Temporomandibular Joint Outcomes Following Orthognathic Surgery

by

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ABSTRACT

Problem: Orthognathic surgery is the definitive treatment for the correction of dentofacial deformity. The effect of orthognathic surgery on temporomandibular joint pain and dysfunction is a controversial topic with inconclusive evidence despite numerous studies on the topic. The ability to identify risk factors for poor TMJ outcome pre-operatively would be beneficial. This could improve pre-operative information for our patients, and set realistic expectations regarding TMJ function following surgery. It may also have an impact on surgical treatment planning.

Purpose: To determine the effect orthognathic surgery has on TMJ-related pain and function, and identify pre-operative patient risk-factors to predict TMJ outcome.

Methods: Prospective data collection of demographic, surgical and outcome variables was collected pre-operatively and 6 months post-operatively on our patient population. Comparisons were made between pre and post-operative data to find correlations or associations between patient and surgical variables, and TMJ outcomes, with a focus on mandibular range of motion and TMJ related pain.

Results: Of 152 patients that enrolled in the study 56 completed the 6 month post-operative visit. MIO, right and left lateral excursion and pain VAS did not show statistically significant differences pre vs. post operatively. Pre-operative overjet ≥6mm ($p<0.01$), Class II malocclusion ($p<0.01$), and completion of FG ($p<0.01$) were associated with a decreased MIO. There was a correlation with increasing magnitude of mandibular movement and decreasing MIO. Protrusion decreased a mean of 2.5mm ($p<0.01$). Self reported frequency of joint pain ($p=0.034$), clenching ($p<0.01$), and objective clicking ($p=0.034$) decreased. Clenching ($p=0.021$) and objective clicking (<0.01) decreased more in those undergoing double vs. single jaw surgery. Self-reported joint noises were significantly decreased ($p=0.01$), but Class II patients were as likely to see the disappearance of joint noise as they were to develop new joint noises ($p=0.026$).

Conclusion: TMJ related pain is likely to remain unchanged following surgery, however patients may notice a decrease in their pain frequency. Some patients experience a small decrease in mandibular mobility, but this reduction is usually not reported by the patient, and is likely inconsequential functionally. A decrease in opening requiring further intervention can occur, but is rare. TMJ clicking and parafunctional clenching may decrease following surgery, especially when performing two-jaw surgery.

LIST OF ABBREVIATIONS USED

ANOVA	Analysis of Variance
BSSO	Bilateral Sagittal Split Osteotomy
CROS	Condylar Resorption following Orthognathic Surgery
CI	Confidence Interval
CT	Computed Tomography
CBCT	Cone Beam Computed Tomography
FG	Functional Genioplasty
IAN	Inferior Alveolar Nerve
i.e.	Id est
IVRO	Intraoral Vertical Ramus Osteotomy
max	Maximum
min	Minimum
mm	Millimeters
MIO	Maximum Incisal Opening
MMF	Maxillomandibular Fixation
MRI	Magnetic Resonance Imaging
PPV	Positive Predictive Value
RCT	Randomized Controlled Trial
Sig.	Significance
SD	Standard Deviation
SSO	Sagittal Split Osteotomy
TMD	Temporomandibular Joint Disorder
TMJ	Temporomandibular Joint
VAS	Visual Analog Scale
OGS	Orthognathic Surgery
OQOL	Orthognathic Quality of Life
QOL	Quality of Life

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CHAPTER 1 – INTRODUCTION

1.1 PREAMBLE

Orthognathic surgery (OGS) is the definitive treatment for dentofacial deformities. OGS changes the position of the maxillomandibular complex and thus has the potential to affect the temporomandibular joint (TMJ). These changes to the TMJ have the potential to either improve or negatively affect the function and subjective pain associated with the TMJ. At our institution approximately 350 of cases of orthognathic surgery are performed annually. Patients presenting for orthognathic surgery often enquire about the expected outcome of pre-existing TMJ pain following surgery. Therefore, realizing the effects of OGS on TMJ pain and dysfunction can have a large impact on our patient population. Secondarily, comparing our institutional surgical outcomes with that in the published literature may allow us to identify strengths or weaknesses in our surgical techniques that could be modified.

1.2 DENTOFACIAL DEFORMITY

Dentofacial deformities are defined by Proffit et al. as "facial and dental disproportions great enough to significantly affect the individual's quality of life"[1]. These jaw deformities inevitably affect the soft-tissue profile of the individual, causing a relative imbalance of the nose, lips and chin. Dentofacial deformities can be identified on the basis of its three main components: vertical excess or deficiency, sagittal excess or deficiency, and transverse discrepancies and asymmetry. Description of the dental occlusion is a common reference for defining these distortions[1]. As first described by Angle in 1899, a Class I occlusion is defined as the mesiobuccal cusp of the maxillary first molar occluding with the buccal groove of the mandibular first molar.[2] Class II malocclusion is defined as the mesiobuccal cusp of the maxillary first molar occluding mesial to the buccal groove of the mandibular first molar. Class III malocclusion is defined as a maxillary mesiobuccal cusp that occludes distal to the buccal groove of the mandibular first molar[2].

If the dental or facial components of this deformity are far enough outside the range of normal the population may be considered socially or functional handicapped[1]. Patients with a dentofacial deformity have been found to be at a disadvantage in society due to low self-esteem and decreased levels of self confidence[3]. Daily effects of their deformity may include thoughts of embarrassment when eating in public or being perceived as stupid, mean, or angry because of their facial appearance. Surveys of patients with a dentofacial deformity demonstrate self-categorization of functional impairment in 50.4%, and esthetic impairment in 43%, and overall lower quality of life (QOL)[4]. Severe dentofacial deformity cases (those requiring orthognathic surgery in addition to orthodontic treatment for correction) also demonstrate masticatory deficiency[5]. Dentofacial deformities have a statistically significantly affect on general oral health related QOL[6]. This includes domains of functional limitation, psychological discomfort, and psychological disability. Not surprisingly, there is also a statistically significant difference in condition specific QOL when patients with dentofacial deformity are assessed with an Orthognathic QOL questionnaire[6].

1.3 ORTHOGNATHIC SURGERY

1.3.1 IMPROVEMENT IN QUALITY OF LIFE

Quality of life surveys are a measure used to quantify conditions that are not necessarily fatal but can cause considerable physical, social and psychological dysfunction[7]. With finite resources available to deliver healthcare, measures of impact and success of interventions must be undertook to ensure efficient use of health care funds. Clinician based evaluation of objective outcomes may overlook the patient's own perception of changes in QOL and therefore QOL-type questionnaires are useful for evaluating overall treatment impact on the patient[8]. With a shift to patient centered care, QOL questioning is becoming increasingly important. Subsequently, orthognathic surgery has been shown to increase patient quality of life[9]. This was demonstrated by Silva et. al in their prospective quality of life survey of 50 patients undergoing orthognathic surgery[10]. Patients were asked to complete a 'Short Form Oral Health Impact Profile (OHIP-14)' (Appendix A), an 'Orthognathic Quality of Life Questionnaire[11] (OQLQ)' (Appendix B), and a social-demographic questionnaire at 3 time points (pre-surgical, 6 weeks post operative, and 6 months post-operatively). The results showed that there was a statistically significant improvement in oral health impact profile at 6 months post-operative compared with pre-operative status. Also, improvement in OQOL was shown to be statistically significant at both 6 weeks and 6 months post-surgery[10]. Lee et. al in 2008 also applied a 36-item Short Form Health Survey (SF-36) to 36 patients and found that although at 6 weeks post-surgery there was a significant reduction in SF-36 physical and mental scores, at 6 months post surgery SF-36 scores had returned to baseline[12]. Lee et. al's results of OHIP-14 and OQLQ also showed statistically significant reductions (interpreted as improvements) in scores at 6 months post-operatively[12]. Soh et. al completed the only comprehensive literature review of the topic in 2013. They looked at 21 articles and concluded that "orthognathic surgery patients experience an improvement in quality of life after surgery"[3].

One may ask why TMJ outcomes cannot be simply extracted from the data collected on these questionnaires, but a review of questioning reveals the answer. Looking at the OHIP-14 questionnaire, 3 of the 14 questions could possibly be attributed to the TMD-type symptoms (Appendix A: b, c, d), and similarly, the OLOQ questionnaire has 4 of 23 questions that could have TMD association (Appendix B: 3,4,5,7). Unfortunately, the questions are closed-ended, not allowing specific explanation for the improvement in symptoms, and therefore it is impossible to associate any improvement in TMD-type symptomatology with the general improvement in oral health impact observed. Therefore, the overall improvements in QOL seen may include improvements in TMD symptomology, although impossible to confirm from these questionnaires alone due to the non-specific line of questioning.

Subjective improvement in oral function (ie. masticatory efficiency) has been shown to be present in the majority of cases (87.8%)[13]. Also, subjective positive esthetic changes are also observed in the majority (92.7%) of patients having undergone surgical correction of their dentofacial deformity[13].

Orthognathic surgery is performed in combination with orthodontic treatment for best results. The orthognathic surgeon has the ability to reshape the facial profile by aligning the underlying skeletal framework of the maxilla and mandible through well-described osteotomies, as explained in the next section.

1.3.2 MAXILLARY SURGERY

1.3.2.1 LEFORT 1 OSTEOTOMY

The classification system of maxillary fracture patterns was created by Rene LeFort in 1901[14]. The first description of LeFort 1 osteotomy was by Wassmund in 1927[15]. A major advancement in the stability was described by Obwegeser, who recommended complete mobilization of the maxilla so that repositioning could be accomplished without soft tissue or bony resistance[16].

A brief description of the procedure as performed by the surgeons involved in this study is as follows; A maxillary vestibular incision is made from first pre-molar to first pre-molar region and a subperiosteal dissection is carried superiorly to the level of the infra-orbital foramen bilaterally and posterior until the pterygoid plates are encountered. Nasal mucosa is dissected free of the nasal floor and horizontal osteotomies are created with a reciprocating saw from the zygomaticomaxillary buttress through the lateral nasal walls. Following this, thin chisels are used to complete the osteotomies posteriorly at the lateral nasal walls and posterior maxillary walls until the solid resistance of the pterygoid plates. The nasal septum is relieved with a nasal septal osteotome. The surgeons involved in the study achieve separation of the pterygoid plates from the posterior maxilla without the use of an osteotome[17,18]. This technique was first described by Precious in 1991 and is now being advocated as a safer alternative to the use of the osteotome technique in the UK[19]. The maxilla is down-fractured with digital pressure. If more force is required a Tessier spreader is introduced into the piriform rim region and opened. Fixation typically includes 2.0mm L-shaped semi-rigid fixation plates adapted to the piriform rims bilaterally. The posterior maxilla is typically secured with double-twisted 28 gauge wire, but semi-rigid fixation plates are used when additional fixation is deemed necessary.

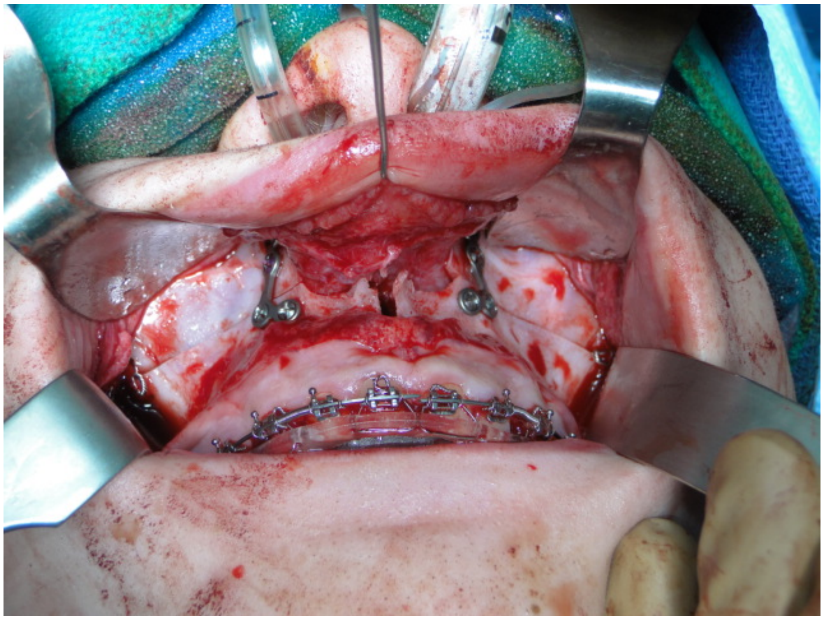

Figure 1. Intra-operative view of LeFort 1 osteotomy following fixation. Image Copyright Fisher, B 2018.

1.3.3 MANDIBULAR SURGERY

Mandibular orthognathic surgery has its beginnings in 1849 as described by Hullihen performing the anterior subapical osteotomy for the correction of mandibular retrognathia in a burn victim[20]. The emergence of mandibular orthognathic surgery did not occur until 50 years later when VP Blair described an extra-oral approach to a mandibular body osteotomy[21]. Progression and modifications to mandibular osteotomies continued including a shift to an intra-oral approach with preservation of the inferior-alveolar neurovascular bundle[22].

1.3.3.1 SAGITTAL SPLIT OSTEOTOMY

The present day workhorse of mandibular orthognathic surgery is the sagittal split osteotomy. The earliest description of an intraoral approach to a ramal osteotomy was published in German literature by Schuchart in 1942[23]. This technique was refined and popularized by Obwegeser, and subsequently introduced to North American surgeons in the 1960s[24]. Modifications to this technique were introduced by DalPont in 1961 and Hunsuck in 1968 to improve the predictability of adequate split and overlap of segments for stability[25,26]. The present day technique includes the modification by Bell, Schendel and Epker to extend the vertical cut through the inferior border of the mandible[27,28].

A brief description of the surgical steps to the technique as performed by the surgeons in the study are as follows (Figure 2); The surgeon makes an incision along the external oblique ridge from the midportion of the anterior border of the ramus to the area of the first molar. Subperiosteal dissection is carried laterally and then with the aid of a ramus stripper the tendinous attachement of the temporalis muscle is released from the anterior border of the ramus. Dissection is then carried medially and posterior to the region of the lingula. A Lindemann bur is used to complete at horizontal osteotomy cut into the retrolingualar fossa, through the lingual, halfway through the mid-portion of the ascending ramus. Dissection is then carried inferiorly to the inferior border of the mandible in the region of the first molar where again a Lindemann bur is used to create a vertical osteotomy through the inferior border. These two cuts are then joined in the sagittal plane using a 701 bur on a rotating handpiece. Separation of the segments is then completed in an anterior-to-posterior and superior-to-inferior direction with spatula and ¼ inch chisels, and Smith and Tessier spreaders. The IAN is then freed from the proximal segment and stripping of medial pterygoid from the proximal segment takes place to the angle of the mandible.

Fixation of the proximal and distal segments takes place with the teeth in maxillomandibular fixation (MMF), using heavy elastics and wire loops if necessary. Fixation is achieved by applying a 2.0mm semi-rigid fixation plate with a minimum of 4 mono-cortical screws (2 in each of the proximal and distal segments). While plating, the inferior borders are aligned. Exceptions to inferior border alignment may be made by the surgeon when there is significant counter-clockwise rotation of the maxilla-mandibular complex. The mandibular condyle is maintained with positive seating in the mandibular fossa throughout plating. This is accomplished by pressure in a posterior-superior direction on the proximal segment with an instrument held by the surgical assistant. Confirmation of proper position of the mandibular condyle is then checked by releasing the MMF and checking the occlusion with light digital pressure on the chin.

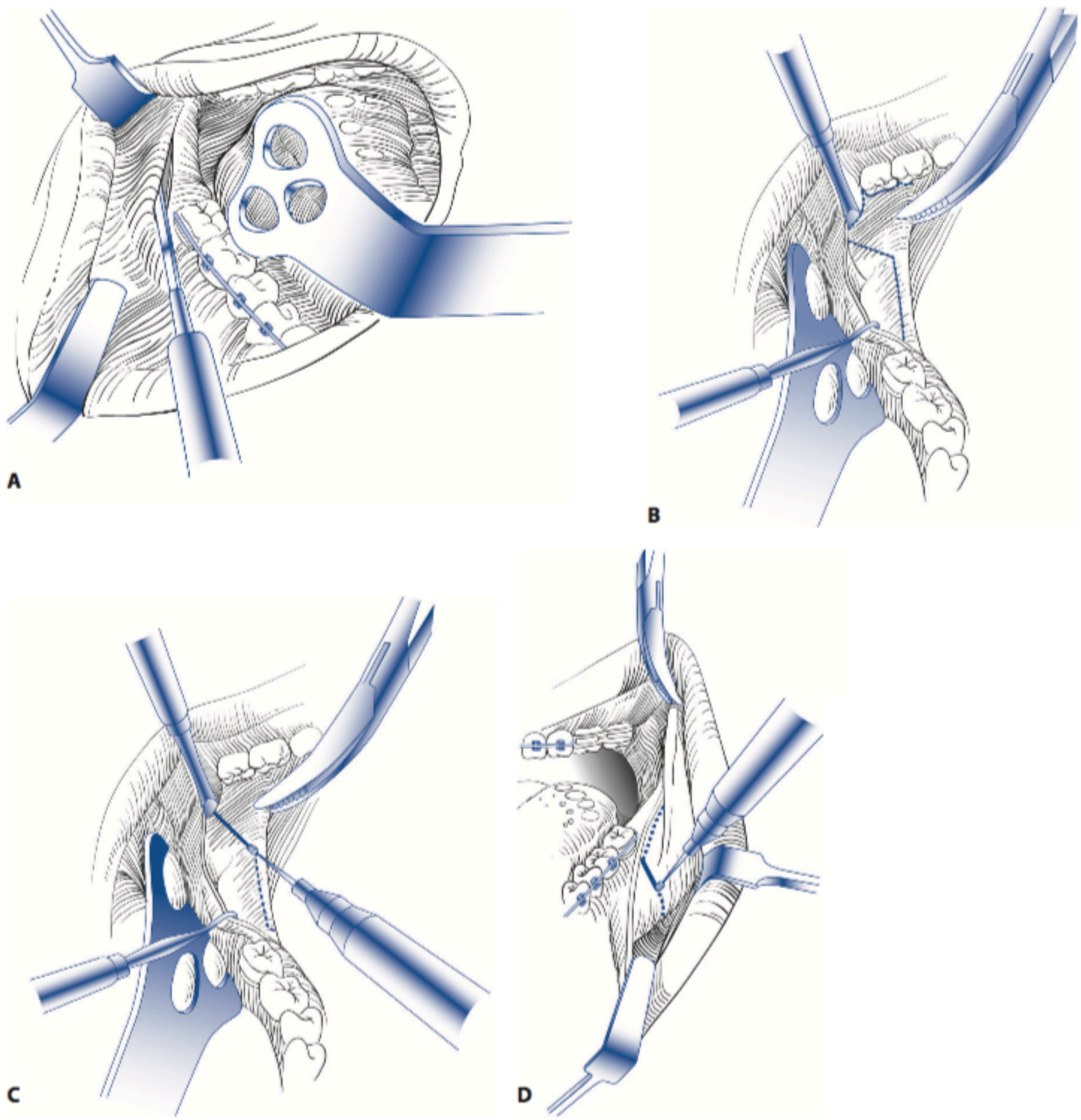

Figure 2. The bilateral sagittal split osteotomy. **A,** Incision. **B** and **C**, Medial exposure and horizontal cut. **D,** Vertical cut. Adapted from Bloomquist DS. Principles of mandibular orthognathic surgery. In Peterson LJ, Indresano AT, Marciani RD, Roser SM, editors. Principles of Oral and Maxillofacial Surgery. Vol 3. Philadelphia: JB Lippincott; 1992; pp. 1436–1437

1.3.3.2 INTRAORAL VERTICAL RAMUS OSTEOTOMY

Developed in 1954 by Caldwell and Letterman the Intra-oral vertical ramus osteotomy (IVRO) has been used in orthognathic surgery for small mandibular advancements (<2mm) as well as mandibular setbacks[29]. The procedure has a low rate of complications, but one of the drawbacks is a lack of rigid internal fixation requiring MMF for a minimum of 2 weeks post-operatively[30]. The IVRO has also become a popular extra-capsular treatment option for patients with painful internal derangements in which the disc is intact[31]. While the surgeons involved in the present study perform this operation frequently, it is less frequently used to reposition the mandible during orthognathic surgery, resorting to the more familiar SSO to accomplish mandibular movements.

1.3.3.3 FUNCTIONAL GENIOPLASTY

Trauner and Obwegeser described the sliding genioplasty via an intra-oral approach in 1957 for the correction of microgenia[32]. With the advent of plate and screw fixation in the 1980s segment stabilization was improved[33]. Functional genioplasty is indicated to maintain facial balance in orthognathic surgery[34,35]. Also, improvement in support for the mandibular incisors and gingival health from assuring lip competence are functional benefits.

The procedure begins with a mandibular vestibular incision from canine to canine, through mucosa and mentalis muscle, to the boney symphysis. Subperiosteal dissection is carried out posteriorly and inferiorly to the level of the mental neurovascular bundles. A reciprocating saw is used to create the horizontal osteotomy bilaterally through both the buccal and lingual mandibular cortices. Following this the free symphyseal segment is positioned according to the surgical treatment plan and fixated with a pre-bent 6-hole 2.0mm fixation plate[36]. Alternative fixation techniques that may be employed include 4 double-twisted 28-gauge wires or 2 bi-cortical positional screws of sufficient length (usually 15-19mm).

