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Masticatory Muscles Activities in Temporomandibular Joint Internal Derangement

by

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DÉDICACE / DEDICATION

Cette thèse de Maîtrise est spécialement dédiée à mon fils Jérémi né au cours de ma dernière année de rédaction. En espérant qu'un jour il la lira en souvenir de moi et de son année de naissance.

De même, j'aimerais me dédier ce mémoire et son accomplissement pour avoir tenu jusqu'au bout malgré les choses de la vie.

PREFACE

Cette thèse est écrite en anglais pour plusieurs raisons pratiques malgré ma connaissance réduite de sa forme écrite: il était plus avantageux d'utiliser le langage scientifique pour ce domaine spécialisé; la majorité sinon la totalité des documents et la littérature utilisés sont en anglais; et les deux articles présentés seront soumis pour fin de publication dans des journaux scientifiques anglophones.

This thesis was written in English for different practical reasons even if my knowledge of its written form is limited: it was more convenient to use the existent English scientific terminology of this specific topic; the majority if not the totality of the documents and literature used are available in English; and the two articles presented here will be submitted to English scientific journals for publication.

This thesis is written in article format style which consists of two scientific articles and a general introduction and conclusion as well as extensive appendices. The appendix A consists of the traditional three first chapters of a master thesis: Introduction, Review of literature, Methodology and other appendices.

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Chantal

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TERMINOLOGY

Abbreviations

TMJ	: TemporoMandibular Joint
ID	: Internal Derangement
SLP	: Superior Lateral Pterygoid (muscle)
ILP	: Inferior Lateral Pterygoid (muscle)
LE	: Linear Envelope
EMG	: Electromyography
MVC	: Maximum Voluntary Contraction
OCC	: Open-Close-Clench (cycle)

Definitions

In order to understand the glossary of the Temporomandibular Joint, knowledge of the current specific vocabulary and meaning is mandatory. These definitions and explanations are as follow:

Temporomandibular Joint (TMJ). Class three lever articulation between the mandible and the cranium (Bourbon, 1988). It is described as a compound synovial joint with an intra-articular disc which divides the cavity into two distinct units: the upper joint remains a freely movable sliding joint (arthrodial) while the lower is converted into a pure hinge joint. It can also be termed as a "socket joint with a movable hinge joint" (Bell, 1983). The TMJ is described as bicondylar or ellipsoid in type. The TMJ involves the articular tubercle and the anterior portion of the mandibular fossa of the temporal bone above and the condyle of the mandible below (Gray's Anatomy, 1987). This term also refers as the cranio-mandibular complex or cranio-mandibular joint.

Dysfunction: is defined as disturbed, impaired or incomplete function (Butterworths Medical Dictionary, 1980). Synonyms of malfunction and parafunction.

Temporomandibular Joint Dysfunction. Temporomandibular joint and muscle dysfunction implying that the disorder takes place either in the muscles of mastication, the joints or both as long as there is a functional disorder. The condition includes the following clinical signs: pain, muscle and joint tenderness, joint sounds during condylar movements, limitation and/or uncoordinated mandibular movements. It differs from other terms in its diagnostic sense and do not need the presence of subjective symptoms. Also synonymous to TMJ syndrome or disorder.

Myofascial-pain dysfunction syndrome. This expression is defined as a chronic soft tissue fascial pain which refers to a masticatory fibromyalgia condition (Truta, 1989). Weinberg (1990) specified that this syndrome specifically eliminates joint involvement, and should be limited to early simple muscular pain disorders.

Craniomandibular and Temporomandibular (Joint) Disorders are more global diagnostic terms that include the whole variety of syndromes of the TMJ. Lately, clinicians and authors tend to categorise the whole scope of TMJ disorders into two etiologic and clinical patterns: Arthrogenous and Myogenous TMJ disorders. The American Academy of Craniomandibular Disorders defines craniomandibular disorders as a term embracing a number a clinical problems that involves the masticatory musculature, the TMJ or both. Craniomandibular disorders have been identified as a major cause of non-dental pain in the orofacial region and are considered to be a subclassification of musculoskeletal disorders (Bell, 1990).

Internal Derangement (ID). An intra-articular disc-condyle incoordination that invclves an abnormal relationship between the articular disc and the mandibular condyle, fossa and articular eminence without regards to condylar position. The disc is, in fact, displaced anteromedially (Benson, 1988). More specifically, we are dealing with an arthrogenous diagnostic with or without soft tissues involvement: clicking, local joint pain and tenderness, discal displacement and motion dysfunction.

Anterior Disc Displacement: relates to the majority of ID involving an abnormal disc displacement in the anterior joint space. The term "displacement" is used to describe disc position by opposition to dislocation which should be reserved for condylar position relative to joint structures. ADD can be viewed as a natural progression of events that occurs as a result of changes in the integrity of the disc-condyle assembly (Benson, 1988). The disc is displaced anteriorly and medially to a certain extend.

Lateral Pterygoid Muscles . One of the four masticatory muscles that lies deep to, and largely behind, the zygomatic arch. It is a short, thick muscle, somewhat conical in shape, that extends almost horizontally between the infratemporal fossa and the condyle of the mandible (Bourbon, 1988). The anatomy and functions of its two portions are discussed in chapter II, literature review (appendix A).

Superior Lateral Pterygoid (SLP): refers to the small upper head of the lateral pterygoid muscle. The fibres originate from the infra-temporal surface of the greater wing of the sphenoid bone, run backward and outward to insert onto the articular disc and the condylar head (Bell, 1983; Bourbon, 1988). Some authorities (Honee, 1972) refute the condyle connection and others refute the discal attachments (Carpentier, 1988).

Inferior Lateral Pterygoid (ILP): refers to the inferior head of the lateral pterygoid muscle. This

portion is three times larger than its counterpart, the SLP, and arises from the lateral lip of the lateral pterygoid plate. It then converges upward and outward to reunite with the SLP and insert onto the neck of the condyle.

Synergist Muscle: that actively provides an additive contribution to a particular function, movement or to another muscle in its action (Basmajian & DeLuca, 1985).

Co-contraction: Simultaneous increased contraction of muscles which are normally antagonist in their primary action. Both agonist and antagonist are activated simultaneously to increase muscle control on a joint (Basmajian & DeLuca, 1985). Usually used to stabilize a movement.

Agonist Muscle: initiate the desired contraction. It Is the prime mover or prime contributor of a movement or action (Basmajian & DeLuca, 1985).

Antagonist Muscle: that actively provides a negative or opposite contribution to a particular function or to the prime mover (Basmajian & DeLuca, 1985).

Postural Muscle: is a fatigue resistant and slow twitch muscle used for balance and postural tonus (Travell & Simmons, 1983)

Power Muscle: is a fast twitch muscle used for greater forces (Travell & Simmons, 1983)

Integrated LE EMG: Refers as the integration of the area under the curve of a linear envelop signal (second order low pass filter with a cut-off at 6Hz) from a full wave rectified raw EMG signal. In the present investigation the time of integration is one second.

INTRODUCTION

Masticatory Muscles Activities in Temporomandibular Joint Internal Derangement

This introduction will serve as an overview relating to this specific field and as a guide to place the reader in the context of the study. This general introduction of the two article format papers is combined with a general conclusion.

Literature Review: Overview

Historically, the first attempt to document a temporomandibular joint (TMJ) pathology was done in the 1930's by Costen: the spirit of TMJ syndrome arose. Today, craniomandibular practice is in a state of confusion primarily because there is no consensus as to the nature and cause of TMJ dysfunction. Unfortunately it places clinicians in a difficult position since they must face the problems, the patients and the underlying factors in the presence of controversial and often conflicting ideas concerning etiology, diagnosis and management of the TMJ (Farrar, 1983; Krauss, 1988).

Clinical and epidemiological investigators have shown that 41 to 79% of the general population have clinical signs of TMJ syndrome, from which 60 to 80% are female aged between 20 and 40 years old (Greene, 1982; Magnusson, 1986; Locker & Slade, 1988). The disorder presents some of the most complex and frustrating diagnostic and therapeutic problems encountered by the dental, medical and paramedical professions (Foreman, 1985). Hence, the majority of the patients are misdiagnosed or mistreated because of the lack of professional training and biomechanical knowledge, including muscle mechanics (Rocabado, 1981; Foreman, 1985).

It is generally agreed that the etiology of TMJ disorders is multifactorial (Schwartz, 1956; McNeil et al., 1980; Mikhail, 1980; Okeson, 1981; Greene, 1982; Foreman, 1985; Clark, 1987; Weinberg, 1980). On the other hand, psychophysiologic and "tooth/occlusion theories" supporters point to their unique causal factor (Schwartz, 1956; Laskin, 1969; Helkimo, 1975; Clarke, 1982;

the causal factors presented in the literature are: muscle hyperactivity, trauma, oral parafunction, nutritional and mechanical stresses, cranial fault, postural stress, congenital defects, environmental and developmental factors, psychological and physiological (chemical stress, systemic diseases) factors with dental work and malocclusion dominating literature (Owen, 1990). In parallel, an extensive list of predisposing, activating and perpetuating factors that repeats from the above list have also been proposed (McNeil 1980; Foreman, 1985). However the issue of the etiology of TMJ dysfunction remains unclear. Overall, the "muscle theory" or "neuromuscular theory" are predominant, whether it is primary or secondary (Carlsson, 1980; Bergamini, 1990): it has been the central feature of all the different theories. It is now accepted that the most common TMJ disorder is internal derangement (ID) or arthrogenous disorders; it represents 80% of all TMJ syndromes; this ID relates to an anterior (and medial) discal displacement process (Farrar, 1972; Weinberg, 1980; Dolwick, 1983; Rocabado, 1983; Isberg, 1985; Owen, 1987; Gage, 1989).

The lateral pterygoid muscle seems to be the cause of numerous temporomandibular disorders; in the human specie it is a muscle with an history of controversies. Although, small in size, it has a very large functional impact on the mechanics of the whole joint. Anatomically, the superior head of the lateral pterygoid muscle (SLP) is most commonly described as inserting onto the articular disc only or on the disc along with the condyle (Grant, 1973; MacNamara, 1973; Juniper, 1981; Bell, 1983; Bourbon, 1988). However recent dissection studies depict its attachment onto the neck of the condyle and not on the disc itself, thus contradicting numerous educational and scientific representations (Carpentier, 1988; Wilkinson, 1988). Yet, debate still exists in regard to the insertion of the superior head of the lateral pterygoid (SLP) and its relationship with the intra-articular disc. Hence, the activity of the SLP is not likely to be causing ID by pulling the disc anteriorly. It is also well recognized that it is the most difficult masticatory muscle to assess either by clinical testing, palpation or by electromyograpy (EMG). Undoubtedly, this lack of certainty about the lateral pterygoid muscle structure makes it difficult to understand how it functions and what the EMG recordings actually represent.

The existence of controversies regarding the role of the SLP in the pathological and etiologic process of the disc-condyle system opposes two leading hypotheses: an hyperactivity of the SLP pulling onto the articular disc still dominates over the newly concept of an hypo or an altered activity of the SLP. Both of them are neither understood as a cause nor as a consequence of anterior disc displacement. There is also an existing dilemma as to the cause of ID: wether passive due to a mechanical factor displacing the condyle posteriorly or active due to parafunctional hypertonicity of the lateral pterygoid muscle. Both of these hypothesis lead to disc displacement and arthralgia (Eversole & Machado, 1985). For more than five decades, studies have shown that myofascial pain-dysfunction (MPD) patients have higher EMG activity in the masticatory muscles that do non-MPD subjects (Moyers, 1950; Pancherz, 1980; Dahlstrom, 1989; Gervais *et al.*, 1989; Glaros *et al.* 1989; Jankelson, 1990).

It is suggested that the presence of hyperactivity/spasm is due to stress, grinding, trauma, malocclusion, or pain. The psychophysiological theory of TMJ dysfunction as developed by Schwatrz and Cobin in the 1950's provides one of the major theoretical frameworks for understanding its etiology: EMG hyperactivity is a response to stress and causes joint and muscles signs and symptoms (Gervais *et al.*, 1989). This school of thought, is in agreement with the discal insertion of SLP, hence suggesting that the hyperfunctional SLP pulls the disc creating the anterior disc displacement process. However, all of these studies have used surface EMG of the temporal and the masseter muscles as a basis for their theory. Isberg (1985) suggested that an altered EMG activity is a consequence of ID probably to restore and stabilize the deranged joint. On the other hand, according to Juniper (1984), hyperactivity of the SLP would cause the ID which would produce occlusal premature contacts. Posterior occlusion could be a result, not a cause of TMJ dysfunction, leading to considerable disagreement concerning the role of malocclusion in the genesis of TMJ pain and dysfunction. More specifically, Bergamini (1990) suggested hyperactivity at rest and hypoactivity or weak bite force during function but only for the masseter and the temporalis.

Conversely, it has been suggested by Carpentier (1988), a proponent of the condyle attachment of the SLP, that hypotonicity (weak condyle protraction), not hyperactivity, of the upper head may contribute to ID. Furthermore, Mahan *et al.* (1983), Gibbs and Mahan (1984) and Juniper (1987) believe that tension in SLP could not be an etiologic factor in ID because of its mechanical design. This excessive tension on the condyle's neck and disc would not displace the disc excessively forward with respect to the condyle. It was hypothesized by Mahan *et al.*(1983), Juniper (1984), Gibbs and Mahan (1984) and Carpentier (1988), who support the condyle attachment of the disc, that SLP dysfunction is the result of ID instead of it's cause.

Other investigators have reported uncoordinated and altered activity of the SLP (Isberg, 1985; Zijun, 1989). The disc dislocation would put the muscle in a "wrinkle state" and then stop it from contracting efficiently. As a consequence of an ID of the TMJ, the structure and function of the SLP could be altered and possibly maintain this ID. Isberg (1985) explained that the SLP is not involved in dislocating the disc at final closure because of its attachment to the condyle. Thus, SLP dysfunction could be a result of ID not its cause. While attempting to solve this cause/consequence controversy, Zijun (1989) found SLP altered EMG (hyper and hypo) in most patients with TMJ disorders and as a proponent of the discal insertion suggest that muscle dysfunction of the SLP is the cause of ID. From these studies, it is obvious that there is no agreement as to whether the SLP is attached onto the disc or not, nor if it can pull the disc anteriorly or not. Consequently, major disagreement persists as to whether the pathological SLP displays hyper, hypo or uncoordinated muscle activity.

In parallel, bite force studies have led to various and contradictory results. It has been shown that the masticatory muscles exhibited lower maximum biting forces in TMJ disorders cases with respect to controls as well as hyperactivity of the masseter and the temporalis (Sheikoleslam *et al*, 1982; Dahlstrom, 1989; Jankelson, 1990). Most of the studies were not done on TMJ ID specifically but more on dental interferences cases (Pancherz, 1980). Moreover, data were collected for an occlusal\teeth purpose and not a biomechanical one, therefore only various clenching forces were measured and no data on opening forces was found.

Consequently, it seems essential to measure the muscle activities of the masseter, temporalis, SLP and ILP during static and dynamic conditions as well as masticatory bite forces to have a clearer picture of the muscular mechanics of TMJ ID compared to controls in order to better understand this disorder.

Justification of the study

EMG studies in TMJ ID have been applied mainly to the masseter and anterior temporal muscles, probably because of their presumed importance, their large size and their accessibility by surface electrodes. It is well known that these muscles have a less significant impact on the displacement of the disc due to the absence of anatomic linkage to the mandibular condyle and intra-articular disc itself.

The majority of EMG studies on the masticatory system have aimed at normal function of muscles of undefined normal subjects. TMJ dysfunction subject criteria were generally subjective, based on occlusal patterns, or not described at all. An effort should be made regarding sample choices as we are dealing with specific biomechanical joint disorders.

Furthermore, previous studies have failed to consider the subject's head position, which has been proven to cause hyperactivity of the muscles of mastication. Forward head posture has seldom been rigidly controlled in EMG studies of masticatory muscles. This could explain the presence of hyperactivity in the majority of TMJ muscular studies. Therefore, the testing position of the head is of prime importance for TMJ disorders patients in EMG studies.

Rationale

The relevance of this study is to provide the clinicians and the researchers with a sound understanding of the muscle mechanics of the SLP and ILP in relation to the deranged disk-condyle functional unit. This will allow for a better comprehension of the etiologic process as well as the

possible consequences of the disorder and will enable effective management of the patients suffering from TMJ syndromes.

TMJ specialists will gain valuable information regarding the ID pathogenesis and its close relationship with the SLP and ILP muscles. It concerns dentists, orthodontists, maxillo-facial surgeons, physiotherapists as well as other therapeutic professionals dealing with TMJ disorders. Moreover, this investigation should lead professionals to consider the TMJ syndrome with a multidisciplinary collaboration to improve the quality of life of the increasing number of mistreated patients.

Lastly, this study should guide future research projects in the TMJ field and lead to improved methodological techniques.

Purpose

The purpose of this study is to investigate and analyze different static and dynamic EMG activities of the two heads of the lateral pterygoid muscles, the masseter and temporalis muscles in subjects with and without TMJ ID. Concurrently, this investigation discusses the role of the SLP and ILP and their relationships to the deranged disc-condyle unit providing us with a clinical and etiologic understanding of the muscle mechanics of the two heads of the lateral pterygoid muscles in TMJ ID.

Presentation of the study

The present thesis is divided into two papers each one with their own title and header. The first article is entitled "The Lateral Pterygoid and other Masticatory Muscles Activities in Temporomandibular Joint Internal Derangement during Static Conditions". The secind article is entitled " The Lateral Pterygoid and other Masticatory Muscles Activities in Temporomandibular Joint Internal Derangement during Dynamic Conditions".

THE LATERAL PTERYGOID AND OTHER MASTICATORY MUSCLES ACTIVITIES IN TEMPOROMANDIBULAR JOINT INTERNAL DERANGEMENT DURING STATIC CONDITIONS

ARTICLE NUMBER ONE

Running Head: *TMJ Static Analysis*

ABSTRACT

Intramuscular EMG of the lateral pterygoid muscles, surface EMG of the temporalis and masseter muscles, electrogoniometry and force measurements of the TMJ were synchronously used to investigate the biomechanical role of the two portions of the lateral pterygoid muscle in relation to internal derangement (ID) of the temporomandibular joint (TMJ). This study dealt with the EMG analysis of five static conditions: resting, resisted protraction, maximum voluntary contraction (MVC) in opening, in molar and incisor clenching of TMJ ID and control subjects. Three maximum isometric masticatory forces were also recorded during the MVC in opening, molar clenching and incisor clenching to compare forces and muscular activity between the two groups. The analysis of variance results of the integrated linear envelop (LE) EMG showed no significant differences between the two groups. Therefore, there is no apparent reason to believe that the temporalis and masseter muscles are hyperactive in TMJ ID. The integrated LE EMG of the SLP was significantly lower in the TMJ group during molar clenching ($104\mu V \pm 60.0$ over $159\mu V \pm 68.8$ for a $p = .020$). The SLP seemed to have lost its discal stabilizing function during clenching. The integrated LE EMG signals of the ILP were significantly higher in the TMJ ID group during rest, resisted protraction and incisor clenching ($p = .029$, $p = .046$, $p = .031$ respectively). The ILP muscle has probably adapted to control the inner joint instability while continuing its own actions. The ILP muscle seemed to have lost its functional specificity. The results of the isometric forces showed that TMJ ID subjects exhibited significantly lower molar bite forces (297.1N over 419N, $p = .042$) confirming that they have less muscle strength and tissue tolerance than subjects with healthy masticatory muscle system. Incisor bite forces, however, showed a tendency to be higher in the TMJ ID group (233N over 180.5N, $p = .168$), possibly resulting from the training of a protracted bite and/or hyperactivity of the ILP associated with ID. Therefore an neuromuscular adaptation could be occurring in TMJ ID masticatory system affecting muscular actions and forces.

KEY WORDS: Temporomandibular joint disorders, internal derangement, electromyography, isometric jaw forces, lateral pterygoid muscles, masticatory muscles.

The Lateral Pterygoid and other Masticatory Muscles Activities in Temporomandibular Joint Internal Derangement During Static Conditions

The prevalence of signs and symptoms of TMJ disorders is reported in 41 to 79% of the general population with the most common disorder being Internal Derangement (ID) [1-4]. Despite a considerable amount of research, there is presently no consensus as to the nature and cause of ID of the TMJ. It is generally agreed that the etiology of TMJ disorders is multifactorial [1,5,6]. However, muscular etiology of TMJ ID dominates the literature, whether primary or secondary to stress, malocclusion, posture, parafunction or trauma. Other authors in the field of TMJ, suggested a muscular disturbance in discal displacement [7-9]. A close relationship was observed between the etiology of TMJ ID and the pathological function of the lateral pterygoid muscles [7,10-11]. More specifically, the SLP has been suspected to be the cause of TMJ ID by pulling on the disc anteriorly [7,8]. Adding to the confusion, anatomical uncertainty regarding the insertion of the SLP on the intra-articular disc and/or the mandibular condyle [12-16] makes it difficult to understand how these muscles function and what the EMG recordings actually represent.

Overall, there are two leading hypotheses as to the role and muscular activity of the SLP in the pathological and etiologic processes of ID: 1) hyperactivity of the SLP causing an anterior displacement of the disc [7,8,17,18], and 2) hypoactivity or altered tonus of the SLP secondary to joint incoordination and/or laxity [9,11,14,19-22]. However, neither of these hypothesis are understood as a cause nor as a consequence of ID. Presently, the ID pathogenesis is mainly based on theoretical interpretation as opposed to experimental knowledge [3,8,10,12,15,23,24]. Clinically, these issues need to be addressed prior to adequately understanding and treating TMJ patients.

In parallel, research on masticatory bite forces have led to wide and contradictory results. Compared to control groups, subjects with TMJ disorders demonstrated that their masticatory muscles exerted weaker clenching forces, lower maximum biting strength, lower levels of EMG activity as well

as showing hyperactivity and altered contraction patterns [18,25-31]. Thus, secondary to fatigue, the masticatory system of TMJ subjects could potentially possess weak primary force muscles and hyperactive postural muscles.

The various limitations of previous masticatory EMG and bite force studies are as follow. Experimental groups, if present, were not well defined thus limiting the interpretation of the findings [18]. Most studies were also conducted on the masseter and temporalis muscles; if studies involved the SLP and ILP, only normal subjects were studied. Uncontrolled head and neck posture may have led previous authors to believe erroneously that the muscles were hyperactive [32,33]. In addition, there have been a general tendency to ignore the uncertainty regarding the insertion of the SLP. The relationship between EMG and bite force has never been explicitly explained. So far, bite forces were only measured to study teeth/occlusal issues instead of the global joint system. Thus, up to date, opening forces have never been measured or documented.

Therefore, muscular mechanics of the ILP, the SLP, the masseter and the temporalis along with jaw kinetics must be examined as they relate to TMJ ID. Consequently, the purpose of this study was to record and analyze the EMG activities and the TMJ kinetics of the whole jaw system during static tasks in subjects with and without TMJ ID disorders. The SLP and ILP muscles were investigated because of their anatomical and functional relationship with TMJ discal derangement and because they are believed to be abnormally recruited in TMJ ID. EMG of the masseter and the temporalis muscles were also recorded in order to provide a more complete profile of masticatory muscles activity, to correlate their activity with the SLP and ILP and to assess their recruitment pattern and contribution in subjects with TMJ ID compared to control subjects.

Moreover, the present investigation discussed the role of the lateral pterygoid muscles and their relationship to the ID dysfunctional unit providing a clinical and etiologic understanding of the biomechanical role of the SLP and ILP. These experiments are part of a larger study dealing with the investigation of EMG, kinematic, kinetic and sound recordings during MVC, static and dynamic conditions.

METHODOLOGY

Subjects

Twenty one female subjects participated voluntarily in the study. The ten subjects affected by TMJ ID were aged between 19 and 37 years old (mean: 27.1 years; S.D:5.7). However, two subjects were excluded from this experimental group: in one subject the EMG signal was too noisy to be analyzed while the slippage of an intramuscular electrode affected the second subject. Eleven healthy females, aged matched (criteria modified from Goldstein[34]) served as control subjects (20 to 33 years old; mean: 25.1years; S.D:4.2). Female subjects were chosen as they are the best representative group reported in epidemiological studies. The selection criteria for ID were: 1) a diagnosis of a TMJ disorder; 2) pain, tenderness or discomfort in one TMJ; and 3) arthrogenous clicking with a corresponding movement dysfunction[2,3,7,21,33,35,36]. This last criterion implied either deviation on opening deviation, limited opening, limited contralateral motion, and/or limited protrusion. Subjects with closed-lock, hypermobility or head and neck orthopaedic problems were excluded from the study.

Prior to the study, two questionnaires were filled out by all subjects to avoid medical complications (particularly those related to the needle insertions) and to record specific conditions that could account for variations in the results, and lastly, to precisely identify our experimental sample. All pertinent clinical information from these questionnaires are summarized in Table 1. In addition, subjective and objective assessments of the TMJ, including clinical characteristics and range of motion of all the subjects are shown in Table 2. The TMJ ID group were all diagnosed with unilateral TMJ ID. Other reported symptoms aside from local pain, tenderness, clicking and movement dysfunctions, were headaches, cervical pain, muscular tension and fatigue. Jaw range of motion data in the control group were within normal limits. In the case of the TMJ ID group, movement dysfunctions was present in all cases. This research proposal was reviewed and approved by the Human Research Ethics Committee of the Faculty of Health Science of the University of Ottawa.

Table 1: Clinical information from the pre-test questionnaires and the TMJ musculoskeletal assessments of the two groups.

Information	TMJ ID Group	Control Group
Subjects with at least 28 teeth	10 (100%)	11 (100%)
Subjects missing back teeth	3 (30%)	1 (9%)
Subjects with bite discomfort	5 (50%)	0 (0%)
Subjects being grinders or clenchers	4 (40%)	1 (9%)
Subjects who had had major dental work	0 (0%)	0 (0%)
Subjects who had orthodontic or occlusal treatment	6 (60%)	7 (64%)
Subjects with an history of head/neck and/or TMJ therapy	5 (50%)	0 (0%)
Subjects with osteoarthritis or other related health problems including medication intake	0 (0%)	0 (0%)

*Values indicate number of subjects. Numbers in parentheses are percentages of the group

Materials

Surface EMG signals of the temporalis and masseter muscles and intramuscular EMG signals of the SLP and ILP muscles were collected simultaneously with jaw forces. Figure 1 is a schematic

diagram of the apparatus and the data acquisition system used for these experiments. Unilateral data collection was performed as TMJ ID is usually a one-sided disorder. Intramuscular EMG was used for the two heads of the lateral pterygoid muscle. Two bipolar fine-wire electrodes threaded into a hypodermic needle (26 gauge;4.5 cm) were inserted into the SLP and ILP on the affected side. The indwelling nickel-chromium electrodes were custom-made using the method described by Giroux & Lamontagne [37]. The needles and wires were sterilized in a TIME 250 AUTOCLAVE. EMG signals of the temporalis and masseter muscles were collected by surface electrodes (pairs of silver-silver chloride, MEDI-TRACE) placed one cm apart along the muscle fibres. One surface electrode was fixed to the ipsilateral clavicle as the reference electrode. The raw EMG signals of each muscle were amplified through a differential bioamplifier (High performance AC preamplifier GRASS P511, input impedance of 20 megohms differential) with adjustable gain selected from 2000 to 10 000 and filtered with 0.03-3kHz bandpass filter and then sampled at a frequency of 1000 Hz with the BIOAD data collection system [38].

Table 2: Clinical characteristics and range of motion of the TMJ from the subjective and objective assessments of the two groups.

Information	TMJ ID Group	Control Group
Mean Age	27.1 years	25.2 years
ID side	3 left/5 right	N/A
Duration of ID	2-11 years	N/A
Opening (deviation > 2mm)	52.8mm (5 left,3 right)	53.4mm
Protraction/Retraction	4.1/3.5mm	4.0/3.9mm
Left/Right Translation	9.6/8.7mm	8.6/9.8mm

N/A: non-applicable

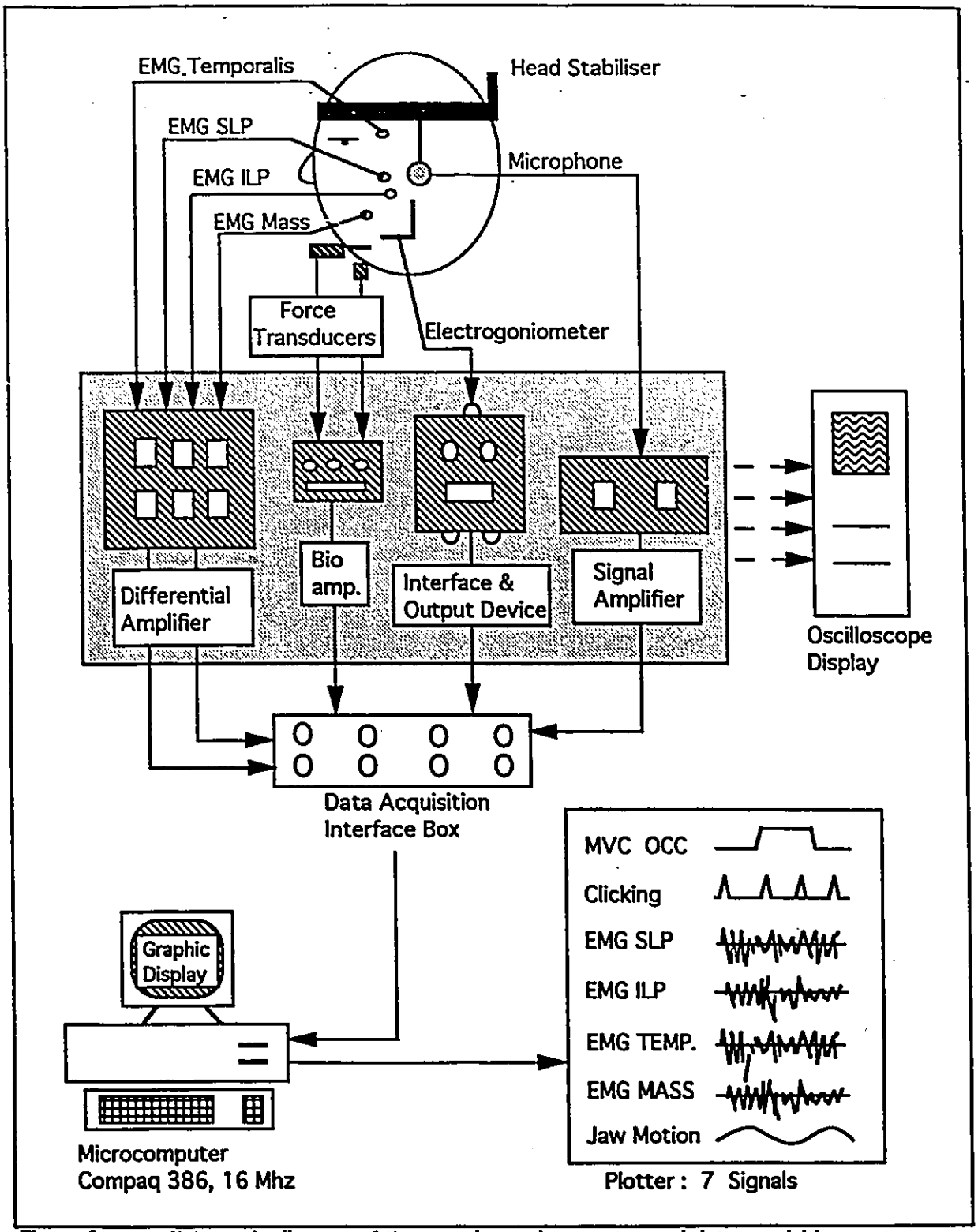

Figure 1. Schematic diagram of the experimental apparatus and data acquisition system.

Kinetic data were obtained using two custom-made, calibrated force transducers to measure maximum voluntary contractions (MVC) during isometric contractions: 1) in molar clenching; 2) in incisor clenching; and 3) in resisted opening. It is pertinent to state that the ILP is a prime opener and that the SLP, the masseter and the temporalis are prime closers, hence, we are measuring all directional forces of the four muscles. The closing bite force transducer (Figure 2) consisted of two steel beams mounted together (4.8mm inter-space) with a method similar to that described by Dechow & Carlson [30]. The full wheatstone bridge configuration of the strain gauges (high resistance 350 Ω elements) was bonded on both inner surfaces of the two steel plates to allow for direct strain measurement. The bite surface of the transducer was covered with a foamy self-adhesive material to allow teeth comfort. This material was changed for every subject. The steel plates were placed between the ispilateral upper and lower first molars and between the upper and lower incisors. The total thickness of the device was 12 mm.

The opening jaw force device (Figure 3) consisted of an eight cm wide and one cm thick steel plate with a chin shaped plastic sitter at one end and solidly fixed with two large clamps to a horizontal beam at the other end. A full bridge configuration strain gauge system was bonded to the top and bottom surfaces of the beam near the clamps. The cephalad stabilization of the head permitted the subject to produce pure downward force and movement with the chin. The strain gauge system was calibrated and the voltage signal amplified and fed to an A/D conversion interface controlled by the BIOAD data acquisition program. The calibration of the two devices were done by loading three trials of six upgrading and downgrading known weights on the steel plate and registering the voltage through an amplifier. A linear relationship between the load and the voltage was obtained and a regression equation for each device was computed. The bite force transducer was linear ($r=0.990081$) and the regression equation was $y=56.664x - 0.687$. Whereas the opening jaw force device showed also linear response ($r=0.999$) with a regression equation described as $y=627.714x + 0$. transducers respectively.

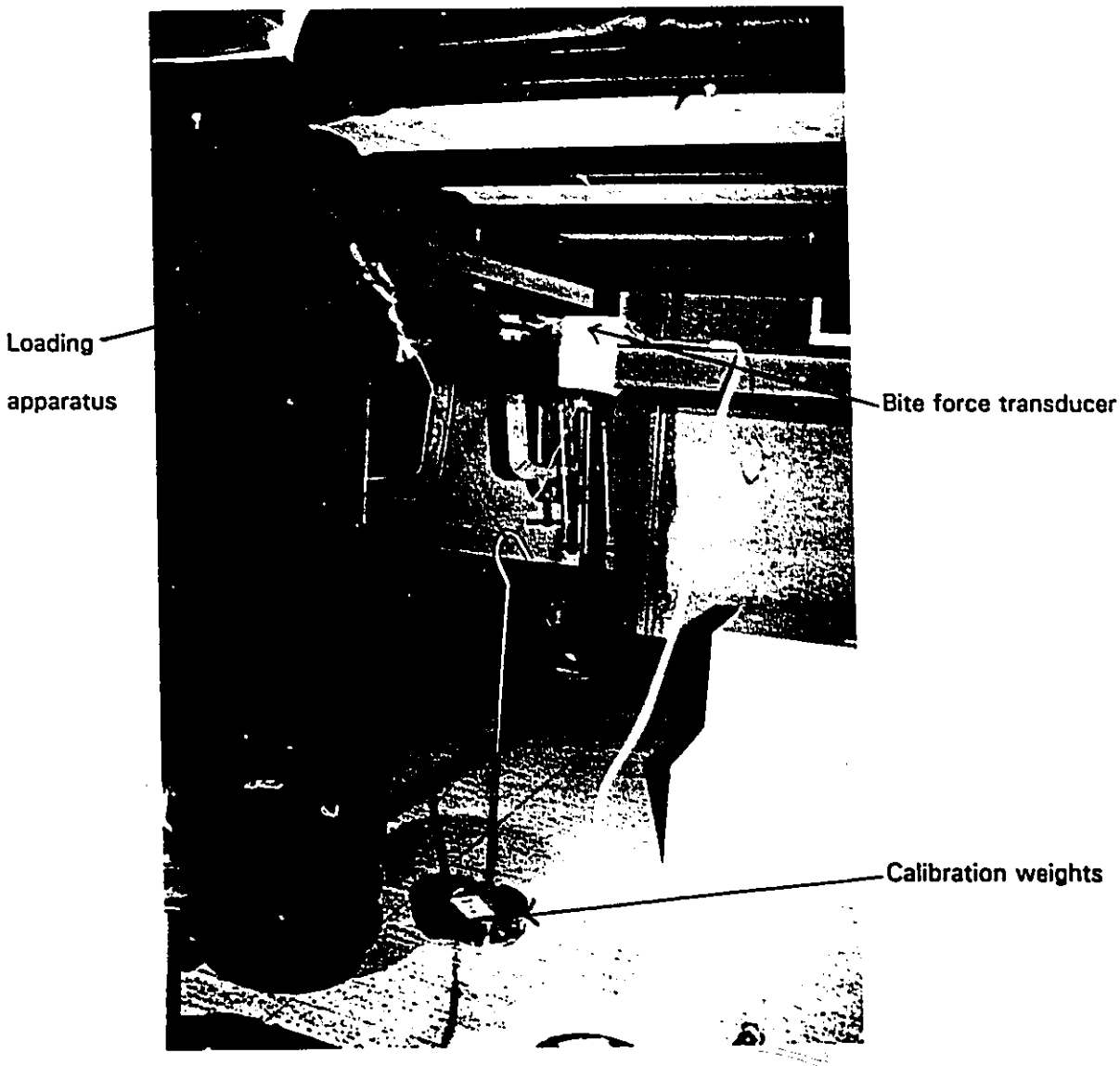

Figure 2. Clenching bite force transducer during the calibration procedures.

During testing session, the subjects were seated in confined, yet straight custom-made testing chair. The chair's ergonomic characteristics along with the head stabilizer and visual feedback minimized the hyperactivity effect of posture and therefore controlled head motion during opening performance.

Procedures

EMG Electrodes and Insertion Application

Prior to testing, all subjects were asked to read an information letter and sign a consent form. One hour before the insertion, the subjects applied an eutectic mixture of local anaesthetics (EMLA, Astra Pharma Inc., Toronto Canada) on the insertion site as instructed. According to the subjects, EMLA cream was efficient in relieving pain due to wire insertion. The relief depended on analgesic time and the area of application. The selection of an insertion technique for both portions of the lateral pterygoid muscle was made following an extensive theoretical review [9,20,39-41] and was achieved extra-orally through the sigmoid notch. This technique was preferred because it is relatively simple and does not lead to post-insertion sequelae. A physiatrist specialized in indwelling EMG performed the insertions of the fine wire electrodes in the ILP and SLP while the subjects laid in a comfortable supine position. The mandibular or sigmoid notch provided the simplest access for insertion in respect to placement, direction and soft tissue mass. Some of the subjects experienced discomfort on maximum opening and maximum closing along with a resistance sensation on maximum opening. Therefore, after the first five subjects and careful studying of an anatomical atlas of the TMJ[42], the approach was modified by adding intraoral palpation for guidance. The second approach consisted of proceeding higher and more posteriorly, close to the condyle and inferior to the zygomatic arch, immediately above the coronoid process, this without being exactly into the notch. After touching the sphenoid bone, the needle was drawn back and the wires carefully pulled so they would hook well before the needle was withdrawn. On a post-testing questionnaire, subjects reported discomfort (an average of 2/10 on the visual

analogue scale) including pulling, tightness and apprehension during the insertion, during the testing period and the needle removal.

Before data collection, proper insertion of the electrodes into the ILP was verified by observing on an oscilloscope, strong activity during opening and protrusion and no activity during clenching[39-43]. Verification of wire placement in the SLP was obtained by monitoring strong activity during clenching and an absence of activity during protrusion and opening [39,43]. The verification of electrode placement was more difficult in the TMJ ID subjects since their EMG signals were expected to be abnormal. Therefore, technical experience, clear EMG signals, and elimination of other muscles activity were used to verify electrode placement. None of the wires were left in the masseter because the thick fascia and the deep portion of the masseter were obvious to sense; thereafter, a clear contact on the greater wing of the sphenoid bone confirmed the correct anatomical space. However, it was impossible to be over the SLP because there is no space between the zygomatic arch and the SLP, contrary to most anatomical books representation. To reach the SLP, we inserted as close to the caudal border of the arch and good EMG signals confirmed the appropriate position of the wires electrodes. Some signals were slightly noisy, especially the SLP ones, and we were able to remove the interference by pulling and the wire. Once the wires were inserted in the ILP and SLP muscles, they were isolated and fixed with adhesive tape. The surface electrodes were fixed on the temporalis and masseter muscles and the EMG signals tested and calibrated.

Conditions

Subjects were then stabilized in the testing chair and the EMG amplifier outputs of the four muscles along with the two force transducers were connected to the data acquisition interface which was controlled by a micro computer (Compaq 386, 16MHz). All signals were collected simultaneously by BIOAD, a data acquisition system to allow synchronisation of the different recordings.

The subject performed five static tasks, chosen because they either elicit an activity of the lateral pterygoid muscle, or are known to cause ID.

1- A maximum voluntary contraction (MVC) of jaw opening consisted of opening the mouth forcefully. A steel bar was used as a resistive device and force transducer while cranium cephalad motion was immobilized. This tasks measured the opening force by bending the steel bar instrumented with strain gages. This also assessed, the ILP recruitment during opening not forgetting the contribution of the accessory jaw openers (hyoid and digastric muscles). Figure 3 shows the experimental set-up with a subject ready to perform the MVC in opening. Notice the head stabilisation mechanism used to isolate mandibular depression and eliminate maxillary or head elevation.

2 and 3- Two MVC of jaw clenching in the molar and incisor position were executed by biting onto the closing bite force transducer that was covered with cushioning tape. These resisted clenching task assessed the basic bite forces, the SLP participation along with joint loading and a proposed causal factor: incisal biting [7,16,21].

4- Resting activity of the SLP, the ILP, the masseter and the temporalis muscles were recorded in order to determine general resting hyperactivity or postural tonus. The resting EMG activity was subtracted from other tasks to obtain the net EMG activity.

5- Active resistive protrusion was isolated to monitor the translation arthrokinematic component of the TMJ and to measure the function of the ILP muscle. The subjects were asked to maximally protrude their jaw against the examiner's fist while the head was stabilized.

Standardized verbal instructions about the tasks and procedures were given to each subject, as were cues to correct movements when needed. All movements were demonstrated by the investigators, then practiced by the subject to a satisfactory level. Each static jaw condition was held for three seconds and repeated three times. Unilateral collection was performed as all the subjects involved in the study were diagnosed with a one sided disorder.

