speeds differ upon being subjected to external as well as internal factors. External factors could be the type and texture of the food while internal factors could be the salivary flow rate and the occlusal relationships (Bradley, 1981).

The central nervous system retains the capacity for controlling the masticatory process through a sensory feedback mechanism. Information is generated from numerous inputs and co-ordination occurs in the trigeminal sensory motor complex within the brain stem (Yamada et al., 2005). The trigeminal motor nucleus lies in the floor of the upper part of the fourth ventricle of the brain.

The motor nerve fibres from this nucleus supply the muscles of mastication, whilst the sensory fibres innervate the face, anterior scalp, mouth, teeth and nasal cavity (Boyd et al., 1966).

### Anatomy of the trigeminal sensory motor complex

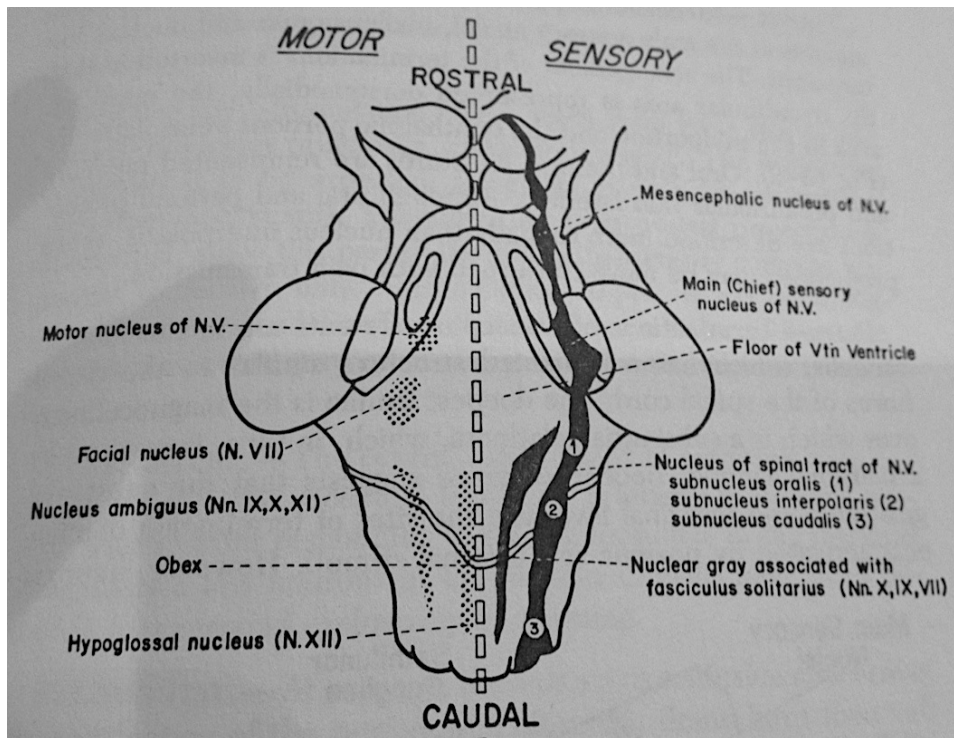


Figure 9: Sensory and motor nuclei of the brain stem (Bradley, 1981)

The trigeminal sensory motor complex is situated in the brain stem, it is divided into sensory and motor components as depicted in Figure 9 above (Bradley, 1981). For ease of understanding the sensory nuclei are shown on the right and the motor nuclei on the left in this figure and these nuclei occur bilaterally (Bradley, 1981).

*Trigeminal control of oro-facial sensation relative to mastication (Bradley, 1981)*

- Muscle spindle: afferent nerves from the muscles have their cell bodies located within the mesencephalic nucleus. These cells project directly to the V<sup>th</sup> motor nucleus and elicit a monosynaptic arc.
- Temporomandibular joint: sensory information from the rotational movements of the joint is transferred to the main sensory and spinal nuclei. An ill-defined region of cells located between the principal and motor nuclei referred to as the supra-trigeminal nucleus has also been found to receive temporo-mandibular joint afferents.
- Motor output: the nervous outputs required for the functioning of the muscles of mastication are situated in the trigeminal motor nucleus. Muscles of mastication are grouped together distinctively within the nucleus. Larger temporalis and masseter muscle fibres would logically occupy a larger part of the nucleus. Jaw openers are situated on the ventral part and jaw closers can be located to the dorsal part of the nucleus as depicted in Figure 10 below (Bradley, 1981).

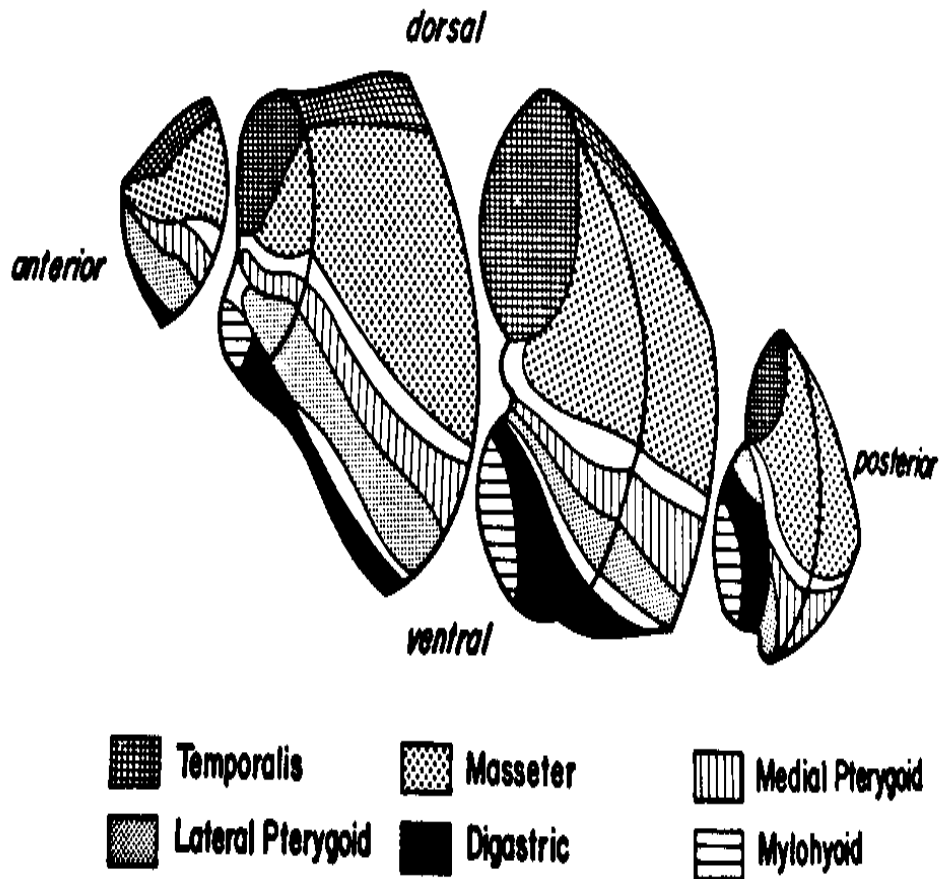


Figure 10: Enlarged perspective of the motor nucleus of the Vth cranial nerve (Bradley, 1981)

### Neural co-ordination of the masticatory process

The brain stem forms an integral component for the neural control of mastication. All efferent nerves to muscles of mastication are found in the pons and the medulla and all the afferent sensory information is projected to brainstem. Thus the brain stem is essential for mastication (Lund, 1991).

Mastication can be initiated in response to sensory input such as food, taste or smell. Involuntary chewing occurs in response to the presence of food. Chewing can also be a voluntary action initiated by an individual without any presence of food, eliciting a more conscious form of mastication. In the case of involuntary mastication, chewing continues subconsciously till the point where deglutination takes place and the rhythmic cycles cease.

Mastication is initiated by the stimulation of numerous brain sites, the cerebral cortex, basal ganglia, hypothalamus, cerebellum and the brain stem. The stimulation of these brain sites are not confined or limited to the initiation of masticatory control, they are multifactorial in their function (Lund, 1991).

### Reflex theory of masticatory movements

Cortical activity initiates the masticatory process by exciting the motor neurons to muscles responsible for jaw opening. After opening occurs, the stretch in the muscle spindles inhibits the motor neuron activity to the jaw closing muscles. This is then followed by the jaw jerk reflex, which closes the jaw by activation of the jaw closing muscles. “Jaw jerk” reflex has been described by Lund to be a jaw movement reflex that is initiated by the opening of the mandible, resulting in a reflex closure (Lund, 1991).

Once the mandible closes and the stretch in the opening muscles has been relieved, the mechanoreceptors and periodontal receptors are stimulated to open the jaw again. This is a complete cycle, which then continues to produce mastication. This theory has however since been updated and is depicted in Figure 11.

Figure 11 illustrates the sequence and direction of events

1. Corticobulbar tracts (C.O)
2. Jaw opening motor neurons (J.O)
3. Muscle spindle afferents (Ia)
4. Jaw closing afferents (J.C)
5. Periodontal or mucosal afferents (G)

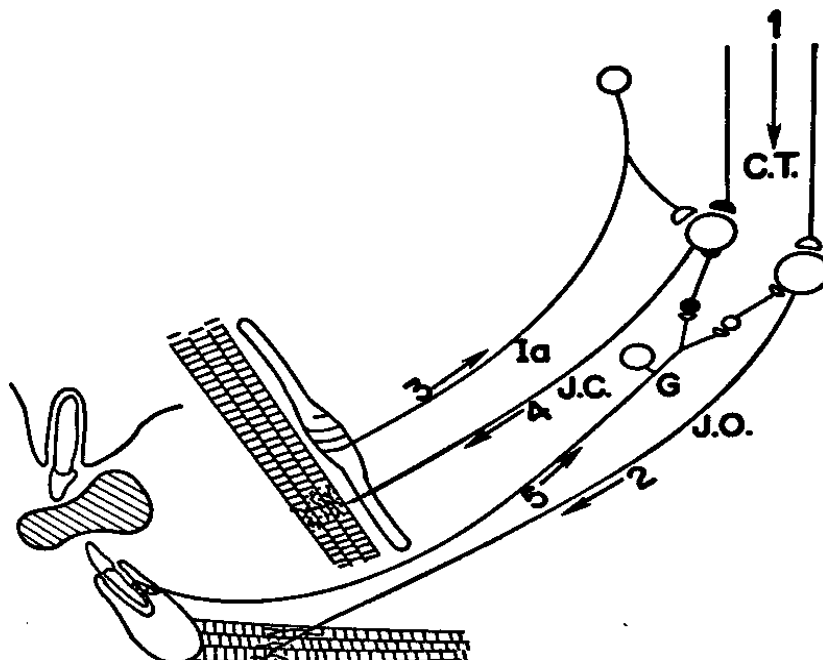


Figure 11: Diagram of the reflex theory of masticatory control (Bradley, 1981)

## The central pattern generator theory

As previously mentioned there is a neural pattern generator located in the brain stem that is said to be responsible for the cyclic movements of the masticatory system called the central pattern generator (C.P.G) (Lund, 1991). This generator is able to function independently of inputs, but regulated by the inputs received either centrally or peripherally. Rhythmic excitation of the jaw elevators and jaw depressor muscles by the central pattern generator elicits the cyclic jaw movements. These movements can be modified by the reception of sensory information in a feedback control manner (Lund, 1991).

Figure 12, illustrates the central neural pattern generator. C.T.I is the indirect cortico-bulbar input to the bulbar motor neurons on which information is transferred via the pattern generator (P.G). Information is sent to the jaw opening motor neurons (J.O). J.C illustrates the jaw closing motor neurons. C.T.D supplies the direct corticobulbar input. O.A are oral afferents that are capable of activating the P.G.

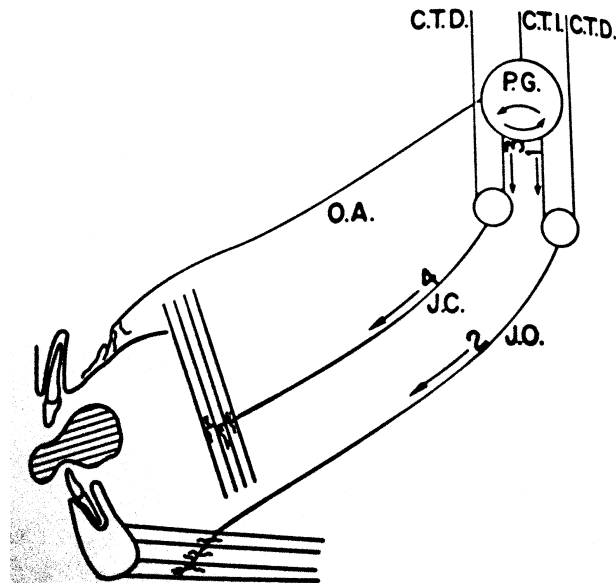


Figure 12: Central pattern generator control of the masticatory system (Bradley, 1981)

### Sensory feedback

It has been established that the rate and force of the masticatory cycle is dependent on oral feedback with respect to the texture and quantity of food present (Van der Bilt et al., 1995). Oral receptors responsible for generating this feedback are the periodontal ligament; temporomandibular joint, tactile and muscle spindle feedback receptors. An alteration in the intra-oral condition has to be reflected in the pattern of mastication, making each new cycle different to the former and prospective cycles (Lavigne et al., 1987). Sensory information in the oral cavity also contributes to the positioning of the food bolus (Van der Bilt et al., 1995). Movement of the lips, tongue and buccinator muscles contribute to positioning of the food on the table of the posterior teeth (Matsuo & Palmer, 2009).

The periodontal receptors then come into play in the assessment of pressure or muscle force required to crush the food substrates. Whilst the presence of all these sensory receptors working together as a unit is ideal, the removal or exclusion of either one or multiple does not render the masticatory system decrepit. Mastication is still able to continue (Matsuo & Palmer, 2009).

### Co-ordination of other activities during mastication and muscle forces

Due to the pathways for food ingestion and the airway crossing at the pharynx, the function of the pharynx is twofold. During mastication, the pharynx converts into a food channel as the bolus accumulates in the pharynx prior to swallowing. It is essential that the structures in this area be under constant and strict neural control to avoid aspiration of food particles into the lungs. During the masticatory process respiration occurs through the nasal cavity and the pharynx. The anatomical structure of the pharynx is dynamic in that during breathing, it remains accessible and patent allowing for the passage of air in and out of the lungs (McFarland & Lund, 1995). However, during mastication and the accumulation of food in the pharynx for bolus development, the pharynx constricts producing the pressure or force necessary to drive the bolus down to the oesophagus.

During deglutination the airway is safeguarded by the velopharyngeal closure sealing of the nasal cavity. The laryngeal closure, which includes the glottal closure, arytenoid adduction and epiglottal folding, seals off the lower airway. The tongue pushes up against the soft palate yielding a further seal. This inhibits aspiration during deglutination (Bradley, 1981).

Matsuo and co-worker reported that adult swallowing is generally initiated during the expiration stage of breathing; breathing is then continued post swallowing by the perpetuation of expiration. Breathing that recommences with expiration is considered an airway protective mechanism, in the event that there are any food particles still present in the pharyngeal area they are thrust out with expiration rather than aspirated with the next inhalation. Thus in healthy adults, swallowing occurs during expiration (Matsuo & Palmer, 2009).

In infants, swallowing ensues in the inspiratory phase of breathing. This is as a result of the difference in the temporal coordination (Kelly et al., 2007). The larynx in infants is positioned higher and posterior to the oral cavity. As the larynx descends in infancy, a shift in the swallow breathing mechanism can be observed and the infant develops an “exhale-swallow-exhale” pattern that matches that pattern of adults (Kelly et al., 2007).

From the detailed analysis of mastication and its control it is evident that the masticatory system is a highly complex system. Movements vary between individuals and variation exists subjective to the nature of the food substance being chewed. Characteristics of food are influential in the masticatory process. Complex muscular activity results in a variety of movements. These movements differ from cycle to cycle, as the food particles are being further crushed and disintegrated and thus morphologically changed.

The presence of saliva is tantamount in the facilitation of taste, mastication and bolus formation. The position of the teeth in the mouth should be ideal so as to maximise the surface area available for the crushing process. Having ideal occlusal contacts reduces the time required to crush a food substance as well as the force or period of intense activity on the masticatory muscles.

