Université de Montréal

Effets post-orthodontiques de l'EPRAC sur les troubles respiratoires de sommeil sur jeunes adultes tel qu'observé en laboratoire de sommeil

Post Orthodontic effects of SARPE on sleep-disordered breathing in young adults as observed in a sleep laboratory

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Ce mémoire intitulé :

Effets post-orthodontiques de l'EPRAC sur les troubles respiratoires de sommeil sur jeunes adultes tel qu'observé en laboratoire de sommeil

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Résumé

Introduction: L'expansion palatine du maxillaire a beaucoup d'effets positifs sur la respiration et la qualité du sommeil, mais peu d'études ont examiné ces données sur des adultes ayant dépassé l'âge permettant de bénéficier d'une expansion palatine conventionnelle. Le but de cette recherche est d'évaluer la stabilité de l'EPRAC (expansion palatine rapide assistée chirurgicalement) et son effet sur les troubles respiratoires après l'ablation des appareils orthodontiques. **Méthodes:** Neuf patients (Âge moyen 21, entre 16-39 ans) nécessitant une EPRAC ont passé des nuits dans un laboratoire de sommeil, et ce avant l'EPRAC, après l'EPRAC, et après l'ablation des appareils fixes. Les radiographies céphalométriques postéroantérieures ainsi que les modèles d'étude ont été pris pendant ces trois périodes de temps. **Résultats**: L'analyse des modèles d'étude a démontré une récidive significative au niveau des distances inter-molaires et inter-canines au niveau du maxillaire seulement. Les analyses céphalométriques ont démontré une récidive au niveau de la largeur maxillaire. Aucun changement important n'a été observé dans les stades de sommeil, mais une réduction importante dans l'index de ronflement a été notée. De plus, il y avait moins de changements entre les stades de sommeil. **Conclusions:** La récidive squelettique est minime et cliniquement non significative. Par contre, les changements dans les distances intermolaires et intercanines sont cliniquement importants. Il semble également qu'une EPRAC ait un effet positif sur la qualité de sommeil par la réduction de l'indice de ronflement ainsi que sur la diminution des changements entre les stades de sommeil.

Mots-clés : ERPAC, Expansion Rapide Palatine Assisté Chirurgicalement, Sommeil, Ronflement

Abstract

Introduction: Orthopedic expansion appears to have several positive effects on respiration as well as sleep quality, but a lack of studies examine these findings using SARPE on skeletally mature individuals. The aim of this study was to evaluate post-SARPE stability as well as its effect on sleep disordered breathing after completing full fixed orthodontics. **Methods:** 9 patients (average age 21, range 16-39) requiring SARPE underwent polysomnographic testing in sleep laboratory before SARPE (T0), after SARPE (T1), and after removal of full fixed appliances (T2). Study models and anteroposterior cephalometric radiographs were also taken at the 3 time points. **Results:** Study model analysis showed significant relapse for intermolar and intercanine widths. Anteroposterior cephalometric results were significant only for effective maxillary width. There were no significant changes in any sleep stages, however a dramatic reduction in snoring as well as fewer stage shifts were observed. **Conclusions:** Although statistically significant relapse was observed on study models and anteroposterior cephalometric radiographs, the dental relapse appears to be more clinically significant than the skeletal relapse. SARPE appears to have a positive effect on sleep quality by reducing the snoring index as well as reducing transitions between sleep stages.

Keywords : SARPE, Surgically Assisted Rapid Palatal Expansion, Sleep, Snoring

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List of Abbreviations

AG	Antegonial Notch
AHI	Apnea-hypopnea index
CT	Computed tomography
CBCT	Cone beam computed tomography
EEG	Electroencephalogram
EOG	Electro-occulogram
EKG	Electrocardiogram
EMG	Electromyogram
IC	Inter-canine
ICC	Intra class coefficient
IM	Inter-molar
JR	Jugale right
JL	Jugale left
Mand W	Mandibular width
Max W	Maxillary width
MMWD R	Maxillo-mandibular width differential right
MMWD L	Maxillo-mandibular width differential left
MTD	Maxillary transverse deficiency
NREM	non rapid eye movement
OSA	Obstructive sleep apnea
PA	Postero-anterior
REM	Rapid eye movement
RPE	Rapid palatal expansion
SARPE	Surgically assisted rapid palatal expansion
TAD	Temporary Anchorage device

To my wife and daughter, for without your support, none of this could have ever happened. To my mom and dad, who put their children before themselves, so they could succeed.

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1. Introduction

Many patients present to the orthodontist with several problems. Crowding, spacing, rotations as well as excessive or insufficient overbite and overjet are commonly seen. However the most common skeletal problem encountered by orthodontists is a transverse deficiency of the maxilla. [1]

The treatment for this problem varies with the age of the patient as well as the severity of the problem. Treatment options can range from a simple occlusal adjustment to a modified LeFort I surgery.

The effect of rapid palatal expansion (RPE) has been extensively studied in growing children. Aside from correcting occlusal problems such as crossbites, other studies have shown changes in palatal depth, arch perimeter, and even nasal volume after this treatment. Recent studies have shown changes in sleep as well.[2]

A more invasive and necessary approach is required for adults to correct problems in the transverse dimension. With increasing age, the relatively non-invasive orthopedic RPE is not an option and surgical intervention is required.[3, 4]

The goal of this project is to evaluate sleep disordered breathing after orthodontic treatment on patients that have received a surgically assisted rapid palatal expansion (SARPE). All patients have had polysomnographic testing, before SARPE (T0) and 6 months after SARPE (T1).

The patient sample was extracted from the master's theses for Dr Normand Bach and Dr Chantal Gautier. Preliminary data from Bach 6 months post SARPE showed increased deep sleep as well as a tendency towards less microarousals.[5]

In this paper, we will be evaluating any changes in sleep and sleep disordered breathing once fixed appliances have been removed (T2). Our results will show any long-term changes that may or may not be present. We will be also analyzing any changes in stability of the SARPE and quality of sleep.

2. Literature Review

2.1 The Maxilla

2.1.1 Anatomy

The maxilla forms the majority of the skeleton of the upper face and contains the paranasal sinuses.[6] The maxilla is a paired bone, separated by the intermaxillary suture, and thus articulates with the opposite maxilla, as well as the ethmoid, sphenoid, nasal, vomer, palatine, lacrimal, and zygomatic bones. It also articulates with the inferior nasal conchae and the septal and nasal cartilages.

The maxilla is made up of five parts: the body, frontal process, zygomatic process, palatine process, and alveolar processes.[6]

2.1.2 Growth and Development

The maxilla is formed by intramembranous ossification and is a derivative from the first pharyngeal arch, which appears at week four in utero.[7] The two maxilllary prominences, two mandibular prominences, and frontonasal prominences all grow as a result of the migrating neural crest cells which proliferate in the first pharyngeal pouch (see Figure 1).[7]

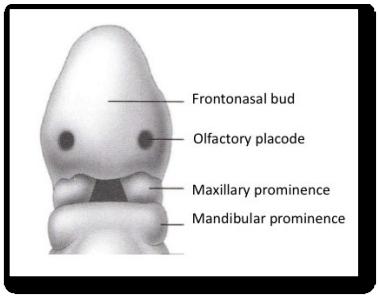

Figure 1. Four weeks in utero. Adapted from Bishara.[7]

The continued proliferation of the maxillary prominences and their medial migration toward one another forms the majority of the maxilla with the exception of the premaxilla. The premaxilla is formed by the migration of the medial nasal prominences towards each other which happens between seven and ten weeks in utero.

The secondary palate, located posterior to the premaxilla and incisive foreamen, is formed after the fusion of the medial nasal and maxillary prominences. Between the tenth and twelfth week, the lateral palatine shelves of the maxillary process continue to proliferate and move medially, as the tongue moves inferiorly. The fusion of both palatine shelves medially, and the fusion with the primary palate anteriorly, completes the formation of the palate (hard and soft) as shown in Figure 2.[7]

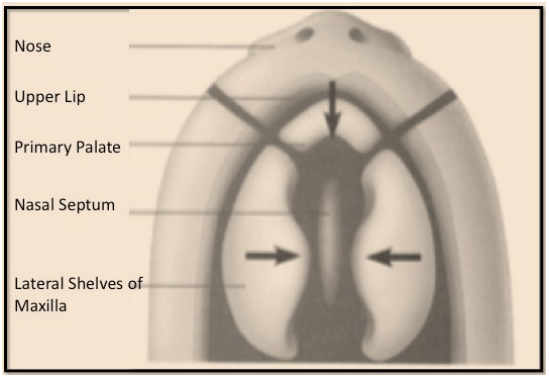

Figure 2. Primary and Secondary Palate. Adapted from Bishara.[7]

2.1.3 Post Natal Growth of the Maxilla

The direction of growth of the maxilla is accepted to be in a downward and forward direction. After birth, growth of the maxilla is thought to be due to two mechanisms. The

first is due to a push from behind from the cranial base, while the second is thought to be due to active growth at the maxillary sutures and nose. [4]

The growth of the cranial base occurs at the sphenooccipital synchondroses, which is essentially a two-sided epiphyseal plate. Proliferating cartilage cells migrate in both directions and endochondral ossification is the result.[8] This passive displacement is most important and contributes to the majority of growth until approximately 7 years of age.[4] After 7 years of age, about two thirds of the maxillary forward movement can be attributed to active growth at the maxillary sutures in response to stimuli from the enlarging soft tissues. This continues until age 15.

The dimensional increase of the maxilla occurs as bone is deposited posteriorly at the tuberosities. [8, 9] This makes room for the developing third molars.[10] Anteriorly, there is marked resorption that occurs just below the anterior nasal spine. Implant studies have shown that for every 11 mm of midfacial displacement, the orbital floor drifts superiorly 6mm and the nasal floor drifts 5 mm inferiorly.[11] Therefore, height increase of the midface is due to combined inferior drift and displacement.[12]

At birth, the width of the midface appears disproportionately large due to the advanced development of the eyes. The brain continues its expansion within the first few years of life in the sagital, frontal, and coronal directions.[12] As this occurs, the metopic suture (separates the frontal bones), the internasal suture (separates the nasal bones), the intermaxillary (separates the two maxillae), and midpalatal suture, all respond by secondary, compensatory bone formation. The bi-jiugale widths have been shown to increase 6mm from age 7 to 15 while the mid-alveolar widths increase 5 mm.[13] In the roof of the mouth, apposition dominates while resorption occurs on the floor of the nose.

Growth continues as long as sutures remain unfused. The premaxillary sutures are the earliest maxillary sutures that fuse at approximately 3-5 years of age.[14] The midpalatal and transpalatal sutures both fuse after the pubertal growth spurts at 15 to 19[15] and 20 to 25 [16] years of age respectively. It has been postulated that closure of the circumaxillary sutures occurs after the intermaxillary sutures.[12]

The growth of the maxilla is completed before the growth of the mandible. The antero-posterior growth is complete by approximately 14 years of age.[17-21] The vertical growth of the maxilla continues indefinitely, but at an immensely slower rate.

2.2 Maxillary Transverse Deficiency

2.2.1 Diagnosis of Maxillary Transverse Deficiency

Skeletal maxillary transverse deficiency (MTD) can be diagnosed in an individual with a narrow palatal vault, bilateral crossbite (Figure 3), unilateral crossbite (Figure 4), chin asymmetry with or without a functional shift, and excessive curves of Monson and Wilson. Frontal cephalometric radiographs are also another method used for diagnosis.[22]

2.2.2 Skeletal Crossbite

The crossbite can involve one or several teeth. The etiology of the crossbite can be dental or skeletal or both. Most of the time, the presence of a bilateral crossbite (Figure 3) indicates the presence a moderate to severe maxillary deficiency in the transverse dimension. The incidence of crossbite in the deciduous and mixed dentition is 7-23% and is not self-correcting as the child progresses from one dentition to the next.[23-27]The etiology of crossbites is multifactorial and includes genetics as well as habits and environmental influences.[28] Some causes can to be due to prolonged retention of primary teeth, premature

loss of primary teeth, symmetric growth of the maxilla or mandible, abnormalities in eruption sequence, impaired nasal breathing during critical growth periods, deviations in tooth anatomy, and improper functioning of the temporomandibular joint.[25, 29, 30]

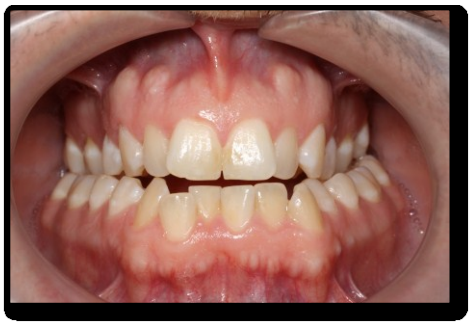

Figure 3. Bilateral Posterior Crossbite (Photo by Dr P. Ross Fiore)

The presence of a unilateral posterior crossbite that involves multiple teeth is probably skeletal in origin, while the presence of a single tooth in crossbite is usually dental in origin. In general, the more teeth involved in the crossbite, the higher the chance the problem is skeletal in nature and the higher the chance that a transverse deficiency exists.[4]

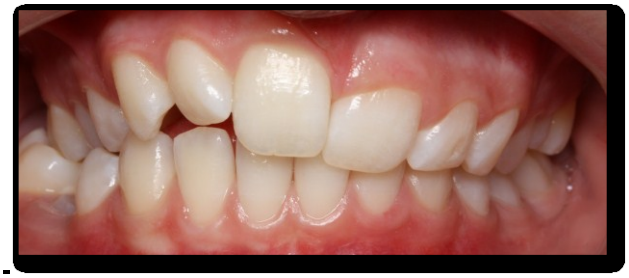

Figure 4. Unilateral Posterior Crossbite (Photo by Dr P. Ross Fiore)

2.2.3 Dental Crossbite

On some occasions, the crossbite is purely dental in origin. In these instances, most likely only one tooth is involved. The lower tooth usually has the proper torque while the

maxillary tooth has excessive palatal crown torque or vice versa. There is no transverse discrepancy in these situations. Skeletal (orthopedic) correction is not always necessary.