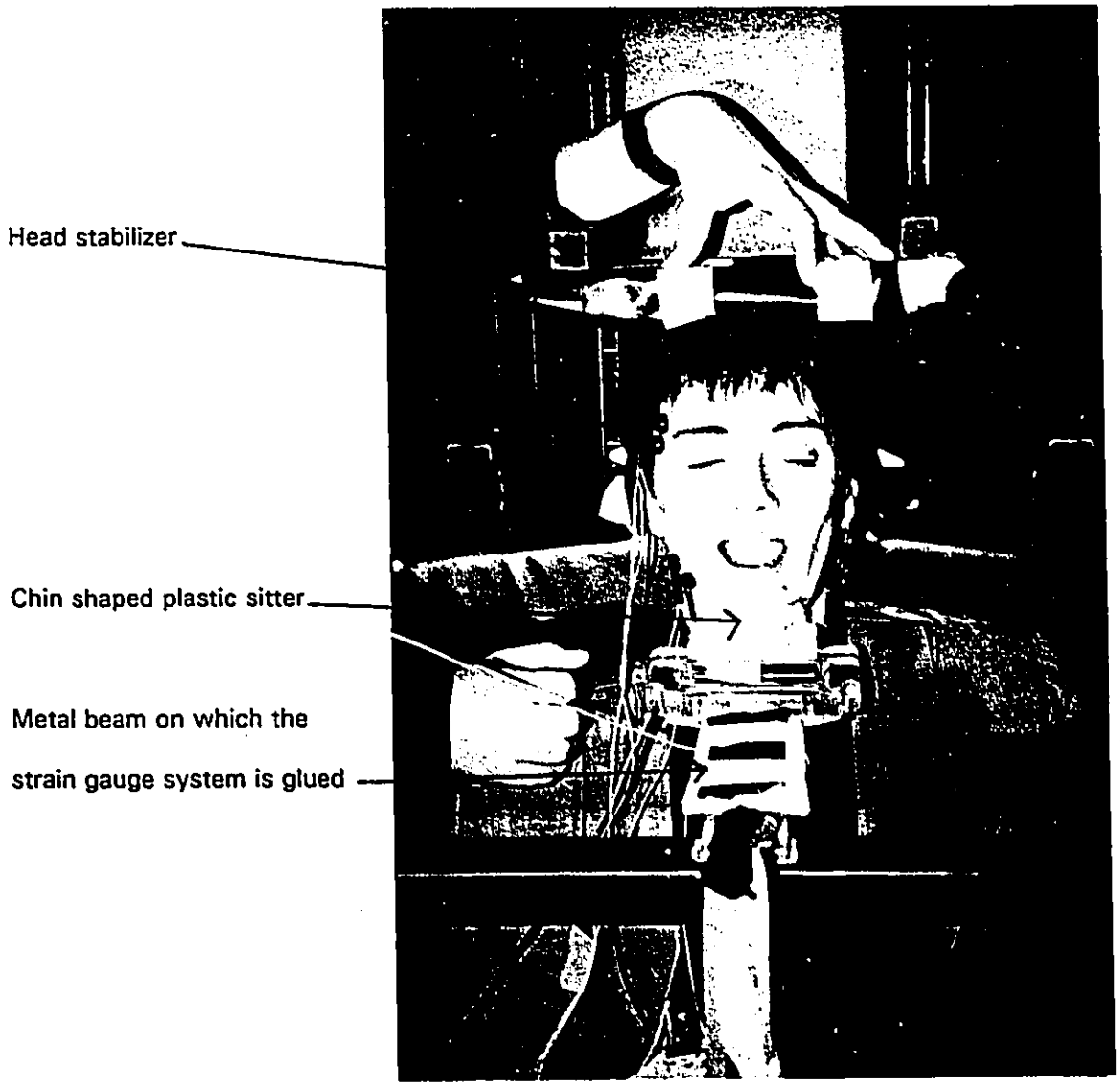

Figure 3. Experimental set-up: The subject is ready to perform a MVC in opening using the opening device.

Data Reduction and Analysis

Two sets of measurements were computed from the five static conditions: the integrated linear envelope (LE) EMG of the four muscles and the isometric force values from the three MVC tasks. To obtain the integrated LE EMG, three trials of raw EMG signals of each muscle for the five conditions were processed using the BIOPROC processing program [38]. The raw EMG signal was high-pass filtered at a cutoff frequency of 10 Hz; the bias was removed by using the mean; and the individual amplification gain obtained from the amplifier (P511 Grass amplifier) was scaled by a factor to convert all signals in μV. The signals were then full wave rectified and filtered with a fourth order, dual passes and critically dampened with a cutoff frequency of 6Hz to obtain the linear envelope (LE). The most constant one second window of the LE EMG was selected and integrated. For the three MVC's tasks, the one second window used for processing was the one with the most stable and stronger maximum force. The average resting integrated LE EMG signal was removed from the mean integrated LE EMG signals to obtain the EMG signal provided by the specific movement. The mean integrated LE EMG of the three trials for each condition was computed for each subject and used in further analysis. Averages were first computed across trials for each subject per condition per muscle. Grand ensemble averages were then computed across subjects for the two groups for the four muscles at each condition in order to compare the results between groups.

The isometric force signals were processed from the three MVC conditions. The bias was removed to level the signal at zero. The mean force signal of the selected one second window was measured for each trial per subject for the three MVC: in opening, molar and incisor clenching. For each three MVC condition, the mean force value (in voltage) of the three trials was inserted in the regression equation obtained from the calibration curves and converted to Newton. Ensemble averages were computed across subjects per group to compare the forces exerted by the TMJ ID and the control groups for each MVC task.

Statistical Design

The two groups were characterized as random samples of two specific populations: TMJ disorders and matching (sex and age) surrounding volunteers of the general population. Statistical analyses were conducted on the dependent variable mean integrated LE EMG expressed in μV for each four muscles for the five static conditions. General linear model ANOVA (four 2x5 with post-hoc Fishers tests) were contrasted with twenty simple one way ANOVA. Statistical analyses were also conducted on the three isometric forces expressed in Newton using one 2x3 ANOVA model and one-way ANOVA. All tests were done through NCSS software package using a significant level of $\alpha \leq 0.05$ to determine if the two groups differ on their muscle activity for the five conditions and on their opening and closing bite forces. The independent variables were TMJ ID and control groups. Two tailed tests were used as the TMJ ID group might present with hyperactivity or hypoactivity, lower or higher forces, compared to the control group. The simple or one way ANOVA comparing the two groups means for each muscle in each condition separately seem to be the best way to draw the information we were looking for without fading the contrasts and loosing the meaning. It was well understood that some conditions and some muscles do interact but sometimes in opposite directions, therefore cancelling the effect of interest. The factors are considered independent and should not be melted together. The only effect of interest being studied was the muscular activities and forces generated by the two groups.

Independent scores and normality of distribution were tested positive. Although, we had large standard deviations, probably due to the large inter-subject variability[44] and the influence of many factors, the homogeneity of variance between samples was valid most of the time (tested within the simple ANOVA procedures with NCSS). If not, a corrected and more-conservative, p value was provided by NCSS in cases of unequal variance and was used to assess significance.

RESULTS AND DISCUSSION

Statistical and Descriptive Analysis of the integrated LE EMG

The statistical results of integrated LE EMG for all muscles in each of the five static tasks are summarized in Table 3. Each muscle will be discussed separately.

Table 3. One-way ANOVA results between the two groups for the integrated LE EMG of the four muscles for the five static conditions.

TMJ ID vs CONTROLS	Rest	MVC in Opening	Resisted Protrac-tion	MVC in Molar Clench	MVC in Incisor Clench
Masseter	=	=	=	=	=
Temporalis	=	=	=	=	=
ILP	↑↑	↓	↑↑	↓	↑↑
SLP	=	=	=	↓↓	↓

=: no significant difference;

↑↑ or ↓↓: significant difference;

↑ or ↓ : tendency.

Masseter and Temporalis Muscles

For the masseter muscle integrated LE EMG, there was no significant difference between the two groups for all the static tasks (Figure 4 and Table 3). As for the anterior temporalis muscle, there was also no significant difference between the two groups for the five static tasks (Figure 5 and Table 3).

Overall, muscular disturbance of the temporalis and the masseter does not seem to be involved in TMJ ID, neither as a cause, nor as a consequence. On the contrary, other EMG investigations of

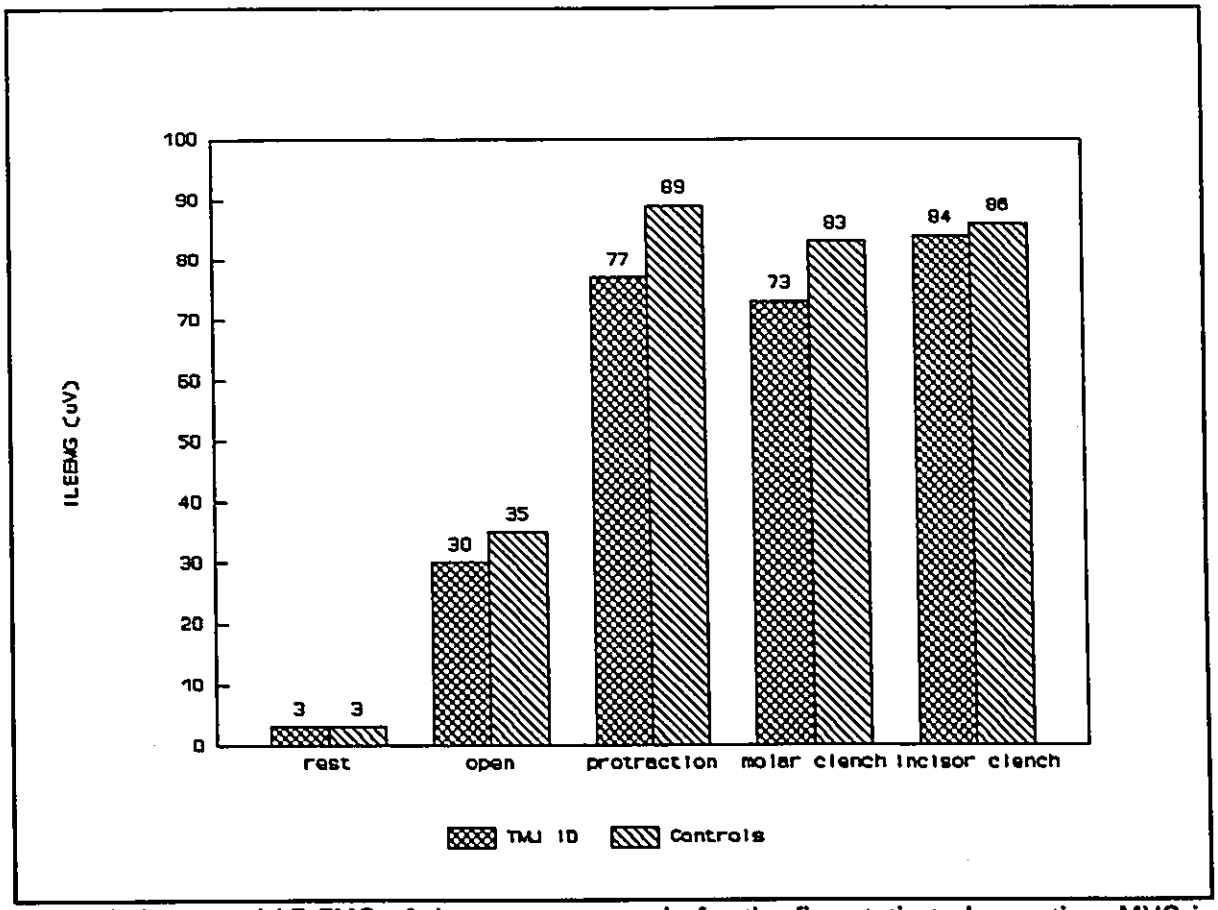

Figure 4. Integrated LE EMG of the masseter muscle for the five static tasks: resting, MVC in opening, resisted protraction, MVC in molar and incisor clench of the TMJ ID and the control groups.

craniomandibular disorders, regardless of the population, have reported significant hyperactivity of the masseter and temporalis in TMJ disorders compared to control subjects[18,21,25,46,47]. This may be explained by the fact that our TMJ ID sample involves specifically ID of the TMJ, which implies an arthrogenous as opposed to a myogenous problem. Previous EMG studies have described their populations as TMJ disorders or malocclusion problems; these can include different muscular dysfunctions such as muscle tension, occlusal interferences, parafunction, muscular pain and myofascial syndrome all which may be associated with hyperactivity. It is unlikely that any centrally mediated masticatory muscle hyperactivity as previously proposed[48] occurs in TMJ ID, but it might be possible in myofascial or myogenous TMJ disorders.

The masseter and the temporalis muscles yielded similar results with the exception that the

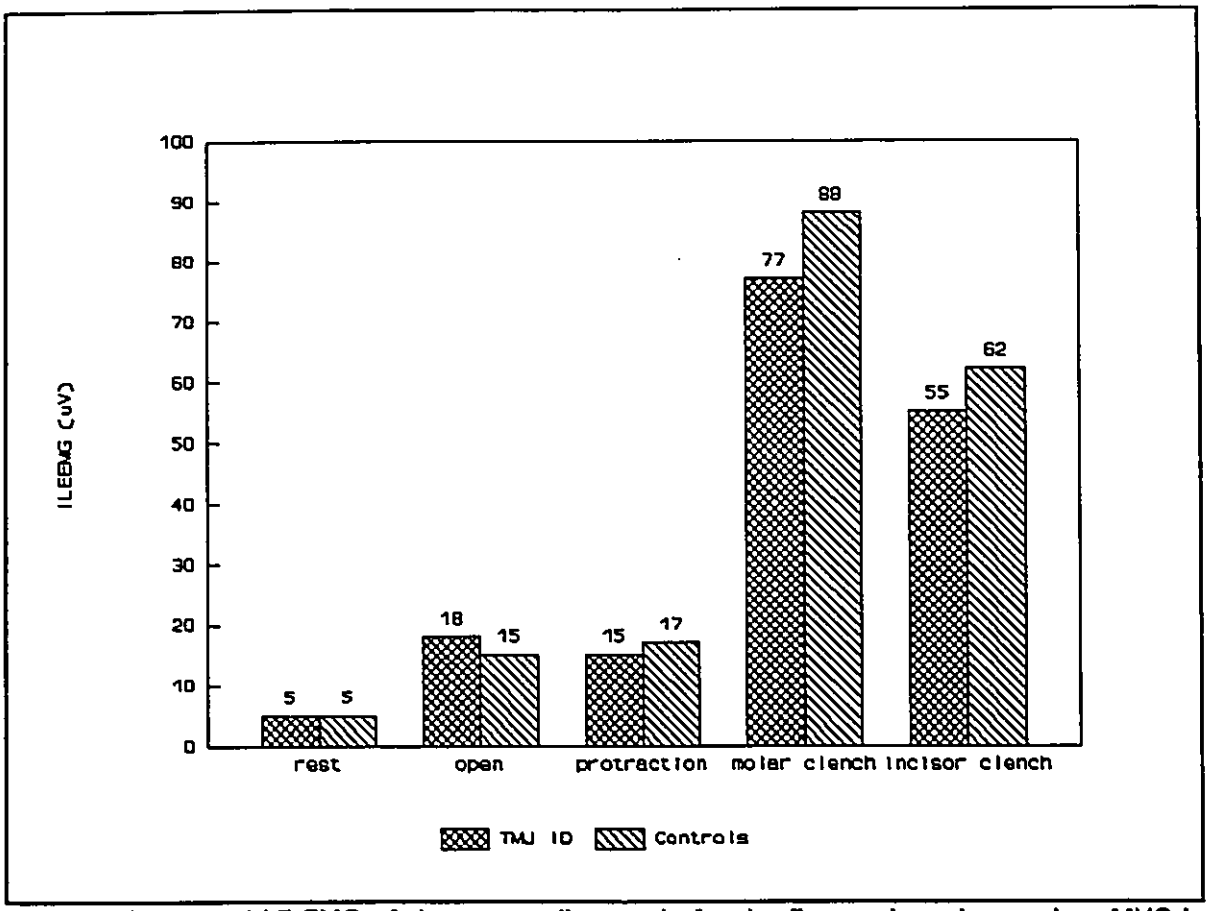

Figure 5. Integrated LE EMG of the temporalis muscle for the five static tasks: resting, MVC in opening, resisted protraction, MVC in molar and incisor clench of the TMJ ID and the control groups.

superficial masseter was functionally more active in opening and protraction for the two groups. These results were understandable and expected, as the superficial masseter is considered a synergist of those two movements based on muscle mechanics and fibre direction [40].

Inferior Head of the lateral pterygoid muscle (ILP)

Despite reports linking the lateral pterygoid muscles to TMJ disorders, the activities of these muscles have not concretely been studied. However, the concept of increased and altered muscular activity in TMJ disorders dominates the literature of the masticatory system [7,11,18,19,21]. Our findings of the ILP muscular activity for the TMJ ID and control groups for the five static tasks are shown in Table 3 and Figure 6.

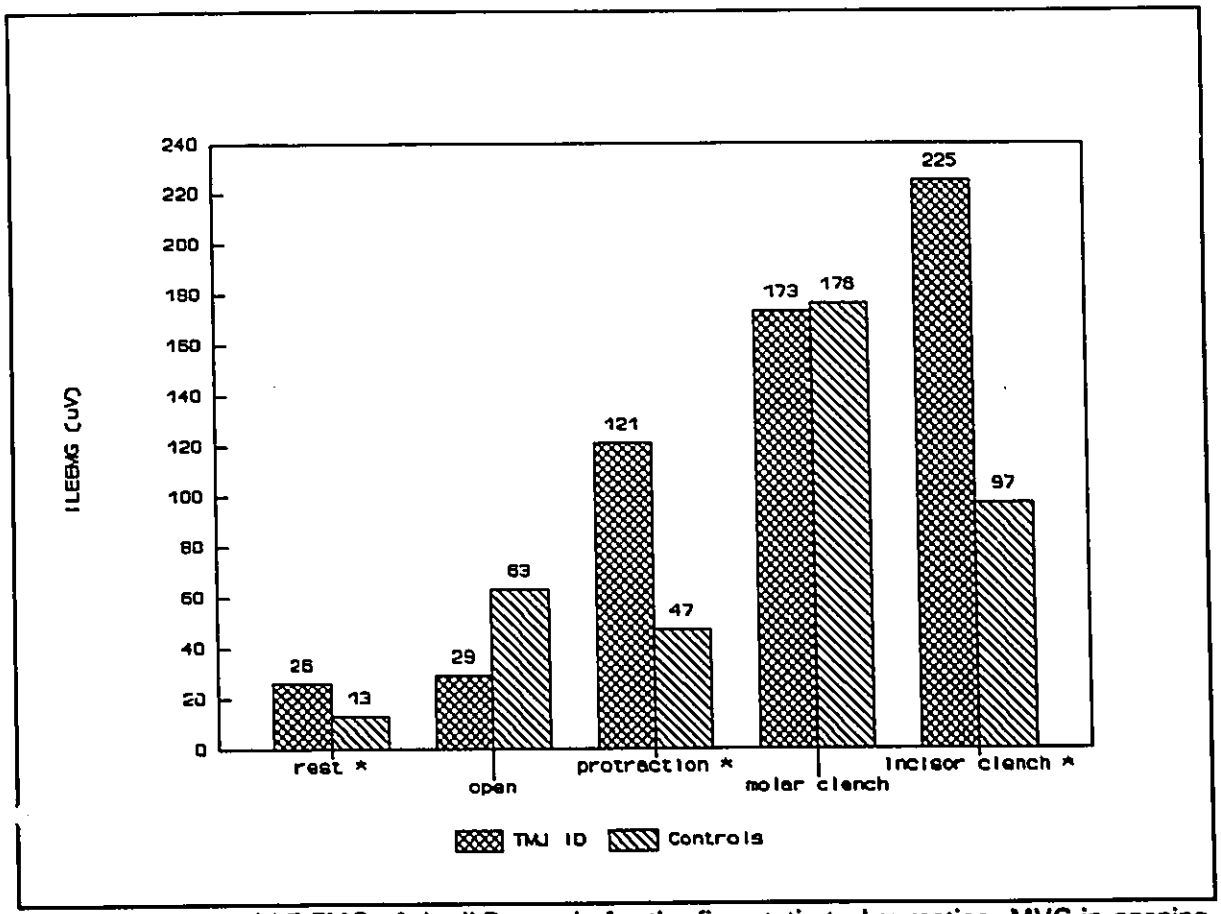

Figure 6. Integrated LE EMG of the ILP muscle for the five static tasks: resting, MVC in opening, resisted protraction, MVC in molar and incisor clench of the TMJ ID and the control groups.
* : significant difference.

In the resting condition, mean integrated LE EMG of the ILP is significantly higher ($p = .029$) in the TMJ ID group ($26\mu V \pm 11.8$) compared to the control group ($13\mu V \pm = 8.8$) (Figure 6). Although one particular TMJ ID subject had a very strong hyperactive ILP, excluding this value from the mean still resulted in a significant difference between the two groups. Our results supports numerous studies reporting resting or postural muscular hyperactivity in patients suffering from TMJ disorders but in other masticatory muscles [18,21,31,47,48]. As head position was controlled to avoid centrally and postural induced hypertonicity, it is considered as a true hyperactivity. Unfortunately, it is not possible to clarify if this hyperactivity of the ILP at rest is a cause, a consequence or maintains TMJ ID, Consequently, we can only suggest that a relationship exists.

During the MVC in opening, there was no significant difference between the two groups ($p = .296$, TMJ ID:29μV $\pm$34.5 and control:63μV $\pm$86.2) (Figure 6), keeping in mind that one function of the ILP is to open the mandible in synergy with the supra-hyoid muscles.

During resisted protraction, a primary action of the ILP, the integrated LE EMG activity is significantly higher ($p = .046$) in the TMJ ID group (121μV $\pm = 83.3$) compared to the control group (47μV $\pm$33.1) (Figure 6). In addition, the integrated LE EMG for the control group in protraction is significantly higher than its own resting EMG activity. This simple ANOVA demonstrated that the ILP EMG activity of the control subjects are contracting normally in protraction as expected. However, the ILP activity in of the TMJ ID subjects are hyper-active. This exaggerated anterior pulling on the condyle by the ILP in the TMJ ID group could be interpreted as a muscular/joint system trying to get the condyle in place under its anteriorly displaced disc or simply compensating for inner-joint instability. Interestingly, protractive biting has been previously interpreted to be responsible for ID [7,16,21] adding to the confusion over the cause and effect issue.

In the two clenching tasks, MVC in incisor and molar clenching, non-negligible myoelectrical activity was recorded in the two groups even though the ILP has been reported to be silent in jaw closing motions[8,20]. Similarly, Wood et al.[50] have reported that the ILP contracts up to 75% of his maximum in anterior clenching. It is suggested that during maximal clenching conditions where muscular strength demand is higher, the co-contraction activity of the ILP is required. In addition, the ILP has been reported to be electrically active during the first half of closure during ID [15]. This eccentric contraction would control the return of the condyle and the ILP would stop contracting while the SLP would terminate the closing motion.

During the molar clenching task, there was no significant difference ($p = .959$) between the two groups (TMJ ID : 173μV $\pm$ 61.1 and control: 176μV $\pm$ 94.1). During the incisor clenching, the integrated LE EMG of the ILP were significantly higher ($p = .031$) in the TMJ ID compared to the control group (225μV $\pm$129.5, and 97μV $\pm$16.5 respectively) (Figure 6). The ILP muscle seems to be a stronger synergist of the jaw elevators in TMJ ID subjects incisor clenching. This supports results that found

ILP activity in incisor clenching in control subjects [23-50]. It is suggested that the protracted position of the mandible is at cause. It was proposed that this action added to the elevators produces a clenching system as well as condyle stabilization [50]. Determining if this new muscular action is eccentric or concentric cannot be answered here, but an eccentric contraction of the ILP during clenching to control the posterior and superior return of the condyle is suspected, as partly reported previously [9,15,20]. A miss-insertion in the SLP or in the deep masseter is highly unlikely as EMG activity was registered in opening and protraction. In addition, the ILP still contracts in its usual functions: opening and resisted protraction. Therefore it functioned in a dual role and was always active wether it is primarily an agonist or an antagonist of the movement. Similar observations were reported by Zijun[11] and Wood et al.[50] . This phenomenon was recorded in eight out of ten TMJ ID subjects.

Therefore the normal function of the ILP in normal subjects needs to be refined and its role in TMJ disorders needs to be understood and defined. In our normal subjects, the ILP muscle was contracting during clenching and during opening as well as in tasks with maximum clenching demands [50]. In the TMJ ID subjects where joint stability and balance are uncontrolled, the ILP appeared to function continuously to control condyle coaptation and to assist its stabilization while its regular duties are amplified. Therefore, the ILP contributes actively to its secondary and opposite functions and is still active in opening and protraction as an agonist. This compensation from the ILP for the SLP is quite possible since both muscles have very similar direction and insertions. The hyperactivity recorded in the ILP muscle in the TMJ ID subjects might be due to one-sided chewing on the better dental side as suggested previously [51] but is impossible to determine at this point. However, evidence of centrally initiated hyperactivity has been shown to lead to muscle dysfunction and TMJ disorders [52].

The reliability of the fine wire recording techniques may be questionned at this point as the wires tend to move in the muscle during function and the cross talk effect of the deep anterior temporalis and medial pterygoid [50] may exist. Explanations used by other authors [50] to explain the variability of their EMG signals may be used here: anatomical and biomechanical constraints of the masticatory system demands variations in muscle contraction to satisfy the goal of the individual; and

it may depend on the position of the occlusal contact points.

Superior Head of the lateral pterygoid muscle (SLP)

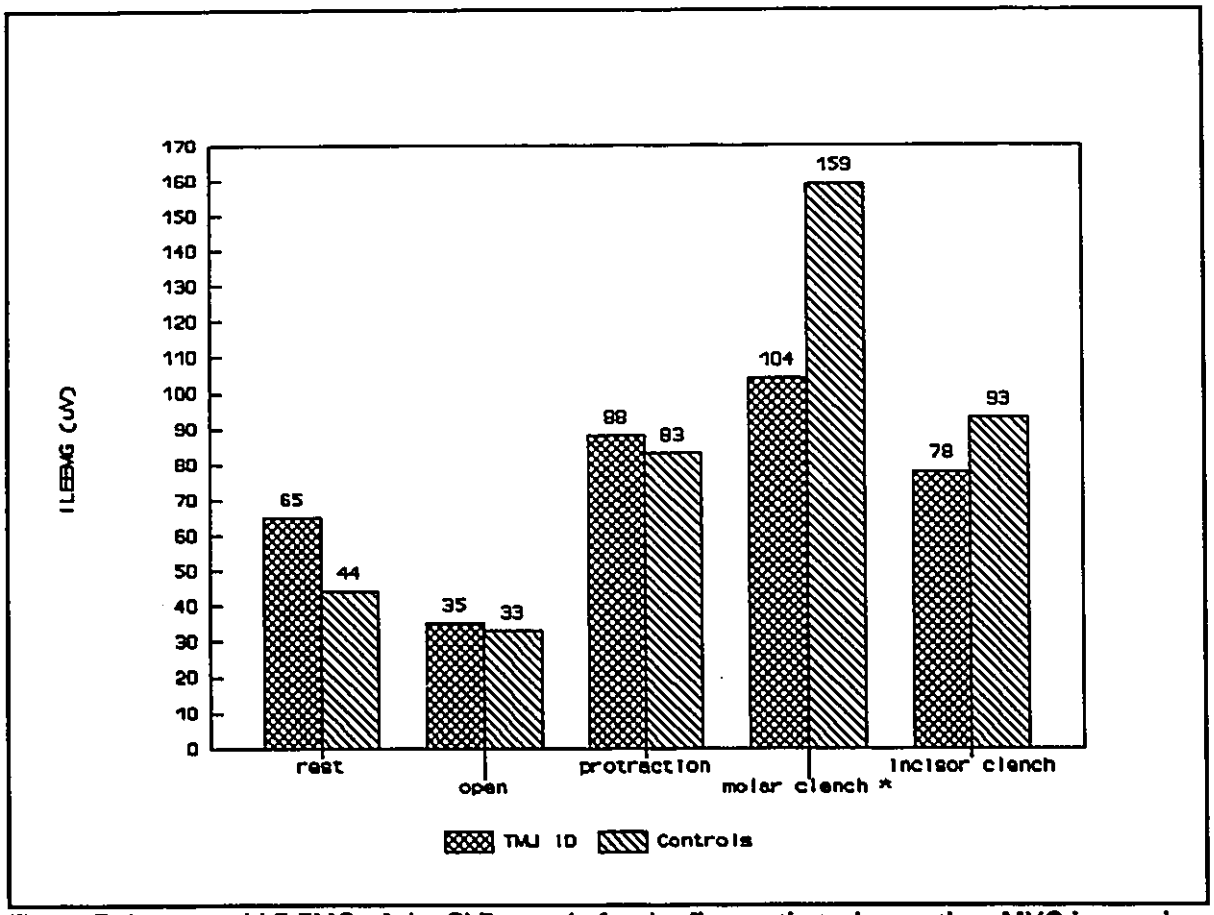

Figure 7. Integrated LE EMG of the SLP muscle for the five static tasks: resting, MVC in opening, resisted protraction, MVC in molar and incisor clench of the TMJ ID and the control groups.
*** : significative difference.**

During the resting condition, there was no significant ($p=.523$) difference between the two groups SLP integrated LE EMG (TMJ ID: 65μV $\pm$81.4 and control: 44μV $\pm$44.8) (Figure 7 and Table 3). Therefore, the SLP had a similar resting tonus whether the disc/condyle relationship is normal or not, in contrast to the ILP, which is hyperactive at rest in TMJ ID subjects. Our results also showed a basic resting tonus for both groups which is consistent with the histopathology and physiology of the lateral pterygoid muscle made of 70% slow twitch muscle fibres[23,53], a postural and fatigue

resistant muscle. The signals show more activity in the resting condition compare to the opening, probably because during opening, there is a reflex inhibition of the antagonist muscles.

During the MVC in opening, there was no significant ($p=.926$) difference in the EMG results of the SLP between the two groups (control: $33\mu V \pm 39.9$ and TMJ ID: $35\mu V \pm 43.0$) (Table 3). Moreover, there was no significant difference in their respective resting tonus (Figure 7). These results were expected, as opening is not a primary function of the SLP which allows the disc to rotate back during the rotation phase.

During protraction, our results demonstrate mild EMG activity of the SLP in the two groups (Figure 7). Normally, the SLP should be inactive during opening and protraction as both movement involve an anterior motion of the disc/condyle unit which is usually achieved by the ILP alone[9,20,44,46]. The experimental group did not differ significantly ($p=.895$) from the control one ($88\mu V \pm 82.5$ and $83\mu V \pm 54.1$ respectively). It is suggested that healthy and pathological SLP muscles are contracting mildly during protraction, probably to control and stabilize the disc/condyle relationship as it is its main function. In parallel, continuous stabilizing activity of the SLP has been previously reported[23].

During both clenching MVC conditions, the SLP muscle demonstrated lower EMG activity in the TMJ ID group: A significant difference ($p=.020$) for the molar clench (TMJ ID:$104\mu V \pm 60.0$; and control group $159\mu V \pm 68.8$); and a tendency for the incisor clenching condition (TMJ ID:$78\mu V \pm 74.7$; and control group $93\mu V \pm 68.9$) (Figure 7). In healthy clenching masticatory systems, the SLP acts as a horizontal force stabilizer of the mandibular condyle/disc complex to prevent posterior dislocation[64], consequently our results suggest a loss of this discal and condylar control by the SLP muscle. The most chronic TMJ ID subjects exhibited silent EMG periods in the middle of a the clenching contractions. This may indicate that the SLP is loosing its ability to control the disc in TMJ ID disorders.

At rest, the lateral pterygoid muscle is almost fully extended and reference to the traditional length/tension curve would indicate that this is its most powerful position, supporting an eccentric type of contraction. Therefore, we believe that the normal function of the SLP is to counteract and control (eccentric contraction) the elastic pull of the posterior discal tissues on mouth closure and to stabilize

during clenching [23]. Our results demonstrate a tendency of the SLP to lose this control function. [47,48]

It appears anatomically impossible for the SLP to pull the disc without affecting the condyle. Accordingly, we could be dealing with a passive non-muscular mechanical origin for TMJ ID. Moreover, after a prolonged ID, the structure and function of the SLP would be altered, a wrinkled inefficient muscle could maintain or progress the development of the discal displacement[21]. This would support our results. If our subjects are true cases of discal ID, it is suggested that the muscle fibres of the SLP would probably be somewhat wrinkled and gradually change into non-contractile, atrophied or fibrous tissues with corresponding altered EMG signals and less efficient pulling and eccentric control of the disc as previously proposed [14,21,49]. The condyle could be positioned posteriorly first and then push the disc forward affecting the contractions of the lateral pterygoid muscles. Alternatively, other causal factors could lead to muscular imbalances which in turn could produce anterior disc displacement by lack of control of the disc/condyle system. Another possible explanation is that the SLP would be overused with incisal biting which in turn would stretch the retrodiscal tissues by posterior condylar compression resulting in a gradual forward displacement of the disc.

Statistical and Descriptive Analysis of the Isometric Forces

The mean isometric force values for the three MVC conditions (opening, molar clench and incisor clench) obtained in this study are displayed in Newton in Figure 8. The values for the TMJ ID and control group respectively are 59.0N±16.1 and 68.9N±20.1 for the MVC in opening; 297.1N±115 and 419.1N±47.2 for the MVC in molar clench; and 233.0N±82.7 and 180.5N±60.4 for the MVC in incisor clenching. Large standard deviations within a group may suggest the influence of many factors, in particular the inability of the subjects to perform a true maximum biting. The 2x3 ANOVA was significant at $p = 0.0493$ but the one-way ANOVA was preferred as there was only one independent variable (TMJ pathology) of interest; it seemed non-logical to blend opposite forces and tendencies.

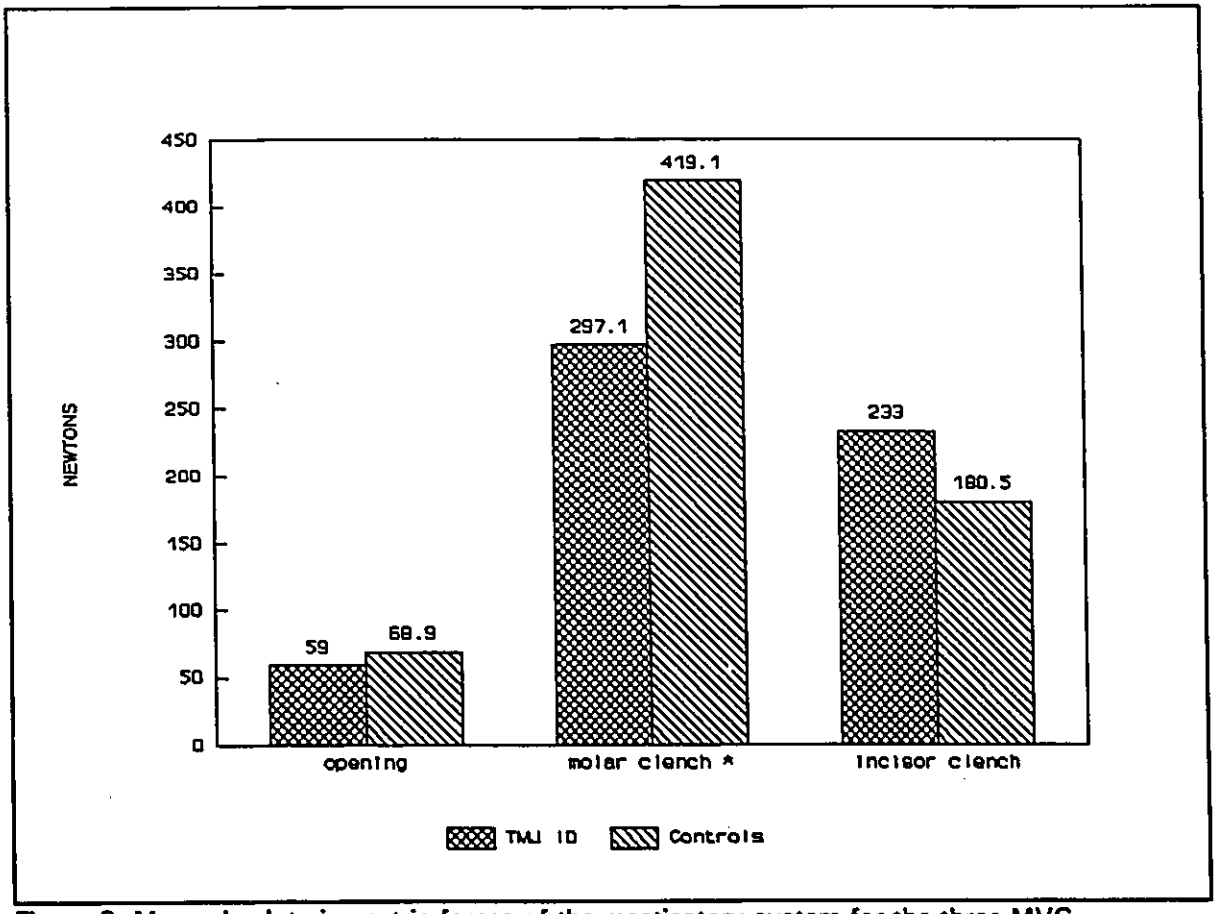

Figure 8. Mean absolute isometric forces of the masticatory system for the three MVC conditions: molar and incisor clenching and opening of the TMJ ID and the control groups. *: significative difference

All our recorded forces are higher than those reported by Helkimo and Carlsson [29] and Throckmorton and Dean [52] in healthy females, for molar clenching (216N and 372N) and incisor bite forces (108.0N and 119.4N). Our corresponding values are 419.1N and 180.5N (Figure 8). Again, in healthy females, Linderholm et al. [59] reported isometric molar and incisor bite forces of 421.0N ± 91.1 and 196.0N ± 60.8 respectively. These values are very close to ours and within our standard deviation ranges. But, Ringqvist et al. [60] observed much greater force values at the incisor and molar levels in 29 healthy females aged 19 to 23, a sample very similar to the one we studied (incisor: 294.0N ± 58.8 and molar: 764.4N ± 92.1 respectively). Disparities in the equipment and procedures, diet and masticatory habits, as well as masticatory training may account for the wide range of maximum bite

forces reported in the literature [27,59,60].

Molar Clenching Isometric Force

The molar clenching forces were statistically different ($p = .042$, one-way ANOVA) between groups with the TMJ ID subjects generating lower forces than control subjects (297.1N and 419.1N respectively) (Figure 8). It has been reported that molar clenching forces is a bias picture of the strength of the masticatory muscles because it is limited by the inflammation state and tolerance of the supporting tissues as well as pain [27,61]. Consequently, TMJ ID subjects would be expected to generate less maximum clenching [61] (39 to 80% in 71% of the subjects [25]) force because of pain and inflammation of the supporting tissues, muscular inhibition, fear, which allows lower compressive forces. In addition, the masticatory musculature of the TMJ ID subjects is probably weaker since the subjects do not use heavy jaw pressures and they demonstrate impaired neuromuscular control and pain inhibition [18,25,31]. Moreover, the linear relationship between the integrated EMG activity and the force exerted in isometric conditions has been reported [62]. Consequently, we can suggest that the masticatory system of the TMJ ID group is weaker, less tolerant and more sensitive compared to control subjects. However, it is impossible to state the extent of each of these factors which might influence our results. For example, there may be no true strength difference, but rather a tissue tolerance problem but the TMJ ID subjects are still using less force than healthy ones. Variation of maximum bite force can also be explained by anatomic variation of the jaws [60], functional factors and state of dentition [27]. This can certainly apply in the present study since the TMJ ID group have greater chances of possessing anatomic variations in the stomatognathic system, a poorer state of dentition, as well as altered masticatory function. These factors have all been reported to be associated with TMJ ID. Moreover, a protective inhibitory reflex mechanism may also be contributing in subjects with TMJ ID.

Incisor Clenching Isometric Forces

It has been suggested that normal incisor biting is controlled by sensory receptors within the periodontal ligaments, within the TMJ, and within the muscles of mastication [61]. It has also been hypothesized, that pathological jaw joints with altered muscles and/or soft tissues secondary to overuse, underuse, inflammation, such as those present in TMJ ID subjects would lead to lower bite forces compared to those generated by control subjects [63]. Therefore, differences in forces might not reflect a loss of pure strength, but also more sensitive and altered proprioception as seen in molar clenching. As shown in our results, these explanations may not be fully applicable for the incisor clenching results (Figure 8). Mean isometric force for the TMJ ID group (233.0N ± 82.7) and the control group (180.5N ± 60.4) compared for a non-significant difference ($p = .168$) at $\alpha \leq 0.05$.

Juniper[23] stated that incisor biting is a function of the ILP muscle which is supported by our LE EMG data where the ILP was hyperactive in TMJ ID subjects. This hyperactivity would result in higher bite forces. Also, it has been proposed, as a causal factor, that overusing the incisor bite, or protracted bite, overstimulates the lateral pterygoid muscles causing anterior discal displacement [7]. TMJ ID subjects could definitely be biting in a more incisor manner as they tend to protract their jaw to reduce the disc, therefore training their masticatory muscles and supporting tissues more than non-TMJ subjects [64]. In addition, TMJ ID subjects who exert great forces during parafunction such as grinding [18,25], could also be training and increasing their incisor bite forces.

for both groups, we obtained lower values in incisor clenching conmpared to the molar clenching. This relationship has been reported and rationalized by other investigators who have addressed the basic muscle mechanics (e.g. lever arm), occlusion contact and pressure point differences as well as neurophysiological reasons such as proprioceptor distribution[60,65].