As the teeth are housed in bony structures and chewing is effected by the action of the muscles attached to the jaws it is pertinent to have an understanding of growth and the theories surrounding craniofacial growth. This knowledge would enable evaluation of interactions between teeth, musculature and the underlying skeletal components, in the establishment of normal and abnormal relationships.

## Theories of growth

### Wolff's law:

Julius Wolff (1836-1902) was a German anatomist who proposed the principle that every change in the form and function of a bone, or a change in the functioning of bone alone will result in alterations in the internal structure of that bone, and its resulting internal form (Prendergast & Huijskes, 1995). This principle results in bone deposition and resorption in a pattern that mimics or follows the differences in pressure and stress (Huijskes, 1995). The viscoelastic property exhibited by bone confirms that as the load exerted on a bone is increased, the response of that bone is to increase its strength, which often results in an increase in the brittleness of the bone (Prendergast & Huijskes, 1995). The muscle relative to the skeletal tissue undergoing a load is of valuable importance as they neutralise the tensile load, and allow functioning of the bone whilst under a load. A consequence of increased loading is that the bone undergoes regular remodelling, which this allows recovery from the load force (Frost, 1990).

### Sutural Theory:

The source of craniofacial growth has been claimed by Sicher to be at the sutures.

The crux of this theory provokes the idea that sutural growth at the juxtaposition of the facial skeletal components and the skull is responsible for the growth of the naso-maxillary complex in sync with mandibular growth. Sicher's theory on sutural growth does not disregard the influence of genetics on the extent of growth that occurs amongst individual subjects (Bhalajhi, 2012).

#### Cartilaginous theory:

The cartilaginous theory negates the primary role of sutures in the growth by the proposition that cartilage and periosteum possess growth factors inherently. This theory suggested and studied by James Scott propagates the idea that sites constituted of cartilage as a base structure, form the primary centres of growth. Scott establishes the nasal septal cartilage as the centre of growth of the naso-maxillary complex. The mandible is akin to a long bone, with growth occurring at the condyles, growth occurs in the partial epiphyseal plate of which the main component is cartilage. Scott summarises that in many bones, the growth takes place in a cartilaginous model, which is then replaced by bone. He noted that should a part of an epiphyseal plate be transplanted, growth does not halt, and this is therefore indicative of the innate potential for growth. (Bhalajhi, 2012)

## Remodelling:

Enlow and Hunter conducted a study to assess the contribution of sutural growth and bone remodelling within the growth systems of the craniofacial region (Enlow & Hunter, 1966). Their aim was to question pre-existing growth theories and manifestos, not with the purpose of disregarding them, but to assess the symbiosis of theories relevant to achieve increase in the total size of the upper face. Enlow and Hunter proposed the idea that there is rather continuous and successively simultaneous remodelling present. As new bone being deposited at the suture site the growth site increases and the adjacent bones will separate. The increase in size often will result in an alteration in the position of the relative parts of the bone.

To prevent the manifestation of disproportionate growth, this increase in size has to be accompanied by continuous remodelling. (Enlow & Hunter, 1966). Enlow and Hunter also reported that sutural growth is a somewhat passive process. It is not directly responsible for any displacement of bone but rather consequential to a displacement. An example of this scenario in craniofacial growth can be observed in the nasal area. Expansion of the nasal septum results in separation of the sutural contact surfaces. The separation then acts as a stimulus for the deposition of bone in this area (Enlow & Hunter, 1966).

Facial growth thus occurs according to the following pattern, growth at the sutural contact surfaces results in relocation of various components of each bone.

Relocation leads to remodelling, producing an increase in the overall size of the bone, resulting in an increase in the craniofacial skeleton (Enlow & Hunter, 1966), thus combining Sicher and Scott's theories on growth.

#### Genetic theory:

One of the primary theories of growth initially proposed, it focuses centrally on the notion that all growth manifests as a consequence of particular genetic makeup and contributory genes. Moss (1981) suggests that there is a combination of genomic and epigenetic factors, which contribute and are necessary for craniofacial growth. Through the synthesis and fusion of various genetic concepts and data studies the following was postulated by Moss (1981).

Firstly there are small, non-conforming and additive mutations that occur at specific points on the genome, these occur spontaneously. Secondly the origins in development of the genome have been naturally selected thus ensuring better survival of the stronger genes relative to the environment. This adaptation allows for successful reproduction and propagation of the gene, whilst ensuring evolutionary change.

Using this information as a foundation of understanding genetics and craniofacial growth we may now understand why there is an inclination to observe familial resemblance amongst individuals, thus ascertaining the genome responsible as the principal regulator of craniofacial growth. (Moss, 1981)

However further study or understanding of genomic influences postulates that the post-fertilization genome doesn't contain inherently sufficient information to regulate the development. Thus it is the combined function of genomic and epigenetic information that results in growth and development of the craniofacial system (Moss, 1981).

#### Functional matrix concept:

Melvin Moss introduced the idea, which is actually complementary to the concept of a functional cranial component involved in the growth process as proposed by Van De Klaaus (Bhalajhi, 2012). The idea of this concept is aimed at bridging the gap between form and function. The theory suggests the following: the tissues, organs and spaces are the loci of the primary action of growth. Separately these role players are related to a corresponding skeletal component (Moss, 1981). Consequentially the effect on the skeletal tissue can be identified in the origin, form, positioning and maintenance within the spectrum of the functioning system. Ipso facto the craniofacial system functions as a unit, each component is responsible for an independent action however

still influential in the greater matrix. The functional matrix is composed of all tissues, organs and functioning spaces. The Skeletal unit originate and grow from a functional matrix.

The Skeletal unit is comprised of all bone, cartilage and tendons. Whilst the accessory functional matrix consists of the supportive, glands, nerves, vessels fat, teeth and associated functioning tissue spaces. This matrix is then further divided into a functional and capsular matrix.

- The Functional matrix: consists of glands, nerves, vessels, fat, teeth and functioning spaces. This matrix is further divided into the periosteal matrix, which acts directly on a particular skeletal unit. Action in the periosteal muscle, gland, and blood vessel system can be observed in the deposition and resorption of bones in the related skeletal component.

#### Van Limborgh's theory:

Van Limborgh proposed the following factors that are influential in the control of growth.

1. Intrinsic genetics
2. Local epigenetic factors
3. Environmental factors
4. General environment factors

According to this theory, intrinsic genetic factors are primarily in control of chondro-cranial growth while desmocranial growth is controlled by the intrinsic genetic factors. Van Limborgh went on to agree with the role that cartilaginous parts of the skull play in the growth mechanism.

According to Van Limborgh: the chondro-cranial growth is controlled primarily by the intrinsic genetic factors. Because this is primary control, the genetic makeup has the most influence on resulting growth and development. Desmocranial growth is then controlled less by the intrinsic genetic factors. The role of the cartilage and sutures that influence adjacent skeletal components play an influential role in the growth mechanism and control. (Van Limborgh, 1972)

Maxillary growth and mandibular growth follow a similar path of movement i.e. downward and forward movement. The movement is antagonistic to growth occurring in a posterior direction. To simplify-posterior growth shifts the entire bone forwards. (Enlow & Bang, 1965)

As these skeletal components increase in size, the remodelling process mirrors the growth process. Remodelling maintains the proportions of the components, as well as their position relative to other skeletal structures.

## Chapter 3: Study design

### Aim:

This study aims to assess the maximum bite force of subjects exhibiting different Skeletal relationships and growth directions. Comparisons in the maximum bite forces amongst Skeletal form, growth direction, age and gender are to be carried out to establish whether any relationship or similarities exist amongst and between these various factors.

### Hypothesis:

There is no difference and no significance in magnitude of bite force between Skeletal Classes, malocclusions and genders.

### Objectives:

1. To determine the occlusal bite forces of patient in all three types of previously classified malocclusions i.e. Skeletal Class I, II and III.
2. To determine the occlusal bite force of patients exhibiting Horizontal growth patterns.
3. To determine the occlusal bite forces of patients presenting with Vertical growth patterns and anterior open bite.

4. To determine the difference between bite forces in males and females.
5. To compare forces amongst Skeletal classes within each growth direction type.
6. To compare forces amongst growth direction types within Skeletal classes.

## Bio-mechanical variables

### Equipment

The occlusal force meter was sourced from a Japanese based company, Nagano Keiki Co. LTD (**Figure 13**). The readings were done in Kilo newton.

With the meter set to a digitally calibrated setting of F0, 1 kilogram of force would equate to 9.80662N. The approximation of 1 kilogram of force equating to 10N is accepted as a rule of thumb amongst engineers. 1kN is a common expression of force, as the bite meter readings are expressed in kN. 1kN equates to 1000N and by simple factorisation we are able to deduce that 1kN equates to 1kg of force. So the exact ideal is that 1 kN be equal to 100kg of force.

10 N = 1Kg of force

100N = 10kg of force

1000N = 100kg of force

1000N = 1 kN = 100kg of force

A 1Kg weight was used to calibrate the bite meter. 1 Kg weight registered a force of 0.012kN. 0.012kN equates to 12 N. Therefore 1kg registered a force of 12 N. Due to method of calibration, the additional 0.002kN registered can be accounted for due to the positional action of the weight on the occlusal bite meter. Thus for

scientific study purposes we can confirm that the bite meter calibration corresponds to the theoretical ideal ratios and relationships between kilograms of force and newton.



Figure 13: Occlusal force meter with case



Figure 14: Close up of meter, with sterile casing



Figure 15: Weight used for calibration

### Bite force measurement:

The apparatus has a “set zero” and also “peak” registers that facilitate the record of the maximal force. The peak register is depicted by a beep. The bite meter comes with a plastic covering that forms a protective covering over the transducer arm where the force meter contacts the teeth (**Figure 14**). The plastic covering has on either side a circular embossed area, on which the subject has to bite. The presence of this covering ensures that there is standardisation in the readings between different teeth and amongst different subjects. These plastic coverings were sterilized prior to use to ensure that the strictest infection control policies were adhered to.

Bite forces were measured with the subjects sitting in the dental chair, in the upright position with the Frankfort Horizontal line parallel to the floor. The patients were instructed to sit with their arms extended along the body, hands were placed on their thighs and not clutching the arms of the dental chair. Patients were then requested to bite down on the meter, and progressively increase the bite force till a beep was heard. Test readings were carried out before the study readings were measured to familiarise the patients with the procedure.

## Study design

An observational analytical study was conducted to determine the relationship between maximum occlusal bite forces and different malocclusions i.e. the Class I, Class II and Class III Skeletal profiles, deep bite (Class II) and anterior open bite (any Class) patients. The study sample was restricted to patients without any major dental or alveolar abnormalities and this restriction included patients presenting with any syndromes. The extra oral examination entailed looking at the shape of the head and assessing according to the following three Horizontal types namely Class I, II and III and three Vertical types namely:

1. Monocephalic: exhibiting average shape and normal arch form  
Figure 25.
2. Brachycephalic: broad and shorter head with accompanying broader dental arches Figure 26.
3. Dolichocephalic: long and narrow head associated with narrow dental arches Figure 24.

The facial form was also assessed according to whether it conformed to an average, broader or longer form. The value in assessing the facial profile is imparted in the identification of gross maxillary to mandibular relationship deviations.

The facial profile was assessed clinically in the following manner. An imaginary line joining the forehead and the soft tissue A point on the maxilla was established. This line is then linked to a second line joining the soft tissue a point and the soft tissue pogonion i.e. the most anterior point of the chin. There are three types of profiles that can be identified using this method: the straight profile, convex profile and concave profile. A convex profile occurs due to a prognathic maxilla and a normal or retrognathic mandible often seen in a Class II. A concave profile is associated with the opposite maxilla to mandible relationship more likely in a Class III malocclusion. A straight profile is indicative of a Class I maxilla to mandibular relationship. The assessment of an anterior to posterior jaw relationship in the clinical examination is done by estimating the relationship between the soft tissue A point on the maxilla and the soft tissue B point on the mandible. When the index and middle finger placed on these two points respectively clinical detection of the skeletal relationship can be established. In a Skeletal Class II it is often found that the index finger is anterior to the middle finger thus resulting in an upward tilt of the fingers. In a Class III patient the middle finger lays anterior to the index finger, thus the fingers will point downwards. In patients with Skeletal Class I relationships the fingers should be parallel to the floor.

The Vertical relationship was addressed by looking as the angle that is formed by the lower border of the mandible and the Frankfort Horizontal plane. The Frankfort Horizontal plane can be established by connecting

the most superior point of the external auditory meatus and the inferior border of the orbit. They should intersect at the occipital region. This is considered a normal relation. If there is an intersection beyond the occipital region it is indicative of Horizontal growth and if there is an intersection anterior to the occipital region this is then Vertical due to the indication of a high angle. The assessment of growth was supported by an evaluation of the facial proportions. These should be of the ration 1:1:1 as measured from the hairline, the supraorbital ridge, the base of the nose and the inferior border of the chin. Any change in this ratio will then enable the establishment of where the discrepancy in the growth lies. For safety and ethical reasons radiographs were not prescribed for the purpose of this study. Skeletal classifications were however confirmed on those patients who had radiographs to meet other treatment objectives. Clinical diagnosis was confirmed on every fifth patient by an independent observer

### Setting

Patients were selected by a process of random sampling, done in both the undergraduate and postgraduate patient wards of the Orthodontic Department at the University of Pretoria Oral and Dental hospital.

The sample consisted of 360 of individuals, age range of 6 years to 45 years, equally divided into 180 females and 180 males. All participants were healthy and showed no major facial malformations. They had

complete or almost complete dentitions, either in the primary mixed or permanent stages. There was no presence of extreme malocclusion or functional abnormalities. Each patient's occlusion, temporomandibular joint function, craniofacial form, and state of dentition were evaluated.

### Patient Selection

- Patients from each malocclusion group were selected based on their Skeletal and dental relationships using standard orthodontic clinical examination parameters described. Radiographs when available confirmed this. Selection was done randomly i.e. every second patient who presented at the undergraduate and postgraduate orthodontic screening clinic.
- Patients were between the ages 6-45 years
- Brachifacial patients (Horizontal growth assessed and confirmed clinically and with radiographs if available)
- Dolico-facial patients (Vertical growth assessed and confirmed clinically and with radiographs if available )
- Mesofacial patients (Normal growth assessed and confirmed clinically and with radiographs if available)
- Patients in the study were equally split between male and female
- Patients had to have their first and permanent molars fully erupted and in occlusion, with no restorations on these teeth.
- Patients with severe skeletal deformities requiring surgical correction were excluded.

Radiographically the following parameters were used to evaluate facial height in those patients who had cephalograms. Linear distances from nasion to anterior nasal spine and anterior nasal spine to the menton were included to calculate the upper and lower face height, total anterior face height. The distance from the sella to gonion indicated posterior face height. Angular parameters included deflection from the sella nasion line, to the palatal, occlusal and mandibular planes. The y-axis was included in the assessment criteria. Horizontal Skeletal relations were evaluated with the ANB angle, Wits analysis and Facial plane angle.

### Measurements

Using the digital occlusal bite meter, measurements were taken over the central incisor area at the meeting of maxillary and mandibular midlines, over the first premolars or deciduous primary molars and lastly over the first molar region. Three readings were taken on each point of interest as mentioned above and a mean reading was deduced. For the posterior teeth, the readings were taken bilaterally. Patients were instructed to bite down on the embossed area of the sterile plastic bite meter cover. It is over this area that the reading registers on the meter. They were instructed to bite in a normal fashion and relax once a beep was heard. The beep on the bite meter is indicative of maximum force registration.

## Data analysis

Analysis of covariance was used (ANCOVA) with the age as a covariate set to mean 15,6333 years comparing bite forces in Class I, II and III malocclusions in Brachifacial and Dolicofacial and Mesofacial subjects.

