Université de Montréal

OROFACIAL MORPHOLOGIC FEATURES OF SLEEP-BRUXERS VERSUS NON-BRUXERS: A CONTROLLED MORPHOLOGICAL AND SLEEP LABORATORY STUDY

par

Cristina Iafrancesco

Département de santé buccale Faculté de médecine dentaire

Mémoire présenté à la Faculté des études supérieures en vue de l'obtention du grade de Maître és sciences (M.Sc.) en médecine dentaire, option orthodontie

Copyright Février, 1999



2011. 27+0.6

Avendé de Nordeni

WU 5

058

1999

ORGENCIAL MORPHOLOGIC YEATHRES OF

V.002

mg

satemental missis i

Department de sante buccale

Memine présente à la facul é des cinites a mémor es en vue la l'édiction du grade de Malive es essences (M.Sg.) est médecant dentants, spison ormodostice

wight Permis 1819



Université de Montréal

Faculté des études supérieures

Ce mémoire intitulé:

OROFACIAL MORPHOLOGIC FEATURES OF SLEEP-BRUXERS VERSUS NON-BRUXERS: A CONTROLLED MORPHOLOGICAL AND SLEEP LABORATORY STUDY

présenté par:

Cristina Iafrancesco

a été évalué par un jury composé des personnes suivantes:

Président du jury: D^r Arto Demirjian
Directeur de recherche et co-directeur de recherche:
D^r Gilles Lavigne et D^r Jack Turkewicz
Membre du jury: D^r Oleg Kopytov

Mémoire accepté le: 99,04.16

SUMMARY

The object of this research project was to further identify if any orofacial morphological variables could characterize sleep bruxism subjects. Sleep bruxism is defined in the literature as a stereotyped movement disorder characterized by nonfunctional grinding or clenching of teeth during sleep, with approximately 85 % to 90 % of the population grinding their teeth to some degree during their lifetime. Of these, there is a deterioration into a pathological phenomenon for about 5 % of the population.

Following confirmation, or not, of the original clinical diagnosis of "bruxism-grinding" by sleep laboratory recordings, post hoc analysis of dento-facial morphological parameters were done between sleep-bruxer subjects (SBs) and non-bruxers. The hypothesis was that SBs have a different orofacial morphology than age-gender matched non-bruxers. The null hypothesis was that there is no significant difference in the orofacial morphology between groups.

For this study a total of 20 subjects, 10 SBs and 10 controls were invited to participate. All subjects had previously submitted for polysomnographic recordings at the Centre d'étude sur le sommeil of the

Hôpital du Sacré-Coeur. This is unique to this study, no other related publication has ever used polysomnographic recordings to differentially classify the subjects into their respective groups. Published studies relied on self report of grinding or tooth wear patterns to classify the subjects into their respective groups.

Once classified into either the SBs group or the control group, all subjects then underwent the following procedures: thorough intra-oral examination, intra-oral and extra-oral photographs, alginate impressions for models of their teeth (subsequently poured into orthodontic plaster), and a lateral cephalometric head radiograph.

The variables being studied included number of teeth, overbite, overjet, Angle classification, amount and direction of any slide from centric relation to centric occlusion, occlusal scheme present, lower anterior crowding, dental measures (from the dental casts), and skeletal measures (from the cephalogram).

Reliability of measures were estimated using Intraclass Correlation Coefficients (ICC) on all cephalometric measurements of interest: the results

revealed no significant intra-tracer, nor any significant inter-tracer differences (ICC ≥ 0.86572).

For most variables in the study, the data was normally distributed. The variables were assessed with the use of descriptive statistics, including the mean and standard deviation. Using the Two-Sample T- test, no statistically significant differences ($p \le 0.01$) between the SBs and the control groups were found for any of the variables.

For the data that was not normally distributed, such as Angle canine classification, Angle molar classification, occlusal scheme, and determination of lower anterior crowding, either the Pearson Chi-Square test or the Yates' Corrected Chi-Square test was used. No statistically significance differences $(p \le 0.01)$ were observed between the SBs and control groups for these variables.

In conclusion, after clinical examination and polysomnographic assessment of bruxism/grinding status, none of the oro-morphological variables seem to further discriminate the SBs from control subjects. Since this study looked at changes in dento-facial morphology as the effect and not the

cause of SB, perhaps the human masticatory system has a self-adaptive mechanism whereby it can withstand parafunctional activities, before irreversible morphological changes take place. However, our study did not include severe tooth wear bruxism subjects with loss of vertical dimension, which may induce changes in morphology over time, secondary to the parafunctional activity. The expected change in morphology would not be major, only subtle changes would be expected.

Further investigations of these hypotheses would be warranted with the following extensions to our study:

- designing a longitudinal study between low grade and severe tooth wear bruxism subjects
- including a larger sample size (as expected, our calculations showed our sample size may have been too small to show statistically sound differences between the SBs and control groups for certain variables)
- having an independent, naïve to study, objective, and highly trained staff person to measure all data in blind manner
- have the polysomnographic recordings done immediately prior to orofacial data collection.

RÉSUMÉ

Ce projet de recherche est une étude comparative des morphologies orofaciales de sujets symptomatiques (bruxeurs nocturnes) et de sujets asymptomatiques sélectionnés en laboratoire de sommeil. L'objectif de ce projet de recherche était d'identifier s'il y avait des variables morphologiques orofaciales qui pourraient caractériser les sujets symptomatiques.

Le bruxisme nocturne est défini comme un désordre de mouvement stéréo-typique caractérisé par un grincement ou serrement non-fonctionel des dents durant le sommeil. Approximativement 85 % à 90 % de la population grince leurs dents à un certain degré durant leur vie. Il y a 5 % de la population qui subit une détérioration de cela à un phénomène pathologique.

Après confirmation, ou non, par les données polysomnographiques du laboratoire de sommeil, du diagnostique clinique original de bruxisme nocturne, une analyse post-hoc des paramètres morphologiques dento-facials a été faites entre les sujets bruxeurs nocturnes et les sujets asymptomatiques. L'hypothèse était que les sujets bruxeurs nocturnes avaient une morphologie orofaciale différente des sujets asymptomatiques (appariés selon l'âge et le sexe). L'hypothèse nulle était qu'il n'aura pas de différences significatives

dans la morphologie orofaciale des sujets bruxeurs nocturnes et les sujets asymptomatiques.

Pour cette étude, un total de 20 sujets, 10 sujets bruxeurs nocturnes et 10 sujets asymptomatiques, ont été invité à participer. Tous les sujets se sont présentés préalablement pour un recueil de données polysomnographiques au Centre d'étude sur le sommeil à 1'Hôpital du Sacré-Coeur. Cela est unique à cette étude; pas d'autre publication (lié à ce sujet) a utilisé des données polysomnographiques d'un laboratoire de sommeil pour classifier les sujets différentiellement (soit en groupe des bruxeurs nocturnes ou en groupe des contrôles asymptomatiques). Les études publiées dans la littérature ont utilisé une méthodologie plus subjective, le reportage du bruxisme par le sujet ou le patron d'usure de dents, pour classifier leurs sujets soit dans le groupe des bruxeurs ou dans le groupe des contrôles asymptomatiques.

Une fois classifié, soit en groupe des bruxeurs nocturnes ou en groupe des contrôles, tous les sujets ont eu ces procédures : examen intra-oral complet, photographies intra-orales et extra-orales, empreintes en alginate des dents (coulées en plâtre orthodontique), ainsi qu'une radiographie céphalométrique latérale de la tête.

Les variables étudiées dans cette étude étaient le nombre de dents, surplomb vertical, surplomb horizontal, classification d'occlusion selon Angle, montant et direction de glissement de relation centrique à relation d'occlusion, schéma d'occlusion, le montant de chevauchement présent en la région antérieure inférieure, mesures dentaires (prises des modèles dentaires en plâtre), et les mesures céphalométriques.

Des statistiques de fiabilité, utilisant l'Intra Class Correlation Coefficients (ICC), ont été faites sur toutes les mesures céphalométriques d'intérêt. Les résultats démontrent aucune différence significative intra-traceur ou inter-traceur (ICC ≥ 0.86572).

Pour la majorité des variables, puisque les données étaient distribuée normalement, elles ont été étudiées à l'aide des statistiques descriptives, telles que la moyenne et la déviation standard. A l'aide des statistiques de type Two-Sample T-test, pas de différences significatives ($p \le 0.01$) ont été trouvées entre les sujets bruxeurs et les sujets asymtomatiques pour aucune des variables.

Des fréquences ont été calculées pour ces variables: classification d'occlusion selon Angle, région canine et région molaire; le type de schéma d'occlusion; le montant de chevauchement présent en la région antérieure inférieure; soit avec le test de Pearson Chi-Square ou le test de Yates' Corrected Chi-Square. Pas de différences statistiquement significatives $(p \le 0.01)$ ont été observées entre les sujets bruxeurs nocturnes et les sujets asymtomatiques pour ces variables.

En conclusion, à part de l'examen clinique et les variables polysomnographiques, aucunes des variables oro-morphologiques ne semble discriminer les sujets bruxeurs nocturnes et les sujets asymtomatiques. Puisque cette étude a regardé le changement de la morphologie dentofaciale comme l'effet du, et pas la cause du bruxisme nocturne, peut-être le système humain masticatoire a un système d'adaptation où il peut résister les activités parafonctionelles, avant qu'il y ait des changements morphologiques irréversibles. Cependant, notre étude n'a pas inclus les sujets bruxeurs nocturnes avec usure de dents avancée et perte de dimension verticale, qui secondairement à l'activité parafonctionelle, pourrait induire des changements dans la morphologie avec le temps. Les changements de morphologie auxquels on s'attendrait ne sont pas majeurs, on s'attendrait a des changement subtil.

D'autres investigations de ces hypothèses devraient être faites avec certaines extensions à notre étude:

- faire une étude longitudinale entre sujets bruxeurs nocturnes avec usure de dents initiale et avancée
- échantillonnage plus important (comme on s'attendait, nos calculs
 ont démontré que notre population était trop petite pour montrer des
 différences statistiquement significatives entre les sujets bruxeurs
 nocturnes et les sujets asymptomatiques pour certaines variables)
- avoir une personne indépendante, naïve à l'étude, l'objectif, et entraînée à faire le recueil des données à l'aveugle
- faire le recueil des données polysomnographiques immédiatement avant le recueil des données orofaciales.

TABLE OF CONTENTS

Summary	111
Résumé	vii
List of tables	XV
List of figures	xvi
List of signs and abbreviations	xix
Acknowledgments	
Dedication	xxiii
CHAPTER ONE	
PROJECT OBJECTIVES	2
GENERAL INTRODUCTION	
1.1 DEFINITION OF SLEEP BRUXISM	3
1.2 CLINICAL FEATURES OF SLEEP BRUXISM	4
1.3 DIAGNOSIC FEATURES TO ASSESS SLEEP BRUXISM	5
1.4 SLEEP BRUXISM AND SLEEP STRUCTURE	6
1.5 TREATMENT OF SLEEP BRUXISM	9
1.6 EPIDEMIOLOGY	10
1.7 ETIOLOGY AND PATHOPHYSIOLOGY OF SLEEP BRUX	ISM 11
1.8 GENETICS OF SLEEP BRUXISM	15
1.9 CAUSE-AND-EFFECT RELATIONSHIPS	17
1.10 MORPHOLOGY OF SUBJECTS WITH SLEEP BRUXISM	
1.10.1: Morphology and Genetics	19
1.10.2: Morphological Variations and Bruxism	20
1.10.3: Morphological Variations and Bite Force	22

1.10.4: Arch Form Morphological Variations and Bruxism 2	24
1.10.5: Morphological Variations by Pathology, and Parafunction 2	25
1.11 CONCLUSION	29
CHAPITER TWO	
MATERIALS AND METHODS	
2.1 POPULATION	31
2.2 ORO-MORPHOLOGICAL MEASURES	12
2.3 CEPHALOMETRIC MEASUREMENTS	54
2.4 STATISTICAL METHODS	32
CHAPITER THREE	
RESULTS8	35
CHAPITER FOUR	
DISCUSSION AND CONCLUSION	
4.1 OROFACIAL MORPHOLOGY9	98
4.2 LIMITATIONS OF SAMPLE SIZE)2
4.3 LIMITATIONS OF METHODOLOGY)6
4.4 CONCLUSION	
CHAPITER FIVE	
BIBLIOGRAPHY11	10
	-

APPENDIX ONE
CONSENT FORMxxv
APPENDIX TWO
MEDICAL HISTORY AND PATIENT QUESTIONNAIRE xxxiv
APPENDIX THREE
CLINICAL SLEEP REPORTxli

LIST OF TABLES

<u>Table I Population Information</u>
Table II Polysomnographic Orofacial Data
<u>Table III</u> Sleep Variable Data
<u>Table IV</u> Intra-tracer and Inter-tracer Reliabilities
<u>Table V</u> Oro-Morphological Data
<u>Table VI</u> Oro-Morphological Data
<u>Table VII</u> Oro-Morphological Data
<u>Table VIII</u> Cephalometric Data
Table IX Sample Size

LIST OF FIGURES

Figure 1 Polysomnographic Tracing: Nocturnal Bruxism	37
Figure 2 ABSOLUTE Digimatic Calipers	46
Figure 3 MITUTOYO Specifications	47
Figure 4 Canine Widths	48
Figure 5 Molar Widths	49
Figure 6 Arch Depth	51
Figure 7 Horizontal Plane of Reference	52
Figure 8 Palatal Depth: Measurement	52
Figure 9 Cephalometric Points	57
Figure 10 Cephalometric Planes	58
Figure 11 Interincisal Angle	62
Figure 12 Upper Incisor to SN	62
Figure 13 Lower Incisor to MP	64

Figure 14 SNA Angle 64
Figure 15 SNB Angle
Figure 16 ANB Angle 65
<u>Figure 17</u> NA (<i>ll</i> FH)
<u>Figure 18</u> NB (<i>ll</i> FH)
<u>Figure 19</u> AB (<i>ll</i> FH)
<u>Figure 20</u> NPg (<i>ll</i> FH)
<u>Figure 21</u> Cf - Go
Figure 22 PFH & AFH
Figure 23 Jarabak Craniofacial Growth Spheres with FHR Values
Figure 24 Hyperdivergent, Neutral, & Hypodivergent Growth Patterns
Figure 25 UFH & LFH
Figure 26 Effective Midfacial & Mandibular Lengths

Figure 27 Gonial Angle	79
Figure 28 FMA	79
Figure 29 Angle of Facial Convexity	81

LIST OF SIGNS AND ABBREVIATIONS

A: Point A or subspinale

AD: Arch depth

AFH: Anterior facial height

ANS: Anterior nasal spine

ar: articulare

B: Point B or supramentale

BMI: Body mass index

CAP: Cyclic alternating pattern

Cd: Condylion

CO: Centric occlusion

CR: Centric relation

CW: Canine width

EEG: Electroencephalography

EMG: Electromyography

EOG: Electro-oculography

FH: Frankfort horizontal line

FHR: Facial height ratio

FMA: Frankfort horizontal to mandibular plane angle

Gn: Gnathion

Go: Gonion

Hz: Hertz

Ii: Incisor inferius

Is: Incisor superius

LFH: Lower facial height

Me: Menton

mm: Millimetre

MP: Mandibular plane line

MW: Molar width

N.S.: Non-significant

N: Nasion

OB: Overbite

OJ: Overjet

Or: Orbitale

OSA: Obstructive sleep apnea

p: Alpha

PD: Palatal depth

PFH: Posterior facial height

Pg: Pogonion

Po: Porion

psi: Pound per square inch

Ptm: Pterygomaxillary fissure

REM: Rapid eye movement

RMMA: Rhythmic masticatory muscle activity

S: Sella

SBs: sleep-bruxer subjects

UFH: upper facial height

ACKNOWLEDGMENTS

I would like to express my gratitude to Gilles Lavigne, Jack Turkewicz, Pierre Rompré, and Francine Guitard for their help in the realization of this project.

A special thanks is due to Gilles Lavigne who guided this novice through the ropes of her Masters degree research project!

As well, Pierre Rompré was instrumental in the statistical analysis of this project. His hard work and patience is much appreciated.

This project was funded by the Paul-Geoffrion Fund of the Université de Montréal and partly supported by both the Canadian Medical Research Council and the Fonds de la recherche en santé du Québec.

DEDICATION

Though the words are few, with all my heart I dedicate this thesis to my very special daughter Victoria, my loving husband Carlos, our expectant baby, and my parents Frank & Raffaela, for their love and support throughout my life. With their patience, support, and encouragement, I have been given the chance to reach for the stars and fulfill my aspirations.

Thank-you, vi voglio bene.

CHAPTER ONE

PROJECT OBJECTIVES

and

GENERAL INTRODUCTION

PROJECT OBJECTIVES

The object of this research project was to further identify if any orofacial morphological variables could characterize sleep bruxism. Our initial hypothesis was that SBs have an orofacial morphology that is different from non-bruxers. Unique to this research project was the inclusion criteria for the subjects: subjects who were chosen had previously participated in sleep laboratory studies at the Sacré Coeur Hospital. These subjects were then classified in the "sleep bruxer with grinding" group or in the "control" group based upon the initial selection criteria, and confirmed by the polysomnographic findings. Through an intra-oral examination evaluation, an analysis of dental arches from the plaster models, and cephalometric radiographs, we hoped to find differences between the two groups that could have been due to the effects of sleep bruxism.

GENERAL INTRODUCTION

SECTION 1.1: DEFINITION OF SLEEP BRUXISM

Sleep bruxism is defined as a stereotyped movement disorder characterized by a nonfunctional grinding or clenching of teeth during sleep (Lavigne and Montplaisir, 1995; Rugh and Harlan, 1988; Thorpy, 1990). It is classified as a parasomnia, defined as a phenomenon that intrudes into or occurs during sleep without being a primary disorder of the states of sleep or wakefulness (Thorpy, 1990). Bruxism may be partly considered as a normal behavior which is quite commonly found in the population, with 85 % to 90 % of the population grinding their teeth to some degree during their lifetime (Hublin *et al.*, 1998; Lavigne and Montplaisir, 1994). However with an increased frequency and force of masseter muscle contraction episodes, the normal behaviour deteriorates into a pathological phenomenon for about 5 % to 8% of the population (Hublin *et al.*, 1998; Lavigne and Montplaisir, 1994).

SECTION 1.2: CLINICAL FEATURES OF SLEEP BRUXISM

It has been found that bruxers do not clench or grind their teeth every night, bruxism decreases with age, and that its frequency is influenced by life stress, drugs, alcohol, disease, and personality (Lavigne and Montplaisir, 1995; and Harlan, 1988). Bruxism can also be associated with temporomandibular joint disorders, headaches, toothaches, hypersensitive teeth, and sleep disorders such as sleep apnea and restless leg syndrome. Bruxism can result in pain, tooth wear, disruption of sleep and tension (Lavigne and Montplaisir, 1995; Rugh and Harlan, 1988). In cases where tooth wear has severely affected the dentition, a clinical appearance of loss of facial height may be apparent (Crothers, 1992). Whereas the usual functional activities of the masticatory system such as mastication and deglutition provide the necessary functional stimuli for the maintenance of healthy periodontal structures, parafunctions (such as bruxism) produce additional stress on these components (Graf, 1969). This additional stress may or may not increase loss of tooth structure, temporomandibular joint disorders, and headaches. In a normal daily cycle, it is estimated that tooth contact duration is in fact very low: approximately 17 minutes per day, including meal times and all swallowings (Graf, 1969). It is not clear yet if in SBs it is the duration of tooth contact that is significantly longer or the additive effect of muscle force applied on tooth that causes the problems (tooth wear, pain, and jaw discomfort, etc.) It is a possible explanation since the forces that can be generated on the teeth are substantial: the average biting force is 162 pounds per square inch (psi), while values as high as 975 psi during grinding have been reported (Thompson *et al.*, 1994). Another alternative hypothesis that has never been tested could explain why in some intense bruxers little tooth wear is noted, in contrast to others in whom there is advanced wear: a less densely calcified tooth structure that could increase the rapidity of tooth substance loss.