Opening Isometric Forces

To our knwoledge, force values of jaw opening have never been reported in the dental nor the orthopaedic literature probably because of technical difficulty, absence of a clear rationale or because

it was not a teeth-occlusion issue. Our results show that TMJ ID subjects exhibited a non-significant tendency ($p = .283$) to lower opening maximum isometric bite forces compared to control subjects ($59.0N \pm 61.1$ and $68.9N \pm 20.1$, respectively). The reasons could be similar to those explaining the comparison observed during molar clenching (ie, muscular weakness due to non-use, tissue tolerance and pain). It is important to note that during the testing period, the majority of the TMJ ID subjects felt resistance while trying to perform the opening MVC. This could explain, in part, the lower values found in the TMJ ID group. It is suggested that a mechanical interference of the wires between the coronoid process and the mandibular condyle could compress soft tissues, and therefore cause a resistance sensation and discomfort thus inhibiting maximum opening. Significantly lower opening forces were recorded compared to the two clenching positions for both groups. This is understandable as the masticatory system is physiologically built to clench and bite and that the opening motion is primarily and neurophysiologically guided by gravity first, and upon exertion by the digastric and the hyoid muscles.

Finally, TMJ ID subjects who probably exert greater forces during parafunction and show hyperactiva EMG signals are not necessarily reinforcing their muscles. Hyperactive muscles are usually more fatigued and overused rather than stronger. Also, irritation due to parafunction could be a constant cause of inflammation of the supporting tissues. This could definitely be the case in molar clenching, though not in incisor clenching since parafunction has not been reported in that position. Therefore we may be dealing with a true training effect in incisor biting as mentioned previously. Finally, the neuromuscular strategy for controlling mandibular forces is still intangible.

It is suggested that a NEUROMUSCULAR ADAPTATION might occur in TMJ ID masticatory muscles (especially the SLP and ILP as they contain 90% of the jaw proprioceptive endings) to control internal joint instability. Therefore, they are uniquely equipped to permit fine neural proprioception [25]. This plasticity termed "INTERNAL REARRANGEMENT" has been formerly reported by Ogus [24]. Muscular combinations are expected to be different in TMJ ID to minimize joint reaction forces and to protect

the tissues from being overloaded in order to continue maintaining vital functions such as breathing, eating, and communication.

CONCLUSIONS

1- Our results do not support the hypothesis that the temporalis and the masseter muscles are hyperactive in TMJ ID. There was no significant differences in integrated LE EMG activities between the two groups for all the static tasks.

2- TMJ ID is a syndrome that definitely affects the lateral pterygoid muscles, directly or indirectly. The presence of altered muscular activities of the SLP and ILP muscles in TMJ ID subjects was present in half of the static tasks performed. In the TMJ group, the ILP muscle was significantly more active during resisted protraction ($p=.046$), in incisor clenching ($p=.031$) and at rest ($p=.29$). As for the SLP, it was significantly less active in TMJ ID group during incisor clenching only ($p=.020$). Hypoactivity of the SLP has been reported previously [14]. It is suggested that the term hyperactivity commonly used to describe the muscles in TMJ ID be used for the ILP only.

3- According to our results, the normal role of the ILP muscle needs to be refined as it is very active during maximum clenching (integrated LE EMG of the control group: molar clenching $176\mu V \pm 94.1$ and incisor clenching $97\mu V \pm 16.5$ respectively).

4- Our results suggest that the normal function of the SLP includes activation during protraction (integrated LE EMG of the control group: $83\mu V \pm 54.1$) probably to maintain joint congruence[15].

5- Pathological function of the ILP muscle dominates over the SLP muscle because of its strong dual role. In TMJ ID subjects, the ILP muscle is actively contributing during opening and protraction as expected. The ILP muscle may become hyperactive in specific positions to help in stabilizing and positioning the condyle and the disc in ID cases.

6- TMJ ID subjects exerted lower isometric forces in molar clenching possibly due to lower tissue tolerance, pain, muscle weakness and inhibition of the masticatory system (control group: 419.1 ± 47N and TMJ group: 297 ± 115, p = .042). Incisor bite forces, however, showed a tendency to be higher in the TMJ ID group (233N over 180.5N, p = .168), possibly resulting from the training of a protracted bite and/or hyperactivity of the ILP associated with ID.

The present study has attempted to elucidate the sophisticated muscular control of TMJ ID during basic jaw positions. It can be suggested that a larger sample size would help to obtain more concluding results. More studies are still required to elucidate the interaction between the SLP and ILP muscles in TMJ disorders. More research will needed to find out if muscular dysfunction is a cause or a consequence of TMJ ID. Moreover, increased attention should be addressed on the normal and pathological role of the lateral pterygoid muscles and how they influence the disc and condyle as well as clarifying the insertion of the SLP and ILP onto the disc.

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THE LATERAL PTERYGOID AND OTHER MASTICATORY MUSCLES ACTIVITIES IN TEMPOROMANDIBULAR JOINT INTERNAL DERANGEMENT DURING DYNAMIC CONDITIONS

ARTICLE NUMBER TWO

Running Head: *TMJ Dynamic Analysis*

ABSTRACT

Surface and intramuscular electromyography (EMG), electrogoniometry, kinetics and joint sound recordings were used to compare the role of the superior and inferior lateral pterygoid muscles (SLP & ILP), the masseter and the temporalis muscles between subjects affected by temporomandibular joint (TMJ) internal derangement (ID) and control subjects during jaw dynamic motion. To determine if muscular activity differed between the two groups, the EMG signals were analyzed through four EMG data treatments used as our dependent variables: 1-the integrated linear envelope (LE) EMG normalized by 100% maximum voluntary contraction (MVC) per open-close-clench phases; 2- the integrated LE EMG normalized by MVC by primary function; 3- the integrated LE EMG normalized by peak; and 4- the descriptive analysis of the LE EMG curves. Results showed, that there is no strong reason to believe that the temporalis and the masseter muscles are involved in a hyperactive manner in TMJ ID. However, they exhibited muscular incoordination that could be related to ID and clicking. Results of the different EMG treatments showed that the SLP and ILP exhibited uncoordinated and altered contraction patterns. The ILP became an agonist in clenching along with the SLP possibly to stabilize the condyle against the eminentia. The normalization by peak which seems the most sensitive and reliable EMG treatment used, demonstrated that in TMJ ID, the masseter, the temporalis and more so the ILP muscles contracted closer to their peak amplitude. In other words, they needed more muscular contribution to perform dynamic mastication compared to control subjects. However, the SLP results depended on the phase. It showed hypercontraction during the dynamic phases (opening and closing) and hypocontraction during clenching. The results also demonstrated that the ILP and SLP muscles roles were complex. During dynamic motion, they act as stabilizers of the joint and discal units. It is suggested that in TMJ ID, clicking may be a consequence of muscular incoordination and that a neuromuscular adaptation is occurring to preserve effective masticatory function.

KEY WORDS: Electromyography, Lateral Pterygoid Muscles, Masticatory muscles, Temporomandibular joint disorders, internal derangement,

The Lateral Pterygoid and Other Masticatory Muscles Activity in Temporomandibular Joint Internal Derangement and Control Subjects during Dynamic Conditions

Presently, cranio-mandibular practice is in a state of confusion, primarily due to a lack of theoretical consensus as to the nature and cause of internal derangement (ID) of the temporomandibular joint (TMJ). The leading voices have proposed a muscular and active cause as opposed to a passive and non-muscular one, whether it is primary or secondary to posture, malocclusion, trauma, stress and parafunction in opposition to a possible passive or non-muscular origin. The most popular hypotheses are that hyperactivity of the masseter and temporalis [1-9] cause or is a consequence of TMJ disorders. In addition, weaker clenching forces as well as lower levels of activity in TMJ compare to control subjects have been reported [4,8,10]. On the other hand, the lateral pterygoid muscles have been described as being hyperactive, hypoactive or exhibiting altered activity [11-17]. To add to the puzzlement, anatomical uncertainty regarding the insertion of the SLP muscle onto the intra-articular disc and/or mandibular condyle makes it difficult to understand how it functions, and what the EMG recordings actually represent. Undoubtedly, a close relationship between the functions of the lateral pterygoid muscles and TMJ ID has been reported. More specifically, the SLP has been identified as the cause of ID by pulling the disc anteriorly [18,19]. Muscle incoordination during mastication has also been reported as causing TMJ ID clicking. The importance of these two muscles in internal joint function of the jaw cannot be overestimated as they are attached to the intra-articular disc and/or the head of the mandibular condyle. The topic of dynamic muscle activity of the lateral pterygoid muscle in ID has only been lightly touched but it is of prime necessity to understand and better define the muscular pathomechanics in TMJ ID.

Various limitations of previous studies have restricted their interpretation such as: unexplicit samples and uncontrolled head and neck posture which may have lead previous authors to believe erroneously that the masticatory muscles were hyperactive. The relationship between EMG and jaw

forces has never been explored. In addition, most of the hypotheses regarding the function of the lateral pterygoid muscle associated with TMJ ID were either based on theoretical reports or on experimental studies without defining the population. Most of those were simply conducted on normal subjects, on the masseter and temporalis or finally during static tasks alone. Very few investigations studied lateral pterygoid muscle EMG activity during dynamic motion. Significant contribution in this field were done by Isberg *et al.* [13] and Zijun *et al.*[15] by specifically studying the masticatory muscles in TMJ disorders during dynamic conditions. No noticeable EMG activity was noted in the temporalis and the masseter during normal opening and closing of the mouth, but in association with discal displacement, continuous muscle activity was provoked when the disc was displaced [13]. Moreover, Zijun *et al.* [15] published that all patients with TMJ anterior disc displacement with reduction demonstrated different quantitative and qualitative EMG signals. Some recordings of the SLP and ILP showed inverse, equal, synchronous, hypoactivity and hyperactivity. However, there is still a definite lack of information concerning the role of the lateral pterygoid muscles in pure arthrogenous ID cases during dynamic motion of the TMJ.

Therefore, there is a necessity to fill the gap for a further understanding of the TMJ muscle mechanics in regards to ID. Hence, there is a need to measure and compare masticatory muscle activity during dynamic and functional jaw motion between TMJ ID and control subjects.

Consequently, the purpose of this study was to analyze the myoelectric activity of four masticatory muscles with special attention to the ILP and SLP during functional dynamic jaw motion in subjects with and without TMJ ID disorders. These muscles were investigated because they are anatomically and functionally related to TMJ ID and because they are believed to be abnormally recruited in TMJ ID. Surface EMG of the masseter and the temporalis muscles were also recorded to compare their activity with the lateral pterygoid muscle and to assess their recruitment pattern and contribution in order to have a complete dynamic masticatory profile in TMJ ID. It was, *a priori*, hypothesized that TMJ ID subjects would exhibit altered and irregular muscle activities compare to the control group. The present investigation also intends to discuss the role of the ILP and SLP as well as

their relationship to the ID dysfunctional joint providing a clinical and etiologic understanding of their normal and pathological roles.

METHODOLOGY

Subjects

Twenty one volunteer female subjects (11 control and 10 TMJ ID subjects) aged between 19 and 37 years (mean age and standard deviation 25.1 years ±4.2 for control and 27.1 years ±5.7 for TMJ ID) participated in the study. The selection criteria for ID were: 1- TMJ pain, tenderness or discomfort on at least one TMJ, 2- a diagnosis of a TMJ disorder 3- discal clicking with corresponding specific movement dysfunction or deviation. The concept that ID syndrome of the TMJ can be assumed from these three criteria is now widely accepted [13,14,18,20-22]. Two subjects were excluded from the experimental sample: in one subject the EMG signal was too noisy to analyze while the slippage of an intramuscular electrode affected the second subject. Eleven healthy females, aged matched (criteria modified from Goldstein[23]) served as control subjects. Female subjects were chosen as they are the best representative group reported in epidemiological studies [24].

Prior to the study, two questionnaires were filled out by all subjects in order to avoid medical complications (particularly those related to the needle insertions) and to record specific conditions that could account for variations in the results, and lastly, to precisely identify our experimental sample. All pertinent clinical information from these questionnaires and from the subjective, objective assessments of the TMJ are summarized in Table 1 and 2. The TMJ ID group were all diagnosed with unilateral TMJ ID and movement dysfunctions was present in all cases. This research proposal was reviewed and approved by the Human Research Ethics Committee of the Faculty of Health Science of the University of Ottawa. Prior to the one hour testing period, the subjects were asked to read an information letter and sign a consent form.

Table 1: Clinical information from the pre-test questionnaires and the TMJ musculoskeletal assessments of the two groups.

Information	TMJ ID Group	Control Group
Subjects with at least 28 teeth	10 (100%)	11 (100%)
Subjects missing back teeth	3 (30%)	1 (9%)
Subjects with bite discomfort	5 (50%)	0 (0%)
Subjects being grinders or clenchers	4 (40%)	1 (9%)
Subjects who had major dental work	0 (0%)	0 (0%)
Subjects who had orthodontic or occlusal treatment	6 (60%)	7 (64%)
Subjects with an history of head/neck and/or TMJ therapy	5 (50%)	0 (0%)
Subjects with osteoarthritis or other related health problems including medication intake	0 (0%)	0 (0%)

*Values indicate number of subjects. Numbers in parentheses are percentages of the group

In order to carry out the objectives of the study, the following measurement techniques were used simultaneously to collect data: intramuscular and surface electrode EMG, kinematics using a flexible electrogoniometer, kinetics using two custom-made force transducers and sound recordings with a contact microphone.

Table 2: Clinical characteristics and range of motion of the TMJ from the subjective and objective assessments of the two groups.

Information	TMJ ID Group	Control Group
Mean Age	27.1 years	25.2 years
ID side	3 left/5 right	N/A
Duration of ID	2-11 years	N/A
Opening (deviation > 2mm)	52.8mm (5 left,3 right)	53.4mm
Protraction/Retraction	4.1/3.5mm	4.0/3.9mm
Left/Right Translation	9.6/8.7mm	8.6/9.8mm

N/A: non-applicable

Electromyography

Two sterilized bipolar fine-wire electrodes were inserted into the SLP and ILP on the affected side in the experimental sample and on the left side or the most used one for the control group. The electrodes were made according to Giroux & Lamontagne [26]. The selection of an insertion technique was guided by an extensive theoretical and practical review of numerous investigators [1,11,12,26-28]. The needles and wires were sterilized in a TIME 250 AUTOCLAVE. They were inserted with the aid of a 26 gauge hypodermic needle into each muscle via an extraoral approach through the sigmoid notch. No post-insertion sequelae or technical difficulties were encountered. A physiatrist specialized in EMG implantation performed the insertions with the subject in a comfortable supine position with slight head rotation. A topical cutaneous analgesic cream (EMLA, Euctetic Mixture of Local Anesthetic, Astra Pharma Inc., Toronto, Canada) was used with success to decrease or eliminate the insertion pain. The analgesia efficiency depended on the area of and time since application. Bipolar surface EMG electrodes, pairs of silver-silver chloride (MEDI-TRACE, Graphic Controls, Ganaboque Canada), were

placed 1 cm apart along the muscular fibres of the masseter and the temporalis. One surface electrode was fixed to the ipsilateral collar bone as the ground reference electrode.

The raw EMG signals of each muscle were amplified through a differential bioamplifier (High performance AC preamplifier GRASS P511, input impedance of 20 MΩ differential) with adjustable high gain ranging from 2000 to 10,000 and filtered with a 0.03-3kHz bandpass filter, low noise common mode rejection) and were recorded at a frequency of 1000 Hz.

Kinematics

A digital-based system using a flexible electrogoniometer (Penny & Giles, BIOMETRICS, Toronto, Canada) was used to record jaw angular displacement in the sagittal and frontal planes during dynamic jaw opening and closing. The electrogoniometer consisted of a central strain gauge encased in a flexible steel strip surrounded by a spring with two end plates for attachment to the skin. This device allowed the simultaneous and independent measurement of joint rotation in the sagittal and frontal planes providing two analogue output signals. The two end plates were fixed to the fronto-temporalis and mandibular surfaces by double sided tape. The calibration was within the Biometric system (1 volt= 90°; positive reading correspond to the closing period and negative reading to the opening period). A 100 Hz sampling frequency was selected for the angular displacement recordings.

Kinetics

The force signal of the three MVC's in opening, molar and incisor clenching were registered with two custom-made, calibrated force transducers. The bite force transducer was linear ($r=0.990081$) and the regression equation was $y=56.664x - 0.687$. Whereas the opening jaw force device showed also linear response ($r=0.999$) with a regression equation described as $y=627.714x + 0$. transducers respectively. The closing bite force transducer consisted of two steel beams mounted together (4.8mm inter-space) with a method similar to that described by Dechow & Carlson [29]. The opening jaw force device consisted of an eight cm wide and one cm thick steel plate with a chin

shaped plastic sitter at one end and solidly fixed with two large clamps to a horizontal beam at the other end. A full wheatstone bridge configuration strain gauge system (high resistance 350 Ω elements) was bonded to the two surfaces of the opening beam and the two closing steel plates. In the present investigation, the force data are not presented, the EMG signals registered while performing the MVC were used to normalized our data.

Sound recordings

Jaw clicking was detected by a sensitive contact microphone fixed directly onto the skin at the TMJ joint line and secured with tape. Correct placement was defined by the investigators after palpation and reproducibility of the sound. The microphone was connected to an adjustable signal amplifier which fed into our analogue to digital (A/D) conversion interface.

Mandibular displacement, EMG and clicking signals were collected simultaneously using a specialized data acquisition system (BIOAD [30]) to allow synchronisation of the input recordings and for better data analysis. Figure 1 depicts a schematic diagram of the testing set-up and apparatus used and the data acquisition system.

Procedures

Two dynamic tasks were performed by both groups of subjects: 1) maximum (slow and full) open-close-clench cycle and 2) functional gum chewing. The two tasks were executed to assess the dynamic muscular coordination of the SLP and ILP, the muscular activity of the masseter and temporalis and the interaction of all muscles during clicking. Gum chewing relates to normal mastication which is the most important function of the masticatory system.

Subjects were asked to chew normally but were encouraged to produce a discal interference sound. Figure 2 depicts the experimental set-up showing a subject performing the dynamic gum chewing. These cycles were registered three times during five seconds periods in order to collect at least three full cycles each time.

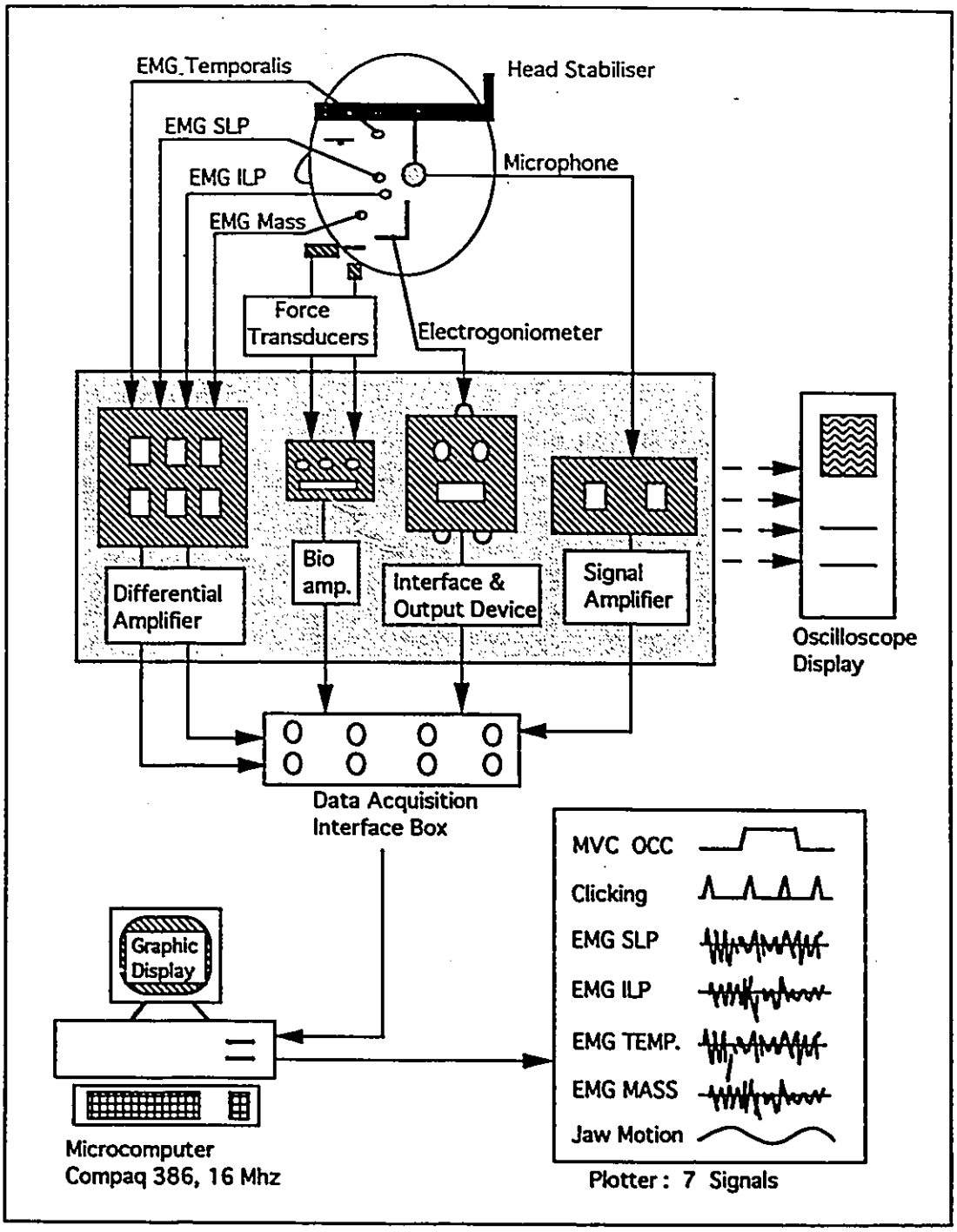

Figure 1. Schematic diagram of the experimental apparatus and the data acquisition system.

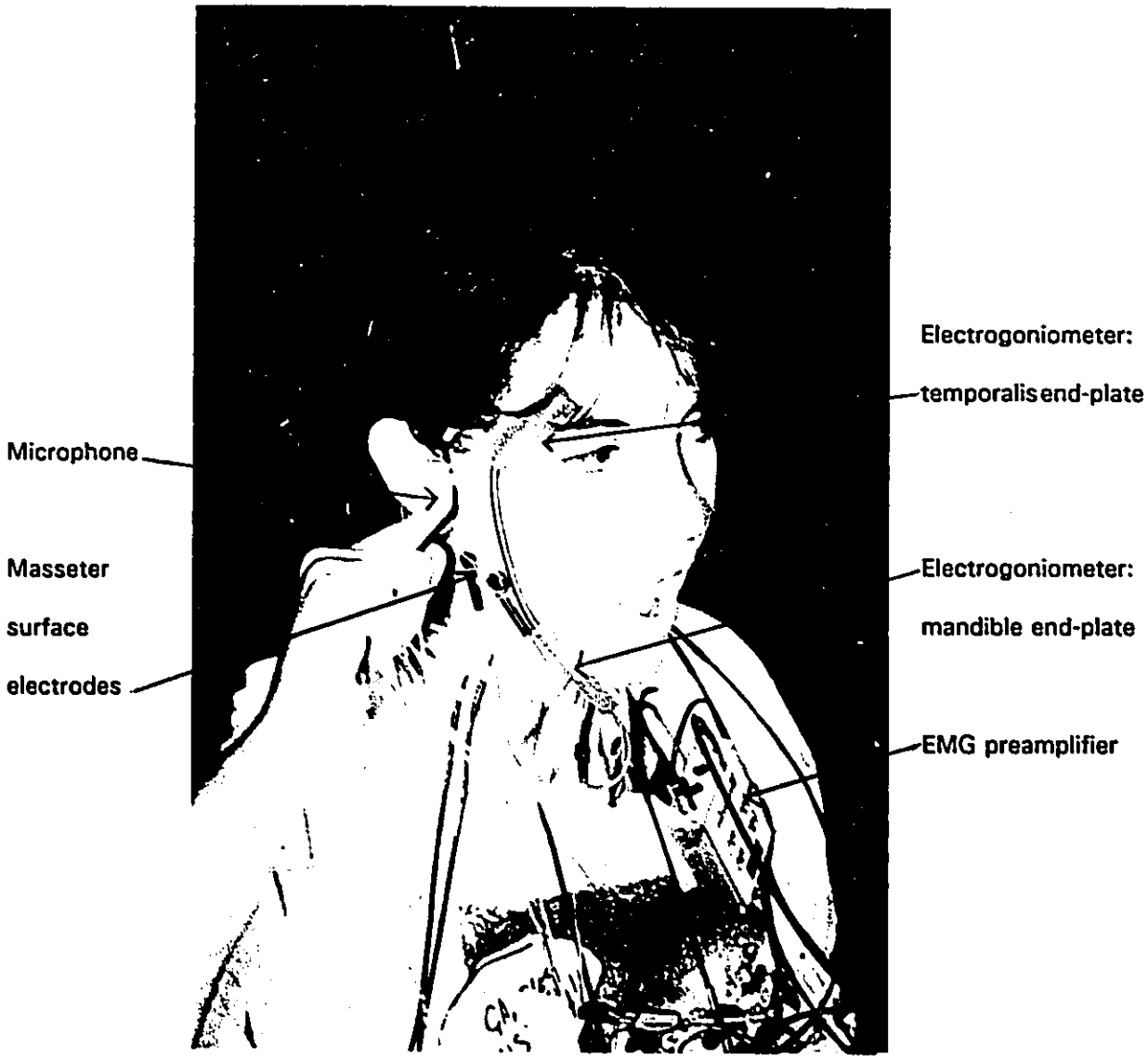

Figure 2. Experimental set-up showing a subject performing the dynamic gum chewing.

Three trials of the three MVCs were performed by all subjects: in opening and in molar and incisor clenching. The MVC during jaw opening was performed by asking the subjects to open forcefully onto an instrumented steel plate used as the resistive device to appraise the depression force. A head stabilisation was used to limit any maxillary or head cephalad motion and to isolate mandibular motion. The two MVCs during jaw closing (molar and incisor) were measured by biting onto a custom-made steel fork force transducer covered with a cushioned material. The MVC were used to normalized our dynamic EMG signals to allow better intersubjec' comparison.

The resting EMG activities of the four muscles (SLP, ILP, masseter, temporalis) were also recorded to be subtracted from the dynamic signals. For the three MVC and the rest condition EMG raw signals, a constant one second window, averaged over three trials and time normalized, was used for data analysis. Once inserted, the wires were insulated with adhesive tape, surface electrodes were applied and EMG signals were tested and calibrated. The subject was then seated in a custom-made straight chair, the EMG amplifier output of the four muscles along with the electrogoniometer and force transducer were connected to the data acquisition system which was controlled by a micro computer (Compaq 386, 16MHz). Before data collection, verification of the insertion of the ILP and SLP was done according to Gross and Lipke [26] and McNamara [31]. This testing was most applicable in the normal subjects as we had to rely on the inserter's experience of the electrode placement as well as by EMG signal interpretation for the TMJ ID subject group. All signals were analyzed on a microcomputer (Compaq 386, 16MHz) through a custom-made biomechanics software package (BIOPROC [30]) which is fully compatible with the data acquisition system used.

Data Reduction and Statistical Design

Theoretically, the presence of myoelectric signals indicates if a muscle is active or not and how active it is in comparison with a reference value such as at the MVC or at its peak value for a given task. In this investigation, the EMG signals of the open-close-clench and gum chewing cycles were computed into four EMG data treatments.

The raw EMG was filtered with a high-pass filter (Butherworth, dual passes) at 10Hz. The bias was removed by mean using the BIOPROC software [30], then scaled by a factor obtained from the different Grass amplification gain for each muscle. The best cycles were selected for processing by viewing the full scaled signal (5 seconds). For the three MVCs tasks, the same processing techniques were undertaken but the window used for processing was selected from the maximum force signal registered. The EMG signals were full wave rectified and filtered with a fourth order, dual passes, critically dampened low-pass filter (cutoff frequency = 6Hz), producing a LE EMG curve. The area under this curve was computed to obtain the integrated LE EMG. The average integrated value of the rest condition was subtracted to obtain the net integrated LE EMG provided by the specific movement. From these EMG signals and data, four different dependant variables were computed during the dynamic motions to compare differences between the two samples.

1- *Integrated LE EMG normalized by 100% MVC and by phase*. Each open-close-clench cycle was divided in three phases: open, close and clench. The three phases were defined from the electrogoniometer signal. Each phase was normalized by time in percentage. For each phase, the integrated LE EMG for each muscle was computed and then normalized by the integrated LE EMG of the specific MVC (100%). The open phases were normalized by the MVC obtained in opening. The close and clench phases were normalized in percentage of MVC obtained in molar clench. For the mandibular muscles, the normalization technique using maximal isometric contraction was previously accepted and used by Jankelson and Isberg *et al.* [8,32].

2- *Integrated LE EMG normalized by 100% MVC by primary functions*. The three best trials of the gum chewing cycle were first normalized by time in percentage, then averaged. The integrated LE EMG were computed for each muscle and each subject. The net integrated LE EMG of the masseter muscle was normalized by the MVC EMG signal obtained from incisor clenching. For the temporalis and SLP muscle, the net integrated LE EMG were normalized by the MVC EMG signal obtained from molar clenching. And for the ILP muscle, the normalization was done by the MVC EMG signal obtained from

resisted opening.

3- *Integrated LE EMG normalized by peak*. The three best trials of the gum chewing cycle were first normalized by time in percentage, then averaged. The LE EMG for each muscle was computed and then normalized by peak amplitude of the chewing cycle. This normalization technique differed from the first ones as we are normalizing a dynamic cycle by a dynamic value obtained from the peak value of the cycle.

For those three families of dependent variables (all expressed in % of the MVC), ensemble averages were computed by subjects of the two groups for the four muscles. To compare means of the two samples, general linear model ANOVA (simple or one-way) were computed with NCSS software package for statistical analysis using a significant level of $\alpha \leq 0.05$ and two tailed testing. The independent variables is ID or TMJ health. The simple or one way ANOVA comparing the two groups means for each muscle in each condition separately seemed to be the best way to draw the information we were looking for without fading the contrasts and loosing some meaningful characteristics. It was well understood that some conditions and some muscles do interact but sometimes in opposite directions, therefore cancelling the effect of interest if grouped together in a two-way ANOVA. The factors are considered independent and should not be melted together. The only effect of interest being studied were the muscular activities.

Knowing that in the practical application of EMG, the pattern of the ensemble LE EMG is often a more important consideration that its amplitude, descriptive and qualitative analysis were also done.

4- *Descriptive analysis* of the gum chewing cycles were completed to withdraw qualitative characteristics of the EMG signals of the four muscles between the two samples. The whole gum chewing raw signal was normalized by time in percentage. The LE EMG for each muscle were computed and normalized by peak amplitude. Descriptive analysis of simple subjects and group average are presented. Surely, these descriptive features are very adequate and pertinent for dynamic tasks analysis and are expressed in similarities and dissimilarities between groups and muscles.

RESULTS AND DISCUSSION

The result and discussion section exposes the findings by EMG data treatments and muscles. Statistical analyses were used to analyze EMG responses with different treatments for both groups (control and TMJ ID) during different dynamic motion of the mandible: open-close-clench and gum chewing cycles.

1-Integrated LE EMG normalized by 100% MVC and by phase

Masseter

The normalized integrated results for the three phases are shown in Figure 3 for the TMJ ID and the control group respectively: 40.6% ± 31.7 and 30.7% ± 15.7 for opening, 36.6% ± 42.4 and 31.7% ± 24.7 for closing and 186.1% ± 121 and 145.0% ± 63.7 for clenching.

In the three phases, there was no significant difference ($p = .498$ for opening, $p = .809$ for closing and $p = .466$ for clenching phases) between the two groups in percentage of masseter muscle activity used (Figure 3). Therefore, both groups are using their masseter at a similar percentage of their respective MVC during the dynamic open-close-clench phases. Our results do not support the so-called masseter hyperactivity present in TMJ disorders [1,2,4-6].

High values of standard deviation possibly refers to an increase intersubject variability reported in EMG normalization by 100% MVC [33]. This could account for the non-significant differences. In cases of heterogeneity of variance, a more conservative p value provided by the NCSS software was used for our statistical analysis. This also applies to other treatments and muscles.

In both the opening and closing phases, the masseter used 30 to 40% of the MVC for both groups (Figure 3) likely to be the resting tonus and/or an eccentric jaw support or control. In the clenching phase, values of 186% and 145% were measured in the TMJ ID and control subjects respectively. These percentages over 100% can be explained by two factors: 1- a much greater force can be applied in full intercuspation (open-close-clench task) without the presence of the transducer

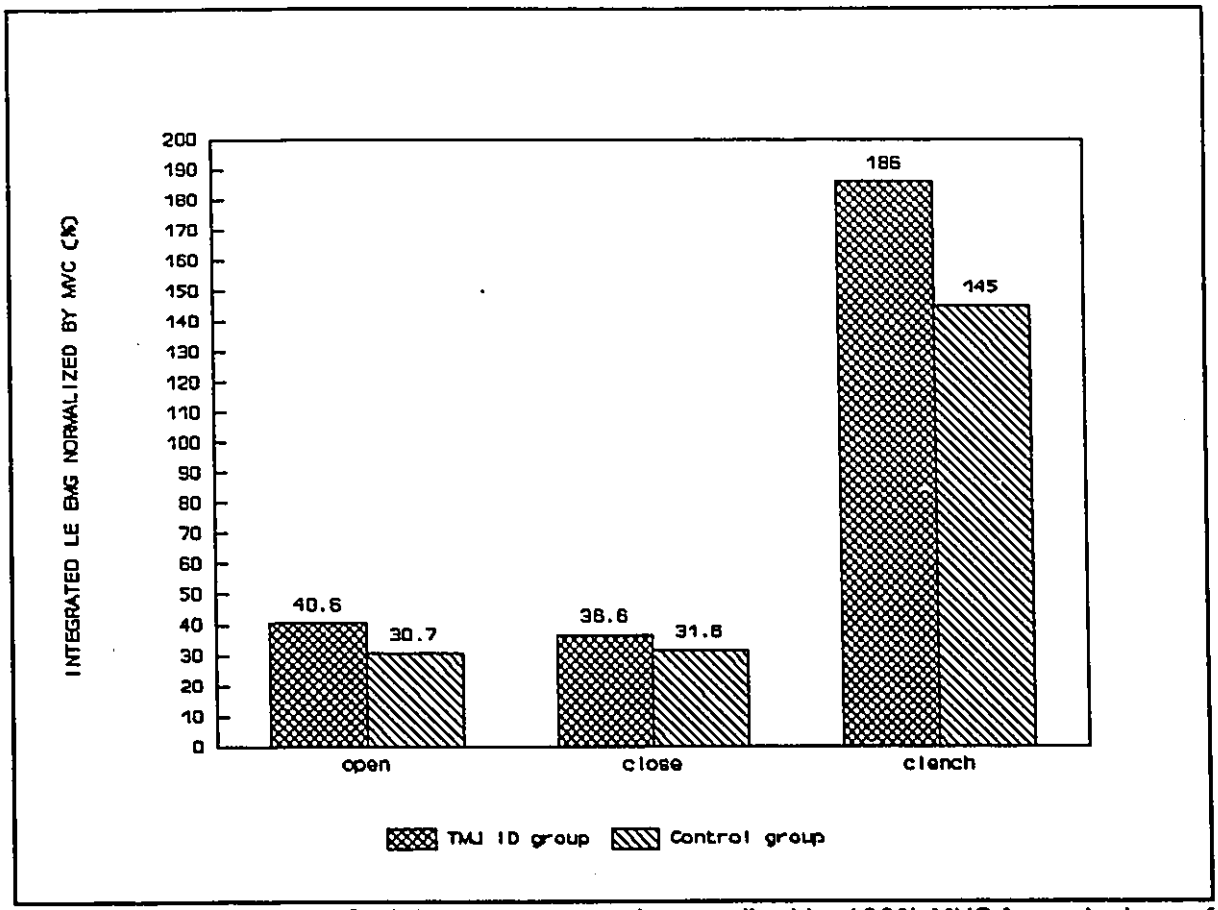

Figure 3. Integrated LE EMG of the masseter muscle normalized by 100% MVC for each phase of the OCC cycle of both groups. The open phase was normalized by the MVC in opening, the close and clench phase were normalized by the MVC in molar clench.

(12mm) between the teeth (MVC task), which prevents complete closure and lowers the lever arm fibres efficiency; 2- dynamic maximum contraction produces more EMG activity than the static MVC task that can be obtained by a greater fibre recruitment in dynamic situations [33]. The same explanations remains for the other muscles.

Temporalis

Figure 4 shows that during the open phase the TMJ ID group is using 64.3% ± 37.3 of their MVC compared to 118.2% ± 51.1 for the control group that represents a significant difference ($p = .018$). It is suggested that the temporalis muscle is more active and efficient in control compared

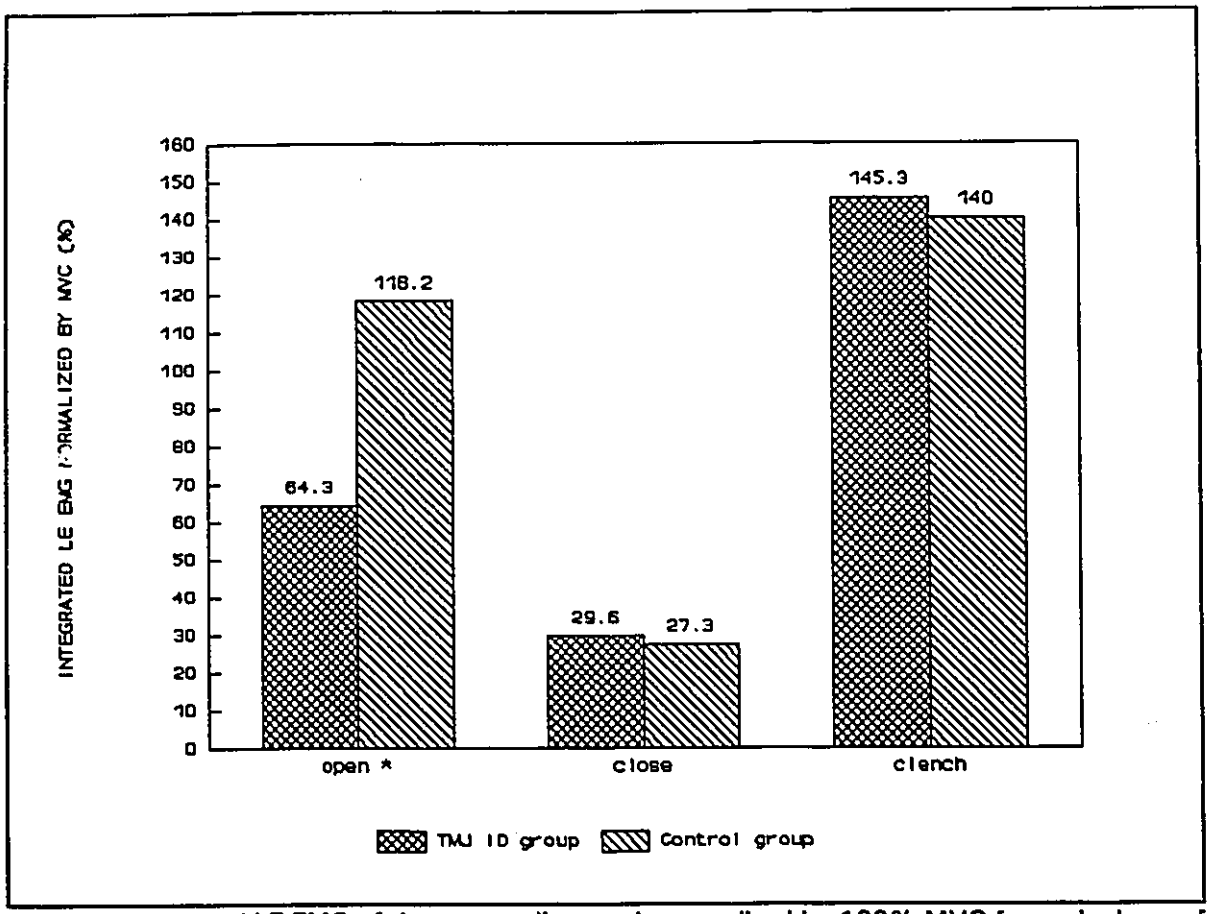

Figure 4. Integrated LE EMG of the temporalis muscle normalized by 100% MVC for each phase of the OCC cycle of both groups. The open phase was normalized by the MVC in opening and the close and clench phase were normalized by the MVC in molar clench.

*: significant difference $\alpha \leq 0.05$

to TMJ ID subjects. There was no significant differences ($p = .849$) between the groups during closing (TMJ ID: 29.6% ± 17.7 and control: 27.3% ± 23.6) and no significant difference ($p = .929$) during clenching (TMJ ID: 145.4% ± 134.6 and control: 139.9% ± 86.8). Consequently, our findings do not support the concept of higher postural temporalis activity in TMJ disorders [1,2,4-6]. However, it is suggested that the masseter and temporalis muscles normally exert an eccentric control on the mandible during closing [34] using around 30% of their MVC.

Superior Lateral Pterygoid

No significant difference was detected for the opening phase ($p = .690$) and closing ($p = .913$) phases (Figure 5). But, during the clenching phase, the SLP EMG contribution is significantly lower ($p = .029$) in the TMJ ID group (TMJ ID: 108.9% ± 41.5 over control: 158.8% ± 53.9). It is understood that in TMJ ID cases the SLP is not functionning efficiently or has a tendency to loose its stabilizing function during clenching. Interestingly, it was proposed that the SLP muscle would be less active in ID because a contracture would occur decreasing its ability to contract [13-15].