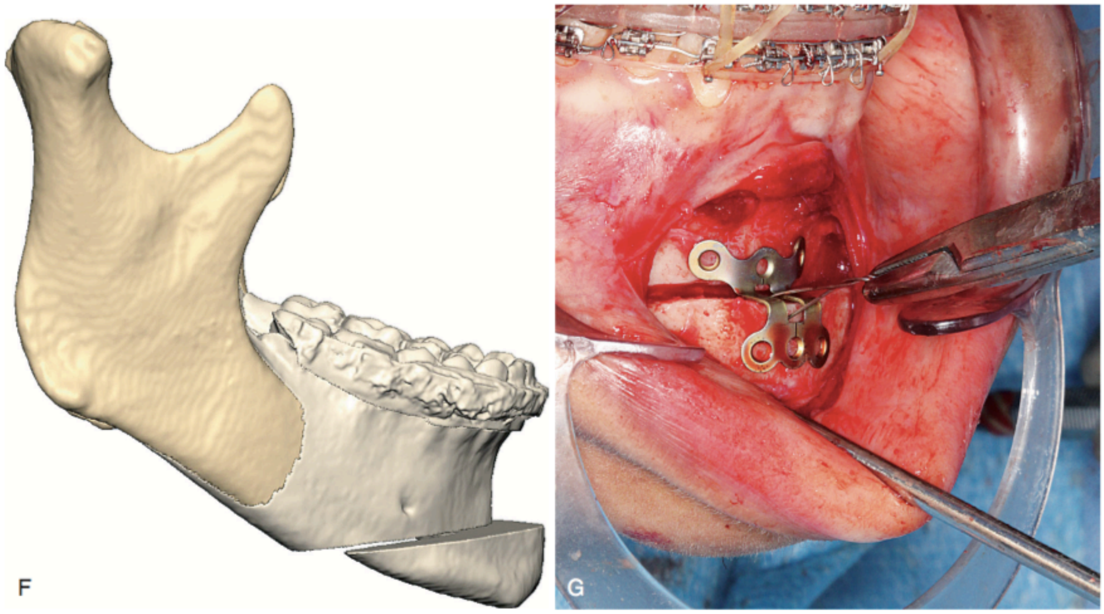

Figure 3. F, Sliding Functional Genioplasty computer animation demonstrating intended plane of horizontal cut for symphyseal advancement. **G**, Typical pre-bent 6-hole 2.0mm plate used for fixation of genioplasty. Image Copyright Kademani D, Tiwana P eds: Atlas of Oral and Maxillofacial Surgery. First Edition. St. Louis, Missouri: Saunders, an imprint of Elsevier, Inc, 2016. Chapter 29: Genioplasty. Pg. 287

1.4 TEMPOROMANDIBULAR JOINT

1.4.1 ANATOMY

The anatomy of the TMJ is complex. The temporomandibular joint (TMJ) is a ginglymoarthroidal joint that permits hinging and gliding of the mandible by translational and rotational movements. This allows for movement of the mandible to occur in 3 different planes[37]. Temporomandibular articulation is composed of bilateral, diarthrodial joints. Each of these synovial joints are formed by a mandibular condyle and corresponding mandibular fossa and articular eminence of the temporal bone[38]. The TMJ plays an essential role in guiding mandibular motion and distributing stresses produced by everyday tasks, such as talking, chewing and swallowing[39].

The articular disc lies in the space between the mandibular condyle and the mandibular fossa separating the two bones to create an inferior joint space and a superior joint space. The inferior joint space is responsible for helping create rotational movement during early opening, thought to represent the first 25mm of mouth opening. The superior joint space is responsible for translation between the disc and the fossa during later opening. Each compartment is filled with plasma-like synovial fluid secreted by Type B synoviocytes of the synovial lining within the joint capsule, which surrounds and protects the TMJ. Synovial fluid minimizes friction within the joint capsule and smooth movement of the joint by acting as a lubricant within the superior and inferior joint spaces[40]. It also aids in delivering metabolic requirements for the disc, as the disc itself is avascular[41]. The shape and morphology of the articular disc is determined by the condylar head and mandibular fossa. It is concave inferiorly and convex superiorly to fit over the condylar head and within the convex surface of the mandibular fossa of the temporal bone. The articular disc can be divided into three separate regions based on thickness. The anterior region is 2mm thick, the middle region is 1mm thick, and the posterior region is 3mm[41]. In a normal joint, the middle region, which is the thinnest region, is compressed between the mandibular condyle and fossa. From the anterior view, the disc is thicker medially than laterally. Its shape, as well as its firm attachment to the medial and lateral poles of the condyle help prevent the disc from displacement during function. The temporomandibular ligament exists at the lateral side of the articular capsule to prevent excessive movement of the mandible beyond normal range[41].

The articular disc is comprised of dense fibrous connective tissue and largely lacks innervation and blood supply. Conversely, the posterior attachment called the retrodiscal tissue is made up of loose connective tissue, is highly innervated, and has rich blood supply[38]. The retrodiscal tissue is superiorly bordered by the superior retrodiscal lamina which is made up of elastic fibers to allow translatory motions of disc in concert with the condyle. It is bordered inferiorly by the inferior retrodiscal lamina which is composed of collagenous fibers to increase rigidity[42]. The retrodiscal tissue is well recognized as being a major source of pain in TMD due to its vast innervation and close proximity to articulation within the TMJ[42,43].

1.4.2 UNIQUE QUALITIES OF THE TEMPOROMANDIBULAR JOINT

Unlike other hinged-typed joints found within the body, the TMJ is a bilateral joint meaning the left side cannot move independently of the right side[39]. The TMJ is unique when compared to other load-bearing articulations within the body with respect to its cartilaginous articular surfaces. Within most synovial joints in the body, the articular surfaces are covered by hyaline cartilage[38]. The TMJ is unique as its articular surfaces are composed of fibrocartilage. Fibrocartilage is unique with respect to its composition because it is made up of both type I and type II collagen, compared to articular hyaline cartilage, which only contains type II collagen[38]. This difference in composition may be one of the reasons why the TMJ can manifest unique symptoms or diseases unrelated to other joints in the body.

1.5 TEMPOROMANDIBULAR JOINT DISORDERS

1.5.1 ETIOLOGY AND DEMOGRAPHICS

Temporomandibular disorders (TMD) encompasses pathology of the TMJ, masticatory musculature and associated head and neck musculoskeletal structures[38]. The TMJ may be affected by inflammatory, traumatic, infectious, congenital, developmental, and neoplastic diseases[44]. TMD is a subgroup of craniofacial problems, with pain localized to the jaw, TMJ and muscles of mastication[44]. Presenting complaints of patients with TMD can include pain, limited or asymmetric mandibular motion, and TMJ sounds. Surrounding anatomical structures often give associated symptoms such as otalgia, aural fullness, tinnitus, dizziness, neck pain and headache[44]. TMD is the second most common cause of orofacial pain, following dental pain[45]. The prevalence of TMD is 6-12% of adults in the United States. It has also been found to be twice as prevalent in females than in males[38].

In general, TMD can be grouped into articular and non-articular disorders. These are also commonly referred to as intracapsular (intra-articular) and extracapsular conditions. Non-articular disorders are theorized to commonly be the result of parafunctional habits such as clenching and grinding[44]. Emotional stress predisposes to these parafunctional habits, therefore contributing to increasing TMD symptoms.[46]

Remodeling of the TMJ is an essential adaptation process needed for appropriate stress distribution and function[39]. Mechanically induced remodeling is a normal process to achieve optimal function. When the capacity for the joint to remodel has been exceeded, structural mal-alignment occurs[38,39]. Insufficient adaptation may result in the constellation of findings seen in osteoarthritis. Changes to the TMJ as a result of osteoarthritis include alterations in shape and size by flattening of the condyle, flattening of the articular eminence and decreased condylar volume[47].

The etiology of TMD is not well understood but multiple correlations exist between several parafunctional habits like nocturnal bruxing, tooth clenching, lip or cheek biting as well as osteoarthritis, neurogenic inflammation, age, sex and genetics[38,41].

1.5.1.1 AGE

Clinical signs of degenerative TMJ disease for both males and females increases with age[48]. This increased prevalence of degenerative TMJ disease with age probably reflects the reduced synthetic capacity of these aged articular tissues. Cell density in articular tissues of the TMJ has been shown to decrease steadily with aging. Concomitantly, a progressive loss of the cartilaginous matrix of these fibrocartilages occurs[48]. The fibrocartilaginous matrices of the mandibular condyles and temporal bone are gradually replaced with fibrous tissue over time, further reducing the inherent biological and mechanical properties that are advantageous to a heavily loaded joint[49].

1.5.1.2 GENDER

TMD occurs primarily between ages 18 and 45 in females. While other joint disease also have a female predilection, they typically occur following menopause[38]. Several lines of evidence point out that hormonal influences from estrogen, progesterone, and relaxin may make an individual susceptible to degeneration of the TMJ[50]. Multiple estrogen and progesterone receptors have been localized in the TMJ. Exogenous increase in estrogen by estrogen replacement therapy, or the use of oral contraceptives are associated an increase in TMD incidence[50]. It has been found that estrogen and relaxin may contribute to TMJ degeneration by enhancing the expression of specific fibrocartilage tissue degrading enzymes called matrix metalloproteinases (MMP)[50]. Supporting this idea of hormonal contribution, polymorphisms of multiple estrogen receptors have shown to be correlated with a greater intensity of pain associated with TMD[51]. Matrix degradation by MMPs is considered to be a primary event in the initiation and progression of joint disease, and this hormone-mediated loss in matrices likely affects the ability of the joint to sustain normal function leading to progressive degenerative changes within the joint. Together, MMPs with elevated levels of estrogen in women with TMJ disease suggest a potential role of specific sex hormones in causing TMJ degeneration[47,51].

1.5.1.3 INFLAMMATION

The TMJ is highly innervated at the anterior region of the articular disc as well as at the posterior retrodiscal tissues. These densely populated sensory neurons contain pro-inflammatory neuropeptides. These neuropeptides are thought to be released from the sensory neurons to adjacent articular tissues and synovial fluid when alteration of occlusal patterns and forced protrusion and retrusion of the mandible result in rapid remodeling of articular surfaces within the TMJ[48]. This causes a biochemical cascade resulting in secretion of other soluble molecules that cause a series of local inflammatory responses. However, the boundary separating normal adaptive responses from those of excessive or sustained mechanical loads is currently ill-defined[52].

Evidence of TMJ involvement in systemic inflammatory disease is well documented. Yildizer et al. in 2017 examined 79 patients with confirmed rheumatic diseases (rheumatoid arthritis, primary Sjogren's syndrome, and akylosing spondylitis) compared with age and gender matched controls. They found the prevalence of subjective symptoms of TMD in patients with rheumatic diseases (73.4%) was significantly higher than the controls (22.8%)[53]. Similar differences were noted with objective findings of lateral TMJ palpation, mandibular movements, and muscle pain.

1.5.1.4 GENETIC FACTORS

It is highly probable that an individual's genetic backdrop governs to a very large extent that individual's susceptibility to TMJ disease and pain. Certain mutations of collagen genes predispose affected individuals to degenerative arthritides[48]. Interestingly, pain sensitivity may also be genetically determined by assessing specific alleles implicated with masticatory muscle pain. It is plausible that future rapid genetic screening methods may be employed to assess an individual's susceptibility to degenerative TMJ disease and pain[47,51]. However, these methods of individual diagnosis are currently experimental.

1.5.2 EVALUATION AND DIAGNOSIS

Diagnostic, prognostic and therapeutic strategies for alleviating symptoms of TMJ disorder are best achieved by undertaking a thorough history and physical examination. Understanding the bio-medical basis for TMJ disorders is becoming clearer with computer and imaging technologies that provide novel insights into the pathogenesis of degenerative TMJ diseases. Additionally, appropriate use of imaging modalities such as plain and orthopantomographic radiography, cone beam computed tomography (CBCT), magnetic resonance imaging (MRI), and arthrography may help improve diagnosis[38,39,54]. MRI is considered the most beneficial imaging device for soft tissues, while CT is considered the most beneficial for imaging bony anatomy[55]. CT and CBCT additionally allow the joint to be visualized as sections in different planes. A 3-dimensional image can be rendered to enhance the diagnosis of dense osseous tissues of the TMJ by showing, in great detail, the internal derangement and joint dysfunction of the diarthroidal joint[54]. MRI has the added advantage over CT for improved viewing of soft tissues such as the disc, ligaments and muscles[55].

Various attempts at standardization of TMJ examination for diagnosis of TMD have been made. Arguably the most famous of these is the Helkimo index[56]. Helkimo developed the Anamnestic Index, Clinical Dysfunction Index and Occlusal State Index based off epidemiological studies during the early 1970s in Sweden[57,58]. There intended use was to evaluate the population for increasing severity of symptoms of jaw pain and dysfunction and occlusal instability in a retrospective fashion. Since these instruments were designed for epidemiological surveys, they have been criticized as being of limited use in clinical outcome studies because they are not sensitive enough to measure small changes in the condition[59]. They have also been described as neither easy to understand, nor simple to score[59]. Most concerning, the reliability for the non-parametric components of the index have demonstrated an unacceptably high degree of inter-observer variability[60]. Fricton attempted to overcome these issues with the development of the Craniomandibular Index, which did demonstrate improved inter-observer reliability[59]. However, this index requires the palpation and scoring of 22 defined points on each side of the patient. They also mention their own shortcoming of palpation of such well defined sites as the lateral pterygoid muscle, which were almost universally painful in all patients tested regardless of presence of TMD[59].

Another attempt at standardization of TMD diagnosis was the creation of the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) in 1992[61]. This questionnaire and examination provides the patient with a physical diagnosis (axis I) and a psychological diagnosis (axis 2). This tool was subsequently proven to be below the acceptable validity of ≥0.70 for axis I diagnosis and was revised to the DC/TMD in 2014[62]. The DC/TMD requires the patient to answer 32 questions, which unfortunately is a deterrent to patient participation.

The lack of adoption to standardized questioning and clinical examination is common throughout the literature on TMD. In a recent meta-analysis regarding orthognathic surgery and TMJ effects only 9 of 76 articles included used the Helkimo index or RDC/TMD[63].

1.5.3 TREATMENT

After gathering and assessing clinical data and making a correct diagnosis, an analysis of current non-invasive, minimally invasive and fully invasive management options can be explored. The ultimate goal of these modalities is to: 1) increase mandibular range of motion, 2) decrease inflammation and muscle pain, 3) prevent further degeneration of the articular tissues[39]. Initial non-invasive treatment modalities are often first employed[39,64]. Non-invasive techniques implemented most commonly include physical therapy, occlusal splints and non-steroidal anti-inflammatory medications[65]. Physical therapy aims to manually exercise the masticatory muscles that control the TMJ to help improve range of motion. Physical therapists may complement these techniques with behavioral changes by drawing awareness to the patient's posture, diet, and stress-related habits[39,64,65].

1.5.3.1 INTRA-ARTICULAR PATHOLOGY

When diagnosed with intra-articular TMJ disease, minimally invasive modalities are usually first exhausted. These include TMJ arthrocentesis, arthroscopy, lysis and lavage, and joint injection. Injection modalities for management of TMD symptoms include sodium hyaluronate and corticosteroid injections. Injections of corticosteroids are designed to treat osteoarthritic symptoms by reducing localized inflammation while high molecular weight sodium hyaluronate in the superior joint space provides lubrication similar to synovial fluid, as well as stimulation of synoviocytes to create more endogenous hyaluronic acid[66]. Typically these procedures require sedation or general anesthesia but can completed in an out-patient setting[67].

Patients who are non-responsive to minimally invasive surgery or have advanced disease may require invasive procedures to restore mandibular motion and mitigate the related orofacial pain[39]. Invasive "open-joint" procedures can include discopexy, discoplasty, discectomy, synovectomy, and arthroplasty. Endstage disease is typically treated with total joint reconstruction. Common modalities include autogenous costochondral grafts or alloplastic total joint replacement[67].

1.5.3.2 MYOFASCIAL PAIN DYSFUCTION SYNDROME

Myofascial pain dysfunction syndrome (commonly referred to as myofascial pain disorder (MPD)) is a psychophysiological disease associated with muscular structures. It is categorized as a regional soft-tissue pain syndrome, and can involve the muscles of mastication. It is categorized by extensive pain, decreased pain relief, sleep disruption, exhaustion, psychosomatic distress, and chronic headache[68]. These patients are typically diagnosed based on the presence of numerous fascial trigger points and taut muscular bands throughout the head and neck[69]. The development of the disorder is thought to originate in a combination of stress-related increased muscle tension and the existence of a parafunctional habit which manifest in muscular spasms and fatigue, leading to mandibular dysfunction[70]. The pain is frequently unilateral and headaches are a common complaint. The highest prevalence is found in females aged 20-40 years[71]. A 2015 study of 180 patients revealed that the number of comorbidities is positively associated with TMD pain duration and intensity, especially the conditions of migraine and chronic fatigue syndrome[72]. Chronic fatigue syndrome is contrast from MPD by displaying more generalized involvement, the presence of muscular tender-points in more than 11 spots, increased fatigue, and an increased association with irritable bowel syndrome[72]. Although they are different syndromes, they can co-exist.

Counseling the patient on the psychophysiological basis of the disease is extremely important. First line treatment may include the use of occlusal bite plane therapy for relief of parafunctional habits such as bruxism, along with a short course of a muscle relaxant, most commonly cyclobenzaprine. Modalities such as dry needling and acupuncture, and more recently ultrasound, have been shown restore blood flow to trigger point areas, decreasing pain[73]. Lidocaine and Botox injections to trigger point areas are also an accepted modality[74].

CHAPTER 2 – REVIEW OF LITERATURE

2.1 EFFECT OF ORTHOGNATHIC SURGERY ON THE TEMPOROMANDIBULAR JOINT

The most recent systematic review and meta-analysis on the subject was completed by Al-Moraissi et. al in 2017[75]. Their review of 29 studies, encompassing 5,029 total patients, showed 5 studies that supported an improvement of TMDs, 2 studies that showed no difference in TMDs, and 6 studies that supported worsening of TMDs. The main outcome variables evaluated were the signs and symptoms of TMD before and after orthognathic surgery and change in MIO. Follow-up ranged from 4 months to 4 years. Results of the meta-analysis showed retrognathic patients that underwent BSSO advancement showed a reduction in TMDs, but those undergoing BSSO and LeFort 1 osteotomy did not display improvement. Prognathic patients undergoing BSSO only setback did not have improvement in TMDs, while those undergoing BSSO and LeFort 1 displayed a reduction in TMDs. Overall, those with pre-existing TMDs tended to improve[75]. They also showed that there was a statistically significant increase of MIO in both Class II (5.7mm) and Class III (7.12mm) patient groups.

Al-Moraissi and colleagues postulate the reduction in TMDs may be due to better masticatory efficacy and muscular-balance, and fewer centric relation-centric occlusion discrepancies. They did find however, that Class II patients with high occlusal plane angles and pre-existing articular disc-displacements may have a poorer outcome compared with low or normal angle mandibular planes. They concluded that although there was an overall statistically significant TMD improvement following orthognathic surgery, not all patients will improve with orthognathic surgery and in fact some patients may actually worsen, including the development of TMD in those patients that were previously asymptomatic. Their recommendation was that surgeons must inform patients that orthognathic surgery may or may not improve pre-existing TMD signs and symptoms[75].

The Index of Orthognathic Functional Treatment Need (IOFTN), which was created in the United Kingdom in 2014 to help stratify patients in need of orthognathic surgery[76]. It was created in response to reduced government funding for orthognathic surgery as functional benefits of the surgery were not realized by government funding sources. It was based off a previous index that had been used to stratify patients that would benefit most from orthodontic treatment[77]. It consists of 5 groups of severity based on malocclusion that is not amenable to orthodontic treatment alone (Appendix E). Noted changes from the previous orthodontic based version include the provision of surgery for obstructive sleep apnea, skeletal anomalies resulting in occlusal disturbance as a result of trauma or pathology, facial asymmetry, and appearance of excessive upper labial segment gingival exposure. This tool has been proven to be valid in the categorization of those patients that would most benefit from orthognathic treatment[78,79]. It is interesting to note the "treatment purely for TMD" is listed as a Category 1 indication, which is stratified as "No Need for treatment". Therefore, it could be inferred that patients should not be undergoing orthognathic surgery solely for the purpose of treatment of TMD, unless a higher indication for orthognathic surgery is present.

2.1.1 TEMPOROMANDIBULAR JOINT PAIN

Complications associated with orthognathic surgery are numerous. Preoperative, intraoperative, and postoperative complications have all been described. In a 2014 review of 44 papers on the topic the incidence of the most common complications was compiled. This included cranial nerve injury/sensitivity alteration (50%), temporomandibular joint disorders or impairment (13.64%), hemorrhage (9.09%), auditory tube dysfunction and hearing problems (6.82%), infection (6.82%), bad split (4.55%), non-union of osteotomy gap (4.55%), skeletal relapse (4.55%), septum deviation (2.28%), bone necrosis (2.28%), soft tissue injuries (2.28%), positional vertigo (2.28%), and psychological depression (2.28%)[80]. With regards to the complications of the TMJ, the review found that a consensus on TMJ dysfunction has not been achieved. In a RCT comparing 30 healthy subjects with that of 30 pre-operative patients undergoing orthognathic surgery there was found to be no significant difference in the incidence of TMJ sounds, deviation of mouth opening, and tenderness of the TMJ and masticatory muscles between patients and volunteers at 3 and 6 month postoperative intervals[81]. Class II patients showed statistically significant subjective worsening in subjective TMJ symptoms compared to the Class III patients at 3 months. One third of TMJ joint noises decreased over the 3 month period, where as only one patient developed new joint noise. However, during the 3 to 6 month follow-up period one third of patients developed new TMJ sounds.

A retrospective study by Dujoncqouy et al. in 2010 showed that in 57 patients whom underwent orthognathic surgery 19.3% of patients reported a decrease in TMJ pain following surgery, while 17.5% reported an increase in TMJ pain[82].