While the presence of crossbites is a good indicator that a MTD exists, it is not always true. A *relative* maxillary transverse deficiency exists in cases where the patients presents with a unilateral or bilateral crossbite and a Class III malocclusion.[31] At first glance, one can observe an apparent MTD when in fact there is no MTD and the crossbite is due to mandibular excess. This is confirmed when placing the study models in a class I canine relationship and the crossbites are no longer present.[31]

2.2.4 Maxillary Transverse Deficiency in the Absence of Crossbites

The absence of crossbites does not rule out the presence of a MTD either. An *absolute* maxillary transverse deficiency represents a true MTD[31] as compared to a *relative* MTD where there is no lack in the transverse dimension. This is observed when the models are placed in Class I canine relationship and crossbites still exist. Another example of an *absolute* maxillary transverse deficiency is when patients present with a Class II malocclusion and no apparent MTD. But, when placing the models in Class I occlusion, one can see the presence of a crossbite while representing a true insufficiency in the transverse dimension.[31]

The removal of dental compensation during orthodontic therapy can often reveal absolute transverse deficiencies, even though no crossbite was apparent at the initial exam. Many patients present with lingually inclined mandibular posterior teeth and buccally inclined maxillary teeth that can camouflage a MTD. If the MTD is not accounted for, and the patient receives conventional fixed therapy, then a crossbite may result as shown in Figure 5.

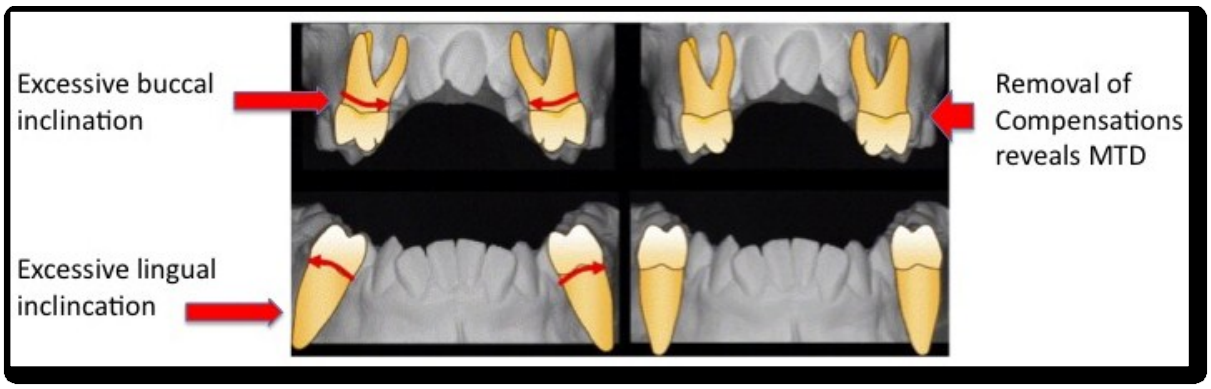

Figure 5. Camouflaged MTD. Adapted from Marshall[23]

2.2.5 Mandibular Asymmetry

On frontal exam, it is possible that the patient may have a chin deviation. [23] This may be due to either a skeletal deviation or functional shift leading to the appearance of a chin deviation (usually with crossbite). Placing the patient in centric relation should eliminate the deviation. If there is no improvement, then a true skeletal chin asymmetry exists. The true skeletal asymmetry could have been due to an initial functional shift that caused a permanent change. Other causes include hemifacial microsomia, hemifacial hypertrophy, condylar hyperplasia, previous condylar fractures, and juvenile rheumatoid arthritis.[32-36]

It is always prudent to test for a functional shift of the mandible when a crossbite is present. This will be an important factor when deciding when to treat and will be discussed later. Some patients show a slide from centric relation to centric occlusion for better maximal intercuspation which gives rise to a chin deviation, crossbite, and dental midline deviation. In figure 6 below, we can see a posterior crossbite as well as a lower midline deviation when the patient is in maximal intercuspation. When the patient is in centric relation, the midlines are well aligned and a cusp to cusp relationship is observed in the buccal

segments. Since a cusp to cusp relational in the buccal segments is observed, an obvious MTD exists.

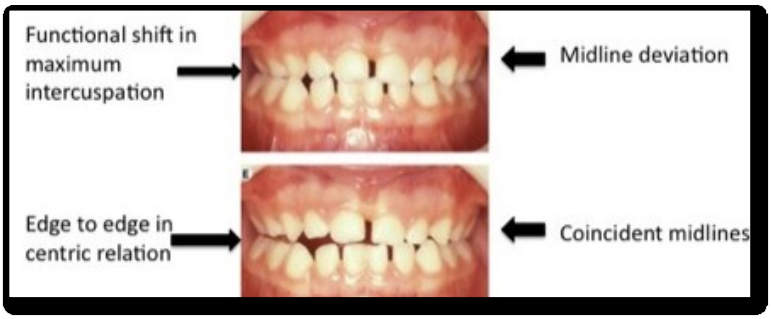

Figure 6. Functional Shift and Unilateral Posterior Crossbite. Adapted from Marshall.[23]

In adults, it is especially important to determine the nature of any mandibular shift on closure.[3] Their muscular kinestetic memory and proprioceptive influences are more ingrained than in children. In some cases, a muscular deprogramming device such as an occlusal plate may be necessary to allow the muscles to move the mandible in coordinated function that is undisturbed by any tooth contacts.[37, 38]

2.2.6 True Unilateral Skeletal crossbite

Although rare, in some cases the MTD is asymmetric. For these patients conventional expansion would produce asymmetric results and requires a different biomechanical approach.[23] Specific treatment for these patients will be discussed later.

2.2.7 Rocky Mountain Analysis

Together with study casts and P-A radiographs, one is able to evaluate the widths of the dental arches, alveolar arches, and the jaws.[39] This is a valuable diagnostic tool but

underutilized because clinicians normally use the P-A cephalometric radiograph for evaluating symmetry. [40] The Rocky Mountain Analysis, shown in figure 7, and as proposed by Ricketts, is composed of the following indicators:

a. Effective Nasal Width
b. Effective Maxillo-Mandibular transverse relation
c. Effective Mandibular Width
d. Skeletal Symmetry
e. Arch Width at the First permanent Molar
f. Arch Width at Lower Cuspids
g. Harmony of Arch width with Jaws
h. Midline drift of the lower Arch
i. Indicator of Molar Crossbite

Mandibular Width- The mandibular width is evaluated by choosing one point on each side of the mandible, at the antegonial tubercle (Ag). These are located just inferior to the trihedral eminence.

Maxillary Width- The maxillary width is evaluated by tracing two points, which intersect between the zygomatic process and maxillary tuberosity. These are called the Jugale points. They are labeled Jugale left (JL) and Jugale right (JR) as appropriate. The effective maxillary width is the distance between JL and JR.

Maxillomandibular difference-The age-appropriate AG-GA distance minus the age-appropriate JR-JL distance.

Maxillomandibular Transverse Differential Index- This is the expected maxillomandibular difference minus the actual maxillomandibular difference. Since our entire sample was

older than 16 years of age, the expected maxillomandibular difference was 19.6 and the actual was the measured effective mandibular width minus the effective maxillary width.

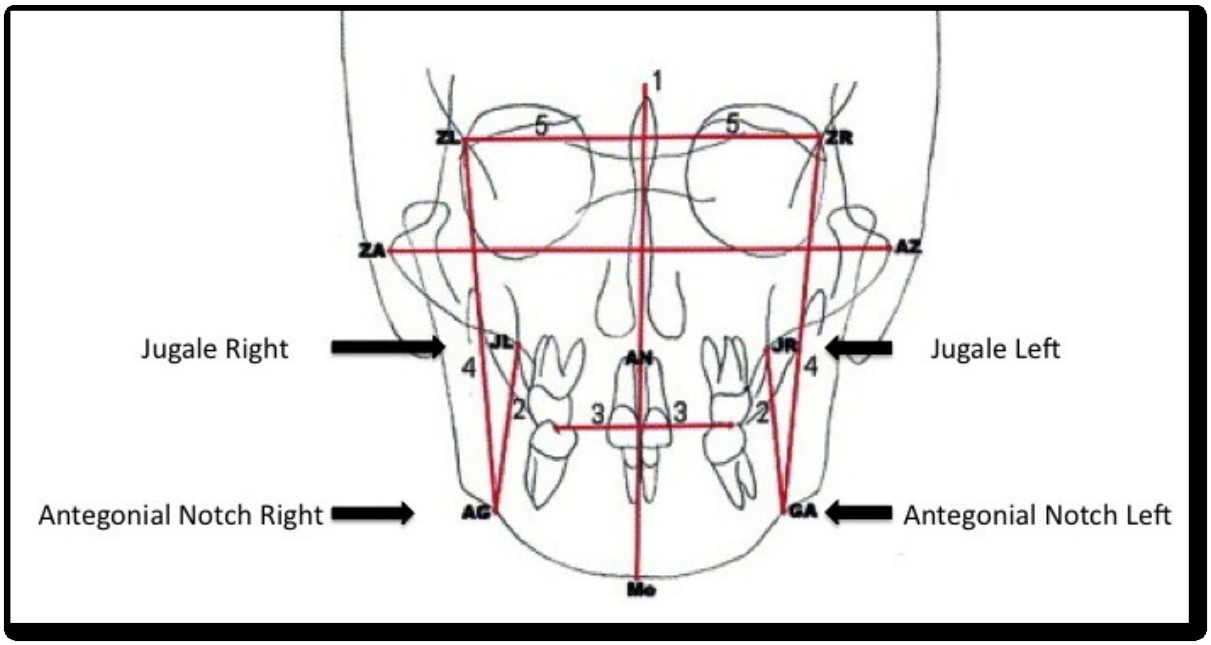

Figure 7. Landmarks and planes for PA ceph. Adapted from Ricketts.[41]

2.3 Treatment of Maxillary Transverse Deficiency

2.3.1 Treatment Goals

The objective for patients with a maxillary transverse deficiency is to increase the transverse dimension, allowing the teeth to be over basal bone, with proper inclination

while in maximum intercuspation. This is accomplished by surgical and non surgical methods, as well as with removable or fixed appliances.

2.3.2 Treatment Options

Several treatment options are available for the clinician to treat MTD. This paper will discuss:

a) Rapid Palatal Expansion (Hyrax and Haas)
b) Slow Expansion (Quad helix)
c) Semi rapid expansion (removable plate)
d) Surgically assisted rapid palatal expansion (SARPE)
e) Occlusal adjustment for treatment of functional shift
f) Implant born expansion devices
g) Expansion Appliance combined with elastics (true unilateral crossbite)

Rapid palatal expansion consists of using an appliance with a jackscrew that opens 0.5mm per day (2 turns). With semi-rapid expansion, 0.25mm (1 turn) of expansion per day is obtained. Slow expansion is obtained by 1 turn every other day, or by activating a quad helix 1 molar width activation.[42]

2.3.3 Treatment Timing

The presence of a functional shift is an immediate sign of a MTD and warrants treatment at the time of diagnosis for children and young adolescents. Because growth is occurring, the functional shift and mandibular deviation could possibly become permanent once growth has ended. Some clinicians prefer to expand the arches early since an increase in arch perimeter will also be gained and facilitate eruption of the permanent dentition.[43]

In the absence of a functional shift, treatment of the MTD can wait until adolescence when comprehensive treatment with full fixed appliance therapy is planned, provided that it occurs before the age of 15.

After the age of 15, the treatment of a severe MTD is not possible without surgical intervention.[4] For the purpose of this paper, we will discuss treatment at 3 stages: the primary and mixed dentition, early permanent dentition, and adulthood.