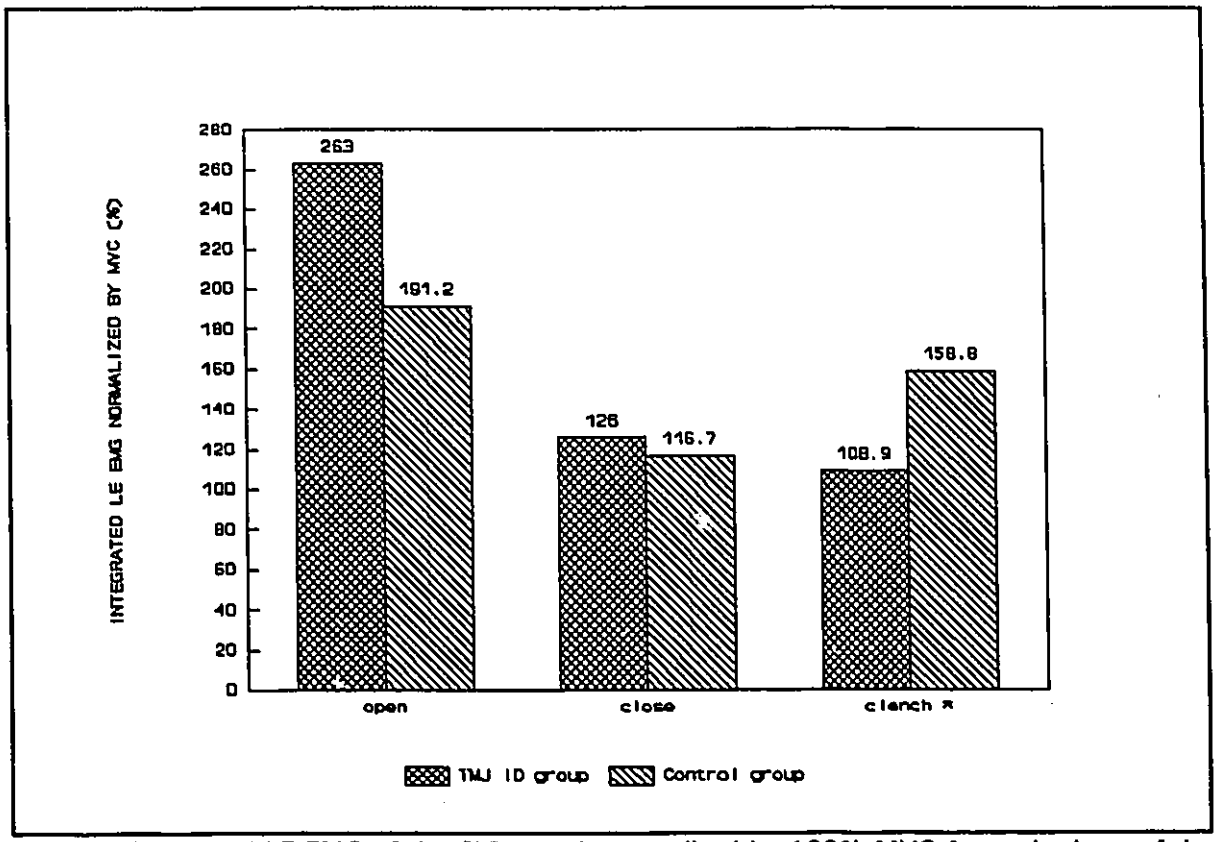

Figure 5. Integrated LE EMG of the SLP muscle normalized by 100% MVC for each phase of the OCC cycle of both groups. The open phase was normalized by the MVC in opening, the close and clench phases were normalized by the MVC in molar clench.

*: significant difference $\alpha \leq 0.05$

Inferior Lateral Pterygoid

Figure 6 shows the integrated LE EMG of the ILP muscle during the open, close and clench phases. The TMJ ID subjects used significantly more muscular activity in the open ($p = .050$) and close ($p = .045$) phases compared to the control group. In ID cases, the ILP seems to be "overworking" probably to increase joint and discal stability during movement. The fact that patients suffering from craniomandibular disorders use greater relative masticatory forces than normal subjects during chewing activities have been reported previously [11,26,36,38]. This may be attributed to a lack of strength in dysfunctional muscles of mastication, lower efficiency of their masticatory system and/or lower MVC forces due to pain and inhibition. It has been mentioned that the ILP muscle plays an important role in

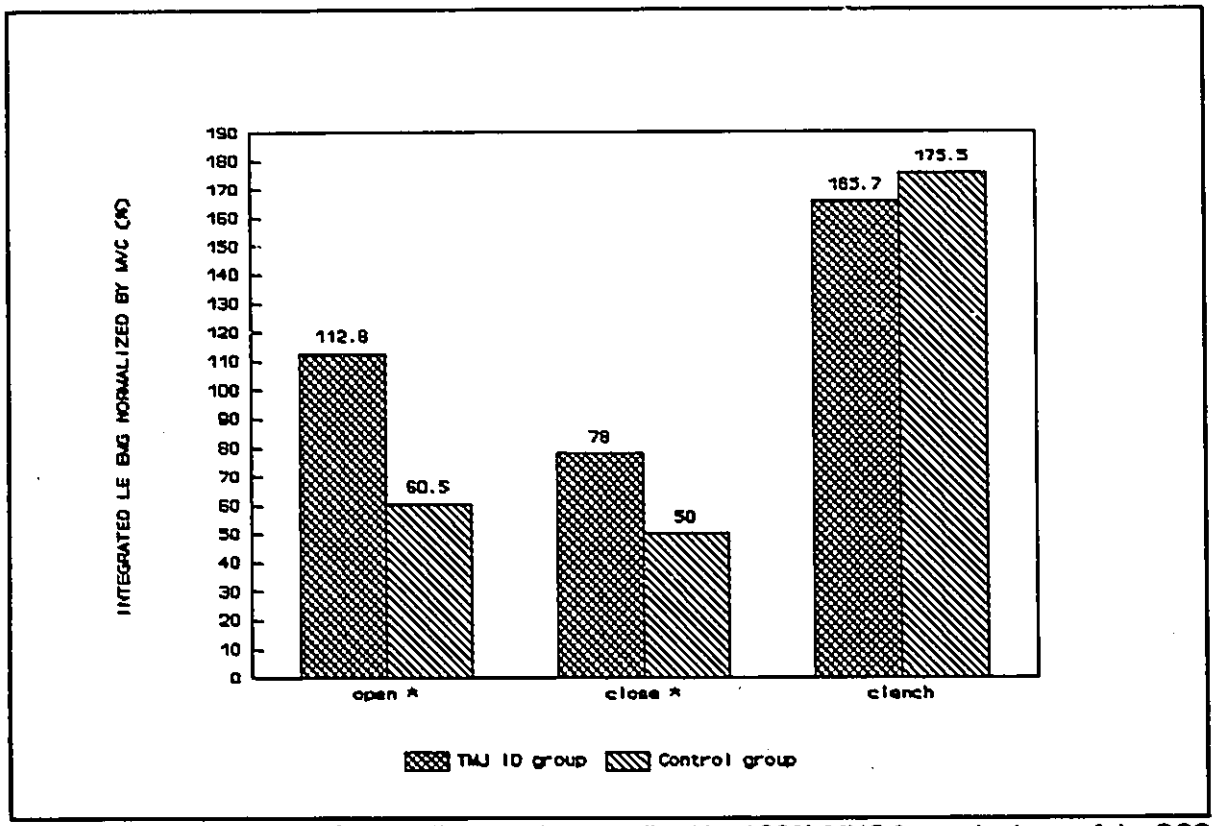

Figure 6: Integrated LE EMG of the ILP muscle normalized by 100% MVC for each phase of the OCC cycle of both groups. The opening phase was normalized by the MVC in opening, the close and clench phases were normalized by the MVC in molar clench.

*: significant difference $\alpha \leq 0.05$

stabilizing the condylar head and disc against the eminentia during closing movement and is the most frequently involved in TMJ dysfunction [37]. This correlates with our findings.

2- Integrated LE EMG normalized by 100% MVC per primary function

The results of the muscular activity of each muscle during gum chewing normalized by the MVC of their primary action are shown in Figure 7. These muscular contribution data give information regarding the extent (% of MVC) each muscle contributes to functional mastication comparing TMJ ID and control subjects. Results for TMJ ID and control subjects are as follow: masseter normalized

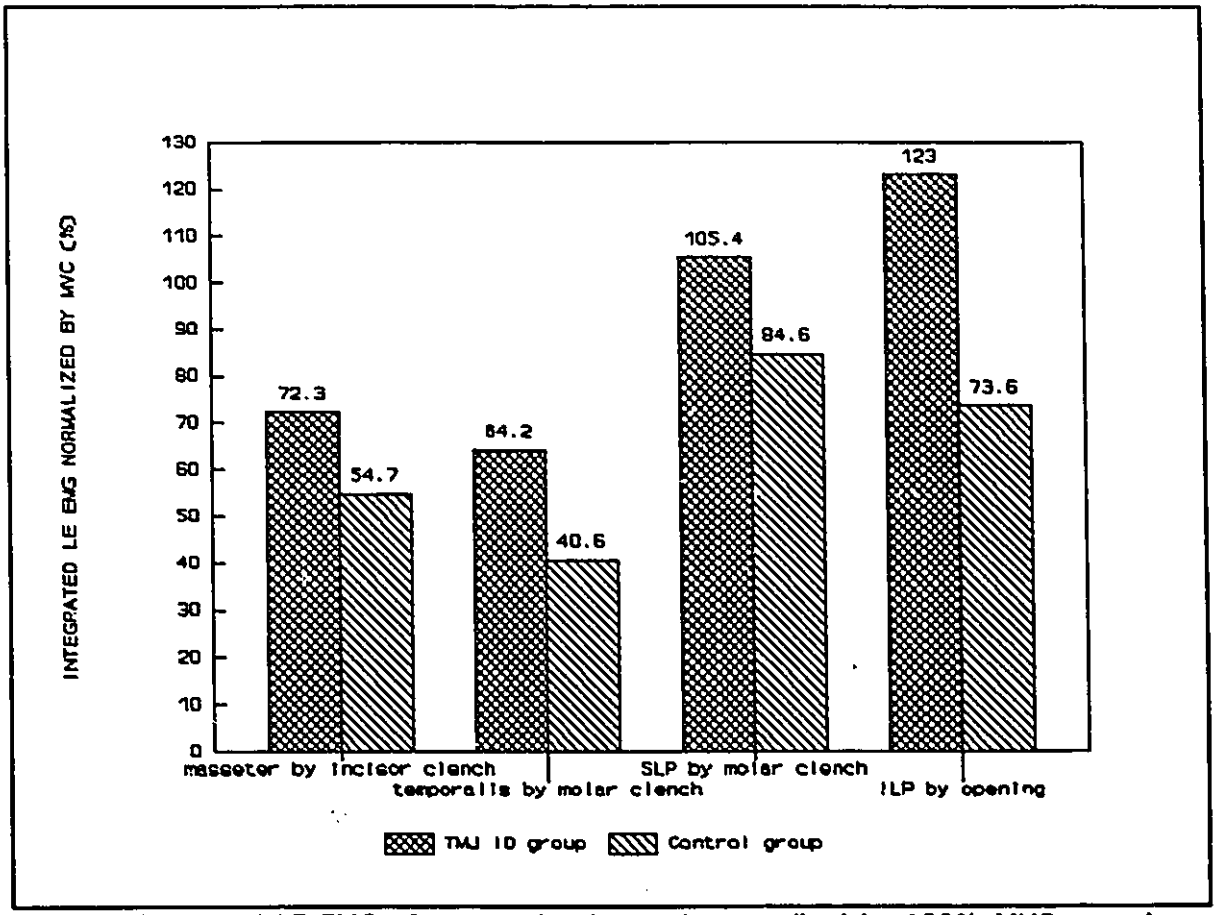

Figure 7. Integrated LE EMG of a gum chewing cycle normalized by 100% MVC per primary function. The masseter was normalized by the incisor clench MVC, the temporalis and SLP by the molar clench MVC and the ILP by the opening MVC.

by incisor clench 72.3% ± 25.3 and 54.7% + 26.1, temporalis by molar clench 64.2% ± 33.7 and 40.6% ± 16.5, SLP by molar clench 105.4% ± 35.8 and 84.6% ± 46.6 and ILP by opening 122.0% ± 85.7 and 73.0% ± 37.3 respectively.The four simple ANOVA revealed non-significant differences between the two groups for the four muscles (masseter, p = .206; temporalis, p = .116; SLP, p = .386;, ILP, p = .322). Yet, there is a small but consistent tendency for the four muscles in the TMJ ID sample to use more muscular activity (or to contract closer to their MVC) compared to the control group. This trend was expected if in fact dysfunctional muscles are weaker, hyperactive and fatigue and need more muscular contribution to chew effectively. In other words, normal muscles which are generally stronger, need less muscle activity to perform mastication. To our knowledge, there are no comparable published data with respect to these specific muscles and samples.

Normalization is usually done to facilitate comparison between subjects (and groups) and to decrease inter-subject variability. But as stated by Yang and Winter [33], normalization by 100% MVC actually increases the intersubject variability due to fatiguability, co-contraction, non-linearity of EMG-force relationship and the problem of performing a true maximum. Moreover, the normalization by 100% MVC do not reflect EMG amplitude of dynamic movement. All of which limits the interpretation of the results. Therefore, normalization by peak was conducted as it reduces the intersubject variability. It has been demonstrated that the EMG normalized by peak is more sensitive and more reliable to compare EMG signals [33].

3-Integrated LE EMG normalized by peak

The integrated LE EMG normalized by peak for each muscle (of both groups) are shown in Figure 8. Simple ANOVA showed significant differences between the two groups for the masseter (p = .002), the temporalis (p = .031) and the ILP (p = .046) muscles. The TMJ ID group are contracting closer to their peak amplitude (in percentage) during chewing compared to the control group. Similar findings were reported previously suggesting that patients suffering from craniomandibular disorders use greater relative masticatory forces than normal subjects during chewing activities [4,8,10,11,26,35,36].

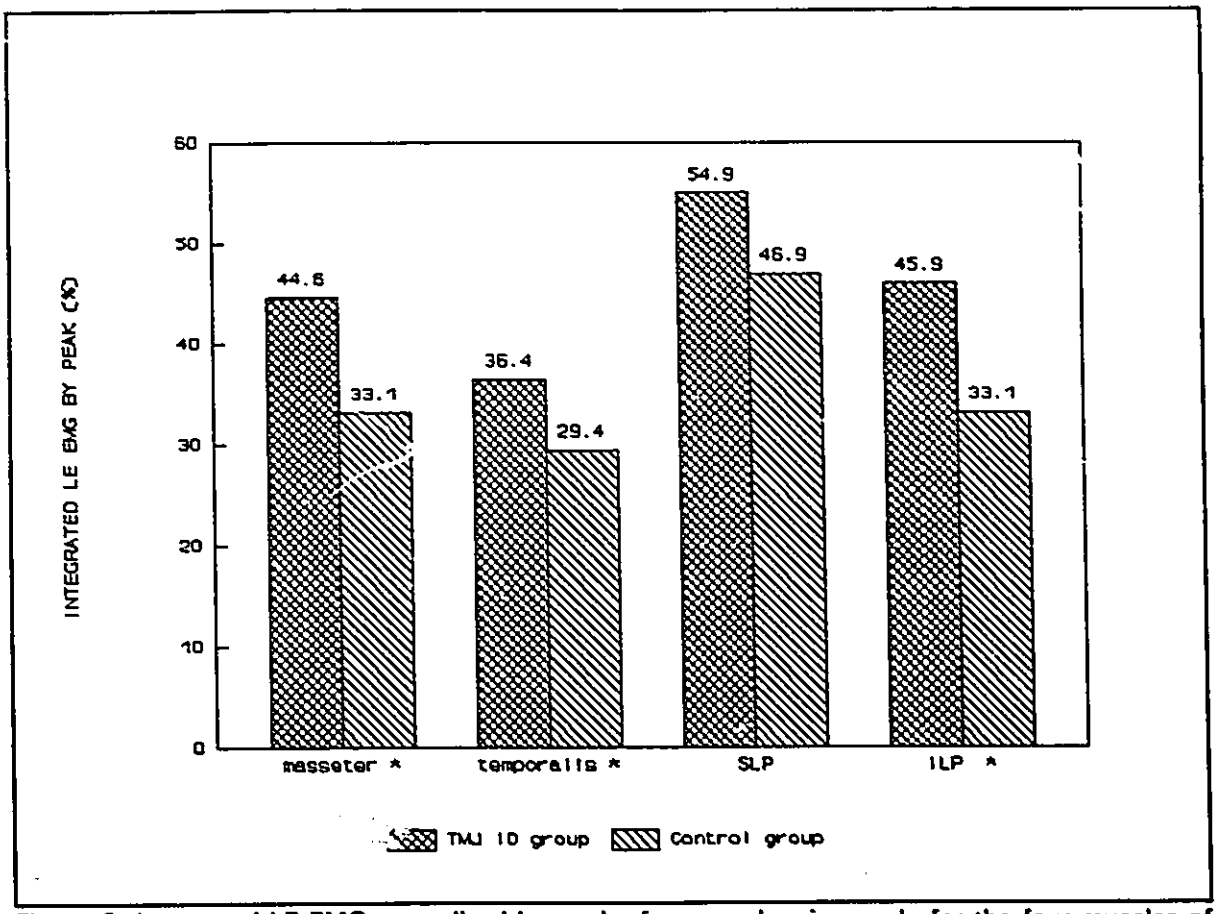

Figure 8. Integrated LE EMG normalized by peak of a gum chewing cycle for the four muscles of both groups.

*: significant difference $\alpha \leq 0.05$

These results can also be explained by the fact that TMJ ID subjects have dysfunctional masticatory muscles that are weaker, have a poor or inadequate endurance, a tendency to be fatigued, a low contraction efficiency, and therefore require more contraction to perform mastication. For muscles comparison, the SLP values are greater than the masseter, temporalis and ILP ones which might reinforce the importance of the SLP function during dynamic chewing, as a controller of the disc\joint unit.

With normalization, the comparison of EMG amplitude measures remains difficult. The shape of the ensemble LE EMG is also an important consideration with its amplitude.

4-Descriptive and Qualitative Results

Figures 9 and 10 show the mean LE EMG signal (normalized by peak and by time in percentage) of each muscle during gum chewing for the two groups.

In the control group (Figure 9), the masseter and temporalis muscles are active in closing but mainly in the clenching phase, as expected. These findings are in agreement with previous ones [12]. The closing motion probably requires minimal activity of those two muscles [38,39] or could be guided by the elastic return of the soft tissues stretched during opening.

The SLP's primary functions are to elevate or close the mandible, to clench and to chew [12,26,31]. One other function is to stabilize the disc/condyle complex especially during closing while the disc pulled by the posterior discal tissues is returning with the condyle under the eminentia [21,31]. It has also been described as being only active in forceful strokes; otherwise it is at a resting tonus [26]. Therefore, it was expected that to be activated more in closing and clenching and less in opening. This hypothesis

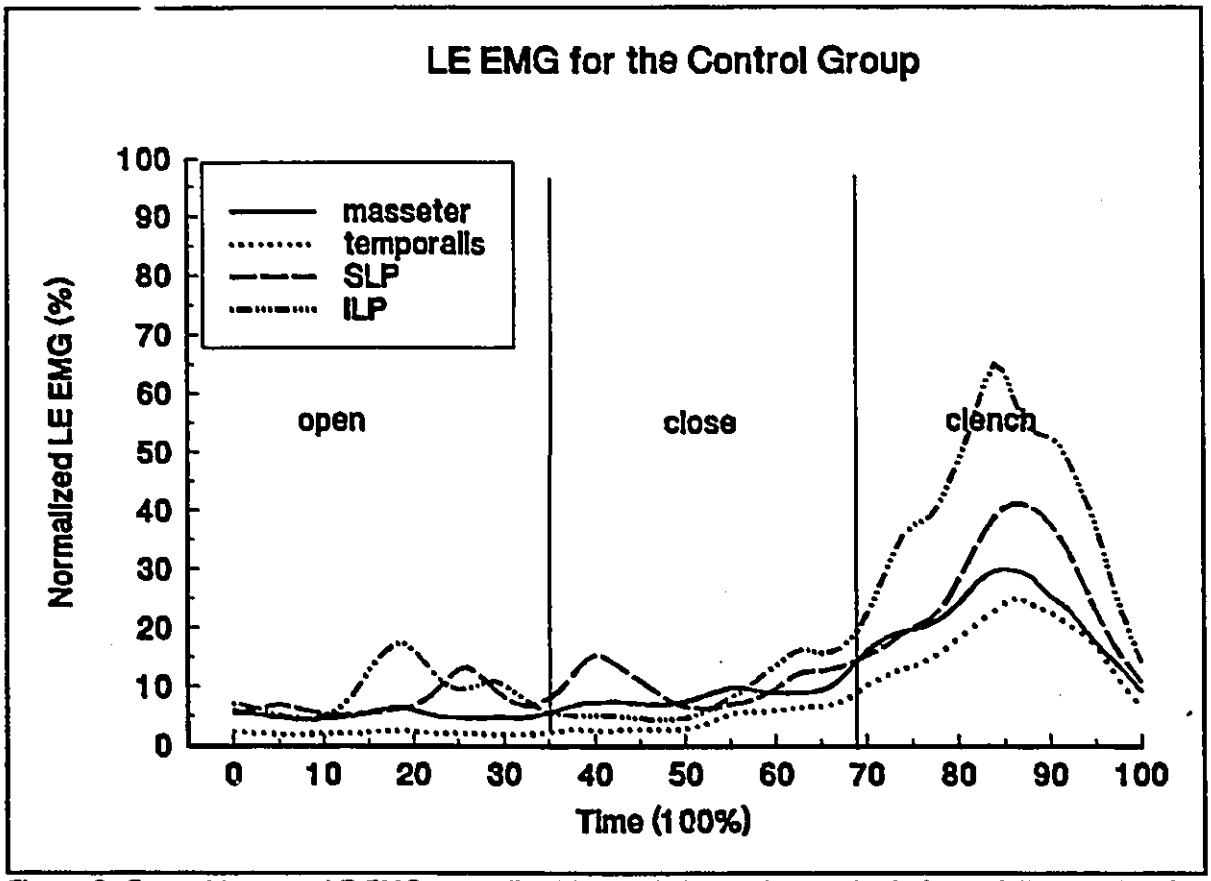

Figure 9. Ensemble mean LE EMG normalized by peak for each muscle during a full gum chewing cycle for the control group.

was not perfectly confirmed. From the LE EMG curve of the control subjects, the SLP was strongly active in clenching but was also moderately active at the end of opening and at the beginning of closing. Thus it is probably contracting to control the disc near complete opening, when the disc is more vulnerable to displacement. Therefore, it is suggested that the normal function of the SLP in dynamic motion is to control the disc and condyle to provide good intra-articular mechanics.

Again in the control group, the ILP was active during opening, as expected, but also during clenching. In both groups, the mean LE EMG curve of the ILP showed high activity during clenching. Juniper's [40] registered EMG activity of the ILP muscle during opening and SLP EMG activity during closing in control subjects. This was not confirmed with this study. Lehr and Owens [41] reported separate roles of the two lateral pterygoid muscles. This was not confirmed either. In some control subjects during clenching both lateral pterygoid heads are active (Figure 9). It is suggested that the ILP is acting as a synergist during clenching. The ILP muscle would contribute to a vertical force vector to stabilize the condyle under the eminentia, as previously mentionned [42]. This activation of the SLP and ILP during clenching is a good example of cocontraction to achieve an end result. This concept of co-contraction has also been reported by Gibbs *et al.* [12] in order to gain stability, fine motor control, smooth coordinated movement and protective splinting. These findings support Zijun [16] conclusion who stated that SLP and ILP dysfunction exist in normal subjects as and early stage of muscular dysfunction leading to ID.

In the TMJ ID group (Figure 10), the masseter muscle was more active compare to its peak value in the three phases compared to the controls. But, the temporalis activity difference was mainly during clenching. The SLP muscle seemed to contract all through the cycle at a relatively high peak % level probably to maintain control of the disc throughout joint motion and clenching. This is understandable as the SLP muscle is considered as a postural muscle, built for stability and control of the disc and joint [21,31]. The ILP muscle was active during opening and closing and very active through clenching. It is advocated that in ID cases where disc\condyle incoordination exist, the cocontraction function of the ILP muscle mentioned earlier is increased in all phases to further stabilize the deranged joint system.

The TMJ ID LE EMG curve showed an overall increase in percentage of peak used by all muscles. Moreover, each of them is contracting earlier (40% of the cycle) compared to the control

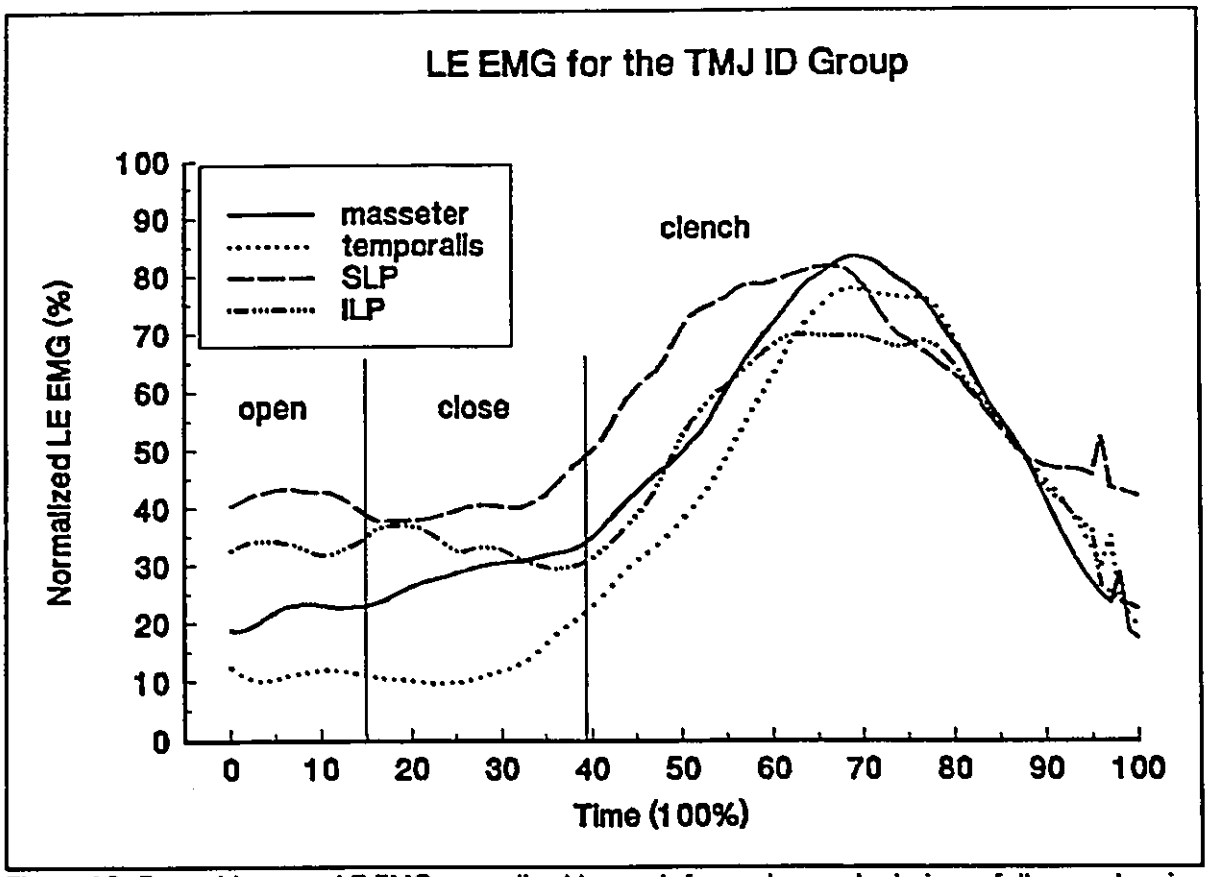

Figure 10. Ensemble mean LE EMG normalized by peak for each muscle during a full gum chewing cycle for the TMJ ID group.

subjects (70% of the cycle). This could be because more stabilization is needed in ID and the masticatory system compensates by contracting earlier and at a higher level.

As presented in Figure 9 and 10, the mean LE EMG for the gum cycles of different heterogenous cycles smoothen specific characteristics of our TMJ ID sample. Therefore, a qualitative analysis of each subjects open-close-clench and gum chewing cycles was found more appropriate to withdraw interesting features of our dysfunctional muscles. EMG noise was more present in the experimental group, especially in the SLP muscle, and less in the ILP muscle. The contraction pattern of the SLP was quite variable: it could be silent in closing yet contract in opening; it contracted strongly in clenching; it was occasionally active in clenching for a longer period of time than the other muscles and it even had completely silent periods during closing and clenching. All these observations were definite signs of muscular deficiency, and incoordination of the SLP in TMJ ID. For the ILP, the LE EMG curves of the different TMJ ID subjects did not show clear opening EMG signals on all cases.

Clicking and Kinematics

It is well recognized that the first or opening click is considered to be due to disc reduction and the second or closing click reflects the occurrence of re-subluxation. In other words, between the first and the second click, the disc/condyle relationship is considered normal, but after the closing sound and before the jaw clicks on reopening, the disc is anteriorly displaced [13,38]. In all individuals of the TMJ ID group, the clicking sounds were recorded and synchronized with our electrogoniometer and EMG signal. Only one subject had multiple crepitus underlying a discogenic click. The opening or subluxation click was usually present around the mid-opening.

The closing click was sometimes so soft that in a minority of cases it could not be recorded even at maximum amplification, but the opening click was always registered. Final opening sounds have been associated with ILP hyperactivity [15,38]. The closing click position was interpreted by observing obvious lateral deviation of the angular displacement in the frontal plane of the mandible. This closing click was usually heard from the middle closing phase to the beginning of the clenching one.

It is proposed that these middle range clicks may be associated with muscular incoordination as suggested previously [11,16,37,43]. Some of these explanations are as follow: a faulty reciprocal activation of the SLP and ILP implying that some muscle are being abused or overused at the expense of the rest of the system, incoordinate activity of the SLP and ILP muscles so that the condyle cannot remain in its normal relationship with the disc and desynchronisation of the masticatory muscles create joint sounds. From the LE EMG curves of the TMJ ID cases, ILP and temporalis muscles had a tendency to start contracting as the disc re-displaced over the condyle after the closing click. This come in agreement with Isberg *et al* [13] who found similar correlation between the activity of the temporalis and masseter muscles during the period where the disc is displaced and while the disc reduces over the condyle.

In the EMG signal of the ILP muscle more than the SLP muscle, there is movement artefact during both discal clicks. Simultaneous registration of mandibular displacement and joint sounds showed a clear frontal (lateral) mandibular displacement of few degrees as the joint was clicking. This relates well with the medial component of anterior displacement of the disc.

Overall, a change in the normal function of the SLP and ILP would affect the maintenance of the joint surfaces of the disc, condyle and eminentia in dynamic motion. Or ID would demand variation in muscle contraction patterns in order to satisfy mandibular vital functions [42]. More precisely, the results of the present study show that the ILP muscle seems to compensate for an uncoordinated inner-joint mechanics. This cooperation and cocontraction of antagonist muscle is typical in jaw movement to gain stability and fine motor control [12]. On the other hand, it is impossible to state if these altered muscular activities cause, maintain or result from ID.

In summary, Table 3 represents the statistical and descriptive results between the two groups for all muscles and all EMG treatments.

Table 3. Statistical and descriptive results summary between the two groups for the four EMG treatments of the four muscles during dynamic tasks.

TMJ ID vs CONTROL		Masseter	Temporalis	SLP	ILP
Normalization by MVC by phase	open	=	↓↓	=	↑↑
	close	=	=	=	↑↑
	clench	=	=	↓↓	=
Normalization by MVC by function		=	=	=	=
Normalization by peak		↑↑	↑↑	↑	↑↑
Descriptive Analysis	open	↑	=	↑	↑
	close	↑	=	↑	↑
	clench	↑	↑	↑	↑

=: no significant difference $\alpha \leq 0.05$

↑↑ or ↓↓: significant difference

↑ or ↓ : tendency.

There is no strong reason to believe that the temporalis and the masseter muscles are involved in a hyperactive manner in TMJ ID. However, the fact that the masseter and temporalis exhibit

muscular incoordination related to reciprocal clicking has been considered. It is suggested that the term hyperactivity be changed to altered function to qualify the muscular activity in ID.

The results of the different EMG treatments of the dynamic conditions showed that the SLP and ILP muscles exhibited uncoordinated and altered contraction patterns as reported previously [4,13,15,16]. In control and TMJ subjects, the ILP muscle is very active in clenching (along with the SLP) possibly to stabilize the condyle against the eminentia. This cocontraction function is increased in TMJ ID to further stabilize the deranged joint system.

The normalization by peak, which seems the most sensitive and reliable EMG treatment used [33], demonstrated that in TMJ ID, the masseter, the temporalis and more so the ILP muscles contract closer to their peak amplitude. In other words, they need more muscular contribution to perform dynamic mastication compared to control subjects. However, the SLP integrated LE EMG findings fluctuated among phases and demonstrated hypercontraction in the dynamic phases (opening and closing) and hypocontraction during clenching.

In conclusion, it is suggested that the ID pathology should be reconsidered as primarily a muscular disorder. Clinically, there is always an abnormality of one or more jaw muscles and\or joint involvement. The presence of a neuromuscular imbalance and functional incoordination of the masticatory muscles may be associated with TMJ clicking and TMJ ID. A neuromuscular adaptation could affect the SLP and ILP muscles to maintain basic jaw function. This concept of "internal rearrangement" was well explained by Ogus [44] and is supported by Yemm [7] and Owens [18]. It may be that joint involvement is secondary to functional muscular adaptations particularly of the lateral pterygoid muscles [7]. For the clinician, successful treatment would likely involve neuromuscular reeducation and promotion of muscular healing by means such as: muscles stimulation, specific coordination and stability exercises, stretching, biofeedback, relaxation, deep heat and electrotherapy. This would also explain why the Rocabado [20] regimen of exercises is so successful in controlling joint clicking, stabilizing the derangement and reducing TMJ muscular and joint pains.

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CONCLUSIONS

Masticatory Muscles Activities in Temporomandibular Joint Internal Derangement

This general conclusion presents the scientific contribution of the two presented papers. For clarity, the conclusions are presented in the same format as presented in both papers. The last section, a general discussion, briefly presents our suggestion on ID pathology derived from our results.

Masseter and Temporalis Muscles

The findings of the static EMG and the dynamic EMG analysis as well as the isometric force analysis of subjects with and without TMJ ID cannot support the hypothesis that the masseter and temporalis muscles are hyperactive in TMJ ID, as reported by many investigators (Moyers, 1950; Gervais *et al.*, 1989; Glaros *et al.*, 1989; Dahlstrom, 1990). There was no significant difference in integrated LE EMG activities between the two groups for all the five static tasks. One potential cause for this finding may be due to carefully chosen subjects as arthrogenous cases (not myogenous). In the dynamic task analysis, the integrated LE EMG normalized by peak demonstrated that in TMJ ID subjects, the masseter (TMJ ID 44.8% and control 33.1%, $p=.002$) and the temporalis (TMJ ID 38.4% and control 29.4%, $p=.031$) muscles contracted significantly closer to their peak amplitude. Hence, they need more muscle contribution to perform gum mastication compare to controls. The fact that the masseter and temporalis muscles exhibit muscular incoordination in ID is considered.

The inferior lateral pterygoid and superior lateral pterygoid muscles

The results of this study for both analysis demonstrated the complexity of the lateral pterygoid muscles during static and dynamic conditions as primary muscles of inner TMJ mechanics.

The author strongly believes that normal function of the ILP and SLP muscles should be redefined. The ILP muscle is known to be active in opening and silent in closing and clenching

(McNamara, 1973; Wood *et al.*, 1986). Moreover, the ILP muscle was found to be active in the second half of the clenching phase (Wood *et al.*, 1986). According to our results the ILP muscle is very active during the maximum clenching task (integrated LE EMG of the control group: molar clenching 176.0μV $\pm$ 94.1 and incisor clenching 97.0μV $\pm$ 16.5) and dynamic clenching (175.5% of MVC). Consequently, clenching demands co-contraction of the ILP and the SLP muscles.

Our results also suggest that the normal role of the SLP includes activation in protraction as well as in closing. It was noted that the SLP muscle is mostly activated in the final part of closing. During protraction, the SLP muscle slightly contracts (control group: 83.0μV $\pm$ 54.1). Possibly to control the disc rotating back on the condyle to maintain joint congruence (Wilkinson, 1988). Overall, in control subjects, the SLP muscle appears to be more active during dynamic motions (opening and closing) than static conditions, reinforcing its disc-controller role.

Therefore, our findings cannot fully support separate roles of the two lateral pterygoid muscles, especially during dynamic function as advocated previously (McNamara, 1973; Gibbs et al. 1984; Juniper, 1984). Instead, a cocontraction between the SLP and ILP muscles in normal cases was demonstrated during clenching and protraction. As mentioned by Gibbs *et al.* (1983), this is necessary in basing jaw position and in clenching to gain stability and fine motor control.

The presence of altered and uncoordinated muscular activity of the SLP and ILP muscle in TMJ ID was present in half of the static task and all the dynamic ones. This has been reported previously (Gervais *et al.*, 1989; Isberg *et al.*, 1985;Zijun *et al.*, 1989; Juniper, 1984). The results of the static integrated LE EMG analysis demonstrated that the ILP was actively contributing in all the movements whether it was performing its normal actions of opening and protraction, or when performing closing and clenching. In the TMJ group, the ILP muscle was significantly more active during resisted protraction ($p = .046$), incisor clenching ($p = .031$) and at rest ($p = .029$) . Therefore, it lost its functional specificity, during clenching as it was acting in synergy with the SLP instead of reciprocal. In TMJ ID, the two lateral pterygoid muscles had become dependent. Similar interpretations have been reported

earlier (Isberg *et al.*, 1895; Juniper, 1987; Gibbs *et al.*, 1989; Zijun, 1989). In control and TMJ subjects, the ILP muscle is very active in clenching possibly to stabilize the condyle against the eminentia. The cocontraction function, mentionned in the control subjects, is increased in TMJ ID to further stabilize the deranged joint system. The ILP may become hyperactive in specific positions to help in stabilizing and positioning the condyle and the disc in TMJ ID cases.

As for the SLP muscle, it was significantly less active in the TMJ ID group during incisor clenching only ($p = .020$). Hypofunction of the SLP muscle has been reported previously (Carpentier, 1988). The SLP muscle appeared to lose efficiency in controlling and stabilizing the disc in ID.

In the dynamic analysis, the normalization by peak seems the most sensitive and reliable EMG treatment used (Yang & Winter, 1984). It demonstrated that in TMJ ID, the masseter, the temporalis and more so the ILP muscles contract closer to their peak amplitude. In other words, they need more muscular contribution to perform dynamic mastication compared to control subjects possibly because of a lack of strength and joint stability (Sheikholeslam *et al.*, 1982; Dahlstrom, 1989; Jankelson, 1990). However, the findings form the SLP muscles varied per phase and demonstrated hypercontraction in the dynamic phases (opening and closing) and hypocontraction during clenching.

Isometric Forces

Compared to the control group, the TMJ ID subjects exerted significantly ($p = .042$) lower molar bite forces (419N ± 47 and 297N ± 115). This could be secondary to lower muscle strength or atrophy, lower tissue tolerance and higher pain reported in TMJ disorders. However, incisor bite forces showed a tendency to be higher in the TMJ ID group (233.0N ± 82.7 and 180.3N ± 60.4, $p = .168$). The causes may include the training of a protracted bite and/or hyperactivity of the ILP associated with ID.

Internal Derangement

In summary, TMJ ID is a syndrome that definitely involves the lateral pterygoid muscles,

directly or indirectly. The presence of muscular imbalance/incoordination or altered activity of the SLP and ILP muscle in TMJ ID is undeniable, whatever the causal factors. Muscular combinations were expected to be different in TMJ ID to minimize joint reaction forces and to protect the tissues and continue to maintain vital functions. It is suggested that the term hyperfunction be changed to altered-function to qualify the muscular activity in ID. However, it remains unclear whether these altered muscular activities cause, maintain or result from ID.

The author proposes that the existence of a muscular imbalance and a functional incoordination of the masticatory system in ID. Moreover, adaptation of the masticatory muscles (especially the SLP and ILP) would happen in order to control the loss of internal joint stability. This is similar to the "internal rearrangement" suggested by Ogus (1987).

It is also proposed that TMJ clicking is associated to SLP and ILP muscles incoordination as suggested previously (Zijun, 1989). Consequently, the concepts of arthrogenous and myogenous TMJ disorders are closely related.

Finally, it is suggested that the ID pathology should be reconsidered as primarily a muscular disorder. Clinically, there is always an abnormality of one or more jaw muscles in TMJ disorders. It may be that joint involvement is due to functional muscular adaptations particularly of the lateral pterygoid muscles. For the clinician, successful treatment would likely involve neuromuscular reeducation and promotion of muscular healing by means such as: muscles stimulation, specific coordination and stability exercises, stretching, biofeedback, relaxation, deep heat, electrotherapy. This would also explain why the Rocabado (1989) regimen of exercises is so successful in controlling joint clicking, stabilizing the derangement and reducing TMJ muscular and joint pains.

Future studies are still required to better understand the interaction between the disc reduction and the muscular activity in ID and to better understand the muscular dysfunction if it a cause or a consequence of TMJ ID. Moreover, special attention should be dedicated on the lateral pterygoid muscles to understand their role in normal and pathological jaw motion.

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APPENDIX A

THESIS PROPOSAL

THE LATERAL PTERYGOID AND OTHER MASTICATORY MUSCLES ACTIVITY IN TEMPOROMANDIBULAR JOINT INTERNAL DERANGEMENT AND CONTROL SUBJECTS DURING STATIC AND DYNAMIC CONDITIONS

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CHAPTER I

INTRODUCTION

Overview

Historically, the first attempt to document a temporo-mandibular joint (TMJ) pathology was done in the 1930's by Costen: the spirit of TMJ syndrome arose. Today, craniomandibular practice is in a state of confusion primarily because there is no consensus as to the nature and cause of TMJ dysfunction. Unfortunately it places clinicians in a difficult position since they must face the problems, the patients and the underlying factors in the presence of controversial and often conflicting ideas concerning etiology, diagnosis and management (Farrar, 1983; Krauss, 1988; Gelb, 1989).