2.1.2 TEMPOROMANDIBULAR JOINT HYPOMOBILITY

Maximum incisal opening has been documented to decrease following orthognathic surgery, as shown in a RCT of 30 patients compared with healthy volunteers at 3 and 6 month post-operative times points[81]. In this study, only lateral excursive movements were found to be statistically significantly decreased in the patient group vs. the healthy volunteers at 6 months[81]. Although a small sample size, the group of 10 patients with a correction of Class II malocclusion with mandibular advancement MIO decreased from an average of 48.7mm to 40.6mm at 6 months post-operative ($p<0.01$). The same effect was seen in the group of 14 patients in which mandibular setbacks were performed to correct Class III malocclusion. The MIO in this group decreased from 50.1mm to 41.3mm at 6 months ($p<0.01$). Clicking of the TMJ was associated with a larger decrease in MIO at 6 months, while other TMJ symptoms did not show an association with reduction in MIO. The authors concluded that there was not a large difference in TMJ symptoms between subjects with malocclusion and those without. Also, they observed that following correction of malocclusion, changes in TMJ symptoms did not always improve and in fact some patients showed changes for the worse[81].

The reduction of MIO following orthognathic surgery has been postulated to be due to two mechanisms[83]. First, intra-articular causes, mainly progressive internal derangement, such as anterior disc-displacement without reduction. Second, myofibrotic contracture resulting from the surgery. In a follow up ranging from 6-42 months post surgery, BSSO for mandibular advancement patients showed the greatest reduction in MIO compared to Lefort 1 osteotomy and BSSO mandibular setback patients.

Counterclockwise rotation of the proximal mandibular segment can cause the anterosuperior condylar surface to become located more superiorly in the glenoid fossa. It is also known that posteriorly positioned condyles are more likely to be associated with anterior disc displacements[84]. Therefore, incorrect condylar positioning at the time of surgery may predispose patients to development or worsening of internal derangements of the TMJ. The supine positioning of the patient at time of surgery as well as empirically directed posterior force on the proximal segment may inadvertently place the condyle in the posterior malposition.

2.1.3 IMPORTANCE OF CONDYLAR POSITIONING

Malposition of the condyle due to forces placed on the proximal segment by rigid internal fixation has been postulated to occur due to an unnatural torque on the condylar head when the fixation is applied. With historical wire fixation it was felt that the condyle is more likely to settle in a muscularly favorable position, not experiencing this external torqueing phenomenon. However, Nemeth et. al showed that when compared to wire fixation, rigid internal fixation did not show any increased risk for TMD and 2 year follow-up[85].

Condylar positioning devices have been developed to help control the movement of the proximal segment, ensuring proper condylar positioning. A review of the literature on these devices was reported by Costa et. al in 2008. They found only 3 studies looking at the use of condylar positioning devices and skeletal stability. None of the reviewed studies looked the use of condylar positioning devices and TMD following orthognathic surgery. Their review found 6 papers that compared the use of condylar repositioning devices and empiric condylar placement. They concluded there was no scientific evidence to support the routine use of condylar positioning devices in orthognathic surgery[86].

Positional plates and navigation guided condylar positioning systems generally rely on an assumed correct condylar position. Proper condylar positioning has also investigated through intra-operative use of sonography[87]. Advocates say advantage of such a technique allow real-time intraoperative monitoring and correction of condylar position. Landes showed that sonography guided condylar positioning was comparable to splint and plate positioning at 12 month follow-up with regards to condylar translation and recovery, dysfunction, and disc dislocation. A reported advantage was seen when comparing average time to use the sonography (5 minutes) with the conventional plate and splint placement (25 minutes).

Another method of assuring correct condylar positioning that has been investigated is that of intra-operative patient awakening to reduce condylar sag. The belief behind such a technique is that muscle tone will maintain contact across the TMJ. The process is described by Politi et. al in their comparison of empirical condylar placement versus intra-operative awakening of the patient[88]. The mandible was fixated with bicortical screws following manual positioning of the mandibular condyle into the glenoid fossa. The MMF was then released and the occlusion was checked with light digital pressure on the chin, which confirms proper mandibular positioning. In the study group of 76 patients, they were rapidly awakened while maintaining the intubation in a state of conscious analgo-sedation. The patient was then asked to open and close as well as perform lateral excursive movements. If the examination confirmed suitable occlusion then anesthesia was reinforced and the operation was completed. They found that in 11 of 76 patients malocclusion was observed after checking with digital pressure on the chin. In 8 patients there was a noted malocclusion when the patient was awoke that was not previously identified on digital manipulation. This allowed appropriate correction intra-operatively prior to conclusion of the operation. In the control group, 7 patients were noted to have malocclusion in the immediate post-operative period

(12-24hrs) that was not identified with digital manipulation. The author concluded that the combination of muscle tone, muscular activity, and proprioception appear to have important roles condylar positioning and therefore intraoperative awakening can help to correct incorrect condylar position[88]. No patients reported recall of the intraoperative event.

Condylar positioning following orthognathic surgery has been evaluated and shown to be stable during 1 year follow up periods via CBCT analysis[89]. Chen et. al showed that immediately following BSSO advancement combined with Lefort 1 osteotomy found that immediately following surgery the condyles tended to move posterio-inferiorly. However at 3 months, the condyles moved in an anterosuperior direction following occlusal splint removal. An overall trend was seen of posterosuperior movement of the condyles compared with the pre-operative position. This may be due to masticatory muscle stretching, resolution of edema, and removal of the occlusal splint.

2.1.4 EFFECT OF AGE

Peacock et al. reported a retrospective study of 911 patients undergoing orthognathic surgery divided into two groups, those <40 years of age and those ≥40 years of age. They found the group ≥40 years of age were more likely to seek treatment for functional reasons[90]. They also noted this increased age group had longer average hospital stays and 2.72 times a likely to require hardware removal at 6 months post-operatively. Verweij et al. found that an age >30 years was also associated with decreased neurosensory recovery than that of younger patients following sagittal spilt osteotomies.

2.1.5 EFFECT OF GENDER

It is known that the overall prevalence of TMD is greater in females than males by a ratio of ~3.3:1[91]. Despite this known fact, most studies do not separate outcomes by gender, making gender specific-outcomes difficult to infer. However, it has been well documented that females are vastly more affected by idiopathic condylar resorption. Idiopathic condylar resorption, or more appropriately termed condylar resorption following orthognathic surgery (CROS) is a condition with progressive degeneration in the morphology of the condyle after orthognathic surgery. Its development can be associated with functionally limiting pain. A systematic review of CROS by Catherine in 2015 concluded that the condition mainly occurred in 14 to 50 years old women with pre-existing TMJ dysfunction, estrogen deficiency, class II malocclusion with a high mandibular plane angle, a diminished posterior facial height and a posteriorly inclined condylar neck[92]. Additionally, mandibular advancements >10mm, counterclockwise rotation of the mandible and posterior condylar repositioning were found to be surgical risk factors for the development of CROS. A retrospective study by Hwang et. al demonstrated that pre-existing internal derangement was not found to be a risk factor for the development of CROS[93].

2.1.6 EFFECT ON INTERNAL DERANGEMENT

A retrospective study by Dujoncqouy et al. in 2010 showed that in 57 patients whom underwent orthognathic surgery 15.8% reported an improvement in TMJ noise and clicking following surgery[82]. The disappearance in TMJ joint noises in the initial healing period following orthognathic surgery may be due to reduced MIO. This was postulated by Onizawa et. al who also showed that when MIO recovered to near pre-operative ranges, the incidence of TMJ clicking also increased. This would make anatomical sense and we are aware the first 25mm of MIO is a rotational movement of the condylar head on the temporomandibular disc. MIO greater than 25mm must utilize translational movement, and therefore a reducing anterior disc displacement as evidenced by a click, would become apparent.

2.1.7 EFFECT ON PARAFUNCTIONAL HABIT

Self reported parafunctional habits such as clenching or bruxism are seen in 10.4-33.0% of patients displaying TMD symptoms[94]. Also, it has been shown that a bruxism habit is associated with the presence of TMD[95]. A 2001 study by Yamada et. al evaluated 94 female patients, all with signs or symptoms of TMD, undergoing orthognathic surgery. They assessed bony change of the condyle as seen on CT volumetric analysis. This study showed that the presence of self-reported clenching or grinding was a statistically significant risk factor for bony changes of the condyle[94]. Arthroscopically, bruxism has been shown to increase the presence of osteoarthritis of the TMJ[96]. Increased condylar loading, decreased local blood flow due to microcirculation disorder and increased pain due to ischemia have been postulated as the sources of bruxism related TMD signs and symptoms[95]. Data on the effect of orthognathic surgery on self-reported bruxism habit is lacking, with no specific studies in the literature found investigating this topic.

2.1.8 EFFECT OF TYPE OF ORTHOGNATHIC SURGERY PERFORMED

Recovery of MIO has been shown to be dependent on the type of orthognathic surgery performed. This is demonstrated by Ueki et. al whom showed a larger recovery of pre-operative MIO at 6 months of those that underwent only a BSSO compared with those that underwent a Lefort 1 osteotomy and BSSO[97]. They also demonstrated that a longer period of post-operative MMF was correlated with a longer recovery of MIO[97]. The standard length of MMF used at our institution is 14days, after which light guiding elastics, permitting increased function, are applied.

Correction of vertical maxillary excess demonstrated a difference in the type of surgery on the change in prevalence of TMD at 6 months post-operative. Those undergoing LeFort 1 and BSSO for correction of VME had an increased prevalence of TMD of 15%, while those only undergoing a LeFort 1 osteotomy alone had a decreased prevalence of 18%[98].

A 2016 systematic review of 22 articles on mandibular advancement surgery's effect on the TMJ concluded that despite the large number of studies on the subject, it can neither be said to improve nor to worsen TMJ health[99].

2.1.9 EFFECT OF MAGNITUDE OF SURGICAL MOVEMENTS

It has been thought that with increasing magnitude of mandibular advancement there is an increased incidence of TMD symptomology after orthognathic surgery due to increased condylar compression of the bilaminar tissue against the superior and posterior walls of the fossa, thus contributing to pain and inflammation[100].

A review of 126 patients that underwent mandibular advancement procedures found that there was no correlation of magnitude of mandibular advancement with development of muscular of joint related symptoms of TMD post-surgically[101]. However, this study did find that the combination of large magnitude of advancement (>7mm) in addition to a counter-clockwise rotation of the mandibular plane was associated with increased joint sounds, palpable capsular pain and decrease mandibular range of motion. These results are similar to those of a review completed in 2013 on the subject which stated that young females with high mandibular plane angles and larger retrognathic discrepancies may experience less improvement of TMD symptoms following surgery[102].

2.1.10 EFFECT OF THIRD MOLAR REMOVAL

At our institution we elect to remove third molars at the time of orthognathic surgery. This is based on studies which demonstrated no increase in unfavorable splits of the mandible as well as lower incidences of neurosensory dysfunction[103,104]. This has even been demonstrated in patients greater than 30 years of age[105]. A literature search with regards to concomitant removal of third molars at the time of orthognathic surgery does not reveal any results with regards to TMJ outcomes or recovery of MIO. However, independently performed third molar removal has been shown as a risk factor for the development of TMD[106]. Therefore, it would be hypothesized that minimizing multiple interventions that stress the TMJ would be another potential positive effect of removing third molars in a single surgery. Additionally, the positive social implications of performing just one surgical procedure have been realized[107].

CHAPTER 3 – PURPOSE OF THE STUDY

The purpose of this study is to determine the effect orthognathic surgery has on TMJ-related pain and function, and identify pre-operative patient-related factors that may be used to help predict TMJ outcome following orthognathic surgery.

CHAPTER 4 – PATIENTS AND METHODS

4.1 SUBJECTS

Nova Scotia Health Research Ethics Board approval for study enrollment was granted on October 17, 2016. All patients scheduled for orthognathic surgery from November 2016 until September 2017 in the Department of Oral and Maxillofacial Surgery at Queen Elizabeth Health Sciences Centre In Halifax, NS were eligible for study inclusion. Only those patients with complete pre and post-operative records were included in the analysis. There were no exclusion criteria.

4.2 METHODS

4.2.1 SUBJECT SELECTION

Patients were asked at their pre-operative visit (typically 1-14 days pre-surgery) if they would like to participate in the study. The study consent was reviewed by a clinical nurse, resident or staff surgeon in the Department of Oral and Maxillofacial Surgery and was completed by the patient. Following their agreement to participate, the Pre-operative Temporomandibular Joint Pain and Function Questionnaire was completed by the participant (Appendix C). Record taking at the pre-surgical visit was completed as per the department's normal Pre-operative Orthognathic Surgery Assessment (Appendix D). The consent in the study allowed the data from this form to be extracted as it related to TMJ function. Data collected included age, gender, maximum incisal opening (MIO), lateral excursive and protrusive movements, deviation/deflection on opening, presence of clicks/pops/crepitus of the TM joints, pre-operative Angle classification of occlusion, overbite, overjet, presence of crossbite, dental and facial midlines as well as vertical and horizontal positions of the maxilla and mandible. This information was used to compare changes in pre and post-operative values for each patient. Poor outcomes would be characterized by an increase in pain VAS, combined with a decrease in MIO or TMJ mobility.

4.2.2 DATA COLLECTION

All subjects underwent an objective clinical examination and completed a self-administered questionnaire. Both of these modalities focused on TMJ sounds, pain, and range of motion. A subjective questionnaire (Appendix C) focused on the primary interests of the study including TMJ-related pain, joint noise, locking, and the presence of a parafunctional habit.

Clinical examination were performed and completed by one of 3 examiners, all of whom were Oral and Maxillofacial Surgery residents. These examiners were calibrated for inter-observer reliability. The same examiner may or may not have re-examined the same patient pre and post-operatively. TMJ sounds, including clicks and crepitation were determined by means of palpation of both TMJs laterally during opening and closing motions. The presence of vibrations were interpreted as joint noise. TMJ pain occurring during opening or closing was evaluated by pressure placed on the lateral pole of the mandibular condyle and lateral capsule of the TMJ by the examiner. The patient was asked "Does this hurt?" and asked for a Yes or No response. Objective measures of TMJ range of motion were recorded by the examiner with aid of a flexible ruler. Maximum incisal opening was recorded as the distance between the right maxillary central incisal edge and right mandibular central incisal edge. Overbite (vertical overlap) and overjet (horizontal overlap) were also measured in the standard fashion from these points of reference. These points were also used to determine protrusive movement. The maxillary and mandibular dental midlines were used to determine lateral excursive movements and mandibular deviation upon opening.

Orthognathic surgery was planned and performed in the standard fashion as undertaken by the Department of Oral and Maxillofacial Surgery. This includes pre-operative occlusal models, photographs and radiographs. The Delaire method of cephalometric analysis was used to aid in determination of the operative plan[34]. Occlusal splints fabricated with poly-methyl-methacrylate were generated from models mounted on a Galetti articular. These splints were used intra-operatively to ensure the final occlusion was achieved as per our pre-surgical planning. For cases with more complex movements of the maxilla and mandible, intermediate splints were generated. This is done at the discretion of the operating surgeon. Using an Erikson Model Block and Platform Model Measuring Kit (Great Lakes Orthodontics. Tonawanda, NY) model surgery was performed using the Analytic Model Surgery method as outlined by Erickson and Bell[108]. A total of 6 attending oral & maxillofacial surgeons at our institution performed the procedures in the study.

Dentofacial deformities were surgically corrected by three main procedures. This includes the Le Fort 1 osteotomy, the bilateral sagittal split osteotomy and the functional genioplasty, as discussed in Chapter 2. The method of surgery was the same for all patients, regardless of the surgeon performing the procedure. All surgeons followed the same general orthognathic surgery procedure and fixation techniques. It was also recorded whether third molars were removed at the time of surgery. The magnitude and direction of surgical movements of the mandible, maxilla and symphysis was recorded by the operating surgeon in the operative note. These values were recorded as interval (continuous) data points. All patients are placed in maxillomandibular fixation (MMF) with the aid of heavy elastics (6.5oz) prior to emergence from general anesthetic.

Usual recovery from surgery included 1-3 nights in hospital. Those with mandible only surgery typically stayed 1-2 nights in hospital, while those that had a Le Fort 1 osteotomy performed typically stayed 2-3 nights in hospital. Discharge home was contingent that self-care could be assured. Post-operative care instructions were reviewed with the patient and caregiver prior to discharge. These instructions included strict instructions for oral hygiene, limitation of heavy lifting, and to have elastics replaced should any break during the initial post-operative period. Standard prescriptions upon discharge included Acetaminophen 650mg po q4h prn, Ibuprofen 600mg po q6h prn, Hydromorphone 2-4mg po q4-6h prn and Chlorhexidine 0.12% oral rinse 10ml BID x 14days. Patients were given contact information in case of emergency.

Follow-up was undertaken at 2, 4 and 8 weeks post-operatively. A follow up visit at 6 months post-operative was undertook, which usually coincided with the removal of orthodontic brackets. MMF was released at 2 weeks post-operative and patients are given a TMJ physiotherapy regimen. This regimen included stretching the jaw via active opening for 10 seconds, then resting for 10 seconds, moving the mandible side-to-side, as well as protrusive movement. These active physiotherapy exercises were to be completed with the guiding elastics removed, 5 times daily. All surgeons in the study used the same physiotherapy regimen. At these follow-up visits TMJ function was assessed and recorded in the clinical note. These notes were accessed via the patient's electronic health record for clinical data extraction. Participants of the study also completed a 6 month Post-operative Temporomandibular Joint Pain and Function Questionnaire as well as the same TMJ examination that was completed prior to surgery. These pre and post-operative values were then recorded for data analysis.

4.2.3 STATISTICS

Using IBM SPSS Statistics Version 25.0 (IBM Corporation. Armonk, NY), data analysis was completed to assess for variations from expected pre-operative and post-operative (6 month) values. Variations from expected values were investigated to see if they represent a relationship between outcome measures (ie. MIO) and variables of patient factors (ie. age) as well as surgical factors (ie. completion of BSSO). A confidence interval of 95% and p value of <0.05 were selected to be statistically significant (as is generally accepted in medical research), to indicate that the values seen in pre-operative and post-operative data was different than would could be expected by random chance alone.

Continuous data, which consisted of MIO, associated mandibular range of motion (lateral excursion and protrusion) and pain VAS, was recorded in regular intervals (ie. 1mm) for both ease of reporting and data analysis. These values were taken at two time points, that being pre-operative and 6 months post-operative.

Ordinal data comprised the rest of the recorded data. This included all the answers (excluding pain VAS) to the Temporomandibular Joint Pain and Function Questionnaire (Appendix C). It also included the data collected (in a binary system) from the TMJ examination portion of the Orthognathic Surgery Evaluation (Appendix D). This ordinal data was collected at the same time points as the interval data, pre-operative and 6 months post-operative.

Population and variable analysis included descriptive analysis including mean with standard deviation, median and mode. Age and gender were described. The change in MIO, excursion, protrusion, and pain VAS scores pre-operative vs. post-operative were analyzed by Paired Sample T-Test to identify any statistically significant change. One-Sample Chi-square Test with cross-tabulation was used to identify statistically significant change in pre-operative vs. post-operative ordinal data. (ie. TMJ questionnaire with binary responses). If values were <5, a Fisher's Exact test was instead used. Bivariate Pearson Correlation analysis was completed to identify any statistically significant relationship between continuous variables (ie. change in MIO vs. magnitude of mandibular advancement). The One-way Analysis of Variance (ANOVA) test was used to determine if each ordinal variable (ie. gender) had a relationship with the interval data (ie. Change in MIO).

CHAPTER 5 – RESULTS

5.1 STUDY POPULATION

Nova Scotia Health Authority Research Ethics Board (REB) approval was granted October 17, 2016, with a subsequent one-year renewal on October 17, 2017. Enrollment of patients in the study commenced November 1, 2016. During the months of enrollment, November 2016 until September 2017, 290 orthognathic surgery patients were approached to enroll in the study. A total of 152 patients agreed to participate and were consented for enrollment in the study. This was an initial acceptance of 52.4%. All of these subjects completed the pre-operative temporomandibular joint outcomes questionnaire (Appendix B) as well a complete orthognathic evaluation (Appendix A) in preparation for their orthognathic surgery. Of these 152 patients, 56 (36.8%) completed the 6 month post-operative follow up visit, along with completion of the questionnaire and TMJ examination at that time period. The statistical analysis was completed with IBM SPSS Statistics Version 25.0.

Only those 56 patients whom completed the post-operative questionnaire and TMJ evaluation were included in the complete statistics. However, an analysis of those 96 (63.1%) patients that did not complete their 6 month visit was also done to compare with the patient population that did complete the entirety of the study (Table 1).

Of the patients that completed only the pre-operative visit (n=96), the mean age was 25.4 year (SD=11.2). The median age was 20 years and the mode was 16 years (frequency=14). The range was 40 years, with the minimum being 15 years and the maximum being 55 years. Of these patients, 33.3% (n=32) were male and 66.7% (n=64) were female. The pre-operative pain VAS was a mean of 1.25 (SD=2). The median and modes were both 0 (frequency=60).

Table 1. *Patient Pre-operative Data of Study Drop-outs (n=96) and Patients Completing 6 Month Follow-up Visit (n=56)*

		Mean	Median	Mode	Std. Deviation	Range	Min.	Max.
Age (years)	Drop-outs	25.4	20.0	16	11.2	40	15	55
	Completed Study	28.1	24.5	16	13.3	50	12	62
Pre-operative Pain VAS	Drop-outs	1.25	0	0	2.0	8	0	8
	Completed Study	1.14	0	0	2.1	8	0	8

5.1.1 AGE

For those completing the study (n=56) the mean age was 28.1 years and the standard deviation was 13.3 years. Median age was 24.5 years, and mode of 16 years (frequency=6). The age range was 50 years, with a minimum age of 12 years, and a maximum age of 62 years. As demonstrated by Figure 4, the distribution of age was more normally distributed in the age range of 12-30 years, and therefore two age range groupings of Age <30 years or Age ≥30 years were created for further sub-analysis of certain variables. Age <30 years contained 66.1% (n=37) patients, while Age ≥30 years contained 33.9% (n=19) patients.