2.3.4 Treatment During the Primary and Early Mixed Dentition

As mentioned earlier, the presence of a functional shift warrants immediate treatment. This can be quite challenging in the early mixed dentition, as retention of the appliance may be difficult. The appliance and activation depend on age[4] as rapid palatal expansion on a young child could have deleterious esthetic results on the nose.[44] A more conservative approach is to use slow expansion (1 mm per week). [4]

Slow expansion can be carried out in the primary dentition or early mixed dentition by using an active palatal arch or quad helix as shown in figure 8. The appliances will separate the palatal suture so that skeletal and dental expansion are obtained.

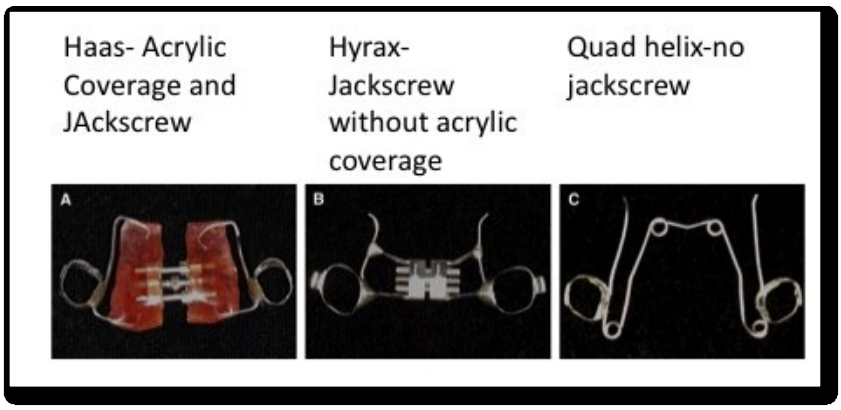

Figure 8. a) Haas b) Hyrax c) Quad helix. Adapted from Huynh.[42]

The use of a removable appliance with a jackscrew is also possible, but this must be activated slowly to permit the tissues and teeth to adjust to the forces (Figure 9). Normally, due to poor patient compliance, this appliance has problems with retention because the Adams clasps will not fit even after a short period out of the mouth.[4] In order for the appliance to fit once adjusted, it is recommended to turn the screw with the removable appliance in place in the mouth and to keep it in place throughout the evening and night.[45]

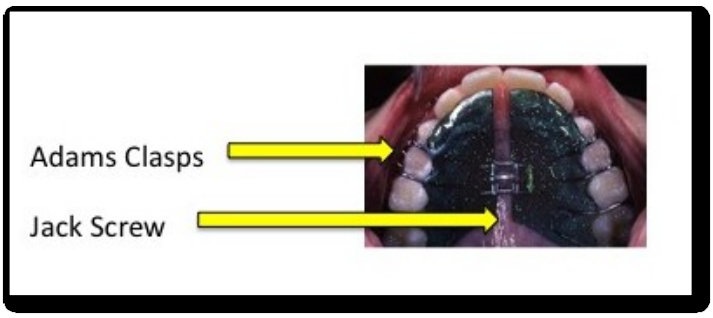

Figure 9. Removable Expansion Plate. Adapted from Sophia[46]

In the primary dentition, sometimes a simple occlusal adjustment may suffice to eliminate the presence of a functional shift (Figure 10). Most of the time, an interference is apparent on one of the mandibular canines. An enameloplasty of the deciduous canine is likely to be

sufficient. The clinician can then wait until the mixed dentition to treat any transverse deficiency since compliance and retention may be an issue for patients of a young age.

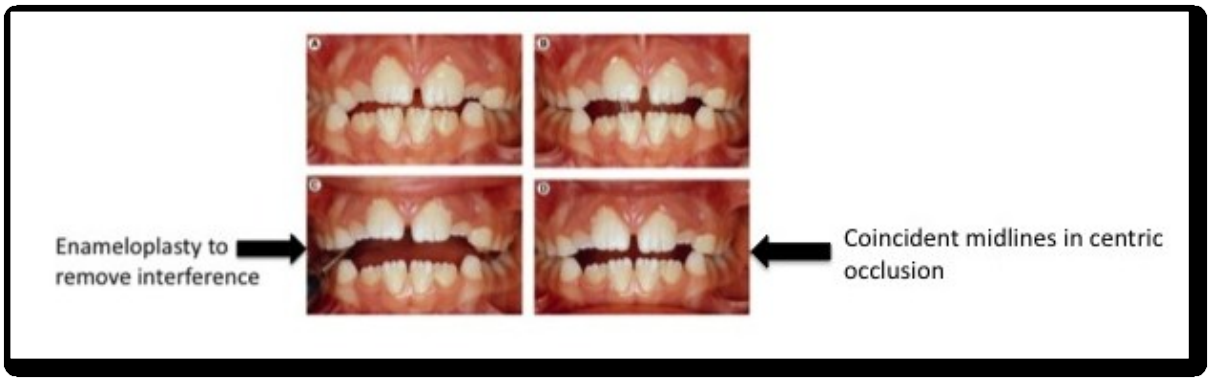

Figure 10. Enamelplasty to remove a functional shift. Adapted from Marshall.[23]

2.3.5 Treatment of True Unilateral Crossbite

As mentioned earlier, special considerations are required for a true asymmetric maxillary transverse deficiency (upon careful evaluation, it is usually observed that the majority are due to a bilateral asymmetry). By employing a conventional expansion appliance, correction of the constricted side would ensue, but over expansion of the normal side would result in a buccal crossbite.

A possible technique to correct a true unilateral crossbite would be to use a conventional maxillary expander in the upper arch, while a lingual arch is cemented in the lower arch. Regular activations occur, while crossbite elastics are worn on the normal side. This allows for correction of the constricted side, while maintaining the side with normal occlusion.[23]

2.3.6 Treatment During Adolescence

As the patients age, the facial sutures become more interdigitated, especially after puberty.[47] This includes the palatal suture as well as the circumaxillary sutures and midface sutures (Figure 11). [45] It must be remembered that girls mature faster than boys, so skeletal maturity and treatment non-surgically should be expedited in females. Great variation between individuals exists however and some individuals can show orthopedic expansion well into their twenties.[16]

It has been suggested that an occlusal radiograph may be a failsafe way to determine ossification of the midpalatal suture.[48] Unfortunately, due to the two dimensional nature of the image, as well as the superimposition of several cranial base structures, proper evaluation may not be possible.[49, 50]

As a general rule, non-surgical treatment of adolescents with a transverse discrepancy should be attempted before age 15 using a rapid palatal expander (Hyrax or Haas type).[4] After this, and depending on the degree of transverse deficiency, surgical treatment may be the only option to correct this insufficiency.[3]

Recently, with the increased use of temporary anchorage devices (TADs), skeletal anchorage has been used to fix the expansion appliance in place. A recent study compared traditional tooth anchored maxillary expanders to bone anchored maxillary expanders.[51] After 12 months, no significant difference in the amount of expansion at the molars was observed. Interestingly, expansion across the premolars was greater in the traditional tooth anchored maxillary appliance group. This study showed no advantage to using bone anchored maxillary expanders over the conventional tooth supported expanders. One indication remains in the use of this type of appliance for periodontally compromised cases such as those with gingival recession.[52]

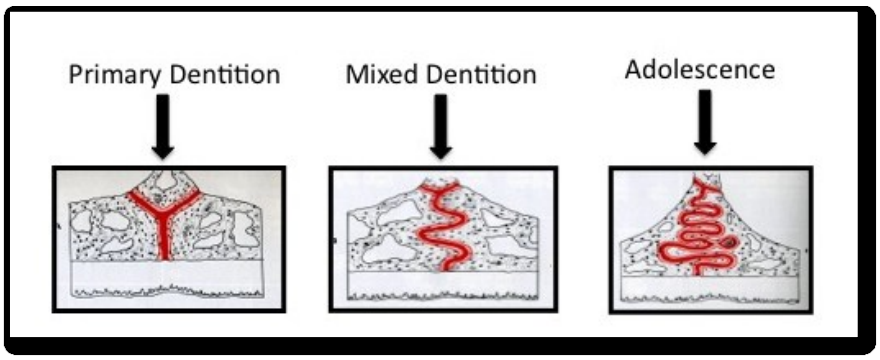

Figure 11. Increased Palatal Interdigitations with Age. Adapted from Proffit.[45]

2.3.7 Treatment in Adults

In the "adult stage," histological sections of the palatal suture have noted synostoses and numerous bony bridge formations across the suture.[53] One can conclude from these findings that orthopedic expansion may prove to be difficult or impossible when the patient has reached this stage of development.[54]

If one were to attempt conventional orthopedic expansion in a skeletally mature individual, a myriad of problems can occur. It would be normal to see extreme bending of the alveolus, dental tipping, and minimal maxillary expansion.[55-57] Other problems despite overcorrection include relapse, periodontal defects, and malocclusion.[58] [59] [60]Skeletally mature individuals are also subject to buccal root resorption, fenestration of the buccal cortex, pain, and palatal tissue necrosis. [3, 61]

The only option in cases of moderate to severe cases of maxillary transverse discrepancies is surgery. Today SARPE (Surgically Assisted Rapid Palatal Expansion) is used for these patients.[44] In cases where the transverse discrepancy is less than 5 mm, orthodontics alone can camouflage the discrepancy and surgery is not necessary.[55] Therefore, for a MTD greater than 5 mm, SARPE is the recommended option.

The appliance typically used is a Hyrax or Haas appliance, with bands on both the first upper molars and either bands or occlusal rests on the first premolars. It is cemented intraorally prior to the day of surgery. The appliance is then activated during the surgery (after the osteotomy).

2.4.0 Surgery

2.4.1 Surgical Technique

The surgery is essentially a Lefort I without the maxillary downfracture. The pterygoid plates are usually left intact, but this varies between surgeons. An osteotome splits the median palatine suture from the buccal side. The appliance is usually turned approximately 12 times until a diastema confirms a successful osteotomy.[62] Five days after the surgery, the patient then continues turning the appliance twice per day (0.5 mm) until the desired expansion is achieved.

There is no agreement on the exact surgical technique.[3] Some authors leave the pterygoid plates intact,[63-66] while several authors make dysjunction of the pterygoid plates part of their surgical procedure.[56] The rational for dysjunction is to reduce resistance and allow for a more parallel expansion.[62] Other authors believe that less resistance contributes to less periodontal injury, tipping, as well as relapse.[62, 67, 68] A large difference appears to be whether or not the pterygoid plates are fractured.

It was once thought that the main resistance to expansion was the midpalatal suture, but other studies have shown the additional resistance coming from the zygommatic buttress[69-71] and the circumaxillary sutures.[58, 72, 73]

2.4.2 Segmental Lefort Osteotomy versus Two Stage Surgery

It has been shown by some authors that 2 surgeries (SARPE followed by another jaw surgery) can yield better results. Due to the stretch of the palatal tissues, a segmented Lefort I may lead to increased transverse relapse.[74] In addition, a one piece Lefort has been shown to have less chances of aseptic necrosis than a segmental.[75, 76] Studies have shown increase in compromise of the vascular palatal pedicle during surgery where the maxilla is segmented.[55] Furthermore, less control is offered to the orthodontist since the arch wires must be segmented and special retention is required by the patient after the surgical procedure.[74]

High relapse rates have been shown in the transverse dimension when significant expansion in required with a multipiece maxilla. Phillips showed as much as 40% with a multipiece maxilla.[77]

Proffit et al suggests an opposing view.[4] In the case of a moderate MTD, they suggest that the increased costs and risks of two surgeries are not justified if acceptable results can be obtained with one surgery.[4] Two separate maxillary surgeries are only justified in cases of severe MTD where a segmental LeFort would compromise the vascular supply.[4]

2.5.0 Stability

2.5.1 Skeletal versus Dental Expansion

Regardless of the type of expander used, the goal is the same-skeletal transverse expansion of the maxilla. As with all movements in orthodontics, relapse occurs after active expansion has been completed.

With rapid palatal expansion (0.5mm/day or 2 turns), the forces exceed physiological adaptations of the teeth, as 10 to 20 pounds of force is delivered.[4] The result is primarily skeletal expansion for the first two to three weeks as a result of the opening of the palatal suture. After this, the jackscrew is usually tied to stabilize the appliance. During this time, the bony segments reposition themselves and some skeletal expansion is lost. By 10 weeks, 50% of the expansion is dental, and 50 % is skeletal.[4]

With slow expansion (0.25mm every other day), the forces are such that the tissues of the mid palatal suture can adapt, and only 2 to 4 pounds of force is delivered.[4] As such, the amount of expansion achieved is 50% dental and 50% skeletal,[4] which is equal to what is achieved with rapid palatal expansion.