## Independent group design

The analyses was structured around the following:

- Independent Variable: malocclusion type
- Dependent Variable: bite force
- Confounding factors: gender, race
- Statistical tests: t test, ANCOVA and m test

## Sample size

- Two groups – 180 males and 180 females
- 9 X 20 subjects in each group
- 9 classes made up as follows:
  - Class I Mesofacial
  - Class I Brachifacial
  - Class I Dolicofacial
  - Class II Mesofacial
  - Class II Brachifacial

- Class II Dolicofacial
- Class III Mesofacial
- Class III Brachifacial
- Class III Dolicofacial

## Ethical considerations

Patient anonymity was respected. Sterile protocol was established to ensure no cross infection amongst subjects. Plastic coverings were washed and soaked in cold sterilant, according to instructions advised by the manufacturing company. Radiographs were not taken for the purpose of this study. Consent and assent was received from patients to comply with the code of ethics laid out by the Research ethics committee at the University of Pretoria. Consent and assent forms have been included in the addenda,

This study was approved by the Research Committee of the School of Dentistry at the University of Pretoria (approval number: 2012/12). Ethical consent to conduct the study was obtained from the University of Pretoria Ethics committee (approval number: 220/2012). Copies of both documents have been included in the appendix.

## Chapter 4: Results

### Data analysis plan

Data from this two-factor study design with main effects Growth direction (Horizontal, Normal, and Vertical) and Skeletal Class (I, II, III) were analysed using an analysis of covariance (ANCOVA) t-test with age as a covariate and the inclusion of the interaction between the main effects. A secondary analysis (ANCOVA m-test) was carried out to analyse the data categorically within each growth direction category to assess the interaction between Skeletal classes and within Skeletal classes.

### Demographic data and distribution of subjects

In order to identify associations between demographic variables (Age, sex, ethnicity) and the main variables of interest (bite force and growth directions) a series of preliminary analysis were conducted. A significant positive correlation was found between Growth direction and Age ( $P < 0,05$ ). However when analysis of subject distribution of subjects aged 14 years and above those below the age of 14 years, it was found that 61,94% of subjects were below the age of 14 years and 38,06% were equal or above the age of 14 years (Figure 16). This information disclosed a trend associated with age, the non-uniform distribution of ages amongst the categories of the main effects of this

study (Growth direction and Skeletal Class), the significance of age could affect the outcome of the interaction and for this reason age as a covariate was set to a mean of 15.6333 years to be used in all the tests.

180 Males and 180 Females were tested, thus the sex distribution of subjects was equally split (50%). Within each sex category the ethnic distribution of subjects was categorized into the following categories: African, White, Indian and Coloured. The female subject group consisted of 53% African, 28% White, 17% Indian, and 2% Coloured (Figure 17). Ethnic subject distribution amongst males was found to be 48% African, 22% White, 22% Indian and 8% Coloured (Figure 18). Analysis of the data to test for difference in mean bite forces between male and female subjects found no significant difference or interaction as the  $P > 0,05$ .

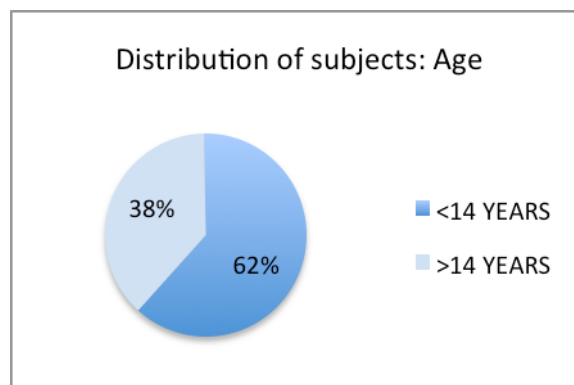


Figure 16: Age distribution of subjects

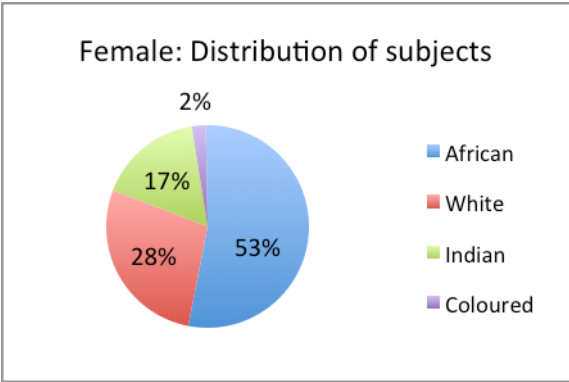


Figure 17: Ethnic distribution amongst females

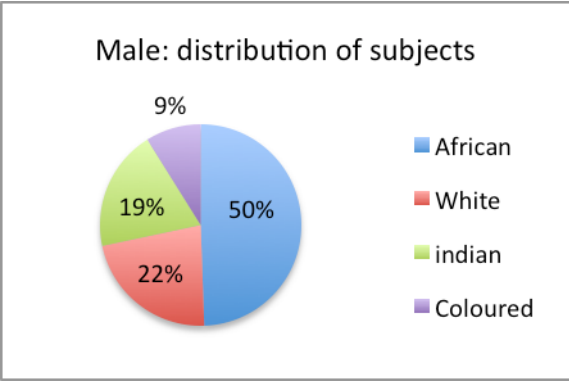


Figure 18: Ethnic distribution of male subjects

Bite force comparisons were carried out over the incisor, premolar and molar area of the subject population group, consisting of 360 subjects. Readings were taken on both the left and right sides of each subject.

### Incisors:

**Table 5: Main effect interaction (Incisor)**

Source	Partial SS	df	MS	F	Prob > F
Model	.063904323	9	.00710048	5.53	0.0000
grwth_dir	.012294971	2	.006147485	4.79	0.0089
skel_Class	.009760745	2	.004880372	3.80	0.0232
grwth_dir#skel_Class	.021832218	4	.005458055	4.25	0.0022
age	.02331131	1	.02331131	18.17	0.0000
Residual	.449065905	350	.001283045		
Total	.512970228	359	.001428886		

The level of statistical significance was set at  $P < 0,05$ . Growth directions differed significantly ( $P = 0,0089$ ), Skeletal Class showed significance but to a lesser extent than observed in growth direction ( $P = 0,0232$ ). (Table 5) However there was a significant interaction between growth direction and Skeletal classification ( $P = 0,0022$ ), as seen in Table 5. From the linear plot of the Mean bite force over the incisor area visible (Figure 19) it is evident that Horizontal and Normal groups perform relatively similarly; the interaction can be seen in the Vertical category, specifically relative to the performance of the Skeletal Class II subjects.

Table 6 below displays the mean bite force over the incisor area for all subjects of the study group.

**Table 6: Mean bite force incisor**

grwth_dir	skel_Class		
	Class I	Class II	Class III
Horizontal	0.053	0.069	0.049
Normal	0.057	0.073	0.046
Vertical	0.055	0.036	0.045

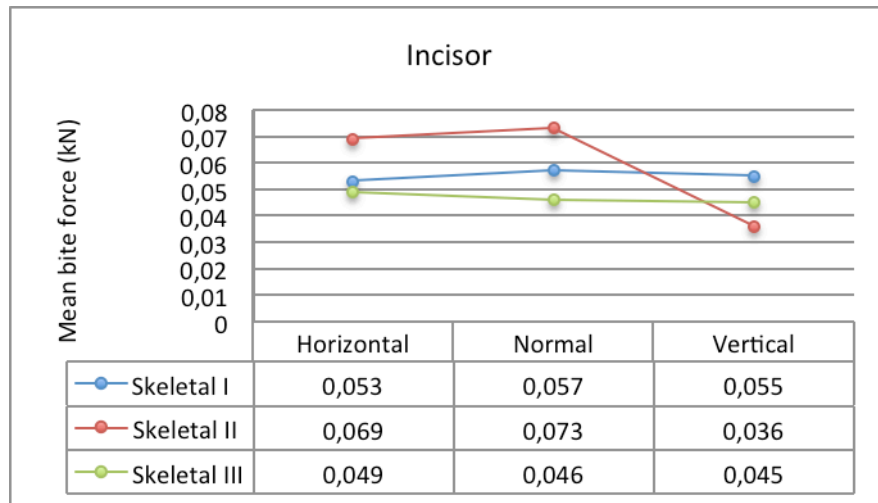


Figure 19: Incisor mean bite force linear graph

Left Premolar:

**Table 7: Main effect interaction (Left PM)**

Source	Partial SS	df	MS	F	Prob > F
Model	.350360153	9	.038928906	6.51	0.0000
grwth_dir	.011686344	2	.005843172	0.98	0.3773
skel_Class	.128314129	2	.064157064	10.73	0.0000
grwth_dir#skel_Class	.089017217	4	.022254304	3.72	0.0056
age	.134342913	1	.134342913	22.47	0.0000
Residual	2.09255959	350	.005978742		

Analysis of the left premolar found a greater significance between the Skeletal classes (P=0,0000) and while the growth direction did not differ significantly (P=0,3773) Table 7 and Table 8. There was a significant interaction between growth direction and Skeletal classes. Investigation of the linear graph identifies this interaction in the Skeletal Class I Horizontal group, whilst we can also note a slight increase in the mean bite force of the Horizontal Skeletal Class II group (**Figure 20**)

**Table 8: Mean bite force (PM\_L)**

grwth_dir	skel_Class		
	Class I	Class II	Class III
Horizontal	0.083	0.119	0.076
Normal	0.142	0.103	0.073
Vertical	0.137	0.093	0.077

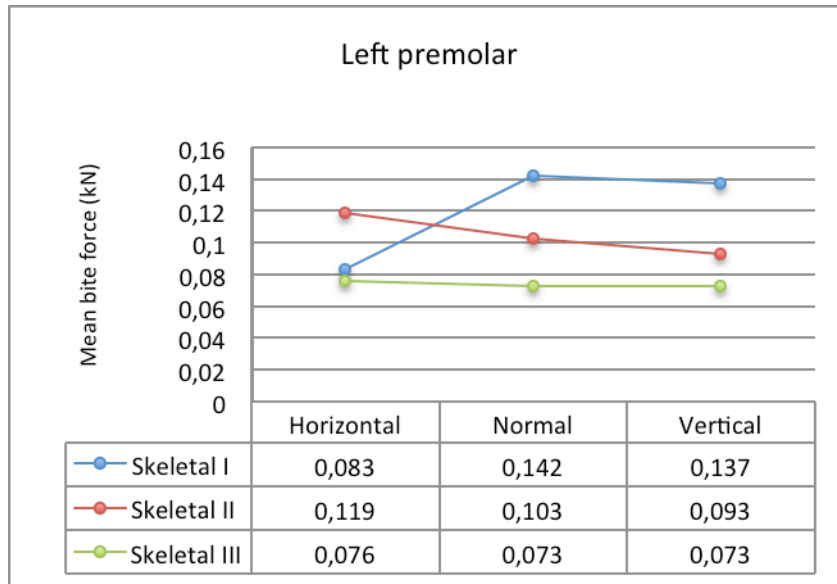


Figure 20: Linear graph mean force PM\_L

Left molar:

**Table 9: Main effect comparison (Molar L)**

Number of obs =		360	R-squared =		0.0301
Root MSE =		.099727	Adj R-squared =		0.0051
Source	Partial SS	df	MS	F	Prob > F
-----+-----					
Model	.107935622	9	.011992847	1.21	0.2901
grwth_dir	.008386651	2	.004193326	0.42	0.6563
skel_Class	.035631612	2	.017815806	1.79	0.1683
grwth_dir#skel_Class	.032655614	4	.008163904	0.82	0.5125
age	.044108197	1	.044108197	4.43	0.0359
Residual	3.48092747	350	.009945507		
-----+-----					
Total	3.5888631	359	.009996833		

**Table 10: Mean bite force (Molar\_L)**

-----			
	skel_Class		
grwth_dir	Class I	Class II	Class III
-----+-----			
Horizontal	0.197	0.201	0.171
Normal	0.193	0.172	0.175
Vertical	0.200	0.157	0.178
-----			

Analysis of the Left Molar mean bite force Table 9 and Table 10, found no significance between growth direction (p=0,6563) and No

significance between Skeletal classes. There was also no interaction of the growth direction and Skeletal Class.

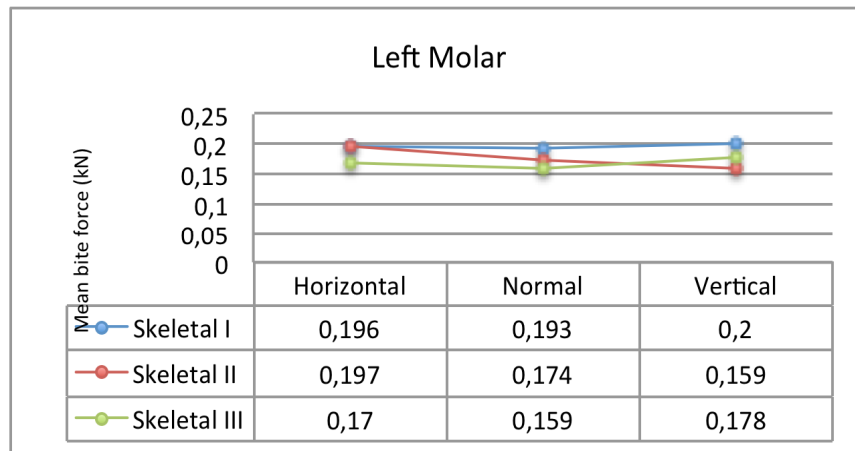


Figure 21: Linear graph mean force Molar\_L

There was however significance in the age ( $P=0,0359$ ), the covariate i.e. age was set to mean. Linear depiction of the values display these findings, with Class I and III behaving rather similarly, a drop in the mean values of Skeletal Class II subjects as they progress from Horizontal to Vertical can be observed, however this has no statistical significance ( $P>0,05$ )

Right Premolar:

Table 11: Main effect comparison (PM\_R)

Source	Partial SS	df	MS	F	Prob > F
Model	.234354751	9	.026039417	5.22	0.0000
grwth_dir	.013482215	2	.006741107	1.35	0.2605
skel_Class	.059887788	2	.029943894	6.00	0.0027
grwth_dir#skel_Class	.055263401	4	.01381585	2.77	0.0274
age	.138816616	1	.138816616	27.81	0.0000
Residual	1.7471898	350	.004991971		
Total	1.98154455	359	.005519623		

Analysis of the left premolar found that the growth direction did not differ significantly  $P=0,2605$ . However there was a significant difference in the Skeletal Classes with  $P=0,0027$ . There was a significant interaction between the growth direction and Skeletal Class ( $P=0,0274$ ); in particular the interaction can be attributed to the Horizontal Class II (Table 11 and Table 12).

A slight interaction in the Vertical growth group can also be observed where there is a decrease in the Force of the Skeletal II Class and an increase in Force of the Skeletal Class III (Figure 22).

Table 12: Mean bite force (PM right)

grwth_dir	skel_Class		
	Class I	Class II	Class III
Horizontal	0.119	0.149	0.089
Normal	0.126	0.104	0.086
Vertical	0.121	0.097	0.102

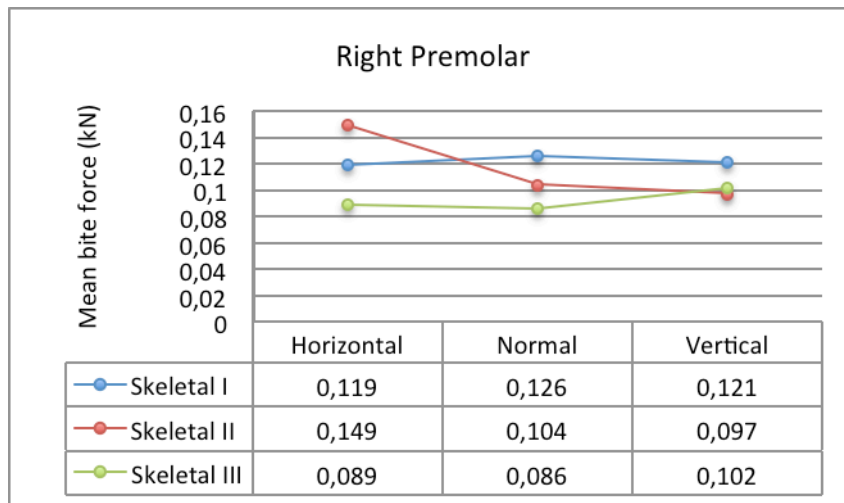


Figure 22: Linear graph mean force PM\_R

Right Molar:

Table 13: Main effect comparison (Molar\_R)

Source	Partial SS	df	MS	F	Prob > F
Model	.277697554	9	.030855284	2.73	0.0043
grwth_dir	.027086903	2	.013543452	1.20	0.3025
skel_Class	.05254643	2	.026273215	2.33	0.0991
grwth_dir#skel_Class	.150676285	4	.037669071	3.34	0.0106
age	.083233123	1	.083233123	7.37	0.0069
Residual	3.95115534	350	.011289015		
Total	4.2288529	359	.011779535		

The analysis of the Right Molar displayed similar finding to the Left Molar. Growth direction did not differ significantly with a P=0,3025. The Skeletal Class though not quite significant, was marginally close with a P=0.0991. There was however a significant interaction between growth direction and Skeletal pattern (P=0,0106) (Table 13). When the data was delineated we are able to locate the source of this interaction, it appears to be attributed to Horizontal Class II. There is also an interaction visible in the Vertical Class II group (Figure 23).