Clinical and epidemiological investigators have shown that 41 to 79% of the general population have clinical signs of TMJ syndrome, from which 60 to 80% are female aged between 20 and 40 years of age (Greene, 1982; Magnusson, 1986; Locker & Slade, 1988). The disorder presents some of the most complex and frustrating diagnostic and therapeutic problems encountered by the dental, medical and paramedical professions (Foreman, 1985). Hence, the majority of the patients are misdiagnosed, miss-oriented or mistreated because of the lack of professional training and biomechanical knowledge, including muscle mechanics of the craniomandibular complex (Rocabado, 1981; Foreman, 1985).

It is generally agreed that the etiology of TMJ disorders is multifactorial (Schwartz, 1956; McNeil et al., 1980; Mikhail, 1980; Okeson, 1981; Greene, 1982; Foreman, 1985; Clark, 1987; Gelb, 1989; Weinberg, 1980). On the other hand, psychophysiologic and "tooth/occlusion theories" supporters point to their unique causal factor (Schwartz, 1956; Laskin, 1969; Helkimo, 1975; Clarke, 1982; Eversole & Machado, 1985); both of these sharing a common outcome: myospasm. Currently, muscle hyperactivity, trauma, oral parafunction, nutritional and mechanical stresses, environmental and developmental factors, psychological and physiological factors and malocclusion dominate the literature (Figure 1). In parallel, an extensive list of predisposing, activating and perpetuating factors have also been proposed (McNeil 1980; Foreman, 1985). However the issue of the etiology of the TMJ dysfunction remains unclear. Hence, the "muscle theory" or "neuromuscular theory" is predominant, whether it is primary or secondary (Carlsson, 1980; Bergamini, 1990): it has been the central feature of all the different theories. It is now accepted that the most common TMJ disorder is ID or arthrogenous disorders; it represents 80% of all TMJ syndromes; this ID relates to an anterior (and medial) discal displacement (ADD) process (Farrar, 1972; Weinberg, 1980; Dolwick,

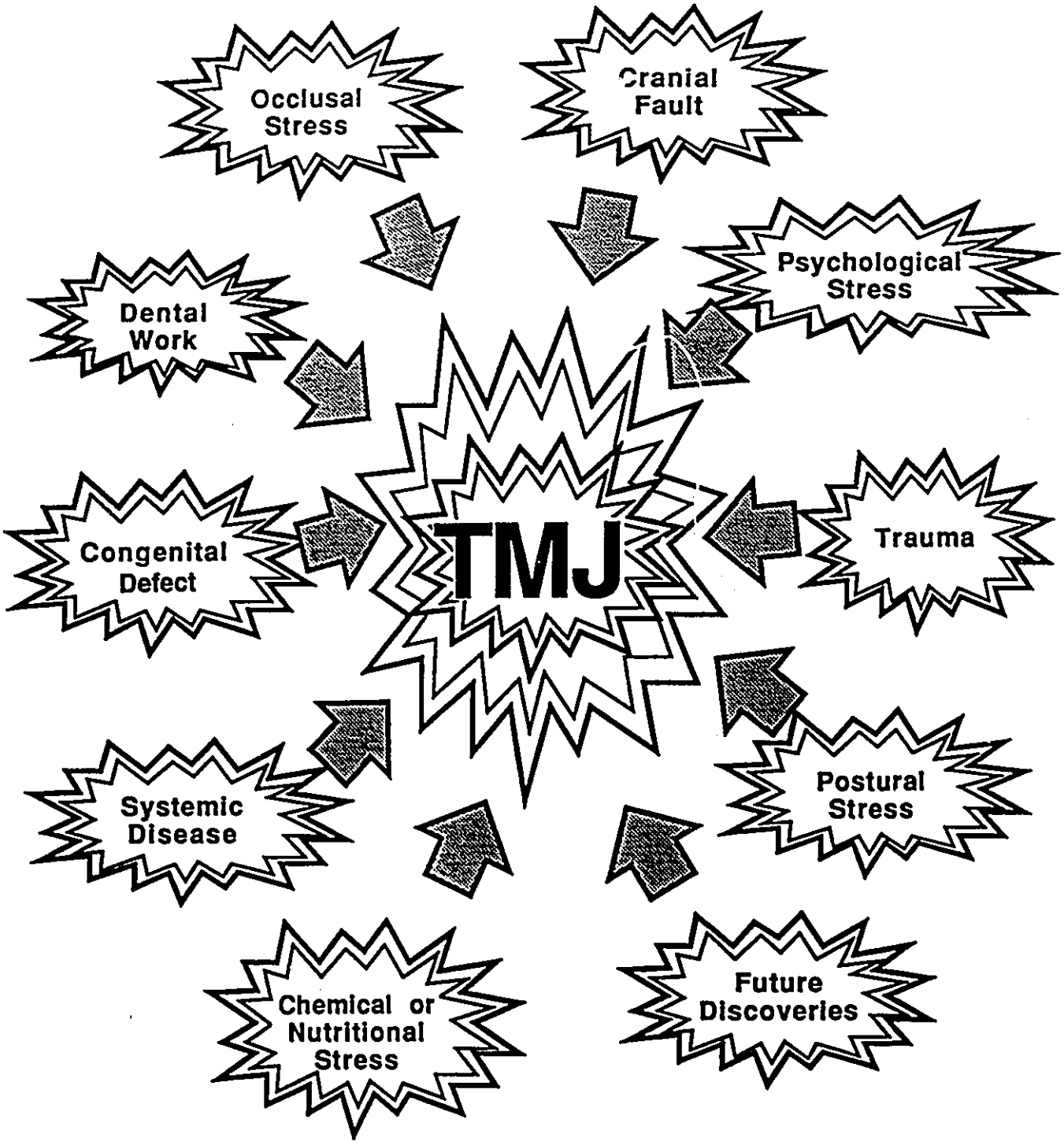

Figure 1: This drawing summarises the major causes of TMJ disorders as we understand them today.

Note: From Its all in your head (p.7) by AH Owen. 1988 Austin, Texas USA

1983; Rocabado, 1983; Isberg, 1985; Owen, 1987; Gage, 1989) (Figure 2).

The lateral pterygoid muscle (LPM) seems to be the cause of numerous temporomandibular disorders; in the human specie it is a muscle with an history of controversies. Although, small in size, it has a very large functional impact on the mechanics of the whole joint. Anatomically, the superior head of the lateral pterygoid muscle (SLP) is most commonly described as inserting onto the articular disc only or on the disc along with the condyle (Grant, 1973; MacNamara, 1973; Juniper, 1981; Bell, 1983; Bourbon, 1988). However recent dissection studies depict its attachment onto the neck of the condyle and not the disc itself, thus contradicting numerous educational and scientific representations (Carpentier, 1988; Wilkinson, 1988). Yet, debate still exists in regard to the insertion of the superior head of the lateral pterygoid (SHLP) and its relationship with the intra-articular disc. Hence, the activity of the SLP is not likely to be causing internal derangements by pulling the disc anteriorly.

It is also well recognized that it is the most difficult masticatory muscle to assess either by clinical testing, palpation or by electromyograpy (EMG). Undoubtedly, this lack of certainty about the LPM structure makes it difficult to understand how it functions and what the electromyographical (EMG) recordings actually represent.

The existence of controversies regarding the role of the SLP in the pathological and etiologic process of the disc-condyle system opposes two leading hypotheses: an hyperactivity of the SLP pulling onto the articular disc still dominates over the newly concept of an hypo or an altered activity of the SLP. Both of them are neither understood as a cause nor as a consequence of ADD. There is also an existing dilemma as to the nature of the cause of the ID: whether it is passive (secondary to hyper-opening of the jaw) or active (parafunctional hypertonicity of the LPM), both leading to ADD and arthralgia (Eversole & Machado, 1985). For more than five decades, studies have shown that myofascial pain-dysfunction (MPD) patients have higher EMG activity in the masticatory muscles that do non-MPD subjects (Moyers, 1950; Ramfjord, 1961; Griffin & Monro, 1971; Kotani *et al.*, 1980; Pancherz, 1980; Sherman, 1985; Dahlstrom, 1989; Gervais *et al.*, 1989; Glaros *et al.* 1989; Jankelson, 1990).

It is suggested that the presence of hyperactivity/spasm is due to stress, grinding, trauma, malocclusion, or pain. The psychophysiologicaltheory of TMJ dysfunction as developed by Schwatrz and Cobin in the 1950's provides one of the major theoretical frameworks for understanding its etiology: EMG hyperactivity is a response to stress and causes joint and muscles signs and symptoms (Gervais *et al.*, 1989). These schools of thought, is in agreement with the discal insertion of SLP, hence suggesting that the hyperfunctional SLP pulls the disc creating the ADD process. However, all of these studies have used surface EMG of the temporal and the masseter muscles as a basis for their theory.

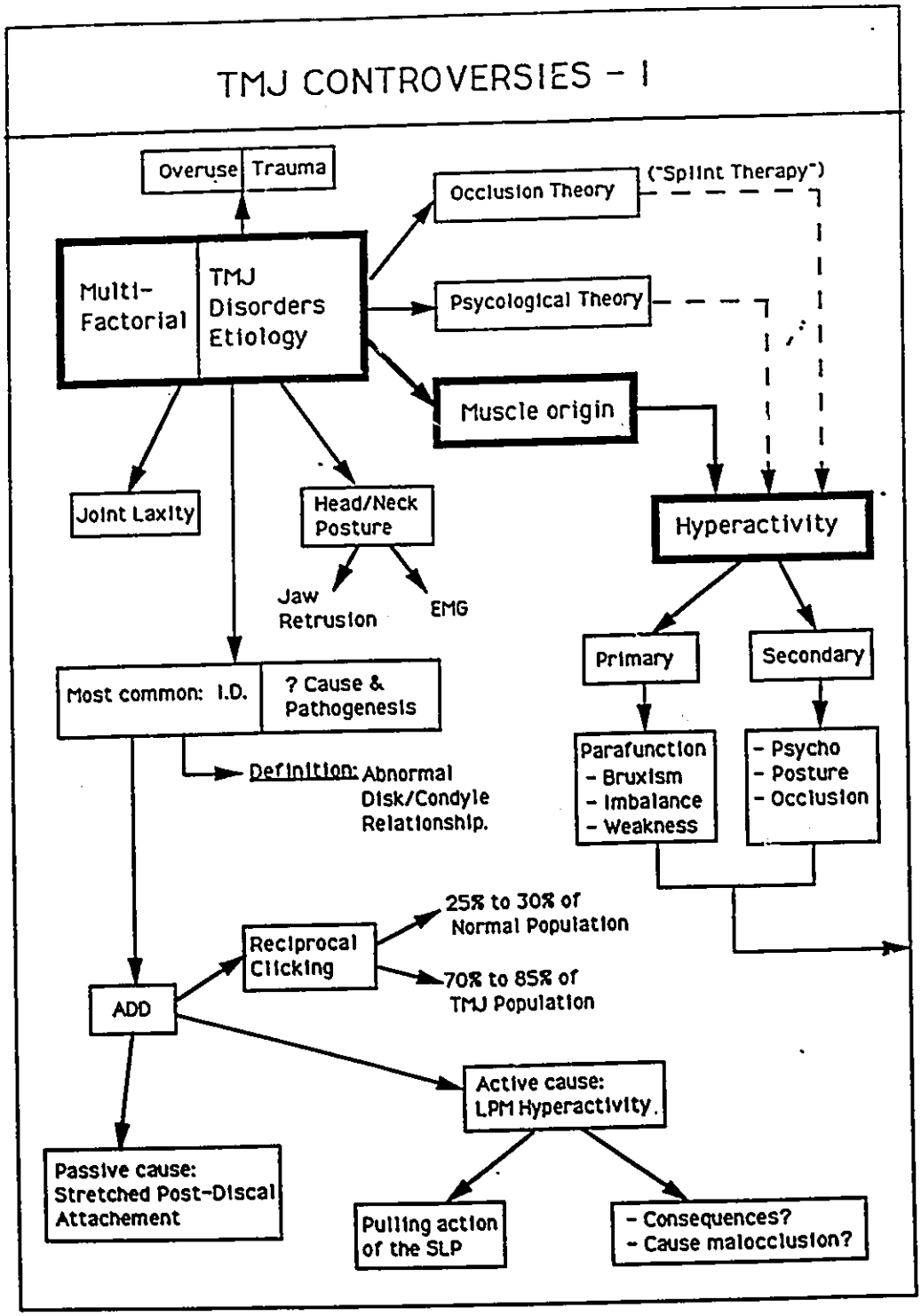

Figure 2:Diagramm of TMJ controversies, part I;

Illustrating a summary of the present knowledge in the world of TMJ pathology.

Isberg (1985) suggested that an altered EMG activity is a consequence of anterior displacement of the disc unit probably to restore and stabilize the deranged joint. On the other hand, according to Juniper (1984), hyperactivity of the SLP would cause the ADD which would produce occlusal premature contacts. Posterior occlusion could be a result, not a cause of TMJ dysfunction, leading to considerable disagreement concerning the role of malocclusion in the genesis of TMJ pain and dysfunction. More specifically, Bergamini (1990) suggested hyperactivity at rest and hypoactivity or weak bite force during function but only for the masseter and the temporalis.

Conversely, it has been suggested by Carpentier (1988), a proponent of the condyle attachment of the SLP, that hypotonicity (weak condyle protraction), not hyperactivity, of the upper head may contribute to an ADD. Furthermore, Mahan *et al.* (1983), Gibbs and Mahan (1984) and Juniper (1987) believe that tension in SLP could not be an etiologic factor in ADD because of its mechanical design. This excessive tension on the condyle's neck and disc would not displaced the disc excessively forward with respect to the condyle. It was hypothesized by Mahan *et al.*(1983), Juniper (1984), Gibbs and Mahan (1984) and Carpentier (1988), who support the condyle attachment of the disc, that SLP dysfunction is the result of ADD instead of it's source.

Other investigators (Isberg, 1985; Zijun, 1989) have reported uncoordinated and altered activity of the SLP. The disc dislocation would put the muscle in a "wrinkle state" and then stop it from contracting efficiently. As a repercussion of an ID of the TMJ, the structure and function of the SLP could be altered and possibly maintain this ID. Isberg (1985) explained that the SLP is not involved in dislocating the disc at final closure because of its attachment to the condyle. Thus, SLP dysfunction could be a result of ID not its cause. While attempting to solve this cause/consequence controversy, Zijun (1989) found SLP dysfunction (hyper- and hypo- activity) in most patients with TMJ disorders. Zijun (1989), proponent of the discal insertion on the SLP, stated that altered activity of the SLP muscle is the cause of ID. From these studies, it is obvious that there is no agreement as to whether the SLP is attached onto the disc or not, nor if it can pull the disc anteriorly or not. Moreover, a major disagreement persists as to whether the pathological SLP displays hyper, hypo or uncoordinated muscle activity (Figure 3).

This muscular activity being increased in some studies, decreased in others but also reported as irregular and altered. Moreover, fite force studies have shown that the masticatory muscles exhibited low maximum forces in TMJ disorders cases and hyperactivity of the masseter and the temporalis (Sheikoleslam *et al*, 1982;Dahlstrom, 1989; Jankelson, 1990). Therefore, it seems essential to measure the muscle activities of the masseter, temporalis, SLP and ILP during static and dynamic conditions as well as masticatory bite forces to have a clearer picture of the muscular mechanics of TMJ IC compare to controls.

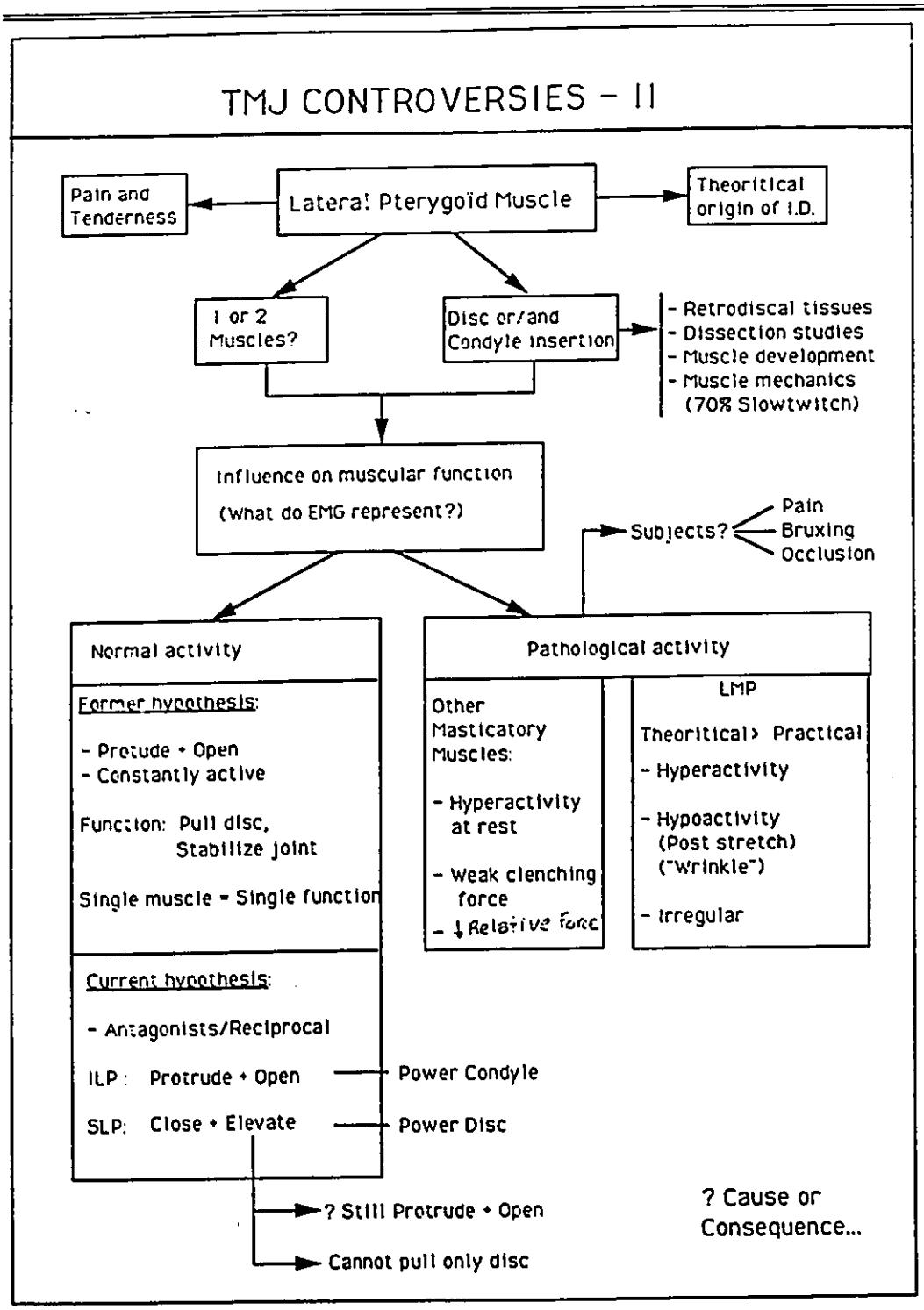

Figure 3: Diagramm of TMJ controversies, part II;

Illustrating a summary of the present knowledge in the world of TMJ pathology.

One difficulty encountered when evaluating factors involved in TMJD is to analyze and quantify the behaviour of the masticatory muscles. EMG represents the sensitive research tool for measuring muscle function and has been widely used since it was first introduced in dental research by Moyers (1950). The uses of EMG have thus substantially increased our knowledge of the function and dysfunction of the masticatory system. Probably because of the difficulty to record and insert in the SLP, the majority of the studies have used surface EMG on the masseter and temporalis muscles without any regard to muscle effects on the pathogenesis of TMJ ID.

Statement of the Problem: Purpose

The purpose of this study is to investigate and analyze different static and dynamic EMG activities of the two heads of the LPM's, the masseter and temporalis muscles in subjects with and without TMJ ID.

Concurrently, the present investigation discusses the role of the LPM's and its relationships to the deranged disc-condyle functional unit providing us with a clinical and etiologic understanding of the muscle mechanics of the two heads of the LPM's in TMJ ID.

Hypothesis

It is hypothesized that during static and dynamic tasks, the integrated and normalized linear envelope EMG (ILEEMG) signal of the SLP and the ILP will be significantly different in the two groups: the TMJ ID and control group. Hyperactivity or hypoactivity of the pathological muscles otherwise uncoordinated, altered and irregular myoelectric activity are anticipated to be associated with ID. In addition, for all the conditions, we expect uncoordination and qualitative differences in the EMG recordings between the two groups.

For the absolute isometric forces values, according to previous studies and discussions, it is hypothesized that the TMJ ID group will exhibit significantly different values than the control group. The pathological subjects are expected to exhibit lower bite and opening forces.

Rationale

The relevance of this study is to provide the clinicians and the researchers with a sound understanding of the muscle mechanics of the two portions of the LPM in relation to the deranged disk-condyle functional unit. This will allow for a better comprehension of the etiologic process as well as the possible consequences of the disorder and will enable effective management of the patients suffering from TMJ syndromes.

TMJ specialists will gain valuable information regarding the ID pathogenesis and its close relationship with the SLP and ILP muscles. It concerns dentists, orthodontists, maxillo-facial surgeons, physiotherapist as well as other therapeutic professionals dealing with TMJ disorders. Moreover, this investigation should lead professionals to consider the TMJ syndrome with a multidisciplinary collaboration to improve the quality of life of the increasing number of mistreated patients.

Lastly, this literature contribution should guide future research projects in the TMJ field and lead to improved methodological techniques.

Justification

EMG studies have been applied mainly to the masseter and anterior temporal muscles, probably because of their presumed importance, their large size and their accessibility by surface electrodes. It is well known that these muscles have a less significant impact on the displacement of the disc due to their absence of anatomic linkage to the mandibular condyle and intra-articular disc itself.

Because of a lack of anatomic certainty regarding the LPM, EMG recordings should be reinterpreted or simply recollected and properly analyzed in the light of current knowledge.

The majority of EMG studies on the masticatory system have aimed at normal function of muscles of undefined normal subjects. TMJ dysfunction subject criteria were generally subjective, based on occlusal patterns, or not described at all. An effort should be made regarding sample choices as we are dealing with specific biomechanical joint disorders.

Furthermore, previous studies have failed to consider the subject's head position, which has been proven to cause hyperactivity of the muscles of mastication (Robinson, 1964; Hairston *et al.*, 1983; Winnberg & Pancherz, 1983; Frosberg *et al.*, 1985; Boyd *et al.*, 1987; Darlow *et al.*, 1987). Forward head posture has seldom been rigidly controlled in EMG studies of masticatory muscles. This could explain the presence of hyperactivity in the majority of TMJ muscular studies. Therefore, the testing position of the head is of prime importance for TMJ disorders patients in EMG studies.

Limitations and Assumptions of the study

Data were collected for this project in a laboratory environment, for instrumentation convenience which inevitably induced some limitations. The principal limitations within which this investigation was conducted can be summarized as follows:

1) For practical and accessibility reasons, the experimental sample was selected from a population of TMJ ID syndrome patients of an Ottawa physiotherapy clinic positively diagnosed by dental, medical and physiotherapy professionals. Only those subjects who conformed with specific criteria entering ID category were selected.

2) The presence of reciprocal clicking is imperative for the subject to be considered as having an anterior disc displacement. This assumption is made without the help of radiography, arthrography, CT scan or magnetic resonance imaging (MRI) as this concept is now widely adopted by researchers and clinicians (Ireland, 1951; Farrar, 1972; Weinberg, 1980; Dolwick, 1983; Rocabado, 1983; Isberg, 1985; Owen, 1987; Gage, 1989; Bell, 1990). Other objective signs of ADD include possible limitation and ipsilateral deviation of the opening in addition to limited contralateral gliding and protrusion.

3) The observations were gathered in laboratory settings and the tasks were done on purpose, not as part of daily living activities. In addition, the implanted electrodes could alter the path of the specific tasks due to physical disturbance or secondary discomfort. These factors can modify the patient EMG recordings by causing stress/discomfort making it difficult to relax.

4) The EMG technique implies minor limitations. In our favour, intramuscular electrodes decreases the crosstalk effect observed with surface electrodes and is highly selective of the motor units in which it is implanted. The signal of a few fibers is representative of the whole muscle (Perry &Bekey, 1981). The electrodes, with movements of the muscle fibres, are likely to record some signal discrepancies. Lastly, the position of the electrode itself and the position of the joint increases the variability of the EMG signals (Perry & Bekey, 1981).

5) The insertion of the SLP may accidentally occur in the ILP muscle because of its larger size, but hardly in the tendon of the temporal which is crossing the ILP or even in the medial pterygoid muscle underlying the SLP. In order to decrease the effect of these technical difficulties, preliminary experiments were done on fresh cadavers and insertion verifications with the oscilloscope were done

according to Mahan *et al.* (1983) and Gibbs & Mahan (1984). This procedure was more applicable for the healthy muscles as the TMJ ID ones could exhibit variations in the EMG signal. Therefore, we relied on the inserter expertise and feeling and the signal for precise insertion.

6) It is important to keep in mind that EMG measures the level of muscular activity and cannot be used as a direct measurement of force. There is a linear relationship between an appropriately quantified EMG measure and isometric force for the intramuscular electrode configuration (Perry & Bekey, 1981). However, the occurrence of myoelectric signals demonstrates whether the muscle is active or not and how active it is compared to a reference value (i.e. MVC).

7) In our inferential statistics (ANOVA), we assumed homogeneity of variance, population normality and independent measures. Test for homogeneity of variance and of homogeneity of correlation were done if needed. The practice effect due the number of trials and conditions was also taken in account. The power of the statistical test were readjusted in case of violation of any assumptions.

Consequently, the limitations of the study will restrain the inference of the results. Moreover, the conclusions drawn from this investigation are limited to the different techniques and instrumentation used in the process of data collection and analysis.

Definitions

In order to understand the glossary of the Temporomandibular Joint, knowledge of the current specific vocabulary and meaning is mandatory. These definitions and explanations are as follow:

TMJ (TemporoMandibular Joint): Class three lever articulation between the mandible and the cranium (Bourbon, 1988). It is described as a compound synovial joint with an intra-articular disc which divides the cavity into two distinct units: the upper joint remains a freely movable sliding joint (arthrodial) while the lower is converted into a pure hinge joint. Can also be termed as a "socket joint with a movable hinge joint" (Bell; 1983). The TMJ is described as bicondylar or ellipsoid in type and involves the articular tubercle and the anterior portion of the mandibular fossa of the temporal bone above and the condyle of the mandible below (Gray's Anatomy, 1987). This term also refers as the cranio-mandibular complex or cranio-mandibular joint.

Dysfunction: disturbed, impaired or incomplete function (Butterworths Medical Dictionary: 1980). Synonyms of malfunction and parafunction.

TMJD (TemporoMandibular Joint Dysfunction): Temporomandibular joint and muscle dysfunction implying that the disorder takes place either in the muscles of mastication, the joints or both as long as there is a functional disorder. The condition includes the following clinical signs: pain, muscle and joint tenderness, joint sounds during condylar movements, limitation and/or uncoordinated mandibular movements. It differs from other terms in its diagnostic sense and do not need the presence of subjective symptoms. Also synonymous to TM syndrome or TMJ syndrome or disorder.

Fascia: refers as a soft tissue called fascia which is an thin fibrous envelope of an organ, a muscle or an area (Medical Dictionary, Flammarion; 1982).

Myofascial-pain dysfunction syndrome (MPD) : This expression encompasses a variety of problems which include the entire scope of TMJ disorders, whether intra-articular or extra-articular (Mikhail, 1980). Laskin (1969) considered the pain dysfunction syndrome as essentially a functional psychophysiologic disease with organic changes that may later be noted on the teeth and joints as secondary rather than primary phenomena. Truta (1989) defined MPDS as a chronic soft tissue fascial pain which refers to a masticatory fibromyalgia condition. Weinberg (1990) specified that MPD syndrome specifically eliminates joint involvement, and should be limited to early simple muscular pain disorders. It is not synonymous with craniomandibular pain or TMJ dysfunction which includes disturbed function. According to Travell and Simons (1983), this term is widely used by the dental profession to identify facial pain with a muscular component. The syndrome is characterized by preauricular pain; muscle tenderness; joint clicking and movement dysfunction.

Craniomandibular and TemporoMandibular (Joint) disorders are more global diagnostic terms that include the whole variety of syndromes of the TMJ either TMJD or MPDS. Lately, clinicians and authors tend to categorise the whole scope of TMJ disorders into two etiologic and clinical patterns: Arthrogenous and/or Myogenous TMJ disorders. The American Academy of Craniomandibular Disorders defines craniomandibular disorders as a term embracing a number a clinical problems that involves the masticatory musculature, the TMJ or both. It is synonymous to TM disorders. CMD have been identified as a major cause of non-dental pain in the orofacial region and are considered to be a subclassification of musculoskeletal disorders (Bell, 1990). CMD's are a cluster of related disorders in the masticatory system that have many features in common. In 1985, Foreman, stated: "the terms

TMJD and MPD have been used synonymously by various authors, and this can be confusing. To add to this semantic problem other terms which have been used include TMJ syndrome, craniomandibular syndrome and more rarely TMJ arthritis or arthroses, myofasciitis, myalgia, fibrositis and Costen's syndrome. All refer to a condition which involves the TMJ and surrounding structures. They are obviously unresolved problems of semantics concerning conditions affecting the TMJ area and it is to be hoped that an internationally acceptable terminology can be agreed upon before long."

ID (internal derangement): An intra-articular disc-condyle discoordination that involves an abnormal relationship between the articular disc and the mandibular condyle, fossa and articular eminence without regard to condylar position. The disc is, in fact, displaced anteromedially (Benson, 1988). More specifically, we are dealing with an arthrogenous diagnostic with or without soft tissues involvement: clicking, local joint pain and tenderness, discal displacement and motion dysfunction.

ADD (anterior disc displacement): Relates to the majority of ID involving an abnormal disc displacement in the anterior joint space. The term "displacement" is used to describe disc position by opposition to dislocation which should be reserved for condylar position relative to joint structures. ADD can be viewed as a natural progression of events that occurs as a result of changes in the integrity of the disc-condyle assembly (Benson, 1988). The disc is displaced anteriorly and medially to a certain extend.

LPM (lateral pterygoid muscle) : One of the four masticatory muscles that lies deep to, and largely behind, the zygomatic arch. It is a short, thick muscle, somewhat conical in shape, that extends almost horizontally between the infratemporal fossa and the condyle of the mandible (Bourbon, 1988). The anatomy and functions of its two portions are discussed in chapter II, literature review.

SLP (superior lateral pterygoid): Refers to the small upper head of the LPM. The fibres originate from the infra-temporal surface of the greater wing of the sphenoid bone, run backward and outward to insert onto the articular disc and the condylar head (Bell, 1983; Bourbon, 1988). Some authorities (Honee, 1972) refute the condyle connection and others refute the discal attachments (Carpentier, 1988).

ILP (inferior lateral pterygoid): Refers to the inferior head of the LPM. This portion is three times larger than its counterpart, the SLP, and arises from the lateral lip of the lateral pterygoid plate. It then converges upward and outward to reunite with the SLP and insert onto the neck of the condyle.

CHAPTER II

LITERATURE REVIEW

The beginning of this chapter is a review and a criticism of the recent literature on TMJ disorders etiology which leads towards the pertinence of the present study wether the cause of ID is passive or non-muscular or active secondary to muscular dysfunction. Internal Derangement (ID) and Anterior Disc Displacement (ADD) of the TMJ are also discussed in order to understand the facts and controversies of the physiology, biomechanics and muscular characteristics of the disorders. Furthermore, we will look at the different aspects regarding the LPMs and other masticatory muscles discussed in parallel with static and dynamic EMG findings of normal and pathological functions. Finally, as an overview and a conclusion, the inter-relationship between the pathological function of the LPM and the pathogenesis of ID of the TMJ will be address.

I-TEMPOROMANDIBULAR JOINT DYSFUNCTION

Epidemiological studies reported the prevalence of clicking and TMJ related muscular symptom in the general population to range from forty-one to seventy-nine percent (Clarke, 1982; Greene, 1982; Foreman, 1985; Magnusson, 1986). Sixty-five to eighty-five percent are female and fall into the age group of 20 to 40 years old. Median age for men is 27 years and for women 33 years; middle and upper social classes are clearly over-represented (Heloe & Heloe, 1975). As a result of this high incidence, more health professionals have specialised and have developed treatment plan for the clicking, painful and stiff jaw joints. For many clinical and scientific journals, it has become a fashionable disease.

The first section covers the conceptual ideas suggested to explain the cause of TMJ dysfunction and their controversies. The pivotal point being the muscular origin is of major important, especially as this investigation aims in providing better knowledge on the muscular aspect of this pathology. Thereafter, the rationale of this study derives. A brief outline on how the lateral pterygoid muscle is believed to be responsible for ID serves as the key issue in this review.

Etiology of TMJ Disorders

The first major concepts that provide the rationale for this study is the proposed and controversial explanation of the origins of TMJ disorders. In a larger sense, researches proposed different factors in cause as well as a multifactorial origin. Unfortunately, the issue is still disputable. One of the major involvement of that confusion is, of course, the inability to cure these patients.

The issue of treating effectively TMJ disorders have motivated researchers, for almost a century, to scrutinize the cause of mandibular imbalance. Back in 1918, Prentiss recognized that the loss of occlusal vertical dimension, mainly due to missing posterior teeth, resulted in TMJ dysfunction, thus, introducing the mechanical displacement theory in opposition to the psychological theory. A few years later, Goodfriend and Costen as reported by Mikhail (1980) suggested that overclosure and posterior displacement of the condyle were part of the degenerative process of the TMJ surfaces and of the compression of hearing structures. Though, occlusal abnormalities were considered to be prime etiological factors of the Costen's syndrome leading to virtually an entire sub-speciality of dentistry, that of occlusion (Foreman, 1985) and to a greater extend the tendency to over-investigate, overcomplicate and over-treat the TMJ situation.

However, Costen's syndrome was not accepted unanimously; pain was also proposed to arise from muscles spasm secondary to malocclusion. In 1937, Schultz introduced the concept of "lax ligaments" realizing the high prevalence of hypermobile joints to explain jaw clicking and dislocation. He emphased treating the joint more than the teeth contacts, believing it was only secondary to the joint problem. In 1969, Laskin, the founder of the myofascial-pain-dysfunction syndrome (MPDS) revised the whole concept of the origin and stressed the role played by muscles. He considered the condition as a functional physiologic disease noted in the joints, teeth as a secondary phenomena to muscle overexertion. Basically, spasm is a physiologic tension-relieving mechanism produced by oral parafunctional habits. According to Laskin, this psycho-physiologic condition is self-perpetuating. Gradually the muscle origin was brought up and Schwartz suggested the psycho-physiological predisposition of spasm (Mikhail, 1980).

Therefore current views on etiologies dispute two leading school of thought: Inadequate and unsatisfactory occlusion are the most frequent cause of TMJ syndrome; and TMJ disorders are usually related to dysfunction of the masticatory muscles caused by muscular parafunction and/or emotional stress. Parafunctional activities can be defined as masticatory activities other than chewing, swallowing and speaking such as pencil chewing, nail biting, playing a musical instrument, clenching and grinding. They produce much greater forces over a longer period of time thereby damaging the joint components (Okeson, 1981).

It is important to mention that traumatic events cannot be forgotten as to acutely and chronically causing TMJ dysfunction, such as whiplash, blow to the jaw, viola playing and motion style (Bryant, 1989). Gross bony architectural malformation of the mandible is, of course, another origin of TMJ disorders. In addition, trauma was found to induced TMJ disorders primarily or secondary, due to muscle hyperfunction (Laskin, 1969; Pullinger *et al.*, 1985; Ash, 1986).

Knowing that the etiology of TMJ dysfunction is multiple, the factors have been divided into predisposing, precipitating and perpetuating ones. Predisposing ones are the anatomical and physiological ones including size and shape of the different tissues of the masticatory system as well as the state of the neurological, the vascular, the nutritional and the metabolic systems. In addition, pathological and behavioral factors fall into this first category. Precipitating factors include trauma and stress. Perpetuating basically means the vicious circle of stress, spasms and pain (Figure 4).

- The Occlusion Concept

Even if clinical experiences testified the importance of occlusal disharmony, in TMJ disorders, no consensus has been developed. Bell (1990) suggested that in the absence of recent changes or activation, evidence of occlusal disharmony in acute muscle disorders may be disregarded etiologically. Newton (1969) reported no correlation between occlusal irregularity and the incidence of joint symptoms by statistical comparison. Clarke (1982) used biological, clinical and therapeutic observations to show that occlusal factors are not involved in the etiology of bruxism or TMJ dysfunction. He even stated that the success of occlusal therapy may be due to the placebo effect. Moreover, the school of occlusal disharmony cannot explain why muscular pain is so uncommon comparatively to malocclusion in the general population.

It is unanimous that some degree of occlusal discrepancy exist in nearly every TMJ patient (Wassell, 1989). Consequently, several authors believed in the occlusion theory as the most frequent cause of TMJ disorders (Solberg, 1972). However, ninety percent of the population demonstrated some occlusal inequality. Relaxation, physical therapy, and medication have had equal success and failure (Wassell, 1989). The fact that occlusal factors causes TMJ dysfunction is therefore questionable.

Indisputably, occlusal harmony is essential to normal masticatory function. Bell (1990) stated that malocclusion may result from spasticity of the ILP or from occlusion interferences of the teeth and is potentially damaging the disc and the joint surfaces. The causal role of occlusal interference is important but serves primarily as a predisposing factor that requires activating before it becomes significant. Emotional tension and bruxism are the important activating factors (Bell, 1990).

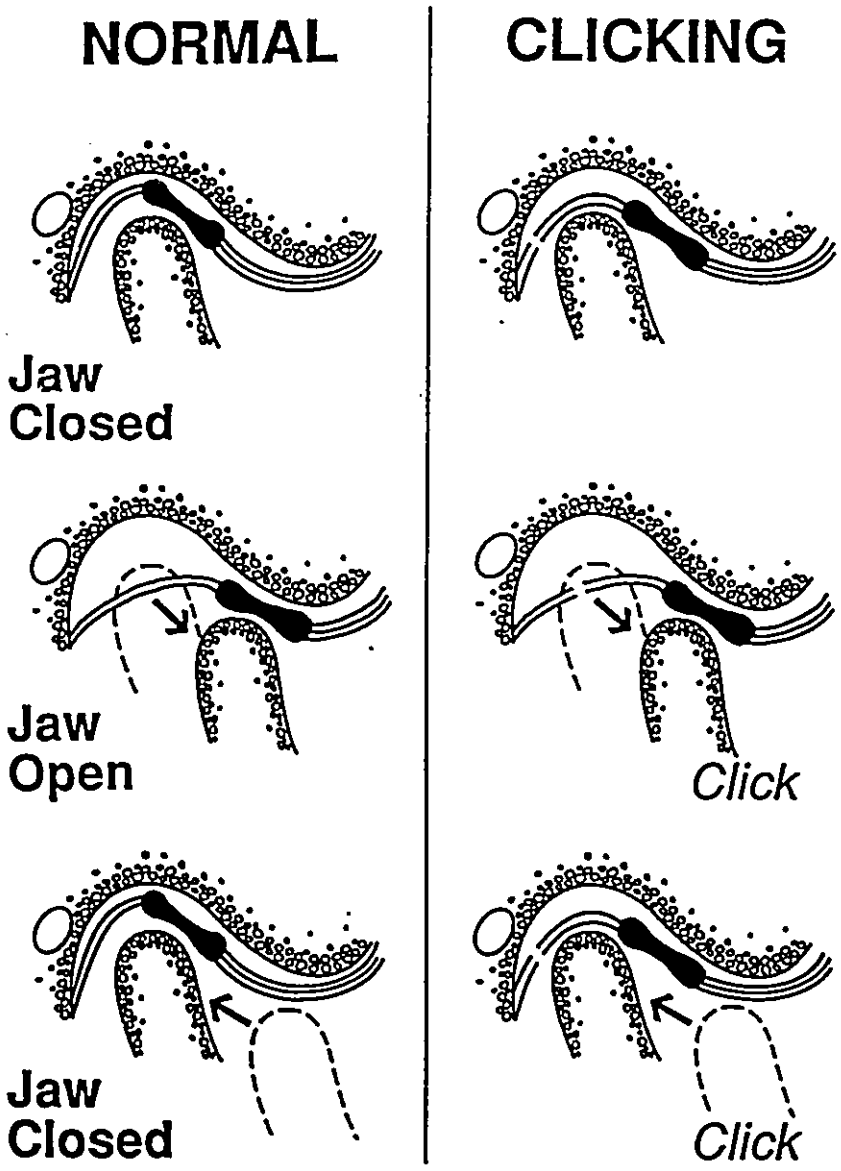

Figure 4 : TMJ views showing intra-articular normal and ID manifestations

Note: From "Its all in your head" (p.6) by A. H. Owen. 1988, Austin, Texas USA

- The Psychological Concept

There is convincing findings to support the psychophysiologic theory. Hall (1984) reported that 80% of TMJ patients had associated psychophysiologic disease. Emotional and stress factors were first considered alone, then, combined with malocclusion and later on as a consequence and a cause of occlusal pathosis. EMG studies showed that stress and environment induce muscle spasm and hyperactivity (Schwartz 1956; Gervais, 1989). Thompson (1969) explained physiologically how the supraspinal control and the gamma system can modify motorneuron activity. He suggested a secondary contribution from the natural unaltered dentition. Therefore a stressful life situation will definitely manifest in the bodily expression by increasing the tone in the mandibular elevator muscles.

Investigations of TMJ ID patients have shown that they are tense, anxious people who are failing to cope adequately with stress (Clarke, 1982). Clarke (1982) hypothesized that during periods of stress two unfavourable factors occur: raised muscle tone within all voluntary muscles, and severe bruxing activity during sleep. Therefore TMJ problems could simply be a difficulty to adapt to changes, to parafunction or an inability to handle stress. (Mikhail & Rosen, 1980; Clarke, 1982).