2.5.2 Stability of Orthopedic Expansion

According to Proffit, relapse is due to the elasticity of the palatal mucosa[4]. It has been suggested that over expansion of the buccal segments is necessary to account for this phenomenon. The upper arch is expanded until the upper lingual cusps touch the buccal cusps of the mandibular molars.[4] Other studies have shown that with expansion, increased pressure from the cheek occurs at the molars which may contribute to loss of the gain in the transverse dimension.[78]

It has been shown that more skeletal effects are achieved and maintained if the treatment is started before the pubertal growth spurt.[54] Approximately 25% of the expansion achieved is maintained if begun before the growth spurt.[79] Retention of the expansion is required for at least three month after expansion.[4]

Relapse rates are much higher in skeletally mature patients, thus showing its questionable clinical applicability. Some older patients receiving orthopedic expansion can experience relapse rates of approximately 63%.[80-82]

Rapid palatal expansion is a biologically sound procedure, with good stability if used at the proper age, even without retention appliances in some cases.[58]

2.5.3 Stability of Surgical Expansion

Several studies have reported varying degrees of SARPE stability, which range from 9-30 % relapse in the canine area and 8-36% relapse in the molar area.[66, 67, 74, 83-88] A major reason for the differences found in the literature is due to the varying treatment protocols as well as retention periods. [86, 89, 90] It is the clinical preference of some clinicians to leave the expansion appliance cemented until 6 months after the SARPE[91] while others prefer to remove the appliance and replace it with a removable acrylic appliance or transpalatal arch to maintain the transverse gain.[92]

Controversial data exist as to changes in equilibrium pressure affecting relapse. One study evaluated cheek tongue and lip pressure and found a significant increase in cheek pressure after expansion, but this increase in pressure slowly returned to pre-expansion values while in retention.[93]

Byloff showed slightly higher stability of transverse expansion at the second premolar showing a relapse rate of 28%, while the most significant relapse was observed at the first molars (36%).[85] They also demonstrated significant buccal angular changes which almost completely relapsed (from 9.6 degrees to 9.4 degrees).[85]

Northway showed a mean increase of 3.3 mm at the maxillary canines as well as 5.5 mm at the maxillary molars with the follow up being approximately 2 years after orthodontic therapy.[67] The depth of the palate was also significantly reduced. [67] Relapse was shown to be between 5-33%.[67]

In a 2.4 year follow up, once fixed appliances were removed, Bays showed that in 19 adults aged 30, relapse from SARPE in the molar and canine regions was 7.7% and 8.8% respectively.[83]

Recently, Chamberland and Proffit have shown similar results with a larger sample of patients, in whom all pterygoid plates were separated.[89] This study had a large sample size of 39 patients with 2 years post orthodontic treatment follow up. They showed that the only clinically significant relapse occurred at the maxillary first molars, where 17% or 1.1mm of relapse occurred.

2.6.0 Biomechanics and Other Effects of SARPE Procedure

2.6.1 Biomechanics

As orthodontic clinicians, we would ideally like pure lateral movement of the maxillary shelves, but this is clearly not what happens during expansion. While it is true that some dental tipping occurs, there is also tipping of the maxillary shelves since the force applied by the expansion screw is below the centre of resistance of each half of the maxillae.[94] The result is a fan shape opening of the palate.[95]

It has also been shown, as verified by cone beam tomography six months post SARPE

significant tipping of maxillary posterior teeth (4-5°) and their supporting alveolar bone and alveolar bending occurs(5-6°).[96]

In the sagittal plane, it has been shown that maxilla is displaced foreward.[58, 97-99] There is also statistically significant retroclination of the upper incisors that takes place is well.[100]

Some authors have also shown that greater stress distribution occurs if the pterygoid plates are not fractured, which can then be a source of surgical instability and relapse[101, 102]. Some have also noted that the increased stress could actually result in fracture of the cranial base resulting in eye mobility problems.[76]

Interestingly, finite element analysis has been recently used to show the stress distribution of various osteotomies that can be done during a SARPE. Holberg et al set up simulation with three possible osteotomies: one without any osteotomy, one with a lateral osteotomy, and one with a lateral osteotomy with separation of the pterygoid plates. Their results clearly show significant reduction in stresses at most structures of the midface, the sphenoid pterygoid process, as well as the foraminae of the cranial base, when there is a release of the pterygomaxillary junction. [101]

Therefore, in order to obtain posterior expansion of the maxilla, the pterygoid process on both sides needs to be released.

2.7.0 Changes in Nasal Resistance

Given the fact that the maxillary bones form over half of the structure of the nasal cavity, it is logical to assume that a change in the maxillary bones would affect the anatomy and physiology of the nasal fossa.[103]

Until recently, only subjective evaluation of nasal breathing pre and post expansion could be evaluated. The need for a more objective method was introduced with acoustic rhinometry.

Acoustic rhinometry is a simple, reproducible and non-invasive way to evaluate nasal volume.[104, 105] The underlying principle is the analysis of the acoustic pulse response that arises through impedance discontinuity inside hollow spaces.[106]

The nasal valves are the minimal cross-sectional areas of the nose, which give the greatest resistance to nasal airflow. Maxillary expansion causes a pyramidal type expansion with the greatest expansion at the level of the incisors, which are just inferior to the nasal valves.[103] The widening of the maxillary shelves also give rise to a widening of the lateral walls of the nose, thus increasing the total nasal volume. [107]

Oliveira et al measured nasal airway resistance (NAR) at several time points, pre and post expansion. They showed a 25.5% reduction in NAR after expansion, which was stable up until 12 months showing no signs of increased resistance. They also showed increases in nasal volume. The nasal volume increased by 18% after expansion, which was stable until a further 7% increase occurred. This was attributed to growth.[103]

Doruk et al found similar results 8 months following retention. On a study with 22 patients, they found a 35% decrease in NAR. Like the study of Oliveira,[103] they found a large portion of the sample subjectively felt they could breath better through the nose after the expansion.

While most authors used acoustic rhinometry to measure resistance, Basciftci et al used lateral and frontal cephalograms to measure nasopharyngeal area.[108] They also compared

rapid palatal expansion with surgically assisted rapid palatal expansion. They showed statistically significant increases in nasal cavity width following RPE and SARPE of 3.47mm and 2.93mm respectively.

While most reports show an increase in nasal volume as verified by acoustic rhinometry, some studies have been able to confirm this by CT.[109] Deeb et al. used a bone born expansion device to provide expansion after SARPE in a study of 16 patients. After a 6-month follow up, another CT was performed and showed an average increase in nasal volume of 5.1%.[109]

Where appropriate, some have even suggested maxillary expansion may be justified on the basis of treating oral respiration. While it has been shown that expansion can increase nasal breathing, approximately 33% of mouth breathers maintained oral respiration. This suggests maxillary expansion for breathing purposes alone is not justified.[110]

A recent unpublished study, as verified by CT, revealed a significant gain in the volume of the nasal fossae and nasopharynx.[111] A 9.88% increase in the volume of the nasal cavity was seen 6 months post SARPE with no significant increases in the volume of the oropharynx.[111]

2.8.0 Studying Sleep

2.8.1 History

It is important to note that sleep is a brain process.[112] While we know it is important that the body rests during sleep, it is the brain that does the sleeping. Since sleep is a brain process, its study involves studying brain activiy.[112]

An electroencephalogram (EEG) was pioneered in 1930 by Dr Hans Berger, an Austrian psychiatrist who performed the first human EEG and discovered the alpha wave disappeared when his subject passed from wakefulness into sleep. Another milestone came in 1937 with a publication by Loomis who summarized their data into "sleep stages." [113]

By 1953, the electro-oculographic activity was observed by Aserinsky and Kleitman to occur every 90 to minutes during stage B sleep.[114] These eye movements became later known as rapid eye movements (REMs).[112, 114]

The last of the major discoveries came in the late 1950's when Jouvet had described postural changes in cats during sleep. He then noticed complete muscle atonia during REM as recorded by electromyography (EMG).[115] Amazingly, this emerged as a third state of consciousness. The first occurs during the day when an individual is awake and the body muscles respond. The second occurs during the majority of sleep, where the brain and body are asleep and unresponsive. The third state of consciousness, as described by Jouvet, occurs during REM sleep, where an active brain is in an inactive body.[115]

Therefore by 1959, an EEG, EOG, and EMG were all routinely used and now part of a standard sleep protocol.

2.8.2 Purpose of Sleep

It is widely accepted that sleep is necessary to restore alertness and cognitive performance, yet the precise physiological mechanisms are not fully understood.[116] It is clear however that the sleep duration, or TST (total sleep time), has a profound effect on recuperation, but this relationship may not be linear.[117] While not linear, longer sleep duration will result in improved performance and alertness during subsequent wakefulness.[118, 119] Studies have

also shown that in addition to total sleep time, sleep continuity independently mediates the recuperative sleep processes.[120]

2.8.3 Sleep Stages

Sleep is divided into 2 main groups, REM and non-REM (NREM). NREM is made up of stages 1-4. Physiologic measures are what define the stages of sleep and wakefulness, and are determined by the polysomnograph, which is considered the "Gold Standard" for detecting abnormalities in sleep architecture.[121] The polysomnogragh measures brain, eye, and muscle activity. In the detection of other diseases and sleep disorders it can also measure: airflow at the nose and mouth, respiratory effort, oxygen saturation, electric activity of the heart and tibial muscle.

Wakefulness or stage 0 is characterized by having the eyes closed with alpha range EEG activity. This is normally low voltage mixed frequency (8-13 Hz). During wakefulness, muscle activity, as well as eye activity, can be present.

The disappearance of the alpha activity can mark the transition from wakefulness to stage 1.[112] The exception to this, is if the person is performing some sort of mental task or opens their eyes. Sleep onset is determined when alpha activity decreases to less than 50%. Sleep stage 2 is characterized by k complexes, delta complexes and sleep spindles.[112]

Stage 1 sleep is without alpha waves, is delta-less and with no sign of sleep spindles. Vertex sharp waves may be present. Low voltage mixed frequency theta waves (4-8 Hz) are the dominant rhythm. During this stage, which is also known as a shallow sleep, the individual can be easily aroused.[121]

Stage 2 sleep is characterized by sleep spindles, which are 12-14 Hz bursts of EEG waveforms of 1.5 second duration. K complexes also appear in stage 2, but the origin is unknown. There is less than 20% delta activity. There are no eye movements and muscular activity is decreased. The threshold for arousal is also augmented.[121]

Stage 3 is marked by slow waves (EEG delta activity 0.5-2 Hz), which occupy 20-50%. High amplitudes (>75uV) are seen. Sleep spindles are also present. There is decreased muscle activity and no eye movement.

Stages 3 and 4 are both known as deep sleep, or delta sleep, and the threshold for arousal is at its highest.

REM is scored when there is complete loss of muscle tonus but with rapid eye movements. The EEG shows low voltage, mixed frequency background activity. Saw-tooth theta waves may be present. During REM, there may also be some middle ear muscle activity; periorbital integrated potentials as well as erections.

2.9.0 Sleep Disordered Breathing

The American Academy of Sleep Medicine Task Force released a document in 1999 in an attempt to standardize definitions due to the large number disordered breathing events that can occur during sleep. The following are excerpts from this document. [122]

2.9.1 Arousal

An arousal is defined as a return of alpha or theta waves for three seconds or more. Electromyography (EMG) tone elevations are not required in NREM sleep but are required to score arousals in REM sleep.[123]

2.9.2 Apnea

Total airway obstruction despite inspiratory effort. This event should last ten seconds or longer. It is recorded as either a 50% decrease of any valid sleep measure or an observed oxygen desaturation of >3% or an arousal.

2.9.3 Hypoapnea

Partial airway obstruction despite inspiratory effort. This event should last ten seconds or longer. It is recorded as either a 50% decrease of any valid sleep measure or an observed oxygen desaturation of >3% or an arousal.

In routine clinical practice, it is not necessary to distinguish between apneas and hypopneas because they have the same effects and similar pathophysiology.[122]

2.9.4 Respiratory Effort-Related Arousal (RERA) event

This event must last ten seconds or longer. It is characterized by a sudden change in esophageal pressure and an arousal.

2.9.5 Central Sleep Apnea-Hypopnea Syndrome

This is an extremely uncommon condition characterized by recurrent apneas in the absence of upper airway obstruction. They can be divided in those who become hypercapnic and those who become hypocapnic/normocapnic. Normocapnic or hypocapnic forms are either idiopathic or seen in those with Cheyne-Stokes breathing.

2.9.6 Obstructive Sleep Apnea

A condition characterized by repetitive obstruction of the upper airway often resulting in oxygen desaturation and arousals from sleep. Symptoms include daytime fatigue, feeling unrefreshed from sleep, and poor concentration.

2.9.7 Relationship Between Expansion and Sleep

Since it has previously been shown that expansion may increase one's ability to breathe, one can extrapolate that some links exists between sleep disordered breathing and expansion.