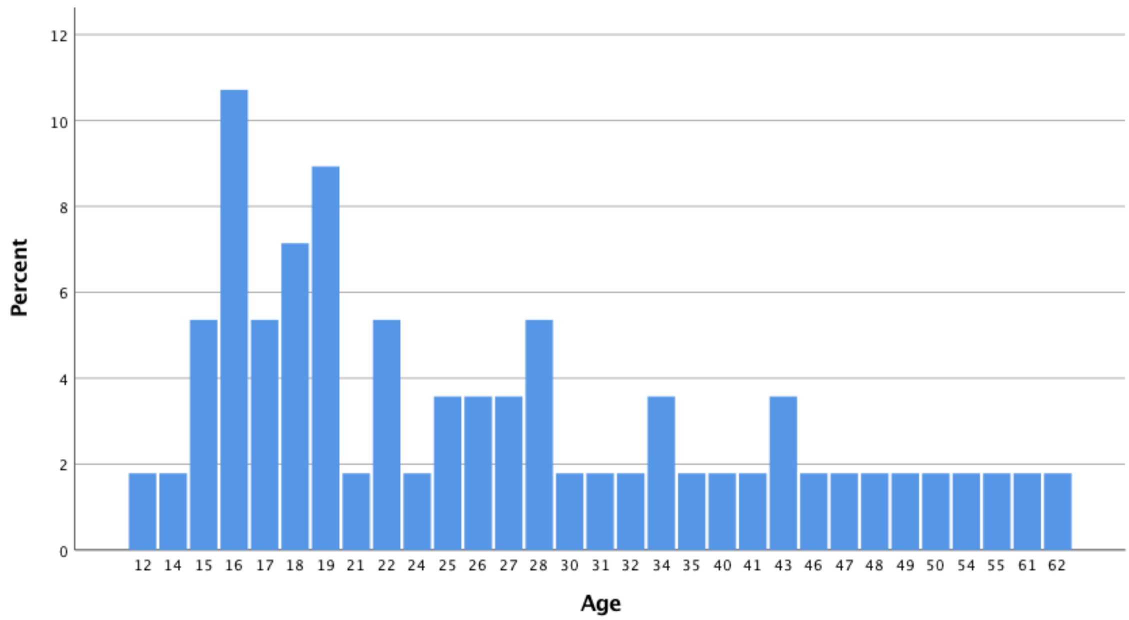

Figure 4. Distribution of patient age.

5.1.2 GENDER

Of the 56 patients that completed the study 37.5% (n=21) were male and 62.5% (n=35) were female.

5.1.3 MALOCCLUSION AND DENTOFACIAL DEFORMITY

Pre-operative malocclusion was classified as Class II in 64.3% (n=36) and Class III in 35.7% (n=20). Class II patients (n=36) showed a mean pre-operative OB of 2.7mm (SD=2.8mm, range -3 to 8mm). The Class II mean pre-operative OJ was 6.6mm (SD=2.4mm, range 2 to 11mm). Class III patients (n=20) showed a mean pre-operative OB of -0.2mm (SD=2.2mm, range -6 to 4mm). The Class III mean pre-operative OJ was -1.6mm (SD=4.7mm, range -7 to 11mm).

Table 2. *Pre-operative Overbite and Overjet by Pre-operative Malocclusion*

(mm)		Mean	Median	Mode	Std. Deviation	Range	Min.	Max.
Pre-operative OB	Class II	2.7	3	-	2.8	11	-3	8
	Class III	-0.2	0	-	2.2	10	-6	4
	Total	1.7	2	4	3.0	14	-6	8
Pre-operative OJ	Class II	6.6	6	-	2.4	9	2	11
	Class III	-1.6	-1	-	2.9	11	-7	4
	Total	3.7	5	6	4.7	18	-7	11

The total mean pre-operative overbite was 1.7mm (SD=3.0mm), mode of 4mm (frequency=10), with a range of 14mm (minimum -6mm, maximum 8mm). The total mean pre-operative overjet was 3.7mm (SD=4.7mm), mode of 6mm (frequency=10), with a range of 18mm (minimum -7mm, maximum 11mm). Vertical maxillary deficiency was diagnosed in 26.8% (n=15). Vertical maxillary excess was diagnosed in 19.6% (n=11) of patients. The patient presented with a diagnosis of obstructive sleep apnea in 7.1% (n=4) of cases.

5.2 SURGICAL MOVEMENTS

Of the 56 operations performed, LeFort 1 osteotomy was performed in 83.9% (n=47) of patients. BSSO was performed in 89.3% (n=50) of patients. Functional genioplasty was performed in 32.1% (n=18) of patients. The combination of Lefort 1 osteotomy combined with BSSO was performed in 71.4% (n=40) cases. LeFort 1 osteotomy without BSSO was performed in 10.7% (n=6) cases. BSSO without LeFort 1 osteotomy was performed in 17.9% (n=10) cases. Third molars were extracted in 48.2% (n=27) of patients.

Table 3. *Magnitude of Surgical Movements*

(mm)	Mean	Median	Mode	Std. Deviation	Range	Min.	Max.
Maxillary Anterior Movement	2.8	2	0[a]	2.7	9	0	9
Maxillary Superior Movement	0.3	0	0	1.6	7	-3	4
Mandibular Anterior Movement	4.7	3	5	4.6	18	-6	12
Genioplasty Movement	4.3	5	5	3.2	12	-4	8

a = multiple modes present, smallest is shown.

The mean magnitude of maxillary anterior-posterior movement was an advancement of 2.8mm (SD=2.7mm). The modes were 0 and 2mm (frequency=12). The range was 9mm (minimum 0mm, maximum 9mm). The mean magnitude of maxillary vertical movement was a superior movement (impaction) of 0.3mm (SD=1.6mm). The mode was 0mm (frequency=34). The range was 7mm (minimum -3mm, maximum 4mm).

The mean magnitude of mandibular anterior-posterior movement was an advancement of 4.7mm (SD=4.6mm). The mode was 5mm (frequency=11). The range was 18mm (minimum -6mm, maximum 12mm).

The mean magnitude of functional genioplasty anterior-posterior movement was an advancement of 1.7mm (SD=0.5mm). The mode was 5mm (frequency=10). The range was 12mm (minimum -4mm, maximum 8mm).

Table 4. *Maxillary and Mandibular AP Surgical Movement by Pre-operative Malocclusion*

	Maxillary AP Movement					Mandibular AP Movement				
	N	Mean	Max.	Min.	Sig.	N	Mean	Max.	Min.	Sig.
Class II	28	2.6	9	0	p=0.591 (One-way ANOVA)	35	7.0	12	2	p<0.01 (One-way ANOVA)
Class III	18	3.1	6	0		15	-0.7	5	-6	
Total	46	2.8	9	0		10	4.7	12	-6	

Class II patients that had maxillary surgery (n=28) showed a mean maxillary AP advancement of 2.6mm (range 9-0mm), while Class III patients had a mean maxillary advancement of 3.1mm (range 6-0mm). This difference was not statistically significant between groups (p=0.591).

Class II patients that had mandibular surgery (n=35) showed a mean mandibular advancement of 7.0mm (range 12-2mm), while Class III patients (n=15) had a mean mandibular AP change of -0.7mm (range -6-5mm). This difference was found to be statistically significant between groups (p<0.01).

5.3 MANDIBULAR RANGE OF MOTION

Table 5. *Summary of TMJ Interval Data Pre-operative, Post-operative, and Change*

(mm)		Mean	Median	Mode	SD	Range	Min.	Max.	Sig.
MIO	Pre-op	45.6	45	45	4.3	18	37	55	p=0.144 (paired sample T-Test)
	Post-op	44.0	45	45	7.8	39	26	65	
	Change	-1.6	-0.5	0	7.9	42	-27	15	
Right Lateral Excursion	Pre-op	8.2	8	8	2.8	14	2	16	p=0.096 (paired sample T-Test)
	Post-op	7.6	8	10	2.5	11	1	12	
	Change	-0.6	0	2	2.7	14	-9	5	
Left Lateral Excursion	Pre-op	8.9	9	10	2.7	14	2	16	p=0.095 (paired sample T-Test)
	Post-op	8.1	8	10	2.5	12	2	14	
	Change	-0.7	-0.5	-4[a]	3.2	16	-8	8	
Total Excursion	Pre-op	17.1	16	15.0[a]	4.9	28	4	32	p=0.061 (paired sample T-Test)
	Post-op	15.8	16	16	4.4	21	3	24	
	Change	-1.3	-1	-4[a]	5.2	24	-14	10	
Protrusion	Pre-op	7.6	7.5	6	2.2	10	2	12	p<0.01 (paired sample T-Test)
	Post-op	5.0	5	4	2.3	10	0	10	
	Change	-2.5	-2	-4[a]	2.6	11	-9	2	

a = multiple modes present, smallest is shown.

5.3.1 MAXIMUM INCISAL OPENING

The pre-operative mean MIO was 45.6mm. The standard deviation was 4.3mm. The median was 45mm, and the mode (frequency=7) was 45mm. The range was 18mm, with minimum opening of 37mm and maximum opening of 55mm. As displayed by Figure 5, the pre-operative MIO was normal distributed with a skewness of 0.308.

Post-operative mean MIO was 44.0mm. The standard deviation was 7.8mm. Median MIO was 45mm and mode (frequency=10) was 45mm. The range was 39mm with a minimum MIO of 26mm and a maximum MIO of 65mm. The data was evenly distributed (skewness=-0.005) as displayed in Figure 6. Overall, 12.5% (n=7) of patients had a post-operative MIO of <35mm, while 23.2% (n=13) of patients had a post-operative MIO of <40mm.

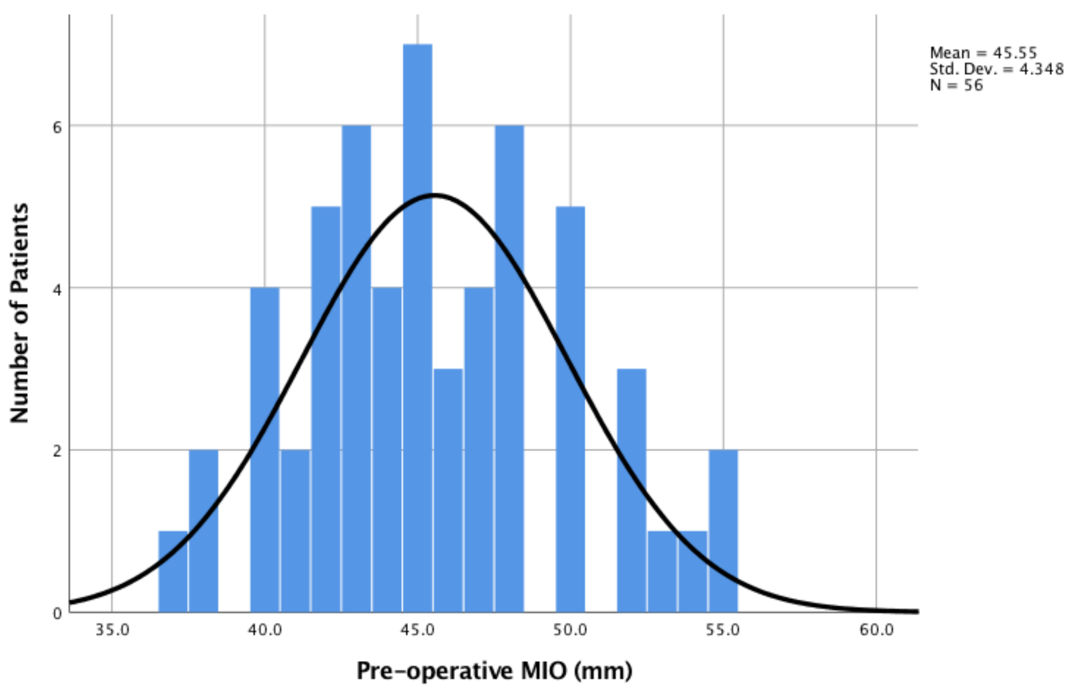

Figure 5. Distribution of pre-operative MIO (mm) displaying normal curve.

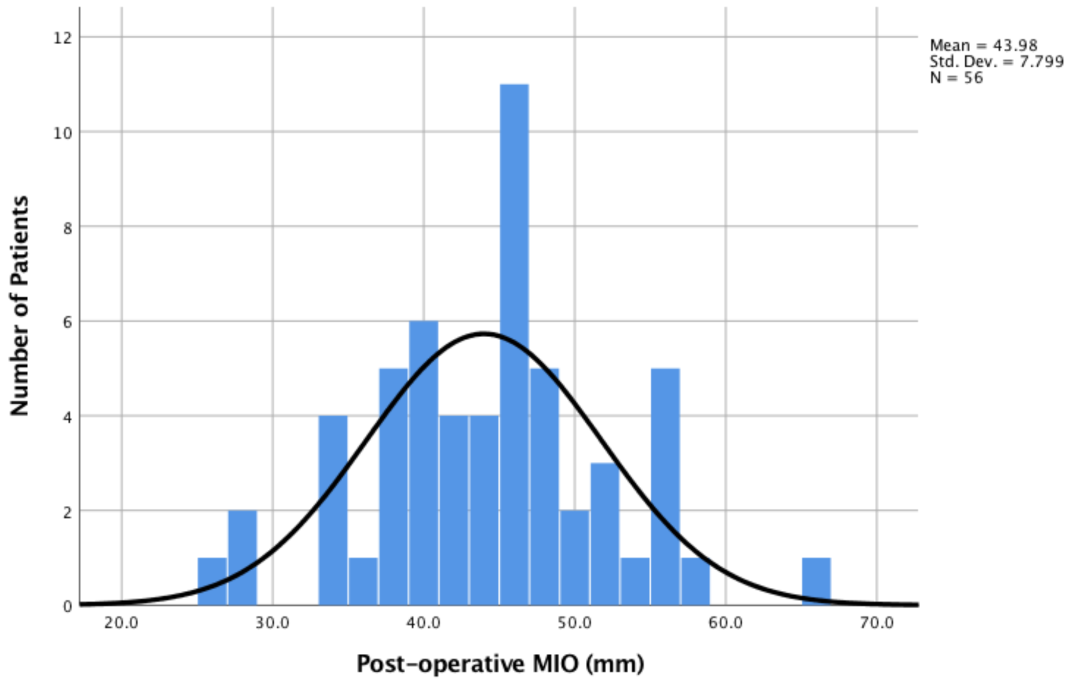

Figure 6. Distribution of post-operative MIO (mm) displaying normal curve.

No change in MIO was seen in 12.5% (n=7) of patients. An increase >5mm of MIO was seen in 19.6% (n=11) of patients, and an increase of >10mm in 5.4% (n=3). While a decrease of >5mm of MIO was seen in 28.6% (n=16) of patients, and a decrease of >10mm in 16.1% (n=9).

The mean change in MIO was -1.6mm. The Paired Sample T-Test of the pre-operative and post-operative MIO means showed that this was not a statistically significant change (p=0.144). The median change was -0.5mm and the mode (frequency=7) was 0mm. The standard deviation was 7.9mm. The range was 42mm with the largest decrease in MIO of -27mm, and the largest increase in MIO of 15mm. The distribution is displayed in Figure 7.

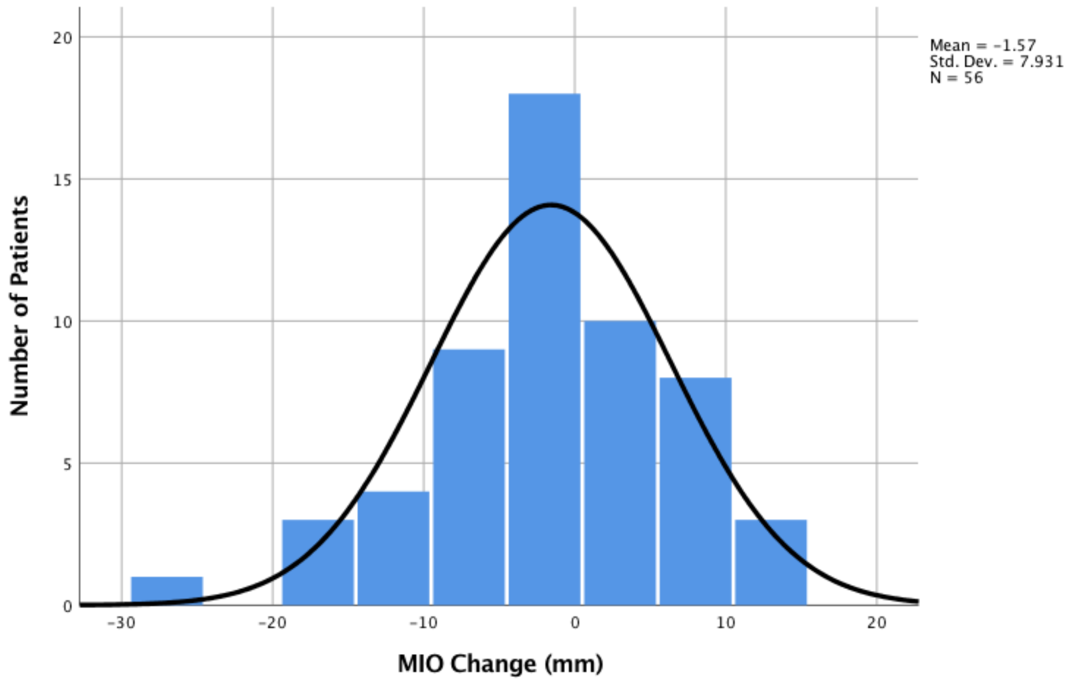

Figure 7. Distribution of MIO change (mm) displaying normal curve.

5.3.2 EXCURSION

Mean pre-operative right-lateral excursion was found to be 8.2mm (SD 2.8mm). Mean post-operative right-lateral excursion was found to be 7.6mm (SD 2.5mm). The mean difference of these pre-operative vs. post-operative values was -0.6mm. This difference did not reach statistical significance (p=0.096).

Mean pre-operative left-lateral excursion was found to be 8.8mm (SD 2.7mm). Mean post-operative left-lateral excursion was found to be 8.1mm (SD 2.5mm). The mean difference of these pre-operative vs. post-operative values was -0.7mm. This difference did not reach statistical significance (p=0.095).

Mean pre-operative total-excursion (sum of right and left lateral-excursion) was found to be 17.1mm (SD 4.9mm). Mean post-operative total-excursion was found to be 15.8mm (SD 4.4mm). The mean difference of these pre-operative vs. post-operative values was -1.3mm. This difference did not reach statistical significance (p=0.061).

5.3.3 PROTRUSION

Mean pre-operative protrusive movement was found to be 7.6mm (SD 2.2mm). Mean post-operative protrusion was found to be 5.0mm (SD 2.3mm). The mean difference of these pre-operative vs. post-operative values was -2.5mm. This difference represents statistical significance ($p<0.01$).

5.3.4 EFFECT OF AGE

Using bivariate correlation age did not correlate with change in MIO ($p=0.064$). Age <30 had a mean reduction in MIO of -0.27mm, whereas age ≥30 years had a mean reduction in opening of -4.1mm. This difference did not reach statistical significance ($p=0.087$).

5.3.5 EFFECT OF GENDER

Using the One-way ANOVA test the change in MIO was compared between male (mean=0mm) and female (-2.51mm) but this did not reach statistical significance ($p=0.254$).

5.3.6 EFFECT OF PREOPERATIVE MALOCCLUSION

Table 6. *Change in MIO by Pre-operative Malocclusion*

Malocclusion	N	Mean	Std. Deviation	Min.	Max.	Significance
Class II	36	-3.7	8.0	-27	11	p<0.01 (One-way ANOVA)
Class III	20	2.3	6.3	-10	15	
Total	56	-1.6	7.9	-27	15	

Patients with a pre-operative Class II malocclusion (n=36) had a mean change in MIO of -3.6mm (SD=8.0mm). The range was a minimum of -27mm and a maximum of 11mm. Those patients with a pre-operative Class III malocclusion (n=20) had a mean change in MIO of 2.3mm (SD=6.3mm). The range was a minimum of -10mm and a maximum of 15mm. This difference between groups in MIO change was found to be statistically significant (p<0.01).

Table 7. *Change in MIO by Pre-operative Overjet ≥6mm or <6mm*

Overjet	N	Mean	Std. Deviation	Min.	Max.	Significance
≥6mm	26	-4.9	8.0	-27	11	p<0.01 (One-way ANOVA)
<6mm	30	1.3	6.8	-17	15	
Total	56	-1.6	7.9	-27	15	

Patients with a pre-operative overjet of ≥6mm showed a change of MIO of -4.9mm (SD=8.0mm, range -27-11mm). Patients with a pre-operative overjet of <6mm showed a mean change in MIO of 1.3mm (SD=6.8mm, range -17-15mm). The difference between these groups was found to be statistically significant (p<0.01).

5.3.7 EFFECT OF TYPE OF SURGERY PERFORMED

Those that underwent a LeFort 1 and BSSO (n=40) showed a mean change in MIO of -2.1mm (SD= 8.7mm). Those that underwent a LeFort 1 without BSSO (n=6) showed a mean change in MIO of 3.8mm (SD=3.1mm). Those that underwent BSSO without LeFort 1 showed a mean MIO change of -2.9mm (SD=5.5mm). The differences between these groups did not show statistical significance (p=0.203).

Patients who underwent a LeFort 1 osteotomy (n=46) showed a mean reduction of MIO of -1.5mm vs. those that did not undergo a LeFort 1 osteotomy (n=10) had a mean reduction of -1.89mm. This did not represent statistical significance (p=0.897). Patients who underwent segmentalization of the maxilla (n=24) had a mean reduction of -3.25mm, vs. -0.31mm in those with a single-piece Lefort 1. This difference did not reach statistical significance (p=0.172).