- The Neuromuscular Concept

A large number of authors believe that masticatory muscles hyperactivity is the primary factor responsible for TMJ dysfunction. The muscle ideology suggests that hyperactivity and/or parafunctional habits (ex. bruxism) of the muscles of the head neck and jaw is the primary cause of TMJD leading to pain and spasm which in turn produces excessive contraction of the muscles establishing a vicious circle (Clarke, 1982; Mikhail & Rosen, 1980; Guttu & Speaktor, 1981; Okeson, 1981; Travell, 1983; Bell, 1990). The muscle concept also includes secondary muscle spasm and contracture due to mechanical, emotional or metabolic stimulation (Travell, 1983).

The hypothesis was first viewed as a stress releasing mechanism by Laskin (1969). This conception has the advantage to explain pain, deviation and limitations regardless of the occlusal contact. Conversely, Toller (1976) and Foreman (1985), showed that even with a higher prevalence of nocturnal bruxism in patients with TMJDS the significance of the relationship between bruxism and TMJD is not conclusive. Many authors reported by Mikhail & Rosen (1980) suggested that the type of occlusion and missing teeth do not contribute to TMJ dysfunction. They believed in a neuromuscular origin. Many authorities believed that muscle spasm and parafunctional activities can be responsible for malocclusion. Therefore it should be regarded as symptomatic rather than etiologic. (Laskin, 1969; Nanthviroj, 1976; Okeson, 1981; Clarke, 1982; Isberg, 1985; Juniper, 1987; Bell, 1990).

Nanthviroj, 1976; Okeson, 1981; Clarke, 1982; Isberg, 1985; Juniper, 1987; Bell, 1990).

It is generally accepted that the LPM is most often involved in the pain-dysfunction syndrome (Guttu & Spektor, 1981). Spasticity, specifically of the LPM causes firm bracing of the condyle against the articular eminence; causing the clicking and grinding sound disturbances. (Travell, 1983). Therefore, the neuromuscular concept is a good compromise between the occlusal and the muscle explanations.

- The Posture Concept

Many researchers (Robinson, 1964; Preiskel, 1965; Lean and Brenamn, 1972) had found how body posture affects the position of the mandible, the occlusion pattern and masticatory muscular activity using the techniques of EMG, occlusal gauge, casting wax and clinical judgement. Since then, numerous studies (Mohl, 1976: Darnell, 1983; Rocabado, 1983; Krauss & Darling, 1984; Araki & Araki, 1987) have shown that movements or postures of the head influence and the rest position of the mandible. Several studies reported EMG hyperactivity of the mandibular postural muscles with extension of the craniocervical spine and forward head posture (FHP)(Darnell, 1983; Rocabado, 1983; Boyd, 1987; Krauss, 1988; Parker, 1990). Furthermore, clinical and scientific belief that one of the basic predisposing factor of TMJ dysfunction syndrome is forward head posture (FHP) and its biomechanical effects of the head-neck-jaw complex.

The condition of the head and neck muscles can thus influence the posture and function of the mandible (Darnell, 1983; Rocabado, 1983; Krauss, 1988; Shiau, 1990). Body posture also has its effect: as its recline, the ILP muscles draw the mandible forward to safeguard the airway (Bell, 1990). Foreman (1985), stated that the mechanism by which this painful and debilitating condition arises is a result of the adverse physical posture and emotional stresses of daily living. The individual responds with head and neck stressed and fatigue muscles and other soft tissues.

Presently, the parafunction is well recognized and could result from environmental tension, occlusal fault, proprioceptive disturbance and may lead toward muscular hyperactivity and joint dysfunction. In turn these can be affected by stress and posture. The lack of standards for comparison of various treatment techniques causes a difficulty to assess their effectiveness, and consequently the factors in cause. The possibility that the etiology is multifactorial seems to be on top of the list presently, the muscular component being always present (Laskin, 1969; Weinberg, 1980; Vanderas, 1988). The two major causes of TMJ disorders are occlusal disharmonies and psychologic stress factors, both theories sharing a common outcome: myospasm leads to pain and dysfunction (Eversole & Machado, 1985).

- Internal Derangement (ID)

Internal derangement (ID), the most common TMJ disorder (25 to 80%), is an intra-articular disc displacement (Farrar & MacCarty, 1979; Rocabado, 1981; Dolwick, 1983; Owen, 1987; Benson, 1988; Gage, 1989). The relevance of this understanding lies within the fact that the disc is anteriorly displaced as a possible consequence of the contraction of the SLP. This study aims, amongst other objectives, towards refuting or supporting this hypothesis.

Joint sounds are the most common symptom of TMJ dysfunction with at least a third of the whole population displaying clicking, popping or crepitus (Agerber, 1985). Clicking in the TMJ the first and principal indication of altered function inside the joint (Gage, 1989). ID is defined in various ways and it basically involves a disc interference disorders usually non-inflammatory in which the articular disc is chiefly responsible for the symptoms whatever the cause (Bell, 1990). It is the second most common problem after muscle disorders and includes the noisy, clicking, popping, locking joints with or without pain demonstrating altered movements such as limitations or deviations (Bell, 1990).

Dolwick (1983) defined ID of the TMJ as an abnormal relationship of the articular disc to the mandibular condyle, fossa and articular eminence. Usually the disc is displaced anteriorly and there is a frequently associated medial displacement and an elongation of the posteriorly attached tissues. It must be realized that ID may occur together with muscle hyperactivity disorders (Dolwick, 1983; Bell, 1990). It is generally agreed that the most common manifestation of ID is characterized by an abnormal anterior disc displacement (ADD) and an associated condyle retrusion (Weinberg, 1979) in the closed jaw position (Rocabado, 1983; Gage, 1989). Reciprocal clicking or locking are the diagnostic presentation of temporary and permanent ADD. This mechanism has been described by Farrar (1972), by Farrar & Macarty (1979), Weinberg (1979, 1980), Rocabado (1981, 1983), Dolwick (1983) and Gage (1989).

Over the years, the classification of disc-condyle derangement has been divided either into one syndrome or as a whole progressive disc displacement with various manifestations. As early as 1951, Ireland described a progression of disease from reciprocal clicking (displacement with reduction) to clicking with intermittent limited opening (locking), to permanent limited opening (closed-lock, displacement without reduction), and finally to degenerative joint disease with crepitus and osteoarthritis. This proposed classification is still the best one and the most used nowadays (Isberg, 1985; Hellsing, 1985), with the addition of a myogenic state, a muscle-pain-dysfunction disorder with soft tissue tear, as the most minor and beginning disorder (Eversole & Machado, 1985). All the common TMJ disorders are now interpreted as a single pathological entity (Farrar, 1972; Rocabado, 1981; Dolwick, 1983; Ogus, 1987; Benson, 1988).

- Causes and Pathogenesis of ID

Many causes of ADD have been reported. They are controversial and still at the centre of major debates regarding treatment of disc-condyle derangements.

According to a number of authors (Rocabado, 1981; Dolwick, 1983; Ogus, 1987; Benson, 1988; Gage, 1989; Bell, 1990), ADD usually results from either chronic repetitive jaw microtrauma or acute macrotrauma that would force the mandible postero-superiorly or a combination of both. The overuse microtraumatic syndrome extended over a prolonged period of time would result from loss of posterior teeth, excessive para-functional habits (grinding, clenching, excessive biting force, repetitive overloading, yawning, bruxism), occlusal interference, incisal contacts, deep overbite, forward head posture (Ireland, 1951; Farrar, 1972; Rocabado, 1981, 1983; Isberg *et al.*, 1985; Thompson, 1986; Ogus, 1987; Owen, 1987; Gage, 1989, Weinberg, 1990; Bell, 1990). This continual overloading of the joint has been advocated to drive the mandibular condyle to overseat posteriorly. On the other hand, macrotrauma includes external forces or sudden stresses of the postero-superior mandible, stretching the post attachments or collateral ligaments. It can be a consequence of whiplash, a blow to the jaw, a prolonged dental procedure (difficult extraction), forced mouth opening during general anaesthesia (intubation) etc.(Rocabado, 1981, 1983; Ogus, 1987; Benson, 1988; Gage, 1989). Hargreaves (1986), suggest other causes of TMJ syndrome: hesitation in movement of the disc, functional incoordination of muscles.

In both situations, the lateral and posterior connective tissue attachments elongate abnormally and the disc is displaced anteriorly. This will induce further migration and loosening of the disc leading to clicking on movements, leading to a vicious circle. As discussed earlier in TMJ dysfunction etiology, malocclusion and altered muscle activity has been suggested as a cause of ADD.

Muscle hyperactivity and occlusal disharmony have been proposed as primary and secondary causes of ADD. It was also mentioned that stress induces neuromuscular overload and eventually increased muscle contraction. Confusion still remains.

It is also suggested that the TMJ disorders results from one of several pathological states: 1) a loss of synovial fluid viscosity due to an increased shear rate from muscular overloading, increased congruity of the surfaces and sticking movement causing the clicking sound (Ogus, 1987; Gage, 1989); 2)ligament laxity or a detached lateral ligament. The condylar head would continue to move forward without the restrain of normal ligaments slides over the anterior band and produces a click (Rocabado, 1981; Ogus, 1987; Gage, 1989). Once the collateral ligament has become elongated, the disc can be pulled forward and anteriorly by the contraction of the SLP (Benson, 1988). Obviously, the first two hypotheses imply a malfunction of the masticatory muscles, more precisely a hyperfunction

of the lateral pterygoid muscle. 3) Thinned posterior band of the disc by repeated condylar compression (overloading) leading to a loosening of the retrodiscal attachments predisposing the condyle to slide behind the disc, thereby forcing the disc anteriorly, ID and hypermobile TMJ develops (Farrar, 1972; Rocabado, 1981, 1983; Hellsing, 1985, Isberg, 1985; Ogus, 1987; Benson, 1988; Weinberg, 1990). This condition is aggravated by a muscle imbalance (Rocabado, 1981).

While the above findings suggest that the condyle is forced posteriorly at the initial stages of ADD, it is also quite feasible that the disc slips forward first, thereby allowing the condyle to overseat posteriorly. It appears that either situation occurs at first (Owen, 1987), possibly allowing for an anterior pull of the disc by the LPM. It is still a matter of conjecture and it points to the complexity of TMD and the need for further understanding of the etiology and pathogenesis.

Sound Disorders in TMJ ID

Joint noise, the most common sign of TMJ ID, is found in approximately 85% of TMJ dysfunction patients, whereas epidemiologic studies report an incidence of 14 69% in the normal population (Weinberg, 1980; Owen, 1987; Rinchuse, 1990, Agerberg, 1985). The significance of a click in the TMJ is not fully understood, nor its presence or absence is an indicative of joint pathology. A correlation has been established between the dysfunction of the SLP or both head of the LPM and TMJ clicking in mandibular disorders.

- The Significance of TMJ Clicking

The clinical significance of TMJ sounds depends on its association with other signs and symptoms. Alone, it does not necessarily indicate a need for treatment (Rinchuse et al., 1990). For example, it is associated with deviation of the midline incisal path and not with a deflection towards the stiff side during opening. Nevertheless, reciprocal clicking was found a significant clinical sign of an ADD with reduction (Hellsing & Holmlund, 1985). Numerous other studies considered patients with reciprocal clicking as suffering from ADD (Ireland, 1951; Farrar, 1972, Weinberg, 1980; Dolwick, 1983; Rocabado, 1983; Isberg, 1985; Owen, 1987; Gage, 1989; Bell, 1990). The natural progression or pathogenesis of ID begins with a simple clicking due to ADD, followed by a locking condition degenerating to crepitus osteoarthritic noises (Dolwick, 1983). In addition, the concept of ADD has been widely supported by anatomic findings, condylar path studies as well as radiographic, arthrographic and surgical findings (Dolwick, 1983; Owen, 1987). The relation between TMJ noise and pathology is undeniable but the etiology remains unclear.

- The Physiologic meanings of TMJ Clicking

Joint clicking is recognized to be caused by the reduction of disc derangement. As the disc is displaced anteromedially, a sound is heard during opening, anterior translation and contralateral movements as the condyle reduces under the posterior band of the disc. A sound is also heard as the condyle dislocates itself on the way back to its resting position (Isberg, 1985).

Other causes postulated are deviations in the form of the articular surfaces, condylar subluxation, loose bodies, posterior position of the condyle, folding or wrinkling of the disc, anterior hypermobility of the condyle, adhesions within the joint space, roughness of the joint surfaces, trauma to the jaw, a vacuum, an obstruction under pressure, the condyle hitting the temporal bone at maximum opening or hitting a bulge in the condylar path and prolonged opening during dental procedure (Isberg et al., 1981; Farrar, 1983; Dolwick, 1983; Sigaroudi & Knap, 1983; Bell, 1990; Rinchuse et al., 1990; Weinberg, 1990). In most of the above, joint clicking is an indication of a lack of coordination between the condyle and the disc and is still attributed to ID in the dynamic state. The exact origin of the click is still not fully explained. They might even be considered within the range of the normal variation (Rinchuse et al., 1990).

In the literature, different types of TMJ noises are described. These can be grouped into 1) reciprocal clicking, popping or soft clicking; 2) snapping or wide opening clicking; and 3) crepitus or grating (Weinberg, 1980, Rinchuse *et al* 1990). A closed lock is silent, it is therefore not considered a pathological sound. This condition eventually evolves into crepitus or resolves back into reciprocal clicking with intermittent locking.

- The LPM as a Cause of TMJ Clicking

It has been speculated that clicking is caused by ADD as a consequence to a damaged posterior capsular element and a hyperfunction of the ipsilateral SLP (Vincent & Lilly; 1988). Or clicking might be caused by an inadequate pull of the LPM which creates an unstable disc prone to become displaced and heavily loaded (Berry & Watkinson, 1978). Sigaroudi & Knap (1983) proposed an incoordination of the LPM and temporalis muscles. However, whether or not the click is caused by the weakening of the attachment of the disc and / or uncoordinated function of the SLP and ILP has not been demonstrated but just stated theoretically. For others (Hellsing & Holmlund, 1985; Bell, 1990), the process is more active: the SLP contract strongly and dislocate the disc anteriorly during power strokes and maximum intercuspation.

On the other hand, it was postulated that SLP dysfunction is the result of the clicking disc

interference (Juniper, 1984, 1987; Mahan, 1983; Wilkinson, 1988). Zijun (1989) found abnormal LPM EMG activity in normal arthrograms refuting the above opinions. He proposed a hyperfunction of both heads instead of the SLP alone as the ILP tries to over-control the pathological situation.

Many investigators did not record the sounds for the purpose of establishing a difference in EMG before and after the click. As the SLP is supposedly inserted on the disc, variations before and after the disc reduction are expected. However, Isberg et al. (1985) reported EMG activity in the elevators before the condyle slid over the posterior thick band of the disc (opening click). This phenomenon could be interpreted as an arthrokinematic reflex caused by distraction. Continuous muscle activity could be provoked by TMJ disc displacement and ceased when the disc position is normalized during mouth opening only to occur again every time the disc became displaced on mouth closure. Non-functional EMG hyper-activity was seen when a closed lock occurred in the ipsilateral and contralateral elevators inhibiting the condylar movement necessary to achieve reduction. Vitti and Basmajian (1977) showed that slow opening and closing movements could be performed without any notable muscle activity in the temporalis muscles.

These studies demonstrate the importance of examining the role of the LPM in synchronisation with the articular click as there are definite grounds to believe that the latter is related to ADD pathogenesis.

- Methodology of Sound Recordings

Most investigators studying patients with TMJ click patients did not use appropriate means to register and relate noises with other parameters. Clinical palpation, the use of a stethoscope and condylar path tracing can merely provide a classification of types of patients. Joint sound has not been regarded as a separate entity with respect to synchronisation of EMG or movement.

Different simple methods have been previously used in registering jaw clicking as an important parameter. Some investigators determined the position of the click in time solely by manual palpation (Sigouradi & Knap, 1983; Hellsing & Holmlund, 1985; Alsawaf, 1989; Zijun, 1989; Weinberg, 1990). Sigaroudi & Knap (1983) determined the positions of all clicks visually looking at the optoelectric tracings. On the other hand, other investigators (Isberg-Holm & Westesson, 1982; Isberg, 1985; Woods & West, 1986; Merlini & Palla, 1988) used a contact microphone fixed to the surface of the temporalis to register the disc sound alone or simultaneously with other parameters such as mandibular movements. Therefore our choice of frequency, filter, amplification, and output voltage were partially based on their techniques and recommendations. A piezo-electric microphone attached to the patient's forehead (LF 16Hz, HF 800 Hz was also used (Isberg et al., 1985).

In summary, the significance of the click remains controversial. It probably implies a pathologic condition such as an internal disc derangement, but may also be normal anatomic behaviour. TMJ studies focused on clicking have been mostly concerned with the movement of the joint components, but none have provided a satisfactory explanation as to its exact relationship with the muscle activity of the disc-condyle power complex.

The literature review of the section leads to the main objective of this investigation which is to assess the EMG activity of the SLP and the ILP in patients with clinical evidence of sound disorders indicating ADD. Furthermore, we will analyze EMG signals in association with the sound of disc incoordination as measured simultaneously by a microphone. We will study the relationship between TMJ sound disorders, patho-biomechanics and muscle activity. This will improve our understanding of the significance of the click in TMJ ID.

II-THE LATERAL PTERYGOID MUSCLES (LPM's) AND OTHER MASTICATORY MUSCLES

The understanding of the present project requires a complete review of all the facts and debate existing over the LPM. The most important aspects to cover will be its anatomic relationship with the intra-articular disc as well as the different concepts on its normal and impaired function on the articular disc. In parallel, reviewing the normal and pathological functions of the masseter, temporalis and other masticatory muscles in relation to static, dynamic and force situation is imperative to appreciate the present study.

The Anatomy of the LPM (Figure 5)

- Facts and Controversies

The LPM is most often quoted as being the source of pain and tenderness in mandibular muscle examinations. Mostly it is accused of being the origin of temporomandibular joint dysfunction (TMJD). As cited by Bell (1990), an overview of the LPM issue: "The lateral pterygoid muscle has been mercilessly incriminated as the cause of numerous temporomandibular complaints. This probably stems from lack of understanding of how the muscle is constructed and what its normal functions are. "

It is also been mentioned that it is a very difficult muscle to assess, palpate and to record by

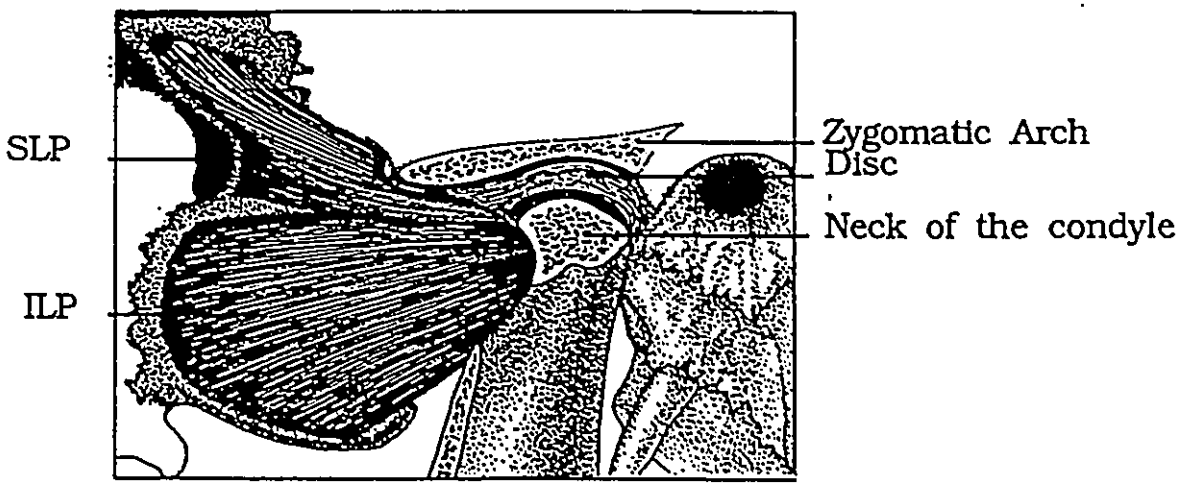

Figure 5 : Anatomy of the Lateral Pterygoid Muscles with their respective attachments. The zygomatic arch and the superficial portion of the condyle have been removed to show attachments of the SLP and ILP to the neck of the condyle, the articular capsule and the intra-articular disc..

Note: From The Trigger Point Manual (p.261) by J.T. Travell and D.G. Simons, 1983. Baltimore: Williams & Wilkins. Copyright 1983 by W & W. Adapted by permission.

EMG. Anatomic descriptions and research have not yet led to a consistent concept over this very peculiar muscle. Is it the power of the intra-articular disc or is it only attached to the condyle? Can it pull the disc forward? What are the joint mechanical implications of its insertions and functions? In order to answer these questions, the first step to consider is the a review of the anatomic structures and relationships of the LPM.

The poor understanding of mandibular basic anatomy can be demonstrated by the disagreement remaining over the nature of the intra-articular fibrocartilage. It is sometimes referred to as a meniscus, but mostly recognized as a disc (Bell, 1990) and even as a "cap", but, to avoid confusion and to follow convention, he used the term meniscus or disc are used.

The human LPM is also a structure with an history of controversies. Over the last 50 years it was conventionally described as being a single functional unit (Grant, 1973; Basmajan, 1970) as well as a double headed muscle (Gardner et al., 1963; Last, 1966; Gross, 1966). In 1973, Grant and Macnamara were the first to separate the LPM in two different entities. Nowadays, results confirm beyond doubt, that it is a short and thick conical muscle that consists of two independent bellies: a flat superior head and a three times larger belly-shaped inferior head (Grant, 1973; Macnamara, 1973; Juniper, 1981, 1984; Carpentier, 1988). Consequently, they should be considered as two distinct functional muscles.

The facts on the fusion of the two bellies and their directions has also been revised considerably. Anatomically, the LPM appears to be separated by a horizontal septum of connective tissue up to 10mm thick surrounded by its own fascia (Juniper, 1981). The two heads have quite different orientations and relationships. Behind the zygomatic arch, the small upper head fibres originate from the upper third of the lateral pterygoid plate, from the infratemporal crest of the greater wing of the sphenoid bone and run almost horizontally backward and outward in close relation to the cranial base. Secondly, the larger inferior head arises from the inferior 2/3 of the lateral surface of the pterygoid plate, the palatine bone and the maxillary tuberosity. These lower fibres converge horizontally upward, outward and backward and inserts on the neck of the condyle anteriorly and medially. According to anatomical observations, the two heads fuse in a single, short tendinous insertion 10 mm in front of the joint, in the medial aspect where they constitute a strong medial wall (Grant, 1973; Juniper, 1981; Burton, 1988; Carpentier, 1988; Hertling, 1990). Grant (1973) described the fibres of the SLP as being mainly red, mostly parallel with small pinnation angles.

The important practical relevance of the LPM incited Johnstone (1980) to determine the feasibility of accurately palpating the LPM in patients after dissecting cadavers and taking lateral radiographs. He emphasized that it is impossible to palpate this muscle directly by clinical techniques without applying pressure through the overlying medial pterygoid and the sensitive temporalis muscle.

- Origin and Insertions of the LPM

Most of all, it is surprising that there is still no agreement with regard to the anterior insertion of the SLP and its relationship with periarticular structures. The question remains as to whether the upper head attaches to the disc only, to the condyle only or to the condyle and disc, and in the affirmative, in which proportions. Some authors report that the SLP inserts only into the capsule and disc (Thilander, 1964; Honee, 1972), whereas the most common view is that it inserts into the condyle, capsule and mainly into the intra-articular disc (Grant, 1973; Macnamara, 1973; Juniper, 1981; Bell, 1983, 1990; Hertling, 1990; Bourbon, 1988). For these reasons, the SLP is sometimes referred to as the sphenomeniscus head (Bell, 1990). On the contrary some authors described an attachment on both structures (Hargreaves, 1986) but mainly on to the condyle (Krauss-Grant, 1988).

A recent dissection investigation evaluating the anatomic relationship of the LPM with the disc-condyle complex using acrylic embedding techniques and serial cuts were conducted by Carpenter in 1988. His study demonstrated that the main insertion of the SLP are not in the disc but into the condyle and that the fibres which seems to go to the disc were all ending on the capsule. This study suggested that the insertion of the SLP is on the neck of the condyle and not the disc itself denying all educational and scientific representations that support the "functional theories". Adding to the confusion, radiographs and textbook illustrations often display a poor impression of the shape and structural relationship of the joint (Rayne, 1987).

Wilkinson (1988) in a cadaver dissection investigation, tried to lighten the issue and assessed the nature of insertion of both heads of the LPM as well as the nature of the anterior surface of the disc in relation to the SLP. He found that in one third of his subjects the SLP had a single insertion on the condyle fovea either directly or by fusing with the ILP, the anterior disc capsule being attached to the roof of the muscle. In the remaining two thirds, the major insertion was identical to the one just described, but the uppermost 20% fibres were implanted in the capsule without any discal connection.

Nevertheless, Bell (1990) believed in a discal insertion of the SLP: "the meniscus is also said to be essential to normal joint functioning because it has its own power system: the superior head of the lateral pterygoid muscle". The disc is described as a jockey's cap, with the border of the cap attachments to the condyle and the peak providing the tendon attachment of the SLP muscle. The disc is firmly attached to the head of the condyle medially and laterally. It is possible for the cartilaginous and inextensible disc to rock backwards and forwards over the condyle between two opposing and balancing mechanical forces: the elastic lower part of the retrodiscal bilaminar zone and the contractile tendon of the SLP muscle (Rayne, 1987; Bell, 1990)

The superior retrodiscal lamina (also called the upper part of the post bilaminar zone) is fibro-

elastic and act as a passive tractive force controlling the disc in a posterior direction only when it is in a stretched position. It is the mechanical counterbalance force of the active SLP muscle that controls the disc in an antero-medial direction (Rayne, 1987; Bell, 1990). This concept is, of course, true only if one accepts the discal insertion of the SLP.

The above controversies are the most relevant anatomic findings leading to the rationale of the present study: is the SLP inserted on the disc or not? What would then be its muscular mechanical function? Lastly, recent anatomical studies describe that the muscle can, in some cases, blend into the orbitalis muscle bridging the intra-orbital groove and fissure explaining retro-orbital pain in TMJ disorders.

- Innervation and Vascularisation

The ILP and SLP are innervated from the same supply: the anterior division of the mandibular branch of the trigeminal nerve (cranial nerve #5); they may have filaments from the buccal and lingual nerves (Travell & Simons, 1983). The trigeminal passes through the oval foramen and has a mandibular division which has one meningeal branch and four masticatory branches. These last ones are the masseteric nerve, the medial pterygoid nerve, the lateral pterygoid nerve (arising from the buccal nerve) and the deep temporal nerve. The arterial supply of the TMJ comes from the external carotid that divides into the maxillary artery for the ILP and SLP, the facial artery for the masseter and the superficial temporal artery for the temporalis.

Consequently, it must be kept in mind that the nerve and blood supply of the four masticatory muscles are all part of one vessel system. Therefore they are all related together for function and pathology.

- Muscle Development

It was interesting and appropriate to cover certain aspects on the development of the TMJ. In the human foetuses at 10 to 20 weeks, of age, the tendon of the LPM passes superiorly to the condylar cartilage and is inserted into Meckel's cartilage posteriorly, thus protruding the primitive jaw. It can therefore be argued that the meniscus was formed from the tendon and this suggestion is supported by the embryological assumption that mesenchyme cells differentiate into fibroblast when subjected to tension and into chondroblast when subjected to compression (Rayne, 1987). It has been shown earlier by the same author (Rayne, 1971) that contractile elements are present in the human

embryo at 6 weeks age and that jaw movements were reported at 9 weeks which must involve the joint between Menckel's cartilage and the chondrocranium. Griffin and Sharpe (1960) indicates that the meniscus is predominantly a posterior extension of the LPM, the medial part of the meniscus is thicker due to the tendon of the LPM being incorporated in its substance (Karger, 1975).

Therefore, if the disc appears to be the continuation of the SLP tendon or the modified tendon of the SLP, the concept of the discal insertion would be confirmed by embryological studies (Juniper, 1984). Another interesting point is that histologically the jaw muscles of the infant are uniquely equip with a very large concentration of muscle spindles, therefore the fine neural proprioception limit the damage and discomfort of force on the joint (Kubota et Masegi, 1977) and allows for massive adaptation.

Newly described muscles inserting on the articular disc have been reported lately. One is called the mandibular capsular muscle (Koritzer) and may provide balance against the action of the SLP or work as a tripodized "suspension system" affecting joint function (Myers, 1988). Another one, the zygomaticodiskal muscle described by Myers (1988), tension of these fibres would move the disc forward. Another muscle called the mandibular discal muscle which would hold the disc anteriorly (Bell,1990). Consequently, these new muscles could affect ID and the LPM pulling onto the disc. Moreover, intramuscular EMG could be registering their myoelectric activities instead of the SLP which brings certain limitations to our methodology.

- Muscle Mechanics

The biomechanics of masticatory muscles has received little attention in the TMJ literature beyond a consideration of leverage. Rayne (1987) has reported various muscle parameters obtained from cadaveric human masticatory muscles. The mean fibre length of the LPM is 23 mm, its index of cross sectional area is 17 %, the smallest and nearly one fifth, of the four masticatory muscles. It has been shown that the greatest tension is exerted when the fibres are about 90% of their maximum sarcomere length (the variability being from 1.8 to 3.2 μm). The LPM at rest, has one of 2.9 μm being almost fully extended and reference to the traditional length/tension curve would indicate that this is its most powerful position. Whether or not this anatomical oddity has any clinical significance is debatable, but it does not indicate that the forward posture adopted by many patients would approximate the theoretical rest position of the LPM (Rayne, 1987). The LPM is made of 70% type I slow twitch muscle fibres, therefore it is anatomically made to be a postural muscle, i.e. a fatigue resistant non-nil rest tonus with an inevitable tendency to myospasticity.

In the jaw elevators muscles (masseter, temporalis and medial pterygoid) type I fibres (slow

contracting, fatigue resistant, low force, postural) predominate in the anterior part and type II fibres predominate in the posterior part (larger, stronger, anaerobic, quickly fatigue). In the LPM's, fibres are smaller and mostly of type I (Mao et al., 1992).

The Normal Function of the LPM

Previously, the LPM has been looked upon as a single muscle with a single function (Moyers, 1950; Carlsoo, 1956; Zenker, 1955; Moller, 1971). None of the previous studies distinguished between the two heads in their recordings. In addition, they were all in agreement that the LPM is inserted on the disc. The discrepancies in these findings are related with the fact that the SLP has been considered as a single functional entity and the results were probably due to an electromyographic placement in the ILP only as the SLP is thinner and less accessible. It was only in the late seventies that the insertion technique started to be refined. Furthermore, there is a question as to whether the constant and the antagonist electric activity found by Carlsoo (1956) and the three EMG patterns recorded by Miller and Vargervik (1980) were either noises captured by their primitive EMG equipment or true EMG signal from variation in electrode placement in the two different heads.

The first worker to record independent muscle activity from each head of the LPM was McNamara (1973, 1974) on rhesus macaques. He innovated in describing independent functions for the two heads. Consequently, a large number of EMG studies distinguished between the two bellies of the LPM in their recordings. As a result, very different if not antagonist and reciprocal EMG activities of the two heads were reported. (McNamara, 1973; Lipke et al., 1977; Gross & Lipke, 1979; Gibbs, 1984; Juniper, 1981, 1984; Mahan et al., 1982; Mahan et al., 1983; Gibbs et al., 1984; Williamson, 1988; Bell, 1983, 1990; Dahan & Boitte, 1986; Bourdon, 1988). Auf der Maur (1980), reported results contradicting Petrovic et al. (1974, 1975) which is that the LPM play an important role in controlling condylar and mandibular growth.

Very interesting and totally contradictory findings were described by Lehr and Owens (1988) who investigated the EMG activity of the human LPM's. They suggested that separate roles for the SLP and ILP cannot be supported with EMG which questions the functional differentiation of the two portions described previously. The study demonstrated that the LPM is active in protrusive movements, including an incisor clench and non-active on retrusion or on molar clench. In addition, both lateral pterygoid muscles initiate depression and contralateral transversion. Therefore, they denied that the muscle has any stabilizing role on the joint and had two different functions.

Carpentier et al. (1988) supported the theory stated that the LPM has only one function: to depress and protrude the mandible as if it had a single head of origin. They attributed the repeatable

activity recorded by other authors, who favour separate functions, to a neuromuscular adaptation to an occlusal abnormality rather than a separation of two normal behaviours. It is suspected that the ones reporting one function only misplaced their electrodes into the ILP, as the SLP is just a few millimetre thick and probably that they wanted to avoid inserting in the elevators. It is important to keep in mind, especially since the basic anatomy has still not been clearly established, that mandibular biomechanics depends enormously on the LPM insertion which in turn will have a major effect on disc displacement, diagnosis and treatment.

The controversy over whether the muscle is mechanically a depressor or a protractor was sparked by functional analyses using a stationary axis of rotation located at the condyle (Moyers, 1950; Carlsoo, 1956). In a biomechanical study, Grant (1973) used an instantaneous axis of rotation and found significantly different results in reinterpreting previous EMG findings. He described two heads exhibiting quite different mechanical properties: the inferior head has a strong opening moment (as expected) which increased as the mouth opened. The superior head has a closing moment (opposite to an opening moment using the condyle as a stationary rotational axis) and a small opening moment on full opening opposition. Consequently, the two heads are functional antagonists. But from the EMG studies described above, no electrical activity in opening or in protrusion have been reported for the SLP. Therefore the SLP cannot be associated with this opening movement.

- The Inferior Head of the LPM (ILP)

Many experts (McNamara, 1973; Lipke et al., 1977; Gross & Lipke, 1979; Gibbs, 1981; Juniper, 1981, 1984; Mahan *et al.*, 1982; Mahan *et al.*, 1983; Gibbs *et al.*, 1984; Bell, 1983, 1990; Dahan & Boitte, 1986; Bourbon, 1988) recognized the fact that the two muscle bellies have quite different mechanical properties and contract independently. The ILP is synergically active with the digastric and suprahyoid group in opening movements; it also contracts in protrusive and contralateral motion of the mandible. It was found inactive in closing, clenching and swallowing. More specifically, it exerts a downward and inward pull on the mandible.

McNamara (1973) interpreted that it assisted translation of the condyle downward, anteriorly and on contralateral motion during opening. Wood et al.(1986) also reported that the ILP was well suited to move the condyle over the articular surface with minimal friction. Therefore it seems to be the power force of the mandibular condyle. In a intramuscular EMG study, Wood et al. (1986), found that the ILP was mostly active during anterior and contralateral clenching and jaw opening. Thus their interpretation was that the ILP participates in bracing the condylar head against the articular eminence during clenching involving condylar displacement and not in compressive phases.

- The Superior Head of the LPM (SLP)

On the other hand, the SLP elevates and closes the mandible. No activity was noted in the SLP during protrusion and opening movements, though it acts as an antagonist to the suprahyoid group (McNamara, 1973; Lipke *et al.*, 1977; Gross & Lipke, 1979; Auf der Maur, 1980; Juniper, 1981, 1984; Mahan *et al.*, 1983; Gibbs *et al.*, 1984). However, it was active concurrently with the elevator musculature during closing, clenching, swallowing and chewing and intercuspal clench. The SLP may stabilizes and positions the condyle and disc up and forward against the articular eminence in closing movements as well as during protruded and retruded clenching (McNamara, 1973). Mahan et al. (1983) and Gibbs et al.(1984) specify that the SLP is moderately active in ipslateral movements. Lipke *et al.* (1977) stated that the SLP normally remains inactive (muscle tonus only) at all times except during power strokes and/or maximum intercuspation.

As a verification tool in order to recognize a precise insertion position, Mahan *et al.* (1983) suggested a differentiation between the two portions of the LPM. The principle lies in comparing EMG recordings during a retruded clench where the ILP is completely inactive and the SLP is constantly very active with a resisted protraction where the opposite activities are found.

Juniper (1981) suggested that the SLP may be doing more than just contributing to the TMJ stability. It must be primarily a closer of the mouth by directing the force of mastication on the loading articular eminence, the SLP was almost identical to the masseter and temporalis muscles in the exception that it is involved mostly in the terminal closing movement and that it acts through the meniscus. Moreover, there is a slight but constant activity of the SLP in the resting position which could only be abolished on opening and protrusion. Thereby, he assumed that the anterior translated position is important for a normal rest of the masticatory musculature. Bell (1983, 1990) stated that this muscle is the active power system of the articular disc for movement that is independent of the bony components of the joint. Therefore the SLP does not move the joint but the meniscus. As such, it is a separate, functioning unit of the joint. He believes that it is a misconception to think of the SLP as a protractor of the jaw only because, technically, its force is in an anterior direction. It protracts the disc simultaneously with the protracting force of the ILP on the condyle during forward translation movements.

The elasticity of the superior retrodiscal lamina creates a traction on the disc in a posterior direction when it is stretched by an anterior translation of the disc condyle complex. The SLP normally balances the posterior elastic traction of the lamina to ensure that the disc occupies the most relaxed rotatory position on the condyle in the articular space (Bell, 1983; Lehman, 1991). In other words, the SLP permits the disc-mandibular condyle assembly to translate forward when it contracts. Contraction

of the SLP anteriorly rotates the disc on the condyle during the closing movement as far as the width of the articular space permits which is in concordance with Bourbon (1988).

Carpentier (1988), by considering the condyle attachments of the upper head onto the condyle only, regards the anterior disc pulling action as improbable. On the basis that the fibres of the upper head run under the anterior band of the disc, and seem to be deflected by it when the jaw is in the intercuspal position, Carpentier suggests that their contraction binds and pulls the disc up over the medial pole and stabilizes the condyle during closing movements. At the superior pole, the SLP exerts tension on both the disc and the condyle binding them tightly together. This "homing system", as Carpentier calls it, may help the disc to rotate back into place and thus reach its final position in the intercuspal position without any damage to the ligaments. On mouth closure, the SLP counteracts the elastic pull of the bilaminar zone of the disc along with the retrodiscal tissues. The contraction possibilities of the SLP being 30 to 35% of its 40 mm length is exactly what (10-12mm) the bilaminar zone can be stretched when the condyle rotates and translate. Under normal function, the SLP only works on closure, repositioning the disc and the condyle in a close-packed position to prevent joint damage by in the best shock-absorber position.

In attempting to understand the function of the SLP it is important to clarify whether its major attachment is to the disc to the condyle.If the muscle is attached to the disc, its contraction will pull the disc forward on the condyle. If the major insertion is to the condyle with the disc attached to the upper surface of the muscle, its contraction will pull the disc and condyle forward together. This understanding is of great importance regarding the concept of ADD.

Though, the normal single or independent functions of the LPM's has been frequently investigated compared to its function in pathological joints. However, two important facts can be brought up: even if specific EMG findings have been reported, EMG activity of coactivation, of synergist and antagonist have also been found. Secondly, whatever the myoactivity is, the role of the muscle still highly depends on its attachment to the disc and condyle.

The Pathological Function of the Masticatory Muscles

As mentioned earlier, there are many hypotheses for the etiology of TMJ dysfunction. Several factors may also interact to produce and perpetuate a dysfunction. The implication of the masticatory muscles, their activity and reactivity in the etiology and development of TMJ disorders have long been recognized. Abnormal muscle contraction of the LPM has been reported as the cause as well as the consequence of craniomandibular disorders. Confusion over its role in the pathological process is understandable due to the anatomo-physiologic controversies discussed earlier and its presumed pulling action on the disc.

Since Joret·ack (1956), who first studied abnormal jaw muscle contraction by means of EMG, a large number of scientists have tried to clarify the cause, consequence and muscle involvement in the TMJ syndrome. Unfortunately basic EMG studies on masticatory muscles were done either on normal subjects, either on the masseter and temporalis muscles of symptomatic patients. These muscles were probably chosen because of their easy accessibility and their large size which presumed more influence on jaw function. Moreover, intramuscular insertion techniques were primitive, inefficient and provoked complications. Those early studies were not concerned with quantification of the EMG response. Control groups were not always used and the populations sampled were not properly defined. In addition precise or valid normalisation techniques were not used and results were often presented in the form of case reports. Finally, the presence of occlusal interferences was improperly regarded as a true pathological condition.

- The Pathological Muscle Activity of the Masseter, the Temporalis and other Masticatory Muscles

EMG has been widely used to demonstrate abnormal muscle activity in the jaw elevators, mainly the masseter and temporalis. Two major findings were: an increased resting or postural muscular activity associated with TMJ dysfunction and a low maximum clenching and resisted strength as compared to controls. The latter finding may be interpreted as muscle fatigue secondary to hyperactivity.

In a literature review on EMG studies of craniomandibular disorders, Dahlstrom (1989) stated that sleep and basic laboratory studies support a correlation between masticatory muscle hyperactivity and TMJ syndrome concluded that the average postural muscular activity of patients was significantly higher than controls, mostly pronounced in the anterior temporalis. Many investigators studied resting EMG activity in patients with myofascial-pain dysfunction syndrome and reported a significantly higher (more than twice) EMG level than in asymptomatic patients before and even after dental treatments (Sheikholeslam and Moller, 1982; Gervais et al., 1989; Glaros et al., 1989; Jankelson, 1990). Sheikholeslam and Moller (1982) interpreted this increased rest activity either as a reflex response to occlusal interferences, to ID or to psychological stresses.

Griffin & Munro (1971) and De Laat et al. (1985) EMG findings suggested that these abnormal muscle behaviour are due to inadequate stimulation of dental pressoreceptors (premature contact of gross tooth loss) or to hyperactivity of the retinacular formation associated with malalignement of the TMJ. They attributed their results to hyperactivity of the motoneuron from a suppression of the inhibitory reflex. In a study treating myotatic contracture with neuromuscular stimulation, Jankelson (1990) found a decrease in the EMG activity of the masseter and the temporalis with the patient at rest

(meaning that they were hyperactive without treatment) and also that there was an increase in EMG on clenching after treatment.

On the other hand, the most common cause of muscular hyperactivity appeared to be muscle fatigue produced by chronic parafunctional oral habits such as clenching and grinding. These contractions are often seen as an involuntary tension-relieving mechanism (Laskin, 1969; Kotani et al., 1980). Sherman (1985), noted that bruxing and clenching patients with or without TMJ problem had far higher EMG levels than subjects with TMJ problem alone. This point implies that parafunctional habits and not pure TMJ ID causes muscle hyperactivity.