In a study of 32 patients with moderate OSA, children received polysomnographic testing before and after treatments with both orthodontic expansion and adenotonsillectomy.[124] After expansion alone, the apnea-hypopnea index (AHI) was drastically reduced from 11.0 events per hour to 5.1 events per hour. After adenotonsillectomy, the AHI reduced from 5.1 events per hour to 0.94 events per hour.[124]

Another study that controlled for obesity as well as large tonsils tested the effects of expansion on obstructive sleep apnea (OSA). Thirty-one children who were shown to have OSA by polysomnographic testing received rapid palatal expansion as part of orthodontic treatment and had a final polysomnographic test 4 months after expansion. The AHI was reduced from 12 events per hours to less than one event per hour.[125]

Villa et al. also studied the effects of RPE on OSA in 14 patients in the early mixed dentition. [126] They found that most children switched from oral to nasal respiration and

experienced significant reduction in their AHI.[126] Other changes twelve months post expansion included a decrease in hypopnea index as well as fewer arousals.[126]

Sleep microstructure and arousals are other important components of sleep. Miano et al showed that children slept longer with a lower number of stage shifts 1 year post expansion, showing improved sleep architecture.[2]

Few studies exist that examine the effect of SARPE on sleep. Our study will show the long-term effects of SARPE on sleep, with a minimum of 3 years post expansion polysomnographic testing.

3. Materials and Methods

This project is a prospective non-controlled clinical study. The subjects will act as controls to themselves.

3.1 Inclusion Criteria

1. Patients that possessed a severe maxillary transverse deficiency.
2. Patients of advanced skeletal maturity for a rapid palatal expansion.
3. Patients in good health with no contraindication to surgery.
4. Patients who received a SARPE and are followed at the Orthodontic Clinic of the University of Montreal for complete orthodontic treatment.
5. Patients who passed multiple sleep studies before and after SARPE.

3.2 Exclusion Criteria

1. Patients whose health contraindicated surgery.
2. Previous history of orthodontic treatment.

3.3 Initial Sample Selection

The patients were selected during the screening sessions in the Orthodontic Department. Those that presented with the above inclusion criteria had conventional orthodontic records taken (cephalometric radiographs or CBCT, intra-oral and extraoral photos, study models). After a complete case analysis, and confirmation that a SARPE was necessary, the patient

was invited for a meeting where the research project was explained. If the patient accepted, the consent form was signed. The presence or absence of sleep-disordered breathing was not a prerequisite.

The patients were then booked for 2 nights in the sleep laboratory to record baseline figures (T0). After spending 2 nights in a sleep laboratory all patients received a bonded Hyrax appliance. The patients then underwent a SARPE and subsequent maxillary expansion. Six-twelve months after removal of the appliance, the patients passed two more consequent nights in the sleep laboratory to evaluate any changes in sleep-disordered breathing (T1).

3.4 Current Sample

This study began with all nine patients (3 male, 6 female) in active orthodontic treatment with full fixed appliances. These patients (average age 21, range 16-39) were deemed not to require a second stage surgery as part of the treatment plan. Their final results (T2) will be compared to previous post SARPE data (T1). The average treatment time in fixed appliances was 2.7 years (range 1.7-3.9y) and the average time post SARPE was 3.5 years (range 2.5-4.4 years)

3.5 Treatment Protocol

All patients were treated using conventional orthodontic techniques. Patients were treated with edgewise fixed upper and lower braces. Specific mechanics were used on a case-by-case basis. Cases that merited extraction had extractions performed at the request of the clinical demonstrator. Four out of the 9 patients had extractions.

3.6.1 Post Treatment Protocol

Once the case was deemed complete by the supervising clinician, post orthodontic records were taken including:

a) Intra oral and extra oral photographs

b) Alginate Impressions (Jeltrate©, Dentsply, York, PA USA) for study models

c) Panoramic, cephalometric, and anteroposterior radiographs

d) Alginate Impressions (Jeltrate©, Dentsply, York, PA USA) for a retention appliance

3.6.1 Post Treatment Sleep Study

Approximately 2-3 months after all fixed appliances had been removed, the patients were sent back to the sleep laboratory for a final night of recordings. There was no habitual night given the fact that this was the patient's 5th time returning to the sleep laboratory. The retention appliance was not worn during the sleep study night.

3.6.2 Sleep Laboratory Recordings

All patients were prepared in the usual manner by a sleep technician and the following were analyzed:

a. Electroencephalogram (EEG)

b. Mentalis Electromyogram (EMG)

c. Electroocculogram (EOG)

d. Right and left anterior tibial EMG

e. Electrocardiogramme (EKG)

f. Oxygen saturation (SaO2)

g. Breathing effort as measured by oral, nasal, thoracic and abdominal sensors

3.6.3 Sleep Analysis

The sleep analysis also consisted of the following measurements and were scored according to the standards set by the American Academy of Sleep Medicine:

a. The number and duration of hypoapneas and apneas
b. The number of arousals
c. The sleep duration
d. The sleep efficiency
e. The time spent in each sleep stage
f. The Sleep Latency time
g. The snoring index

3.7 Study Model Analysis

Once the study models were returned from the laboratory, the following measurements were made:

a. Intercanine distance for maxillary arch
b. Intercanine distance for the mandibular arch
c. Intermolar distance for the maxillary arch (central fossa)
d. Intermolar distance for the maxillary arch (palatal groove)
e. Intermolar distance for the mandibular arch (central groove)

3.8 Cephalometric Analysis

The anteroposterior cephalometric X-ray was analyzed according to the Rocky Mountain Analysis as proposed by Ricketts. For the purpose of analyzing transverse figures, the MMWD was used, which is known as the maxillo-mandibular width differential, as well as the Maxillary and Mandibular width. From these, the Maxillo-mandibular transverse differential index was determined.

The following anatomic landmarks were identified before calculating both right and left MMWD, as well as the maxillary and mandibular widths:

- Jugula right and left
- Antegonion right and left (AG and GA)
- Zygomatico-frontal suture (ZR and ZL)

The Jugula points were traced at the point of intersection of the tuberosity and zygommatic process. The distance between JR and JL is the Effective Maxillary Width.

The antegonial notches were located just inferior to the trihedral eminence. The distance between AG and GA is the Effective Mandibular Width.

A vertical line was drawn from each of ZR and ZL (right and left zygomatico-frontal sutures). The distance from this perpendicular line to JR and JL represented the Maxillo-Mandibular Width differential (MMWD- right and MMWD left).

The maxillomandibular width differential index, a calculation of transverse deficiency, is given by subtracting the theoretical index (Effective mandibular width-effective maxillary width) with the measured index (Effective mandibular width-effective maxillary width). The theoretical index in all cases was 19.6 since all were over the age of sixteen.

3.9 Statistical Analysis

All anteroposterior cephalometric measurements as well as study model measures were taken on 2 occasions by the author (PRF) in a randomized fashion. They were then measured a third time by an orthodontic staff member (NB). The following statistical tests were used to determine inter and intraevaluator reliablity: intraclass correlation, Dahlberg, and Bland Altman.

Data from the cephalometric and study model analysis was analysed using Shapiro-Wilk testing for normality. For all cephalometric differences between T1 and T2, that followed a normal distribution, a paired t-test was used. For all study model measurements between T1 and T2, that followed a normal distribution, a pair t-test was used.

Data from the sleep laboratory was analyzed using Shapiro-Wilk testing for normality. For data following a normal distribution, repeated measures ANOVA was used. For data that did not follow a normal distribution, the Friedman test was used.

4. Results

4.1 Inter and Intraoperator Reliability

All anteroposterior cephalometric radiographs were retraced by the author on 2 occasions at random. They were traced again by an orthodontic staff member. Three measures of repeatability were used: Bland Altman, intraclass coefficient (ICC), and Dahlberg. Results of these tests are presented in Table 1 and Table 2. These results show excellent repeatability and reliability. For the intraevaluator reliability, the ICC was close to 1, while Dahlberg below 0.5, and Bland-Altman range between 0.5 and 1.4. Similarly, for the interevaluator reliability, Bland-Altman ranged from 0.3-2.3, ICC was close to 1, and Dahlberg below 0.9. All measures were within the accepted norms for cephalometric evaluation.

Intraevaluator Reliability						
	Avg diff	**Min diff**	**Max diff**	**Bland-Altman**	**ICC**	**Dahlberg s**
MAXIC	0,113	-0,270	0,610	0,529	0,997	0,187
MAXIMF	-0,173	-1,030	0,360	0,892	0,990	0,315
MAXIMS	-0,058	-0,650	0,710	0,710	0,994	0,251
MDIC	-0,050	-0,590	0,720	0,639	0,978	0,226
MDIMF	0,070	-0,280	0,850	0,642	0,995	0,227
MDIMS	-0,003	-0,750	0,740	0,651	0,994	0,230
MMWDD	-0,183	-0,500	0,500	0,827	0,980	0,292
MMWDG	-0,017	-0,500	0,500	0,592	0,992	0,209
MAXW	0,033	-0,500	1,000	0,856	0,993	0,303
MANDW	-0,133	-1,000	1,000	1,033	0,995	0,365
INDEX	0,167	-1,000	1,500	1,483	0,991	0,524

Table 1. Intraevaluator reliability.

Interevaluator Reliability						
	Avg diff	**Min diff**	**Max diff**	**Bland-Altman**	**ICC**	**Dahlberg s**
MAXIC	0,283	-0,530	1,340	1,103	0,986	0,390
MAXIMF	0,401	-0,090	0,920	0,990	0,987	0,350
MAXIMS	-0,241	-1,750	1,440	1,338	0,978	0,473
MDICR	0,074	-0,890	0,590	0,729	0,972	0,258
MDIMF	0,452	-0,040	0,960	1,054	0,986	0,373
MDIMS	-0,055	-0,250	0,270	0,320	0,999	0,113
MMWDD	0,170	-0,800	1,000	0,983	0,974	0,347
MMWDG	0,110	-0,500	0,700	0,790	0,986	0,279
MAXW	-0,433	-1,500	0,800	1,592	0,979	0,563
MANDW	-0,067	-1,500	1,400	1,464	0,991	0,518
INDEX	-0,367	-2,000	2,300	2,387	0,978	0,844

Table 2. Interevaluator Reliability.

4.2 Cephalometric Results

All cephalometric results are presented in Table 3 and Table 4. The statistical analysis between T1 and T2 follows in Table 5.

The maxillo-mandibular width differential for both right and left sides were analyzed between T1 (post SARPE) and T2 (post removal of fixed appliances). No statistical difference was seen. The same non-statistically significant results were observed for the mandibular width as well as maxillo-mandibular width differential index.

The only statistically significant result observed was the reduction in effective maxillary width which was 1.23mm (p=0.026) (Table 5).

Patient	Maxillo-mandibular Width Differential Right		Maxillo-mandibular Width Differential Left		Effective Maxillary Width	
	Before	After	Before	After	Before	After
1	16	15.5	17	16.5	60.5	60
2	15.5	16	15	18	66	64.5
3	15.5	15	16	16	63.5	64
4	13.5	16.5	16	14	61	59.5
5	18	18.5	18	19	65	65
6	12	11	12.5	14.5	71.5	68.6
7	13.6	14.5	14.6	13.5	72.7	71
8	11.5	12	13	12.5	72	68.5
9	15	14.5	16.5	13	65	65

Table 3. MMWD Right, MMWD Left, Maxillary Width

See Table 5 for statistical analysis

Patient	Effective Mandibular Width		Maxillomandibular Width Differential Index	
	Before	After	Before	After
1	95	95	-14.9	-15.4
2	99	99	-13.4	-14.9
3	95.5	95.5	-12.4	-11.9
4	88	87.5	-7.4	-8.4
5	108	108	-23.4	-23.4
6	99	99	-7.9	-10.8
7	98	98	-5.7	-7.4
8	96	94	-4.4	-5.9
9	101	98	-16.4	-13.4

Table 4. Effective Mandibular Width and MMWD Index

See Table 5 for statistical analysis

Paired Samples Test				
				Sig. (2 tailed)
	Mean	Std. Deviation	Df	p value
MMWD R BEFORE - MMWD R AFTER	-0.3222	1.19	8	0.441
MMWD L BEFORE - MMWD L AFTER	0.1778	1.99	8	0.795
MAX W BEFORE - MAX W AFTER	**1.23**	**1.36**	**8**	**0.026***
MAND W BEFORE - MAND W AFTER	0.611	1.11	8	0.138
INDEX BEFORE - INDEX AFTER	0.6222	1.69	8	0.301

Table 5. Statistics for Cephalometric Results

* denotes statistical significance

4.3 Study Model Analysis

The intercanine, intermolar (fossa), and intermolar (grooves), widths were measured for both the maxilla and mandible (Table 6 and Table 7). The statistical analysis for the maxillary and mandibular widths is shown in table 8. All measurements for the maxilla had decreased between T1 (post SARPE) and T2 (post removal of fixed appliances). The intercanine distance decreased 4.14mm while the intermolar distance decreased 3.57mm. The differences were statistically significant. No significant changes were seen in any mandibular measurements.