Table 8. MIO Change with Type of Surgery Performed

	N	Mean	Std. Deviation	Range	Min.	Max.	Significance
LeFort 1 + BSSO	40	-2.1	8.7	42	-27	15	p=0.203 (One-way ANOVA)
LeFort 1 only	6	3.8	3.1	8	0	8	
BSSO only	10	-2.9	5.5	16	-12	6	
LeFort 1 ± BSSO	46	-1.5	8.4	42	-27	15	p=0.897 (One-way ANOVA)
BSSO only	10	-2.9	5.5	16	-12	6	
BSSO ± LeFort 1	50	-2.2	8.1	42	-27	15	p=0.077 (One-way ANOVA)
LeFort 1 only	6	3.8	3.1	8	0	8	
FG	18	-5.5	8.7	33	-27	6	P<0.01 (One-way ANOVA)
No FG	38	0.3	6.8	42	-12	15	
Total	56	-1.6	7.9	42	-27	15	

Patients who underwent a bilateral sagittal split osteotomy (n=50) showed a mean reduction in MIO of -2.2mm, vs. those that did not have a BSSO showed a mean increase in MIO of 3.8mm. This difference did not reach statistical significance (p=0.077).

Those undergoing a functional genioplasty (n=18) showed a mean reduction in MIO of -5.5mm, vs. those that did not have a functional genioplasty (n=38) have a mean increase in MIO of 0.3mm. The change in MIO was associated with the completion of a functional genioplasty (p<0.01).

5.3.8 EFFECT OF MAGNITUDE SURGICAL MOVEMENTS

Using bivariate Pearson correlation analysis the change in MIO was found to be correlated with the magnitude for mandibular advancement ($p<0.01$). This relationship is displayed by Figure 8 showing a trend toward a decreased mouth opening with increased anterior movement of the mandible.

Using bivariate correlation analysis the magnitude of maxillary advancement ($p=0.671$), magnitude of maxillary vertical movement ($p=0.522$). or the patient's pre-operative overjet ($p=0.391$) or overbite ($p=0.985$) were all shown to not be related to change in MIO. Change in MIO was not found to be correlated to the magnitude of genioplasty movement ($p=0.214$).

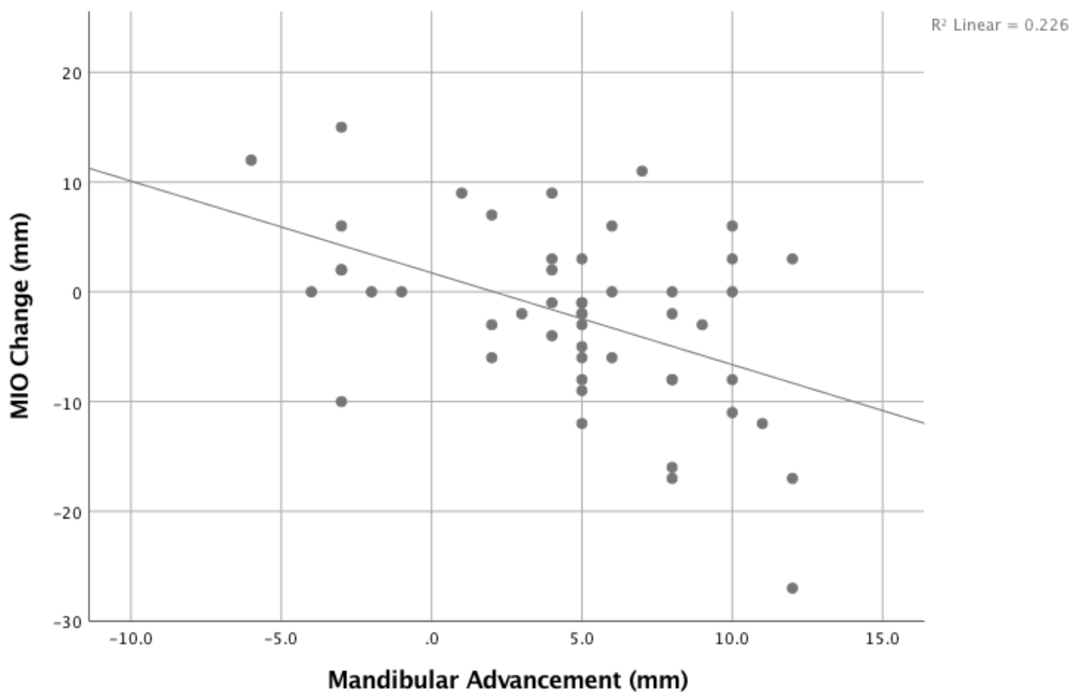

Figure 8. Simple scatterplot with line of best fit displaying MIO change (mm) against Mandibular Advancement (mm).

5.3.9 EFFECT OF THIRD MOLAR REMOVAL

Patients that had third molar removal at the time of surgery (n=27) had a reduction in opening of -0.1mm vs. no third molar removal of -2.9mm. This did not reach statistical significance ($p=0.186$).

5.3.10 EFFECT OF OBSTRUCTIVE SLEEP APNEA

Surgery for correction of OSA (n=4) showed a mean reduction in opening of -5.3mm, vs. -1.29mm. This difference was not found to the statistically significant (p=0.34).

5.4 TEMPOROMANDIBULAR JOINT PAIN

5.4.1 VISUAL ANALOG SCALE

Table 9. *Pain VAS Data Pre-operative, Post-operative, and Change*

		Mean	Median	Mode	SD	Range	Min.	Max.	Sig.
Pain VAS	Pre-op	1.14	0	0	2.1	8	0	8	p=0.254 (paired-sample T-Test)
	Post-op	0.86	0	0	1.5	8	0	8	
	Change	-0.3	0	0	1.9	10	-7	3	

The mean pre-operative pain VAS was 1.14 (SD=2.1). The range was 8 (minimum 0, maximum 8). The mean post-operative VAS was 0.86 (SD=1.5). The range was 8 (minimum 0, maximum 8). The mode for both pre and post operative VAS was 0. The mean difference (pre. vs post-operative) was -0.3. Although lower, this difference did not reach statistical significance (p=0.254) when compared with paired-sample T-Test.

Of the 56 patients, 66.1% (n=37) had no change in their pain VAS scores pre vs. post-operatively. A decrease in pain VAS was seen in 19.6% (n=11) with a range of decrease from -7 to -1. An increase in pain VAS was seen in 14.3% (n=8) patients with a range of increase from 1 to 3.

5.4.2 EFFECT OF AGE

Change in Pain VAS and Age of the patient was analyzed using Bivariate Pearson Correlation test to see if a relationship was demonstrated. There was found to be no correlation between the change in pain and age (p=0.238).

Table 10. *Pre vs. Post Operative Pain VAS Change by Age Grouping*

	N	Mean	Std. Deviation	Min.	Max.	Significance
<30 years	37	-.41	2.02	-7	3	p=0.506 (One-way ANOVA)
≥30 years	19	-.05	1.51	-4	3	
Total	56	-.29	1.86	-7	3	

Age <30 years (n=37) showed a mean reduction in pain VAS of -0.41 (SD=2.0). Age ≥30 years (n=19) showed a mean VAS reduction of -0.05 (SD=1.5). The difference between age groupings was not statistically significant (p=0.506).

5.4.3 EFFECT OF GENDER

Table 11. *Pre vs. Post Operative Pain VAS Change by Gender*

	N	Mean	Std. Deviation	Min.	Max.	Significance
Male	21	-.14	1.23	-5	2	p=0.66 (One-way ANOVA)
Female	35	-.37	2.16	-7	3	
Total	56	-.29	1.86	-7	3	

One-way ANOVA was used to compare the effect of ordinal variables on change in pain VAS. Males (n=21) showed a -0.14 (SD=1.2) mean decrease in pain VAS (range 7, minimum -5, maximum 2). Females (n=35) showed a -0.37 mean decrease in pain VAS (range 10, minimum -7, maximum 3). The differences between genders was not statistically significant (p=0.66).

5.4.4 EFFECT OF PRE-OPERATIVE MALOCCLUSION

Table 12. *Pre vs. Post-operative Pain VAS Change by Pre-operative Malocclusion*

	N	Mean	Std. Deviation	Min.	Max.	Significance
Class II	36	-.11	1.79	-6	3	p=0.350 (One-way ANOVA)
Class III	20	-.60	2.0	-7	2	
Total	56	-.29	1.86	-7	3	

Class II patients showed a mean change in Pain VAS of -0.11 (SD=1.79, maximum increase of 3, maximum decrease of -6). Class III patients showed a mean change in Pain VAS of -0.60 (SD=2.0, maximum increase 2, maximum decrease -7). This difference between groups was not found to be statistically significant (p=0.350).

When grouped into those that had pre-operative overjet of ≥6mm (n=26) or those <6mm (n=30) there was no statistically significant difference in pain VAS change observed (p=0.951).

Table 13. *Pre vs. Post Operative Pain VAS Change by Presence of Anterior Open Bite Malocclusion*

	N	Mean	Std. Deviation	Min.	Max.	Significance
AOB	13	-.46	2.07	-7	2	p=0.700 (One-way ANOVA)
No AOB	43	-.23	1.81	-6	3	
Total	56	-.29	1.86	-7	3	

Patients with a pre-existing AOB (n=13) showed a mean change in Pain VAS of -0.46 (SD=2.07, maximum increase of 2, maximum decrease of -7). Patients without a pre-existing AOB showed a mean change in Pain VAS of -0.23 (SD=1.81, maximum increase 3, maximum decrease -6). This difference between groups was not found to be statistically significant (p=0.700).

5.4.5 EFFECT OF TYPE OF SURGERY PERFORMED

Table 14. *Pre vs. Post Operative Pain VAS Change by Type of Surgery Performed*

	N	Mean	Std. Deviation	Min.	Max.	Significance
LeFort I and BSSO	40	-.25	2.01	-7	3	p=0.734 (One-way ANOVA)
LeFort I only	6	-.83	2.04	-5	0	
BSSO only	10	-.10	.99	-2	2	
Total	56	-.29	1.86	-7	3	

Those patients that had a LeFort 1 osteotomy and BSSO (n=40) showed a mean reduction in pain VAS of -0.25 (SD=2.01). Those that had a LeFort 1 osteotomy only (n=9) showed a reduction in Pain VAS of -0.83 (SD=2.04). Those that had a BSSO only (n=10) showed a reduction in Pain VAS of -0.10 (SD=0.99). The difference between these groups was not statistically significant (p=0.734).

Table 15. *Pre vs. Post Operative Pain VAS Change by Completion of LeFort 1 Osteotomy*

	N	Mean	Std. Deviation	Min.	Max.	Significance
LeFort I	46	-.34	1.98	-7	3	p=0.619 (One-way ANOVA)
No LeFort I	10	.00	1.00	-2	2	
Total	56	-.29	1.86	-7	3	

Those patients that had a LeFort 1 osteotomy (n=46) showed a mean reduction in pain VAS of -0.34 (SD=1.9), compared with those that did not have a LeFort 1 osteotomy (n=10) showed no difference in VAS (0, SD=1). The difference between these groups was not statistically significant (p=0.619).

Table 16. *Pre vs. Post Operative Pain VAS Change by Completion of BSSO*

	N	Mean	Std. Deviation	Min.	Max.	Significance
BSSO	50	-.22	1.84	-7	3	p=0.449 (One-way ANOVA)
No BSSO	6	-.83	2.04	-5	0	
Total	56	-.29	1.86	-7	3	

Patients that had a BSSO (n=50) showed a mean reduction in pain VAS of -0.22 (SD=1.8). Patients that did not undergo a BSSO (n=6) showed a mean pain VAS reduction of -0.83 (SD=2.0). The difference between these groups was not statistically significant (p=0.449).

Table 17. *Pre vs. Post Operative Pain VAS Change by Completion of Functional Genioplasty*

	N	Mean	Std. Deviation	Min.	Max.	Significance
Functional Genioplasty	18	.22	1.99	-6	3	p=0.161 (One-way ANOVA)
No Functional Genioplasty	38	-.53	1.77	-7	3	
Total	56	-.29	1.86	-7	3	

Patients undergoing a functional genioplasty (n=18) showed a mean increase in pain VAS of 0.22 (SD=1.9). Those that did not have a functional genioplasty performed (n=38) showed a reduction in pain VAS of -0.29 (SD=1.9). The difference between these groups was not significant (p=0.161).

5.4.6 EFFECT OF MAGNITUDE OF SURGICAL MOVEMENTS

Change in pain VAS was compared with the magnitude of surgical movements with a Bivariate Pearson Correlation analysis to identify if a relationship existed.

Change in pain VAS and magnitude of maxillary anterior-posterior movements did not demonstrate a correlation (p=0.505). Also, magnitude of maxillary vertical movements did not demonstrate a correlation (p=0.794). Change in pain VAS and magnitude of mandibular anterior-posterior movements did not demonstrate a correlation (p=0.132). Change in pain VAS and magnitude of symphysis anterior-posterior movements did not demonstrate a correlation (p=0.352).

5.4.7 EFFECT OF THIRD MOLAR REMOVAL

Table 18. *Pre vs. Post Operative Pain VAS Change by Extraction of Third Molars*

	N	Mean	Std. Deviation	Min.	Max.	Significance
Third Molars Extracted	27	-.37	1.88	-7	3	p=0.745 (One-way ANOVA)
No Third Molars Extracted	29	-.21	1.86	-6	3	
Total	56	-.29	1.86	-7	3	

Those patients that had third molars extracted (n=27) showed a reduction in pain VAS of -0.37 (SD=1.88). Those that did not have third molars extracted (n=29) showed a reduction in pain VAS of -0.21 (SD=1.86). The difference between these two groups was not statistically significant (p=0.745)

5.4.8 EFFECT OF OBSTRUCTIVE SLEEP APNEA

Table 19. *Pre vs. Post Operative Pain VAS Change by Diagnosis of OSA*

	N	Mean	Std. Deviation	Min.	Max.	Significance
OSA	4	.50	1.00	0	2	p=0.384 (One-way ANOVA)
No OSA	52	-.35	1.90	-7	3	
Total	56	-.29	1.86	-7	3	

Those patients that had a diagnosis of OSA (n=4) showed a mean increase in pain VAS of 0.5 (SD=1). Those that did not have a diagnosis of OSA showed a mean reduction of pain VAS of -0.35 (SD=1.8). The difference between these groups was not statistically significant (p=0.384).

5.5 TEMPOROMANDIBULAR JOINT DYSFUNCTION

The results of the pre-operative and post-operative TMJ Pain and Function Questionnaire (Appendix C) were compared using Chi-square analysis. If any of the values were less than 5, Fisher's Exact test was used.

5.5.1 SUBJECTIVE TEMPOROMANDIBULAR JOINT PAIN FREQUENCY

Of the patients that did not complete the post-operative visit, self-reporting of pre-operative joint pain frequency in the prior 30 days consisted of 61.5% (n=59) reporting no pain, 33.3% (n=32) reporting intermittent pain, and 5.2% (n=5) reporting constant pain.

Of those completing the study (n=56) the pre-operative joint pain frequency in the past 30 days showed 67.9% (n=38%) reported no pain, while 23.2% (n=13) reported intermittent pain, and 8.9% (n=5) reported constant pain. Post-operative results showed 66.1% (n=37) reporting no pain, 33.9% (n=19) reporting intermittent pain, and no reports of constant pain. These differences were found to be statistically significant (p=0.034). These differences were found to be statistically significant in both Class II patients (p=0.048) as well as Class III patients (p=0.013).

Table 20. *Pre vs. Post-operative Joint Pain Last 30 Days Crosstabulation*

		Post-operative Joint Pain Last 30 Days		Total	Significance
		No Pain	Intermittent Pain		
Pre-operative Joint Pain Last 30 Days	No Pain	30	8	38	p=0.034 (Chi-Square)
	Intermittent Pain	7	6	13	
	Constant Pain	0	5	5	
Total		37	19	56	

When further evaluated for the influence of type of surgery performed there was a statically significant difference in reported pain frequency only in those that under went LeFort 1 and BSSO ($p<0.01$), compared with LeFort 1 only ($p=0.624$) or BSSO only ($p=1.00$).

5.5.2 SUBJECTIVE TEMPOROMANDIBULAR JOINT NOISE

Of the patients that did not complete the post-operative visit, pre-operative joint noise was reported present in 53.1% (n=51).

Of those completing the study pre-operative joint noise was reported as present in 44.6% (n=25) while it was only reported present in 33.9% (n=19) post-operatively. This change was statistically significant ($p=0.01$) when compared with Chi-square test. Those patients with a pre-operative AOB (n=13) did not show a statistically significant difference compared to those without AOB by Fisher's Exact Test ($p=0.068$).

Table 21. *Pre-operative vs. Post-operative Joint Noise Crosstabulation*

		Post-operative Joint Noise			
		Yes	No	Total	Significance
Pre-operative Joint Noise	Yes	13	12	25	p=0.01 (Chi-square)
	No	6	25	31	
Total		19	37	56	

When these results were further evaluated for those patient with a pre-operative Class II malocclusion (n=36) it was found that 41.6% (n=15) of patients that did not report pre or post-operative joint noise, 27.7% (n=10) of patients with pre-operative joint noise continued to have joint noise following surgery, 16.6% (n=6) of patients that had pre-operative joint noise did not have joint noise following surgery, 13.9% (n=5) of patients that did not have pre-operative joint noise reported the development of post-operative joint noise (Figure 9). These changes were found to be statistically significant by Fisher's Exact test (p=0.026).

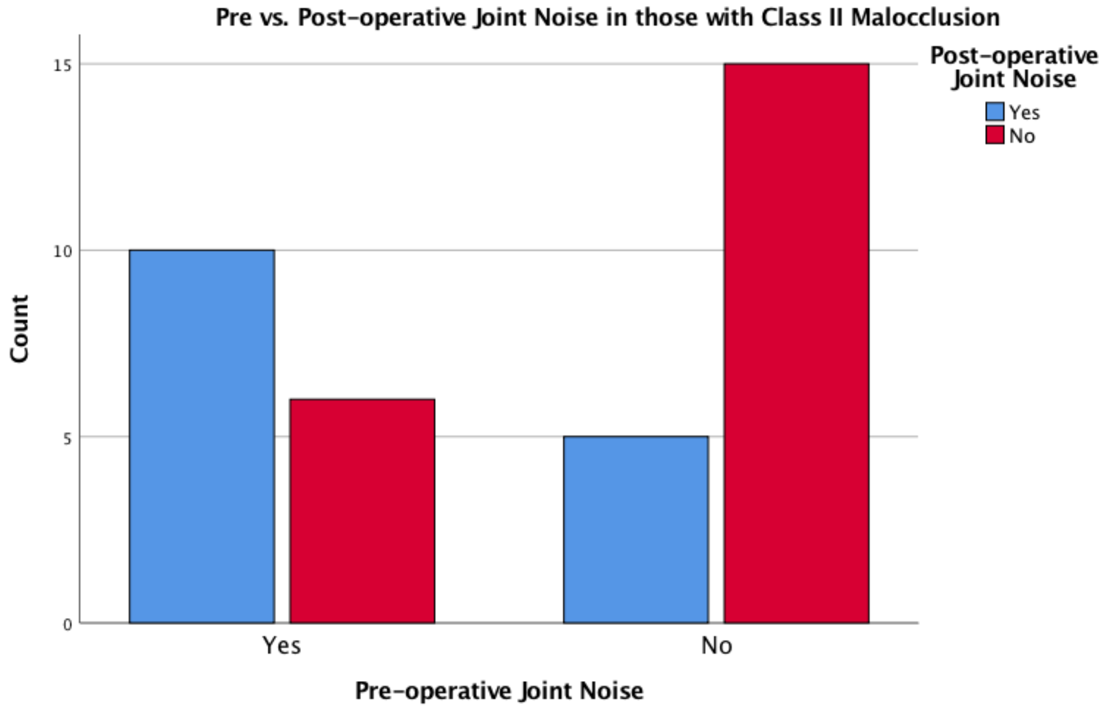

Figure 9. Bar graph displaying change in pre vs. post operative reported joint noise in Patients with Class II malocclusion.

When these results were further evaluated for those patient with a pre-operative Class III malocclusion (n=20) it was found that 50% (n=10) of patients did not report pre or post-operative joint noise, 15% (n=3) of patients with pre-operative joint noise continued to have joint noise following surgery, 30% (n=6) of patients that had pre-operative joint noise did not have joint noise following surgery, and 5% (n=1) of patients that did not have pre-operative joint noise reported the development of post-operative joint noise (Figure 10). This was found not to be statistically significant by Fisher's Exact test (p=0.217).