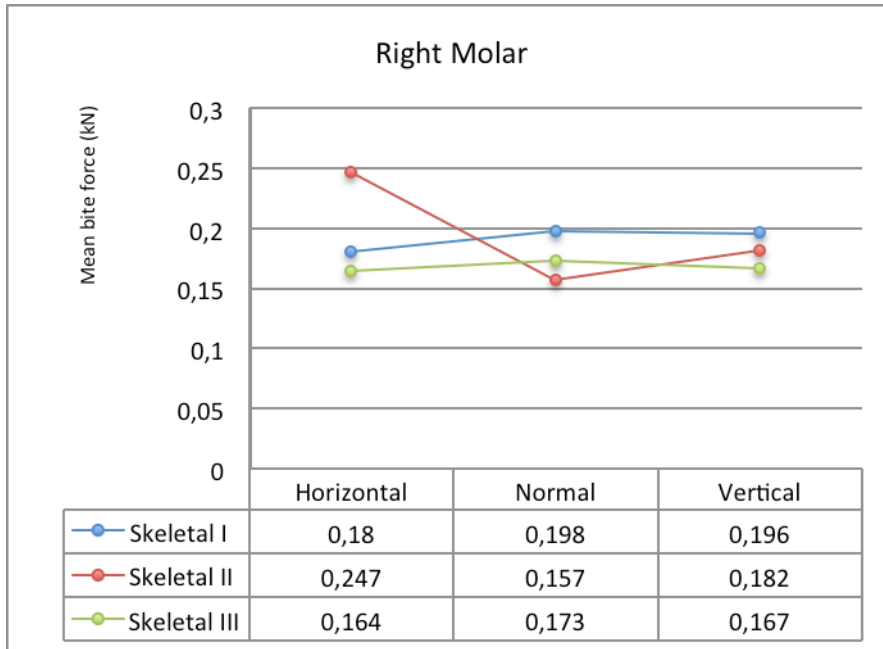


Figure 23: Linear graph mean force Molar\_R

Further testing using the ANOVA (m test) was carried out on the data of the five parameters (i.e. bite forces) to assess whether a significant difference in the readings of the 5 parameters between the three Skeletal classes within each growth direction and furthermore to assess differences between different growth directions within individual Skeletal classes. In the tables below (1) Horizontal: Normal (2) Horizontal: Vertical and (3) Normal: Vertical are compared. The Skeletal classes are compared as (1) Class I: Class II, (2) Class I: Class III and (3) Class II: Class III.

## Incisors:

**Table 14: Horizontal growth (Incisors)**

	F(df, 350)	df	p
(1)	3.88	1	0.0498 #
(2)	0.34	1	0.5575 #
(3)	6.34	1	0.0122 #
all	3.48	2	0.0319

In the Horizontal Growth (Table 14) group-in the incisor region a slight significant difference between Skeletal Class I and Skeletal Class II ( $P=0,0498$ ) can be noted, no significant difference between Skeletal Class I and Class II ( $P=0,5575$ ) and a significant difference between Skeletal Class II and Class III ( $P=0,0122$ ).

**Table 15: Normal growth (Incisors)**

	F(df, 350)	df	p
(1)	4.03	1	0.0454 #
(2)	1.79	1	0.1816 #
(3)	11.37	1	0.0008 #
all	5.76	2	0.0035

In the Normal growth group (Table 17), there was a significant difference between Skeletal Class I and Class II ( $P=0,0454$ ), no

significant difference between Skeletal Class I and III ( $P=0,1816$ ) and a significant difference in Skeletal Class II and Class III ( $P=0,0008$ ).

For Vertical growth (Table 18), we found a significant difference in Skeletal I and II ( $P=0,0140$ ), no significance between Skeletal Class I and III or between Skeletal Class II and III ( $P$  values  $>0,05$ )

**Table 16: Vertical growth (Incisors)**

	F(df,350)	df	p
(1)	6.09	1	0.0140 #
(2)	1.55	1	0.2133 #
(3)	1.50	1	0.2220 #
all	3.05	2	0.0487

**Table 17: Skeletal Class I (Incisors)**

	F(df,350)	df	p
(1)	0.20	1	0.6531 #
(2)	0.07	1	0.7864 #
(3)	0.03	1	0.8584 #
all	0.10	2	0.9021

In Class I (Table 19) there was no significant differences between Growth directions reported between Horizontal and Normal, Horizontal and Vertical or Normal and Vertical ( $P>0,05$ ).

**Table 18: Skeletal Class II comparison (incisor)**

	F(df, 350)	df	p
(1)	0.25	1	0.6176 #
(2)	16.84	1	0.0001 #
(3)	21.84	1	0.0000 #
all	13.07	2	0.0000

In Class II (Table 20), we found no significant difference between Horizontal and Normal groups ( $P=0,6167$ ), however we did find a significant difference between Horizontal and Vertical groups where  $P=0,0001$ . There was also a significant difference between the Normal and Vertical group ( $P<0,05$ ).

In Class III, there was no significant difference found amongst the growth directions all  $P > 0,05$  (Table 21).

**Table 19: Skeletal Class III comparison (Incisor)**

	F(df, 350)	df	p
(1)	0.10	1	0.7491 #
(2)	0.18	1	0.6734 #
(3)	0.01	1	0.9383 #
all	0.08	3	0.9687

*In the left premolar area:*

**Table 20: Horizontal (left pm)**

	F(df, 350)	df	p
(1)	4.50	1	0.0345 #
(2)	0.17	1	0.6778 #
(3)	6.26	1	0.0128 #
all	3.60	2	0.0283

Horizontal growth direction:  
Significant difference was found between Skeletal Class I and Class II (P=0,0345), as well as between Skeletal Class II and III (P=0,0128). However there was no significant difference between Skeletal Class I and III (P=0,6778) (Table 22).

*In the right premolar area:*

The Horizontal growth category found marginally significant differences between Skeletal Class I and Class II subjects (P=0,0558 and P=0,0587 respectively). The difference between Skeletal Class II and Class III subjects displayed a marked difference, P=0,0002 (Table 23).

**Table 21: Horizontal (PM\_R)**

	F(df, 350)	df	p
(1)	3.68	1	0.0558 #
(2)	3.60	1	0.0587 #
(3)	14.06	1	0.0002 #
all	7.03	2	0.0010

In the Normal growth direction group (Table 24), there was a reported significant difference between Skeletal Class I and Class II, Skeletal Class I and III with  $P=0,0264$  and  $P=0,0001$  respectively. However no significant difference between Skeletal Class II and III was found ( $P=0,0825$ ).

Table 22: Normal growth (PM\_L)

	F(df,350)	df	p
(1)	4.97	1	0.0264 #
(2)	15.66	1	0.0001 #
(3)	3.03	1	0.0825 #
all	7.86	2	0.0005

The Normal group (Table 25) showed a significant difference between Skeletal Class I and Class III ( $P=0,0134$ ). There was no significant difference between Skeletal Class I and II, nor Skeletal Class II and III ( $P>0,05$ ).

Table 23: Normal growth (PM\_R)

	F(df,350)	df	p
(1)	1.91	1	0.1683 #
(2)	6.17	1	0.0134 #
(3)	1.24	1	0.2667 #
all	3.10	2	0.0465

In the Vertical growth direction group (Table 26), significant differences were found between Skeletal Class I and II and Skeletal Class I and III with  $P=0,0124$  and  $P=0,0006$  respectively. There was no significant difference between Skeletal II and III ( $P>0,05$ ).

**Table 24: Vertical growth (PM\_L)**

	F(df,350)	df	p
(1)	6.32	1	0.0124 #
(2)	12.12	1	0.0006 #
(3)	0.93	1	0.3364 #
all	6.46	2	0.0018

Analysis of Vertical Growth (Table 27) right Premolar data revealed that there was no clinical significance in any of the interactions. All P values were greater than 0,05.

**Table 25: Vertical growth (PM\_R)**

	F(df,350)	df	p
(1)	2.33	1	0.1281 #
(2)	1.56	1	0.2131 #
(3)	0.08	1	0.7802 #
all	1.32	2	0.2684

Assessment of Premolar values of different growth direction subjects within a Skeletal Class found the following.

For Skeletal Class I (Table 28), Significant differences were found between Horizontal and Normal ( $P=0,0006$ ) as well as between Horizontal and Vertical ( $P=0,0016$ ). No significance was detected between Normal and Vertical ( $P=0,7662$ ).

Table 26: Class I (PM\_L)

	F(df,350)	df	p
(1)	11.92	1	0.0006 #
(2)	10.07	1	0.0016 #
(3)	0.09	1	0.7662 #
all	7.43	2	0.0007

The Right Premolar (Table 29) Growth direction comparison within Skeletal Class I found no significance to growth direction, in comparison to the differences found on the left premolar. ( $P>0,05$ )

Table 27: Class I (PM\_R)

	F(df,350)	df	p
(1)	0.20	1	0.6569 #
(2)	0.03	1	0.8739 #
(3)	0.08	1	0.7769 #
all	0.10	2	0.9040

For Skeletal Class II, no significant difference was detected as P values were larger than 0,05 (Table 30).

**Table 28: Class II (PM\_L)**

	F (df, 350)	df	p
(1)	0.83	1	0.3632
(2)	2.17	1	0.1413
(3)	0.33	1	0.5680
all	1.10	2	0.3332

For Skeletal Class III significance was only found between the Horizontal and Vertical patients with a P=0,0054. No other significance was detected (Table 31).

**Table 29: Class III (PM\_L)**

	F (df, 350)	df	p
(1)	0.02	1	0.8884 #
(2)	7.85	1	0.0054 #
(3)	0.04	1	0.8373 #
all	3.37	3	0.0187

In the Skeletal Class II group a significant difference was found between Horizontal and Normal subjects (P=0,0052) and between Horizontal and Vertical (P=0,0013) (Table 32). There was no significant difference between Normal and Vertical subjects.

**Table 30: Class II (PM\_R)**

	F (df, 350)	df	p
(1)	7.92	1	0.0052 #
(2)	10.46	1	0.0013 #
(3)	0.18	1	0.6735 #
all	6.13	2	0.0024

In Skeletal Class III analysis, no significant difference was reported, all the categories have a P value greater than 0,05 (Table 33)

	F (df, 350)	df	p
(1)	0.03	1	0.8521 #
(2)	0.04	1	0.8323 #
(3)	0.94	1	0.3334 #
all	0.36	3	0.7810

**Table 31: Class III (PM\_R)**

The analysis of Class I Left Molar values within the various growth direction categories established that Skeletal classes did not differ significantly (statistically) within the Horizontal growth direction group, P values all larger than 0,05 (Table 34).

**Table 32: Horizontal growth (PM\_L)**

	F (df, 350)	df	p
(1)	0.04	1	0.8509 #
(2)	1.41	1	0.2366 #
(3)	1.81	1	0.1792 #
all	1.08	2	0.3410

Skeletal Classes (Table 35) did not differ significantly in the Normal growth direction group either ( $P > 0,05$ ).

**Table 33: Normal growth (PM\_L)**

	F (df, 350)	df	p
(1)	0.83	1	0.3633 #
(2)	0.65	1	0.4211 #
(3)	0.01	1	0.9162 #
all	0.49	2	0.6107

Horizontal Right Molar analysis established a significant difference between Skeletal Class I and II ( $P = 0,0054$ ), as well as between Skeletal Class II and Class III ( $P = 0,0006$ ). There is no significant difference between Skeletal Class I and III groups in the Horizontal growth direction ( $P > 0,05$ ) (Table 36).

**Table 34: Horizontal growth (PM\_R)**

	F (df, 350)	df	p
(1)	7.84	1	0.0054 #
(2)	0.50	1	0.4781 #
(3)	11.96	1	0.0006 #
all	6.69	2	0.0014

In Normal growth (Table 37) the Right Molar displayed no statistical significance in the Skeletal Classes comparatively ( $P > 0,05$ ).

**Table 35: Normal growth (PM\_R)**

	F (df, 350)	df	p
(1)	2.93	1	0.0876 #
(2)	1.10	1	0.2949 #
(3)	0.45	1	0.5042 #
all	1.49	2	0.2270

A significant difference between Skeletal I and II patients in the Vertical growth direction group was noted ( $P=0,0545$ ) this is a marginally significant P value, (Table 38).

**Table 36: Vertical growth (PM\_L)**

	F (df, 350)	df	p
(1)	3.72	1	0.0545 #
(2)	1.00	1	0.3186 #
(3)	0.87	1	0.3522 #
all	1.86	2	0.1569

There was no significant difference between Skeletal I, II and III subject comparison of Right Molar values within the Vertical growth group ( $P>0,05$ ) (Table 39).

**Table 37: Vertical growth (PM\_R)**

	F (df, 350)	df	p
(1)	0.37	1	0.5429 #
(2)	1.51	1	0.2196 #
(3)	0.38	1	0.5362 #
all	0.76	2	0.4703

Analysis of Left Molar data values when determining the relationship between different growth patterns within individual Skeletal classes supplied the following findings.

For Class I subjects (Table 40) there were no significant differences between subjects belonging to one of the three growth directions ( $P < 0,05$ ).

In the Skeletal Class I group (Table 41), the f the Right molar displayed no clinically significant differences in all three defined growth directions ( $P > 0,05$ ).

**Table 38: Class I (Molar\_L)**

	F (df, 350)	df	p
(1)	0.04	1	0.8517 #
(2)	0.03	1	0.8687 #
(3)	0.12	1	0.7285 #
all	0.06	2	0.9414

**Table 39: Class I (Molar\_R)**

	F (df, 350)	df	p
(1)	0.58	1	0.4475 #
(2)	0.45	1	0.5051 #
(3)	0.01	1	0.9230 #
all	0.35	2	0.7068

For Class II subjects (Table 42) there was a marginal significant P value for a difference in the comparison of Horizontal and Vertical subjects (P=0,0549). The clinical significance of this relationship bears importance. There are not significant differences between the Horizontal and Normal subjects or between Normal and Vertical subjects.

**Table 40: Class II (Molar\_L)**

	F (df, 350)	df	p
(1)	1.61	1	0.2050 #
(2)	3.71	1	0.0549 #
(3)	0.44	1	0.5065 #
all	1.91	2	0.1500

Skeletal Class II subject analysis (Table 43) produced a significant difference when comparing Horizontal to Normal (P=0,0003) and When comparing Horizontal to Vertical (P=0,0073).

There was no significant difference in the Normal to Vertical comparison (P>0,05).

**Table 41: Class II (Molar\_R)**

	F (df, 350)	df	p
(1)	13.68	1	0.0003 #
(2)	7.29	1	0.0073 #
(3)	1.04	1	0.3091 #
all	7.27	2	0.0008

Amongst Class III subjects (Table 44), no statistical significance differences can be deduced amongst different growth directions. All  $P > 0,05$

Significant differences were also not found in the Vertical subject values, all reporting P values higher than 0,05 (Table 45).

**Table 42: Class III (Molar\_L)**

	F (df, 350)	df	p
(1)	0.03	1	0.8626 #
(2)	0.00	1	0.9967 #
(3)	0.03	1	0.8713 #
all	0.05	3	0.9866

**Table 43: Class III (Molar\_R)**

	F (df, 350)	df	p
(1)	0.16	1	0.6890 #
(2)	0.29	1	0.5923 #
(3)	0.07	1	0.7877 #
all	0.20	3	0.8938

The analysis of Class I Left Molar values with in the various growth direction categories established that Skeletal Classes did not differ significantly (statistically) within the Horizontal growth direction group. P values were all larger than 0,05 (Table 46).