SECTION 1.3: DIAGNOSTIC FEATURES TO ASSESS SLEEP BRUXISM

Criteria used by a typical dental clinician to diagnose bruxism include: tooth wear; tooth or restoration fracture or failure; masseter muscle hypertrophy on voluntary contraction; tooth mobility; periodontal condition; temporomandibular joint disorder; jaw tightness sensation or clenching; grinding sounds as reported by bed partners. In an ideal research situation, a diagnosis of bruxism using the above criteria is confirmed with a polysomnographic recording (Lavigne and Montplaisir, 1995).

In a sleep laboratory setting, subjects can be monitored with both audio and video in a parallel fashion, as well as having polysomnographic data

(recording of jaw muscle activity during sleep) collected in a graphic form. Typically the subject spends two consecutive nights in the sleep laboratory; the first to get used to the new sleep environment, and the second night is typically used for the collection of data.

This is a very specific, expensive technique to objectively discriminate bruxers from non-bruxer subjects. Diagnostic criteria based on polysomnographic recordings were proposed by Lavigne *et al.* (1996). They are:

- more than 4 bruxism episodes per hour of sleep
- more than 6 bruxism bursts per episode and/or 25 bursts per hour of sleep, and
- at least 2 episodes with grinding sounds.

The combination of these criteria gives a prediction of 82.4% (with a sensitivity-bruxer prediction of 83.3 % and specificity control prediction of 81.3 %) (Lavigne *et al.*, 1996).

This technique utilizes polysomnographic and audio-video recordings in a parallel fashion to help rule out various types of orofacial activities (such as coughing, swallowing, sleep talking, snoring, myoclonic contraction, etc.) that may be present during sleep (Lavigne et al., 1996; Velly-Miguel et al., 1992). Up to 40 % of polysomnographic recordings may contain these orofacial activities, and with the audio-video recordings, one can distinguish sleep bruxism from these other confounding orofacial activities in sleep (Lavigne et al., 1996; Velly-Miguel et al., 1992).

Polysomnographic recordings also include electromyography (EMG) tracings of the masticatory muscles (temporalis, masseters, and digastrics). The types of bruxism episodes found on these tracings can be one of three types: tonic, phasic, and mixed (Velly-Miguel *et al.*, 1992). A tonic episode consists of a sustained contraction (one burst lasting more than 2.0 seconds) (Reding *et al.*, 1968). A phasic episode consists of several rhythmic contractions (at least 3 rhythmic bursts of 0.25 to 2.0 seconds duration, separated by two interburst intervals) (Reding *et al.*, 1968; Ware and Rugh, 1988). A mixed episode is a combination of the two types of episodes. The EMG tracings also graphically illustrate the simultaneous contraction of agonist and antagonist muscles (co-contraction phenomenon) during bruxism.

SECTION 1.4: SLEEP BRUXISM AND SLEEP STRUCTURE

Through the use of polysomnographic recordings, it has been found that sleep bruxism can occur during all sleep stages. However, higher levels of involuntary clenching and grinding were observed in sleep stages 1, 2 and were associated with micro-arousals and alterations of sleep structure. (Bader et al., 1997; Hublin et al., 1998; Lavigne and Manzini, 1998; Lavigne and Montplaisir, 1995; Lavigne et al. 1996; Macaluso et al., 1998; Reding et al., 1968; Rugh and Harlan, 1988; Wieselmann et al., 1986). In a study by Lavigne et al., (1996), approximately 80 % of bruxism-like episodes were produced during sleep stages 1 and 2, for both the bruxism group and the control group. Bruxism is also frequently observed during the transition from a deep sleep stage to a light sleep stage (Bader et al., 1997; Satoh and Harada, 1973).

The observation of bruxism episodes during REM sleep is a source of confusion in the literature. Although it was first published that bruxism episodes occurred mainly in REM sleep (Reding et al., 1964), due to a methodological scoring error (redefinition of the masseter EMG criteria of bruxism, with rhythmicity as the central characteristic), this statement was later retracted (Reding et al., 1967). More recently, a high incidence of destructive sleep bruxism was reported during REM sleep by Ware and Rugh

(1988). This study seemed to have evaluated muscle activity indirectly from EEG artifacts, instead of EMG muscle registrations. As well, the study included several subjects who were on medication for clinical depression, which introduces confounding variables. Thus, further investigations with respect to the association between REM sleep and sleep bruxism would be warranted.

SECTION 1.5: TREATMENT OF SLEEP BRUXISM

The treatment of bruxism is generally palliative and non-invasive with the use of an occlusal splint. Other methods can include occlusal equilibration, biofeedback, use of TENS, psychological counseling, and some medications (Lavigne and Montplaisir, 1995). The effects of occlusal adjustments to treat bruxism reveal that this irreversible therapy produces unpredictable results (Thompson *et al.*, 1994) and is the subject of intense controversy (Okeson, 1996).

SECTION 1.6: EPIDEMIOLOGY

The prevalence of bruxism based on self-reports of bruxism, and not objective criteria, has been reported by: Thompson et al. (1994) who claimed a prevalence of approximately 15 % in children and 96 % in adults; by Attanasio (1991) who reported that in children bruxism varied from 7 % to 88 %, while in the adult population, bruxism varied from 15 % to 90 %. More recently, Hublin et al. (1998) compared the prevalence with respect to frequency of occurrence; in children who report that they are frequent bruxers, the prevalence was between 5 % to 20 %; in young adults, it was in the 2 % to 5 % range; while in the adult and elderly population, it was between 1 % to 5 %. These figures are much more in accordance to what was noted here in Montreal, with 14 % of children reporting frequent grinding (more than 5 nights per week); 13 % of young adults were aware of grinding; this dropped to under 3 % for ages 60 and over (Lavigne and Montplaisir, 1994; Migraine et al., 1996).

SECTION 1.7: ETIOLOGY AND PATHOPHYSIOLOGY OF SLEEP BRUXISM

Despite the relative high prevalence of bruxism that occurs in both adults and children, no simple pathophysiology has been clearly demonstrated so far (Hartmann, 1994; Rugh and Harlan, 1988). Various theories have been advanced, such as bruxism being the result of occlusion-related problems, the consequence of psychological stress, under the influence of the central nervous system, and being associated with genetic predisposition (Lavigne and Manzini, 1998; Lavigne and Montplaisir, 1995). Should this study reveal significant differences between the bruxer and control groups, we may suggest that orofacial morphology has an influence as an etiological factor in the initiation of bruxism.

Although once popularly embraced by the dental community that nocturnal bruxism was the individual's subconscious attempt to perform a self-equilibration of the tooth's occlusal surface, it has been further debated that there is no direct relationship between nocturnal bruxism and tooth interferences (Attanasio, 1991; Clark and Adler, 1985; Okeson, 1996).

It has been frequently suggested that bruxism may be a subconscious outlet for life stresses such as unexpressed emotions, anxiety, hate and aggression (Hartmann 1994; Rugh and Harlan, 1988; Thompson *et al.*, 1994).

On one side, it was proposed that the incidence of bruxism was found to be higher in adults who were under stress or who had the Type A personality, a personality characterized by time urgency and achievement compulsion (Hicks and Chancellor 1987; Hicks *et al.*, 1990; Pingitore *et al.*, 1991; Thompson *et al.*, 1994). However, these findings were not corroborated by a recent study involving 100 adult bruxers (Pierce *et al.*, 1995). In the daytime, subjects were evaluated daily for self-reports of stress and were monitored during sleep with EMG for masseter muscle activity for fifteen consecutive nights. Correlations between EMG measures and self-reported stress were found to be statistically significant for only 8 individual subjects, from over the 100 observed. Therefore, the influence of stress from different life experiences, and the influence of emotions, such as anxiety, hate, and aggression on bruxism requires further investigation.

The hypothesis that bruxism is related to a dysfunctional central nervous system is supported by the finding that various drugs that act on the central nervous system can actually precipitate bruxism episodes (Hartmann, 1994; Rugh and Harlan, 1988; Thompson *et al.*, 1994).

Among those drugs, the first are related to dopamine (the catecholamine pre-cursor L-dopa) that was first described as an exacerbating drug by Magee

(1970). It was noted that one patient with Parkinson's disease being treated with L-dopa exhibited as a side effect, tooth grinding, during the day and during sleep (Magee, 1970). However, a recent controlled study showed the opposite happens in non-neurological (non-Parkinsonian) SBs (Lobbezoo *et al.*, 1996, 1997). A significant reduction in the number of sleep bruxism episodes was noted in SBs after administration of L-dopa, as compared to the control group (who were given a placebo).

The second ones are related to serotonin, where the precursor (L-tryptophan) and the non-specific receptor blocker (amyltriptyline) had no effect (Etzel et al., 1991; Mohamed et al., 1997), although it was reported that selective receptor blockers exacerbate clenching/grinding related to bruxism (Ellison and Stanziani, 1993; Por et al., 1996).

In addition, bruxism occurs in subjects with damaged central nervous systems, such as brain-damaged children, comatose patients, and patients with cerebral palsy (Rugh and Harlan, 1988; Thompson *et al.*, 1994). However, to date, no specific central nervous system structure associated with bruxism has been identified (Thompson *et al.*, 1994).

Interestingly, it has been found that 58 % of the non-bruxer population (Lavigne and Montplaisir, 1995; Lavigne et al., 1998) show masticatory muscle activity which is similar to bruxism, referred to as rhythmic masticatory muscle activity (RMMA) during sleep. RMMA is defined as the presence of at least 3 EMG contractions (bursts) repeated at a frequency of 1 Hz, with a lowest amplitude and lasting more than 0.5 seconds, in the absence of tooth grinding (Lavigne and Manzini, 1998). This condition is present in a large segment of the population and seems to be part of a normal sleep pattern; it is therefore considered a natural activity during sleep (Lavigne and Manzini, 1998; Lavigne et al., 1998). Perhaps, bruxism may be an extreme manifestation of a normal motor activity (Lavigne et al., 1995).

Another area of interest is in the periodic changes in several physiological functions that occur approximately once every 20 to 60 seconds. These periodic changes in the EEG are referred to as cyclic alternating pattern (CAP). Several studies suggest this basic rhythm is involved in modulating these physiological functions: heart rate, respiration, motor activity during sleep, among others (Terzano and Parrino, 1993; Terzano *et al.*, 1990, 1997). CAP has been divided into two phases: Phase A is the transient EEG activation, and Phase B is the subdued EEG activity. A study on four SBs by Zucconi *et al.* (1995) observed that 60 % of sleep bruxism episodes occurred

during Phase A of CAP, although there were no differences in the organization of CAP itself. A more recent controlled study by Macaluso *et al.* (1998) reported that although total CAP rates were not different, 88 % of sleep bruxism episodes occurred during Phase A of CAP. Thus bruxism may be a concomitant result of normal sleep fluctuations.

Most likely, the etiology of bruxism is multifactorial and overlapping, enveloping aspects from all the theories put forth. As well, bruxism may very well be an extreme manifestation of a normal motor activity, and not a typical motor disorder of sleep (Lavigne *et al.*, 1995).

SECTION 1.8: GENETICS OF SLEEP BRUXISM

Hublin et al. (1998) studied the role of genetics on bruxism and found a substantial genetic effect on bruxism, both in childhood and adulthood. They concluded that bruxism is quite a persistent trait, with a significant role of genetic liability in bruxism. A drawback to this study is that adult twins were asked to recall their bruxism episodes starting from their childhood onwards. Memory may have diluted or increased the frequency of the episodes, while some individuals may not have been aware of their own bruxism at all. Since

bruxism episodes occur during sleep, only 5 % to 20 % of persons with this condition are aware of their behaviour (Thompson *et al.*, 1994).

Others (Hori and Hirose, 1995; Lindqvist, 1974) have also done twin studies to determine the relative importance that genetics plays on the presence and amplitude of bruxism. These large twin studies: 117 pairs of twins (Lindqvist, 1974), 332 pairs of twins (Hori and Hirose, 1995), 1298 monozygotic pairs & 2419 dizygotic pairs of twins (Hublin et al., 1998) have shown a familial predisposition of bruxism. As well, monozygous twins had a statistically higher frequency of same wear facet pattern than did dizygous twins (Lindqvist, 1974). This underlines the importance of heredity factors in the genesis pattern of masticatory movement in bruxism. However, to date, no specific genetic marker has been found for bruxism, and additionally, no study has been designed to try and study the specific mode of transmission of this nocturnal parafunctional disorder (Lavigne and Montplaisir, 1995). The role that genetics plays in subjects' morphology will be presented in a subsequent section (Section 1.10.1).

SECTION 1.9: CAUSE-AND-EFFECT RELATIONSHIPS

Before we continue with examining the link between morphology and sleep bruxism, this next section will introduce an important methodological concept, that of cause-and-effect relationships. The determination of whether two entities (such as morphology and bruxism) have a cause-and-effect relationship is an objective of many studies. It is highly unlikely to establish a cause-and-effect relationship with 100% certainty, instead the relationship can be expressed as having a high degree of probability. In order to make this conclusion, studies should fulfil these criteria by Spilker (1991):

- Bias, chance, and confounding influences are absent. This is highly dependent on the study's methodology: double-blind, randomized clinical trials are the most preferable design. Because it is not easy to eliminate the factors which bias and influence the study, they are often left undetected. They are exposed when new or different evaluation techniques are used, which also disprove the assumptions made earlier.
- The association should be consistent. If a true association exists, it should be reproducible from study to study.
- The cause must precede the effect. Therefore to effectively validate a cause-and-effect relationship, it is not enough to have single

measurements because they do not demonstrate the relationship between the two entities over time.

- A dose-response gradient is present. One should observe that with a stronger cause, this should lead to a greater effect.
- The association should make epidemiologic sense.
- The association must be specific. The effect should not occur without the cause having previously occurred.

The more these criteria are fulfilled, the more probable the cause-and-effect relationship is. Depending on how well the study fulfils the above causation criteria, the cause-and-effect relationship between the two entities can be described as almost certain, probable, or unlikely. These criteria should be kept in mind when critically evaluating studies and their conclusions.

SECTION 1.10: MORPHOLOGY OF SUBJECTS WITH SLEEP BRUXISM

Sub-section 1.10.1: Morphology and Genetics

It is generally accepted that the shape of the face is determined by both genetic influences and local environmental factors. If one looks at the genetic influences for facial characteristics and dentofacial variables, different heritabilities have been reported. For different facial characteristics and dentofacial variables, there are variations in heritability ranging from 20 % to 90 % (Savoye et al., 1998). On average, higher heritabilities have been found for vertical facial characteristics and dentofacial variables, whereas lower heritabilities have been found for the horizontal and dental characteristics and dentofacial variables (Savoye et al., 1998). The same investigators have also found high heritabilities in twins for the contours of the individual bones, but lower heritabilities for the whole craniofacial complex. Furthermore, a local environmental factor, the masticatory muscle function, was suggested to have a considerable influence on the craniofacial morphology (Ueda et al., 1998). This tends to support the above finding that there is a lower heritability for the craniofacial complex.

Therefore, if a certain variable has a high heritability, it logically stands to reason it will be less influenced by any environmental factors, and those variables with low heritability are at an increased risk of being influenced by the local environmental factors.

Sub-section 1.10.2: Morphological Variations and Bruxism

The review of the literature reveals several studies which looked at the morphologies displayed by bruxer subjects.

In a study by Waltimo *et al.* (1994), 7 male subjects who were self-reported bruxers with severely worn dentitions were evaluated for dento-facial morphology. The results showed that the bruxer subjects with severely worn dentition typically exhibited a "rectangular jaw" with an "anterior rotated mandible" and a "small anterior facial height." This was explained by the possibility that the rate of dental wear exceeded the rate of compensatory tooth eruption and dento-alveolar bone growth, causing an anterior rotation of the mandible and subsequent reduction of the lower anterior facial height. The bruxer subjects had a greater inter-incisal angle (teeth more upright) and the form of the maxillary dental arches were more rectangular. The control group used for the cephalometric and morphological measurement comparisons

consisted of 15 men. They did not exhibit a severely worn dentition, but they were not objectively evaluated as to whether they were nocturnal bruxers or not.

In another study, Kiliardis (1995) looked at subjects with advanced occlusal tooth wear (without comparing them to any control groups). These persons were described as having a functional hyperactivity of the masticatory system but again no objective measures were done. The functional hyperactivity imposed an increased amount of stress on the bony structures of the craniofacial complex, which influenced its structure. Persons with advanced occlusal wear were found to have a small gonial angle, which was said to be the localized effect of an excessive function of the masticatory muscles in the region of their insertion; but this can be accepted only as a speculation since there was no control group.

Other studies looking at the dentofacial morphology of bruxers versus non-bruxers (Menapace et al., 1994; Young et al., 1999) found there were no significant differences with respect to morphology between the two groups. The author did not choose bruxer patients with severe occlusal wear, but rather used the subject's awareness of bruxism and faceting of teeth to differentially place subjects in their respective groups. We have similar results with non-

destructive bruxism without loss of vertical dimension. The selection criteria may be considered problematic in patient classification for two reasons: firstly, most people are not aware if they grind their teeth or not (only 5 % to 20 % of persons with this condition are aware of their behaviour (Thompson *et al.*, 1994)) and secondly, faceting of teeth is a normal process occuring in most individual over a period of time (Kiliardis and Carlsson, 1994; Marbach *et al.*, 1990).

Sub-section 1.10.3: Morphological Variations and Bite Force

The size and shape of the mandible, including mandibular body length, ramus height, and gonial angle, seem to be correlated to bite force (Ueda *et al.*, 1998). In a study by Braun *et al.* (1995), it was concluded that bite force reflects the geometry of the jaws lever system. The adductor muscles of the mandible appear to have a greater mechanical advantage when the ramus is more vertical and the gonial angle is more acute. As the gonial angle increases, the mechanical advantage of the muscles decreases, resulting in less force perpendicular to the occlusal plane. Thus this indicates that bite force may be a reflection of form. Braun *et al.* (1995) also found that square-jawed individuals exhibited greater maximal bite force capacity than those with normal facial proportions. Thus bruxer sujects who exhibit these

morphological characteristics as per the morphology studies (Kiliardis, 1995; Waltimo *et al.*, 1994), typically will exhibit larger bite forces.

The studies looking at the correlation between morphology and bite forces only considered masticatory muscle activity for short periods of time (during mastication or clenching). Ueda et al. (1998) recently looked at low level muscle activity over day time by having subjects wear portable muscle recorders for 3 hours, excluding meals, sleep, and exertion. They found that the activities of the masseter, temporal and digastric muscles may be related to vertical craniofacial morphology. Their conclusion was that the low-level muscle activity during the day were correlated with reduced vertical craniofacial morphology. Therefore this low-level activity, as well as the strong muscle activity during function and parafunctions, should all be taken into account when investigating craniofacial morphology.

Not all studies looking at bite force and morphology found a significant relationship between the two. Looking at a general population, Fogle and Glares (1995) measured craniofacial morphology from lateral cephalograms, as well as looking at their biting force using EMG. They found that the facial morphology variables they studied did not exert a significant

influence on the EMG data. This particular study did not differentiate whether there were any bruxers being studied, and did not have a control group.

Sub-section 1.10.4:

Arch Form Morphological Variations and Bruxism

The size and form of the dental arches exhibit considerable variability within and among human groups. Different arch forms were noted from that relatively short and square ones to others that are relatively long and tapered. Determinants of arch size and shape are not well understood. Arch form is first defined in the fetus and is affected by several processes. The variability in the eruptive paths of the teeth, growth of the supporting bones, and movement of the teeth after emergence due to habits and unbalanced muscular pressures may all contribute to the variations in arch size and shape as suggested in the literature (Cassidy *et al.*, 1998). The study by Waltimo *et al.* (1994) looked at 7 male bruxer subjects (with severely worn dentitions) found that the form of the maxillary dental arches were more rectangular in these subjects.

Sub-section 1.10.5:

Morphological Variations by Pathology, and Parafunction

Since bruxism is only one example of a parafunctional activity, it is interesting to look at other parafunctional activities or pathological situations and see what possible influence they may exert onto the craniofacial morphology.