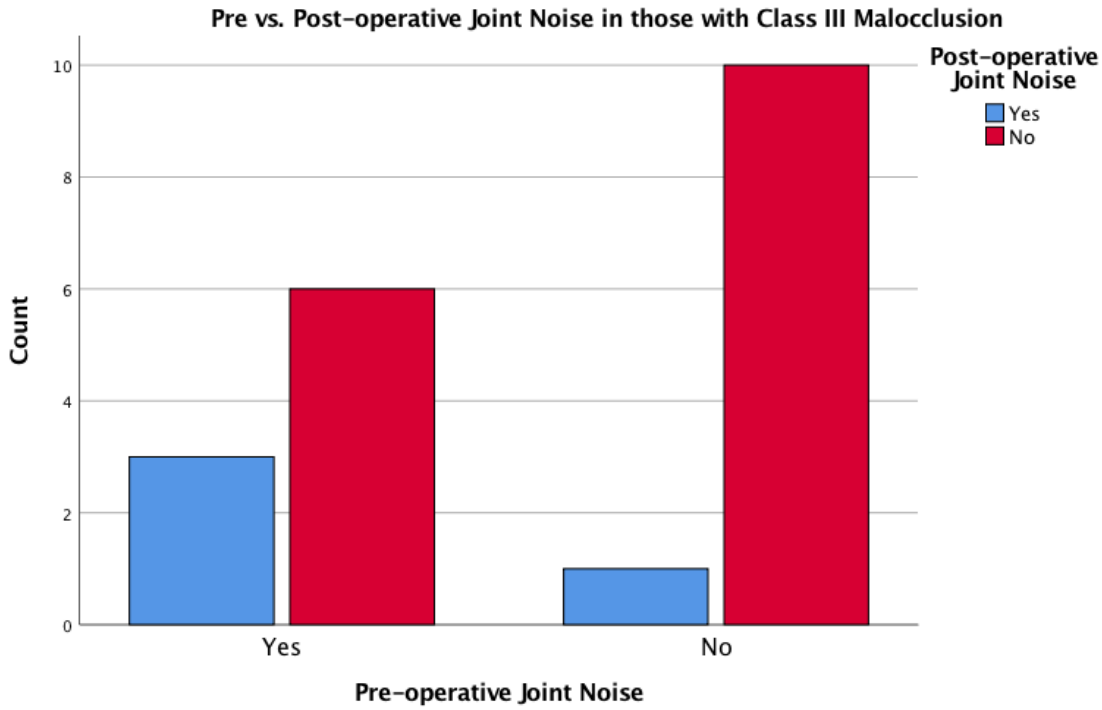

Figure 10. Bar graph displaying change in pre vs. post operative reported joint noise in Patients with Class III malocclusion.

When these results were further evaluated for the type of surgery performed it was found that in those patients that underwent both LeFort 1 and BSSO (n=40), 47.5% (n=19) did not report pre or post-operative joint noise, 22.5% (n=9) reported both pre and post-operative joint noise, 20% (n=8) reported pre-operative joint noise but no post-operative joint noise, and 10% (n=4) reported no pre-operative joint noise while reporting post-operative joint noise (Figure 11). These changes were found to be statistically significant by Fisher's Exact Test (p=0.021). Those undergoing Lefort I only (n=6) and BSSO only (n=10) did not display statistically significant differences in their pre vs. post-operative reported joint noise (p=0.400 and p=0.548 respectively).

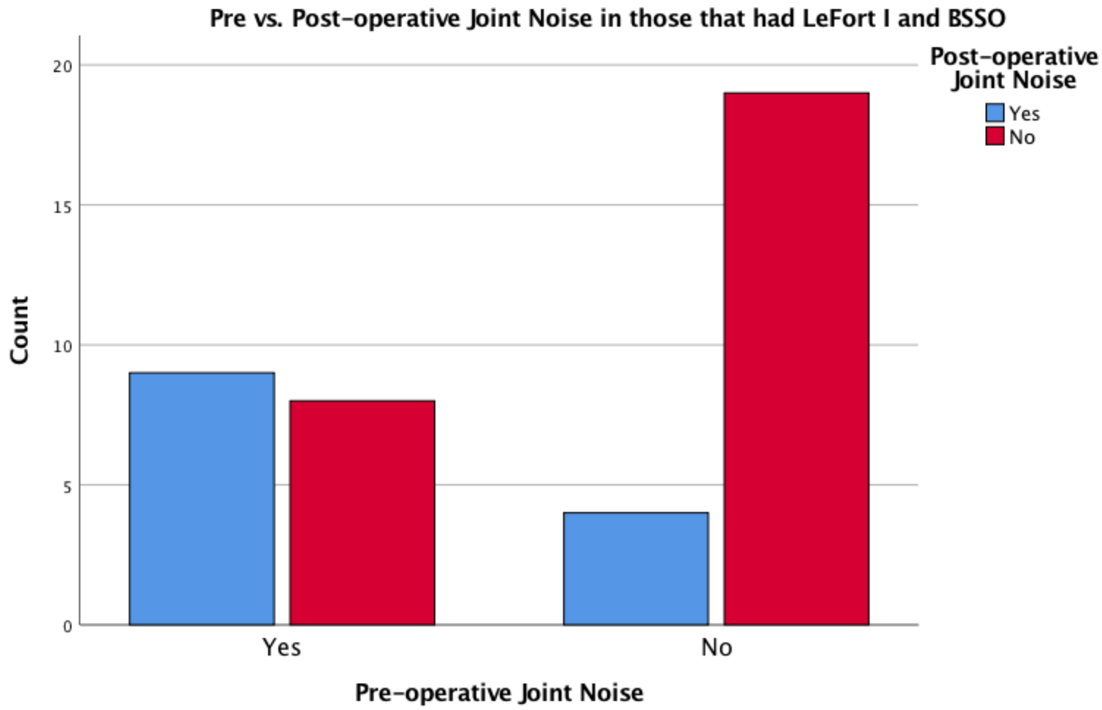

Figure 11. Bar graph displaying change in pre vs. post operative reported joint noise in those patients that underwent LeFort 1 and BSSO procedures.

5.5.3 SUBJECTIVE LIMITED OPENING

Of the patients that did not complete the post-operative visit, pre-operative limited opening was reported as being present in 15.6% (n=15).

Of those completing the study (n=56), pre-operative limited opening was reported in 3.6% (n=2), while post-operative limited opening was reported in 8.9% (n=5). Fisher's exact test did not find this to be a statistically significant difference (p=0.828).

Table 22. *Pre-operative vs. Post-operative Limited Opening Crosstabulation*

		Post-operative Limited Opening Yes	Post-operative Limited Opening No	Total	Significance
Pre-operative Limited Opening	Yes	0	2	2	p=0.828 (Fisher's Exact)
	No	5	49	54	
Total		5	51	56	

5.5.4 SUBJECTIVE OPEN LOCKING

Of the patients that did not complete the post-operative visit, pre-operative open locking was reported present in 7.3% (n=7).

Of those completing the study (n=56) pre-operative and post-operative open-locking was reported in 1.8% (n=1). This was not statistically significant (p=0.982).

Table 23. *Pre-operative vs. Post-operative Open Locking Crosstabulation*

		Post-operative Open Locking Yes	Post-operative Open Locking No	Total	Significance
Pre-operative Open Locking	Yes	0	1	1	p=0.982 (Fisher's Exact)
	No	1	54	55	
Total		1	55	56	

5.5.5 SUBJECTIVE PARAFUNCTIONAL HABIT

Of the patients that did not complete the post-operative visit, pre-operative clenching was reported to be present in 44.8% (n=43), and absent in 29.2% (n=28), and Unsure in 26.0% (n=25).

Of the 56 patients completing the study pre-operative clenching was reported as being present in 50.0% (n=28), absent in 28.6% (n=16), and unsure in 21.4% (n=12). Post-operative clenching was reported being present in 33.9% (n=19), absent in 28.6% (n=16), and unsure in 37.5% (n=21). These differences, displaying a decrease in clenching pre-op vs. post-op, were found to be statistically significant (p<0.01) by Chi-square analysis.

Table 24. *Pre-operative vs. Post-operative Clenching Crosstabulation*

		Post-operative Clenching				
		Yes	No	Unsure	Total	Significance
Pre-operative Clenching	Yes	15	5	8	28	p<0.01 (Chi-square)
	No	3	9	4	16	
	Unsure	1	2	9	12	
Total		19	16	21	56	

When further evaluated for the influence of pre-operative malocclusion Class II patients showed a statistically significant difference in clenching (p<0.01), while Class III patients did not (p=0.448).

When further evaluated for the type of surgery performed it was found that this was a statistically significant difference in those undergoing both a LeFort 1 and BSSO (p=0.021), but not in those undergoing only LeFort 1 (p=0.343) or only BSSO (p=0.392).

5.5.6 OBJECTIVE TEMPOROMANDIBULAR JOINT CLICK, CREPITUS AND TENDERNESS

Presence of a pre-operative click was diagnosed in 28.6% (n=16). Post-operative click was diagnosed in 19.6% (n=11). This reduction was statistically significant (p=0.034) when compared with the Chi-Square test.

Table 25. *Pre-operative vs. Post-operative Click Crosstabulation*

		Post-operative Click Yes	Post-operative Click No	Total	Significance
Pre-operative Click	Yes	6	10	16	p=0.034 (Chi-square)
	No	5	35	40	
Total		11	45	56	

When further evaluated for the influence of pre-operative malocclusion, both Class II (p=0.065) and Class III (p=0.117) did not show statistically significant differences between the groups with regards to change in TMJ clicking.

When the influence of type of surgery was evaluated it found that those undergoing LeFort 1 and BSSO showed a statistically significant decrease in clicking (p<0.01), while those who underwent only LeFort 1 (p=0.273) or BSSO only (p=0.747) did not.

Table 26. *Pre-operative vs. Post-operative Crepitus Crosstabulation*

		Post-operative Crepitus Yes	Post-operative Crepitus No	Total	Significance
Pre-operative Crepitus	Yes	0	2	2	p=0.795 (Fisher's Exact)
	No	6	48	54	
Total		6	50	56	

Pre-operative crepitus was identified in 3.6% (n=2). Post-operative crepitus was identified in 10.7% (n=6). This difference was not found to be statistically significant (p=0.795) when compared with Fisher's Exact test.

Table 27. *Pre-operative vs. Post-operative Tenderness to Palpation Crosstabulation*

		Post-operative Tenderness			
		Yes	No	Total	Significance
Pre-operative Tenderness	Yes	2	4	6	p=0.119 (Fisher's Exact)
	No	4	46	50	
Total		6	50	56	

Pre-operative tenderness to palpation was identified in 10.7% (n=6). It was also identified in 10.7% (n=6) post-operative. This was found to be not statistically significant by Fisher's Exact test (p=0.119).

CHAPTER 6 – DISCUSSION

6.1 EFFECT OF ORTHOGNATHIC SURGERY ON TEMPOROMANDIBULAR JOINT PAIN

66.1% of patients found no difference in pain VAS following surgery, while 19.6% had a decrease in pain VAS, and 14.3% had an increase in pain VAS. These numbers are congruent with that of the most recent meta-analysis on the subject[109]. While the mean pain VAS showed a decrease of -0.3, this reduction did not reach statistical significance (p=0.254). It is interesting to note that when patients were asked about joint pain in the previous 30 days, 8.9% (n=5) showed a reduction from constant pain and 12.5% (n=7) had intermittent pain reduced to no-pain. These differences were found to be statistically significant (p=0.034). This would indicate that the patient population that displayed pain before surgery had a reduction in pain after surgery. Again this is in keeping with the systemic review's finding that those with TMD are more likely to find a decrease in their symptoms than an increase[109].

Objective tenderness to palpation of the lateral capsule remained constant at 10.7% (n=6) pre and post-operatively. Interestingly, only 2 of the patients had tenderness both pre and post-operatively, while 4 patients that displayed tenderness pre-operatively had no tenderness post-operatively. Conversely, 4 patients that had no tenderness pre-operatively displayed tenderness post-operatively. These changes were not statistically significant (p=0.119). Age did not have an effect on the reduction in pain VAS (p=0.238). There was a greater reduction of pain in the age <30 years group (-0.41) than the age ≥30 years group (-0.05). However, this difference was not statistically significant (p=0.506). Females showed a greater reduction of pain (-0.37) than males (-0.14), but this difference was not significant (p=0.66). Pre-operative malocclusion (Class II vs. Class III) did not show a difference in reduction of pain VAS (p=0.350). Also, it appeared that a pre-operative overjet of ≥6mm vs. <6mm made no difference in pain VAS change (p=0.951). The presence of pre-existing anterior open bite did not demonstrate a difference in change of pain VAS (0.700).

Patients that underwent a LeFort 1 osteotomy had a reduction in pain VAS of (-0.34), while those that did not undergo a LeFort 1 osteotomy did not display a change in pain VAS (0). However, this difference was not statistically significant. Those patients that did not have a BSSO showed a greater reduction in pain VAS (-0.83) than those having a BSSO (-0.22), however this was not found to be statistically significant (p=0.449). Interestingly, those that had a functional genioplasty showed an increase in pain VAS (0.22) compared with a reduction in pain VAS for those not undergoing a functional genioplasty (-0.53). However, this difference was not to be statistically significant. This may be due to the fact that the majority of patients undergoing genioplasty had concurrent LeFort 1 and BSSO surgery. This increase in pain may represent the combination of all those procedures and not just the functional genioplasty. Those patients that had third molars removed showed a larger reduction of pain VAS (-0.37) than those that did not have their third molars removed (-0.21), but this difference was not statistically significant (p=0.745). Those patients that had the surgery for correction of OSA demonstrated an increase in pain VAS (0.50), while those that did not have OSA showed a decrease in pain VAS (-0.35), however this was not found to be statistically significant (p=0.384).

In general, the magnitude of surgical movements was not correlated to a change in pain VAS. Magnitude of Maxillary anterior-posterior movement (p=0.505), maxillary vertical movement (p=0.749), mandibular anterior-posterior movement (p=0.132), and genioplasty anterior-posterior movement (p=0.352) did not demonstrate a significant correlation.

A conscious decision was made not to use a standardized questionnaire or examination template such as Helkimo or RDC/TMD. The main reasoning included ease of patient participation in the hopes of increasing study enrollment. Also, it was felt that the main indicators of TMJ pain, presence of joint noise, and range of motion are the most clinically significant changes for this study population.

The criteria used for TMD diagnosis varies greatly in the literature and therefore direct comparisons for improvement of TMD following orthognathic surgery is difficult. An example of this is a systematic review of the topic that noted a prevalence of TMD among patients under going orthognathic surgery to be 7-78% in the studies they evaluated[109]. However, when looking at TMJ related pain they noticed a slight decrease in reported pain post-operatively (8-35%)[109]. These authors mentioned the notion that part of these improvements could be a placebo effect of orthognathic surgery, which has never been studied. A review on the incidence of TMD in patients undergoing orthognathic surgery vs. controls showed that pre-operatively patients with dentofacial deformities have a higher incidence of TMD than controls (RR = 1.6)[110]. However, after surgery there was no difference in incidence of TMD between the two groups. The author proposes several theories on why this may occur. Firstly, there is an improvement in masticatory ability, which may reduce TMD symptoms. Second, the change in the disc/condyle relationship may actually be beneficial in relieving pain, as has been seen following IVROs. Third, there is generally an overall improvement in a patient's self-image and confidence following correction of esthetic defects. The pre-operative state of being dis-satisfied with their appearance and low-self esteem may have contributed to the psychological development of TMD symptoms, which decrease following surgery[110].

The majority of literature seems to support the notion that TMD pain in patient's undergoing orthognathic surgery should be treated in a similar manner as that of a patient not presenting for orthognathic surgery. Ideally, this occurs before the orthognathic surgical intervention. The need to separate treatment of TMD and skeletal malocclusion is stressed by Nale, with his recommendation to treat a pre-existing TMD before undertaking orthognathic surgery as demonstrated in his treatment algorithm[111]. Wolford and colleagues have suggested a more aggressive approach to the problem in those planned for orthognathic surgery with a concomitant intra-articular TMD. They advocate for correction of both conditions in a single (open joint and orthognathic) surgical procedure[112]. The touted benefits include that it only requires one operation and general anesthetic, that it balances the occlusion, TMJs, jaws and neuromuscular structures at one time, that it decreases overall treatment time, that it eliminates unfavorable sequelae that can occur with orthognathic surgery only, and it avoids iatrogenic malocclusion that can occur when performing open TMJ surgery only[112]. However, this approach is not widely accepted in practice. Critics state during the concomitant surgery the condyle-fossa relationship becomes vulnerable to the experience of the surgeon, potentially leading to a higher chance of postoperative malocclusion[111].

While surgeons should not offer orthognathic surgery to patients solely for the purpose of reducing their TMD symptoms, they may find some re-assurance when providing orthognathic surgery to patients with TMD, as our results, as well as findings in the literature, seem to support that patients are more likely to improve with surgery than worsen[109]. Although, it must be stressed that these outcomes are not predictable.

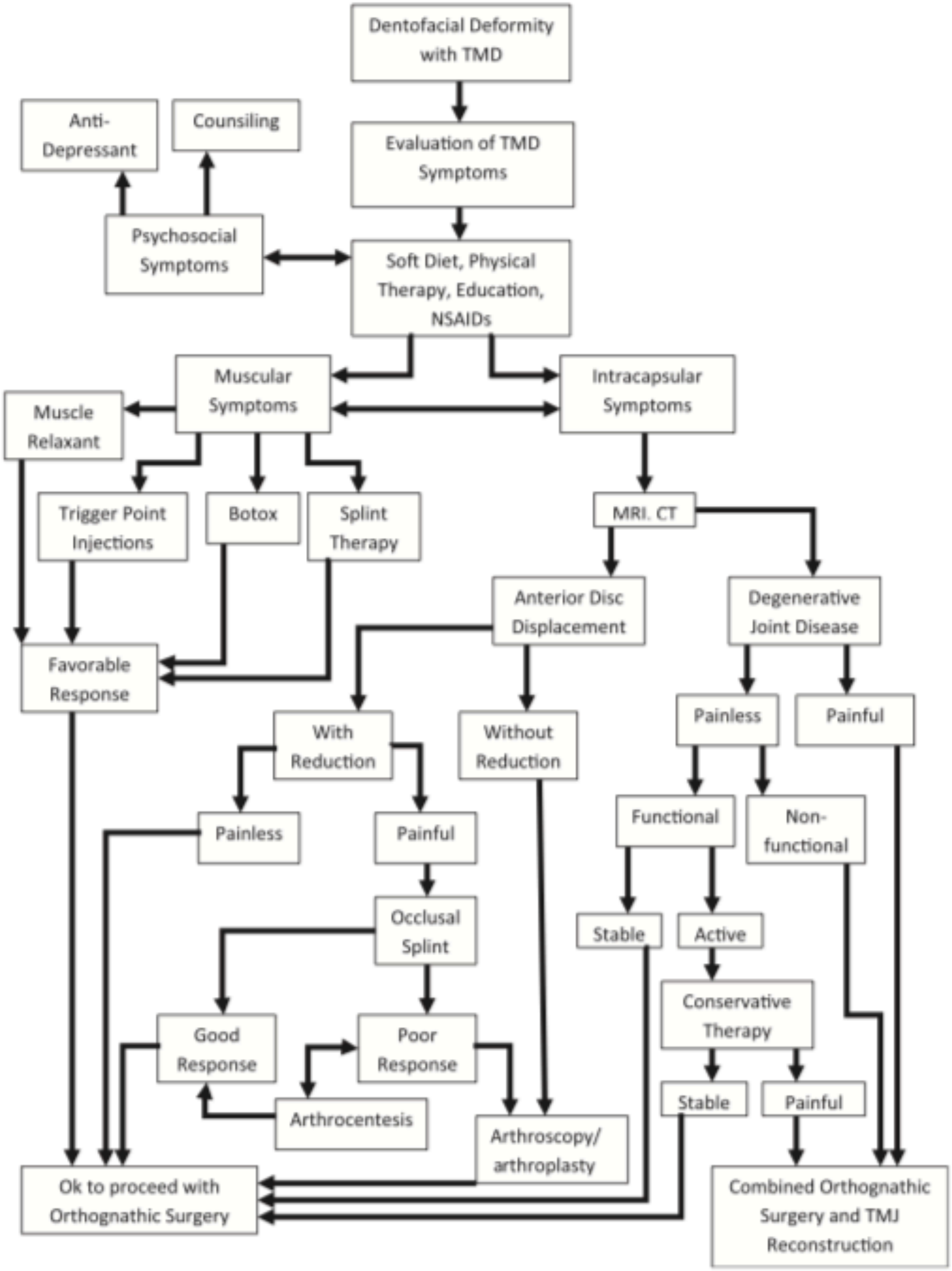

Figure 12. Flow diagram and treatment algorithm for patients with a dentofacial deformity and TMD symptoms. Copyright Nale JC: Orthognathic surgery and the temporomandibular joint patient. Oral Maxillofac Surg Clin North Am 26: 551, 2014.

6.2 EFFECT OF ORTHOGNATHIC SURGERY ON TEMPOROMANDIBULAR JOINT FUNCTION

Pre-operative mean MIO 45.6mm decreased to 44.0mm post-operatively, a mean decrease of -1.6mm. Although, this was not found to be statistically significant (p=0.144). The lowest pre-operative MIO was 39mm. Post-operatively 12.5% (n=7) of patients had a MIO of <35mm, while 23.2% (n=13) of patients had a MIO of <40mm. Similarly, a decrease in MIO of >5mm was seen in 28.6% (n=16) of patients, and a decrease of >10mm in 17.9% (n=7). This decrease in MIO was also seen by a self-reported increase in limited opening pre vs. post operatively, 3.6% to 8.9% respectively. The incidence of open-locking did not change (1.8%). A minimum opening of 35mm has been suggested as a minimum opening in which activities of living will functionally limited.

Right lateral excursion showed a mean decrease of -0.6mm. However, this difference did not reach statistical significance (p=0.096). Left lateral excursion had a mean decrease of -0.7mm, which again was not found to be statistically significant (p=0.095). This mean decrease of -1.7mm in total excursion also did not reach statistical significance (p=0.061). Protrusion showed a mean decrease of -2.5mm which was found to be statistically significant (p<0.01). However, as this is a 6 month post-operative follow-up visit, some surgeons believe there is still potential for improvement in mandibular range of motion. In a 2013 systematic review on mandibular hypomobility following orthognathic surgery, 2 of the studies examined revealed decreased mandibular range of motion at 2 years after surgery, while 5 studies did not support the notion that orthognathic surgery affect range of motion permanently[113]. A mechanism for the decrease in mandibular range of motion was not identified. The authors' conclusion was that longer-term, prospective randomized studies were needed. Storum and Bell found that MIO decreased most markedly in subjects undergoing BSSO vs. controls at time points 6-42months post-operatively. They thought the decreased opening may be due to pre-existing TMD or muscle dysfunction, and stressed the importance of a systematic regimen of muscular and occlusal rehabilitation post-surgically to normalize muscle function, condylar movement and range of mandibular motion[114]. The emphasis on passive range of motion exercises is something that is done well at our institution, with each surgeon relaying to patients in the early post-operative phase of treatment the need to regain their pre-operative opening. Also, by maintaining maxillomandibular fixation for a period of two weeks, a balance between potential decreased mandibular range of motion with the benefit of increased success of osteotomy stability may be reached.