	Maxilla					
	Intercanine		Intermolar (fossa)		Intermolar (groove)	
Patient	**Before**	**After**	**Before**	**After**	**Before**	**After**
1	36.3	33.19	45.27	43.27	36.08	31.87
2	35.5	35.31	43.4	42.23	34.8	31.13
3	38.75	34.23	49.76	46.99	42.19	37.54
4	37.5	32.09	45.8	41.89	36.6	32.91
5	43.02	34.98	49.22	44.01	38.64	34.65
6	37.8	33.74	49.8	43.08	39.8	32.81
7	38.7	36.13	49.2	47.9	41.8	38.07
8	42.9	36.09	50.6	49.8	41	39.01
9	39.1	36.59	51.7	43.42	42.2	33.74

Table 6. Intercanine and Intermolar Widths for Maxilla

See Table 8 for Statistical Analysis

	Mandible					
	Intercanine		Intermolar (fossa)		Intermolar (groove)	
Patient	**Before**	**After**	**Before**	**After**	**Before**	**After**
1	23.53	24.31	39.63	40.4	32.73	31.85
2	26	27.25	32.3	34.78	25	27.12
3	24.39	24.35	40.59	41.14	33.4	33.47
4	24.11	25.17	32.77	35.79	26.02	27.82
5	27.91	26.51	40.32	40.44	32.94	32.15
6	24.8	25.49	45.2	40.44	37.9	32.34
7	26.2	27.66	42.2	41.6	35.4	34.23
8	27.1	23.95	47.3	45.79	39.2	37.67
9	30.7	28.56	42.7	39.97	33.9	30.51

Table 7. Intercanine and Intermolar width for Mandible

See Table 8 for Statistical Analysis

Paired Samples Test			
			Sig. (2 tailed)
	Mean	Std. Deviation	p value
MAX IC BEFORE - MAX IC AFTER	**4.14**	**2.39**	**0.001***
MAX IMF BEFORE - MAX IMF AFTER	**3.57**	**2.70**	**0.004***
MAX IMG BEFORE - MAX IMG AFTER	**4.60**	**1.95**	**0.001***
MAN IC BEFORE - MAN IC AFTER	0.17	1.66	0.773
MAN IMF BEFORE - MAN IMF AFTER	0.30	2.45	0.727
MAN IMG BEFORE - MAN IMG AFTER	1.04	2.39	0.229

Table 8. Statistics for Study Model Results

* denotes statistical significance

4.4 Sleep Results

Sleep data are presented in the Table 9. Shapiro-Wilk testing for normality was performed. For data following a normal distribution, repeated ANOVA was used, and the mean is presented with the standard error of the mean. For data that did not follow a normal distribution, the Friedman test was used, and the median is presented with the minimum and maximum values. N2 represents the sleep night at T0 (pre SARPE), N4 represents the sleep night at T1 (post SARPE), and N5 represents the final sleep night after removal of the fixed appliances.

	Night 2 (N2)	Night 4 (N4)	Night 5 (N5)	p
Sleep duration (min)	454.6 ± 15.5	437.7 ± 14.0	403.6 ± 21.4	0.11
Sleep Efficiency (%)	96.0 (72.5-98.8)	96.7 (81.4-99.1)	90.4 (70.6-97.2)	0.045 (n2-n5 p=0.018)
Sleep Latency (min)	13.0 (4.0-68.7)	14.3 (5.0-77.7)	30.3 (3.3-89.3)	0.9
Number of Awakenings	24.9 ± 3.0	25.1 ± 4.1	32.0 ± 4.9	0.21
Awake (min)	18.3 (6.0-132.7)	15.7 (4.3-83.0)	47.7 (10.7-121.0)	0.045 (n2-n5 p=0.018)
Number of transitions AASM	203.0 ± 14.9	226.9 ± 15.3	189.0 ± 12.6	0.028 (n4-n5 p=0.009)
Number of transitions R&K	235.6 ± 16.1	270.4 ± 21.7	209.0 ± 13.6	0.007 (n2-n5 p=0.011 (n4-n5 p=0.005
Duration of Stage 1 (min)	23.5 ± 2.9	21.6 ± 3.7	32.8 ± 7.1	0.16
Duration of Stage 2 (min)	216.0 ± 16.6	213.9 ± 14.4	201.1 ± 9.5	0.66
Duration of Stage N3 (min)	115.1 ± 11.9	120.0 ± 17.5	92.5 ± 11.8	0.12
Duration of Stage REM (min)	100.1 ± 7.2	82.3 ± 6.0	77.1 ± 9.3	0.077
Duration of Stage1 (%)	5.3 ± 0.7	5.1 ± 1.0	8.2 ± 1.6	0.077 (n4-n5 p=0.019)

Duration of Stage 2 (%)	47.2 ± 2.7	49.0 ± 3.1	50.4 ± 2.4	0.56
Duration of Stage N3 (%)	25.6 ± 2.7	27.3 ± 3.7	22.7 ± 2.3	0.3
Duration of Stage REM (%)	21.9 ± 1.1	18.7 ± 1.1	18.7 ± 1.8	0.092
Number microarousals	60.0 (26-144)	73.0 (26-117)	60.0 (31-186)	0.72
Microarousal index (n/h)	8.4 (3.5-18.7)	11.5 (3.2-15.8)	8.2 (4.0-38.4)	0.72
Number of respiratory events	20.2 ± 5.5	17.0 ± 4.6	8.6 ± 2.2	0.11
Respiratory event index (n/h)	2.73 ± 0.72	2.35 ± 0.61	1.26 ± 0.32	0.093
Snoring Duration (%sleep)	3.0 (0-17.5)	2.1 (0-23.0)	0	0.016 (n2-n5 p=0.034 (n3-n5 p=0.034
Snoring number	14.0 (0-42)	2.0 (0-32)	0	0.013 (n2-n5 p=0.018)
Snoring index (n/h)	1.6 (0-7.2)	0.3 (0-5.0)	0	0.013 (n2-n5 p=0.018)

Table 9. Sleep Data and Statistics

The sleep efficiency was significantly reduced from 97% to 90%, between N4 and N5 (p=0.045). The time before falling asleep also increased but not significantly. The number of transitions was also significantly decreased between sleep stages from 227 to 189 (p=0.028). Light sleep (stage 1) was increased between N4 and N5 from 5.1% to 8.2% (p=0.019). All snoring measurements were significantly reduced between N4 and N5 from 2.1 events to zero (p=0.016). While not statistically significant, there was a reduction in the respiratory index (includes apneas and hypopneas)(p=0.093).

5. Discussion

5.1 Changes Observed on PA Cephalograms

The average maxillary width, as measured from jugale right to jugale left, decreased from 66.36 mm to 65.12 mm. It is worth noting that this 1.2 mm relapse was the average relapse over our 9 patients. In fact, only 6 of the nine patients experienced any relapse (averaging 2.2 mm) while 2 others showed no decrease in the interjugale distance. An unexpected finding was that one patient actually showed a 0.5mm increase. The stability of SARPE in this study as shown by the PA cephalograms at the time of debonding, is quite promising. In this study, our data is on average 3.5 years post SARPE. When comparing the initial effective maxillary width of 63.39 mm, a net skeletal widening of 1.73 mm occurred.

The clinical significance of this measurement needs to be brought into question. Jugale is a skeletal landmark, and even though skeletal changes are brought about, dental compensations occur in order to keep a crossbite corrected. A 1.2 mm skeletal change, on a landmark of questionable validity, is of low clinical significance, especially since a lateral translation of the maxillary shelves is not observed.[89 127]

The skeletal relapse after SARPE at the time of debonding was still higher than that reported by Byloff[85]. Although using different maxillary skeletal landmarks on the anterposterior cephalometric radiograph, they showed an average 0.24mm skeletal relapse, but stated there was extreme variation between patients.[85] This study also showed less initial skeletal expansion (1.2 mm compared to our 2.97 mm). On a percentage basis

however, the relapse of 42% (T0-T2) of the original skeletal expansion in our study is slightly higher than Byloff who showed 20% relapse.

5.2 Changes Observed on Study Models

The mandibular width showed no significant changes, as can be expected, since no major expansion occurred in this arch. In contrast, statistically significant relapse occurred in all width measurements for the maxillary arch after SARPE (T1) to post debonding (T2). The average change of maxillary intercanine width was 4.14mm. The intermolar widths as measured between the central fossae and groove, relapsed an average 3.57mm and 4.60mm respectively. On a percentage basis, between T0 and T2, 61% of the intercanine expansion relapsed and 45.5% of the intermolar distance relapsed.

The relapse data is slightly higher than that reported by Byloff.[85] In their study of 14 subjects, relapse at the molars and canines was 2.62mm and 0.9mm respectively which amounts to half the relapse that we observed. The retention protocol was 6 months after SARPE which was very similar in our study. Chamberland et al reported slightly lower relapse of 2.609mm in the canine region at debonding, which is slightly less than the 4.13 mm relapse that we had observed.[89] In the molar region, Chamberland found again slightly less relapse of 1.8mm compared to our 3.6mm.

These relapse figures should be interpreted with caution. While it is true that the same dental landmarks were used post-SARPE and post orthodontic appliance removal, it does not take into account the tooth's position in the arch, which can change after extraction. For example, if a posterior tooth moves mesially into an extraction space, then it will be translating into a narrower portion of the arch. This could be misinterpreted as relapse. Another reason to interpret the results with caution is regarding arch form and ectopic teeth. If canines for example were initially ectopic, and then moved into the arch with space

created from either SARPE or extraction, then they too would show a significantly lower distance. Again, this is not relapse but simply related to ectopia and arch form.

In this study, patients # 2, 5, 6, and 9 all had upper premolar extractions. It is therefore not surprising to see this significant amount of dental relapse since 44% of our sample had some form of mesial movement into a more narrow portion of the arch.

A very important note is that most articles in the literature do not detail the amount of overexpansion performed, if any. If one was to overexpand, and later let the maxillary arch become coordinated with the mandibular arch, one could interpret this as significant loss of maxillary width when in fact the large difference is simply due to overexpansion. Very few articles in the literature also give no long term SARPE stability results and most have an endpoint before removal of fixed appliance.[84, 86, 128] It is therefore difficult to interpret their results since no final arch coordination or seating of the occlusion in the final position has taken place. A universal consensus detailing an end point should be outlined in the literature so proper evaluation of the relapse can be evaluated. Alternatively, all cases could start expansion after the lower arch had been uprighted so that a final expansion endpoint can easily be determined.[91] In our study, upper and lower fixed orthodontic appliances were placed at the same time, so the end of activation of the expander depended on an evaluation of the maxillary expansion attained as well as the anticipated uprighting of mandibular teeth.

5.3 Changes in Sleep

The *sleep efficiency* decreased significantly from 96.7% to 90.4%. While this change is statistically significant, clinically, this change will not impact a patient. The *time awake*, also increased significantly, indicating that it took much longer for the patients to fall asleep after debonding. These unfavorable results can be explained by the fact that no habitual night was used for the final sleep night and that the patients hadn't become

accustomed to the new surroundings. It was decided that no habitual night was necessary given the fact that it was the patient's 5th time returning to the sleep laboratory.

The number of transitions, scored according to the American Academy of Sleep Medicine as well as Rechtschaffen and Kales both decreased significantly.[129, 130] This is a promising result indicating that patients are not going back and forth between sleep stages and therefore getting better rest.

All sleep stages had the same duration, with the exception of an increase in percentage *stage 1*. This means that patients spent an average of 3% more time in light sleep. Again, the new surroundings and the lack of a habituation night for the final testing may have contributed to this change.

Perhaps the most significant change observed was the decrease in *snoring duration, snoring number, and snoring indices*. Significant decreases were seen post SARPE as well as a decrease to zero after debonding. This effect can have quite significant effects on the health of an individual since snoring has been termed as the cardinal symptom for sleep disordered breathing.[131] Eliminating this negative variable, possibly as a result of decreasing upper airway resistance, will contribute to better sleep quality.

An important feature that was controlled for in this study was mandibular deficiency. Because this sample had a SARPE as the only surgical part of the treatment plan, no mandibular advancements were performed and only fixed appliance therapy was performed after fixed appliance therapy. Therefore, we were confident in controlling the possible contributor of mandibular retrognathia to respiratory events.

5.4 Limitations

One of the greatest limitations in this study was the small sample size. While we were able to show statistically significant results for some sleep parameters as well as skeletal and dental changes, more changes could potentially have been seen with a larger sample size.

Another limitation was the fact that our sample contained a mixture of extraction and non-extraction cases. Ideally, in order to control for anchorage loss, which could be misinterpreted as relapse in the transverse dimension, only non-extraction cases should have made up the sample. This unfortunately would have left a sample size of only 5 patients.

5.5 Future Research

This study demonstrated positive relationship between SARPE and sleep quality. It would be interesting to see if these positive results were maintained. A long-term follow up on these patients would contribute greatly to the understanding of SARPE.

Future research could also be aimed at evaluating the effects of SARPE on patients that currently have OSA or other respiratory issues.

6. Conclusion

1) SARPE is a stable procedure where the maxillary skeletal relapse was minimal and clinically insignificant.
2) Significant transverse maxillary gains were maintained 3.5 years post SARPE.
3) Post SARPE dental relapse was clinically significant. Even in the presence of dental relapse, all patients finished with a buccal occlusion that meets the current standard of care.
4) Positive changes in sleep occurred after SARPE. Significant reductions in the number of sleep stage transitions occurred as well as a drastic reduction in the snoring index.

7. Article

SARPE and Sleep: Effects on healthy patients.