In contrast to the study of bruxer subjects with a functional hyperactivity of the masticatory system (Kiliardis, 1995), another study from the same author (Kiliardis *et al.*,1989) with subjects presenting myotonic dystrophy, a reduced muscle function or hypoactivity of the masticatory system, showed a high incidence of malocclusions (Class II, open bites, crossbites, dental crowding), steep mandibles, and high Frankfort Mandibular Plane angles. This is presents an opposite clinical picture of subjects with excessive occlusal wear and high level of muscle function.

It is also interesting to look at sleep apnea studies, as their research design includes the use of both (as per this study) polysomnographic recordings and lateral cephalograms to evaluate cranio-facial morphology. The main interest here is how cranio-facial and soft tissue morphology affect airway size. This is of vital importance since it is postulated and accepted that

obstructive sleep apnea (OSA) is caused by recurrent upper airway obstruction during sleep (Lowe *et al.*, 1986). This is further supported by a study of 25 male subjects with moderate to severe OSA (Lowe *et al.*, 1986): these subjects presented with an opposite clinical picture of those studies looking at bruxers with severe occlusal wear and high level of muscle function (Kiliardis 1995; Kiliardis *et al.*, 1995; Waltimo *et al.*, 1994). The OSA subjects showed a posteriorly positioned maxilla and mandible, steep occlusal plane, overerupted maxillary and mandibular teeth, proclined incisors, steep mandibular plane, large gonial angles, high upper and lower facial heights, and anterior open bites with long tongues and posteriorly placed pharyngeal walls (Lowe *et al.*, 1986). This study however did not use any control groups.

Another sleep apnea study (Marklund *et al.*, 1998) states that although the exact role of cranio-facial morphological variables in the etiology of OSA is unclear, adults with OSA have been found to have certain characteristics of skeletal open bites, more retrognathic mandibles, steeper mandibular plane angle, and longer anterior facial heights. The OSA subjects seem to reflect many of the same characteristics as the subjects with Myotonic Dystrophy, the opposite clinical picture of the bruxer subjects with excessive occlusal wear and high level of muscle function and bite force.

Interesting studies looking at Inuit (Eskimo) morphological characteristics have been done. These subjects, similar to bruxers, have a powerful masticatory apparatus, which has been hypothesized to be the result of centuries of evolution and selection for those individuals with well developed craniofacial morphology (Hylander, 1977; Mayhall, 1977).

The Inuit skull is different from other skulls of the human population, characterized by large spacious orbits; narrow nasal aperature; reduced nasal bones; increased facial flatness; enlarged zygomaxillary region; high temporal lines; shallow glenoid and canine fossa; robust mandible, condyles, coronoid processes, with a wide, low, oblique ascending ramus; a high incidence of palatal and mandibular tori; increased thickness of the tympanic plate; sagittal keeling; weakly developed brow; pronounced gonial eversion; and a high incidence of third molar agenesis (Hylander, 1977; Moses, 1968). Cephalometric studies also reveal the overall size of the Inuit craniofacial complex is significantly larger than in Caucasian groups, with the Inuits exhibiting larger cranial and facial widths, increased mandibular length, anterior facial height, protrusion of the incisors, and increased posterior cranial development, with less prominent nasal morphology and chin point development (Colby and Cleall, 1974).

To explain some of the above listed features of the Inuit skull, a theory postulates that some of the aforementioned morphological features are associated with powerful chewing (Hylander, 1977). The large attachment fields of the muscles of mastication (high temporal lines); the robust mandible, condyles, and coronoid processes; palatal and mandibular tori; and sagittal keeling are features commonly cited as being importantly related to a powerful masticatory apparatus (Hylander, 1977). The increased facial flatness and flaring zygomas are associated with a more anterior positioning of the temporalis and masseter muscles, which improves the mechanical efficiency of the masticatory apparatus (Hylander, 1977). In fact, the Inuit skull is especially adapted to generate and dissipate large vertical biting forces, with Inuits showing impressively high bite force values in various studies (Hylander, 1977; Moses, 1968). This is seen in their traditional way of life, where they exist on an almost completely carnivorous diet, chewing seal skins, frozen food and bones, and use their jaws as a "third hand" or an all purpose vise, all requiring considerable amounts of occlusal forces (Hylander, 1977; Moses, 1968).

As well there is a documented change in the depth and slope of the mandibular fossa with an increasing functional stress on the dentition (Hinton,

1981). Inuits showed a generalized increase in fossa depth and slope with wear (Hinton, 1981).

Thus many parallels can be drawn between Inuit morphology and that of bruxer subjects; both have changes in their craniofacial morphology due to an increased function of the masticatory system; both are able to generate higher than average bite forces, another reflection of the adaptation of their craniofacial morphology.

SECTION 1.11: CONCLUSION

We have seen through this literature review that morphology is sensitive to change, through examples of studies looking at sleep bruxism, increased bite forces, myotonic dysplasia, sleep apneas, as well as ethnic variations (Inuits). Since subjects suffering from sleep bruxism with its parafunctional grinding or clenching of the teeth, have a concomitant increased use of their masticatory system (muscles, dentition, and supporting bone), this study will seek to establish whether SBs have an orofacial morphology that is different from non-bruxers.

CHAPTER TWO

MATERIALS AND METHODS

MATERIALS & METHODS

SECTION 2.1: POPULATION

This study was conducted using 20 subjects in total, of which 10 were SBs and 10 were non-bruxer control subjects. Both the SBs and control groups consisted of 4 female subjects and 6 male subjects, who were matched for age as much as possible. The mean ages were 33.21 ± 1.9 years for the SBs group and 31.2 ± 2.9 years for the control group. Three of the subjects were classified as slightly obese, one SBs and 2 in the control group, with a Body Mass Index (BMI) greater than 28 kg/m² (Boyer 1998). Table I (p. 32) summarizes the relevant patient information on gender, mean ages, height, weight, and BMI. Note that for these variables, alpha is not less than 0.05, thus statistically, no significant differences exist between the bruxer group and the control group with respect to the above variables.

Subjects were recruited from the Universté de Montréal, Faculté de médecine dentaire and the Centre d'étude sur le sommeil of the Hôpital du Sacré-Coeur. The subjects all signed a consent form, approved by the ethics committees of the Université de Montréal and the Hôpital du Sacré-Coeur. As well, the subjects received financial compensation for any inconvenience their

Table I: Population Information

Variable	Bruxer Group	Control Group	
# Female subjects	4	4	
# Male subjects	6	6	
Total # subjects	10	10	

Variable Bruxer Group		Control Group	Significance
	Mean ± standard deviation	Mean ± standard deviation	(t-test)
Age (years)	33.20 ± 6.01	31.23 ± 9.21	N.S.
Height (m)	1.67 ± 0.09	1.71 ± 1.12	N.S.
Weight(kg)	64.05 ± 13.93	69.73 ± 13.94	N.S.
BMI	22.79 ± 4.27	23.80 ± 4.28	N.S.

participation may have generated. Prior to the data collection for this study, a thorough dental and medical history of all subjects was obtained.

The subjects who were chosen for this study had previously participated in the Centre d'étude sur le sommeil de L'Hôpital du Sacré-Coeur (sleep laboratory studies at the Sacré Coeur Hospital's Sleep Study Center) and thus had already undergone polysomnographic recordings.

The subjects first had to fulfill selection criteria (derived from the literature (Rugh and Harlan, 1988; Thorpy, 1990)) and then were either placed in the bruxism group or control group, with respect to the clinical selection criteria. Later, polysomnographic data was used to confirm the correct placement of the subjects into either the bruxism group or control group.

The selection criteria for inclusion into the bruxism group were (Lavigne *et al.*, 1996):

- Male or female
- Ages, between 20 to 45 years old
- Minimum of 5 nights per week of grinding/bruxism sounds during sleep, in
 the last 6 months (as reported by their bed partners)

 Subjective criteria: observation of tooth wear (enamel) or shiny spots (metal restorations)

and/or

- Subjective criteria: morning masticatory muscle fatigue or pain and/or
- Subjective criteria: masseteric muscle hypertrophy upon digital palpation.

The exclusion criteria was as follows:

- More than 2 missing posterior teeth (excluding third molars)
- Presence of a dental prosthesis
- Presence of gross malocclusion (examples: severe Class III malocclusion, severe Class II malocclusion with bird face profile)
- Use of medication with possible effects on sleep or motor behaviour (benzodiazepines, L-dopa, neuroleptics, tricyclic antidepressants)
- Alcohol or drug abuse
- Ongoing dental or physical therapy
- Major neurological or psychiatric disorders
- Sleep disorders (orofacial or cervical myoclonus, narcolepsy, insomnia, periodic leg movements during sleep (>20 events per hour of sleep), EEG

epileptiform activity, sleep apnea (as confirmed during the first night polysomnographic recordings).

The inclusion criteria for the control group were:

- Male or female
- Matched as much as possible for age and sex to the bruxer group

The exclusion criteria for the control group were:

- Same as for bruxers
- Any signs or symptoms of bruxism

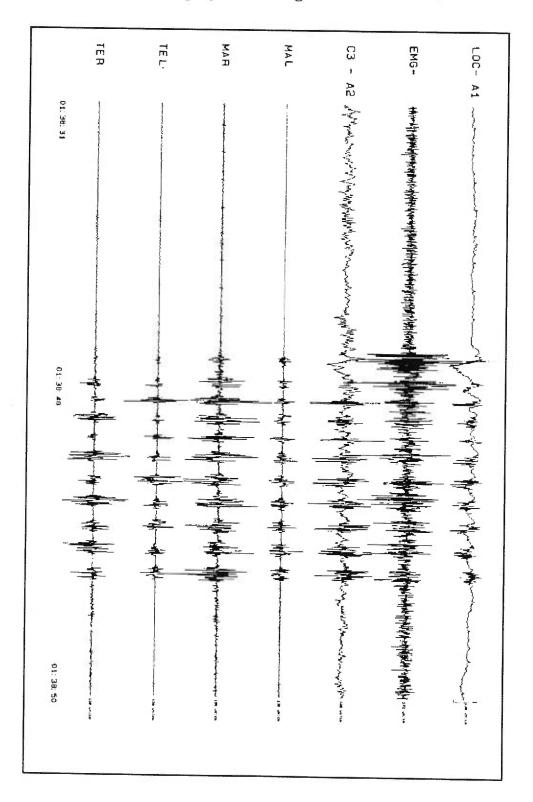
In addition, because there was a variable lag time between the subjects' participation in the sleep laboratory studies at the Sacré Coeur Hospital's Sleep Study Center and this study (ranging from 24 months to 5 years), both the bruxer and control subjects had to respect the following exclusion criteria:

- No loss of teeth since participation in the sleep laboratory studies
- No orthodontic treatment initiated since participation in the sleep laboratory studies
- No maxillo-facial surgery done since participation in the sleep laboratory studies

 No dental prosthesis made since participation in the sleep laboratory studies.

All subjects were studied in the sleep laboratory for a minimum of 2 consecutive nights. The purpose of the first night was for the acclimatization and adaptation of the subject to the environment, as well as to rule out the presence of other sleep disorders (as per the exclusion criteria). The second night was used to collect the experimental data so as to document subjects with respect to their sleep pattern, psychological profile and oromotor activity. All the polygraphic recording was done in a dark, sound-attenuated, and temperature controlled room. The sleep data was recorded and scored using EEG (electroencephalography), EOG (electro-oculography) and EMG (electromyography of the chin, right and left masseter, temporal and anterior tibial muscles), as well as with video and audio recordings. Figure 1 (p. 37) demonstrates a polysomnographic tracing during an episode of nocturnal bruxism.

Figure 1: Polysomnographic Tracing: Nocturnal Bruxism



The clinical diagnosis of sleep bruxism according to the American Association of Sleep Disorders (ASDA) (Thorpy, 1990) is based on the report of tooth grinding or clenching with the presence of at least one of the following secondary criteria:

- positive grinding sounds
- tooth wear
- jaw muscle discomfort

This was initially used to group the subjects and this initial grouping was confirmed by the use of polysomnographic recordings, as per the cut-off values established by Lavigne *et al.*, 1996.

Thus, to be placed in the bruxism group, the subject must have:

• over 4 bruxism episodes per hour

and/or

more than 6 bruxism bursts per episode and/or 25 bruxism bursts
 per hour of sleep

and

• at least 2 events of grinding sounds per sleep period.

Tables II (p. 40) and III (p. 41) summarize the pertinent sleep data recorded from the subjects in this study. This data was obtained from each subject's clinical sleep report, an example of which is included in Appendix Five. Table II (p. 40) refers to polysomnographic orofacial data, and Table III (p. 41) refers to sleep variable data. There are no major differences except, as expected, the number of orofacial activities per night, the number of bruxism episodes per hour, and number of bursts per episode. These variables were used to classify the subject in the bruxer group or the control group, as per the established cut-off values. The values and their significance will be discussed further in the Results section and Discussion section, respectively.

Table II : Polysomnographic Orofacial Data

Variable	Bruxer	Control	Significance
	Mean ± standard deviation	Mean ± standard deviation	(t-test)
# orofacial activities/night	83.10 ± 30.87	40.40 ± 9.65	p≤ 0.01
# bruxism episodes/hour	6.24 ± 3.51	1.12 ± 0.82	p≤ 0.01
# bursts/episode	5.68 ± 3.05	1.30 ± 1.09	p≤ 0.01

Variable	Bruxer	Control	
	Range:	Range:	
	Minimum to	Minimum to	
	Maximum	Maximum	
# orofacial activities/night	23.00 to 120.00	29.00 to 58.00	
# bruxism episodes/hour	0.37 to 12.80	0.29 to 2.90	
# bursts/episode	1.98 to 11.62	0.29 to 3.66	

Table III : Sleep Variable Data

Variable	Bruxer	Control	Significance
	Mean ± standard	Mean ± standard	(t-test)
	deviation	deviation	
Total Sleep Time (minutes)	459.05 ± 42.17	461.03 ± 37.99	N.S.
Sleep Efficiency (%)	95.08 ± 1.79	94.58 ± 4.86	N.S.
Sleep Latency/Onset (minutes)	22.36 ± 23.20	10.50 ± 6.65	N.S.
Sleep Stage Changes (#/hour)	28.48 ± 5.00	27.13 ± 5.23	N.S.
Stage 1 (%)	11.4 ± 3.43	9.43 ± 5.94	N.S.
Stage 2 (%)	55.92 ± 8.49	56.40 ± 8.94	N.S.
Stages 3 & 4 (%)	10.16 ± 6.41	13.84 ± 9.20	N.S.
Stage REM (%)	22.3 ± 4.33	20.29 ± 5.56	N.S.

Section 2.2: Oro-Morphological Measures

Alginate impression of both the maxillary and mandibular dental arches were taken and poured up in white orthodontic plaster. A wax bite in centric occlusion was taken and the models trimmed with their backs parallel.

Using the plaster models, the following measures which were taken during the intra-oral exam were verified: number of teeth present, Angle's dental occlusion for the canines and first molars (both right and left), overbite, and overjet.

The Angle classification of malocclusion was introduced by Edward H.

Angle, using the anteroposterior relationships of the upper and lower first
molars, and for this study, the upper and lower canines as well.

- Canine Class I: lower canine cusp tip occludes with the interproximal contact between upper lateral and upper canine
- Canine Class II: lower canine cusp tip occludes distally with the interproximal contact between upper lateral and upper canine
- Canine Class III: lower canine cusp tip occludes mesially with the interproximal contact between upper lateral and upper canine

- Molar Class I: Mesiobuccal cusp of the upper molar occludes in the buccal grove of the lower molar (Proffit, 1986)
- Molar Class II: Lower molar positioned distally relative to upper molar (Proffit, 1986)
- Molar Class III: Lower molar positioned mesially relative to upper molar (Proffit 1986).

Overbite (OB) and overjet (OJ) were both measured from the maxillary and mandibular right central incisors and are defined as:

- OB: vertical overlap of the maxillary and mandibular right central incisors
- OJ: horizontal overlap of the maxillary and mandibular right central incisors.

As well, the functional occlusal scheme, both right and left, was noted and scored as follows:

1 = cuspid rise

Definition: during the movement made in a lateral excursion, there are no contacts of molars or premolars. The excursion is guided by the contacts the cuspid makes (Ash, 1984)

2 = group function

Definition: during the movement made in a lateral excursion, there are multiple contacts on the working side by either the premolar and/or the molar teeth (Ash, 1984).

The presence of any slide from CR (centric relation) to CO (centric occlusion) was recorded, noting both the direction and amount.

The literature review reveals that the quantitative analysis of dental casts are usually performed by a direct measuring technique after standardized landmarks are individualized on the casts (Ferraio, 1998). Using the plaster models uniquely, the following measures were taken by the principal investigator: maxillary canine width, mandibular canine width, maxillary molar width, mandibular molar width, maxillary arch depth, mandibular arch depth, and palatal depth.

The instrument used to record all the measurements from the dental casts (except for the palatal depth) was the ABSOLUTE DIGIMATIC digital calipers from the Mitutoyo Corporation (Japan), as seen in Figure 2 (p. 46). The digital calipers have a resolution up to 0.01 mm and an error rate of \pm 0.02mm, as per the Mitutoyo Specification Sheet in Figure 3 (p. 47). Two

simple millimetric rulers were used to record palatal depth, as explained below.

For the purpose of this study, the following definitions were used:

• Canine width (CW)

The distance in mm between the canine crown tips or the centers of the corresponding facets, as in Figure 4 (p. 48). If it was found that a canine tooth was missing when calculating the canine width, the corresponding center of the edentulous ridge was used to represent the center of the tooth.

• Molar width (MW)

The distance in mm between the centers of the occlusal surfaces of the first molars, as in Figure 5 (p. 49). If it was found that a molar tooth was missing, when calculating the molar width, the corresponding center of the edentulous ridge was used to represent the center of the tooth.