Not surprisingly the amount of mandibular advancement was statistically significant when comparing pre-operative Class II vs. Class III patients ($p<0.01$). Class II patient's mandibles were advanced on average 7.0mm, while Class III mandibles were set back 0.7mm on average, relying mostly on maxillary advancement for the correction. This significance could be a factor contributing to the statistically significant Change in MIO seen between Class II vs. Class III patients ($p<0.01$). Class II patients showed a mean reduction in opening of 3.7mm while Class III patients actually had an increase in opening of 2.3mm. The amount of maxillary movement was not statistically different between these two groups ($p=0.591$). It has been shown that with increasing size of mandibular advancement there is increased strain on the muscles of mastication[99]. If these muscles are already stretched by their new position, it could result in the difficulty in regaining the pre-operative MIO as evidenced in the Class II patients with large advancement of the mandible. Using a grouping of Pre-operative Overjet ≥6mm or <6mm seems to be an appropriate cut-off point to define those that may experience a reduction in their MIO following surgery. The difference between these groups was found to be statistically significant ($p<0.01$). This, along with a the presence of a Class II malocclusion, could be used by surgeons a marker of those patients would be at higher risk for decreased MIO after surgery.

Age did not correlate with change in MIO ($p=0.064$). There was a difference between age groupings <30 years or ≥30 years old with regards to difference in MIO of 3.8mm, but this was not statistically significant ($p=0.087$). The difference in the mean may be accounted for by the fact that the patients receiving the surgery for correction of OSA had larger mandibular advancements, and were typically older. With regards to the effect of gender, males did not show a mean change in MIO while females showed a mean change of -2.51mm, but this was not statistically significant.

Interestingly, functional genioplasty ($p<0.01$) was the only type of surgery that demonstrated a statistically significant difference in MIO compared with those that did not undergo a functional genioplasty. LeFort 1 ($p=0.897$) and BSSO ($p=0.077$) did not show an effect. The reasoning for this may be that more patients undergoing a functional genioplasty had a combined surgery involving LeFort 1 and BSSO as well, which may compound the effects each of these surgeries have on reduced MIO. This is similar to Ueki et al. in 2008 showing no difference in MIO in 68 patients undergoing different orthognathic procedures, however functional genioplasty was not included in their study[97]. They did however find that increased length of MMF post-operatively was correlation to a reduced MIO. As our standard MMF regimen is 2 weeks this was not a variable we could explore.

The magnitude of mandibular advancement was found to be correlated to the change in MIO ($p<0.01$), with a decrease in MIO seen with larger mandibular movements. The changes in the muscles of mastication that accompany larger advancements, including stretching and re-orientation, are well documented[115]. It has also been showed the there is posterior-superior positioning of the condyle in the mandibular fossa as well as changes in the loading of the articular disc[116]. There is an established association between posteriorly positioned condyles and TMJ dysfuction[117]. The direction of loading in a posterior-superior vector compresses the highly innervated retrodiscal tissues, not only causing pain, but also forcing the disc anteriorly pre-disposing it to anterior displacement. This may contribute to the development of future internal derangement[118]. This may lead to an anterior disc-displacement without reduction which substantially limits opening, as was found in at least 1 patient post-operatively.

There was a reduction in the self-reported presence of joint noise from

44.6% to 33.9% (p=0.01). This is congruent with the objective finding of clicking which shows a reduction from 28.6% pre-operatively to 19.6% post-operatively (p=0.034). This percent reduction is similar the findings of Dujoncqouy et al. of a 15.8% decrease in joint noise[82]. This reduction in clicking was also demonstrated in 22 of 24 studies in a systematic-review by Al-Riyami in 2009. The authors noted various hypothesis on why this finding occurred. One theory was a repositioning of the condyle disc complex during the surgery. However, they also stated that a reduction in click may not relate to a recapture of the disc, but actually a progression to a worse condition of disc displacement without reduction[109]. This may explain the large decreases in MIO that we saw in our four example cases, potentially experiencing an anteriorly displaced disc. Alternatively, Onizawa et al. hypothesized that the reduction in click was due to an initial limitation in MIO, and was expected to return to pre-surgery incidence when the MIO reached the pre-operative state[81]. To definitively diagnose this displacement post-operatively and establish anterior-disc displacement without reduction as the source of limited opening would require MRI imaging, which was not completed.

Interestingly, the change in clicking was found to be statistically significant for Class II patients (p=0.026) and not Class III patients (p=0.217). The change in clicking that was noted with Class II patients was unique in that 16.6% saw a disappearance of joint noise post-operatively, while 13.9% developed new joint new after surgery. This is useful information for Class II patients as they can be informed they are as likely to develop a new click as they are to see the disappearance of clicking, although in the majority (41.6% + 27.7%) their pre-operative clicking status remains unchanged. Those patients undergoing two-jaw vs. single jaw surgery saw favorable reductions of clicking (20%) vs. developments (10%) of new clicks, but again the majority remained unchanged (47.5% + 22.5%).

The benefit of decreased clicking may not be clinically relevant. Most patients with non-painful clicking are treated with observation, and the majority will resolve. However, those patients with a painful click and the need to undergo orthognathic surgery should be given consideration to the use of an IVRO on the affected side, which has been shown to reduce the pain post-operatively more than a sagittal split osteotomy[119]. However, a significant mandibular advancement precludes the use of IVRO.

There was a reduction from 50.0% (n=28) patients that identified the presence of a parafunctional habit pre-operatively to 33.9% (n=19) patients post-operatively. This reduction was found to be statistically significant (p<0.01). The reason for this decrease may be that with the normalization of occlusion, the muscles of mastication have less of a task of finding a stable occlusal interface, thus reducing occlusal interferences which can exacerbate parafunction[120]. This reduction in parafunction, should it be permanent, would be beneficial to the TM joints as it is well documented that parafunction can lead to osteoarthritic changes.

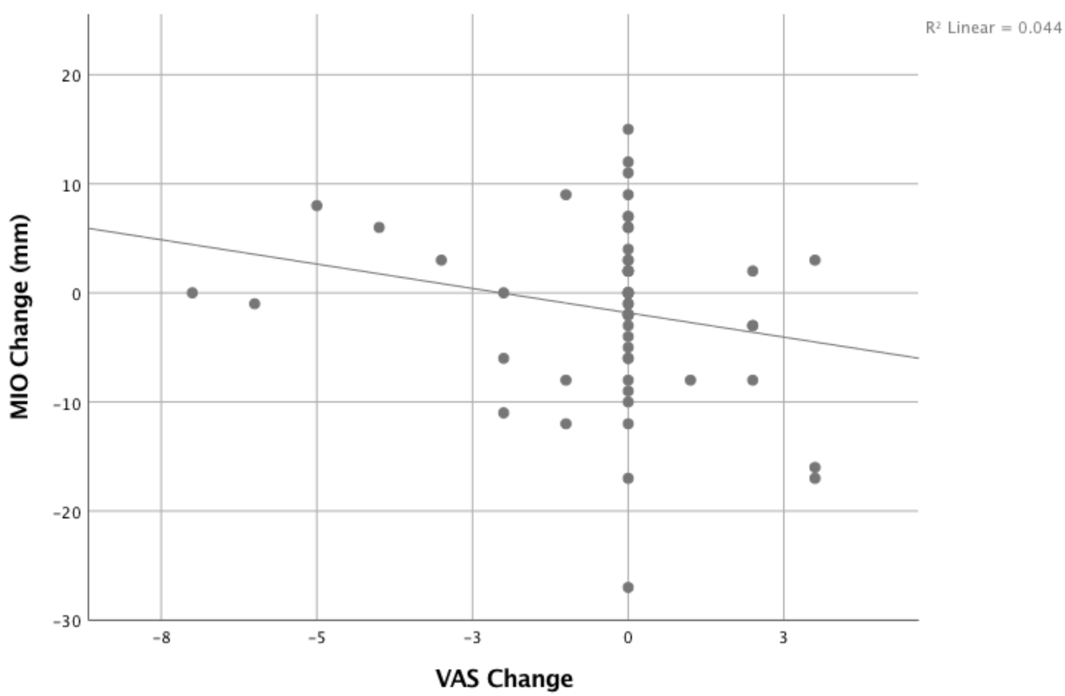

Figure 13. Simple scatter plot with line of best fit displaying MIO change (mm) against VAS change. Small R^2 value indicates no significant correlation.

Although change in MIO and change in pain VAS did not show significant correlation (p=0.122) an analysis of Figure 9 allows a look at case-by-case outcomes. Those patients that had an increase in pain VAS with a decrease in MIO would be classified as having a poor TMJ outcome. The most extreme examples of these cases are as follows: 1) 28year old female with an increase in pain VAS of 3, and a reduction in MIO of -17mm following correction of Class II malocclusion with a segmental LeFort 1 osteotomy, BSSO advancement of 8mm and genioplasty advancement of 6mm, 2) 25 year old female with an increase pain VAS of 3, and a reduction in MIO of -16mm following correction of Class II malocclusion with a LeFort 1 osteotomy, BSSO advancement of 8mm and genioplasty advancement of 8mm, 3) 55 year old male with no change in pain VAS but a decrease in MIO of 27mm following maxillomandibular advancement for correction of obstructive sleep apnea with a Lefort 1 advancement of 8mm, BSSO advancement of 12mm and genioplasty advancement of 5mm, and 4) 34 female in which her pre and post-operative pain scores were 8 and her reduction in MIO was -17mm following maxillomandibular advancement for correction of OSA with a LeFort 1 advancement of 5mm, BSSO advancement of 12mm, and genioplasty advancement of 4mm. This last patient was found on follow-up to have a non-reducing left anteriorly displaced disc, and has subsequently offered TMJ arthroscopy. Interestingly, at the time of orthognathic surgery this patient was noted intra-operatively by the surgeon to have extremely lax TM joints, and was prone to anterior dislocation of the condylar head. Overall, common factors in these poor outcomes are that they were all combinations of LeFort 1, BSSO, and functional genioplasty surgeries with large (8-12mm) advancements of the mandible.

6.3 METHODOLOGICAL ASPECTS

Patient participation was found to be acceptable with approximately half (52.4%) of those asked to participate agreeing to enroll. However, the study participation to completion was lower (36.8%) than expected. This may have been due to some patients requiring an extra follow-up visit at 6 months solely for the purpose of completing the study if their "brace's off" appointment did not fall at the 6 month time frame. When comparing those that completed the 6 month visit to those that dropped out of the study, the mean age was younger in the study drop outs (25.4 vs. 28.1 years). This younger population may have been less inclined to participate or perhaps had more difficulty in arranging transportation for visits, hence the higher drop out rates. Those that completed the study also had a lower pre-op Pain VAS score than those that dropped out (1.14 vs. 1.25). This is a small difference and it would be unlikely to say that this difference accounted for the decrease in study completion. Males comprised 33.3% of those that did not complete the study, which is similar to the composition of those that completed the study at 37.5%, therefore gender differences shouldn't account for the lack of study completion.

Limitations of the study include relatively short length of follow up. It has been demonstrated that some TMD conditions do not develop until later in the post-operative period. Wolford studied a group of 25 patients with displaced TMJ discs whom underwent mandibular advancement procedures and found 84% had joint related pain at a mean of 2.2 years after surgery (vs. 24% pre-operatively)[112]. The new onset of TMJ pain averaged 14 months post surgery. Also, the 6 month follow-up period may have accounted for the reduction in MIO seen post-operatively, as it has been demonstrated in other studies that MIO continues to increase up to 2 years post-operatively. The study did not include a control arm, which could have been used to track the natural course of TMJ range of motion and pain over time. Future studies may make use of a standardized exam and questionnaire for ease in sharing data between studies, however an ideal exam and questionnaire has not yet been developed. It would consist of a tool for diagnosis that is valid, reproducible, and simple to carry out[109].

Future research considerations may include the validation of a shortened format questionnaire and examination. Also, applying the examination and questionnaire before the start of orthodontic treatment would be another useful time point to investigate. Longer-term follow-up, and volumetric analysis of the condylar head and analysis of condylar position from CBCT imaging may also be considered.

CHAPTER 7 – CONCLUSION

The results regarding temporomandibular joint outcomes following orthognathic surgery are congruent with those found in the currently accepted literature on the topic. Our findings corroborate that surgeons and patients should be aware TMJ related pain is likely to remain unchanged following surgery. In the small percentage of patients were pain does change, there is a higher chance it will improve than get worse. The frequency of reported TMJ pain is likely to decrease. Most patients experience a small decrease in mandibular mobility, especially protrusion, but this is likely inconsequential functionally. Patients may also fail to recognize this reduction in opening. A larger magnitude of mandibular advancement, pre-operative overjet $\geq$6mm, Class II malocclusion and completion of a functional genioplasty are risk factors for a decrease in mouth opening. A very small percentage of patients may develop persistent limitations in opening severe enough to require further intervention. Patients with TMJ clicking and/or parafunctional clenching will likely see a decrease in these symptoms after surgery, especially when undergoing two-jaw surgery. Class II patients have an equal chance of clicks disappearing or developing after surgery. TMJ crepitus is likely to remain unchanged.

BIBLIOGRAPHY

1. Proffit W, White R, Sarver D eds: Comtemporary Treatment of Dentofacial Deformity. First Edit. St Louis, Mo: CV Mosby, 2003.
2. Angle E: Classification of malocclusion. Dent Cosm 41: 248, 1899.
3. Soh CL, Narayanan V: Quality of life assessment in patients with dentofacial deformity undergoing orthognathic surgery—A systematic review. Int J Oral Maxillofac Surg 42: 974, 2013.
4. Bock JJ, Odemar F, Fuhrmann RAW: Assessment of Quality of Life in Patients Undergoing Orthognathic Surgery. J Orofac Orthop / Fortschritte der Kieferorthopädie 70: 407, 2009.
5. Bourdiol P, Soulier-Peigue D, Lachaze P, Nicolas E, Woda A, Hennequin M: Only severe malocclusion correlates with mastication deficiency. Arch Oral Biol 75: 14, 2017.
6. Lee S, McGrath C, Samman N: Quality of life in patients with dentofacial deformity: a comparison of measurement approaches. Int J Oral Maxillofac Surg 36: 488, 2007.
7. Allen PF: Assessment of oral health related quality of life. Health Qual Life Outcomes 1: 40, 2003.
8. B.A. W: Use of oral health related quality of life measures in managed dental care organisations. Community Dent Health 15: 27, 1998.
9. Choi WS, Lee S, McGrath C, Samman N: Change in quality of life after combined orthodontic-surgical treatment of dentofacial deformities. Oral Surgery, Oral Med Oral Pathol Oral Radiol Endodontology 109: 46, 2010.
10. Silva I, Cardemil C, Kashani H, Bazargani F, Tarnow P, Rasmusson L, Suska F: Quality of life in patients undergoing orthognathic surgery – A two-centered Swedish study. J Cranio-Maxillofacial Surg 44: 973, 2016.
11. Cunningham SJ, Garratt AM, Hunt NP: Development of a condition-specific quality of life measure for patients with dentofacial deformity: II. Validity and responsiveness testing. Community Dent Oral Epidemiol 30: 81, 2002.
12. Lee S, McGrath C, Samman N: Impact of Orthognathic Surgery on Quality of Life. J Oral Maxillofac Surg 66: 1194, 2008.
13. Lucia A, Barbosa B, Marcantonio E, Eduardo C, Barbosa DM, Real MF, Aparecida M, Gabrielli C: Psychological Scheduled Evaluation For Orthognathic of Patients Surgery.: 1, 1993.

14. Fort R Le: Fractures de la machoire superieure 1901; 4:360. Rev Chir: 360, 1901.

15. Wassmund M: Frakturen und Lurationen des Gesichtsschadels. Leipzig Meusser Verlag, 1927.

16. Obwegeser H: Eingriffe an Oberkiefer zur Korrektur des pro- genen. Zahnbheilk: 356, 1965.

17. Precious DS, Morrison A, Ricard D: Pterygomaxillary separation without the use of an osteotome. J Oral Maxillofac Surg 49: 98, 1991.

18. Precious D, Goodday R, Bourget L: Pterygoid plate fracture in Le Fort I osteotomy with and without pterygoid chisel and a computed tomography scan evaluation of 58 patients. J Oral Maxillofac Surg: 151, 1993.

19. Breeze J, Linares CV, Stockton P: Is an osteotome necessary for pterygomaxillary dysjunction or dysjunction through the tuberosity during le Fort i osteotomy? A systematic review. Br J Oral Maxillofac Surg 54: 248, 2016.

20. Hullihen S: Case of elongation of the under jaw and distortion of the face and neck, caused by a burn, successfully treated. Am J Dent Sci 9, 1849.

21. Blair V: Operations on the jaw bone and face. Surg Gynecol Obs 4, 1907.

22. Dingman R: Osteotomy for the correction of mandibular malrelation of developmental origin. J Oral Surg 2, 1944.

23. Monson LA: Bilateral sagittal split osteotomy. Semin Plast Surg 27: 145, 2013.

24. Obwegeser H, Trauner R: Zur operationstechnik bei der progenie und anderen unterkieferanomalien. Dtsch Zahn Mund Kieferheilkd 23, 1955.

25. DalPont G: Petromolar osteotomy for the correction of prognathism. J Oral Surg Anesth Hosp Dent Serv Jan: 42, 1961.

26. Hunsuck E: A modified intraoral sagittal splitting technique for correction of mandibular prognathism. J Oral Surg 26, 1968.

27. Bell WH, Schendel S a: Biologic basis for modification of the sagittal ramus split operation. J Oral Surg (Chic) 35: 362, 1977.

28. Epker B, Wolford L, Fish L: Mandibular deficiency syndrome. II. Surgical considerations for mandibular advancement. Oral Surg Oral Med Oral Pathol 45: 349, 1978.

29. Caldwell JB, Letterman GS: Vertical osteotomy in the mandibular raml for correction of prognathism. J Oral Surg (Chic) 12: 185, 1954.

30. McKenna SJ, King EE: Intraoral Vertical Ramus Osteotomy Procedure and Technique. Atlas Oral Maxillofac Surg Clin North Am 24: 37, 2016.

31. Park KR, Kim SY, Park HS, Jung YS: Surgery-first approach on patients with temporomandibular joint disease by intraoral vertical ramus osteotomy. Oral Surg Oral Med Oral Pathol Oral Radiol 116: e429, 2013.

32. Trauner R, Obwegeser H: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. Oral Surg Oral Med Oral Pathol: 677, 1957.

33. Strauss RA, Abubaker AO: Genioplasty: A case for advancement osteotomy. J Oral Maxillofac Surg 58: 783, 2000.

34. Delaire J, Schendel SA, Tulasne JF: An architectural and structural craniofacial analysis: A new lateral cephalometric analysis. Oral Surgery, Oral Med Oral Pathol 52: 226, 1981.

35. Precious D, Delaire J: Correction of anterior mandibular vertical excess : the functional genioplasty. Oral Surg Oral Med Oral Pathol 59: 229, 1985.

36. Kademani D, Tiwana P eds: Atlas of Oral and Maxillofacial Surgery. First Edit. St. Louis, Missouri: Saunders, an imprint of Elsevier, Inc, 2016.

37. Ugolini A, Mapelli A, Seg?? M, Zago M, Codari M, Sforza C: Three-dimensional mandibular motion in skeletal Class III patients. Cranio - J Craniomandib Pract: 1, 2017.

38. Wadhwa S, Kapila S: TMJ disorders: future innovations in diagnostics and therapeutics. J Dent Educ 72: 930, 2008.

39. Murphy MK, MacBarb RF, Wong ME, Athanasiou KA: Temporomandibular Disorders: A Review of Etiology, Clinical Management, and Tissue Engineering Strategies. Int J Oral Maxillofac Implants 28: e393, 2013.

40. Bouloux GF: Temporomandibular Joint Pain and Synovial Fluid Analysis: A Review of the Literature. J Oral Maxillofac Surg 67: 2497, 2009.

41. Choi KY, Yang JD, Chung HY, Cho BC: Current concepts in the mandibular condyle fracture management part I: Overview of condylar fracture. Arch Plast Surg 39: 291, 2012.

42. Langendoen J, Müller J, Jull G a.: Retrodiscal tissue of the temporomandibular joint: clinical anatomy and its role in diagnosis and treatment of arthropathies. Man Ther 2: 191, 1997.

43. Candirli C, Demirkol M, Yilmaz O, Memis S: Retrospective evaluation of three different joint surgeries for internal derangements of the temporomandibular joint. J Cranio-Maxillofacial Surg 45: 775, 2017.

44. Scrivani SJ, Keith D a, Kaban LB, Rossi SS De, Greenberg MS, Rcsd FDS, Liu F, Steinkeler A: Temporomandibular Disorders Evaluation and Management. Med Clin N Am 98: 1353, 2014.

45. Ballenberger N, Piekartz H von, Danzeisen M, Hall T: Patterns of cervical and masticatory impairment in subgroups of people with temporomandibular disorders???an explorative approach based on factor analysis. Cranio - J Craniomandib Pract: 1, 2017.

46. RASMUSSEN OC: Description of population and progress of symptoms in a longitudinal study of temporomandibular arthropathy. Eur J Oral Sci 89: 196, 1981.

47. List T, Jensen RH: Temporomandibular disorders: Old ideas and new concepts. Cephalalgia 37: 692, 2017.

48. Milam SB: Pathogenesis of degenerative temporomandibular joint arthritides. Odontology 93: 7, 2005.

49. Attia HS, Mosleh MI, Jan AM, Shawky MM, Jadu FM: Age, gender and parafunctional habits as prognostic factors for temporomandibular joint arthrocentesis. Cranio - J Craniomandib Pract: 1, 2017.