The concept of increased muscle activity, whether secondary to occlusal interferences, muscle spasm, oral parafunctional habits or psychological stress, is quite dominant in the EMG literature of the masticatory system. In the majority of EMG studies done on TMJ disorders, the LPM was not studied despite reports linking it to TMJ disorders. As of EMG results, an increased signal amplitude is still the major finding from the masseter and temporalis muscles.

- The Pathological Activity of the LPM

The role of the LPM in mandibular movement has always been extremely difficult to determine. In the last two decades, many authors agreed on two separate normal biomechanical functions of the LPMs. In contrast, its role in TMJ dysfunction has just started to be seriously investigated. There are two leading hypotheses regarding the muscular activity of the SLP in the pathologic and etiologic process of ID: hyperactivity and hypoactivity/altered of the SLP. Neither are understood as a cause or a consequence of TMJ disorders.

It has long been recognized that SLP hyperactivity is associated with TMJ syndrome. For more than five decades studies have shown consistently that myofascial pain-dysfunction patients have higher EMG activity in the head, neck and jaw muscles than do control subjects. Some authors related this hyperactivity/spasm to stress, grinding, or pain (Moyers, 1950; Ramfjord, 1961; Macnamara, 1973, Sheikholeslam & Moller, 1982; Gervais, 1989; Glaros 1989).
Neurophysiological studies of TMJ dysfunction demonstrated that hyperactivity of jaw-closing muscles may originate in the central nervous system, which could be sufficient to cause malocclusion (Yemm, 1976).

Johnstone (1980) believed that the LPM is inserted on the disc and functions as a mandibular protractor and suggested that a hypercontraction of the LPM would displace the disc anteriorly. He also stated that parafunctional habits of the masticatory muscles, such as clenching and grinding may be responsible for LPM hyperactivity. Conversely, Bell (1990) believed that occasional clenching is not

only an abnormal physiologic activity but a necessary one. He stated that clenching and grinding may be viewed as an inverse stretch reflex of the LPM that is stimulated to maintain full resting length of its fibres. Just as an occasional yawn is necessary to maintain full resting length of the elevators, clenching prevents the loss of the ability to open the mouth normally and to function adequately (Bell, 1990). A question arises: could people with a shorter SLP tend to clench their teeth more as an involuntary response to this stretch reflex? Moreover, could people with short SLP due to ADD exhibit hyperactivity of this masticatory muscle due to this stretch reflex? Furthermore, hyperactivity of the SLP is known to cause ADD which in turn, produces premature contacts (Juniper, 1985). Posterior occlusion could then be a result not a cause of ID. Isberg *et al.*, (1985) combined clinical and radiographic examination with EMG recordings of the masseter and temporal muscles in patients with ADD with reduction. They found that the masticatory muscles could be activated by disc displacement which ceased when the disc position was normalized on mouth opening. In other words, ID causes hyperactivity of the muscles.

According to some modern anatomists (Gibbs & Mahan, 1984; Mahan et al., 1983; Isberg et al., 1985; Wilkinson, 1988; Carpentier, 1988) it is unlikely that the SLP pulls the disc forward without the condyle because its major attachment is onto the head of the condyle. It only pulls the disc forward if the insertion is detached from the pterygoid fovea. This attachment would ensure that the disc remains in normal relation with the condyle, or that it does not protrude far beyond with contraction of the whole muscle but stabilize it just anteriorly to the condyle (Gibbs & Mahan, 1984; Mahan et al., 1983). Consequently, the action of the SLP would prevent the disc from rotating behind the condyle as it moves forward and stretch the bilaminar zone. Wilkinson (1988) suggested that incisal clenching may be a major factor in ADD. According to Isberg (1985) the SLP is strongly active in incisal biting so contraction in this position should displace the disc if the SLP was hyperactive. In the theories of the pathogenesis of TMJ syndrome, we have seen that ID was interpreted to result from LPM hyperactivity. Later work suggested that TMJ dysfunction induce an increases EMG activity. Finally we found out that the presence of dysfunction alone could not produce hypercontraction. While EMG hyperactivity is known to occur in patients with TMJ dysfunction, whether this hyperactivity causes the disc syndrome or the disc displacement causes spastic activity is still unresolved.

Hypoactivity has also been found as a pathological activity of the SLP. This recent finding phenomenon was less common than the hyperactivity one. Carpentier (1988) used a dissection study to demonstrate that the main insertions of the SLP fibres were not in the disc but into the neck of the condyle. Therefore, an anterior displacement of the disc due to a spastic activity of this muscle alone was improbable. Hence, theoretical hypoactivity of the SLP may contribute to ADD (Carpentier, 1988). In other words, ADD could be induce if the SLP was to weak to pull the condyle forward as the jaw

opens. It is anatomically impossible for the SLP to pull the disc without affecting the condyle. Instead, ADD would result from a passive stretched of a causative factor such as the bilaminar zone, creating ligament laxity or causing elongation of posterior disc attachment due to incisal biting (Heeling, 1985; Wilkinson, 1988). Accordingly ADD may tend to "wrinkle" the muscle and stop it from contracting efficiently. Furthermore, it can be interpreted that subsequent to ID, the structure and function of the SLP is altered which could possibly maintain the derangement. Thus SLP patho-activity may be a result of ID not its cause (Mahan *et al.*, 1983; Gibbs & Mahan, 1984; Juniper, 1984; Carpentier, 1988).

Both the LPM and the mandibular elevators, were reported to exhibit irregular activity behaviour in patients with TMJ syndrome. This was attributed to anatomical or psychological factors or may be a consequence of the dysfunction (Moyers, 1950; Mahan *et al.*, 1983; Gibbs & Mahan, 1984; Isberg *et al.*, 1985; Zijun, 1989). EMG analysis and clinical investigations led to conclude that incoordination rather than hyperactivity is a sign of TMJ dysfunction (Nanthaviroj *et al.*, 1976). As early as 1950, Moyers found abberations in the mandibular muscle patterns. He suggested that malocclusion and teeth interferences as causative factors. More recently, Zijun (1989) found abnormal muscle activity (hyper and hypo) in most patients with TMJ sound disorders. He believed in a muscular cause for ADD and a discal insertion of the SLP.

In summary, EMG studies of the masticatory muscles in TMJ disorders have emanated from different paradigms. Despite the divergences in terminology, undefined samples and techniques, little differentiation between the two heads, the use of EMG has substantially increased our knowledge of the normal and abnormal behaviour of the masticatory system. Unfortunately, there are very few studies that have focused on the EMG of the LPM in subjects with ID of the TMJ.

The Masticatory Muscles and TMJ Force Studies

In TMJ disorders, the muscles of mastication were mainly reported by theoretical and experimental studies as being hyperactive, but also as exerting weaker clenching forces compare to normals (Sheikholeslam & Moller, 1982; Dahlstrom, 1989; Jankelson, 1990). Anatomical uncertainty regarding the insertion of the LPM's onto the intra-articular disc and/or mandibular condyle makes it difficult to understand how it functions and what the recordings actually represents.

In the 1970's, an outbreak of research was done on bite force: it was the odontologic subject of the decade. The general objective of those studies were wide and diverse: assessment of the properties of the teeth and gum, craniofacial biomechanics, alterations in vertical dimension, effect of tooth loss, malocclusion, dentures and functional disturbances and general data gathering on different

maximal and sub-maximal pressures of various bites (Linderholm & Wennstrom, 1969; Ringqvist, 1973; Carlsson, 1974; Mansour & Reynik, 1974; Helkimo et al. 1976; Dechow & Carlson, 1983).

The muscles of mastication also incur significantly low maximum biting strength and low average level of activity with respect to controls (Sheikholeslam & Moller, 1982; Dahlstrom, 1989; Jankelson, 1990). Treatment did not affect that strength parameter. Therefore subjects with weak muscles may be more prone to pain and TMJ dysfunction, meaning that the weak muscles have to be hyperactive in order to function effectively, thus causing an overloading condition. Jankelson (1990) found muscle hypoactivity in the anterior temporalis and hyperactivity in the masseter in patients suffering from TMJ disorders during maximal clenching. Therefore, one could expect that a person with TMJ syndrome would have weaker primary force muscle and hypertonic posturing muscle.
Moreover, the average level of activity on maximum biting is decreased in patients with TMJ disorders which could be interpreted as muscle fatigue from continuous hypercontraction.

There have been many more EMG studies on subjects with malocclusion and dental interferences than on true TMJ dysfunction cases. These studies showed that during maximal biting and chewing, the subjects exhibited less EMG activity and a different muscle-contraction pattern than the controls. The impaired muscular activity may be attributed to a diverging dentofacial morphology and unstable occlusal contact condition (a deep anterior overbite causes a posterior displacement of the mandible) (Pancherz, 1980). It is important to note here that we are not dealing with ID but with malocclusion and that hypoactivity not hyperactivity of the masticatory muscles was found. It may be concluded that if malocclusion is a cause of ID, hypoactivity may be present. Conversely, if malocclusion is a consequence of ID, hypoactivity might be the result of this disorder.

A number of methods and apparatuses for studying bite force have been described in the dental literature comprising various types of pressures strain gauge transducers providing satisfactory accurate results. However there has been confusion as to wether one is recording forces or pressures. It was suggested that the force is a measure of the resistance of the supporting tissues and the compressibility of the teeth before it is a true measured force or strength of the masticatory muscles (Carlson, 1974; Williams *et al.*, 1984).

Various limitations of previous masticatory bite force studies are as follow: Unexplicit selected samples has limited the possible interpretation of the data; Uncontrolled head and neck posture may of led previous authors to believe erroneously that the muscles were hyperactive and the position not favourable to optimal forces intake; The relationship between EMG and bite force have never been explicitly explained; Forces of the jaw were oriented towards a teeth/occlusion issue instead of a whole joint orthopaedic point of view such that up to date, opening forces have never been measured or documented.

III-INTERRELATIONSHIP OF TMJ ID AND THE LPM

Overview, Discussion and Conclusion

As an overview of the whole literature review, the significance of the role of the LPM in ID of the TMJ is discussed and summarized. As discussed previously, controversies in this particular subject exist everywhere from the basic anatomy of the LPM to the etiology of TMJ disorders. Overall, authors agree on the facts that TMJ ID is a psychophysiologic syndrome that primarily involves the muscles of mastication.

Causes of ID includes numerous factors such as occlusal stress, bruxism, psychological stress, cranio-cervical posture, trauma, eating habits, dental history, etc. Considering altered masticatory muscle activity alone, it can be seen as a primary cause of TMJ disorders; but it is mainly seen as secondary to forward head posture, masticatory parafunction, psychological tension, malocclusion, bruxism, which changes the contraction pattern of the jaw musculature which in turn alter the normal biomechanics of the joint. In other words, all these factors can lead to ID through muscular imbalance or impaired activity. This increased muscle tension combined with parafunctional habits can results in fatigue, pseudo-spasticity and spasm through muscle overexertion and overcontraction. The pathology usually starts as a non-articular pure muscular disorder and evolves as an ID of the joint itself. Hence, the muscular involvement is undeniable.

Theoretical explanations of ID pathogenesis can be divided in three major categories: 1) a passive explanation where hyper-opening, forward head posture, malocclusion and parafunction causing overstretching of the posterior disc attachments, allows the disc to rotate forward off the head of the condyle which in turns lead to reciprocal clicking, protective muscle splinting and/or altered function of the LPM's (Gage, 1989); 2) an active explanation where parafunctional hyperactivity, power strokes and maximum intercuspation leads the SLP to contract strongly and dislocate the disc anteriorly (Bell, 1983; Eversole & Machado, 1985; Benson, 1988). It is believed that the SLP pulls on the disc instead of stabilizing as proposed by the next group; 3) It is interesting to note how some authors cannot separate the passive and active concepts (Ogus, 1987; Benson, 1988; Bell, 1990; Weinberg, 1990) and came up with the third explanation: Muscle incoordination among agonists, antagonists and synergists can cause ID. Repeated abnormal activity of the SLP leads to either a neuromuscular adaptation or an incoordination. Ogus (1987) stated that there might be a malfunction of the neuromuscular system controlling the joint movements. He referred to Schwarts and Cobin (1957) and Bertry and Yemm (1971) who have proposed that muscular altered activity might be a primary cause

of TMJD. Zijun (1989) found SLP hyper and hypoactivity in patients with joint sound disorders.

Although some instances of muscle dysfunction can be attributed to defective contacts at either the intercuspal position of during excursive movements, it is inappropriate to attribute all clicking signs to malocclusion simply because there is some deviation from a preconceived notion of what is normal occlusion. Since an estimated 90 percent of the population have minor malocclusion, an etiology for overcontraction of the SLP muscle may be suspected only when an easily identifiable occlusal is followed by muscle symptoms.

Nevertheless, recent studies suggested that altered muscle activity is a secondary effect (Juniper, 1984). The damaged discal attachments have led to instability within the joint. To remedy the situation and stabilize the condyle against the articular eminence, the ILP is activated. Similarly, Nanthviroj (1976) proposed that clicking/locking may cause an incoordination of the EMG pattern of the masticatory muscles. It has been shown that masticatory muscle activity can be induced by disc displacement (Isberg, 1985). It is advocated that the muscular incoordination and the malocclusion are results of the ADD and removal of occlusal interference should not be performed until the disc position has been normalized. Consequently, the muscle imbalance is a result of ADD and not a cause. In other words, the forward disc position would wrinkle and shorten the muscle and uncoordinated muscular activity would result.

Overall, the muscular concept is at the top of the list: the hyperactive SLP pulling on the disc forward and medially would create the ADD condition and the reciprocal clicking assuming a discal insertion of the muscle. Which is completely different than seeing the muscular impairment as the consequence of the ID. To determine the sophisticated muscular control of the TMJ, it is critical to clarify whether the SLP is attached to the disc and /or to the condyle to know if a contraction of its fibres will pull forward the disc, the condyle or both together therefore changing the whole pathobiomechanics of the ADD process. A unequivocal correlation exists between an abnormal contraction of the LPM and ID. Therefore as ADD is clinically expressed as reciprocal clicking, recording the clicking sound at the same time as the EMG signal in clicking jaws will give us valuable information regarding the interrelationship of those two parameters in TMJ disorders.

In patients suffering from TMJ disorders, an increased rest muscular activity has been recorded in some masticatory muscles. Is it because the recordings were mainly into the temporalis and the masseter described as postural muscles and tension relieving "clenchers" ? Or can it be because the majority of these subjects were in forward head posture which induces an hypertonus in all mandibular muscles? And again, is that muscle activity responsible for the pathology or is the pathology inducing a muscle hyperactivity? And finally can this abnormal hypercontraction maintain the ID?

A second popular finding is that pathologic TMJ patients exhibit weaker muscular strength and different muscle contraction pattern. Is it due to pain? To a neuromuscular inhibition reaction? Is muscle fatigue a result of the disorder which in turn induces hypertonus at rest and weaker primary force? All these questions are not directly going to be answered but may be somewhat covered into our discussion and conclusion.

In spite of all the different concepts and hypotheses suggested regarding the muscular activity of the LPM's in ID, we are still in a state of confusion. Therefore it is expected that future research will address those issues.

CHAPTER III

METHODOLOGY

Summary and Purpose

The purpose of the present study was to investigate and analyze the intramuscular electrode EMG activity of different static and dynamic tasks of the two portions of the LPM in control and TMJ ID subjects. Surface electrode EMG of the masseter and the temporalis muscles were also recorded in order to have a complete masticatory picture and patho-muscular pattern.

Previous TMJ studies were concerned mainly with the masseter and temporalis muscles and various experimental samples. The undescribed selected samples of previous studies has limited the possible interpretation of the data. Moreover, uncontrolled head and neck posture may of led previous authors to believe erroneously that the LPM was hyperactive. The insertions of the LPM's remains uncertain which makes it difficult to interpret the data.

Biomechanical relationships between muscle activity (especially the LPM as it is probably inserted onto the disc), jaw force, jaw motion and joint noises in regards to TMJ ID were examined in the present investigation. It was expected that patients with TMJ ID demonstrated LPM dysfunctional activity. In order to reach this objective, the following measurement techniques were used simultaneously to collect data: intramuscular and surface electrode EMG, force transducers, electrogoniometry, and sound recording.

The LPM's were investigated because they are anatomically and functionally related to disc derangement of the TMJ and because they are believe to be abnormally recruited in TMJ disorders. If this abnormal activity is a cause of a consequence of the derangement remains unknown. These two larger main masticatory muscles were recorded in order to correlate their activity with the LPM's and to assess their recruitment pattern and contribution in TMJ ID.

INSERTION PROCEDURES

Preliminary experiments

For standardization and precision of the electrode insertion of the two heads of the LPM's, preliminary experiments were performed in order to verify and master the techniques used by Gross & Lipke (1979), Mahan *et al.*, (1982, 1983), Gibbs *et al.* (1984), Travell & Simons (1983), Bell (1990), and by Koole *et al.* (1990).

Facial soft tissue dissection and insertion trials were done on human specimen available at the department of anatomy of the University of Ottawa. As confirmed by these procedures and anatomy books (Cabrol, 1980; Ide & Nakazawa, 1990) no arteries or nerves are in the path of insertion: the external carotid artery and the external jugular vein are at least 3 cm posterior to the insertion entry; the maxillary and transverse facial arteries are at a minimum of two cm below the insertion point; the larger facial vein and artery are five cm anterior and below the path of entry; as for the nervous pathways, the rami of the parotid plexus covers the superficial area of the masseter not entering the mandibular notch area. The insertion trials were very easy to make without approaching any vessels and the muscles were larger than expected. Surface anatomy guidelines were noted such as the fact that the SLP is at the base of the nose level just under the z arch floor. The only structure we had to go through were the masseter muscle fibres and its thick facia overlying the deeper LPM's.

As part of the preliminary experiments, EMLA cream (Astra Pharma Inc., Toronto), a topical anesthetic for dermal analgesia, have been tried on the thigh and then on facial skin with excellent analgesic results using 26 gauge needles.

EMG Electrodes

The muscular activities of the LPM's were captured by bipolar intramuscular fine wire electrodes using the method described by Giroux and Lamontagne (1990). The need for implanted electrodes was not under dispute as the deep anatomical position and underlying situation of the LPM's made surface electrodes inappropriate. The electrodes were inserted into the superior and inferior portions of the LPM on the selected side.

The superiority of wire electrodes over needle electrodes for intramuscular recording is now well established (Kadefors & Herberts, 1977; Notermans, 1984; Basmanjian & De Luca, 1985; Moritani *et al.*, 1985). Intramuscular wire electrodes are known to be selective and record a larger number of

muscular fibres than needle electrodes. Yemm (1977) demonstrated that intramuscular needle electrodes were best suited for observation of low levels of muscular activity.

The bipolar fine-wire electrodes (Biomed wire, Cooner Wire Company C.A., 51 μm in diameter) were custom-made with a platinum-tungsten alloy (92%-8%) possessing both ductility and strength. Both wires, 15 cm long (Ni/Cr, 0.01 cm diameter), were threaded into an hypodermic needle (26 gauge, 1.5 & 1.75 inch). The insulation of both extremities of the wire was burned over an approximate length of 5-6 mm and cut to approximately 1 mm from the bare tip, then the wires emerging from the tip were bent and cut with a scalpel blade so that the two wires are slightly spaced. The indwelling nickel-chromium electrodes were custom-made using the method described by Giroux & Lamontagne (1990) (Figure 6). Before implantation in the muscles, the needles and wires were sterilized at 150°C for 30 minutes at 20 psi in a TIME 250 AUTOCLAVE at the Biology Department (U. of Ottawa).

Insertion protocol

Sixty to ninety minutes before inserting the electrodes, alcohol disinfectant and local skin topical analgesic EMLA (Eutectic Mixture of Local Anesthetics) were applied on the cheek over the sigmoid notch. The selection of an insertion technique for both portions of the LPM's was guided by an extensive theoretical and practical review of numerous investigations (Macnamara, 1973; Lipke *et al.* (1977); Gross & Lipke, 1979; Hannam & Wood, 1981; Mahan *et al.*, 1982, 1983; Gibbs *et al.*, 1984; Travell & Simons, 1983; Wood, 1986; Bell, 1990; Koole *et al.*, 1990). No complications were reported.

The extraoral approach over the intra-oral route was used for several reasons. No post-insertion sequelae (pain, haematoma and difficulty opening) or technical difficulties in electrode placement (repetitive re-insertions) have been reported as opposed to the transoral one. All portions of the LPM are best inserted extraorally through the sigmoid notch, through the masseter and deep into the ramus of the mandible and there is less no interference with movement. The technique has been proven safe and reliable by Gross and Lipke (1979). Based on previous studies, Auf der Maur (1980) reported that the intraoral techniques often leads to erroneous recordings and haemorrhages from the pterygoid venous plexus (Figure 7).

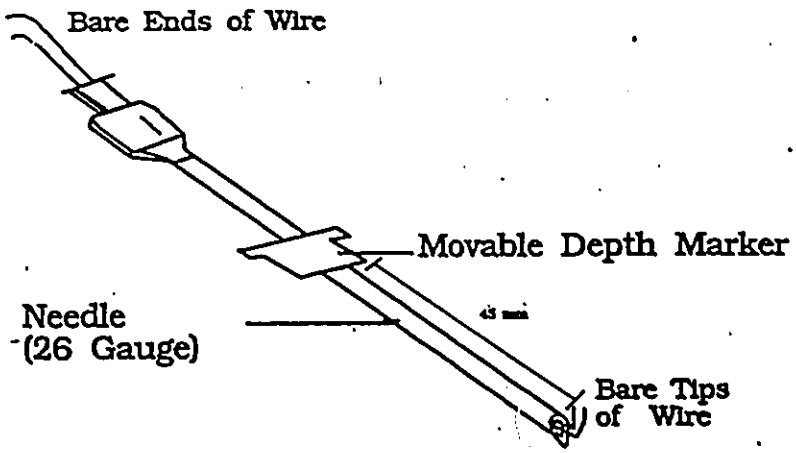

Figure 6 : Fine-wire electrode and needle used for intra-muscular EMG

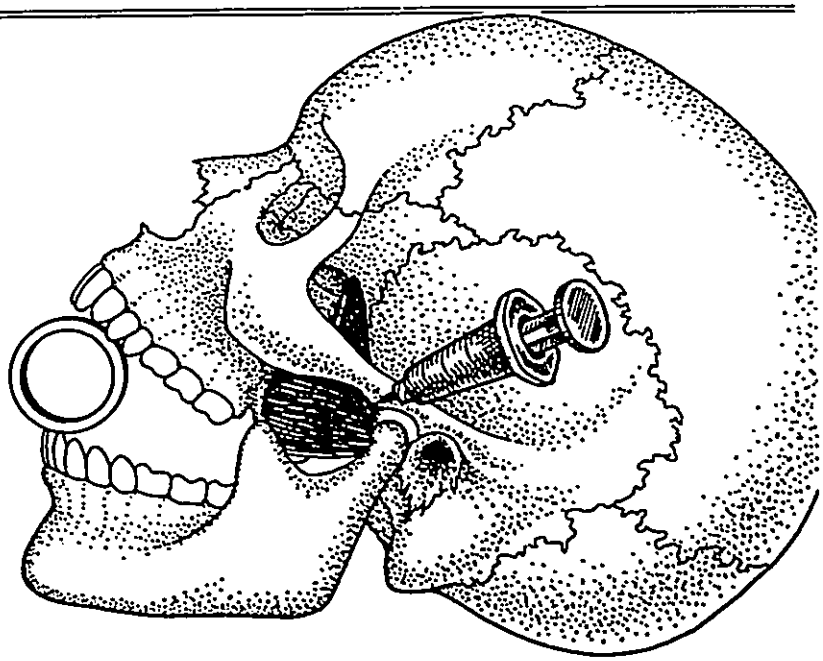

Figure 7a: Insertion technique of the ILP and SLP muscles.

Note: From The Trigger Point Manual (p.265) by J.T. Travell and D.G. Simons, 1983. Baltimore: Williams & Wilkins. Copyright 1983 by W & W. Copied by permission

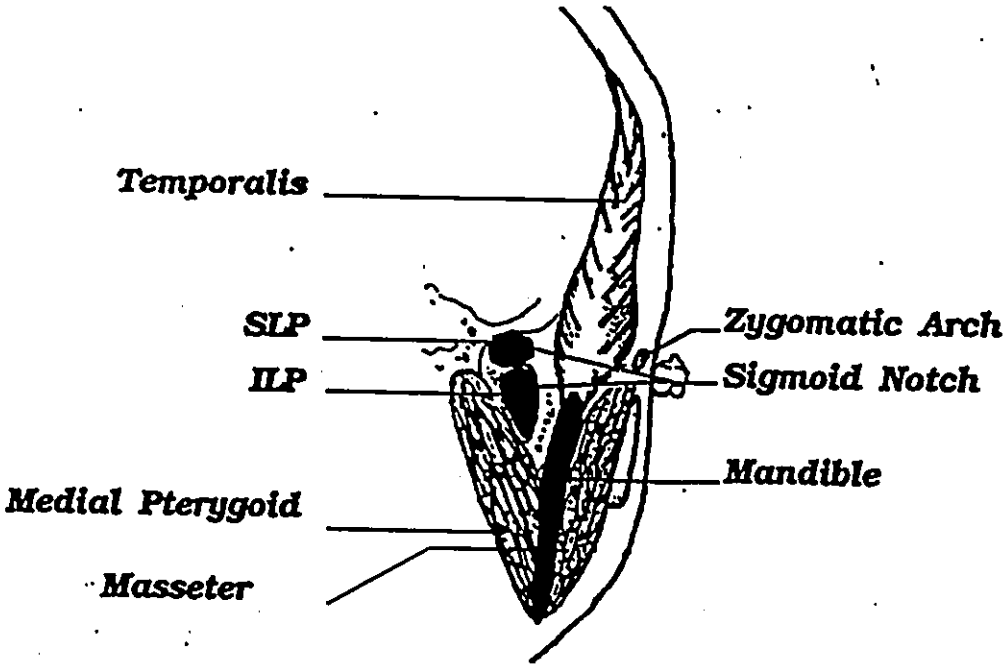

Figure 7b: Electrode placement: diagrammatic frontal section at the sigmoid notch of the mandible.

Note: From "A technique for percutaneous lateral pterygoid electromyography" by B.D. Gross & D.P. Lipke, 1979, Electromyography and Clinical Neurophysiology, 19 (p.52) Copyright 1979 by B.D. Gross. Adapted by permission

- Inferior Head of the Lateral Pterygoid Muscle (ILP)

For the ILP, the extraoral technique was used and described as follows: 1) the zygomatic arch and the lateral aspect of the condyle were palpated in order to localize the sigmoid notch. The sigmoid notch is described as the bony aperture above the mandibular notch between the coronoid process in front and behind the condyle (Travell & Simons, 1983) and below the Z arch. With slight opening of the mouth, two knuckles, (22-30 mm as suggested by Travell & Simons, 1983), the point of entrance was is just anterior to the condyle and 15 mm inferior to the zygomatic arch; 2) the needle was directed nearly perpendicular to the surface towards the lateral aspect of the lateral pterygoid plate of the sphenoid bone which is situated approximately 30° medially and 30° caudally to the point of insertion. The needle was then advanced until the lateral pterygoid plate was felt. The masseter fascia penetration was easily felt and heard by the subject and the inserter. A slightly downward direction toward the angle of the mandible to reach the ILP instead of a straight horizontal, which can get into the superior head; 3) the ILP fibres are reached only after full penetration of the masseter muscle. A gentle contact with the pterygoid plate followed by a gentle retraction ensures that we are in the space within which the muscle lies.

- Superior Head of the Lateral Pterygoid Muscle (SLP)

The extraoral route was also used for the SLP. Mahan et al. (1982) observed many placement errors with the intraoral access, electrodes were misplaced in the ILP or the medial pterygoid muscle. Our method is an adaptation from various techniques used previously by many researchers (Gross & Lipke, 1979; Auf der Maur, 1980; Mahan *et al.*, 1983; Travell & Simons, 1983; Gibbs *et al.*, 1984; Bell, 1990; Koole *et al.*, 1990).

The point of entrance was situated 6 mm below the zygomatic arch, just anterior to the most forward position of the condyle at maximal opening where the lateral pole of the condylar head can be palpated. The needle is directed at 90° to the skin surface and slightly upward and anterior with respect to the zygomatic arch through the sigmoid notch, toward the orbit. The preliminary experiment reveals a direction toward the base of the nose which is the lower border of the zygomatic arch, 3 cm medial and slightly anterior. The needle is inserted through the resistant aponeurosis of the masseter muscle, no bony contact is felt. If the needle goes through the temporalis muscle, the penetration of its fascia can produce an obvious brief resistance. The needle was moved along the roof of the infra-temporal fossa within about 10 mm from the junction of the greater wing and the lateral pterygoid plate. The

needle should attain a depth of approximately 30 mm to 35 mm medial to the skin. The electrodes were then fixed to the cheek skin leaving loops for movements to prevent artifacts and promote comfort of motion. The tips were isolated with medical tape which made our EMG signal clearer.

Insertion Application

The selection of an insertion technique for both portions of the LPM was guided by an extensive theoretical and practical review (Gross & Lipke (1979), Mahan et al. (1982, 1983), Gibbs et al (1984), Travell & Simons (1983), Bell (1990), and Koole et al (1990). Insertion for both portions of the LPM was achieved extraorally through the sigmoid notch. This technique was preferred because it is relatively simple and does not lead to post-insertion sequelae or technical difficulties in placement. A physiatrist specialized in EMG performed the insertions.

Verification of the electrode's position was done according to the studies of McNamara (1973), Gross & Lipke (1979), Mahan *et al.* (1983), Gibbs *et al.* (1984) and Koole (1990). They found strong myoelectric activities of the SLP on clenching; moderate activity on ipsilateral movement and closing in retraction; and no activity on opening and protraction. On the contrary, the ILP exerts no EMG signal on elevation or closing movements but is strongly active on opening and protrusion especially upon resistance. This testing was mostly applicable in the control subjects. Hence, we had to rely on the inserter's certitude of the placement combine with EMG firing for the TMJ ID group.

We followed the insertions techniques described previously. The mandibular notch was the easiest spot to insert as for finding it, direction, soft tissue mass, but two problems arose: pain on maximum opening and closing with a locking sensation on resisted motion, and no feedback on the exact situation of the needle. After the first five subjects and studying carefully the anatomical atlas of the TMJ (Nakazawa & Kamimura, 1991) a different approach was tried. Adding intraoral palpation for guidance, the second approach consisted of proceeding higher and more posterior close to the condyle and just under the zygomatic arch, just above the coronoid process without being exactly into the notch. The palpation was done at the junction of the coronoid process and the arch just above the last molar, feeling the masseter and the needle. Therefore, the SLP was less difficult to insert as there was no muscle in the way during that upward direction under the arch near the posterior teeth with intraoral guidance. It was also easier to locate the lower portion because of its larger size, the needle was simply redirect in a medial and downward direction. To avoid wire deformation during the recording, the subjects were instructed to contract their muscles few times prior to data acquisition.

EXPERIMENTAL PROTOCOL

Subjects

Ten female subjects with ID of the TMJ aged between 19 and 37 voluntarily participated in this investigation. Two subjects were excluded, in one case the EMG signal was qualified too noisy and the other one, the needle was pulled out during testing. This sample was selected from a population of patients seen in a Physiotherapy Clinic specialized in TMJ management in Ontario, Canada . All patients were referred to us by the dental or medical professions. Ten control females, matching in age, from the general population, served as controls. Female subjects were chosen as they are the best representative group reported in epidemiological studies (7 female for 1 male suffering from TMJD).

- Selection Criteria

The experimental subjects were selected on the basis of the following criteria: 1) professional diagnosis of TMJ disorder; 2) pain, tenderness or discomfort should be present in one joint, ear or cheek area; 3) discal clicking and corresponding movement dysfunction. These were regarded as determinant factors to be considered as having ID (Owen, 1987; Gage, 1989; Bell, 1990). Movement dysfunction related to discal displacement consisted of a deviation towards the clicking side on opening, and/or a limited contralateral translation and/or a limited opening and/or a limited protrusion range.

The exclusion criteria were as follows: 1) closed lock conditions and hypermobile joints were not selected. Subjects with major mechanical head and neck disorders were also excluded form the study. Patients with reciprocal clicking and obvious opening discal clicks were considered as having ADD according to the criteria established by authorities (Ireland, 1951; Farrar, 1972; Weinberg, 1980; Dolwick, 1983; Rocabado, 1983; Isberg, 1985; Owen, 1987; Gage, 1989; Bell, 1990).

The ten control subjects were selected according to criteria modified from Goldstein (1984): No signs nor symptoms of TMJ dysfunction, functional occlusion, a full complement of at least 28 teeth, no major dental restoration or cuspal coverage, no history of orthodontic treatments and no history of pain or trauma in the head and neck areas.

- Clinical data

Two questionnaires (appendix B) were filled out by all the subjects to avoid any medical complications and to determine specific conditions that could alter the validity of our results and explain variations in data. All subjects had at least 28 teeth; one control and three TMJ were missing one to four teeth. Pertinent results are as follows: all control subjects reported comfortable bite, however 50% of the TMJ felt biting discomfort. One control and four TMJ participant reported being grinders and clenchers. None of the two groups had major dental work done, but more than 60% of the two groups stated that they went through orthodontics and occlusal appliance. None of the control group and half of the TMJ group reported intermittent necks pains and headaches and had a course of TMJ treatment (dental and/or physio therapy). All subjects were not affected by osteoarthritis or by any other related health problems and were not taking any medications. Nobody reported allergies to anesthetics or blood pressure and bleeding disorders for insertions needs.

Clinical data and range of motion of the TMJ ID and control samples are shown in Table 1a,1b and 2a,b respectively for both samples. Ranges of motion were measured with the Therabite Instrument cardboard curved ruler. Ranges of motion of the control group were within normal limits; for the TMJ ID group, movement dysfunction was present in all cases (Table 1a,b and 2a,b for the control and the TMJ ID group).

Procedures

A few days before testing, all 20 subjects were individually taught how and where to put the ELMA (Eutectic Mixture of Local Anaesthetics, Astra Pharma Inc., Toronto, Canada) cream, 90 minutes before testing time (Appendix B). Unilateral testing was done as the condition generally affects one joint (Laskin, 1986). All subjects involved in our study suffered from only one side, or if bilateral, one side was definitely worst. Prior to the testing period, the subjects were asked to read an information letter and sign a consent form explaining in detail their duties, their rights, and the testing protocol (appendix B).

The subject layed down in supine and the insertions were performed by a physiatrist specialized in EMG electrode insertion (Figure 8). Before data collection, verification of the insertion of the ILP was determined by recording strong activity during opening and protrusion and none during clenching on the oscilloscope (Gross & Lipke, 1979; McNamara, 1973). Verification of the SLP was obtained by recording strong activity during clenching and an absence of activity during protrusion and opening (Gross & Lipke, 1979; McNamara, 1974). For the superficial muscles, the masseter strongly fires

TABLE 1a. CLINICAL DATA: SUBJECTS CHARACTERISTICS

CONTROL SUBJECTS

Subject (Code)	Age	Pain (Left/Right)	Tenderness (Left/Right)	Clicking (Left/Right)	Other Symptoms	Duration of Symptoms (Years)	Probable Etiology
CML	21	0	0	0	NONE	N/A	N/A
JVL	27	0	0	0	NONE	N/A	N/A
CLL	26	0	0	SOFT R	RESOLVED WHIPLASH	N/A	N/A
JQL	30	0	0	0	NONE	N/A	N/A
DCL	33	0	0	0	TORTICOLIS	N/A	N/A
JCL	20	0	0	0	NONE	N/A	N/A
MRL	27	0	0	0	NONE	N/A	N/A
CWL	22	0	0	0	NONE	N/A	N/A
LFL	21	0	0	0	NONE	N/A	N/A
MML	25	0	0	0	SLIGHT OVERALL LAXITY	N/A	N/A
Mean	25.2						

N/A: Non Applicable 0: None/Absent R: right TMJ

TABLE 1b. CLINICAL DATA : SUBJECTS CHARACTERISTICS

TMJ ID SUBJECTS

Subject (Code)	Age	Pain (Left/Right)	Tenderness (Left/Right)	Clicking (Left/Right)	Other Symptoms	Duration of Symptoms (Years)	Probable Etiology (Diagnostic)
GLL	24	R		R>L	Right neck pain	5-10	ID, discal **mechanical**
YCL	28	NO	NO	L	Locking Muscle fatigue	>10	ID **parafunction**
SKL	25	L>>R	L<R	L<R	Severa TMJ pain Muscle fatigue	10-11	ID,discal **occlusion, parafunction**
LAR	28	R	R	R	Muscle tension Neck stiffness	>10	ID, ant. disc displ. **muscular incoord.**
JBR	23	R	R	R	Neck discomfort	3-4	ID, ant. disc displ.
KMR	19	R	R	R	Headaches Local TMJ pain	>3	ID **muscle incoord.**
AGR	33	R	R>L	R>L	Night grinding	2	ID **TMJ laxity**
MLL	37	L	L	L	Neck stiffness Headaches	5	ID, discal **parafunction**
Mean or Total	27.1	3L, 5R	3L, 5R	3L, 5R		2-10	

L: left TMJ R: right TMJ

TABLE 2a. CLINICAL DATA : RANGES OF MOTIONS OF TMJ MOVEMENTS

CONTROL SUBJECTS

Subject (Code)	Opening (mm)	Deviation (Left/Right, ≥2mm)	Protrusion (mm)	Retraction (mm)	Left Translation (mm)	Right Translation (mm)
Normal Ranges	40 – 60	None	2-5	2	8 – 12	8 – 12
CML	45	0	5	2	11	11
JVL	42	0	4	2	7	9
CLL	55	0	2	4	7	7
JQL	51	0	8	2	10	7
DCL	54	0	4	2	6	7
JCL	63	0	4	8	12	15
MRL	60	0	6	3	6	10
CWL	52	0	2	6	10	10
LFL	58	0	2	4	8	13
MML	54	0	3	6	9	9
MEAN	53.4	0	4	3.9	8.6	9.8

TABLE 2b. CLINICAL DATA : RANGES OF MOTIONS OF TMJ MOVEMENTS.

TMJ ID SUBJECTS

Subject (Code)	Opening (mm)	Deviation (Left/Right, ≥2mm)	Protrusion (mm)	Retraction (mm)	Left Translation (mm)	Right Translation (mm)
Normal Ranges	40 - 60	None	2-5	2-3	8 - 12	8 - 12
GLL	55	L2	7	3	12	15
YCL	48	L	2	2	8	10
SKL	54	R,L	5	1	10	7
LAR	52	R	6	2	10	10
JBR	58	R	6	2	11	13
KMR	45	L	2	8	8	10
AGR	58	L	4	6	12	9
MLL	52	L	1	4	6	9
MEAN	**52.75**	**over 2mm**	**4.1**	**3.5**	**9.6**	**8.7**

Figure 8 : Picture of the Insertion techniques.

Figure 9 : Experimental set-up and testing material.

during incisive clenching and the temporalis during molar clenching, both being silent on opening (Travell & Simons, 1983).

The next procedure was to carefully tape and isolate each of the 4 wires and to fix the surface electrodes on the temporalis and masseter. At the same moment, the EMG signal from the four investigated muscle were tested and calibrated. Then, the EMG amplifier outputs for the four muscles were connected to the data acquisition box interface which was connected to a microcomputer (Compaq 386,16MHz) using a custom-made biomechanic data acquisition software (BIOAD, Lamontagne et al., 1989). A straight and confined testing chair, head stabilizer and visual feedback allowed us to minimize the effect of forward head posture on our results. For the resisted maximum opening, the head was immobilized with a frontal velcro harness and a cephalad support to limit any upward compensation (Figure 9). EMG signal, jaw kinematics and kinetics data were collected simultaneously. The temporalis and masseter muscles were registered by surface electrodes (Siver-silver, Meditrace, Ganagoque, Canada) (Figure 10) and the two heads of the LPM by intramuscular electrodes.

Conditions

The experimental procedure, individual session of approximately one hour, consisted of performing five static tasks: resting, resisted protraction, maximum voluntary contraction (MVC) in opening, molar clenching and incisor clenching and two dynamic tasks: open-close-clench cycle and gum chewing. These specific movements were chosen for various pertinent reasons: they exhibit an activity of the LPM's; some are known to cause ID; some are functional daily muscular patterns, some exhibit the clicking sounds; or have any relation with the rationale of this investigation.

The maximum voluntary contraction (MVC) of jaw opening consisted of a maximum jaw depression motion against the opening force transducer (Figure 11). This first task not only estimates the force of the motion, but also appraises the ILP recruitment and the contraction of the accessory jaw depressors, the hyoid and digastric muscles. The MVC were also used to normalize our data for the two muscles, ILP by resisted opening and the SLP by molar clench.

Maximum voluntary contraction (MVC) of jaw closing, in the molar and the incisor positions consisted of a clenching force on the closing bite force transducer (Figure 12 and 13). The resisted molar clenching tasks assesses the bite force and the loading of the condyle in a posterior position. It is pertinent to note that an inflamed and damaged cartilage joint surface will inhibit any strong muscular compression. Furthermore, incisal biting has been accused to cause TMJ internal derangement (Isberg, 1985; Wilkinson,1988; Bell, 1990).

Figure 10. Picture of a subject with surface electrodes on the masseter and the temporalis muscles.

Figure 11. Picture of a subject performing the MVC in opening.

The resting activities of both heads of the LPM, the masseter and the temporalis was then recorded in order to conclude on general resting muscular hyperactivity or postural tonus and to subtract it from other tasks in order to get the pure specific activity signal.

Active resistive protrusion was recorded while the subject was isometrically protracting his mandible against the experimenter's fist in order to isolated the translation arthrokinematic component of the opening TMJ and measured a specific function of the inferior head of the LPM (Figure 14).

A maximum open-close-clench cycle was performed (Figure 15) to assess the EMG activities of the two muscles while clicking (= ID) and to examine the dynamic patterns of the muscular interaction between the two heads of the LPM and the masseter and temporalis. In addition, before and after clicking, qualitative observations could be made.