**Table 44: Horizontal growth (Molar\_L)**

	F (df, 350)	df	p
(1)	0.04	1	0.8509 #
(2)	1.41	1	0.2366 #
(3)	1.81	1	0.1792 #
all	1.08	2	0.3410

Skeletal classes did not differ significantly in the Normal growth direction group either (P>0,05) (Table 47).

**Table 45: Normal growth (Molar\_L)**

	F (df, 350)	df	p
(1)	0.83	1	0.3633 #
(2)	0.65	1	0.4211 #
(3)	0.01	1	0.9162 #
all	0.49	2	0.6107

Horizontal Right Molar analysis (Table 48) established a significant difference between Skeletal Class I and II (P=0,0054), as well as between Skeletal Class II and Class III (P=0,0006). There is no significant difference between Skeletal Class I and III groups in the Horizontal growth direction (P>0,05)

**Table 46: Horizontal growth (Molar\_R)**

	F (df, 350)	df	p
(1)	7.84	1	0.0054 #
(2)	0.50	1	0.4781 #
(3)	11.96	1	0.0006 #
all	6.69	2	0.0014

In Normal growth the Right Molar displayed no statistical significance in the Skeletal Classes comparatively (P>0,05)

**Table 47: Normal growth (Molar\_R)**

	F (df, 350)	df	p
(1)	2.93	1	0.0876 #
(2)	1.10	1	0.2949 #
(3)	0.45	1	0.5042 #
all	1.49	2	0.2270

A significant difference between Skeletal I and II patients in the Vertical growth direction group was noted ( $P=0,0545$ ). While this is a marginally significant P value, it may have clinical significance (Table 50).

**Table 48: Vertical growth (Molar\_L)**

	F(df, 350)	df	p
(1)	3.72	1	0.0545 #
(2)	1.00	1	0.3186 #
(3)	0.87	1	0.3522 #
all	1.86	2	0.1569

There was no significant difference between Skeletal I, II and III subject comparison of Right Molar values within the Vertical growth group ( $P>0,05$ ) (Table 51).

**Table 49: Vertical growth (Molar\_R)**

	F(df, 350)	df	p
(1)	0.37	1	0.5429 #
(2)	1.51	1	0.2196 #
(3)	0.38	1	0.5362 #
all	0.76	2	0.4703

## Chapter 5: Discussion

This research project intended to identify the significant variances of the maximum bite force in malocclusions and junctures of dentofacial development. The aim was to ascertain the existence of a relationship between force and facial form within a sample of the South African population. Such studies have not been previously conducted on the local population, in which anterior open bite (Vertical growth) (Figure 30), as well as deep bite Class II malocclusions are often diagnosed.

Traditional studies connected to facial morphology, specifically when studying Vertical types have been documented, predominantly in the readings of the overbite and over-jet relationships (Fields et al., 1984). Studies have also been conducted on the relative mandibular plane angles associated with specific facial growth types i.e. Vertical or Horizontal or Normal. The assessments were conducted formerly to gauge the differentiations in facial morphology concomitant to different craniofacial growth types (Fields, et al., 1984). It is evident that subjects demonstrating a Vertical facial form have a tendency to present with an increase in the lower facial height, conversely Brachifacial subjects (Horizontal facial form) display a decrease in the lower facial height (Figure 32 and **Figure 41**)

According to Fields and Proffit (1984) the studies that were executed to assess growth, have emphasised on morphological factors associated with long, Normal and short face subjects. Subjects in the study by Fields and co-workers (1984) were in an extensive range, spanning from childhood to adulthood. The study analysis was assenting that an increase in Vertical growth is clear and the subject is referred to having “long face syndrome”. This is synonymous with a decrease in the over bite and often may also consequent in an open bite. (Fields, et al., 1984).

A sample of the facial prototypes and their malocclusion that were used in this study are illustrated below (Figure 24 - Figure 34)



Figure 24: Vertical facial form



Figure 25: Normal facial form



Figure 26: Horizontal facial form



Figure 27: Anterior open bite



Figure 28: Lateral view of anterior open bite

Intra-oral manifestations:



Figure 29: Left molar relationship of open bite



Figure 30: Anterior intraoral of open bite



Figure 31: Right molar intraoral open bite



Figure 32: Deep bite



Figure 33: Posterior view of deep bite occlusion



Figure 34: Intraoral of Skeletal Class III Vertical

Maximum bite force has been defined as “when the mandible is elevated due to an active force by masticatory muscles, there is an effort exerted between the occlusal surfaces of maxillary and mandibular teeth” (Marquezin et al., 2013). In order to identify associations between demographic variables (Age, sex, ethnicity) and the main variables of interest (bite force and growth directions) a series of preliminary analysis were conducted. A significant positive correlation was found between Growth direction and Age ( $P < 0,05$ ) (Table 5). However when analysis of subject distribution of subjects aged 14 years and above those below the age of 14 years it was found that 61,94% of subjects were below the age of 14 years and 38,06% were equal or above the age of 14 years. This information disclosed a trend associated with age and the occlusal force of the growth directions

as well as skeletal classes. However the non-uniform distribution of ages amongst the categories of the main effects of this study (Growth direction and Skeletal Class) coupled with the significance of age could affect the outcome of the interaction and for this reason age as a covariate was set to a mean of 15.6333 years to be used in all the tests.

180 Males and 180 Females were tested, thus the sex distribution of subjects was equally split (50%). Within each sex category the ethnic distribution of subjects was categorized into the following categories: African, White, Indian and Coloured. The female subject group consisted of 53% African, 28% White, 17% Indian, and 2% Coloured. Ethnic subject distribution amongst males was found to be 48% African, 22% White, 22% Indian and 8% Coloured. The distribution of ethnicity amongst the study population was assessed to evaluate the presentation of the randomly selected subject pool and representative of the South African population. Analysis of the data to test for variance in maximum bite forces between male and female subjects found no significant difference or interaction as the  $P > 0,05$ .

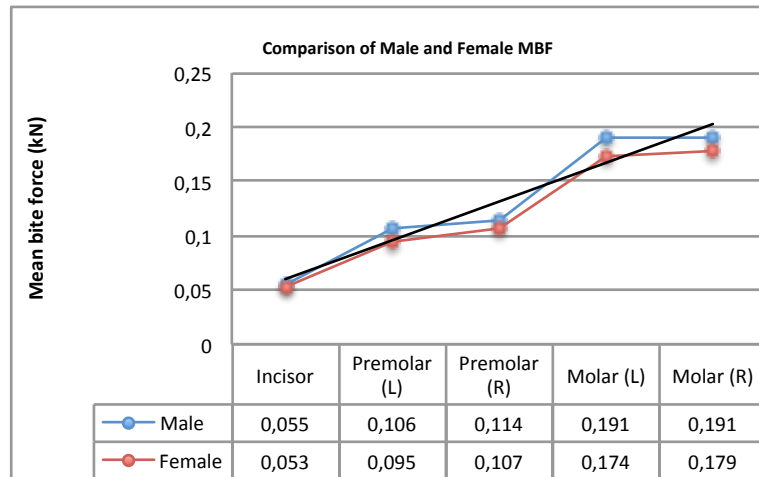


Figure 35: Comparison of gender force

Male and female Bite forces show a similar pattern of increment as they pass from incisor to molar, with male force being slightly increased as displayed in **Figure 35**. In this study there was no significance in the difference of bite forces relative to different genders. These findings do not corroborate with the outcomes of the studies conducted by Palinkas and co-workers in 2010, or with the findings of Braun and co-workers in 1995. The reason for no statistical maximum bite force gender difference significance in this study may have to do with the imbalance of age within the population group of this study. The proximate 60% to 40% child to adult split in the population group indicates that the potential for an increase in susceptibility to operational or subject related factors that influence the predictability of the clinical study. Some examples of relevant factors could be placement of the meter, size of the meter, patient sensitivity, responsiveness and accuracy of the patient (Braun et al., 1995).

Age is significant in the assessment of maximum bite forces however in this study it was not possible to determine the degree of significance due to the inequalities of age amongst the subject pool as heretofore discussed. Garcia-Morales and co-workers (2003) conducted a similar study and identified loopholes in the comparative analysis of bite forces when using younger subjects (Garcia-Morales et al., 2003). A loophole of distinct relevance proposed the notion that focuses on the susceptibility of younger subjects to sensitivity or discomfort. This proposition can be associated with an inconsistency in the increased force execution that is necessary to achieve a maximum bite force. Consequent to which may result in disparities amongst readings taken. The studies conducted by Proffit and Fields as reported by Kiliaridis (Kiliaridis et al, 2003), focused upon the assessment of bite force differences amongst long and short-faced children. Slight discrepancies were detected in the comparison of occlusal force readings, thus rendering the study findings of little contributory value. However, when similar studies were carried out on adult subjects differences were detected. Dolicofacial adults exhibited a lower occlusal force (Kiliaridis et al., 2003).

This information may prove to be useful in establishing a difference in pre-growth and post-growth bite force measurements and a causative change that is only apparent in post pubertal subjects. There may however in the execution of this research project, as in similar previous

studies, be the influence of patient sensitivity influential to the maximum bite force assessment of juvenile subjects.

This study found that Skeletal Class and growth direction both have a significant effect on bite forces. There is also an interaction between Skeletal classes and growth direction. The findings therefore reject the initial hypothesis that a relationship does not exist between maximum occlusal forces and facial form (direction of growth). A second finding was that bite forces do differ amongst Skeletal classes and therefore may contribute to the development of malocclusion. The extent of the differences and interactions are validated by the statistical data analysis and presented in the form of tables and graphs (presented in the results section). Details of interaction and disparity amongst the Skeletal classes and growth directions relative to anterior and posterior occlusal forces will be discussed.

In the anterior segment, force was measured at the midpoint of 11 and 21. It was found that the Horizontal and Normal growth pattern performed similarly (**Figure 19**, Table 16). However in Skeletal Class II Vertical subjects there was decrease in the occlusal force over this area. Previous studies indicate that Vertical subjects with an anterior open bite (**Figure 34**) display a bite force half the strength of subjects from a Normal growth group (Braun, et al., 1995). While the findings of this study did not detect a significant difference in the incisor region of Vertical subjects the linear plot (**Figure 19**), indicates that there is a

clinically significant difference that exists between Vertical and Normal subjects. Measurements in the incisor region of Horizontal growth subjects found a slight significant difference between Skeletal Class I and Skeletal Class II ( $P=0,0498$ ), no significant difference between Skeletal Class I and Class II ( $P=0,5575$ ) and a significant difference between Skeletal Class II and Class III ( $P=0,0122$ ) (Table 14: Horizontal growth (Incisors)). Contrary to the Vertical growth pattern and associated force relationship, Braun and co-workers found that Brachifacial patients with accompanying deep bites and intruded molars present with an increase in force (Braun et al., 1995). Analysis of the data presented is in agreement with this finding of Braun and co-workers, across all the line graph plots. It is apparent that there is a general increase in force of Skeletal Class II Horizontal growth subjects (table 20, **Figure 37** and **Figure 39**). Analysis of data in Horizontal growth direction group also reveals that there is a difference in magnitude of force between Skeletal Class II and Class III subjects ( $P=0,0122$ ). The Skeletal Class II display an increased force in comparison to Skeletal Class III (Table 16, **Figure 39**, **Figure 40**, and **Figure 41**). A comparison of Skeletal Class II subjects amongst growth direction groups report a difference in bite force between Horizontal and Vertical subjects ( $P=0,0001$ ) (Table 20, and Table 21). Bite forces reflect the relation of the jaws geometrically and the mechanical lever-like architecture (Fields et al., 1984). Amongst Vertical growth subjects there is a mainstream profile that is often found. This profile identified by the long face accompanied by an anterior open bite or decrease in the over bite

relationship (Fields, et al., 1984). The presence amongst this profile can be accountable for the decrease in magnitude of force displayed over the incisor region in Vertical growth. Fields et al (1984) suggests according to cephalometric study that this discrepancy can be associated with the morphological development of the mandible. The study conducted by Schendel and associates (Schendel et al., 1976) indicated that an increase in the mandibular plane angle is associated with a decrease in the ramus height.

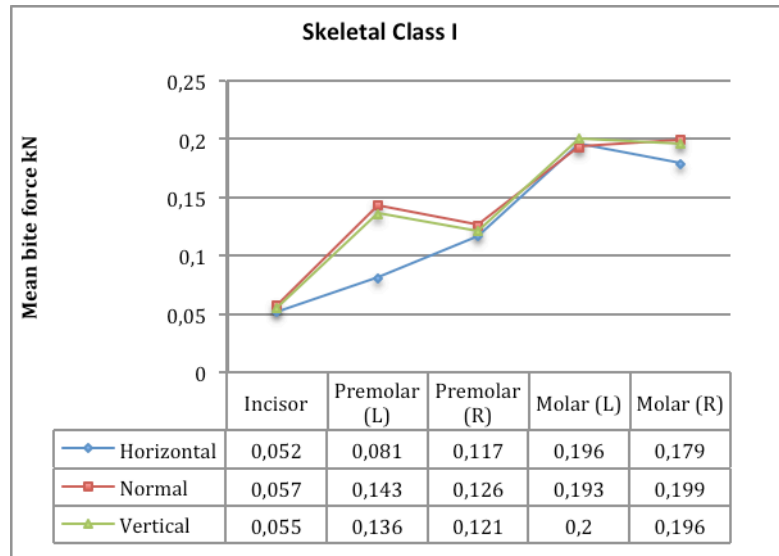


Figure 36: Skeletal Class I comparison

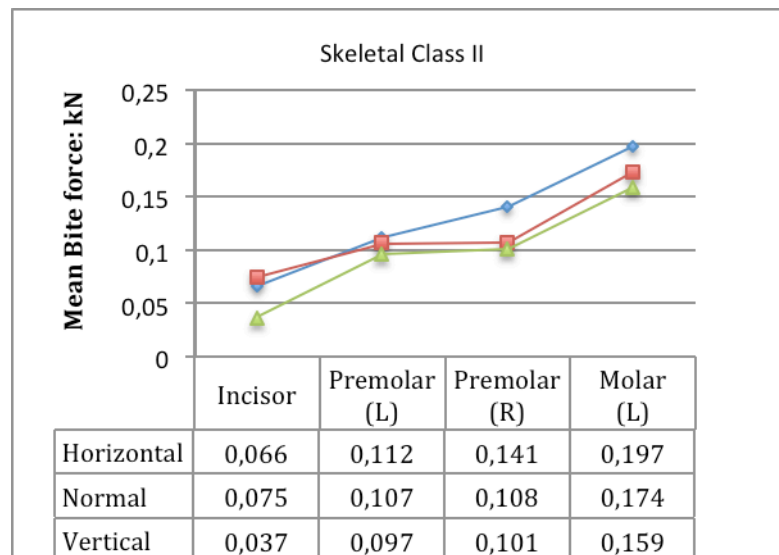


Figure 37: Skeletal Class II comparison

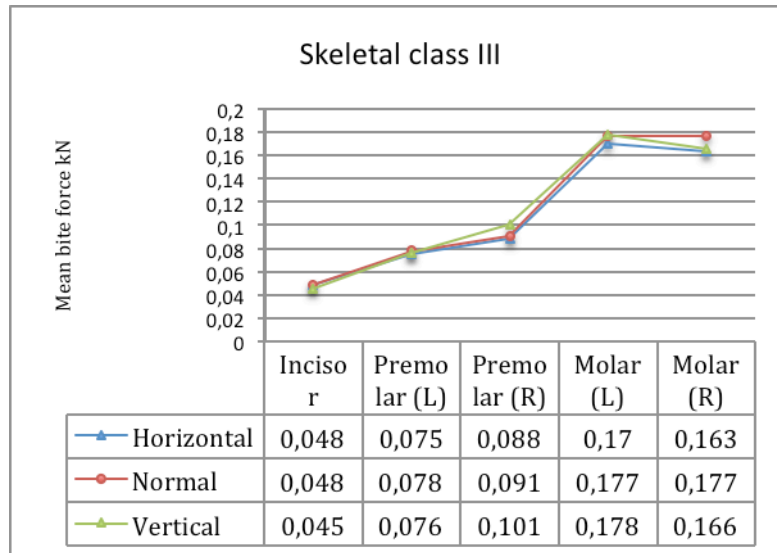


Figure 38: Skeletal Class III comparison

Over the Premolar area in the Vertical growth effect group there is a steady decline in force amongst the Skeletal Classes, from Class I to Class III (**Figure 20**, **Figure 22**). There are significant differences in the magnitude of force between Skeletal Class I and II and Skeletal Class I and III with  $P=0,0124$  and  $P=0,0006$  respectively (Table 26 and Table 27). In the premolar area there is an increase in force of the Horizontal Skeletal Class II subjects, this increase is associated with a progression across the growth directions i.e. Horizontal, Normal and Vertical growth. In the area of the premolar, in Horizontal growth subjects there is no difference in force between Class I and III Skeletal subjects. They display a similar force. However in the Horizontal growth direction group a difference between forces in Skeletal Class I and Class II, as well as between Skeletal Class II and III (Table 22 and Table 23, **Figure 20**, **Figure 22**.) is evident. P values are conducive to significance for differences between Skeletal Class I and II and Skeletal Class I and III with  $P=0,0124$  and  $P=0,0006$  respectively:.