Figure 2: ABSOLUTE DIGIMATIC Calipers

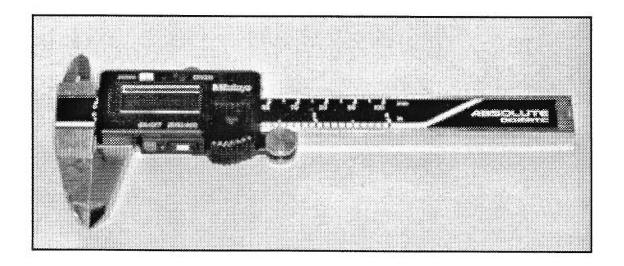


Figure 3: MITUTOYO Specifications

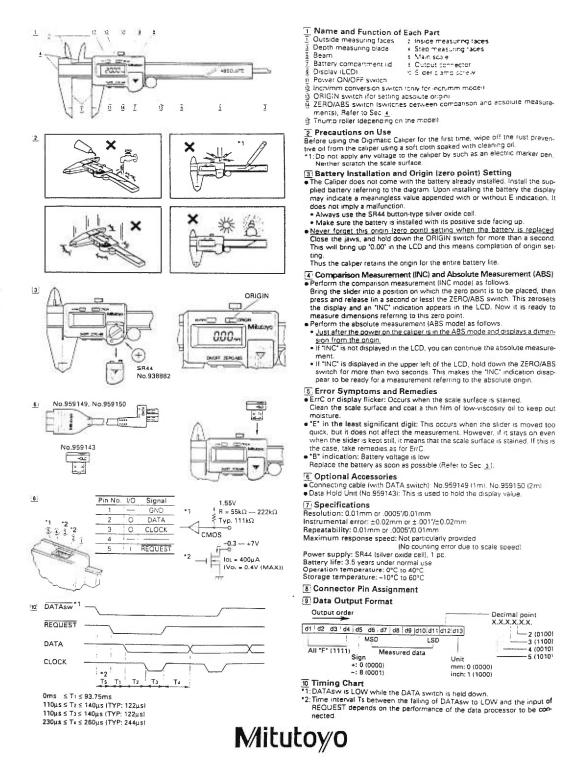


Figure 4: Canine Widths

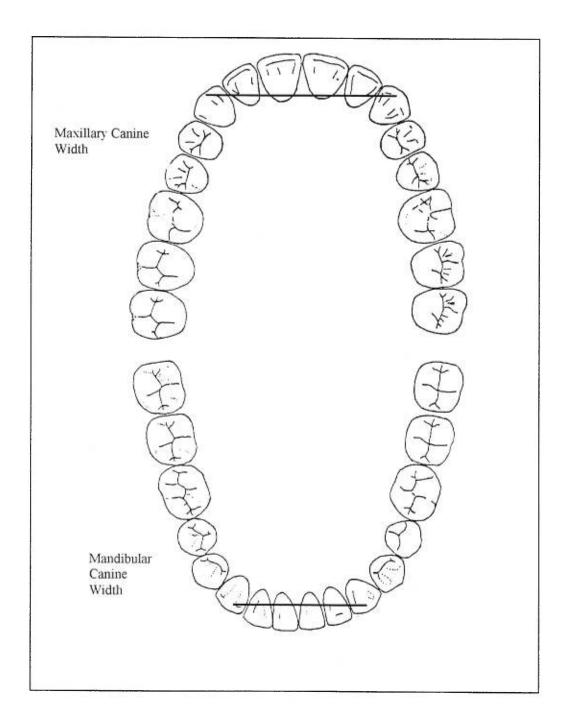
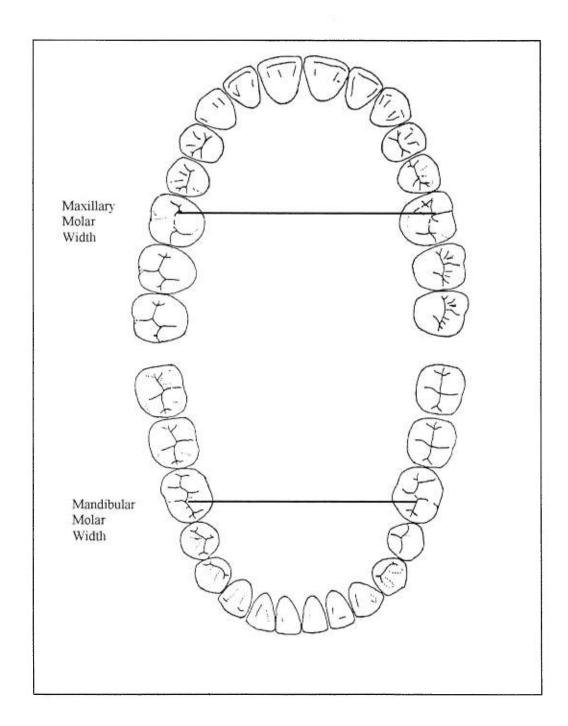


Figure 5: Molar Widths



• Arch Depth (AD)

The distance in mm between a line tangent to the labial surface of the central incisors and a line connecting the most distal points of the first molars, as in Figure 6 (p. 51). If it was found that a molar tooth was missing when calculating the arch depths, the corresponding mesial aspect of the second molar was used instead.

• Palatal Depth (PD)

A vertical measure of the depth of the palate. It was measured from the occlusal plane to a point midpalatally (corresponding to the center of the first maxillary molars). First a rigid millimetric ruler was placed over the occlusal surfaces of the first molars, with one side of the ruler placed over the imaginary line going through the center of the first maxillary molars (Figure 7, p. 52). This is the occlusal plane, or the horizontal plane of reference. Next another millimetric ruler was placed in a vertical direction so that it was perpendicular to the occlusal plane previously established; the ruler's pointed end was placed in the center of the palate (on the mid-palatal raphe). The palatal depth is the measure going from the point on the palate to the occlusal plane (Figure 8, p. 52). If it was found that a molar tooth was missing when

Figure 6: Arch depths

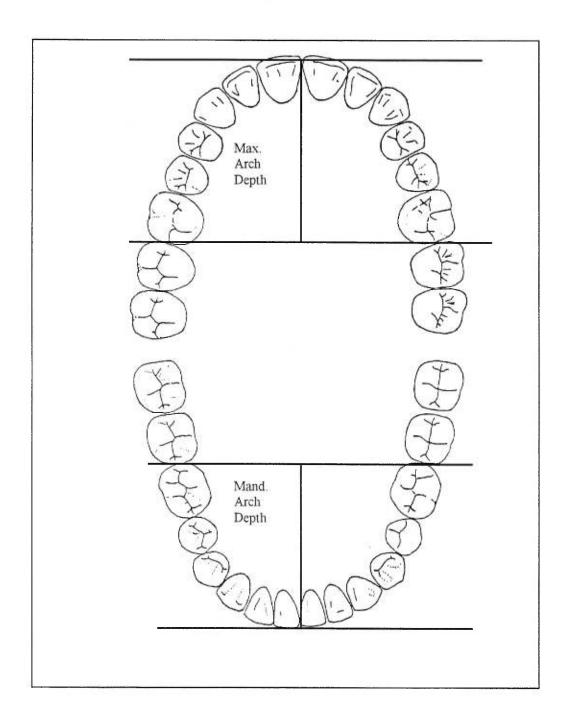


Figure 7: Palatal Depth: Horizontal Plane of Reference

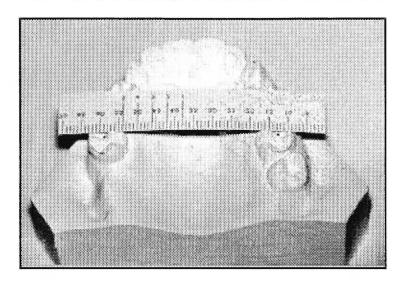
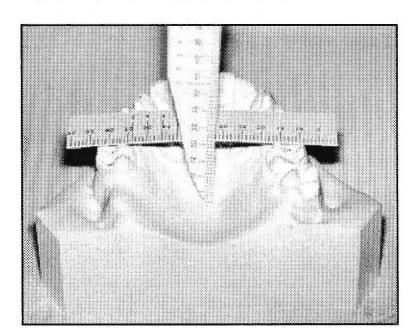


Figure 8 : Palatal Depth: Measurement



calculating the palatal depth, the corresponding height of the edentulous ridge was obtained using the occlusal surface of the adjacent premolar.

These millimetric measures and those measures in a ratio form are presented and assessed in the Results section and Discussion section, respectively.

The presence of anterior crowding was measured in millimetres (mm) and scored as follows:

- 1 no crowding
- 2 1 to 3 mm of crowding
- 3 3 to 5 mm of crowding
- 4 5 to 7 mm of crowding
- 5 greater than 7 mm of crowding

Extra-oral and intra-oral photos were taken as further documentation. The extra-oral views consisted of a straight frontal and a profile, while the intra-oral views consisted of frontal bite, right and left lateral, and maxillary and mandibular occlusal shots.

54

SECTION 2.3: CEPHALOMETRIC MEASUREMENTS

Cephalometrics was initially designed by Broadbent to study serial

growth of the dentofacial complex, and by fractioning the dentofacial

complex, one can examine how the parts are related to one-another (Jarabak

and Fizzell, 1972).

For the purpose of this study, all cephalometric films were taken in a

standard fashion, at the Université de Montréal Dental Clinic Radiology

Section, using the same cephalostat, PICKER GX 300, with the following

settings:

KV major: 80 ± 10

• KV minor: 84 ± 2

• MA: 200 L

• Seconds: 1/30 seconds

The cephalometric film was taken with the left side of the subject's

head placed closest to the film and the subjects looking straight ahead, in the

natural head position (Solow and Tallgren, 1971; 1976). The teeth were closed

together in centric occlusion with a relaxed orofacial musculature.

The films were traced with standard tracing paper in a darkened room, with a fluorescent screen providing the source of illumination. All structures were measured manually using standard equipment.

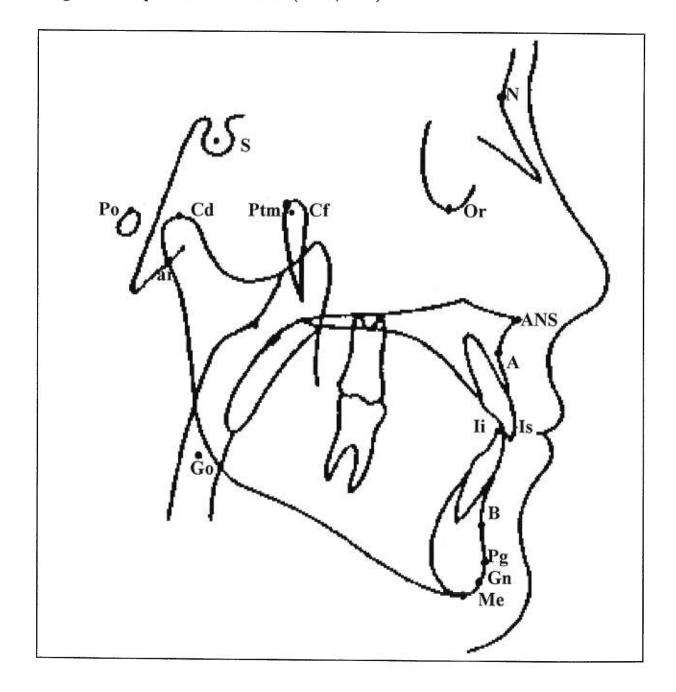
Prior to the study, the principal investigator (Cristina Iafrancesco) and an independent examiner (Jack Turkewicz, Orthodontist) performed intratracer and inter-tracer reliability tests for the cephalometric measures evaluated in this study. The reliability test were done on a sample of 20 randomly chosen cephalometric films, not included in this study. The cephalometric films were traced on two separate occasions, with a time interval of 3 weeks between the two, by the principal investigator. The independent examiner later traced the cephalometric films one time. A reliability estimation was performed so as to determine whether there were any intra-tracer or inter-tracer discrepancies. An intra-tracer discrepancy refers any differences in the measures which were done on two separate occasions by the same person. Inter-tracer discrepancy refers to any differences in the measures as reported by two different persons. As presented in the Results section, no significant discrepancies were found.

Figure 9 (p. 57) reveals the points used to analyze the cephalometric films in this study. Figure 10 (p.58) reveals the planes used to analyze the cephalometric films in this study.

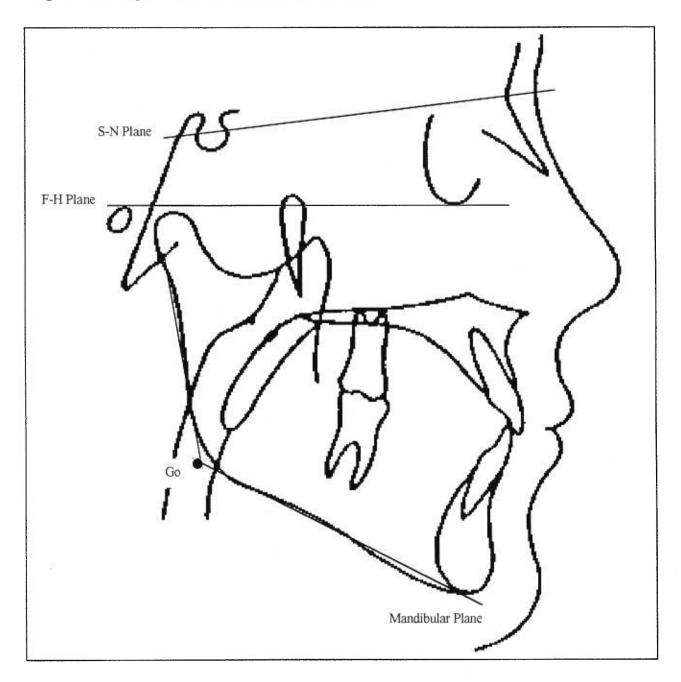
The definition of the points and planes used in this study are as follows:

- A: Point A or subspinale is the deepest midline point in the curved bony outline from ANS to the alveolar process of the maxilla (Rakosi, 1982).
- ANS: The anterior nasal spine is the tip of the bony anterior nasal spine in the median plane (Rakosi, 1982).
- ar: Articulare is the point of intersection of the posterior margin of the ascending ramus and the outer margin of the cranial base (Rakosi, 1982).
- **B**: Point B or supramentale is the most posterior point in the outer contour of the mandibular alveolar process, in the median plane (Rakosi, 1982).
- Cd: Condylion is the most superior and posterior point on the head of the condyle (Rakosi, 1982).
- Cf: This is the point of intersection of pterygomaxillary perpendicular and Frankfort horizontal. Pterygomaxillary perpendicular is constructed by drawing a vertical line perpendicular to Frankfort horizontal, through the pterygomaxillary point (Owen, 1984).

Figure 9: Cephalometric Points (Owen, 1984)







- **FH**: Frankfort horizontal line is the line going through anatomic porion and orbitale.
- Gn: Gnathion is a constructed point corresponding to the intersection of a
 perpendicular line to the midpoint of a line connecting menton and
 pogonion with the bony outline (Rakosi, 1982).
- Go: gonion is a constructed point corresponding to the intersection of the lines tangent to the posterior margin of the ascending ramus and the mandibular plane line (Rakosi, 1982).
- **Ii**: Incisor inferius is the tip of the crown of the most anterior mandibular central incisor (Rakosi, 1982).
- Is: Incisor superius is the tip of the crown of the most anterior maxillary central incisor (Rakosi, 1982).
- Me: menton is the most caudal point in the outline of the symphysis and is regarded as the lowest point of the mandible (Rakosi 1982).
- MP: Mandibular plane is a line going through menton, tangent to the inferior border of the mandible.
- N: Nasion is the most anterior point of the nasofrontal suture in the median plane (Rakosi, 1982).
- Or: orbitale is the lowermost point of the lower border of the orbit of the eye (McNamara, 1984).

- Pg: pogonion is the most anterior point of the bony chin, in the median plane (Rakosi, 1982).
- **Po**: porion is the anatomic porion, which refers to the superior aspect of the external auditory meatus (McNamara, 1984).
- Ptm: pterygomaxillary point is the most superior, posterior point located on the pterygomaxillary fissure, which represents the contour of the fissure projected onto the palatal plane. The anterior wall represents the maxillary tuberosity outline, the posterior wall the anterior curve of the pterygoid process (Rakosi, 1982).
- S: sella is a constructed radiological point in the median plane, corresponding to the midpoint of the sella, or the hypophysial fossa (Rakosi, 1982).
- SN: sella-nasion plane refers to the line going through sella and nasion.

Using the points and planes previously described, several measurements were chosen in order to evaluate the subjects' dental relationships, skeletal antero-posterior relationships and skeletal vertical relationships.

Measurements to evaluate dental relationships:

• Is-Ii°

Interincisal angle (Figure 11, p. 62): the angular measurement between upper and lower central incisor axes, measured posteriorly, with a normative value of 135° (Rakosi, 1982). As the incisors become more upright with respect to one another, the angle assumes a greater value.

• Is-Sn°

Maxillary dental angular measurement: Upper incisor to the sellanasion plane, with a normative value of $104^{\circ}\pm 4^{\circ}$ (Figure 12, p. 62). This measurement has been used as a reasonable description of upper incisor position. Values of 109° or more may indicate protrusion, while values of 99° or less may indicate retrusion. (Owen, 1984).

Ii-MP°

Mandibular dental angular measurement of the long axis of the lower incisor to the mandibular plane, with normative values of 95° ± 5° (Figure 13, p. 64). Values of 101° and more may indicate a mandibular dental protrusion, while values of 89° and less may indicate mandibular dental retrusion (Owen, 1984).

Figure 11: Interincisal Angle (Owen, 1984)

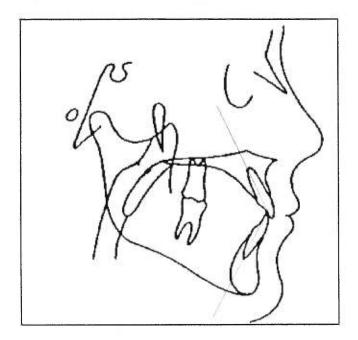
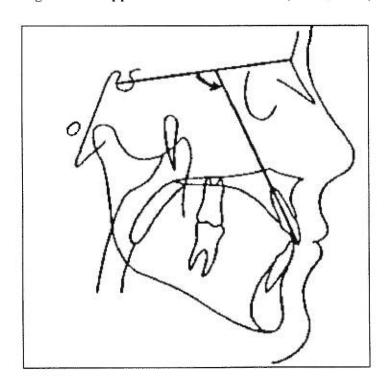


Figure 12: Upper incisor to SN Plane (Owen, 1984)



Measurements to evaluate skeletal antero-posterior relationships:

SNA°

Maxillary skeletal angular measurement from sella to nasion to subspinale, with normative values of 82°± 2° (Figure 14, p. 64). Although sella can vary up or down, independent of the facial skeleton, nasion may be more protrusive or retrusive, and A point can change with treatment. SNA is a commonly used measurement to relate A point to the head and is useful in studies. Values of 85° and more indicate protrusion of point A, while values of 79° and less may indicate retrusion of point A (Owen, 1984).

SNB°

Mandibular skeletal angular measurement from sella to nasion to supramentale with normative values of $80^{\circ} \pm 2^{\circ}$ (Figure 15, p. 65). The same reservations apply to SNB as for SNA. Values of 83° or more may indicate a mandibular skeletal protrusion while values of 77° and less may indicate retrusion (Owen, 1984).

Figure 13: Lower incisor to MP (Owen, 1984)

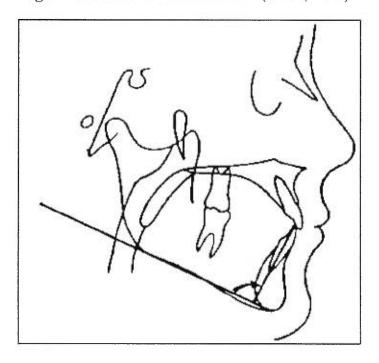


Figure 14: SNA (Owen, 1984)

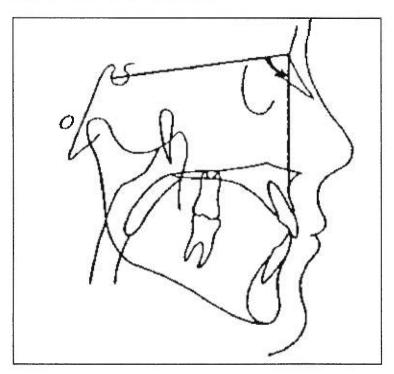


Figure 15: SNB (Owen, 1984)

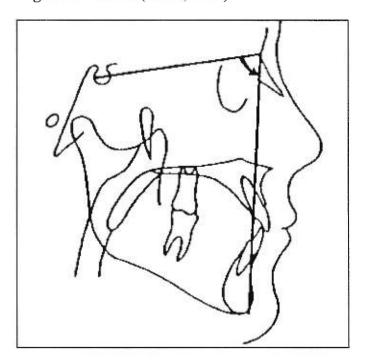
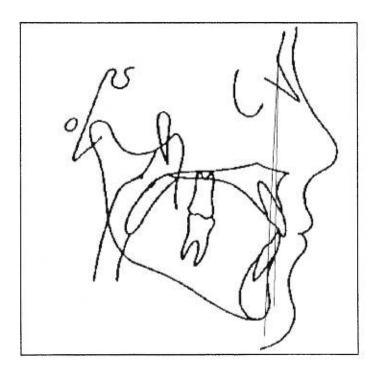


Figure 16: ANB (Owen, 1984)



ANB°

This represents the difference between SNA and SNB angles with a normative value of 2° (Figure 16, p. 65). The ANB angle is positive if point A lies anterior to NB. If NA and NB coincide, the angle will be zero. If point A lies posterior to NB, then ANB will be negative. High positive values occur in Class II skeletal relationships, and high negative values occur in skeletal Class III relationships.

To overcome the deficiencies associated with the angular measurements of SNA, SNB, ANB, we also looked at linear measurements:

• N-A (*ll* FH)

The linear distance from nasion to point A, measured in a parallel fashion to Frankfort Horizontal (Figure 17, p. 68). The procedure is as follows: first Frankfort horizontal is identified. A vertical line (nasion perpendicular) is constructed perpendicular to Frankfort horizontal and extended inferiorly from nasion. Measurements of the linear distance from nasion perpendicular to point A is measured, parallel to Frankfort horizontal (McNamara, 1984). Point A is usually very close to the nasion perpendicular line. Normative values are 0 mm \pm 2. Values of 3 mm or more may indicate

protrusion of Point A, while values of -3 mm or less may indicate retrusion of Point A (Owen, 1984).