Abstract

Introduction: Orthopedic expansion appears to have several positive effects on respiration as well as sleep quality, but a lack of studies examine these findings using SARPE on skeletally mature individuals. The aim of this study was to evaluate SARPE stability as well as its effect of sleep disordered breathing after completing full fixed orthodontics. **Methods:** Nine patients requiring SARPE underwent polysomnographic testing in a sleep laboratory before SARPE (T0), after SARPE (T1), and after removal of full fixed appliances (T2). Study models and anteroposterior cephalometric radiographs were also taken at the 3 time points. **Results:** Study model analysis showed significant relapse for maxillary intermolar and intercanine widths. Anteroposterior cephalometric relapse results were significant only for effective maxillary width. There were no significant changes in any sleep stages, however a dramatic decrease in the snoring index was observed. **Conclusions:** Although statistically significant relapse was observed on study models and anteroposterior cephalometric radiographs, the dental relapse appears to be more clinically significant than the skeletal relapse. SARPE appears to have an effect on reducing the snoring index and reducing transitions between sleep stages.

Introduction

As orthodontists, orthopedic expansion of the maxillary arch is a fairly routine and simple procedure. The purpose of expansion is to correct a maxillary transverse deficiency which

can be present in individuals with: a narrow maxillary arch, uni or bilateral crossbite (Figure 1), chin asymmetry with or without a functional shift, and excessive curves of Monson and Wilson.[23]

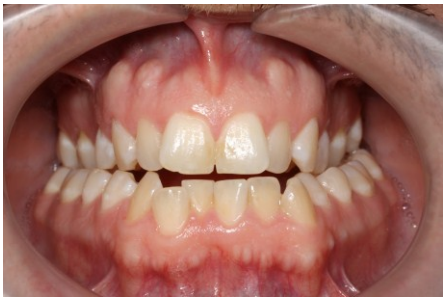

Figure 1. Severe Maxillary Transverse Deficiency.

As the patients age, the facial sutures become more interdigitated, especially after puberty.[31] This includes the palatal suture as well as the circumaxillary sutures and midface sutures.[45, 73, 132] As a general rule, non surgical treatment of adolescents with a transverse discrepancy should be attempted before age 15 using a rapid palatal expander (Hyrax or Haas type). [4]After this, and depending on the degree of transverse deficiency, surgical treatment may be the only option to correct this insufficiency.[3]

If one were to attempt conventional orthopedic expansion in a skeletally mature individual, a myriad of problems can occur. It would be normal to see extreme bending of the alveolus, dental tipping, and minimal maxillary expansion.[55-57] Other problems despite overcorrection include relapse, periodontal defects, and malocclusion.[58] [59] [60]Skeletally mature individuals are also subject to buccal root resorption, fenestration of the buccal cortex, pain, and palatal tissue necrosis [3, 61].

In cases where the transverse discrepancy is less than 5 mm, orthodontics alone can camouflage the discrepancy and surgery is not necessary.[55] However, for a MTD greater than 5 mm, SARPE is the recommended option.

There is no agreement on the exact surgical technique.[3] Some authors leave the pterygoid plates intact[63-65], while several authors make dysjunction of the pterygoid plates part of their surgical procedure. [56] The rational for dysjunction is to reduce resistance and allow for a more parallel expansion.[62] Other authors believe that less resistance contributes to less periodontal injury, tipping, as well as relapse.[62, 67, 68]

Several studies have reported varying degrees of SARPE stability.[66, 67, 74, 83-88] A major reason for the differences found in the literature is due to the varying treatment protocols as well as retention periods. [86, 89, 90] It is the clinical preference of some clinicians to leave the expansion appliance cemented until 6 months after the SARPE[91] while others prefer to remove the appliance and replace it with a removable acrylic appliance to maintain the transverse gain.

Byloff showed second premolar relapse of 28%, while the most significant relapse was observed at the first molars (36%).[85] They also demonstrated significant buccal angular changes which almost completely relapsed (from 9.6 degrees to 9.4 degrees).[85] Northway showed a mean increase of 3.3 mm at the maxillary canines as well as 5.5 mm at the maxillary molars with the follow up being approximately 2 years after orthodontic therapy.[67] Relapse was shown to be between 5-33%.[67]

In a 2.4 year follow up, once fixed appliances were removed, Bays showed that in 19 adults aged 30, relapse from SARPE in the molar and canine regions was 7.7% and 8.8% respectively.[83] Recently, Chamberland and Proffit have shown similar results with a larger sample of patients, in whom all pterygoid plates were separated.[89] This study had a large sample size of 39 patients with 2 years post orthodontic treatment follow up. They showed, after debonding, that the only clinically significant relapse occurred at the maxillary first molars, where 17% or 1.1mm of relapse occurred.

SARPE and sleep

Given the fact that the maxillary bones form over half of the structure of the nasal cavity, it is not surprising that the maxillary bones by expansion would affect the anatomy and physiology of the nose.[103]

The nasal valves are the minimal cross-sectional areas of the nose, which give the greatest resistance to nasal airflow. Maxillary expansion causes a pyramidal type expansion with the greatest expansion at the level of the incisors, which are just inferior to the nasal valves.[103] The widening of the maxillary shelves also gives rise to a widening of the lateral walls of the nose, thus increasing the total nasal volume. [107]

In a study of 32 patients with moderate obstructive sleep apnea(OSA), children received polysomnographic testing before and after treatments with both orthodontic expansion and adenotonsillectomy.[124] After expansion alone, the apnea-hypopnea index (AHI) was drastically reduced from 11.0 events per hours to 5.1 events per hours. After adenotonsillectomy, the AHI reduced from 5.1 events per hour to 0.94 events per hour.[124]

Another study that controlled for obesity as well as large tonsils tested the effects of expansion on obstructive sleep apnea (OSA). Thirty-one children who were shown to have OSA by polysomnographic testing received rapid palatal expansion as part of orthodontic treatment and had a final polysomnographic test 4 months after expansion. The AHI was reduced from 12 events per hour to less than one event per hour.[125]

Villa et al. also studied the effects of RPE on OSA in 14 patients in the early mixed dentition. [126] They found that most children switched from oral to nasal respiration and experienced significant reduction in their AHI.[126] Other changes twelve months post expansion included a decrease in hypopnea index as well as fewer arousals.[126]

Sleep microstructure and arousals are other important components to sleep. Miano et al showed that children slept longer with a lower number stage shifts 1 year post expansion showing improved sleep architecture.[2]

While the effect of expansion on sleep has been studied in young patients, to the authors' knowledge, no such study has been conducted using SARPE on skeletally mature individuals. The aim of this study was twofold: 1) to study skeletal and dental relapse with SARPE after removal of fixed appliances and 2) to study the effects of SARPE on sleep stages and events in healthy young adults.

Materials and Methods

Nine patients from a university outpatient orthodontic clinic (average age 21, range 16-39) with a moderate to severe maxillary transverse deficiency, who had already undergone polysomnographic testing in a sleep laboratory before and after SARPE made up the sample of this study. Other inclusion criteria included: no requirement for second stage orthognathic surgery, good health, no sleep disturbances, and no upper airway abnormalities as verified by an otolaryngologist.

Upon completion of orthodontic treatment and removal of fixed appliances (2.7 years, range 1.7-3.9 years), patients were sent for a final night in a sleep laboratory for polysomnographic testing and scored according to the standards set by the American Academy of Sleep Medicine. [130]

Study models and PA cephalograms were taken after SARPE and after debonding to evaluate stability and relapse (average post SARPE 3.5 years, range 2.5-4.4years). The anteroposterior cephalograms were analyzed according to Ricketts.[22] Effective maxillary and mandibular widths, maxillomandibular width differentials, and maxillomandibular width differential indices were calculated. Study models were evaluated using a digital

caliper. Intercanine widths, intermolar widths (central fossa) and palatal groove were all measured for both arches.

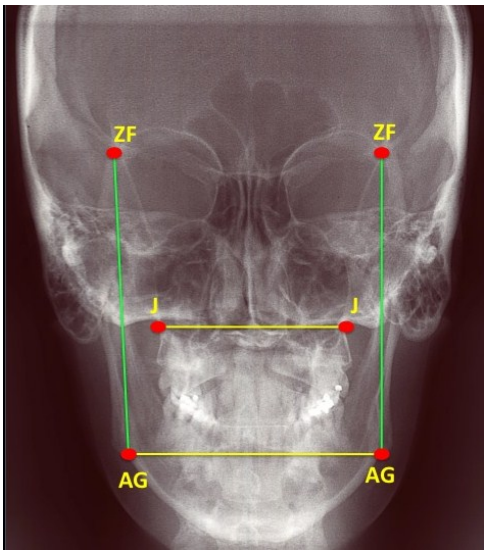

Figure 2 Anteroposterior Cephalometric Radiograph

The post SARPE data (T1) was then compared with data from after removal of fixed appliances (T2).

Results

Inter and Intraoperator reliability

All anteroposterior cephalometric radiographs were retraced by the author on two occasions at random. They were traced again by an orthodontic staff member. Three measures of repeatability were used: Bland Altman, intraclass coefficient, and Dahlberg's formula. Ninety five percent of the differences between the 2 measurements were within the acceptable limits of 1.1mm. The intraclass correlation values are all close to 1.0 showing excellent consistency of measurements. The Dahlberg results were all within acceptable limits.

Post SARPE to Post Bracket Removal Data

Cephalometrics

The only statistically significant result observed was the change in effective maxillary width (p=0.026). The average reduction in interjugale distance was 1.23mm. Table 1 shows statistics for all anterposterior cephalometric measurements.

Paired Samples Test				
				Sig. (2 tailed)
	Mean	Std. Deviation	Df	p value
MMWD R BEFORE - MMWD R AFTER	-0.3222	1.19	8	0.441
MMWD L BEFORE - MMWD L AFTER	0.1778	1.99	8	0.795
MAX W BEFORE - MAX W AFTER	**1.23**	**1.36**	**8**	**0.026***
MAND W BEFORE - MAND W AFTER	0.611	1.11	8	0.138
INDEX BEFORE - INDEX AFTER	0.6222	1.69	8	0.301

Table 1. Statistics for Cephalometric Results.

*denotes statistical significance

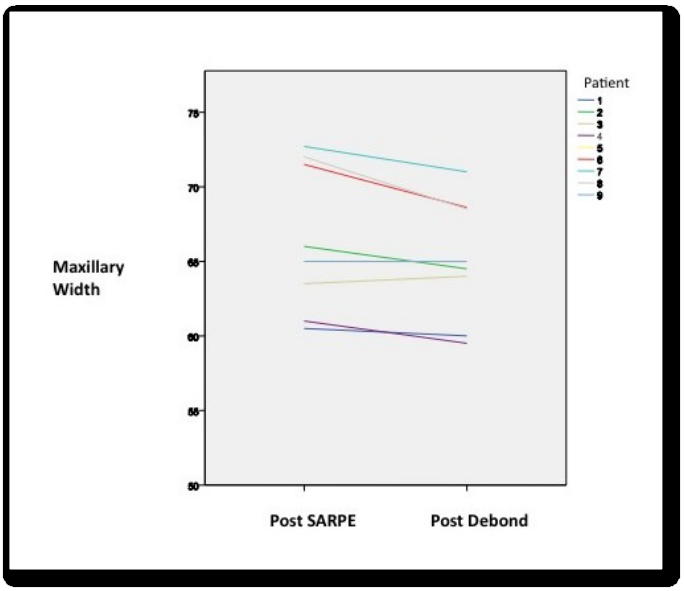

Figure 3 Effective Maxillary Width (Jugale to Jugale)

Study Model Analysis

All measurements for the maxilla width had decreased and were statistically significant.

Paired Samples Test			
			Sig. (2 tailed)
	Mean	Std. Deviation	p value
MAX IC BEFORE - MAX IC AFTER	**4.14**	**2.39**	**0.001***
MAX IMF BEFORE - MAX IMF AFTER	**3.57**	**2.70**	**0.004***
MAX IMG BEFORE - MAX IMG AFTER	**4.60**	**1.95**	**0.001***
MAN IC BEFORE - MAN IC AFTER	0.17	1.66	0.773
MAN IMF BEFORE - MAN IMF AFTER	0.30	2.45	0.727

MAN IMG BEFORE - MAN IMG AFTER	1.04	2.39	0.229

Table 2. Statistics for Study Model Results.