In the Horizontal growth group Skeletal Class I subjects have a weaker force than Skeletal Class II subjects, and Skeletal Class II have a markedly larger premolar force than Skeletal Class III (**Figure 20** and **Figure 22**). Conclusively the Class II Horizontal subjects have increased forces in the premolar area, Class I subjects fall in the middle, and Class III subjects exhibit the weakest forces in the Horizontal growth direction.

For Normal growth in the premolar area observation of interactions between Skeletal Class I and II subjects as well as between Skeletal Class I and III occur with  $P=0,0264$  and  $P=0,0001$  respectively (Table 24 and Table 25). In Vertical growth group there exists a similar pattern between Skeletal Class I and II as well as between Skeletal Class I and III Skeletal Class I and II and Skeletal Class I and III with  $P=0,0124$  and  $P=0,0006$  respectively (Table 26 and Table 27).

Incongruence also arose within the premolar group when comparing left to right sides. This is evident by the mapping of data in **Figure 20**, **Figure 22**. The origin of the discrepancy may be attributed to the presence of subjects in primary, permanent and mixed dentition within the population group. Another important point that should be factored into this study is the tendency for subjects to favour a particular side during function this will need further investigation.

When bite forces were looked at over the Skeletal classes to identify the difference in premolar force between growth directions, in Skeletal Class I, significant differences were found between Horizontal and Normal ( $P=0,0006$ ) as well as between Horizontal and Vertical ( $P=0,0016$ ). The force in Horizontal growers is less than that of Normal growers. Horizontal growers also have a larger force than Vertical growers **Figure 36**.

The premolar mean bite force of Skeletal Class II subjects in the population group indicated a significant difference between Horizontal and Normal subjects ( $P=0,0052$ ), where the magnitude of force in the Horizontal growth direction group was increased over the force recorded in the Normal growth direction population group (Table 30 and Table 32). There is also a significant difference between Horizontal and Vertical ( $P=0,0013$ ), as a result of the magnitude of the force in the Horizontal growth group (Figure 37)

It can therefore be stated that there exists within Skeletal Classes differences in bite force. The magnitude of mean bite force in the premolar area shows in this study that Horizontal growth pattern displays a larger mean bite force over Vertical and Normal growers. Skeletal Class I and Class III display similarity in bite force amongst the different growth directions i.e. Horizontal, Normal and Vertical. Class III Vertical growth direction subjects display a lower force. According to a study carried out in 1984 by Fields and co-workers, weak masticatory muscles are a major cause of Vertical growth patterns (Fields, et al., 1984). A separate study went on to report on the role of the adductor muscles of the mandible relative to the ramus and gonial angle. With a Vertical ramus and relatively acute gonial angle, it was found that there is an inversely proportionate relationship apparent between gonial angle and muscle force. As the gonial angle increase, a reduction in the mechanical advantage of the muscle was observed. The resulting force was less perpendicular to the occlusal plan in this instance (Braun, et

al., 1995). This finding infers that bite force may be reflective of facial form (Braun, et al., 1995). A decrease in the gonial angle as expected in subjects displaying deep bites or Horizontal facial form, may increase the mechanical advantage of the muscle, which will be reflected in the facial form. Great Vertical growth or hyper-divergence consequential to a decrease in the moment arms (Garcia-Morales, et al., 2003) will also cause a decrease or lower maximum bite force. The bite force referred to here is believed to be associated with malocclusions which present with increases in the overbite or decrease in the overbite resulting in open bite and cross bite anomalies

Garcia-Morales, et al, (2003) concluded that independent of the age, larger moment arms of the mandible requires less activity in order to achieve a given force. The relationship between hyper-divergent subjects and lower bite force is an important finding of this study, and can be used to reach a conclusive point regarding the presence of lower bite forces in long faced individuals.

Over the molar region in the Horizontal growth group there are difference in the force of Skeletal Class I and II ( $P=0,0054$ ) with Class II having an increased force over Class I, as well as between Skeletal Class II and Class III ( $P=0,0006$ ), Class II having an increased force over Class III Skeletal Class I and II ( $P=0,0054$ ), (Table 48 and Table 46, **Figure 21** and **Figure 23**)

Subjects in the Normal growth pattern display no marked difference in molar bite force, Skeletal Class I, II, and III exhibits relativity amongst the magnitude of force.

In Vertical growth pattern group there are significant differences between Skeletal Class I and Class III ( $P=0,0545$ ) (Table 50 and Table 51), while this is a statistically marginal value in terms of significance, a graphical presentation elicits a potential clinical significance. The graph displays Class III subjects displaying a decrease in force when compared to Skeletal Class I (Figure 41)

Skeletal Class II subjects with a Horizontal growth direction display in remarked increase in molar force in comparison to the Normal growers and the Vertical growers (Figure 37).

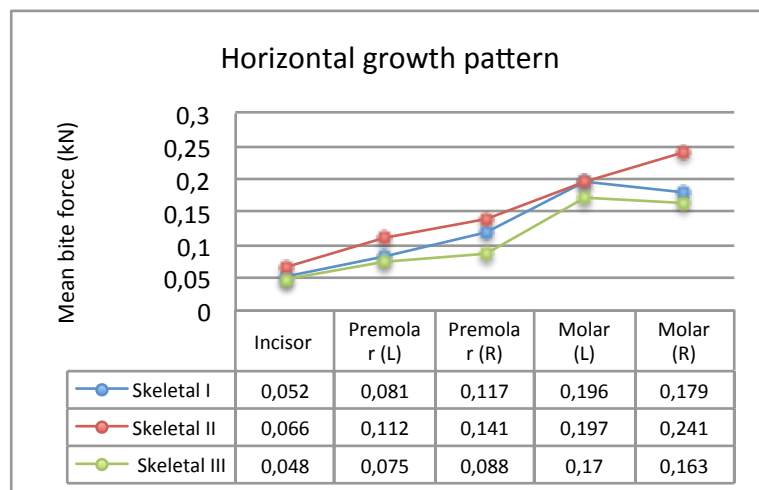


Figure 39: Horizontal growth comparison

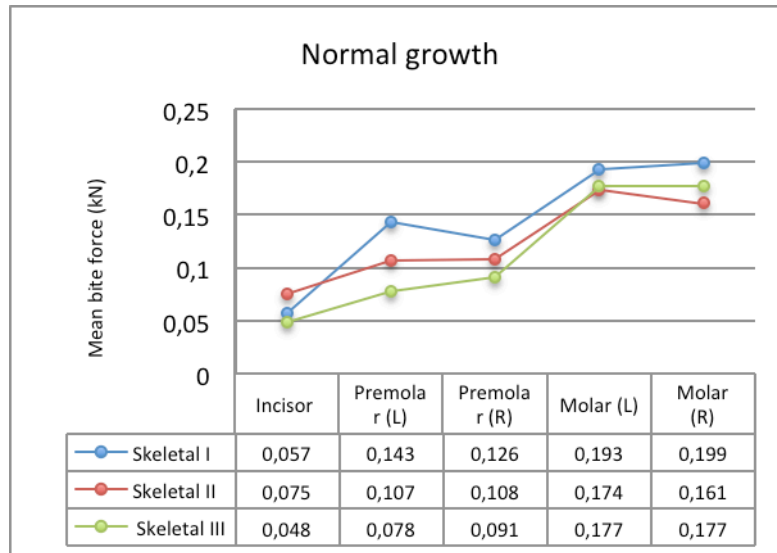


Figure 40: Normal growth comparison

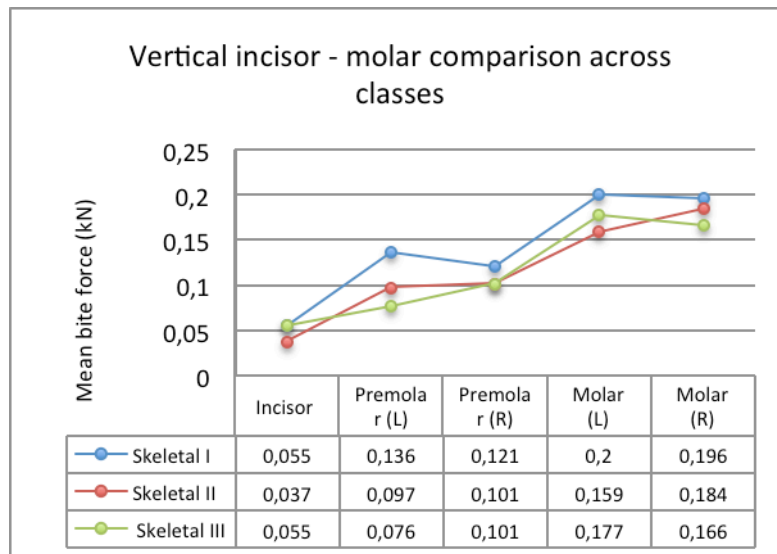


Figure 41: Vertical growth comparison

Skeletal Class I and III subjects display no difference in force amongst the different growth directions; Vertical growth force and Normal growth force run almost parallel over the molars. The greatest difference in molar bite force can be found in Skeletal Class II subjects in the Horizontal growth direction, the increase in bite forces in these subjects

is in agreement with earlier studies. Such studies have already established that the mandibular elevator muscles have an influence on both transverse as well as facial dimensions (Kiliaridis et al., 2003). The point of interest reported by Kiliaridis (2003) was that an increase in load on the jaws and masticatory muscle function resulted in increase of growth at the sutures and an increase in the apposition of bone. Consequentially transverse growth was influenced as a result of the broader bone base of both the maxilla and mandible. This transverse growth results in skeletal changes consequential to the increase in the masticatory muscle function. These changes can also be identified by the anterior mandible and its growth rotation as well as in the coronoid process and the development of the condyle (Kiliaridis et al., 2003). The Skeletal form is maintained post-partum by the continuous remodelling process, which is influenced by the function of the masticatory muscles that act locally on the maxilla and mandible (Kiliaridis et al., 2003). This supports the deduction that form follows function, and the muscles of face particularly the ones responsible for function in the craniofacial skeleton – the muscles of mastication- are influential in the regulation of growth (Kiliaridis et al., 2003).

The data set also provides the opportunity to assess the difference in forces from anterior to posterior, it is apparent that there is an increase of force in most cases from incisor through to molar. A similar study carried out on an indigenous Brazilian population presented analogous results. In the incisive region, the maximum force was found to be of a

smaller magnitude than those measured over the molar region, in both the indigenous and white Brazilian population (Palinkas et al., 2010). Palinkas and co-workers supported these results by prompting the rational thought process centred on molar function versus incisal function. A greater force is required over the molar region, as a result of and increased need of function and close approximation to the masseter muscle. This is the area where the bulk of masticatory activity occurs. It is for this reason that we observe a taper in the mean force from molar, over the premolars to the lowest force found in the anterior segment over the incisors (Palinkas et al., 2010). Proffit and Fields (1983), discovered that the masticatory muscles have a potential influence on the craniofacial growth of man. The terms of this potential influence were centred on a “tension parameter” (Proffit et al., 1983). The tension must be above a certain “threshold overload window” (Proffit et al., 1983).

The changes to components of craniofacial skeleton both in size and shape have been also been studied to reveal that amongst individuals in a population there manifests large varieties in pattern of growth and morphology (Regalo et al., 2008). Through the measurement of the thickness of masseter muscle using ultrasonography Regalo and associates were able to obtain a bilateral mid distance measurement between the zygomatic arch and the gonial angle. This assessment confirmed the hypothesis associating muscle thickness with dimensional changes of the cranio-facial skeleton (Regalo et al., 2008). In another

study by Radsheer and co-workers a relationship or alliance between bite force magnitude and muscle fibre size was identified (Radsheer et al., 1996). These findings uphold those of the additional findings of Ingervall and Thilander (1974) and those of Bolt and Orchardson (1986). These effects can be a literal depiction of “Wollfs’ law”, which has been alluded to. The thickness of the masseter muscle and thus the resulting strength of the muscle weighs in on the width of the dental arch, and should there for be considered in treatment (Satiroglu et al., 2005). This is true for the interaction between an increase in force and growth direction found in this study. Particularly the larger force noted in the Horizontal Class II subject population group.

## Chapter 6: Conclusion

Anthropological studies conducted specifically aimed at analysis of the occlusion and Skeletal structure of primitive humans; reveal a limited presence of malocclusion (Kiliaridis et al., 2003). The increase in incidence of malocclusion correlated with the industrialization of civilization, the progression into modern society. Anthropologists and Orthodontists working together on this subject matter rationalise the manifestation of malocclusion in a more industrialized society as being the consequence of premature and inconsistent exfoliation of deciduous teeth. This untimely occurrence is consequential to the transformation of nutritional regimens between the primitive and industrialised populaces (Kiliaridis et al., 2003). The development and integration of refined sugars into the diet accompanying a modification in the consistency of food are central to this nutritive alteration and consequence. The consistency of nutritional intake is contributory to an alteration in the masticatory muscles functional activity. Consequential hypo-function ensues resultant to the reduction in force requisite (Kiliaridis et al., 2003). The assessment and study of medieval skulls by Kiliaridis (2003) identified a reduction in the Gonial angle. This reduction was found to be concomitant to an increase in the maxillary and mandibular width, contrary to the prevalence of modern day assessment of the long faced modern craniofacial relationships. This Brachifacial façade of medieval craniofacial skeletal structures are

consequential to the superior masticatory muscle activity and force production essential to nutritional consumption relative to that period (Kiliaridis et al., 2003). An increase in malocclusion in modern times is consequential to these factors, thus rendering the contributory role of muscle activity and more importantly force production in the masticatory system a relevant factor in the development of the craniofacial system.

The findings of an increase in force magnitude associated with Horizontal subjects and Skeletal Class II subjects, the incongruences amongst subjects in different growth directions, Skeletal classes, gender groups, age categories within the randomly selected study population group opposes the initial proposed hypotheses. A relationship between facial form and bite force exists. It varies amongst the different Skeletal classes and growth directions. The cause of which can be centred on the function of the masticatory muscles and resultant influence on the growth and development of underlying skeletal structures.

Results of this research project are suggestive to an amendment in the intellectualization of common Skeletal and growth discrepancy identified in the South African scenario. Deliberation on the role and influence of bite force in a particular case during clinical examination and treatment planning is vital. The incorporation of force magnitudes and masticatory muscle activity assessment alluding to the predilection for the development of a Skeletal or growth discrepancy could elicit the need for primary intervention. This primary intervention aimed at modification

of muscle activity may deter the consequential skeletal growth anomalies associated with variances in muscular function.

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## Addenda

### Addendum A

PATIENT / PARTICIPANT'S INFORMATION LEAFLET & INFORMED  
CONSENT FORM FOR CLINICAL TRIAL / NON-INTERVENTION  
STUDY

TITLE OF STUDY:

---

Occlusal bite force measurements in different malocclusions

Dear Mr. / Mrs. .... date ...../...../.....

#### 1) INTRODUCTION

You/your child are invited to volunteer for a research study. This information leaflet is to help you/your child to decide if you would like to participate. Before you/your child agree to take part in this study you should fully understand what is involved. If you/ your child have any questions, which are not fully explained in this leaflet, do not hesitate to ask the investigator Dr. Maryam M. Dawjee. You/your child should not agree to take part unless you are completely happy about all the procedures involved. In the best interests of your health, it is strongly

recommended that you discuss with or inform your personal doctor of your possible participation in this study, wherever possible.