50. Naqvi T, Duong TT, Hashem G, Shiga M, Zhang Q, Kapila S: Relaxin's induction of metalloproteinases is associated with the loss of collagen and glycosaminoglycans in synovial joint fibrocartilaginous explants. Arthritis Res Ther 7: R1, 2005.

51. Ushiyama T, Mori K, Inoue K, Huang J, Nishioka J, Hukuda S: Association of oestrogen receptor gene polymorphisms with age at onset of rheumatoid arthritis. Ann Rheum Dis 58: 7, 1999.

52. Milam B, Schmitz P: Molecular Biology of Temporomandibular Joint Disorders: J Dent: 1448, 1995.

53. Yıldızer Keriş E, Yaman SD, Demirağ MD, Haznedaroğlu Ş: Temporomandibular joint findings in patients with rheumatoid arthritis, ankylosing spondylitis, and primary Sjögren's syndrome. J Investig Clin Dent 8: e12255, 2017.

54. Caruso S, Storti E, Nota A, Ehsani S, Gatto R: Temporomandibular joint anatomy assessed by CBCT images. Biomed Res Int 2017, 2017.

55. Krohn S, Gersdorff N, Wassmann T, Merboldt KD, Joseph AA, Buergers R, Frahm J: Real-time MRI of the temporomandibular joint at 15 frames per second—A feasibility study. Eur J Radiol 85: 2225, 2016.

56. Helkimo M: Studies on function and dysfunction of the masticatory system. II. Index for anamnestic and clinical dysfunction and occlusal state. Swed Dent J 67: 101, 1974.

57. Helkimo M: Studies on function and dysfunction of the masticatory system. I. An epidemiological investigation of symptoms of dysfunction in Lapps in the north of Finland. Proc Finn Dent Soc 70: 37, 1974.

58. Helkimo M: Studies on function and dysfunction of the masticatory system. 3. Analyses of anamnestic and clinical recordings of dysfunction with the aid of indices. Sven Tandlak Tidskr 67: 165, 1974.

59. Fricton JR, Schiffman EL: Reliability of a Craniomandibular Index. J Dent Res 65: 1359, 1986.

60. Kopp S, Wenneberg B: Intra- and interobserver variability in the assessment of signs of disorder in the stomatognathic system. Swed Dent J 7: 239, 1983.

61. Dworkin S, Leresche L: Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. J Craniomandib Disord Facial Oral Pain: 301, 1992.

62. Schiffman E, Ohrbach R, Truelove E, Look J, Anderson G: Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications: Recommendations of the International RDC/TMD Consortium Network* and Orofacial Pain Special Interest Group. J Oral Facial Pain Headache 28: 6, 2014.

63. Veldhuis EC, Veldhuis AH, Bramer WM, Wolvius EB, Koudstaal MJ: The effect of orthognathic surgery on the temporomandibular joint and oral function : a systematic review. Int J Oral Maxillofac Surg 46: 554, 2017.

64. Jung H-D, Kim SY, Park H-S, Jung Y-S: Orthognathic surgery and temporomandibular joint symptoms. Maxillofac Plast Reconstr Surg 37: 14, 2015.

65. Grootel RJ van, Buchner R, Wismeijer D, Glas HW van der: Towards an optimal therapy strategy for myogenous TMD, physiotherapy compared with occlusal splint therapy in an RCT with therapy-and-patient-specific treatment durations. BMC Musculoskelet Disord 18: 76, 2017.

66. Goiato MC, Silva EVF da, Medeiros RA de, Turcio KHL, Santos DM Dos: Are intra-articular injections of hyaluronic acid effective for the treatment of temporomandibular disorders? A systematic review. Int J Oral Maxillofac Surg: 1531, 2016.

67. Miloro M, Ghali G, Larson P, Waite P eds: Peterson's Principles of Oral and Maxillofacial Surgery. Third Edit. Shelton, CT: People's Medical Publishing House-USA, 2011.

68. Khan M, Nishi SE, Hassan SN, Islam MA, Gan SH: Trigeminal neuralgia, glossopharyngeal neuralgia, and myofascial pain dysfunction syndrome: An update. Pain Res Manag 2017, 2017.

69. Goldenberg DL, Burckhardt C, Crofford L: Management of Fibromyalgia Syndrome. JAMA 292: 2388, 2014.

70. Svensson P, Graven-Nielsen T: Craniofacial Muscle Pain : Review of Mechanisms and Clinical Manifestations. J Orofac Pain 15: 117, 2001.

71. Gerwin R: A study of 96 subjects examined both for fibromyalgia and myofascial pain. J Musculoskelet Pain 3: 121, 1995.

72. Dahan H, Shir Y, Velly A, Allison P: Specific and number of comorbidities are associated with increased levels of temporomandibular pain intensity and duration. J Headache Pain 16: 1, 2015.

73. Gazi MCB, Issy AM, Ávila IP, Sakata RK: Comparison of Acupuncture to Injection for Myofascial Trigger Point Pain. Pain Pract 11: 132, 2011.

74. Ho KY, Tan KH: Botulinum toxin A for myofascial trigger point injection: A qualitative systematic review. Eur J Pain 11: 519, 2007.

75. Al-Moraissi EA, Wolford LM, Perez D, Laskin DM, Ellis E: Does Orthognathic Surgery Cause or Cure Temporomandibular Disorders? A Systematic Review and Meta-Analysis. J Oral Maxillofac Surg 75: 1835, 2017.

76. Ireland AJ, Cunningham SJ, Petrie A, Cobourne MT, Acharya P, Sandy JR, Hunt NP: An Index of Orthognathic Functional Treatment Need (IOFTN). J Orthod 41: 77, 2014.

77. Brook PH, Shaw WC: The development of an index of orthodontic treatment priority. Eur J Orthod 11: 309, 1989.

78. Shah R, Breeze J, Chand M, Stockton P: The index of orthognathic functional treatment need accurately prioritises those patients already selected for orthognathic surgery within the NHS. Br J Oral Maxillofac Surg 54: 511, 2016.

79. Borzabadi-Farahani A, Eslamipour F, Shahmoradi M: Functional needs of subjects with dentofacial deformities: A study using the index of orthognathic functional treatment need (IOFTN). J Plast Reconstr Aesthetic Surg 69: 796, 2016.

80. Jędrzejewski M, Smekta T, Olszewski R: Preoperative , intraoperative , and postoperative complications in orthognathic surgery : a systematic review. Clin Oral Invest 19: 969, 2015.

81. Onizawa K, Schmelzeisen R, Vogt S: Alteration of temporomandibular joint symptoms after orthognathic surgery: comparison with healthy volunteers. J Oral Maxillofac Surg 53: 117, 1995.

82. Dujoncquoy J-P, Ferri J, Raoul G, Kleinheinz J: Temporomandibular joint dysfunction and orthognathic surgery: a retrospective study. Head Face Med 6: 27, 2010.

83. Storum KA, Bell WH: Hypomobility after maxillary and mandibular osteotomies. Oral Surgery, Oral Med Oral Pathol 57: 7, 1984.

84. Ren Y-F, Isberg A, Westesson P-L: Condyle position in the temporomandibular joint. Oral Surgery, Oral Med Oral Pathol Oral Radiol Endodontology 80: 101, 1995.

85. Nemeth DZ, Rodrigues-Garcia RC, Sakai S, Hatch JP, Sickels JE Van, Bays RA, Clark GM, Rugh JD: Bilateral sagittal split osteotomy and temporomandibular disorders: rigid fixation versus wire fixation. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 89: 29, 2000.

86. Costa F, Robiony M, Toro C, Sembronio S, Polini F, Politi M: Condylar positioning devices for orthognathic surgery : a literature review. Surg Oral Med Oral Pathol Oral Radiol Endod 106: 179, 2008.

87. Landes CA: Proximal Segment Positioning in Bilateral Sagittal Split Osteotomy: Intraoperative Dynamic Positioning and Monitoring by Sonography. J Oral Maxillofac Surg 62: 22, 2004.

88. Politi M, Toro C, Costa F, Robiony M: Intraoperative Awakening of the Patient During Orthognathic Surgery : A Method to Prevent the Condylar Sag. J Oral Maxillofac Surg 65109-114, 65: 109, 2007.

89. Chen S, Lei J, Wang X, Fu K: Short- and Long-Term Changes of Condylar Position After Bilateral Sagittal Split Ramus Osteotomy for Mandibular Advancement in Combination With Le Fort I Osteotomy Evaluated by Cone-Beam Computed Tomography. J Oral Maxillofac Surg 71: 1956, 2013.

90. Peacock ZS, Lee CCY, Klein KP, Kaban LB: Orthognathic surgery in patients over 40 years of age: Indications and special considerations. J Oral Maxillofac Surg 72: 1995, 2014.

91. Manfredini D, Guarda-Nardini L, Winocur E, Piccotti F, Ahlberg J, Lobbezoo F: Research diagnostic criteria for temporomandibular disorders: A systematic review of axis I epidemiologic findings. Oral Surgery, Oral Med Oral Pathol Oral Radiol Endodontology 112: 453, 2011.

92. Catherine Z, Breton P, Bouletreau P: Condylar resorption after orthognathic surgery: A systematic review. Rev Stomatol Chir Maxillofac Chir Orale 117: 3, 2016.

93. Hwang SJ, Haers PE, Seifert B, Sailer HF: Non-surgical risk factors for condylar resorption after orthognathic surgery. J Cranio-Maxillofacial Surg 32: 103, 2004.

94. Yamada K, Hanada K, Fukui T, Satou Y, Ochi K, Hayashi T, Ito J: Condylar bony change and self-reported parafunctional habits in prospective orthognathic surgery patients with temporomandibular disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 92: 265, 2001.

95. Jiménez-Silva A, Peña-Durán C, Tobar-Reyes J, Frugone-Zambra R: Sleep and awake bruxism in adults and its relationship with temporomandibular disorders: A systematic review from 2003 to 2014. Acta Odontol Scand 75: 36, 2017.

96. Israel HA, Diamond B, Saed-Nejad F, Ratcliffe A: The relationship between parafunctional masticatory activity and arthroscopically diagnosed temporomandibular joint pathology. J Oral Maxillofac Surg 57: 1034, 1999.

97. Ueki K, Marukawa K, Hashiba Y, Nakagawa K, Degerliyurt K, Yamamoto E: Assessment of the Relationship Between the Recovery of Maximum Mandibular Opening and the Maxillomandibular Fixation Period After Orthognathic Surgery. J Oral Maxillofac Surg 66: 486, 2008.

98. Athanasiou AE, Elefteriadis JN, Dre E: Short-term functional alterations in the stomatognathic system after orthodontic-surgical management of skeletal vertical excess problems. Int J Adult Orthodon Orthognath Surg 11: 339, 1996.

99. Bermell-Baviera A, Bellot-Arcís C, Montiel-Company JM, Almerich-Silla JM: Effects of mandibular advancement surgery on the temporomandibular joint and muscular and articular adaptive changes—a systematic review. Int J Oral Maxillofac Surg 45: 545, 2016.

100. Wolford LM, Reiche-Fischel O, Mehra P: Changes in temporomandibular joint dysfunction after orthognathic surgery. J Oral Maxillofac Surg 61: 655, 2003.

101. Frey DR, Hatch JP, Sickels JE Van, Dolce C, Rugh JD: Effects of surgical mandibular advancement and rotation on signs and symptoms of temporomandibular disorder: A 2-year follow-up study. Am J Orthod Dentofac Orthop April: 1, 2008.

102. Valladares-Neto J, Cevidanes LH, Rocha WC, Almeida G de A, Paiva JB de, Rino-Neto J: TMJ response to mandibular advancement surgery: an overview of risk factors. J Appl Oral Sci 22: 2, 2014.

103. Doucet J-C, Morrison AD, Davis BR, Gregoire CE, Goodday R, Precious DS: The Presence of Mandibular Third Molars During Sagittal Split Osteotomies Does Not Increase the Risk of Complications. J Oral Maxillofac Surg 70: 1935, 2012.

104. Doucet JC, Morrison AD, Davis BR, Robertson CG, Goodday R, Precious DS: Concomitant removal of mandibular third molars during sagittal split osteotomy minimizes neurosensory dysfunction. J Oral Maxillofac Surg 70: 2153, 2012.

105. Camargo IB, Sickels JE Van, Curtis WJ: Simultaneous Removal of Third Molars During a Sagittal Split Does Not Increase the Incidence of Bad Splits in Patients Aged 30 Years or Older. J Oral Maxillofac Surg 73: 1350, 2015.

106. Huang GJ, Rue TC: Third-molar extraction as a risk factor for temporomandibular disorder. J Am Dent Assoc 137: 1547, 2006.

107. Morton L, Downie I: Surgical and social implications of extraction of mandibular third molars as a separate procedure before bilateral sagittal split osteotomy. Br J Oral Maxillofac Surg 55: 937, 2017.

108. Erickson K, Bell W, Goldsmith D: Analytical Model Surgery. In: Bell W, ed. Modern Practice Orthognathic and Reconstructive Surgery. 1st ed. Philadelphia: Saunders, 1992:155–205.

109. Al-Riyami S, Cunningham SJ, Moles DR: Orthognathic treatment and temporomandibular disorders: A systematic review. Part 2. Signs and symptoms and meta-analyses. Am J Orthod Dentofac Orthop 136: 626.e1, 2009.

110. Al-Moraissi EA, Perez D, Ellis E: Do patients with malocclusion have a higher prevalence of temporomandibular disorders than controls both before and after orthognathic surgery? A systematic review and meta-analysis. J Cranio-Maxillofacial Surg 45: 1716, 2017.

111. Nale JC: Orthognathic surgery and the temporomandibular joint patient. Oral Maxillofac Surg Clin North Am 26: 551, 2014.

112. Wolford LM: Concomitant temporomandibular joint and orthognathic surgery. J Oral Maxillofac Surg 61: 1198, 2003.

113. Al-Belasy FA, Tozoglu S, Dolwick MF: Mandibular hypomobility after orthognathic surgery: A review article. J Oral Maxillofac Surg 71: 1967.e1, 2013.

114. Storum KA, Bell WH: Hypomobility after maxillary and mandibular osteotomies. Oral Surgery, Oral Med Oral Pathol 57: 7, 1984.

115. Finn R a, Throckmorton GS, Bell WH, Legan HL: Biomechanical considerations in the surgical correction of mandibular deficiency. J Oral Surg 38: 257, 1980.

116. Sickels JE Van, Tiner BD, Keeling SD, Clark GM, Bays R, Rugh J: Condylar position with rigid fixation versus wire osteosynthesis of a sagittal split advancement. J Oral Maxillofac Surg 57: 31, 1999.

117. Hwang SJ, Haers PE, Sailer HF: The role of a posteriorly inclined condylar neck in condylar resorption after orthognathic surgery. J Cranio-Maxillofacial Surg 28: 85, 2000.

118. Katzberg RW, Keith DA, Eick WR Ten, Guralnick WC: Internal derangements of the temporomandibular joint: An assessment of condylar position in centric occlusion. J Prosthet Dent 49: 250, 1983.

119. Al-Moraissi EA, Ellis E: Is There a Difference in Stability or Neurosensory Function Between Bilateral Sagittal Split Ramus Osteotomy and Intraoral Vertical Ramus Osteotomy for Mandibular Setback? J Oral Maxillofac Surg 73: 1360, 2015.

120. Kato T, Thie NM, Huynh N, Miyawaki S LG: Topical review: sleep bruxism and the role of peripheral sensory influences. J Orofac Pain 17: 191, 2003.

Appendix A

Oral Health Impact Questionnaire[10]

Table 1
OHIP-14 questionnaire translated to English.

Have you ever because of your teeth or oral problems:
a) Felt pain in your mouth?
b) Felt discomfort eating food?
c) Had an unsatisfying diet?
d) Felt the need to interrupt a meal?
e) Experienced degradation concerning taste ability?
f) Had difficulties pronouncing words?
g) Been embarrassed?
h) Felt insecure?
I) Felt tense?
j) Found it difficult to relax?
k) Felt irritation against other people?
l) Had problems to complete every day tasks?
m) Felt that life in general has been less satisfying?
n) Been unable to function in general?

Appendix B

Orthognathic Quality of Life Questionnaire[10]

Table 2
OQLQ questionnaire translated to English.

Read following claims carefully and put an X in the column that suits you:
1. I feel ashamed about the appearance of my teeth.
2. I feel ashamed about the appearance of my upper and/or my lower jaw.
3. I have problems biting.
4. I have problems chewing.
5. There are things I avoid eating because of problems with my bite.
6. I don't like eating in public places.
7. I often get pain in my face or my upper and/or my lower jaw.
8. I don't like seeing a side view of my face.
9. I spend a lot of time studying my face in the mirror.
10. I spend a lot of time studying my teeth in the mirror.
11. I spend a lot of time studying my upper and/or lower jaw in the mirror.
12. I dislike being filmed or having my photograph taken.
13. I often take notice at other people's teeth.
14. I often take notice at other people's faces.
15. I often feel insecure because of my facial appearance.
16. I try to cover my mouth when I meet people for the first time.
17. I feel nervous when meeting people for the first time.
18. I worry that people will make hurtful comments about my appearance.
19. I lose confidence when I am with other people.
20. I don't like smiling when I meet people.
21. My appearance makes me depressed sometimes.
22. I sometimes think that people are staring at me.
23. Comments about my appearance upset me, even when I know that it is not meant seriously.

Appendix C

Temporomandibular Joint Pain and Function Questionnaire

Orthognathic Surgery Temporomandibular Joint (Affix Patient Label Here)

Outcome Questionnaire

Today's Date: ________________

Date of Surgery: ______________

1. How would you rate your temporomandibular joint pain on a 0 to 10 scale AT THE PRESENT TIME, that is right now, where 0 is "no pain" and 10 is "pain as bad as could be"?

0	1	2	3	4	5	6	7	8	9	10
(No pain)									(Pain as bad as could be)	

2. In the last 30 days which of the following best describes any pain in your jaw, temple, or in front of the ear on either side?

   No Pain Pain comes and goes Pain is always present

3. In the last 30 days have you had any jaw joint noise(s) when you moved or used your jaw?

   Yes No

4. In the last 30 days did your jaw lock or catch severe enough to limit your jaw opening and interfere with your ability to eat?

   Yes No

5. In the last 30 days did your jaw lock or catch severe enough to limit your jaw closing and interfere with your ability to eat?

   Yes No

6. To the best of your knowledge do you clench or grind your teeth?

   Yes No Unsure

Appendix D

Orthognathic Surgery Pre-operative Evaluation Form

ORTHOGNATHIC SURGERY EVALUATION

Name ______________________ Surgeon _____________________ Date __________________

CONCERNS

Ortho _______________ Ht_____

Length_______________ Wt_____

FACIAL AESTHETICS

Brachycephalic Mesocephalic Dolichocephalic

Profile Convex Concave

MIDFACE

Orbits Zygoma Nose Symmetry Nasal tip

CANT

Maxilla VME VMD A-P

MANDIBLE

Retrognathic Mesognathic Prognathic

Chin Microgenia Macrogenia Asymmetry

LIP FORM Competent Incompetent Asymmetry

NL ML

MIDLINE

TSR TSS TL OB OJ IIO

ANGLE CLASS R I II III

L I II III

TRANSVERSE Deficient Adequate CROSSBITE

DENTAL

PERIODONTAL

RADIOGRAPHIC

WISDOM TEETH

MISSING TEETH

TMJ

PAIN R L CLICK R L CREPITUS R L DEVIATION EXCURSIONS

OSA

AHI RDI ESS Snoring Y N CPAP Y N

SURGICAL PLAN

LeFort

8's

BSSO

FG

Appendix E

Index of Orthognathic Functional Treatment Need[76]

Index of Orthognathic Functional Treatment Need

This index applies to those malocclusions that are **not amenable to orthodontic treatment alone, due to skeletal deformity,** and will ordinarily apply to those patients who will have completed facial growth prior to surgery (commonly 18 years of age and older). It relates only to the **functional** need for treatment and should be used in combination with appropriate psychological and other clinical indicators.

5. Very Great Need for Treatment
5.1 Defects of cleft lip and palate and other craniofacial anomalies
5.2 Increased overjet greater than 9 mm
5.3 Reverse overjet ≥ 3 mm
5.4 Open bite ≥ 4 mm
5.5 Complete scissors bite affecting whole buccal segment(s) with signs of functional disturbance and or occlusal trauma
5.6 Sleep apnoea not amenable to other treatments such as MAD or CPAP (as determined by sleep studies)
5.7 Skeletal anomalies with occlusal disturbance as a result of trauma or pathology
4. Great Need for Treatment
4.2 Increased overjet ≥ 6 mm and ≤ 9 mm
4.3 Reverse overjet ≥ 0 mm and < 3 mm with functional difficulties
4.4 Open bite < 4 mm with functional difficulties
4.8 Increased overbite with evidence of dental or soft tissue trauma
4.9 Upper labial segment gingival exposure ≥ 3mm at rest
4.10 Facial asymmetry associated with occlusal disturbance
3. Moderate Need for Treatment
3.3 Reverse overjet ≥ 0 mm and < 3 mm with no functional difficulties
3.4 Open bite < 4 mm with no functional difficulties
3.9 Upper labial segment gingival exposure < 3mm at rest, but with evidence of gingival/periodontal effects
3.10 Facial asymmetry with no occlusal disturbance
2. Mild Need for Treatment
2.8 Increased overbite but no evidence of dental or soft tissue trauma
2.9 Upper labial segment gingival exposure < 3mm at rest with no evidence of gingival/periodontal effects
2.11 Marked occlusal cant with no effect on the occlusion
1. No Need for treatment
1.12 Speech difficulties
1.13 Treatment purely for TMD
1.14 Occlusal features not classified above