• N-B (*ll* FH)

The linear distance from nasion to point B, measured in a parallel fashion to Frankfort horizontal (Figure 18, p. 68). The procedure is as follows: first Frankfort horizontal is identified. A vertical line (nasion perpendicular) is constructed perpendicular to Frankfort horizontal and extended inferiorly from nasion. Measurements of the linear distance from nasion perpendicular to point B is measured, parallel to Frankfort horizontal (McNamara, 1984) . Normative values are -6 mm \pm 4.

• A-B (*ll* FH)

The linear distance from point A to point B, measured in a parallel fashion to Frankfort horizontal (Figure 19, p.70). The procedure is as follows: first Frankfort horizontal is identified. A vertical line (A perpendicular) is constructed perpendicular to Frankfort horizontal and extended inferiorly from point A. Measurements of the linear distance from A perpendicular to point B is measured, parallel to Frankfort horizontal (McNamara, 1984). Normative values are -6 mm ± 4.

Figure 17 : N-A (*ll* FH)

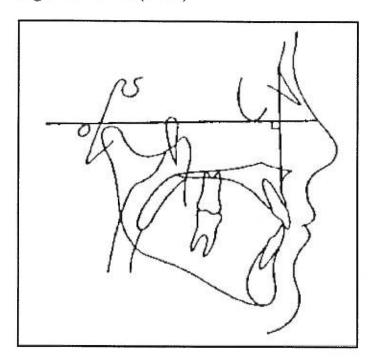
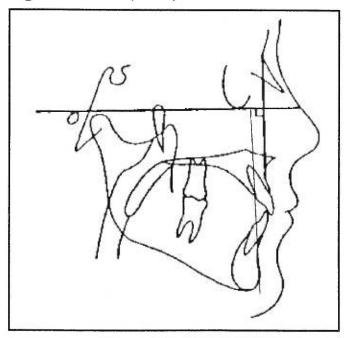


Figure 18 : N-B (// FH)



• N-Pg (*ll* FH)

The linear distance from nasion to pogonion, measured in a parallel fashion to Frankfort horizontal (Figure 20, p. 70). The procedure is as follows: first Frankfort horizontal is identified. A vertical line (nasion perpendicular) is constructed perpendicular to Frankfort horizontal and extended inferiorly from nasion. Measurements of the linear distance from nasion perpendicular to pogonion is measured, parallel to Frankfort horizontal (McNamara, 1984). This value describes chin position well because it is an absolute measure of pogonion. Normative values are -6 ± 3 . Values of -2 or greater may indicate a mandibular skeletal protrusion, while values of -10 mm or less may indicate a skeletal retrusion (Owen, 1984).

Measurements to evaluate vertical skeletal relationships:

Cf-Go

This measurement of the vertical aspect of the ramus, measures the linear distance in mm from CF to gonion (Figure 21, p. 72). Normative values are in the order of 55±3 mm. Values of 59 mm and more may indicate a counterclockwise or brachyfacial tendency, while values of 51 mm and less may indicate a clockwise or dolichofacial tendency (Owen, 1984).

Figure 19: A-B (*ll* FH)

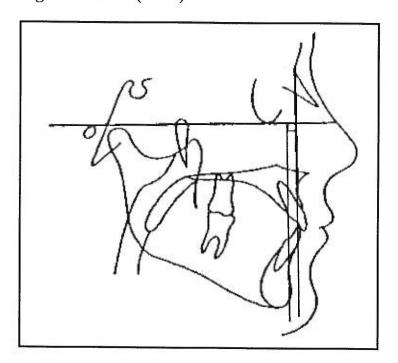
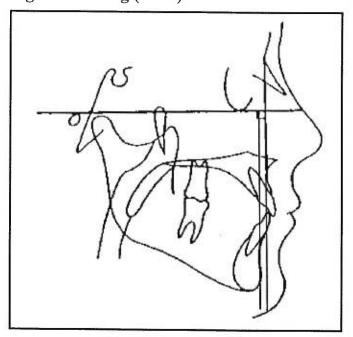


Figure 20: N-Pg (ll FH)



PFH

A linear measurement of posterior facial height (PFH), measuring from sella to gonion in mm (Figure 22, p. 72) (Jarabak and Fizzell, 1972).

AFH

A linear measure of anterior facial height, measuring from nasion to menton in mm (Figure 22, p. 72) (Jarabak and Fizzell, 1972).

The absolute values of the posterior facial height (S-Go) and anterior facial height (N-Me) are not that important, we are interested in the information which can be derived from the ratio they form (Jarabak and Fizzell, 1972).

FHR

The ratio of the posterior facial height to the anterior facial height gives the Facial Height Ratio (FHR) or the Jarabak quotient (Figure 23, p. 74) (Jarabak and Fizzell, 1972).

For a FHR less than 59 %: this demonstrates a hyperdivergent growth pattern, with the face rotating downward and posteriorly with growth, as seen

Figure 21: Cf-Go (Owen, 1984)

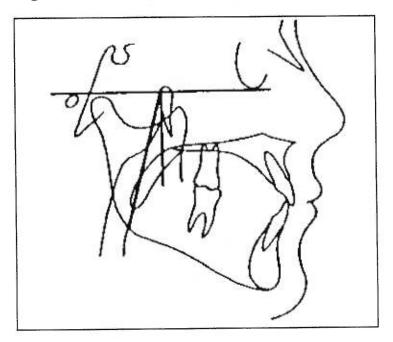
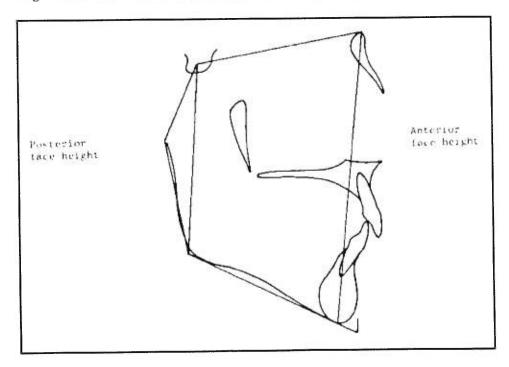


Figure 22: PFH & AFH (Jarabak and Fizzell, 1972)



in Figure 24 (p. 74). The anterior facial height increases more rapidly than the posterior facial height (Jarabak and Fizzell, 1972).

For FHR ranging from 59% to 63%: this demonstrates a neutral growth pattern, the most prevalent pattern. Growth direction is downward and forward, with about the same incremental growth, anteriorly and posteriorly (Figure 24, p. 74) (Jarabak and Fizzell, 1972).

For FHR greater than 63%: This demonstrates a hypodivergent growth pattern, predominantly horizontal growth (Figure 24, p. 74) (Jarabak and Fizzell, 1972).

Prediction of growth pattern is not so clear cut, and there can be an overlap of the different patterns, as shown in Figure 23 (p. 74). The areas of overlap are referred to as the Gray Zones, in which it is difficult to predict in which direction future growth would carry the face. Generally, males would tend towards a more prognathic pattern, while females would tend towards either a more prognathic or neutral growth pattern (Jarabak and Fizzell, 1972).

Figure 23: Jarabak Craniofacial Growth Spheres with FHR Values (Siriwat and Jarabak, 1985)

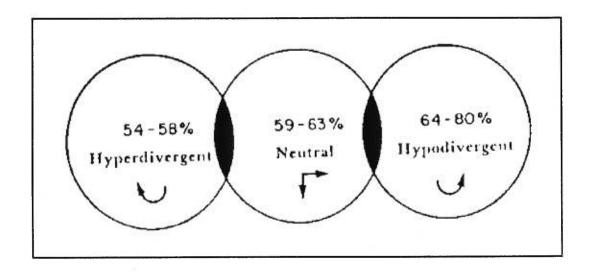
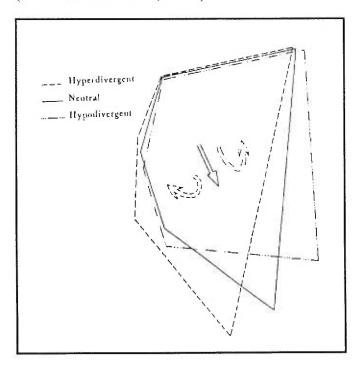


Figure 24: Hyperdivergent, Neutral, & Hypodivergent Growth Patterns (Siriwat and Jarabak, 1985)



UFH

This linear mm measurement of the upper facial height (UFH), measured from nasion to anterior nasal spine, perpendicular to Frankfort horizontal (Figure 25, p. 77).

LFH

This linear mm measurement of the lower facial height (LFH), measured from anterior nasal spine to menton, perpendicular to Frankfort horizontal (Figure 25, p. 77).

These absolute measurements of the UFH and LFH are not what interests us, but will be used to calculate percentages, which will provide us with information relative to vertical relationships of the face.

UFH %/LFH %

The following formulas indicate how the UFH % and LFH% are calculated:

UFH
$$\%$$
 = UFH \div (UFH + LFH) * 100

LFH
$$\%$$
 = LFH \div (UFH + LFH) * 100

The ratio of UFH %/LFH % should be 45%/55 % for well balanced faces (in the vertical dimension) (Figure 25, p. 77). Variations of more than 5% may indicate vertical excess or deficiency in that area (Owen, 1984).

• Cd-A

This linear measurement is the effective midfacial length. It is a measure of the line from cd to point A, with normative values of 85 ± 6 mm for females, and 87 ± 6 mm for males (Figure 26, p. 77) (McNamara, 1984).

Cd-Gn

This linear measurement is the effective mandibular length which is derived by measuring the line from cd to gnathion (Figure 26, p. 77). Normative values are 105 ± 6 mm for females, and 107 ± 6 mm for males (McNamara, 1984).

• Maxillomandibular Differential

The maxillomandibular differential is the the difference between the effective mandibular length and the effective midfacial length. The effective midfacial length is subtracted from the effective mandibular length. Normative value is 20 mm (McNamara, 1984).

Figure 25: UFH & LFH (Owen, 1984)

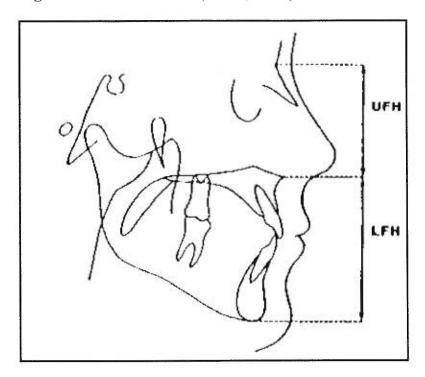
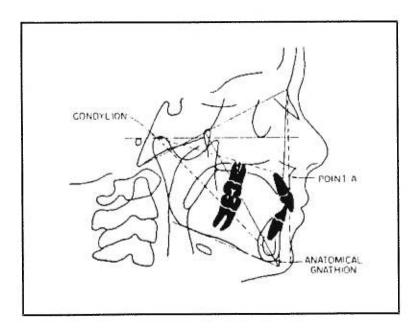


Figure 26: Effective Midfacial & Mandibular Lengths (McNamara, 1984)



LFH /Cf-Go

The ratio of the lower facial height (ANS-Me) to the vertical aspect of the ramus (Cf-Go) was calculated and was used in the comparison between bruxers and non-bruxer groups. To our knowledge, this ratio does not exist in the literature.

Ar-Go-Me°

The gonial angle is an angular measurement of Ar to Go to Me, with normative values of $128 \pm 7^{\circ}$ (Figure 27, p. 79). The gonial angle is an expression of the mandibular form, with respect to the relationship between the body and ramus of the mandible. A large angle indicates more of a tendency to a posterior rotation of the mandible. A small gonial angle indicates a tendency to anterior rotation of the mandible (Owen, 1984).

FMA°

This is an angular measurement of the angle formed by the Frankfort horizontal plane to the mandibular plane (Figure 28, p. 79). Normative values are in the order of $26^{\circ} \pm 4^{\circ}$. Values of 31° and more may indicate vertical excess, while values of 21° or less may indicate vertical deficiency or brachyfacial growth trends (Owen, 1984).

Figure 27: Gonial Angle (Rakosi, 1982)

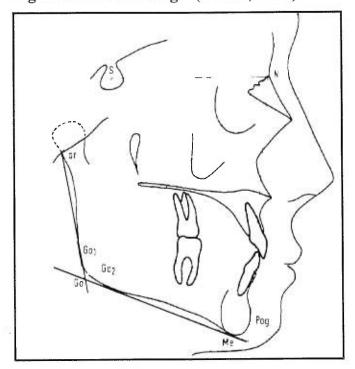
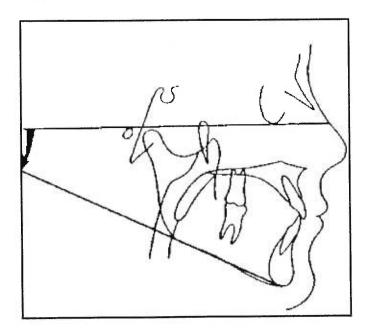


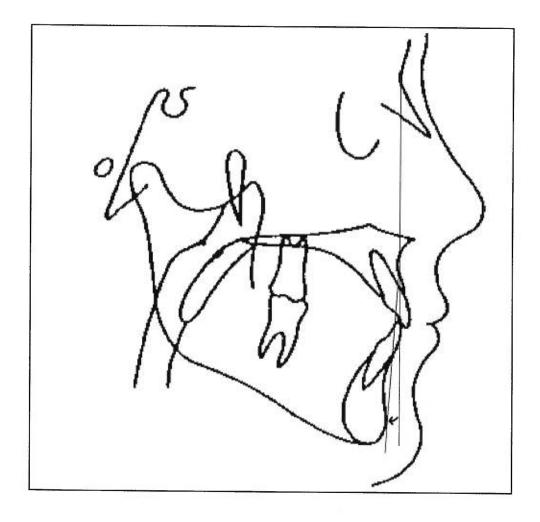
Figure 28: FMA (Owen, 1984)



• N-A-Pg °

Angle of facial convexity. Measures the relative convexity or concavity of the subjects profile by measuring the angle formed by nasion, point A, and pogonion (Figure 29, p. 81). Normative values are $4^{\circ} \pm 2$.

Figure 29: Angle of Facial Convexity



SECTION 2.4: STATISTICAL METHODS

Intraclass correlation coefficients (Deyer et al., 1991) were calculated to assess the intra-tracer reliability (the reliability of double tracing) and the inter-tracer reliability for landmark identification and the calculation of measurements.

For most of the variables in the study, since the data was normally distributed, they were assessed with the use of descriptive statistics, including the mean and standard deviation. The Two-Sample T-Test was used to evaluate significance.

Since their distribution was not normal, frequencies were calculated for the following variables: Angle canine classification, right and left, Angle molar classification, right and left, occlusal scheme, right and left, and determination of lower anterior crowding, with either the Pearson Chi-Square test or the Yates' Corrected Chi-Square test. As previously presented in this chapter, each of these variables had different number of values. For the Angle classification, three values were possible; for the occlusal scheme, two values were possible; for lower anterior crowding, five values were possible. The SBs and control groups were compared using the Pearson Chi-Square test, for the following variables: Angle canine classification left, Angle molar

classification, right and left, and determination of lower anterior crowding. The Yates' Corrected Chi-Square test was used for two-by-two tables in determination of frequencies for the Angle canine classification right and occlusal scheme, right and left.

Taking into account the large number of comparisons done between the two groups, an alpha (p) value of ≤0.01 was considered statistically significant, so as to avoid statistical Type I errors.

As well, the sample sizes needed to detect the differences observed between the groups in this study were calculated for selected variables using the statistical criteria of α =0.05 and β =0.20, with GPOWER (Faul and Rerdfelder, 1992).

CHAPTER THREE

RESULTS

RESULTS

Tables II (p. 40) and III (p. 41) summarize the pertinent polysomnographic and sleep data recorded from the subjects in this study. This data was obtained from each subject's clinical sleep report, an example of which is included in Appendix Five. Table II (p. 40) refers to polysomnographic orofacial data, and Table III (p. 41) refers to sleep variable data. There are no major differences except, as expected, the number of orofacial activities per night, the number of bruxism episodes per hour, and the number of bursts per episode. These variables were used to classify the subject in the sleep bruxer group or the control group, as per the established cut-off values, which were presented in the Material and Methods section.

Sleep bruxers had 83.10 ± 30.87 (range of 23.00 to 120.00) orofacial activities per night, whereas non-bruxers had 40.40 ± 9.65 (range of 29.00 to 58.00) orofacial activities per night (p<0.01). Sleep Bruxers had 6.24 ± 1.11 (range of 0.37 to 12.80) bruxism episodes per hour, versus the control group who had 1.12 ± 0.26 (range of 0.29 to 2.90) bruxism episodes per hour (p<0.01). Sleep Bruxers had 5.68 ± 3.05 (range of 1.98 to 11.62), versus the control group who had 1.30 ± 1.09 (range of 0.29 to 3.66) bursts per episodes

(p< 0.01). Thus, all are significant variables and support the statement SBs oro-motor activity is much more frequent than in non-bruxers.

In comparing various sleep variables between sleep bruxer subjects and control subjects, there were no significant differences (p=N.S.). With such a sample size, these results are in accordance with previous findings that demonstrate no differences between sleep bruxers and non-bruxers with respect to sleep organization (Lavigne *et al.*, 1996). Therefore, as we see in Table III (p. 41), our sleep bruxer group demonstrated no differences with the control group for the following sleep variables: total sleep time, sleep efficiency, sleep latency or sleep onset, number of sleep stage changes per hour, sleep stage by percentage: stage 1, stage 2, stages 3 and 4, and stage REM.

The reliability tests performed for the cephalometric measurements reveal no significant intra-tracer nor any inter-tracer differences (Table IV, p.87). With the exception of two variables, intra-tracer reliability for LFH % (ICC = 0.87339) and inter-tracer reliability for Cd - A (ICC = 0.86572), the rest of the correlation coefficients ranged from 0.91240 to 0.99486. These represent excellent reliability values, and are in accordance with previously

Table IV: Intra-tracer and Inter-tracer Reliabilities

Variable	Intra-tracer Reliability	Inter-tracer Reliability
Is-Ii °	0.99486	0.98292
Is-Sn °	0.97975	0.95561
Ii-MP °	0.96296	0.92328
SNA°	0.95862	0.97308
SNB°	0.97235	0.97285
ANB°	0.96126	0.96180
NA (// FH) (mm)	0.91240	0.93961
NB (// FH) (mm)	0.94033	0.95505
AB (// FH) (mm)	0.96855	0.94544
N-Pg (// FH) (mm)	0.95198	0.94596
Cf-Go (mm)	0.99295	0.98327
PFH (mm)	0.99173	0.96838
AFH (mm)	0.99248	0.97514
FHR	0.98063	0.94247
UFH (mm)	0.97775	0.95319
LFH (mm)	0.99137	0.98019
UFH %	0.97279	0.92587
LFH %	0.87339	0.92728
Cd-A (mm)	0.93119	0.86572
Cd-Gn (mm)	0.98276	0.97190
Cd-Gn – Cd-A (mm)	0.97084	0.96079
LFH/ Cf-Go	0.98957	0.97431

Variable	Intra-tracer Reliability	Inter-tracer Reliability
Ar-Go-Me °	0.98499	0.98544
FMA°	0.98563	0.95727
N-A-Pg °	0.97666	0.97051

published reliability tests in the literature (Fogle and Glaros, 1995; Menapace et al., 1994; Ozbek et al., 1998).

The first set of oro-morphological variables in Table V (p. 90) were statistically analyzed using means, standard deviation, and the Student T-test to evaluate significance. These variables, total number of teeth, number of teeth in the maxillary and mandibular arches, overbite and overjet, slide from centric relation to centric occlusion showed no significant differences between the SBs group and the control group.

The second set of oro-morphological measures in Table VI (p. 91) related the canine and molar Angle classification, occlusal scheme, and amount of crowding present. Using statistical frequencies, no variables showed statistical significance. Additionally, the data is incomplete for several variables such as for the Angle classification of the right canine, as the maxillary canine was missing in 1 SBs; in the Angle classification of the left canine, 1 SBs was missing the maxillary canine; for the Angle classification of the right molar, 3 SBs were missing either the maxillary or mandibular right molar; and for the Angle classification of the left molar, 5 SBs were missing either the maxillary or mandibular left molar.