## 2) THE NATURE AND PURPOSE OF THIS STUDY

You/your child are invited to take part in a research study. The aim of this study is to evaluate the bite force in the malocclusion type which you/ your child presents with. By doing so we wish to learn more about occlusal bite forces and the role they play in the different malocclusions.

## 3) EXPLANATION OF PROCEDURES TO BE FOLLOWED

This study involves a comprehensive orthodontic evaluation that is done in the department as part of your regular assessment. I will also be measuring the force of the bite using a meter on which you will be asked to bite, and a reading will be taken.

## 4) RISK AND DISCOMFORT INVOLVED.

There is no risk or discomfort expected during the execution of this study.

## 5) POSSIBLE BENEFITS OF THIS STUDY.

In identifying whether there are differences in the bite forces found in the various malocclusions we can do further research to aid early treatment.

6) I understand that if I do not want to participate in this study, I will still receive standard treatment for my illness.

7) I may at any time withdraw from this study.

8) HAS THE STUDY RECEIVED ETHICAL APPROVAL?

This Protocol was submitted to the Faculty of Health Sciences Research Ethics Committee, University of Pretoria and written approval has been granted by that committee. The study has been structured in accordance with the Declaration of Helsinki (last update: October 2008), which deals with the recommendations guiding doctors in biomedical research involving human/subjects. A copy of the Declaration may be obtained from the investigator should you wish to review it.

**9) INFORMATION** If I have any questions concerning this study, I should contact:

Dr Maryam Mohamed Dawjee

Cell: 0825681657

10) CONFIDENTIALITY

All records obtained whilst in this study will be regarded as confidential. Results will be published or presented in such a fashion that patients remain unidentifiable.

11) CONSENT TO PARTICIPATE IN THIS STUDY.

I have read or had read to me in a language that I understand the above information before signing this consent form. The content and meaning of this information have been explained to me. I have been given opportunity to ask questions and am satisfied that they have been answered satisfactorily.

I understand that if I/my child do not participate it will not alter my/my child's management in any way. I hereby volunteer to take part in this study/ consent to my child participating in the study.

I have received a signed copy of this informed consent agreement.

.....  
Patient / Guardian signature      Date

.....  
Person obtaining informed consent      Date

.....

Witness

Date

PATIENT / PARTICIPANT'S INFORMATION LEAFLET & INFORMED  
ASSENT FOR PATIENTS AGED 7-18.  
FORM FOR CLINICAL TRIAL / NON-INTERVENTION STUDY

TITLE OF STUDY:

---

Occlusal bite force measurements in different malocclusions

Dear Master. / Miss. .... date ...../...../.....

## 1) INTRODUCTION

You child are invited to volunteer for a research study. This information leaflet is to help you child to decide if you would like to participate. Before you agree to take part in this study you should fully understand what is involved. If you child have any questions, which are not fully explained in this leaflet, do not hesitate to ask the investigator Dr. Maryam M. Dawjee. You child should not agree to take part unless you are completely happy about all the procedures involved. In the best interests of your health, it is strongly recommended that you discuss with or inform your personal doctor of your possible participation in this study, wherever possible.

## 2) THE NATURE AND PURPOSE OF THIS STUDY

You child are invited to take part in a research study. The aim of this study is to evaluate the bite force in the malocclusion type, which you have been diagnosed with. By doing so we wish to learn more about occlusal bite forces and the role they play in the different malocclusions.

3) EXPLANATION OF PROCEDURES TO BE FOLLOWED

This study involves a comprehensive orthodontic evaluation that is done in the department as part of your regular assessment. I will also be measuring the force of the bite using a meter on which you will be asked to bite, and a reading will be taken.

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In identifying whether there are differences in the bite forces found in the various malocclusions we can do further research to aid early treatment.

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**11) CONSENT TO PARTICIPATE IN THIS STUDY.**

I have read or had read to me in a language that I understand the above information before signing this assent form. The content and meaning of this information have been explained to me. I have been given

opportunity to ask questions and am satisfied that they have been answered satisfactorily.

I understand that if I do not participate it will not alter my management in any way. I hereby volunteer to take part in this study.

I have received a signed copy of this assent agreement.

.....  
Patient / Guardian signature      Date

.....  
Person obtaining informed consent      Date

.....  
Witness      Date

## Addendum B:

```
-> anova incisor grwth_dir##skel_Class c.age
```

```

                                Number of obs =    360    R-squared    =
0.1246                                Root MSE    =    .03582    Adj R-squared =
0.1021

```

Prob > F	Source	Partial SS	df	MS	F
-----	-----+-----	-----	-----	-----	-----
0.0000	Model	.063904323	9	.00710048	5.53
0.0089	grwth_dir	.012294971	2	.006147485	4.79
0.0232	skel_Class	.009760745	2	.004880372	3.80
0.0022	grwth_dir#skel_Class	.021832218	4	.005458055	4.25
0.0000	age	.02331131	1	.02331131	18.17
	Residual	.449065905	350	.001283045	
-----	-----+-----	-----	-----	-----	-----
	Total	.512970228	359	.001428886	

```
-> adjust age, by( grwth_dir skel_Class) format(%9.3f)
```

```

-----
-----
Dependent variable: incisor    Command: anova
Covariate set to mean: age = 15.633333
-----
-----

```

grwth_dir	skel_Class		
	Class I	Class II	Class III
Horizontal	0.053	0.069	0.049
Normal	0.057	0.073	0.046
Vertical	0.055	0.036	0.045

```
Key: Linear Prediction
```

```
-> anova prem_1 grwth_dir##skel_Class c.age
```

```

                                Number of obs =    360    R-squared    =
0.1434
                                Root MSE     =  .077322    Adj R-squared =
0.1214

```

Prob > F	Source	Partial SS	df	MS	F
0.0000	Model	.350360153	9	.038928906	6.51
0.3773	grwth_dir	.011686344	2	.005843172	0.98
0.0000	skel_Class	.128314129	2	.064157064	10.73
0.0056	grwth_dir#skel_Class	.089017217	4	.022254304	3.72
0.0000	age	.134342913	1	.134342913	22.47
	Residual	2.09255959	350	.005978742	
	Total	2.44291974	359	.00680479	

```
-> adjust age, by( grwth_dir skel_Class) format(%9.3f)
```

```

-----
Dependent variable: prem_1    Command: anova
Covariate set to mean: age = 15.633333
-----

```

grwth_dir	skel_Class		
	Class I	Class II	Class III
Horizontal	0.083	0.119	0.076
Normal	0.142	0.103	0.073
Vertical	0.137	0.093	0.077

```
Key: Linear Prediction
```

-> anova mol\_1 grwth\_dir##skel\_Class c.age

Number of obs = 360 R-squared =  
 0.0301  
 Root MSE = .099727 Adj R-squared =  
 0.0051

Prob > F	Source	Partial SS	df	MS	F
	Model	.107935622	9	.011992847	1.21
0.2901					
	grwth_dir	.008386651	2	.004193326	0.42
0.6563					
	skel_Class	.035631612	2	.017815806	1.79
0.1683					
	grwth_dir#skel_Class	.032655614	4	.008163904	0.82
0.5125					
	age	.044108197	1	.044108197	4.43
0.0359					
	Residual	3.48092747	350	.009945507	
	Total	3.5888631	359	.009996833	

-> adjust age, by( grwth\_dir skel\_Class) format(%9.3f)

Dependent variable: mol\_1 Command: anova  
 Covariate set to mean: age = 15.633333

grwth_dir	skel_Class		
	Class I	Class II	Class III
Horizontal	0.197	0.201	0.171
Normal	0.193	0.172	0.175
Vertical	0.200	0.157	0.178

Key: Linear Prediction



```
-> anova mol_r grwth_dir##skel_Class c.age
```

```

                                Number of obs =    360    R-squared    =
0.0657
                                Root MSE     =   .10625    Adj R-squared =
0.0416

```

Prob > F	Source	Partial SS	df	MS	F
0.0043	Model	.277697554	9	.030855284	2.73
0.3025	grwth_dir	.027086903	2	.013543452	1.20
0.0991	skel_Class	.05254643	2	.026273215	2.33
0.0106	grwth_dir#skel_Class	.150676285	4	.037669071	3.34
0.0069	age	.083233123	1	.083233123	7.37
	Residual	3.95115534	350	.011289015	
	Total	4.2288529	359	.011779535	

```
-> adjust age, by( grwth_dir skel_Class) format(%9.3f)
```

```

-----
Dependent variable: mol_r    Command: anova
Covariate set to mean: age = 15.633333
-----

```

grwth_dir	skel_Class		
	Class I	Class II	Class III
Horizontal	0.180	0.247	0.164
Normal	0.198	0.157	0.173
Vertical	0.196	0.182	0.167

```
Key: Linear Prediction
```

```
. for var incisor - mol_r: table race gender, c(N X mean X sd X)
format(%9.3f) row col
```

```
-> table race gender, c(N incisor mean incisor sd incisor)
format(%9.3f) row col
```

race	gender		Total
	Male	Female	
African	89	96	185
	0.049	0.055	0.052
	0.019	0.044	0.035
White	40	50	90
	0.068	0.051	0.058
	0.065	0.028	0.048
Indian	35	30	65
	0.042	0.051	0.046
	0.025	0.027	0.026
Coloured	16	4	20
	0.084	0.037	0.075
	0.033	0.003	0.035
Total	180	180	360
	0.055	0.053	0.054
	0.038	0.037	0.038

```
-> table race gender, c(N prem_1 mean prem_1 sd prem_1) format(%9.3f)
row col
```

race	gender		Total
	Male	Female	
African	89	96	185
	0.095	0.107	0.101
	0.047	0.075	0.063
White	40	50	90
	0.128	0.078	0.100
	0.157	0.036	0.110
Indian	35	30	65
	0.089	0.090	0.089
	0.109	0.048	0.086
Coloured	16	4	20
	0.146	0.053	0.127
	0.088	0.004	0.087
Total	180	180	360
	0.106	0.095	0.100
	0.098	0.062	0.082

```
-> table race gender, c(N mol_l mean mol_l sd mol_l) format(%9.3f) row
col
```

race	gender		Total
	Male	Female	
African	89	96	185
	0.167	0.182	0.175
	0.082	0.092	0.088
White	40	50	90
	0.245	0.169	0.203
	0.178	0.071	0.134
Indian	35	30	65
	0.161	0.162	0.161
	0.068	0.059	0.064
Coloured	16	4	20
	0.254	0.155	0.234
	0.095	0.069	0.098
Total	180	180	360
	0.191	0.174	0.183
	0.115	0.081	0.100

```
-> table race gender, c(N prem_r mean prem_r sd prem_r) format(%9.3f)
row col
```

race	gender		Total
	Male	Female	
African	89	96	185
	0.108	0.120	0.114
	0.067	0.078	0.073
White	40	50	90
	0.120	0.094	0.105
	0.088	0.040	0.067
Indian	35	30	65
	0.106	0.095	0.101
	0.104	0.043	0.081
Coloured	16	4	20
	0.148	0.048	0.128
	0.093	0.029	0.093
Total	180	180	360
	0.114	0.107	0.110
	0.083	0.065	0.074

```
-> table race gender, c(N mol_r mean mol_r sd mol_r) format(%9.3f) row
col
```

race	gender		Total
	Male	Female	
African	89	96	185
	0.180	0.190	0.185
	0.096	0.107	0.102
White	40	50	90
	0.223	0.173	0.195
	0.145	0.059	0.108
Indian	35	30	65
	0.140	0.152	0.146
	0.115	0.072	0.097
Coloured	16	4	20
	0.286	0.165	0.261
	0.154	0.131	0.155
Total	180	180	360
	0.191	0.179	0.185

```
. for var incisor - mol_r: anova X grwth_dir##skel_Class c.age \
test, test(gr_hor) mtest \ test, test(gr_norm) mtest \ test,
test(gr_vert) mtest \ test, test(cl_I) mtest \ test, test(cl_II) mtest
\ test, test(cl_III) mtest
```

```
-> anova incisor grwth_dir##skel_Class c.age
```

```
Number of obs = 360 R-squared =
0.1246
Root MSE = .03582 Adj R-squared =
0.1021
```

Prob > F	Source	Partial SS	df	MS	F
0.0000	Model	.063904323	9	.00710048	5.53
0.0089	grwth_dir	.012294971	2	.006147485	4.79
0.0232	skel_Class	.009760745	2	.004880372	3.80
0.0022	grwth_dir#skel_Class	.021832218	4	.005458055	4.25
0.0000	age	.02331131	1	.02331131	18.17
	Residual	.449065905	350	.001283045	
	Total	.512970228	359	.001428886	

```
-> test, test(gr_hor) mtest

( 1) 0b.skel_Class - 1.skel_Class + 0b.grwth_dir#0b.skel_Class -
0b.grwth_dir#1o.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 0b.grwth_dir#0b.skel_Class -
0b.grwth_dir#2o.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 0b.grwth_dir#1o.skel_Class -
0b.grwth_dir#2o.skel_Class = 0
      Constraint 2 dropped
```

	F(df, 350)	df	p
(1)	3.88	1	0.0498 #
(2)	0.34	1	0.5575 #
(3)	6.34	1	0.0122 #
all	3.48	2	0.0319

# unadjusted p-values

```
-> test, test(gr_norm) mtest

( 1) 0b.skel_Class - 1.skel_Class + 1o.grwth_dir#0b.skel_Class -
1.grwth_dir#1.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 1o.grwth_dir#0b.skel_Class -
1.grwth_dir#2.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 1.grwth_dir#1.skel_Class -
1.grwth_dir#2.skel_Class = 0
      Constraint 3 dropped
```

	F(df, 350)	df	p
(1)	4.03	1	0.0454 #
(2)	1.79	1	0.1816 #
(3)	11.37	1	0.0008 #
all	5.76	2	0.0035

# unadjusted p-values

```
-> test, test(gr_vert) mtest

( 1) 0b.skel_Class - 1.skel_Class + 2o.grwth_dir#0b.skel_Class -
2.grwth_dir#1.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 2o.grwth_dir#0b.skel_Class -
2.grwth_dir#2.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 2.grwth_dir#1.skel_Class -
2.grwth_dir#2.skel_Class = 0
      Constraint 2 dropped
```

	F(df, 350)	df	p
(1)	6.09	1	0.0140 #
(2)	1.55	1	0.2133 #
(3)	1.50	1	0.2220 #
all	3.05	2	0.0487

# unadjusted p-values

```
-> test, test(cl_I) mtest

( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#0b.skel_Class -
1o.grwth_dir#0b.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#0b.skel_Class -
2o.grwth_dir#0b.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1o.grwth_dir#0b.skel_Class -
2o.grwth_dir#0b.skel_Class = 0
      Constraint 2 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1) |           0.20         1      0.6531 #
(2) |           0.07         1      0.7864 #
(3) |           0.03         1      0.8584 #
-----+-----
all |           0.10         2      0.9021
-----+-----
# unadjusted p-values
```

```
-> test, test(cl_II) mtest

( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#1o.skel_Class -
1.grwth_dir#1.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#1o.skel_Class -
2.grwth_dir#1.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1.grwth_dir#1.skel_Class -
2.grwth_dir#1.skel_Class = 0
      Constraint 3 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1) |           0.25         1      0.6176 #
(2) |          16.84         1      0.0001 #
(3) |          21.84         1      0.0000 #
-----+-----
all |          13.07         2      0.0000
-----+-----
# unadjusted p-values
```

```
-> test, test(cl_III) mtest

( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#2o.skel_Class -
1.grwth_dir#2.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#2o.skel_Class +
2.grwth_dir#2.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1.grwth_dir#2.skel_Class -
2.grwth_dir#2.skel_Class = 0
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1) |           0.10         1      0.7491 #
(2) |           0.18         1      0.6734 #
(3) |           0.01         1      0.9383 #
-----+-----
all |           0.08         3      0.9687
-----+-----
# unadjusted p-values
```

```
-> anova prem_l grwth_dir##skel_Class c.age
```

```

                                Number of obs =    360    R-squared    =
0.1434
                                Root MSE     =  .077322    Adj R-squared =
0.1214