* denotes statistical significance

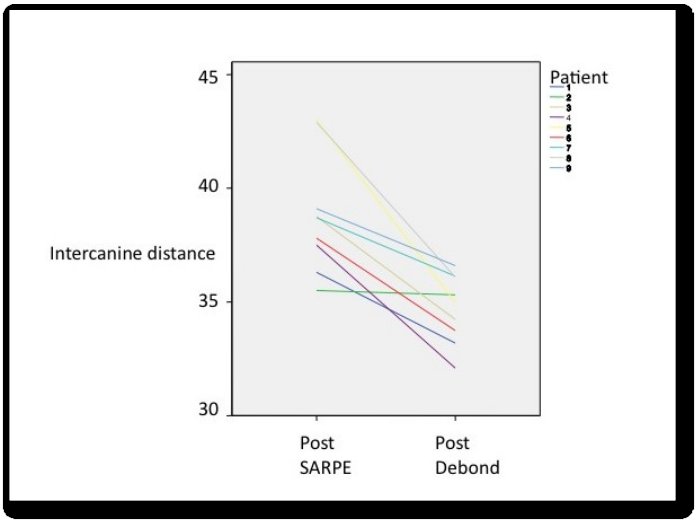

Figure 4. Intercanine Distance

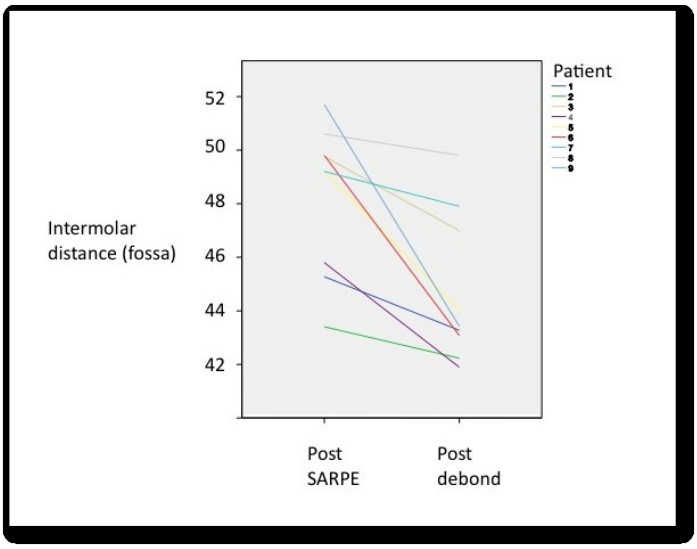

Figure 5. Intermolar changes.

Sleep Results

Sleep data are presented in the table below. Shapiro-Wilk testing for normality was performed. For data following a normal distribution, repeated ANOVA was used, and the mean is presented with the standard error of the mean. For data that did not follow a normal distribution, the Friedman test was used, and the median is presented with the minimum and maximum values.

	Night 2 (N2)	Night 4 (N4)	Night 5 (N5)	p
Sleep duration (min)	454.6 ± 15.5	437.7 ± 14.0	403.6 ± 21.4	0.11
Sleep Efficiency (%)	96.0 (72.5-98.8)	96.7 (81.4-99.1)	90.4 (70.6-97.2)	0.045 (n2-n5 p=0.018)
Sleep Latency (min)	13.0 (4.0-68.7)	14.3 (5.0-77.7)	30.3 (3.3-89.3)	0.9
Number of Awakenings	24.9 ± 3.0	25.1 ± 4.1	32.0 ± 4.9	0.21
Awake (min)	18.3 (6.0-132.7)	15.7 (4.3-83.0)	47.7 (10.7-121.0)	0.045 (n2-n5 p=0.018)
Number of transitions AASM	203.0 ± 14.9	226.9 ± 15.3	189.0 ± 12.6	0.028 (n4-n5 p=0.009)
Number of transitions R&K	235.6 ± 16.1	270.4 ± 21.7	209.0 ± 13.6	0.007 (n2-n5 p=0.011 (n4-n5 p=0.005
Duration of Stage 1 (min)	23.5 ± 2.9	21.6 ± 3.7	32.8 ± 7.1	0.16
Duration of Stage 2 (min)	216.0 ± 16.6	213.9 ± 14.4	201.1 ± 9.5	0.66
Duration of Stage N3 (min)	115.1 ± 11.9	120.0 ± 17.5	92.5 ± 11.8	0.12
Duration of Stage REM (min)	100.1 ± 7.2	82.3 ± 6.0	77.1 ± 9.3	0.077
Duration of Stage1 (%)	5.3 ± 0.7	5.1 ± 1.0	8.2 ± 1.6	0.077

				(n4-n5 p=0.019)
Duration of Stage 2 (%)	47.2 ± 2.7	49.0 ± 3.1	50.4 ± 2.4	0.56
Duration of Stage N3 (%)	25.6 ± 2.7	27.3 ± 3.7	22.7 ± 2.3	0.3
Duration of Stage REM (%)	21.9 ± 1.1	18.7 ± 1.1	18.7 ± 1.8	0.092
Number microarousals	60.0 (26-144)	73.0 (26-117)	60.0 (31-186)	0.72
Microarousal index (n/h)	8.4 (3.5-18.7)	11.5 (3.2-15.8)	8.2 (4.0-38.4)	0.72
Number of respiratory events	20.2 ± 5.5	17.0 ± 4.6	8.6 ± 2.2	0.11
Respiratory event index (n/h)	2.73 ± 0.72	2.35 ± 0.61	1.26 ± 0.32	0.093
Snoring Duration (%sleep)	3.0 (0-17.5)	2.1 (0-23.0)	0	0.016 (n2-n5 p=0.034 (n3-n5 p=0.034
Snoring number	14.0 (0-42)	2.0 (0-32)	0	0.013 (n2-n5 p=0.018)
Snoring index (n/h)	1.6 (0-7.2)	0.3 (0-5.0)	0	0.013 (n2-n5 p=0.018)

Table 3. Sleep Data and Statistics

The sleep efficiency was significantly reduced between N4 and N5. The time before falling asleep was also increased significantly. The number of transitions was also significantly decreased between sleep stages. Light sleep (stage 1) was increased between N4 and N5. All snoring measurements were significantly reduced between N4 and N5.

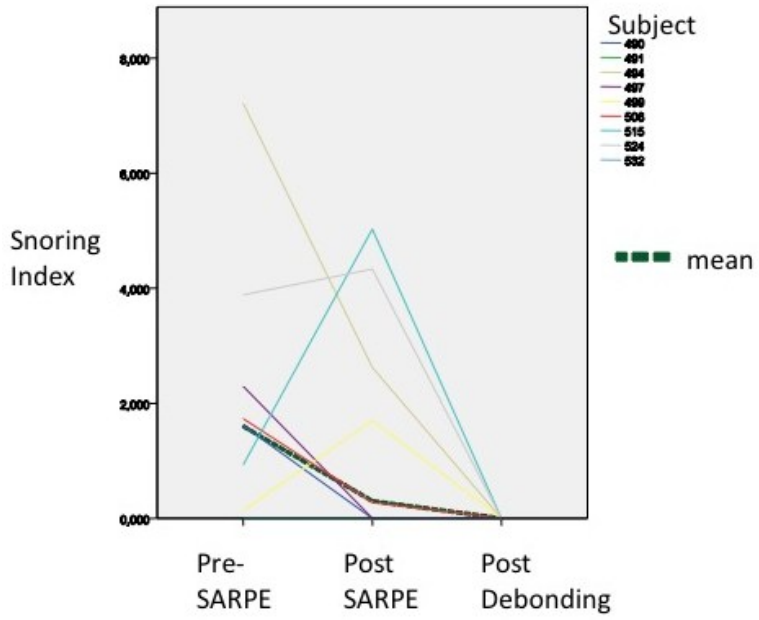

Figure 6 Snoring Index

Discussion

Change observed on PA Cephalograms

The average maxillary width, as measured from jugale right to jugale left, decreased from 66.4mm to 65.1mm, between T1 (post SARPE) and T2 (post removal of fixed ppliances). It is worth noting that this was the average relapse over our 9 patients. In fact, only 6 of the nine patients experienced a relapse of approximately 2.2 mm, while 2 others showed no decrease in the interjugale distance. An unexpected finding was that one patient actually showed a 0.5mm increase. Therefore, according to our observation, we can say that this study found 2.2 mm of skeletal relapse in approximately 56% of the patients. The stability of SARPE in this study as shown by the PA cephalograms at the time of debonding, is quite promising. In this study, our data is on average 3.5 years post SARPE. Comparing the post treatment to initial effective maxillary width of 63.39 mm shows a net skeletal widening of 1.73 mm.

The clinical significance of this measurement needs to be brought into question. Jugale is a skeletal landmark, and even though skeletal changes are brought about, dental compensations occur in order to keep a crossbite corrected. A 2.2 mm skeletal change, on a landmark of questionable validity, is of low clinical significance, especially since a lateral translation of the maxillary shelves is not observed.[89 127]

The skeletal relapse after SARPE to the time of debonding was still higher than that reported by Byloff[85]. Although different maxillary skeletal landmarks were used on the anterposterior cephalometric radiograph, they showed an average 0.25mm skeletal relapse, but stated there was extreme variation between patients.[85] This study also showed less initial skeletal expansion of 1.2 mm compared to our 2.97 mm. On a percentage basis however, the relapse of 42% (T0-T2) of the original skeletal expansion in our study is slightly higher than Byloff who showed 20% relapse.

Changes observed on study models

The mandibular width showed no significant changes as can be expected since no major expansion occurred in this arch. In contrast, statistically significant relapse occurred in all width measurements for the maxillary arch. The average change in maxillary intercanine width was 4.13mm. The intermolar widths as measured between the central fossae and groove relapsed an average of 3.57mm and 4.60mm respectively.

The relapse data is slightly higher than that reported by Byloff.[85] In their study of 14 subjects, relapse at the molars and canines was 2.62mm and 0.9mm respectively which amounts to half the relapse that was observed. The retention protocol was 6 months after SARPE which was very similar to our study. Chamberland et al reported slightly higher relapse of 2.609 mm in the canine at debonding, which is slightly less than the 4.13 mm

relapse that we had observed.[89] In the molar region, Chamberland found again slightly less relapse of 1.8mm compared to our 3.6mm.

These relapse figures should be interpreted with caution. While it is true the same dental landmarks were used post-SARPE and post orthodontic appliance removal, it does not take into account the tooth's position in the arch, which can change after extraction. For example, if a posterior tooth moves mesially into an extraction space, then it will be translating into a narrower portion of the arch. This could be misinterpreted as relapse. Another reason to interpret the results with caution is regarding arch form and ectopic teeth. If canines for example were initially ectopic, and then moved into the arch with space created from either SARPE or extraction, then they too would show a significantly lower distance. Again, this is not relapse, but simply related to ectopia and arch form.

A very important note is that most articles in the literature do not detail the amount of overexpansion performed, if any. If one was to overexpand, and later let the maxillary arch become coordinated with the mandibular arch, one could interpret this as significant loss of maxillary width when in fact the large difference is simply due to overexpansion. Very few articles in the literature also give no long term SARPE stability results and most have an endpoint before removal of fixed appliance.[84, 86, 128] It is therefore difficult to interpret their results since no final arch coordination or seating of the occlusion in the final position has taken place. A universal consensus detailing an end point should be outlined in the literature so proper evaluation of the relapse can be evaluated. Alternatively, all cases could start expansion after the lower arch had been uprighted so that a final expansion endpoint can easily be determined.[91] In our study, upper and lower fixed orthodontic appliances were placed at the same time, so the end activation of the expander depended on an evaluation of the maxillary expansion attained as well as the anticipated uprighting of mandibular teeth.

Changes in Sleep

The *sleep efficiency* decreased significantly from 96.7% to 90.4. While this change is statistically significant, clinically, this change will not impact a patient. The *time awake*, also increased significantly, indicating that it took much longer for the patients to fall asleep after debonding. These unfavorable results can be explained by the fact that no habitual night was used for the final sleep night and that the patients hadn't become accustomed to the new surroundings.

The number of transitions, scored according to the American Academy of Sleep Medicine as well as Rechtschaffen and Kales both decreased significantly.[129, 130] This is a promising result indicating that patients are not going back and forth between sleep stages and therefore getting better rest.

All sleep stages had the same duration, with the exception of an increase in percentage *stage 1*. This means that patients spent an average of 3% more time in light sleep. Again, the new surroundings and lack of habitual night for the final testing may have contributed to this change.

Perhaps the most significant change observed was the decrease in *snoring duration, snoring number, and snoring indices*. Significant decreases were seen post SARPE as well as a decrease to zero after debonding. This effect can have quite significant effects on the health of an individual since snoring has been termed as the cardinal symptom for sleep disordered breathing.[131] Eliminating this negative variable, possibly as a result of decreasing upper airway resistance, will contribute to a better sleep quality.

An important feature that was controlled for in this study was mandibular deficiency. Because this sample had a SARPE as the only surgical part of the treatment plan, no mandibular advancements were performed and only fixed appliance therapy was performed after SARPE. Therefore, we were confident in controlling the possible contributor of mandibular retrognathia to respiratory events.

Conclusion

1) SARPE is a stable procedure where the maxillary skeletal relapse was minimal and clinically insignificant.
2) Significant transverse maxillary gains were maintained 3.5 years post SARPE.
3) Post SARPE dental relapse was clinically significant. Even in the presence of dental relapse, all patients finished with a buccal occlusion that meets the current standard of care.
4) Positive changes in sleep occurred after SARPE. Significant reductions in the number of sleep stage transitions occurred as well as a drastic reduction in the snoring index.

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