Table V: Oro-Morphological Data

Variable	Bruxer	Control	Significance
	Mean ± standard	Mean ± standard	(t-test)
	deviation	deviation	
ОВ	1.37 ± 1.28	1.95 ± 1.71	N.S.
OJ	1.90 ± 0.94	2.13 ± 1.11	N.S.
# Teeth Maxilla	13.70 ± 1.49	14.20 ± 1.23	N.S.
# Teeth Mandibular	13.40 ± 1.35	14.20 ± 1.14	N.S.
# Teeth Total	27.10 ± 2.47	28.40 ± 2.12	N.S.
CR-CO	0.35 ± 0.27	0.10 ± 0.32	N.S.

Table VI: Oro-Morphological Data

Variable	Bruxer	Control	Test	Significance
Angle Canine Right			Yates	N.S.
# Class 1	9	9		
# Class 2	0	0		
# Class 3	0	1		
Angle Canine Left			Pearson	N.S.
# Class 1	4	9		
# Class 2	5	0		
# Class 3	0	1		
Angle Molar Right			Pearson	N.S.
# Class 1	5	6		
# Class 2	1	2		540
# Class 3	1	2		
Angle Molar Left			Pearson	N.S.
# Class 1	3	5		
# Class 2	2	2		
# Class 3	0	3		
Occlusal Scheme Right			Yates	N.S.
# Category 1	1	6		
# Category 2		4		
Occlusal Scheme Left		7	Yates	N.S.
# Category 1	1	6		
# Category 2	9	4		
Anterior Crowding			Pearson	N.S.
# Category 1	5	2		
# Category 2	4	5		
# Category 3	1	2		
# Category 4	0	1		
# Category 5	0	0		

The third set of oro-morphological measures, in Table VII (p. 93) evaluated the following variables: maxillary canine width (CW), mandibular canine width, maxillary molar width (MW), mandibular molar width, maxillary arch depth (AD), mandibular arch depth, palatal depth, ratio of the mandibular CW/MW, ratio of maxillary CW/AD, mandibular CW/AD, ratio of maxillary to mandibular CW, ratio of maxillary to mandibular MW, and ratio of maxillary to mandibular AD, with the use of Student T-tests.

Descriptive statistics using means and standard deviation were done, as well as the Student T-test to evaluate significance. No significant results were recorded.

Table VIII (p. 94) shows the cephalometric values studied, of which, none showed any significant differences between the sleep bruxer and control groups. The variables shown in Table VIII (p. 94) along with their mean values, standard deviations, and significance include the measurements to evaluate dental relationships: the angular measurement Is-Ii, angular measurement Is-Sn, angular measurement Ii-MP; the measurements to evaluate

Table VII: Oro-Morphological Data

Variable	Bruxer	Control	Significance
	Mean ± standard	Mean ± standard	(t-test)
	deviation	deviation	
Maxillary CW (mm)	32.10 ± 2.43	33.37 ± 1.60	N.S.
Mandibular CW (mm)	26.71 ± 3.45	25.42 ± 1.25	N.S.
Maxillary MW (mm)	46.43 ± 2.48	44.84 ± 3.90	N.S.
Mandibular MW (mm)	41.87 ± 2.96	40.05 ± 2.65	N.S.
Maxillary AD (mm)	36.10 ± 2.44	36.61 ± 3.74	N.S.
Mandibular AD (mm)	31.07 ± 1.98	32.09 ± 2.89	N.S.
Palatal Depth PD (mm)	21.00 ± 2.82	21.10 ± 2.72	N.S.
Maxillary CW/MW	0.69 ± 0.04	0.75 ± 0.06	N.S.
Mandibular CW/MW	0.64 ± 0.08	0.64 ± 0.06	N.S.
Maxillary CW/AD	0.89 ± 0.06	0.92 ± 0.08	N.S.
Mandibular CW/AD	0.86 ± 0.10	0.80 ± 0.08	N.S.
Maxillary/Mandibular CW	1.21 ± 0.13	1.31 ± 0.05	N.S.
Maxillary/Mandibular MW	1.11 ± 0.05	1.12 ± 0.06	N.S.
Maxillary/Mandibular AD	1.16 ± 0.05	1.14 ± 0.08	N.S.

Table VIII: Cephalometric Data

Variable	Bruxer	Control	Significance
	Mean ± standard deviation	Mean ± standard deviation	(t-test)
Is-Ii °	135 ± 13.90	133.50 ± 10.89	N.S.
Is-Sn °	95.30 ± 12.90	103.40 ± 7.90	N.S.
Ii-MP °	91.20 ± 7.15	90.00 ± 6.60	N.S.
SNA°	79.70 ± 3.50	80.85 ± 2.43	N.S.
SNB °	77.30 ± 3.62	78.90 ± 2.72	N.S.
ANB°	2.40 ± 2.41	1.95 ± 2.58	N.S.
NA (// FH) (mm)	-0.50 ± 2.37	-0.10 ± 3.44	N.S.
NB (// FH) (mm)	-5.2 ± 5.05	-3.85 ± 8.39	N.S.
AB (// FH) (mm)	4.70 ± 4.57	3.75 ± 6.10	N.S.
N-Pg (// FH) (mm)	-3.20 ± 4.18	-1.90 ± 9.10	N.S.
Cf-Go (mm)	71.40 ± 5.10	69.10 ± 7.00	N.S.
PFH (mm)	84.25 ± 6.23	83.65 ± 8.63	N.S.
AFH (mm)	128.85 ± 8.54	128.25 ± 9.33	N.S.
FHR	65.60 ± 6.17	65.17 ± 3.97	N.S.
UFH (mm)	55.85 ± 4.52	57.15 ± 3.89	N.S.
LFH (mm)	72.10 ± 5.65	70.90 ± 7.61	N.S.
UFH %	43.63 ± 2.29	44.73 ± 2.76	N.S.
LFH %	56.37 ± 2.29	55.27 ± 2.76	N.S.
Cd-A (mm)	89.80 ± 5.45	91.00 ± 6.06	N.S.

Variable	Bruxer	Control	Significance
	Mean ± standard deviation	Mean ± standard deviation	(t-test)
Cd-Gn (mm)	121.95 ± 6.28	124.70 ± 8.96	N.S.
Cd-Gn – Cd-A	32.15 ± 4.83	33.70 ± 5.36	N.S.
LFH/ Cf-Go	1.02 ± 0.11	1.03 ± 0.09	N.S.
Ar-Go-Me °	125.10 ± 7.02	127.95 ± 5.14	N.S.
FMA°	24.30 ± 5.93	22.55 ± 10.45	N.S.
N-A-Pg °	1.95 ± 5.06	1.60 ± 5.21	N.S.

skeletal antero-posterior relationships: angular measure SNA, angular measure SNB, angular measure ANB, linear measure NA (// FH), linear measure NB (// FH), linear measure AB (// FH), linear measure N-Pg (// FH), and the measurements to evaluate vertical skeletal relationships: linear measurement Cf-Go, linear measure PFH (S-Go), linear measure AFH (Na-Me), FHR (the ratio of the PFH/AFH), linear measure UFH (Na-ANS), linear measure LFH (ANS-Me), UFH %, LFH %, linear measure of Cd-A, linear measure of Cd-Gn, difference between Cd-Gn and Cd-A, the ratio of LFH/ Cf-Go, angular measurement Ar-Go-Me, angular measure FMA, and the angular measure N-A-Pg.

The next chapter will discuss the pertinence of the results obtained.

CHAPTER FOUR

DISCUSSION

and

CONCLUSIONS

DISCUSSION and CONCLUSION

SECTION 4.1: OROFACIAL MORPHOLOGY

In accordance with our review of the literature, we can remark that this study is the first (to our knowledge) to assess any differences in oromorphological and cephalometric variables between SBs and control subjects, previously discriminated using polysomnographic parameters and stringent selection criteria to classify subjects into their respective groups. One of the unique features here is that the first selection of subjects was not done with any orofacial, morphological discrimination. A selection bias was therefore eliminated and thus we were able to avoid confounding influences.

Although the assessment of sleep variables was not the goal of our study, these allowed us to base our study on solid ground and avoid the presence of any confounding sleep problems that would have increased data variability. The sleep variable data demonstrates that our results are in accordance with previous findings; sleep bruxer subjects have a normal sleep organization, with normal sleep latency (onset), total sleep time, sleep efficiency, percentage of sleep stages, and various stages, and that bruxer subjects do not complain of poor sleep (Lavigne *et al.*, 1996).

The oro-morphological measures in this study showed that bruxers and control subjects did not differ significantly with regard to number of teeth present, overbite, overjet, occlusal scheme present, slide from centric relation to centric occlusion, canine and molar Angle classification, and amount of anterior crowding. Our results are similar in that regards to the ones of Menapace *et al.* (1994) who also found there were no significant differences for the Angle classification of occlusion, nor were any significant differences found between the SBs and control groups for overbite (Young *et al.*, 1999).

Contrary to what we expected, we found no obvious differences between groups for the for the oro-morphological measures involving the dental casts. Thus there were no significant differences in the maxillary and mandibular canine widths, molar widths, arch depths and ratios of these variables. In contrast, Waltimo *et al.*(1994) found that the form of the maxillary dental arch was more rectangular (since the ratio of maxillary canine width to molar width was higher) in bruxer patients with severely worn dentitions. It must be kept in mind that this study was based on 7 patients with severe attrition (more than 1/3 of the crown lost) caused by nocturnal bruxism; in our study, the SBs were not showing such an advanced loss of dental substance. Though there were controls for the cephalometric and dental

measurements, no mention is made whether these subjects were screened for the presence of nocturnal bruxism.

The cephalometric variables of this study describe dental angular relationships, antero-posterior skeletal relationships and vertical skeletal relationships. In general, our study did not find any significant differences between the bruxer group and the non-bruxer group for any of the variables, as per other studies (Menapace *et al.*1994; Young *et al.*, 1999). For the antero-posterior skeletal relationships, we are in accordance with the findings of Kiliardis *et al.* (1995) who studied dentofacial structure, occlusal traits, and bite force in subjects with extensive occlusal wear. That study did not find any significant differences for any of the antero-posterior skeletal measurements either.

In evaluating our cephalometric measurements, our results did not show as those of Waltimo *et al.* (1994) that the bruxer patients typically exhibited an anterior rotated mandible and a small anterior facial height. Additionally, Waltimo *et al.* (1994), found a larger interincisal angle for his group of 7 bruxer patients with severe wear. Perhaps, since none of our bruxer subjects exhibited a severely worn dentition, the rate of dental wear did not exceed the rate of compensatory tooth eruption and dento-alveolar bone growth.

Therefore our bruxer subjects had no changes in the vertical position of the mandible and nor any reduction of the lower anterior facial height.

Thus, those studies looking at bruxism patients with severely worn dentitions (Kiliardis, 1995; Kiliardis et al., 1995; Waltimo et al., 1994) found significant differences in the interincisal angle, the vertical skeletal relationships, but no significant differences in the antero-posterior skeletal relationships.

In contrast, studies comparing bruxer and non-bruxer subjects, (Menapace *et al.* 1994; Young *et al.*, 1999) without using destructive tooth wear caused by sleep bruxism as a confounding variable, did not find a significant difference in any of the interincisal angle, the vertical skeletal relationships, or the antero-posterior skeletal relationships, as per our results.

The differences in the results from the different studies may be due to the selection criteria for inclusion into the study and subsequent classification into the bruxer group or control group. The studies (Kiliardis, 1995; Kiliardis et al., 1995; Waltimo et al., 1994) looking at bruxer subjects with severe wear included a confounding variable in their results. The studies (Menapace et al. 1994; Young et al., 1999) using the self-report of bruxism for inclusion

into the bruxism group relied on subjective criteria to classify a subject as a bruxer subject. Several studies (Kiliardis, 1995; Kiliardis *et al.*, 1995) were conducted in the absence of a control group. As well there are limitations due to sample size. All these factors decrease the validity of the results obtained by the respective studies.

SECTION 4.2: LIMITATIONS OF SAMPLE SIZE

Most of the results of this study did not show a significant difference between the bruxer group and the non-bruxer control group. However it is interesting to note that by increasing our sample size, real differences between the sleep bruxer group and the control group could be realized (Table IX, p. 103). Sample size calculations were done for the certain variables: interincisal angle, as well as the vertical cephalometric measures. These variables were chosen because significant results for these variables were obtained in other studies (Kiliardis *et al.*, 1995; Waltimo *et al.*, 1994).

The sample size calculations for the following variables revealed enormously large sample sizes would be required:

Table IX : Sample Size

Variable	Total sample size required for significance	
Is-Ii °	2180	
Cf-Go (mm)	226	
PFH (mm)	4958	
AFH (mm)	6996	
UFH (mm)	332	
LFH (mm)	982	
UFH %	170	
LFH %	170	
Cd-Gn (mm)	252	
Ar-Go-Me °	150	
FMA°	744	

- For the variable Is-Ii °: total of 2180 subjects, 1090 SBs and 1090 control subjects
- For the variable PFH: total of 4958 subjects, 2479 SBs and 2479 control subjects
- For the variable AFH: total of 6996 subjects, 3498 SBs and 3498 control subjects
- For the variable LFH: total of 982 subjects, 491 SBs and 491 control subjects
- For the variable FMA: total of 744 subjects, 372 SBs and 372 control subjects.

For these variables, if such large populations are needed to prove that differences exist between the sleep bruxer group and the control group, then the real differences may very well be negligible.

The sample size calculations for the following variables revealed realistic sample sizes could be realized:

• For the variable Cf-Go: total of 226 subjects, 113 SBs and 113 control subjects

- For the variable UFH: total of 332 subjects, 166 SBs and 166 control subjects
- For the variable UFH %: total of 170 subjects, 85 SBs and 85 control subjects
- For the variable LFH%: total of 170 subjects, 85 SBs and 85 control subjects
- For the variable Cd-Gn: total of 252 subjects, 146 SBs and 146 control subjects
- For the variable Ar-G-Me °: total of 150 subjects, 75 SBs and 75 control subjects.

The population size needed to prove that real differences may exist between the two groups is much more feasible for these variables. Moreover, the size of these numbers suggest that real differences between the two groups, with respect to certain variables may exist.

Therefore, we can conclude that for certain variables, the lack of significant findings in this study may be partly due to our sample size.

SECTION 4.3: LIMITATIONS OF METHODOLOGY

This study has some methodological limitations. Ideally we would have done the study with a completely blind protocol. We have to highlight that none of the other similar studies reviewed were done in this manner. One of the major difficulties was to have the model and cephalogram measures done by another person than the one who collected the data; it was very easy knowing the hypothesis, to recognize the bruxism-grinding wear pattern of the SBs. Ideally, one could have used a non-dental trained auxiliary, naïve to the study objectives, for this task. Even if this could have been an interesting alternative, it is at risk of introducing measurement errors. This was shown by Gravely and Benzies (1974) who assessed cephalometric tracing errors; they found that the errors made by the two experienced orthodontists were similar, while the ones made by a "well" trained laboratory technician were significantly higher. It is unknown if this is due to training quality or low precision in performing such tedious and long measurements.

Other limits of this study include the time elapsed (ranging from 24 months to 5 years) between the initial patient screening and polysomnographic recordings, and the collection of dento-cranio data. To reduce the impact of this problem and to maximize the validity of our data, reassessment of selection criteria were re-introduced (see Materials and Methods

chapter) at the time where subjects participated in the measurement session.

Moreover, a questionnaire was re-administered to confirm the presence or absence of initially reported oral habits (see Appendix Four).

SECTION 4.4: CONCLUSION

In conclusion, besides the clinical examination and polysomnographic variables, none of the oro-morphological variables seem to discriminate the SBs from control subjects. For certain variables, the lack of significant findings in this study may be partly due to our sample size. As well, it may be due to the capacity of the human system to accommodate to parafunctional habits and activities before permanent morphological changes take place (Graf, 1969). Perhaps the lack of findings or significant association between dentofacial morphology and bruxism implies that the etiology of bruxism may not be structurally related, which would lend credence to the hypothesis that bruxism is of psycho-reactive origin and/or results from central nervous system influences, rather than due to only morphological features (Menapace et al., 1994). Therefore, even though we postulate that orofacial morphology is probably not an etiologic factor in initiating bruxism, perhaps some morphological changes may occur secondary to parafunctional activity, as evidenced by subjects with severe attrition and loss of vertical dimension have been seen with modified morphological characteristics (Kiliardis, 1995; Kiliardis *et al.*, 1995; Waltimo, 1994); the Inuit population exhibit a changed morphology secondarily to their masticatory habits (Hylander, 1977; Mayhall, 1977); Obstructive sleep apnea subjects have changes in their morphology which may precede or be concomitant with their sleep apnea (Lowe *et al.*, 1986; Marklund *et al.*, 1998).

Finally, if one considers that it is important to assess the influence of sleep bruxism on dento-cranio facial morphology, a longitudinal study with a large sample size would be ideal but this would incur significant costs. One must evaluate the validity of such a project since bruxism is a low impact health problem compared to life threatening problems (such as sleep apnea), although it remains an interesting academic question.

CHAPTER FIVE

BIBLIOGRAPHY

Ash M (1984). Wheeler's dental anatomy, physiology, and occlusion. Philadelphia: W. B. Saunders, 415.

Attanasio R (1991). Noctural bruxism and its clinical management. *Dent. Clin. North Am.* 35(1):245-252.

Bader GG, Kampe T, Tagdae T, Karisson S, Blomqvist M (1997). Descriptive physiological data on a sleep bruxism population. *Sleep* 20:982-990.

Braun S, Bantleon H-P, Hnat WP, Freudenthaler JW, Marcotte MR, Johnson BE. (1995). A study of bite force, part 1: Relationship to various physical characteristics. *Angle Orthod*. 65(5):365-372.

Boyer F (1998). Le ronflement et le syndrome d'apnee obstructive du sommeil. *Med Que*. 33(3):45-50.

Cassidy KM, Harris EF, Tolley EA, Keim RG (1998). Genetic influence on dental arch form in orthodontic patients. *Angle Orthod*. 68(5): 445-454.

Clark GT, Adler RC (1985). A critical evaluation of occlusal therapy: Occlusal adjustment procedures. J. Am. Dent. Assoc. 110:743-750.

Colby WB, Cleall JF (1974). Cephalometric analysis of the craniofacial region of the Northern Foxe Basin Eskimo. *Am. J. Phys. Anthrop.* 40: 159-170.

Crothers AJR (1992). Tooth wear and facial morphology. J. Dent. 20(6):333-341.

Deyer RA, Diehr P, Patrick DL (1991). Reproductibility and responsiveness of health status measures. Statistics and stategies for evaluation. *Controlled Clinical Trials* 12:142S-158S.

Ellison JA, Stanziani P (1993). SSRI-associated nocturnal bruxism in four patients. *J. Clin. Psychiatry* 54:432-434.

Etzel KR, Stockstill JW, Rugh JD, Fisher JG (1991). Tryptophan supplementation for nocturnal bruxism: Report of negative results. *J. Cranjomand. Dis. Fac. Oral Pain* 5:115-119.

Faul F, Rerdfelder E (1992). GPOWER: a priori, post-hoc, and compromise power analyses for MS-DOS (computer program). Bonn, FRG: Bonn University, Department of Psychology.

Ferraio VF, Sforza C, Schmitz JH, Colombo A (1998). Quantitative description of the morphology of the human palate by a mathematecal equation. *Cleft Palate Craniofac. J.* 35(5):396-401.

Fogle LL, Glaros AG (1995). Contributions of facial morphology, age, and gender to EMG activity under biting and resting conditions: a canonical correlation analysis. *J. Dent. Res.* 74(8):1496-1500.

Gravely JF, Benzies PM (1974). The clinical significance of tracing error in cephalometry. *Br. J. Orthod.* 1(3):95-101.

Graf H (1969). Bruxism. Dent. Clin. North Am. 13(3):659-665.

Hartmann E (1994). Bruxism. In: Kryger MH, Roth T, Dement WC, eds. Principles and practice of sleep medicine. 2nd Ed. Saunders, 598-601.