```

Prob > F	Source	Partial SS	df	MS	F
0.0000	Model	.350360153	9	.038928906	6.51
0.3773	grwth_dir	.011686344	2	.005843172	0.98
0.0000	skel_Class	.128314129	2	.064157064	10.73
0.0056	grwth_dir#skel_Class	.089017217	4	.022254304	3.72
0.0000	age	.134342913	1	.134342913	22.47
	Residual	2.09255959	350	.005978742	
	Total	2.44291974	359	.00680479	

```
-> test, test(gr_hor) mtest
```

```

( 1) 0b.skel_Class - 1.skel_Class + 0b.grwth_dir#0b.skel_Class -
0b.grwth_dir#1o.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 0b.grwth_dir#0b.skel_Class -
0b.grwth_dir#2o.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 0b.grwth_dir#1o.skel_Class -
0b.grwth_dir#2o.skel_Class = 0
      Constraint 2 dropped

```

	F(df, 350)	df	p
(1)	4.50	1	0.0345 #
(2)	0.17	1	0.6778 #
(3)	6.26	1	0.0128 #
all	3.60	2	0.0283

```
# unadjusted p-values
```

```
-> test, test(gr_norm) mtest

( 1) 0b.skel_Class - 1.skel_Class + 1o.grwth_dir#0b.skel_Class -
1.grwth_dir#1.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 1o.grwth_dir#0b.skel_Class -
1.grwth_dir#2.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 1.grwth_dir#1.skel_Class -
1.grwth_dir#2.skel_Class = 0
      Constraint 3 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1) |           4.97         1      0.0264 #
(2) |          15.66         1      0.0001 #
(3) |           3.03         1      0.0825 #
-----+-----
all  |           7.86         2      0.0005
-----+-----
      # unadjusted p-values
```

```
-> test, test(gr_vert) mtest

( 1) 0b.skel_Class - 1.skel_Class + 2o.grwth_dir#0b.skel_Class -
2.grwth_dir#1.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 2o.grwth_dir#0b.skel_Class -
2.grwth_dir#2.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 2.grwth_dir#1.skel_Class -
2.grwth_dir#2.skel_Class = 0
      Constraint 2 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1) |           6.32         1      0.0124 #
(2) |          12.12         1      0.0006 #
(3) |           0.93         1      0.3364 #
-----+-----
all  |           6.46         2      0.0018
-----+-----
      # unadjusted p-values
```

```
-> test, test(cl_I) mtest

( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#0b.skel_Class -
1o.grwth_dir#0b.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#0b.skel_Class -
2o.grwth_dir#0b.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1o.grwth_dir#0b.skel_Class -
2o.grwth_dir#0b.skel_Class = 0
      Constraint 2 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1) |          11.92         1      0.0006 #
(2) |          10.07         1      0.0016 #
(3) |           0.09         1      0.7662 #
-----+-----
all  |           7.43         2      0.0007
-----+-----
      # unadjusted p-values
```

```
-> test, test(cl_II) mtest
```

```
( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#1o.skel_Class -
1.grwth_dir#1.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#1o.skel_Class -
2.grwth_dir#1.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1.grwth_dir#1.skel_Class -
2.grwth_dir#1.skel_Class = 0
      Constraint 3 dropped
```

	F(df, 350)	df	p
(1)	0.83	1	0.3632 #
(2)	2.17	1	0.1413 #
(3)	0.33	1	0.5680 #
all	1.10	2	0.3332

# unadjusted p-values

```
-> test, test(cl_III) mtest
```

```
( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#2o.skel_Class -
1.grwth_dir#2.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#2o.skel_Class +
2.grwth_dir#2.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1.grwth_dir#2.skel_Class -
2.grwth_dir#2.skel_Class = 0
```

	F(df, 350)	df	p
(1)	0.02	1	0.8884 #
(2)	7.85	1	0.0054 #
(3)	0.04	1	0.8373 #
all	3.37	3	0.0187

# unadjusted p-values

```
-> anova mol_l grwth_dir##skel_Class c.age
```

```

                                Number of obs =    360    R-squared    =
0.0301
                                Root MSE     = .099727    Adj R-squared =
0.0051

```

Prob > F	Source	Partial SS	df	MS	F
	Model	.107935622	9	.011992847	1.21
0.2901	grwth_dir	.008386651	2	.004193326	0.42
0.6563	skel_Class	.035631612	2	.017815806	1.79
0.1683	grwth_dir#skel_Class	.032655614	4	.008163904	0.82
0.5125	age	.044108197	1	.044108197	4.43
0.0359	Residual	3.48092747	350	.009945507	
	Total	3.5888631	359	.009996833	

```
-> test, test(gr_hor) mtest
```

```

( 1) 0b.skel_Class - 1.skel_Class + 0b.grwth_dir#0b.skel_Class -
0b.grwth_dir#1o.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 0b.grwth_dir#0b.skel_Class -
0b.grwth_dir#2o.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 0b.grwth_dir#1o.skel_Class -
0b.grwth_dir#2o.skel_Class = 0
      Constraint 2 dropped

```

	F(df, 350)	df	p
(1)	0.04	1	0.8509 #
(2)	1.41	1	0.2366 #
(3)	1.81	1	0.1792 #
all	1.08	2	0.3410

```
# unadjusted p-values
```

```
-> test, test(gr_norm) mtest

( 1) 0b.skel_Class - 1.skel_Class + 1o.grwth_dir#0b.skel_Class -
1.grwth_dir#1.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 1o.grwth_dir#0b.skel_Class -
1.grwth_dir#2.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 1.grwth_dir#1.skel_Class -
1.grwth_dir#2.skel_Class = 0
      Constraint 3 dropped
```

	F(df, 350)	df	p
(1)	0.83	1	0.3633 #
(2)	0.65	1	0.4211 #
(3)	0.01	1	0.9162 #
all	0.49	2	0.6107

# unadjusted p-values

```
-> test, test(gr_vert) mtest

( 1) 0b.skel_Class - 1.skel_Class + 2o.grwth_dir#0b.skel_Class -
2.grwth_dir#1.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 2o.grwth_dir#0b.skel_Class -
2.grwth_dir#2.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 2.grwth_dir#1.skel_Class -
2.grwth_dir#2.skel_Class = 0
      Constraint 2 dropped
```

	F(df, 350)	df	p
(1)	3.72	1	0.0545 #
(2)	1.00	1	0.3186 #
(3)	0.87	1	0.3522 #
all	1.86	2	0.1569

# unadjusted p-values

```
-> test, test(cl_I) mtest

( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#0b.skel_Class -
1o.grwth_dir#0b.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#0b.skel_Class -
2o.grwth_dir#0b.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1o.grwth_dir#0b.skel_Class -
2o.grwth_dir#0b.skel_Class = 0
      Constraint 2 dropped
```

	F(df, 350)	df	p
(1)	0.04	1	0.8517 #
(2)	0.03	1	0.8687 #
(3)	0.12	1	0.7285 #
all	0.06	2	0.9414

# unadjusted p-values

```
-> test, test(cl_II) mtest
```

```
( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#1o.skel_Class -
1.grwth_dir#1.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#1o.skel_Class -
2.grwth_dir#1.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1.grwth_dir#1.skel_Class -
2.grwth_dir#1.skel_Class = 0
      Constraint 3 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1) |           1.61         1      0.2050 #
(2) |           3.71         1      0.0549 #
(3) |           0.44         1      0.5065 #
-----+-----
all |           1.91         2      0.1500
-----+-----
# unadjusted p-values
```

```
-> test, test(cl_III) mtest
```

```
( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#2o.skel_Class -
1.grwth_dir#2.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#2o.skel_Class +
2.grwth_dir#2.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1.grwth_dir#2.skel_Class -
2.grwth_dir#2.skel_Class = 0
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1) |           0.03         1      0.8626 #
(2) |           0.00         1      0.9967 #
(3) |           0.03         1      0.8713 #
-----+-----
all |           0.05         3      0.9866
-----+-----
# unadjusted p-values
```

```
-> anova prem_r grwth_dir##skel_Class c.age
```

```

                                Number of obs =    360    R-squared    =
0.1183
                                Root MSE     =  .070654    Adj R-squared =
0.0956

```

Prob > F	Source	Partial SS	df	MS	F
0.0000	Model	.234354751	9	.026039417	5.22
0.2605	grwth_dir	.013482215	2	.006741107	1.35
0.0027	skel_Class	.059887788	2	.029943894	6.00
0.0274	grwth_dir#skel_Class	.055263401	4	.01381585	2.77
0.0000	age	.138816616	1	.138816616	27.81
	Residual	1.7471898	350	.004991971	
	Total	1.98154455	359	.005519623	

```
-> test, test(gr_hor) mtest
```

```

( 1) 0b.skel_Class - 1.skel_Class + 0b.grwth_dir#0b.skel_Class -
0b.grwth_dir#1o.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 0b.grwth_dir#0b.skel_Class -
0b.grwth_dir#2o.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 0b.grwth_dir#1o.skel_Class -
0b.grwth_dir#2o.skel_Class = 0
      Constraint 2 dropped

```

	F(df, 350)	df	p
(1)	3.68	1	0.0558 #
(2)	3.60	1	0.0587 #
(3)	14.06	1	0.0002 #
all	7.03	2	0.0010

```
# unadjusted p-values
```

```
-> test, test(gr_norm) mtest

( 1) 0b.skel_Class - 1.skel_Class + 1o.grwth_dir#0b.skel_Class -
1.grwth_dir#1.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 1o.grwth_dir#0b.skel_Class -
1.grwth_dir#2.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 1.grwth_dir#1.skel_Class -
1.grwth_dir#2.skel_Class = 0
      Constraint 3 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1) |           1.91         1      0.1683 #
(2) |           6.17         1      0.0134 #
(3) |           1.24         1      0.2667 #
-----+-----
all  |           3.10         2      0.0465
-----+-----
      # unadjusted p-values
```

```
-> test, test(gr_vert) mtest

( 1) 0b.skel_Class - 1.skel_Class + 2o.grwth_dir#0b.skel_Class -
2.grwth_dir#1.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 2o.grwth_dir#0b.skel_Class -
2.grwth_dir#2.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 2.grwth_dir#1.skel_Class -
2.grwth_dir#2.skel_Class = 0
      Constraint 2 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1) |           2.33         1      0.1281 #
(2) |           1.56         1      0.2131 #
(3) |           0.08         1      0.7802 #
-----+-----
all  |           1.32         2      0.2684
-----+-----
      # unadjusted p-values
```

```
-> test, test(cl_I) mtest

( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#0b.skel_Class -
1o.grwth_dir#0b.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#0b.skel_Class -
2o.grwth_dir#0b.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1o.grwth_dir#0b.skel_Class -
2o.grwth_dir#0b.skel_Class = 0
      Constraint 2 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1) |           0.20         1      0.6569 #
(2) |           0.03         1      0.8739 #
(3) |           0.08         1      0.7769 #
-----+-----
all  |           0.10         2      0.9040
-----+-----
      # unadjusted p-values
```

```
-> test, test(cl_II) mtest
```

```
( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#1o.skel_Class -
1.grwth_dir#1.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#1o.skel_Class -
2.grwth_dir#1.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1.grwth_dir#1.skel_Class -
2.grwth_dir#1.skel_Class = 0
      Constraint 3 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1)  |           7.92         1      0.0052 #
(2)  |          10.46         1      0.0013 #
(3)  |           0.18         1      0.6735 #
-----+-----
all  |           6.13         2      0.0024
-----+-----
# unadjusted p-values
```

```
-> test, test(cl_III) mtest
```

```
( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#2o.skel_Class -
1.grwth_dir#2.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#2o.skel_Class +
2.grwth_dir#2.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1.grwth_dir#2.skel_Class -
2.grwth_dir#2.skel_Class = 0
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1)  |           0.03         1      0.8521 #
(2)  |           0.04         1      0.8323 #
(3)  |           0.94         1      0.3334 #
-----+-----
all  |           0.36         3      0.7810
-----+-----
# unadjusted p-values
```

```
-> anova mol_r grwth_dir##skel_Class c.age
```

```

                                Number of obs =    360    R-squared    =
0.0657
                                Root MSE     =    .10625   Adj R-squared =
0.0416

```

Prob > F	Source	Partial SS	df	MS	F
0.0043	Model	.277697554	9	.030855284	2.73
0.3025	grwth_dir	.027086903	2	.013543452	1.20
0.0991	skel_Class	.05254643	2	.026273215	2.33
0.0106	grwth_dir#skel_Class	.150676285	4	.037669071	3.34
0.0069	age	.083233123	1	.083233123	7.37
	Residual	3.95115534	350	.011289015	
	Total	4.2288529	359	.011779535	

```
-> test, test(gr_hor) mtest
```

```

( 1) 0b.skel_Class - 1.skel_Class + 0b.grwth_dir#0b.skel_Class -
0b.grwth_dir#1o.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 0b.grwth_dir#0b.skel_Class -
0b.grwth_dir#2o.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 0b.grwth_dir#1o.skel_Class -
0b.grwth_dir#2o.skel_Class = 0
      Constraint 2 dropped

```

	F(df, 350)	df	p
(1)	7.84	1	0.0054 #
(2)	0.50	1	0.4781 #
(3)	11.96	1	0.0006 #
all	6.69	2	0.0014

```
# unadjusted p-values
```

```
-> test, test(gr_norm) mtest

( 1) 0b.skel_Class - 1.skel_Class + 1o.grwth_dir#0b.skel_Class -
1.grwth_dir#1.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 1o.grwth_dir#0b.skel_Class -
1.grwth_dir#2.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 1.grwth_dir#1.skel_Class -
1.grwth_dir#2.skel_Class = 0
      Constraint 3 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1)  |          2.93         1      0.0876 #
(2)  |          1.10         1      0.2949 #
(3)  |          0.45         1      0.5042 #
-----+-----
all  |          1.49         2      0.2270
-----+-----
      # unadjusted p-values
```

```
-> test, test(gr_vert) mtest

( 1) 0b.skel_Class - 1.skel_Class + 2o.grwth_dir#0b.skel_Class -
2.grwth_dir#1.skel_Class = 0
( 2) 0b.skel_Class - 2.skel_Class + 2o.grwth_dir#0b.skel_Class -
2.grwth_dir#2.skel_Class = 0
( 3) 1.skel_Class - 2.skel_Class + 2.grwth_dir#1.skel_Class -
2.grwth_dir#2.skel_Class = 0
      Constraint 2 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1)  |          0.37         1      0.5429 #
(2)  |          1.51         1      0.2196 #
(3)  |          0.38         1      0.5362 #
-----+-----
all  |          0.76         2      0.4703
-----+-----
      # unadjusted p-values
```

```
-> test, test(cl_I) mtest

( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#0b.skel_Class -
1o.grwth_dir#0b.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#0b.skel_Class -
2o.grwth_dir#0b.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1o.grwth_dir#0b.skel_Class -
2o.grwth_dir#0b.skel_Class = 0
      Constraint 2 dropped
```

```
-----+-----
      |      F(df,350)      df      p
-----+-----
(1)  |          0.58         1      0.4475 #
(2)  |          0.45         1      0.5051 #
(3)  |          0.01         1      0.9230 #
-----+-----
all  |          0.35         2      0.7068
-----+-----
      # unadjusted p-values
```

```
-> test, test(cl_II) mtest
```

```
( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#1o.skel_Class -
1.grwth_dir#1.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#1o.skel_Class -
2.grwth_dir#1.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1.grwth_dir#1.skel_Class -
2.grwth_dir#1.skel_Class = 0
      Constraint 3 dropped
```

	F(df, 350)	df	p
(1)	13.68	1	0.0003 #
(2)	7.29	1	0.0073 #
(3)	1.04	1	0.3091 #
all	7.27	2	0.0008

# unadjusted p-values

```
-> test, test(cl_III) mtest
```

```
( 1) 0b.grwth_dir - 1.grwth_dir + 0b.grwth_dir#2o.skel_Class -
1.grwth_dir#2.skel_Class = 0
( 2) 0b.grwth_dir - 2.grwth_dir + 0b.grwth_dir#2o.skel_Class +
2.grwth_dir#2.skel_Class = 0
( 3) 1.grwth_dir - 2.grwth_dir + 1.grwth_dir#2.skel_Class -
2.grwth_dir#2.skel_Class = 0
```

	F(df, 350)	df	p
(1)	0.16	1	0.6890 #
(2)	0.29	1	0.5923 #
(3)	0.07	1	0.7877 #
all	0.20	3	0.8938

# unadjusted p-values

## Addendum C

Additional documentation as required for submission.