Hicks RA, Conti PA, Bragg HR.(1990). Increases in nocturnal bruxism among college students implicate stress. *Med. Hypotheses* 33:239-240.

Hicks RA, Chancellor C (1987). Nocturnal bruxism and Type A-B behaviour in college students. *Psychol. Rep.* 60:1211-1214.

Hinton RJ (1981). Changes in articular eminence morphology with dental function. *Am.J. Phys. Anthrop.* 54: 439-455.

Hori A, Hirose G (1995). Twin studies on parasomnias. Sleep Res. 24A:324.

Hublin C, Kaprio J, Partinen M, Koskenvuo M (1998). Sleep bruxism based on self-report in a nationwide twin cohort. *J. Sleep Res.* 7:61-67.

Hylander WL (1977). The adaptive significance of Eskimo craniofacial morphology. In Dahlbeg AA, Graber TM, eds. Orofacial growth and development. The Hague: Mouton Publishers, 129-169.

Jarabak JR, Fizzell JA (1972). Volume one: technique and treatment with light-wire edgewise appliances. St. Louis: C.V. Mosby, 128-167.

Kiliaridis S (1995). Masticatory muscle influence on craniofacial growth. *Acta Odontol. Scand.* 53:196-202.

Kiliaridis S, Carlsson GE (1994). Bruxing and craniofacial growth. *Angle Orthod*. 64(4):244-245.

Kiliaridis S, Johansson A, Haraldson T, Omar R, Carlsson GE (1995). Craniofacial morphology, occlusal traits, and bite force in persons with advanced occlusal tooth wear. *Am. J. Orthod. Dentofac. Orthop.* 107:286-292.

Kiliaridis S, Mejersjö C, Thilander B (1989). Muscle function and craniofacial morphology: a clinical study in patients with myotonic dystrophy. *Eur. J. Orthod.* 11:131-138.

Lavigne GJ, Lobbezoo F, Montplaisir JY (1995). The genesis of rhythmic masticatory muscle activity and bruxism during sleep. In: Brain and oral functions. Morimoto T, Matsuya T, Takada K, eds. Elsevier Science B.V.; 249-255.

Lavigne GJ, Manzini C (1998). Sleep bruxism and concomitant oro-motor activity. Submitted.

Lavigne GJ, Montplaisir JV (1994). Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. *Sleep* 17(8):739-743.

Lavigne GJ, Montplaisir JV (1995). Bruxism: epidemiology, diagnosis, pathophysiology, and pharmacology. In: Orofacial pain and temporomandibular disorders. Fricton JR, Dubner RB, editors. New York: Raven Press, 387-404.

Lavigne GJ, Rompre PH, Montplaisir JY (1996). Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. *J. Dent. Res.* 75(1):546-552.

Lavigne GJ, Rompre PH, Poirier G, Gosselin A, Nicolas A, Montplaisir JY (1998). Rythmic masticatory muscle activity (RMMA or chewing-automatism) during sleep in normal controls. *J. Sleep Res.* 7(2):151, no 302 (Abstract).

Lindqvist B (1974). Bruxism in twins. Acta Odont. Scand. 32:177-187.

Lobbezoo F, Lavigne GT, Tanguay R, Montplaisir J (1997). The effect of catecholamine precursor L-dopa on sleep bruxism: A controlled clinical trial. *Movement Disorders* 12:73-78.

Lobbezoo F, Soucy JP, Hartman NG, Montplaisir J, Lavigne GJ (1997). Effects of the D2 receptor agonist Bromocriptine on sleep bruxism: Report of two single-patient clinical trial. *J. Dent. Res.* 76:1610-1614.

Lobbezoo F, Soucy JP, Montplaisir J, Lavigne GJ (1996). Striatal D2 receptor binding in sleep bruxism: A controlled study with iodine-123-iodobanzamide and single photon emission computed tomography. *J. Dent. Res.* 75:1804-1810.

Lowe AA, Santamaria JD, Fleetham JA, Price C (1986). Facial morphology and obstructive sleep apnea. Am. J. Orthod. Dentofac. Orthop. 90:484-491.

Macaluso GM, Guerra P, Di Giovanni G, Boselli M, Parrino L, Terzano MG (1998). Sleep bruxism is a disorder related to periodic arousals during sleep. *J. Dent. Res.* 77 (4): 565-573.

Magee KR (1970). Bruxism related to levodopa therapy. J. Am. Dent. Ass. 214:147.

Marbach J, Raphael G, Dohrenwend P, Lennon C (1990). The validity of tooth grinding measures: etiology of pain dysfunction syndrome revisited. *J. Am. Dent. Ass.* 120:327-333.

Marklund M, Franklin KA, Stenlund H, Persson M (1998). Madibular morphology and the efficiency of a mandibular advancement device in patients with slerep apnoea. *Eur. J. Oral Sci.* 106:914-921.

Mayhall JT (1977). The oral health of a Canadian Inuit community: an anthropological approach. *J. Dent. Res.* 56: C55-C61.

McNamara JA. A method of cephalometric evaluation. *Am. J. Orthod.* 86(6):449-469.

Menapace SE, Rinchuse DJ, Zullo T, Pierce CJ, Shnorhokian H (1994). The dentofacial morphology of bruxers versus non-bruxers. *Angle Orthod*. 64(1):43-52.

Migraine D, Scholte AM, Tremblay R, Montplaisir J, Lavigne GJ (1996). Bruxism and onycophagy in children: Prevalence and relative risk factors. *J. Dent. Res.* 75:219 - #1610 (Abstract).

Mohamed SE, Christensen LV, Penchas J (1997). A randomized double-blind clinical trail of the effect of Amitriptyline on nocturnal masseteric motor activity (sleep bruxism). *J. Craniomandib. Pract.* 15:326-332.

Moses CH (1968). Tooth forms and masticatory mechanisms of natural and artificial teeth. *J. Prosthetic Dent.* 19(1): 22-35.

Okeson JP (1996). Management of temporomandibular disorders and occlusion. St. Louis: C.V. Mosby, 152-169.

Owen AH (1984). Diagnostic block cephalometrics part 1. *J Clinical Orthod.* 18(6):400-422.

Ozbek MM, Memikoglu TUT, Gogen H, Lowe AA, Baspinar E (1998). Oropharayngeal airway dimensions and functional-orthopedic treatment in skeletal Class II cases. *Angle Orthod.* 68(4):327-336.

Pierce CJ, Chrisman K, Bennett ME, Close JM (1995). Stress, anticipatory stress, and psychologic measures related to sleep bruxism. *J. Orofacial Pain* 9(1):51-56.

Pingitore MA, Chrobak V, Petrie J. (1991) The social and psychological factors of bruxism. *J. Prosthet. Dent.* 65:443-446.

Por CH, Watson L, Doucette D, Dolovich L (1996). Sertraline-associated bruxism. *Can. J. Clin. Pharmacol.* 3:123-125.

Pracharktam N, Hans MG, Strohl KP, Redline S (1994). Upright and supine cephalometric evaluation of obstructive sleep apnea syndrome and snoring subjects. *Angle Orthod*. 64(1):63-74.

Proffit WR (1986). Contemporary Orthodontics. St. Louis: C.V. Mosby, 4-8.

Rakosi T (1982). An atlas and manual of cephalometric radiography. Philadelphia: Lea & Febiger, 25-68.

Reding GR, Rubright WC, Rechtschaffen A, Daniels RS (1964). Sleep pattern of tooth grinding: Its relationship to dreaming. *Science* 145:725-726.

Reding GR, Zepelin H, Robinson JE Jr, Smith VH, Zimmerman SO (1967). Sleep pattern of bruxism: A revision. *APSS Meeting* abstract 396.

Reding GR, Zepelin H, Robinson JE Jr, Smith VH, Zimmerman SO (1968). Nocturnal teeth grinding: All night psychophysiologic studies. *J. Dent. Res.* 47:786-797.

Rugh JD, Harlan J (1988). Nocturnal bruxism and temporomandibular disorders. In: Jankovic J, Tolosa E, eds. Advances in Neurology, Volume 49: Facial Dyskinesias. New York: Raven Press, 329-341.

Satoh T, Harada Y (1973). Electrophysiological study on tooth grinding during sleep. *Electroencephaly Clin. Neurophysiol.* 35:267-275.

Savoye I, Loos R, Carels C, Derom C, Vlietinck R (1998). A genetic study of anteroposterior and vertical facial proportions using model-fitting. *Angle Orthod.* 68(5):467-470.

Siriwat PP, Jarabak JR (1985). Malocclusion and facial morphology: is there a relationship? *Angle Orthod.* 55(2):127-138.

Solow B, Tallgren A (1971). Natural head position in standing subjects. *Acta Odont. Scand.* 29:591-607.

Solow B, Tallgren A (1976). Head posture and craniofacial morphology. *Am. J. Phys. Anthrop.* 44:417-436.

Spilker B (1991). Guide to clinical trials. New York: Raven Press, 528-535.

Terzano MG, Parrino L (1993). Clinical applications of cyclic alternating pattern. *Physiology & Behaviour* 54:807-813.

Terzano MG, Parrino L, Fioriti G, Orofiamma B, Depoortere H (1990). Modifications of sleep structures induced by increasing level of acoustic perturbation in normal subjects. *Electroenceph. Clin. Neurophysiol.* 76:29-38.

Terzano MG, Parrino L, Mennuni G (1997). Phasic events and microstructure of sleep. Lecie, Italy: Ass. Ital. Med. Del Sonno.

Thompson BA, Blount BW, Krumholz TS (1994). Treatment approaches to bruxism. *Am. Family Physician* 49(7):1617-1622.

Thorpy MJ (1990). Diagnostic and coding manual. In: Parasomnia: International classification of sleep disorders. Rochester, Minnesota: American Sleep Disorders Association, 142-185.

Ueda HM, Ishizuka Y, Miyamoto K, Morimoto N, and Tanne K (1998). Relationship between masticatory muscle activity and vertical craniofacial morphology. *Angle Orthod.* 68(3)233-238.

Velly-Miguel Am, Montplaisir J, Rompre PH, Lund JP, Lavigne GJ (1992). Bruxism and other orofacial movements during sleep. *J Craniomandib Dis Fac Oral Pain* 6:71-81.

Waltimo A, Nyström M, Könönen M (1994). Bite force and dentofacial morphology in men with severe dental attrition. *Scand. J. Dent. Res.* 102:92-96.

Ware JC, Rugh JD (1988). Destructive bruxism: Sleep stage relationship. *Sleep* 11:172-181.

Wieselmann G, Permann R, Korner E, Flooh E, Reinhart B, Moser F, Lechner H (1986). Distribution of muscle activity during sleep in bruxism. *Eur. Neurol.* 25: suppl. 2, 111-116.

Young DV, Rinchuse DJ, Pierce CJ, Zullo T (1999). The craniofacial morphology of bruxers versus nonbruxers. *Angle Orthod.* 69(1)14-18.

Zucconi M, Oldani A, Ferini-Strambi L, Smirne S (1995). Arousal fluctuations in non-rapid eye movement parasomnias; the role of cyclic alternating pattern as a measure of sleep instability. *J. Clin. Neurophysiol.* 12:147-154.

APPENDIX ONE

CONSENT FORM

HOPITAL NOTRE-DAME

Étude d'imagerie des récepteurs dopaminergiques chez les sujets souffrant des désordres du mouvement au cours du sommeil du bruxisme de narcolepsie

Protocole: 92 sml; SPECT- sommeil

Formule de consentement

INTRODUCTION

Nous vous invitons à participer à une étude clinique en collaboration entre le Département de médecine nucléaire de l'Hôpital Notre-Dame, le Centre d'étude du sommeil de l'Hôpital du Sacré-Coeur et la Faculté de médecine dentaire de l'Université de Montréal. Il est important que vous compreniez certains principes généraux qui s'appliquent à toutes les personnes qui participent à nos études:

- votre participation est entièrement volontaire;
- il est possible que vous ne puissiez pas retirer de bénéfices personnels en participant à cette étude, toutefois, vous contribuez à améliorer la qualité des soins prodigués aux personnes souffrant des conditions ci-haut énumérées;
- vous êtes libre, en tout temps, de vous retirer de cette étude, ce sans aucun préjudice ou pénalité de quelque nature;
- votre participation est confidentielle;
- la confidentialité sera aussi de rigueur lors de la présentation des résultats de cette étuce aux congrès ou dans des journaux scientifiques;
- la nature de l'étude, les risques, inconvénients et inconforts associés à la présente étude sont expliqués dans le texte qui suit.

BUT DE L'ÉTUDE

Cette étude a pour but d'identifier les sites où une des substances chimiques de votre cerveau (la dopamine) se localise. Nous espérons être en mesure de mieux comprendre les désordres du mouvement au cours du sommeil et la narcolepsie, et de découvrir la thérapie la plus apte à améliorer ces conditions.

VOTRE PARTICIPATION

Vous recevrez un médicament (IBZM; iodométhylbezamide: similaire à la dopamine), qui associé à de l'iode (I-123) radioactif, nous permettra de filmer les sites où la substance dopaminergique se situe dans votre cerveau. Cette substance semble avoir un rôle dans plusieurs troubles du sommeil et du mouvement. Pour la première visite, nous évaluerons votre condition médicale et dentaire pour déterminer si vous répondez aux critères de notre étude. Votre consentement à participer à la présente étude sera confirmé par la signature de ce formulaire.

Vous aurez à participer à une séance (ou deux si vous êtes un sujet témoin) de 3 heures. Cette étude est effectuée, sous la supervision d'un médecin, par une technicienne de l'Hôpital Notre-Dame.

- Le rendez-vous aura lieu selon un horaire pré-déterminé avec vous. Votre participation est essentielle au succès de l'étude.
- De plus, au cours de l'étude, il est possible que nous vous demandions de remplir différents questionnaires afin de mieux évaluer votre condition.

INCONVÉNIENTS

Voici les inconvénients associés à cette étude:

- vous ne pouvez participer à cette étude si vous êtes allergiques à l'iode ou produits similaires.
- vous ne pouvez pas avoir recours à des médicaments ou à une autre forme de traitement 2 jours avant la séance expérimentale.
- une légère irritation peut survenir au site où l'injection est effectuée; cet inconfort est normalement passager.
- L'I-123-IBZM est une substance radiochimique employée à des doses traceuses, elle ne présente pas de problèmes de toxicité et n'induit aucune réaction pharmacologique connu à ce jour. Les doses de radiation estimées, chez des sujets sains, sont dans les limites acceptables.
- Il n'y a a normalement aucun risque prévu pour votre santé. Nous utiliserons les meilleures conditions possibles pour assurer votre sécurité et confort.

SOINS EN CAS D'URGENCE

Il n'y a normalement aucune urgence prévue avec ce type d'étude, toutefois, si c'était le cas, vous devrez en informer un des membres de l'équipe de recherche aux numéros joints à la page suivante.

HOPITAL DU SACRE-COEUR ET FACULTE DE MEDECINE DENTAIRE, UNIVERSITE DE MONTREAL

PERIODIQUES DES MEMBRES INFERIEURS AU COURS DU SOMMEIL

PROTOCOLE #91-ML-BRUX

FORMULE DE CONSENTEMENT POUR SUJETS RECRUTES A L'UNIVERSITE DE MONTREAL (sujet bruxeur)

INTRODUCTION

Nous vous invitons à participer à une étude clinique à titre de sujet bruxeur, en collaboration avec le "Centre d'étude du sommeil de l'Hôpital du Sacré-Coeur". Il est important que vous compreniez certains principes généraux qui s'appliquent à toutes les personnes qui participent à nos études:

- votre participation est entièrement volontaire;
- il est possible que vous ne puissiez pas retirer de bénéfices personnels en participant à cette étude. Toutefois, par votre participation, vous contribuez à améliorer la qualité des soins prodigués aux personnes souffrant de bruxisme (serrement et grincement des dents);
- vous êtes libre, en tout temps, de vous retirer de cette étude sans aucun préjudice ou pénalité de quelque nature;
- votre participation est confidentielle;
- la confidentialité sera aussi de rigueur lors de la présentation des résultats de cette étude aux congrès ou dans des journaux scientifiques (Ex.: enregistrement, vidéo, photos, etc...);

- la nature de l'étude, les risques, inconvénients et inconforts associés à la présente étude sont expliqués dans le texte qui suit.

BUT DE L'ETUDE

Cette étude a pour but d'étudier la relation qui existe entre l'activité de certains muscles (appareil masticateur, cou et jambes) et les phases du sommeil chez les sujets atteints d'épisodes de bruxisme. Une meilleure compréhension de ce phénomène nous aidera à découvrir la thérapie la plus apte à améliorer cette condition.

VOTRE PARTICIPATION

Vous n'aurez <u>aucun médicament</u> à prendre pour participer à cette étude.

Pour la première visite, nous évaluerons votre condition dentaire et médicale pour déterminer si vous répondez aux critères d'inclusion de notre étude. Vous devrez compléter les questionnaires ci-joints et une radiographie de type panoramique datant de moins de 3 mois sera requise. Votre consentement à participer à la présente étude sera confirmé par la signature de ce formulaire.

Vous aurez à participer pour 2 nuits ou plus (avec votre accord) à des séances où sera enregistrée (par des électrodes collées à votre peau) l'activité électrique de votre cerveau et de vos muscles. Ces enregistrements sont effectués, sous la supervision d'un médecin, par une technicienne en électrophysiologie à la Clinique du sommeil de l'Hôpital Sacré-Coeur.

- Le rendez-vous aura lieu selon un horaire pré-déterminé avec vous. Votre participation est essentielle au succès de l'étude.
- De plus, au cours de l'étude, nous vous demanderons de remplir différents questionnaires afin de mieux évaluer votre condition.

INCONVENIENTS

Voici les inconvénients associés à cette étude:

- Vous ne pouvez pas avoir recours à des médicaments ou à une autre forme de traitement pendant la durée de l'étude; toutefois si votre état nécessite un traitement, nous vous suggérerons, au meilleur de notre connaissance, une thérapie alternative.
- Les électrodes collées à la surface de votre peau peuvent créer une légère rougeur. Une telle irritation est de courte durée et sans conséquence.
- Le fait de dormir deux nuits ou plus à l'Hôpital peut vous occasionner certains problèmes pour votre travail. Une lettre explicative pourra être remise à votre employeur pour justifier votre absence.
- Il n'y a normalement aucun risque prévu pour votre santé. Nous utilisons les meilleurs conditions possibles pour assurer votre sécurité et confort.

N'oubliez pas que vous contribuez à améliorer la qualité des soins pour des personnes qui souffrent d'une condition identique à la vôtre.

SOINS EN CAS D'URGENCE

Il n'y a normalement aucune urgence prévue avec ce type d'étude, toutefois si c'était le cas, vous devrez suivre ce qui suit: pendant toute la durée de l'étude, si vous éprouvez de la douleur intense ou des malaises, veuillez en informer un des membres de l'équipe de recherche.

CONSENTEMENT DU PATIENT

Je déclare avoir lu, compris et discuté les explications concernant cette étude, et que j'ai eu l'occasion de discuter des points qui me semblaient confus.

J'ai complété les questionnaires médical et dentaire ci-joints, et j'informerai les responsables du projet de tout changement à mon état médical ou dentaire pouvant survenir en cours d'étude. Je certifie avoir signé ce formulaire sans pression et en toute liberté. J'accepte de participer volontairement à la présente étude et d'en assumer les inconvénients et les risques inhérents.

Nom du patient:	4 12
Date de naissance:	
Signature du patient:	
Date:	
Témoin:	
Dentiste chercheur:	
Signé à Montréal, Québec, le/_	

Pour toute question concernant cette étude, veuillez contacter:

Dr G. Lavigne (Université de Montréal): 343-2310 Pierre Rompré, MSc (Université de Montréal): 343-6111 ext. 3439 Dr J. Montplaisir (Hôpital du Sacré-Coeur): 338-2693 Formule de consentement - brucisme bromocriptine/placebo

SOINS EN CAS D'URGENCE

Il n'y a normalement aucune urgence prévue avec ce type d'étude, toutefois, si c'était le cas, vous devrez suivre ce qui suit: pendant toute la durée de l'étude, si vous éprouvez de la douleur ou des malaises, veuillez en informer un des membres de l'équipe de recherche par le télé-avertisseur (# à veuit). Si un malaise survient après votre départ du Centre d'étude du sommeil, contactez nous ou rendez vous à l'urgence de l'Hôpital du Sacré-Coeur (ou à l'Hôpital le plus près de chez vous, ou au 911) et s.v.p. téléphonez nous dès que possible.

CONSENTEMENT DU PATIENT

Je déclare avoir lu et compris les explications concernant cette étude, et avoir eu l'occasion de discuter des points qui me semblaient confus.

J'ai complété les questionnaires médicaux et dentaires ci-joints, et j'informerai les responsables du projet de tout changement à mon état médical ou dentaire pouvant survenir en cours d'étude. Je certifie avoir signé ce formulaire sans pression et en toute liberté. J'accepte de participer volontairement à la présente étude et d'en assumer les inconvénients et les risques inhérents.

Nom du patient:	
Date de naissance:	
Signature du patient:	
Date:	
Témoin:	
Dentiste chercheur:	
Signé à Montréal, le:/// En acceptant de participer à cette étude, je ne reno	once à aucun des droits normalement
reconnus à tout sujet inscrit dans un protocole de re	chérche.
Pour toute information supplémentaire concernant o	cette étude, veuillez contacter:
 Dr Gilles Lavigne (Université de Montréal) 	343-2310
Dr Frank Lobbezoo (Université de Montréal)	343-6111 P. 3464 ou télé-avertisseu
 Dr Jacques Montplaisir (Hôpital Sacré-Coeur) 	338-2693
TROUBLE HAPITAL SACRÉ-COEUR (5400 GOU	IN OUEST): 338-2000

NOM:	
22	
Par la présente, je confirme a	u service des finances de l'Université de
Montréal que j'ai participé, à titre	e de volontaire, à un projet de recherche de
l'équipe du Dr. Gilles Lavigne de	la Faculté de médecine dentaire.
Auriez-vous l'obligeance de me	faire parvenir la somme prévue pour mon
dédommagement à l'adresse su	ivante:
40	
ADRESSE:	
Noticode.	
VILLE:	
CODE POSTAL	
SIGNATULE	
Signé à Montréal, le	

APPENDIX TWO

MEDICAL HISTORY

and

PATIENT QUESTIONNAIRE



DOSSIER DENTAIRE QUESTIONNAIRE D'INSCRIPTION CONFIDENTIEL



: M F Alon:	the factor of	Prinom	
ise: No: 1706:	and the state of t	App. Vitie:	
postal: Tél: Dom.:		Trev.: (Poste) Poids:	
The state of the s			
	100	OU Taille:	1.02
	MACAL S		
	No. ass. sc	oc.;	
isé(e) par:		V. A. 1977 7-78-25-77-10-10-10-10-10-10-10-10-10-10-10-10-10-	
ION DE LA VISITE	Miles Septim		
in the second	IISTOIRI	E MÉDICALE	OUI NO
les-vous actuellement sous les soins d'un médecin?		26. Maux d'oralles	
		27. Rhume des foins	
Prénom		28. Asthme 29. Furnez-vous?	
Poste		30. Avez-vous déjà subi des traitements de radiothéraple et / ou	
	OUI NO	chimicthérapie (tumeur)	
enez-vous (présentement) des médicaments ou en avez-vous pris au	As was a	32. Éles vous séro-positif au test du SIDA?	ōō
urs des 6 demiers mois?oul, lesquels?		33. Avez-vous des prothèses articulaires (hanches, genoux, etc.)	
es-vous enceinte?	0 0	OUI NON	OUI NO
enez-vous des anovulants? (pilule anticonceptionnelle)	-	Aliments	
rous souffert ou souffrez-vous de? oubles cardiaques (infarctus, angine, problèmes valvulaires, souffie)	1 4	Pěricifilne Codéine	_
interes cardiaques (interctus, angine, problemes varvualres, soume)		* lode	
ilgnements prolongés	5.5	35. Avez-vous déjà été hospitalisé et / ou subi des interventions chirurgicale	es autres qu
rémie		dentaires? - Si oui, lesquelles? et quand?	
umes fréquents ou sinusite	1 1		and the second
berculose ou problèmes pulmonaires		19	
xubles digestifs		26 Coulcillon up to discuss de texte de	OUI NON
oblèmes du foie (hépatite : virus A, B, C, cirrhose, etc.)		36. Souhaftez-vous discuter de votre santé en privé avec votre dentiste? Remarques:	
xubies du rein		A received to Mary at	
Italies vénériennes (MTS)		Section 2000	Trans.
xubles thyroldiens	55	NASTALA PASSA DE E	
ladies de la peau		À L'USAGE DU PROFESSIONNEL:	
blèmes oculaires (yeux)		ATTENTION!	
llepsie			
xubles nerveux	0 0		-
xurdissements, évanouissements			
	12 34		
revisite: 0-6 m	ISTOIRE	DENTAIRE	
ients recus	47		
- Indiana in the second of the	15, 3		
ous déjà eu des traitements dentaires tels que:	OUI NON	하는 사람들은 사람들이 가득하는 것들다는 것이 없는 사람들이 되었다. 그런 사람들이 있는 것이 없는 것이 없는 것이다. 그런 사람들이 없는 것이다. 그런 사람들이 없는 것이다. 그런 사람들이 없는 사람들이 없는 것이다. 그런	OUI NON
monstration d'hygiène buccale		7. Prothèses complètes et / ou partielles	
itement d'orthodontie		8. Traitement de chirurgie buccale ou extractions mplants dentaires	
homent de canal		10. Radiographies dentaires	
uronne(s) et / ou pont(s)		11. Autres	
	0.0		
ssigné, déclare avoir lu, compris, m'être reneeigné et avoir répondu au qui dentaire ci-dessus au meilleur de ma connaissance. Je m'engage par la prése	uestionnaire ente, à vous	Je reconnais avoir pris connaissance des réponses au questionnaire d'int	roduction e
e tout changement de mon état de santé. le la constitution de mon dossier dentaire, son suivi, ainsi que mon inscription		avoir pris les mesures d'usage, le cas échéant.	
el du (des) dentiste(s) traitant(s).	- A - 4	Signature	
informé(e) que mon dosaler sera conservé au cabinet en tout temps et que it son(leur) personnel auxiliaire y aura(auront) seul(e) accès.		Deniliste tratant	
aussi informé(e) de mon droit de consulter mon dossier, d'y demander une se retirer de la fiste de rappel.	rectification,	Date/	/ An
	107/0		3477
Patient ou responsable	-	Company to the company of the compan	

Partie II. Fonction

1.	Est-ce que l'articulation de votre mâchoire craque (fait des bruits secs) quand vous ouvrez ou fermez ou lorsque vous mastiquez? □ non □ oui
2.	Est-ce que l'articulation de votre mâchoire fait un bruit de grattement (frottement) quand vous ouvrez ou fermez ou lorsque vous mastiquez? □ non □ oui
3	Est-ce que votre mâchoire se bloque de sorte que vous ne puissiez pas ouvrir normalement? non oui, parfois souvent Si oui, est-ce que c'est possible de débloquer votre mâchoire vous-même? non oui Si non, combien de temps le blocage dure-t-il généralement? quelques minutes 4 à 8 heures jusqu'à une heure jusqu'à quelques journées 1 à 4 heures continue
4.	Est-ce que votre mâchoire se bloque de sorte que vous ne puissiez pas fermer normalement? □ non □ oui, □ parfois □ souvent
5.	Mastiquez-vous ou sucez-vous vos lèvres, langue ou joues? □ jamais ou presque □ parfois □ souvent □ toujours ou presque Vos ongles? □ jamais ou presque □ parfois □ souvent □ toujours ou presque De la gomme à mâcher? □ jamais ou presque □ parfois □ souvent □ toujours ou presque Un crayon ou un stylo? □ jamais ou presque □ parfois □ souvent □ toujours ou presque
6.	En êtes-vous conscient de serrer des dents? □ non (passer à la question 7) □ oui, □ presque jamais □ parfois □ souvent □ toujours ou presque Pendant la journée? □ jamais ou presque □ parfois □ souvent □ toujours ou presque Et pendant la nuit? □ jamais ou presque □ parfois □ souvent □ toujours ou presque

7.	En êtes-vous conscient de grincer des dents?
	☐ non (passer à la question 8) ☐ oui, ☐ presque jamais ☐ parfois ☐ souvent ☐ toujours ou presque
	Pendant la journée?
	☐ jamais ou presque ☐ parfois ☐ souvent ☐ toujours ou presque
	Et pendant la nuit?
	☐ jamais ou presque ☐ parfois ☐ souvent ☐ toujours ou presque
	Cela vous dérange-t-il? ☐ jamais ou presque ☐ parfois ☐ souvent ☐ toujours ou presque Sioui, pourquoi?
	brout, postiquor:
	Depuis quand grincez-vous des dents? ☐ l'enfance ☐ l'adolescence ☐ l'âge adulte
	Faites-vous du bruit en grinçant des dents?
	Qui dans votre famille grince des dents? ☐ aucune personne ☐ père ☐ mère ☐ frère ☐ soeur ☐ enfant ☐ conjoint
8.	Selon vous, vos dents sont-elles usées? □ non □ oui, □ partiellement □ complètement
9.	Brisez-vous vos plombages ou couronnes? □ non □ oui, □ parfois □ souvent
10.	La façon dont vos dents ferment est-elle inconfortable ou différente de votre habitude?
11.	Avez-vous des prothèses (dentiers) en bouche? □ non (passez à la question 12)
	☐ oui, à la mâchoire ☐ supérieure ☐ inférieure ☐ les deux Depuis combien de temps?
	ans
	Est-ce que vous les portez durant le nuit? ☐ non
	□ oui, à la mâchoire □ supérieure □ inférieure □ les deux
12.	En général, qu'elle est votre posture lors du sommeil?
	☐ sur le ventre ☐ sur le côté droite ou gauche ☐ sur le dos ☐ variable ou je ne sais pas
	THE TOTAL TOTAL TOTAL STATE OF THE STATE OF

13.	Est-ce que votre mâchoire est endolorie ou raide lorsque vous vous réveillez la matin? ☐ jamais ou presque ☐ parfois ☐ souvent ☐ toujours ou presque					
14.	Ressentez-vous de la fatigu non (passer à la oui Lorsque vous vous réveillez jamais ou presque Et pendant la journée? jamais ou presque Et pendant le soir? jamais ou presque Comment cette fatigue varie augmente din	question 15 I la matin? Parfoi parfoi parfoi parfoi t-elle du n	s souvent s souvent s souvent s souvent natin au soir?	☐ toujours o ☐ toujours o ☐ toujours o	ou presque	ion?
15.	Au cours du dernier mois, i difficile:	indiquez jus	qu'à quel point	t votre problè	me de mâcho	ire rend
		pas du tout	un peu	modéré- ment	beaucoup	extrême- ment
a. b. c. d. e. f. g. h. i.	mastiquer boire exercices physiques manger des aliments durs manger des aliments mous la digestion nettoyer vos dents bâiller avaler parler			000000000		0000000000
17	5. Est-ce que vous ronfler la nuit jamais ou presque 7. Avez-vous l'habitude de mettre crayon, stylo, de	parfois □				
	☐ jamais ou presque {	□ parfois □	□ souvent □ to	oujours ou pre	esque	

Partie III. Santé en général

1.	Comment considérez-vous votre santé en général? ☐ mauvaise ☐ passable ☐ bonne ☐ très bonne	e 🗆 excellente				
2.	Veuillez faire une liste de tous les médicaments que vous prenez actuellement, incluan les médicaments obtenus sans prescriptions, en cochant la case de tous ce que vous prenez uniquement pour la douleur.					
		nombre de comprimés par jour	uniquement pour la douleur			
84						
3.	Avez-vous subi un traumatisme au cou, à la tête ou au vis non oui Si oui, expliquez					
4.	Avez-vous des maux de tête ou des migraines? ☐ jamais ou presque ☐ parfois ☐ souvent ☐ to	oujours ou presque				
5.	Avez-vous des maux au cou ou aux épaules? □ jamais ou presque □ parfois □ souvent □ to Et des limitations des mouvements du cou ou des épaules? □ jamais ou presque □ parfois □ souvent □ to					
5.	Avez-vous des maux au dos? ☐ jamais ou presque ☐ parfois ☐ souvent ☐ to	oujours ou presque	:			
7.	Souffrez-vous d'arthrite rhumatoïde, de lupus ou arthritique? □ non □ oui,					

8.	Avez-vous déjà eu ou avez-vous des articulations autre que celles de votre mâchoire qui enflent ou sont doloreuses? □ non □ oui,
9.	Avez-vous de la douleur ou d'autres plaintes aux oreilles? □ non □ oui,
10.	Pendant la journée, bougez-vous les jambes nerveusement? ☐ jamais ou presque ☐ parfois ☐ souvent ☐ toujours ou presque Au moment du coucher, diriez-vous qu'il vous arrive que des impatiences musculaires puissent retarder la venue de votre sommeil? ☐ jamais ou presque ☐ parfois ☐ souvent ☐ toujours ou presque Lorsque vous vous réveillez la nuit, diriez-vous qu'il vous arrive de ressentir des impatiences musculaire des jambes qui vous forcent à bouger ou à marcher pour vous soulager? ☐ jamais ou presque ☐ parfois ☐ souvent ☐ toujours ou presque
11.	Au cours du dernier mois, avez-vous eu des difficultés à digérer, des maux à l'estomac ou au ventre, ou de la constipation? □ non □ oui,
12.	Y a-t-il eu des gros changement dans votre vie privé(e) ou au travail lors des dernier six (6) mois? ☐ non ☐ oui Si oui, expliquez simplement
13.	Y a-t-il des choses ou des situations récentes qui vous irritent, qui vous déçoivent, ou qui vous sont ennuient? ☐ non ☐ oui Si oui, lesquels?
14.	Y a-t-il des problèmes dans votre environnement qui vous causent du souci? ☐ non ☐ oui Si oui, lesquels?

APPENDIX THREE

CLINICAL

SLEEP REPORT

schr0n2 br Dec 21,1993 22:13:32 SCHR0N2.EEG

```
range = 42 to 1438 (SUBSET) -- 0 unscored epochs in between length = 20.0 sec
```

onset defined as :

least 1 consecutive min of stage 1 - or .0 sec of stage 2,3,4 or REM

cies to stage 1 & 2 computed from lights out cies to stage 3,4 & REM computed from sleep onset

ng values are printed as "9999"

```
(minutes)
                        Arousals
                                Latencies (min)
Onset
             2.3
                       Arousals = 31
                                 [Sleep]
                                       2.3
Sleep Time
                 434.7
                       >= 1 \min = 3
                                   [1]
                                       2.3
mittent Awake
          =
                 28.3
                       >= 3 min =
                                   [2]
                              1
                                       10.0
Period Time
          =
            463.0
                 463.0
                       >= 5 min =
                                   [3]
                                       16.3
Morning Awake
          =
             0.3
                                   [4]
                                       18.7
Time Recorded
          =
            465.7
                      Sleep Eff.=
                              93.9%
                                  [REM]
                                       69.7
```

ion of stages in minutes and as a percentage of Total Sleep Time ent wake is based on duration of Sleep Period)

[1/3] [2/3] [3/3][Total] 0 min 0 min min min 7.3 1.7 ° 10.3 2.4 0 17.0 3.9 ° 34.7 8.0 ° ge 2 ° 53.0 12.2 ° 85.3 19.6 ° 70.7 16.3 ° 48.1 ° 209.0 ge 3° 30.7 7.1 ° 26.3 6.1 ° 0.3 0.1 ° 13.2 ° 57.3 ge 4 ° 57.0 13.1 ° 1.7 0.4 ° 0.0 ° 0.0 58.7 13.5 ° 0.6 ° ge R° 2.7 5.8 ° 25.3 10.8 ° 47.0 75.0 17.3 ° 150.7 tal 34.7 ° 149.0 34.3 ° 135.0 31.1 ° 434.7 100.0 ° 3.7 0.8 ° 5.3 1.2 ° 19.3 4.2 ° 28.3 6.1 °

```
s in percentage by thirds of night
  ° Stage 1 ° Stage 2 ° Stage 3 ° Stage 4 °
13
    12.9
          21.2
               25.4
                     53.5
                          97.2
                                3.6
  0
13
    18.8
          29.8
             0
               40.8
                                   0
                     45.9
                           2.8
                                33.8
13
    68.2
          49.0
               33.8
                     0.6
                           0.0
                                62.7
             0
    100.0
         100.0
               100.0
                        0
                    100.0
                          100.0
```

schr0n2 br Dec 21,1993 22:13:32 SCHR0N2.EEG

```
range = 42 to 1438 (SUBSET)
length = 20.0 sec
onset defined as:
```

least 1 consecutive min of stage 1 - or - .0 sec of stage 2,3,4 or REM

cies to stage 1 & 2 computed from lights out cies to stage 3,4 & REM computed from sleep onset

ng values are printed as "9999"

Recording Time: 466 min

Onset : 2 min (norm: < 30)

Sleep Time : 435 min

mittent Awake : 28 min (norm: < 30)

2 latency : 10 min 3 latency : 16 min 4 latency : 19 min

REM latency : 70 min (norm: 50-120)

° % of total Length(min) ° sleep time (norm) ° Stage 1 ° 34.7 ° 8.0 (0-20) ° Stage 2 ° 0 209.0 48.1 0 (45-65)° Stage 3 ° 57.3 0 13.2 (3-20)° Stage 4 ° 58.7 0 13.5 (0-15)° Stage R ° 75.0 17.3 (15-30)

```
r of REM periods: 3 (norm: 3-6) fficiency: 73.5 % (norm: > 80)
```

r of transitions into Stage 1 or Wake : 71 (norm: < 90)

r of arousals : 31 (norm: < 30)

r of arousals longer than 1 min : 3

ils of REM periods

periods are seperated by at least 15 min of NREM)

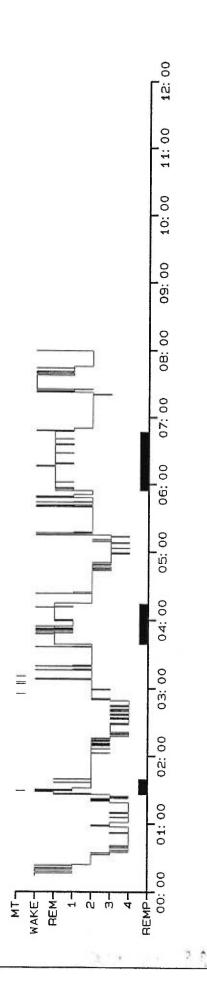
រង់ងង់ងងងងងងងងងងងងងងងងងងងងងងងងងងងងងងង							
	Interv	T REM	T NREM	T wake	T tot	Frag	REM Eff
1	69.7	2.7	10.0	1.0	13.7	4	19.5 %
2	133.0	25.3	7.7	2.7	35.7	6	71.0 %
3	135.3	47.0	5.0	0.7	52.7	10	89.2 %
lääääää	aááááááááá	ááááááááááá			ááááááááá	áááááá	áááááááááá
	112.7	75.0	22.7	4.3	102.0	20	73.5 %
ıaaaaā	aaaaaááááá	ááááááááááá	áááááááááá	áááááááááá	ááááááááá	ááááááá	áááááááááá

: transition matrix Number of transitions ° Trans/hour of sleep period R °Total° W 1 R ° Total° 1 ° 33 ° 13 376 13 ° 428 ° 13 ° 39 ° 156 168 ° 505 ° 5 ° 44 ° 168 65 ° 570 ° 1 ° 52 ° 13 ° 674 ° 0 0 27 ° 0 0 350 ° 0 ° 20 ° 0 ° 259 ° **ేష ప**డే ప్రవర్ణ ప

er of arousals longer than 3 min : 1 er of arousals longer than 5 min : 1 xliv

: 93.9 % (norm: > 90)

Efficiency : er of cycles during night : age cycle duration : 154 min



Stellate Systems - Licence E15 - Facult de mdecine dentaire - U de M