Finally, a natural gum chewing condition was performed to appraised the coordination of the two LPM's with the masseter and the temporalis muscles simultaneously with the recording of joint clicking during functional mastication (Figure 15). This appraised a dynamic task including the disc reduction sound and muscular pattern interaction and coordination. Right and left sided were accepted as no instruction except usual chewing pattern were dictated. Most subject chewed on their affected side in order to reproduce a discal interference sound.

Standardized verbal instructions about the tasks procedures were given to each subject as well as cues to correct the situation when needed. All the movements were demonstrated by the investigators, then practice by the subject to a satisfactory level. Each static jaw condition were held for three seconds and repeated three (3) times. For the dynamic conditions each of the three trial lasted 5 seconds in order to capture one to three (3) full cycles for a total of nine cycles.

Figure 12. Picture of a subject performing the MVC in incisor clench.

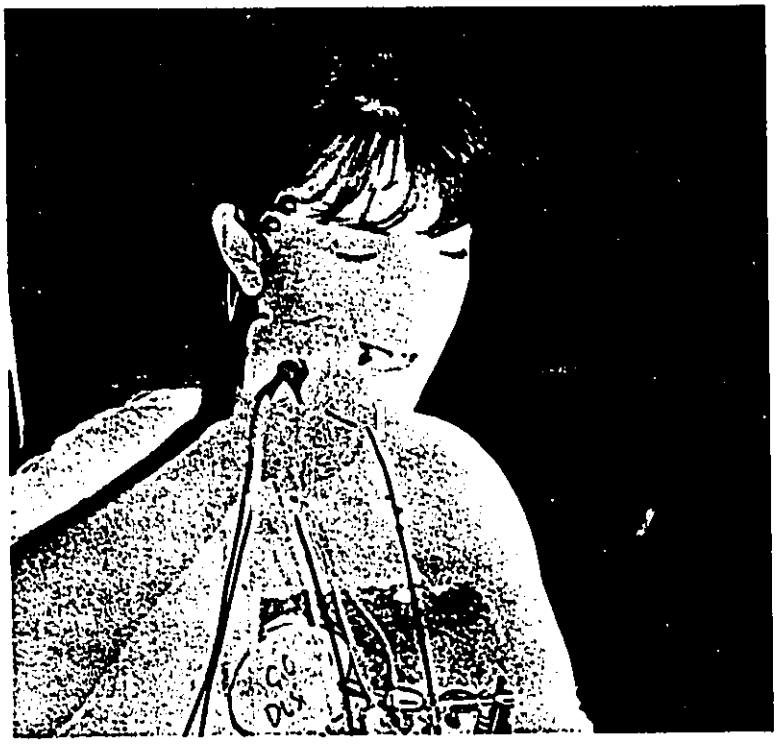

Figure 13. Picture of a subject performing the MVC in molar clench.

Figure 14. Picture of a subject performing the resisted protraction.

Figure 15. Picture of a subject performing the maximum open-close-clench cycle or the gum chewing.

DATA COLLECTION

For all three trials of each static or dynamic task, the EMG signal was recorded simultaneously with the clicking sound by microphone and the jaw kinematics by electrogoniometry for the dynamic conditions and with jaw kinetics by force transducers for the static tasks. A period of three seconds was used for static conditions and the best one second window was chosen for data analysis. A period of five seconds for the dynamic tasks was needed to capture two to three full cycles. The instrumentation used for the static tasks project were EMG and kinetics. For the dynamic study, EMG, kinetics, kinematics and sound recording data were obtained. Figure 16 depicts a schematic diagram of the apparatus used and the data acquisition system.

Electromyography

The muscle activity of the two heads of the LPM were recorded by means of bipolar fine-wire intramuscular electrodes. Bipolar surface EMG electrodes (pairs of silver-silver chloride, MEDI-TRACE) were placed 1 cm apart along the muscular fibres of the masseter and the temporalis. One surface electrode was fixed to the ipsilateral collar bone as the ground reference between the electrodes muscles insertions and the heart. Unilateral collection was performed as all the subjects involved in the study were diagnosed with a one sided disorder.

The raw EMG signals of all four muscles were amplified and filtered through a differential bioamplifier with adjustable high gain (High performance AC preamplifier GRASS P511). The frequency characteristics of the filter band width were carefully selected at 3, 10 or 30 to 100 or 300 to register the dominant frequency components of our myoelectric signal. and different gain (2000 to 10 000) were chosen in order to get to the best output signal. The EMG signal was then converted by analog-to-digital conversion and fed to a microcomputer (Compaq 386, 16MHz).

The data acquisition system allows for 16 configurable channels data acquisition inputs, therefore it enables to record the 4 EMG signals at 2000 Hz simultaneously with one sound, two jaw electrogoniometric signals and one force signal all at 100 Hz sampling frequency. This synchronisation of the kinematics signals was imperative to precisely define a jaw cycle within the EMG signal collected.

Kinetics

The three MVC in opening, molar and incisor clench were recorded through two custom-made force transducers. The forces were amplified by a custom-made high bioamplifier at low voltage

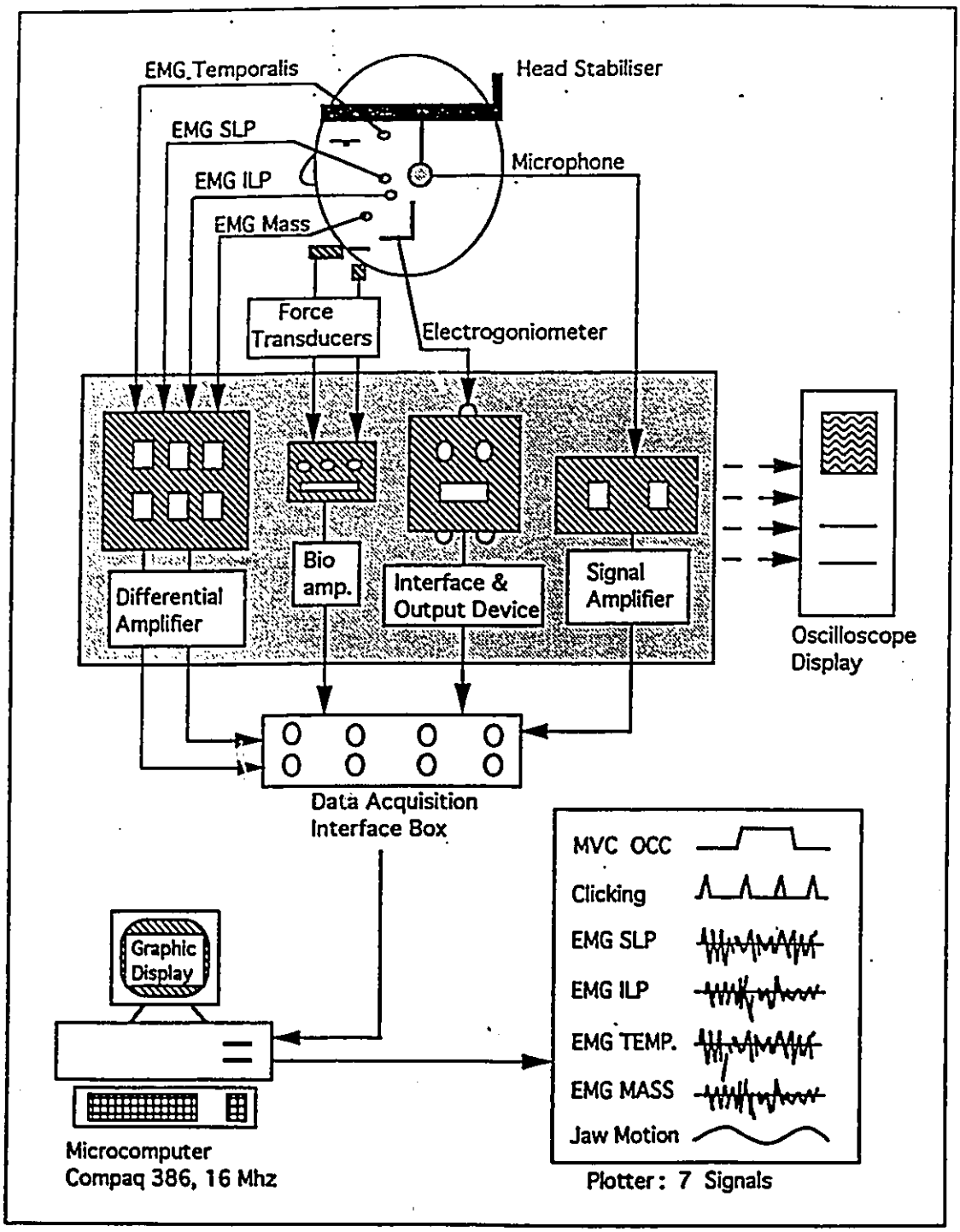

Figure 16. Schematic diagram of the apparatus used and the data acquisition system.

excitability and then recorded through the data acquisition system at a sampling frequency of 100 Hz and a variable gain of 1 or 10 that was automatically scaled in the analysis.

- The closing bite force transducer consisted of two steel beams mounted together (4.8mm inter-space) similar to the one described by Dechow & Carlson (1983). The full wheatstone bridge configuration of the strain gauges (high resistance 350 Ω) was glued along the inner surface of one beam to allow for direct strain measurement. Therefore the voltage output was proportional to the difference in strain between the two gauges. The biting surface of the fork was covered with a foamy self-adhesive material of minimum thickness to allow teeth comfort that was changed for every subject. The beams were placed between the ispilateral upper and lower first molar and between the upper and lower incisors. The total thickness of the device was 12 mm. The inter-beam space was measured after each trial to verify if any permanent bending of the metal had occur. Our first apparatus had one plate of 2mm stainless steel that became permanently bent on our pilot trials. Hence, we had to go for thicker beams and larger inter-space which remained constant through all the experimentation (Figure 17).

- The opening device consisted of an 8cm wide and 1 cm thick metal beam with a chin shaped plastic sitter at one end. The other end was solidly fixed with two large clamps to a horizontal beam part of the chair system. Another bridge configuration strain gage system was glued to the top surface of the beam near the clamps (Figure 18). Therefore the cephal stabilization of the head permitted to produce pure downward force with the chin causing the metal beam to flex. The difference of voltage produced was amplified and collected through an A/D conversion box before entering the microcomputer.

- Calibration

The calibration of the two devices was done by loading known weights on the pressure beams and recording through an amplifier the voltage on the oscilloscope and on the computer. Weights used for the opening device were 0; 1; 3; 5; 10; 19 kilos. For the closing strain gage, the weights used were 0; 3,75; 13,75; 23,75 ;33,75; 53,75 in kilos. The gain of the amplifier was 60 (1000 at a scale of 300μV) (Figure 19). Three trials of six upgrading and downgrading weights comparable with forces exerted by the jaw were used to calibrate the two force apparatus. In the literature, female molar and incisive closing bite force were reported to be of 22 kg and 11 kg respectively (Helkimo et al., 1976). No data was reported for opening maximum force but calibration was made up to higher values than expected.

From these calibrations procedures, a linear relation between the load and the deflexion voltage was obtained. Two regression equations were computed:

- the opening force one:

Y (Newton) = 56.66413 X (data voltage) + b (-0.68749),

the standard error of the Y estimate being 0.415077 and the R square being 0.999517.

- and the closing force one:

Y (Newton) = 627.7142 (volts) + b (0)

the standard error of Y estimates being 1.23597 and the R square value of 0.990081.

Electrogoniometry

A digital-based system using a flexible electrogoniometer (Penny and Gilles, BIOMETRICS) was used to record jaw displacement in the sagittal and frontal plane during dynamic jaw opening and closing. The electrogoniometer consists of a central strain gauge encased in a flexible steel strip surrounded by a spring with two end plates for attachment to the skin. The consequences of polycentric joints and linear skin movements are avoided; therefore, this planar device does not require a specific centre of rotation. It allows the simultaneous and independent measurement of joint rotation in two axes thus providing two analogue output signals representing vertical and lateral translations of the mandible. The measuring range of the goniometer is of +/- 180 degrees with an infinite resolution. For the purpose of our study, we used a 100 Hz sampling frequency in our data acquisition set-up and a total of 30 degrees sagittal and 10 degrees frontal range of motion. Calibration was integrated within the Biometric system: 1 volt = 90°; positive reading correspond to the opening period and negative reading to the closing or jaw elevation period.

The two pieces were attached with a double sided adhesive tape to the temporal/frontal bone and onto the mandible (Figure 20). The wires from the apparatus were restrained to the head and neck using tape in an unobtrusive manner for jaw motion. In this particular study, the purpose was to correlate movement direction and the end of range of each cycle with EMG signals in order to isolate and differentiate between opening and closing motion, simply to recognize precisely to which part of the cycle the EMG and microphone signal correspond.

Microphone

For our study, clicking was detected by a sensitive contact microphone fixed directly onto the skin at the joint line level and secure with tape. Correct placement was defined by the investigators after palpation and reproducibility of the sound. The microphone was connected to an adjustable signal amplifier which fed into our analogue to digital (A/D) conversion interface box, transforming decibels into millivolt. For each clicking sound, a difference of potential was produced and

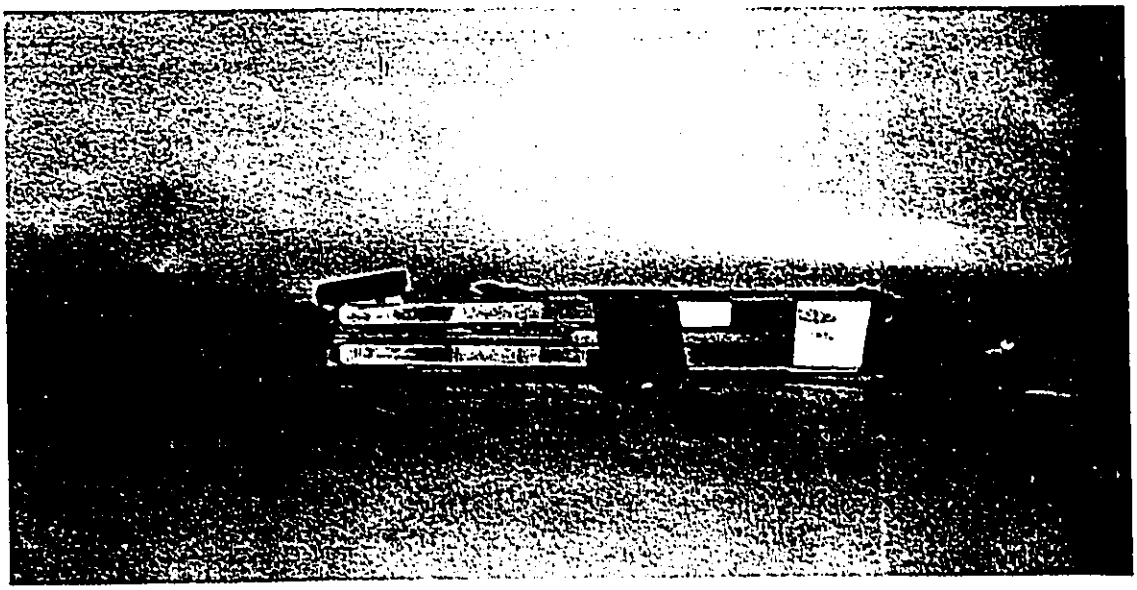

Figure 17. Custom-made closing bite force transducer.

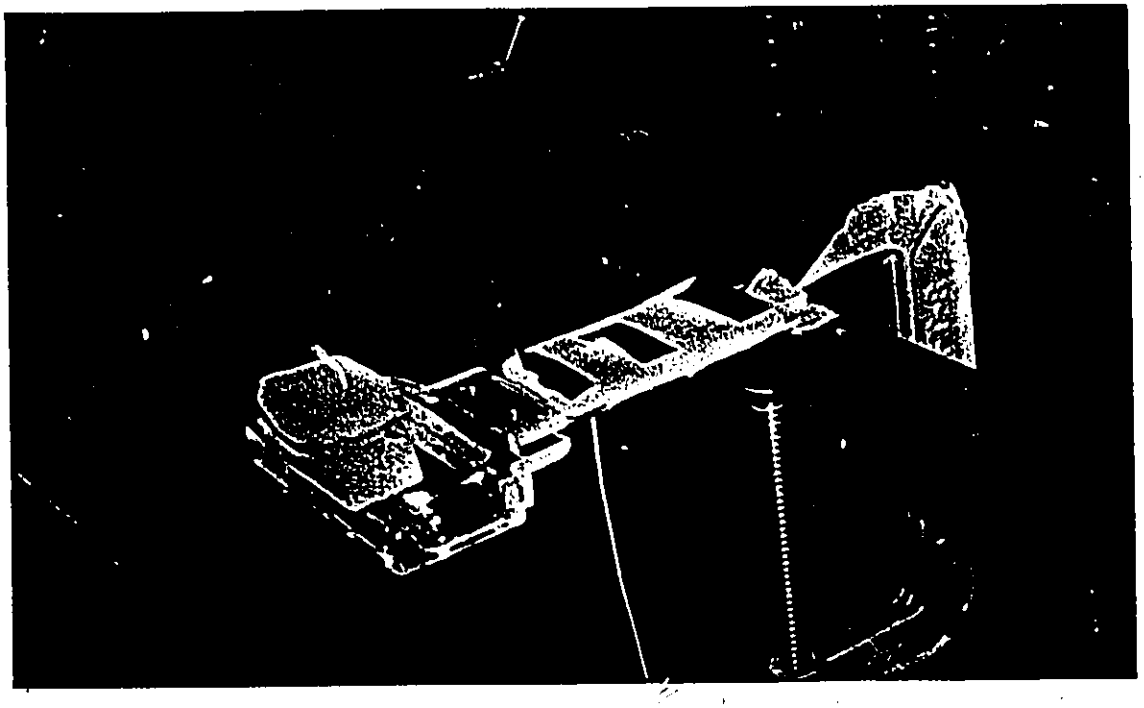

Figure 18. Custom-made opening force transducer.

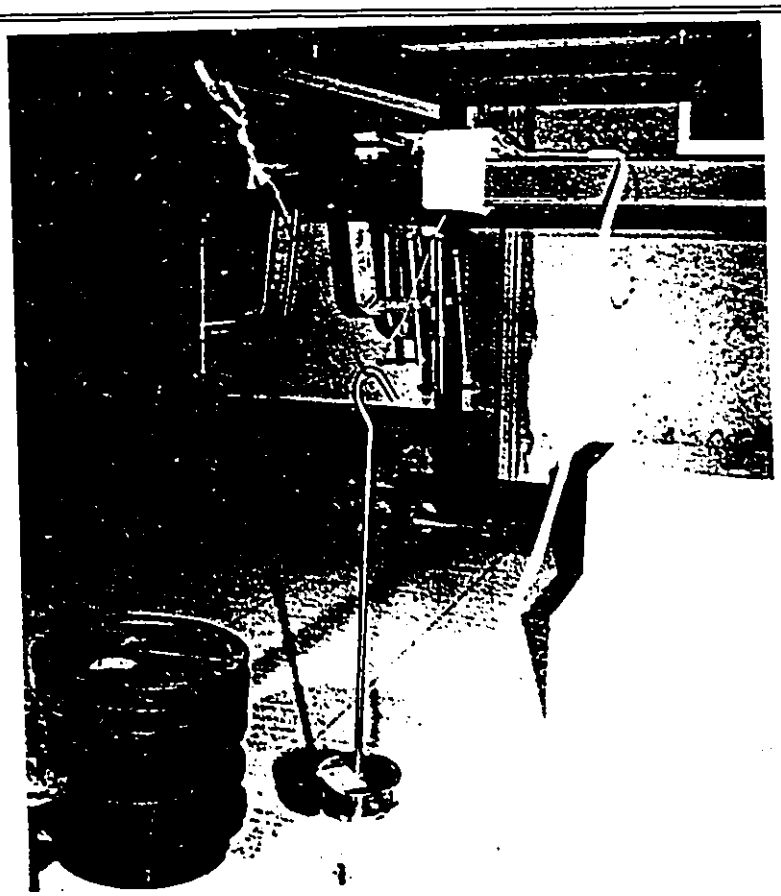

Figure 19. Calibration procedures of the closing bite force transducer.

Figure 20. Electrogoniometer on a subject.

re-amplified as needed through our data acquisition system BIOAD (Lamontagne , M. Bradley, D. & Lemaire, E. D., 1989).

No specific calibration was needed as the position in the cycle and its effect on the myoelectric signal was the factor needed. The intensity of the sound was not important as long as it could be traced. The amplified microphone signal was biased by mean procedure and could be easily detected graphically with the EMG recordings on the computer screen for later measurements and analysis.

The first or opening click was considered as the disc reduction sound and the second or closing click determined the disc re-subluxation occurrence. In other words, between the first and the second click, the disc/condyle relationship is considered normal, but after the closing sound and before the jaw clicks on reopening we have a pathological situation where the disc is anteriorly displaced. The closing click was sometimes so soft that it could not be recorded even at maximum amplification but the opening click could always be registered. The closing click position could sometimes be interpreted with obvious variations of the goniometer signals.

DATA REDUCTION AND ANALYSIS

All signals were analyzed on a microcomputer (Compaq 386, 16MHz), through a custom-made biomechanics software package (BIOPROC, Lamontagne, Bradley & Lemaire, 1989) which is fully compatible with the data acquisition software (BIOAD). This section is divided into two parts: The analysis of the static conditions along with the isometric forces; and the analysis of the dynamic conditions.

Statistical and Descriptive Analysis of the Static Conditions

Two sets of measures were computed from the five static conditions: the integrated LE EMG of the four muscles for the five tasks and the isometric absolute force values from the three MVC tasks. These were done for each subjects of both groups.

- EMG Data Reduction

For the five static conditions (rest, resisted protraction, MVC in opening, MVC in molar clench and MVC in incisor clench), for each three trials and for the four muscles, we digitally filtered and processed the EMG signals with an A/C filter at 10Hz and the bias was removed by mean. The second step consisted of scaling (in μV) each myoelectric signal by its own gain factor obtained from each

channel of the Grass amplifier. The raw EMG signal was converted to a linear envelope EMG (LEEMG) signal which consisted of a full wave rectification and then filtered with a fourth order, dual passes, critically dampened low-pass filter with a cut-off frequency of 6Hz. Each trial was time normalized in percentage in order to average the three trials per muscle per conditions per subject (n=3x4x5x19) and to be able to do inter-subjects comparison. The one second window that had the most constant EMG signal was then selected by viewing the scaled and smoothed signal. For the three MVC's tasks, the same processing techniques were undertaken but more attention was directed towards choosing the one second time interval based upon the maximum force contraction. From that one second window, the integration of the area under the curve was computed to obtain the integrated LE EMG. The integrated LE EMG of the resting condition was then subtracted to the integrated LE EMG of the 4 other static tasks in order to obtain the net integrated LE EMG.

Averages were computed across trials for each subject per condition per muscle to obtain a mean integrated LE EMG value in μV. Grand Ensemble Averages (GEA) were then computed across subjects for the two groups for the 4 muscles and the 5 conditions to reduce the variance of EMG recordings and to compare between the groups. Second order GEA were also calculated over conditions and then over muscles but were not found meaningful and were not used.

- Force Data Reduction

The Isometric force signals were processed for the three MVC conditions. The bias was removed by factor procedure to level the signal at zero. The mean force value of the selected one second was measured for each trial, muscle and condition per subject. For each three MVC conditions the mean forces value (in voltage) was inserted in the regression equation from the calibration procedures and converted in Newton. From the mean force value, GEA were computed across subjects per group to compare the absolute forces exerted by the TMJ ID group and the control one for each MVC condition.

- Statistical Design

The two groups were characterized as random samples of two specific populations: TMJ disorders and matching (sex and age) surrounding volunteers of the general population. Statistical analyses were conducted on the dependent variable mean integrated LE EMG expressed in μV for each four muscles for the five static conditions. General linear model ANOVA (four 2x5 with post-hoc Fishers tests) were contrasted with twenty simple one way ANOVA. Statistical analyses were also conducted on the three isometric forces expressed in Newton using one 2x3 ANOVA model and one-

way ANOVA. All tests were done through NCSS software package using a significant level of $\alpha \leq 0.05$ to determine if the two groups differ on their muscle activity for the five conditions and on their opening and closing bite forces. The independent variables were TMJ ID and control groups. Two tailed tests were used as the TMJ ID group might present with hyperactivity or hypoactivity, lower or higher forces, compared to the control group. The simple or one way ANOVA comparing the two groups means for each muscle in each condition separately seem to be the best way to draw the information we were looking for without fading the contrasts and loosing the meaning. It was well understood that some conditions and some muscles do interact but sometimes in opposite directions, therefore cancelling the effect of interest. The factors are considered independent and should not be melted together. The only effect of interest being studied was the muscular activities and forces generated by the two groups.

Independent scores and normality of distribution were tested positive. Although, we had large standard deviations, probably due to the large subject inter-variability[44] and the influence of many factors, the homogeneity of variance between samples was valid most of the time (tested within the simple ANOVA procedures with NCSS). If not, a corrected and more-conservative, p value was provided by NCSS in cases of unequal variance and was used to assess significance. This logarithmic transformation to normalize the data is necessary before concluding on the inferential value of our results. However, our sample was so small, that generalization was not possible.

Statistical and Descriptive Analysis of Dynamic Conditions

Theoretically, the presence of myoelectric signals demonstrates if a muscle is active or not and how active it is in comparison with a reference value such as the MVC or a peak value. In this investigation, the EMG signals of the open-close-clench and gum chewing cycles were computed into four EMG data treatments.

The raw EMG was filtered with a high-pass filter (Butherworth, dual passes) at 10Hz. The bias was removed by mean using the BIOPROC software (Lamontagne et al. 1989), then scaled by a factor obtained from the different Grass amplification gain for each muscle. The best cycles were selected for processing by viewing the full scaled signal (5 seconds). For the three MVCs tasks, the same processing techniques were undertaken but the window used for processing was selected from the maximum kinetic signal registered. The EMG signal was full wave rectified and filtered with a fourth order, dual passes, critically dampened low-pass filter (cutoff frequency =6Hz), producing a LE EMG data. The area under the curve of the LE EMG was computed to obtain the integrated LE EMG. The average integrated value of the rest condition was subtracted to obtain the net integrated LE EMG

provided by the specific movement. From the EMG signal, four different dependant variables were computed during the dynamic motions to compare differences between the two samples.

1- *Integrated LE EMG normalized by 100% MVC and by phase*. Each open-close-clench cycle was divided in three phases: open, close and clench. Each phase was normalized by time in percentage. For each phase, the integrated LE EMG for each muscle was computed and then normalized by the integrated LE EMG of the specific MVC (100%). The open phases were normalized by the MVC obtained in opening. The close and clench phases were normalized in percentage of MVC obtained in molar clench. For the mandibular muscles, the rationale is confirmed for the EMG protocol using maximal isometric contraction as the normalization guideline (Miller & Brown, 1974; Jankelson, 1990).

Practically, these values are the extend to what each muscle is working during a phase of the OC : cycle in relation to its corresponding MVC based on their respective integrated LE EMG. In other words, these values show how much both groups are using each muscle during a phase. GEA were computed across subjects for the four muscles for each group.

2- *Integrated LE EMG normalized by 100% MVC by primary functions*. The three best trials of the gum chewing cycle were first normalized by time in percentage. The integrated LE EMG were computed for each muscle and each subject. The integrated LE EMG of the resting condition was subtracted. The resulting μV values of the masseter muscles were normalized by the integrated LE EMG from the MVC obtained in incisor clenching (the superficial masseter acts primarily in a protracted clench). For the temporalis and SLP muscle, the resulting integrated LE EMG of the gum chewing were normalized by the MVC obtained in molar clench. And for the ILP muscle, the normalization of the gum integrated LE EMG was done by the MVC obtained in resisted opening.

Hence, each EMG recording is expressed as an amplitude percentage of these 100% MVC values. This muscular contribution data gives information relating to what extend (% of MVC) each muscle contributes to perform functional mastication, again comparing TMJ ID subjects and controls.

It is pertinent to mention that normalization is usually done to facilitate comparison between subjects and groups to eliminate inter-subject variability. But as stated by Yang & Winter (1984), normalization by 100% MVC actually increases the inter-subject variability secondary to fatiguability, co-contraction, non-linearity of EMG-force relationship which definitely limits the interpretation of the results. Therefore, normalization by peak was conducted as it reduces drastically the inter-subject variability and improves the sensitivity of EMG as a measuring tool (Yang & Winter, 1984).

3- *Integrated LE EMG normalized by peak*. The whole gum chewing signal was normalized by time in percentage. The LE EMG for each muscle was computed and then normalized by peak amplitude. This normalization differs from the first ones as we are normalizing a dynamic cycle by a dynamic value within the cycle permitting better between groups comparison.

For those three families of dependent variables (all expressed in percentage of the MVC), ensemble averages were computed through subjects of the two groups for the four muscles. To compare means of the two samples, general linear model ANOVA (simple or one-way) were computed through NCSS software package for statistical analysis using a significant level of $\alpha \leq 0.05$ and two tailed testing. The independent variables are TMJ ID and control groups. The simple or one way ANOVA comparing the two groups means for each muscle in each condition separately seemed to be the best way to draw the information we were looking for without fading the contrasts and loosing the meaning. It was well understood that some conditions and some muscles do interact but sometimes in opposite directions, therefore cancelling the effect of interest. The factors are considered independent and should not be melted together. The only effect of interest being studied were the muscular activities.

Knowing that in the practical application of EMG, the pattern of the ensemble LE EMG is often a more important consideration that its amplitude, descriptive and qualitative analysis were also done.

4- *Descriptive analysis* of the gum chewing cycles were completed to withdraw qualitative characteristics of the EMG signals of the four muscles between the two samples. The whole gum chewing raw signal was normalized by time in percentage. The LE EMG for each muscle were computed and normalized by peak amplitude. Descriptive analysis of simple subjects and group average are presented. Surely, these descriptive features are very adequate and pertinent for dynamic tasks analysis and are expressed in similarities and dissimilarities between groups and muscles.

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APPENDIX B

QUESTIONNAIRES, CONSENT FORM, PATIENT INFORMATION

AND

CERTIFICATION OF THESIS PROPOSAL BY THE ETHICS COMMITTEE

FOR THE PROJECT ENTITLED

THE LATERAL PTERYGOID AND OTHER MASTICATORY MUSCLES ACTIVITY IN TEMPOROMANDIBULAR JOINT INTERNAL DERANGEMENT, AND CONTROL SUBJECTS DURING DYNAMIC AND STATIC CONDITIONS

UNIVERSITÉ D'OTTAWA
UNIVERSITY OF OTTAWA

FACULTÉ DES SCIENCES DE LA SANTÉ
FACULTY OF HEALTH SCIENCES

PART II:CONSENT FORM

Participation Consent to a Biomechanical Analysis of the Temporomandibular Joint (TMJ)

Whenever a research project is undertaken with human participants, the Human Research Ethics Committee of the University of Ottawa (Chairman of the H.R.E.C.,Faculty of Health Science: Dr. M.A. Loyer 613-787-6707) requires that participants provide in writing a proof of their consent. This ensures that participants are aware of the nature of this study, and that they are fully informed about their rights.

INVESTIGATORS: C. M. Lafrenière (pht BSc. MCPA) and Dr. M. Lamontagne PhD
tel.# (613) 564-9232/9105

Please read this form and sign it if you are willing to participate in the study described below.

I am here as a volunteer and I understand that I AM FREE TO WITHDRAW this consent and to discontinue my participation at any time, even during the testing period, without penalty or discrimination.

MY PRIVACY will be protected in the following manner: All research data obtained from me in the course of this study will be kept confidential and will be accessible only to the principal investigators. Should the study be published, my identity will not be released.

I UNDERSTAND the implications required for my participation in the present EMG study during the experimental protocol and the possible physical discomforts involved explained in the letter of information. These were, possible local skin and muscle tenderness, jaw stiffness, and rarely haematoma and local pain.

TESTING PROCEDURES

You will have to be sitting straight on a chair with your forehead surrounded by a comfortable head stabilizer. You will be asked to wear regular clothes excluding a turtle neck and to tie your hair so it doesn't get in contact with the cheek area.

The experiment will not involve future discomfort or stress of any kind. The principal investigators will be with you constantly to fix the apparatus, instruct you and collect the different information. The investigators are prepared to provide assistance should the need arise.

In signing this consent form you acknowledge that you have read and understood the above statements as well as the letter of information. You enter the biomechanical investigation willingly and you may withdraw AT ANY TIME without penalty or discrimination. Any questions about the procedures used in this experiment are welcome.

Under confidentiality, you will receive, freely, the results of our study once analyzed as a appreciation sign.

Printed name of Volunteer: _____

Signature of Volunteer and Date: _____

Signature of Witness: _____

...2/2

ÉCOLE DES SCIENCES DE L'ACTIVITÉ PHYSIQUE
SCHOOL OF HUMAN KINETICS

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PART I: LETTER OF INFORMATION

Participation Consent to a Biomechanical Analysis of the Temporomandibular Joint (TMJ)

INVESTIGATORS: C. M. Lafrenière (pht BSc. MCPA) and Dr. M. Lamontagne PhD
tel.# (613) 564-9232/9105
Chairman of the Human Research Ethics Committee of University of Ottawa:
Dr M. A. Loyer 1-613-787-6707

INFORMATION ABOUT THE STUDY:

The purpose of this study is to examine the muscle activity of four (4) jaw muscles of normal and abnormal jaw joints while measuring motion and joint sounds. One session of approximately thirty (30) minutes will be required for the experimentation.

This research will help dentists, doctors, physiotherapists and other clinicians to better understand and treat TMJ problems. Which should improve the quality of life of the increasing number of mistreated TMJ sufferers.

The only discomfort expected are the insertion of two wires in the two deep muscles located in the cheek area that will become painless with the use of a special cream. The possible risks and inconveniences consist of slight stiffness in the jaws and muscle, skin tenderness and less likely to happen, a local haematoma and a local pain for a maximum of 2 to 3 days. Infection and other complications have not been reported.

EXPERIMENTAL PROCEDURES:

In the present study, you are asked to read and sign the consent form if you are willing to participate. Moreover, you will be asked to fill out a simple questionnaire to make sure you fulfil the criteria to enter the study. One hour prior to the testing, you may apply a analgesic cream on your cheek (area will be shown to you) for the insertion to be completely painless.

Before the testing, you will be asked to perform specific jaw movements in order to familiarise you with the various, below mentioned, conditions required for the study.

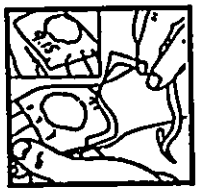
The next step will be to insert the two intramuscular wire electrodes and to stick the two surface ones on your jaw muscles (cheek surface) to register muscle activity. We will then apply a comfortable flexible steel strip inside a plastic box and a small microphone on your cheek skin with medical tape to register motion and joint sounds.

The research protocol will start by determining your maximal jaw strength. You will be asked to perform maximum resisted effort in opening and clenching against a cushioned metal instrument that measures forces. This test takes approximately five (5) minutes including the warm-up period.

Subsequently, you will be asked to perform the following jaw movements: 1- resting your jaw (maximum relaxation), 2- maximum opening-close-clench cycle, 3- forward jaw motion, 4- front teeth biting and 5- gum chewing. The testing will consist of performing the different conditions 3 times each which takes approximately 10 minutes. During the tasks, your muscles will generate a small electrical signal that will be recorded by the four (4) electrodes, you will give us a message, you won't feel anything. Also for each condition, jaw movement will be registered by the plastic piece attached to your cheek and clicking sounds will be recorded with a small microphone.

You may refuse any of the above conditions ...1/2

e) Remove the paper frame from the dressing. The time the cream was put on may be marked on the dressing if you wish.



5. Wash your hands right away so the cream does not get in your eyes.

6. Do not remove the dressing unless your doctor tells you. The 'freezing' effect lasts about 1 hour *after* the dressing and cream are removed. In most cases the dressing and cream will be removed at your doctor's office.

Special Precautions

- *EMLA® should be used only on healthy, unbroken skin.* Do not use it on areas with an infection, skin rash, cuts, scrapes, or wounds. These can change the way EMLA® goes through the skin. Check with your doctor ahead of time if any of these problems are present when you are to use the cream.

- EMLA® should be kept at room temperature. Do not put it in the fridge or freezer.

- KEEP EMLA® OUT OF THE REACH OF CHILDREN. Although you may not think of creams as "medicine", the ingredients of EMLA® can be poisonous if swallowed in large amounts. If possible, keep the tube in a locked place.

- Do not put EMLA® in or near the eyes. If you accidentally get EMLA® in the eyes, rinse them well with clear water.

- A mild reaction (slight puffiness, initial burning, itching or change in skin colour) may occur on the area EMLA® is used. These are normal and will go away in a short while.

- Other reactions to EMLA® are EXTREMELY RARE. However, *check with your doctor right away* if you notice any of the following after the cream is applied: skin rash or hives, swelling in the neck or throat area, or trouble breathing. These may be an "allergic reaction" to local anesthetics.

- Other side effects which cannot be predicted may occur in rare cases. Check with your doctor if you notice any unusual effects after using EMLA®.

EMLA® Cream	1 x 30 gm	078
EMLA® with 20 Tegaderm® dressings	10 x 5 gm	077
EMLA® with 2 Tegaderm® dressings	1 x 5 gm	076

ASTRA

Astra Pharma Inc.
1004 Middlegate Road
MISSISSAUGA, Ontario
L4Y 1M4

02/91
J40

EMLA®

Lidocaine and Prilocaine

Consumer Information

Astra Pharma Inc.

Why EMLA® is used

EMLA® is a local anesthetic *(an-ess-thet-ik)* cream. Local anesthetics are used on the surface of the body to cause a loss of feeling - or "freezing" - in the area they are applied.

EMLA® contains two local anesthetics, called *lidocaine* and *prilocaine*. Its most common use is to freeze the skin before a blood sample is taken, or before a certain procedure is done by a doctor.

Doctors may suggest EMLA® for other reasons. Be sure to ask your doctor if you are unclear about why you were given this cream.

Read this leaflet carefully. It has been prepared by the makers of EMLA® to help you get the most benefit from this cream. If you have any questions or concerns after reading the leaflet, talk to you doctor or pharmacist.

Before you apply EMLA®

Certain people should not use EMLA®. Before you apply the cream, be sure you've told your doctor:

- about *all* other health problems you have now, and have had in the past;
- about *all* other medicines you take by mouth, or use on your skin;
- if you are pregnant or breast feeding;
- if you have ever had a bad, unusual or allergic reaction to:
  - lidocaine or prilocaine;
  - any other "freezing" or local anesthetic medicine;
  - any other medicine ending with *caine*.
- if you think you may be sensitive or allergic to other ingredients in this cream. *[see EMLA® Ingredients]*

EMLA® Ingredients: Most medicines contain more than their active ingredients. These are needed to keep the medicine in a form you can use. For the information of people with certain allergies, the following is a list of all ingredients in EMLA® cream. Check with your doctor if you think you might be sensitive to any of these items: lidocaine, prilocaine, polyoxyethylene hydrogenated castor oil, carboxypolymethylene, sodium hydroxide.

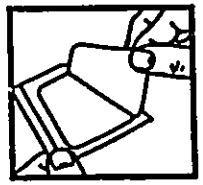
How to use EMLA® cream

- In order for EMLA® to work, it should be used between 1 and 5 hours before you see the doctor. At least 1 hour is needed to cause a loss of feeling in the area being treated. Less than 5 hours is needed to make sure the area is still 'frozen' when the procedure is done. Check with your doctor ahead of time if you have any questions about when you should apply EMLA®.
- EMLA® should be applied the following way:

1. Wipe the area clean. Dry the skin gently and completely.
2. EMLA® comes with a protective seal on the tube opening. This can be opened by firmly pressing the pointed end of the white cap into the seal.
3. Squeeze a thick layer of EMLA® onto the area which needs freezing. That area should be completely white with cream. As a *rough* guideline, put 1/2 of a 5 gram tube on an area the size of a quarter.
4. Cover the cream with an 'occlusive dressing' found in the box. An occlusive dressing is one which completely seals the area and does not let air in. This is needed so the freezing will go deep in the skin. The dressing also protects clothing and prevents cream from getting in the eyes by accident. The cream will spread out to cover an area a little larger than the $1.00 coin ("loonie").

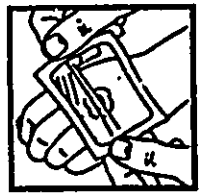
To cover the cream with occlusive dressing:

a) Remove the center piece of the dressing.



b) Peel the paper liner from the paper-framed dressing.

c) Center the dressing over the cream. *Do not spread the cream or rub it into the skin.* Try not to touch the cream with your hands.



d) Press your finger firmly around all edges of the dressing to keep it in place. It is equally important to make sure there are no spots where the cream can leak out.

Faculty of Health Sciences

School of Human Kinetics

HEALTH SCREENING QUESTIONNAIRE

1.	Has a physician ever said you have heart trouble?	Yes	No
2.	Do you frequently have pains in your heart and chest?	Yes	No
3.	Do you often feel faint or have spells of severe dizziness?	Yes	No
4.	Has a physician ever said that your blood pressure was too high?	Yes	No
5.	Do you suffer from any respiratory tract problem such as chronic bronchitis, asthma or emphysema?	Yes	No
6.	Have you ever had or are you now suffering from any nervous disorder?	Yes	No
7.	Do you suffer from any bone or joint problem which either has been or may be irritated by an exercise session?	Yes	No
8.	Do you know of a valid medical reason why you should not be involved in either a regular exercise program or an exercise testing session?	Yes	No
9.	At present, are you taking medication for blood pressure?	Yes	No

If yes, please specify:

Reason:_____

Name:_____

Dosage:_____

10.	At present, are you taking any other type of medication, whether they are prescribed or "over the counter"?	Yes	No

If yes, please specify:

Reason:_____

Name:_____

Dosage:_____

Signature: _____

Date: _____

From: Jetté, M., Quenneville, J., Sidney, K., Fitness Testing and Counselling in Health Promotion, Canadian Journal of Sport Sciences, 1992.

SUBJECT QUESTIONNAIRE

Y N 1- Did you ever had a clicking sound or other noises in your jaw, such as popping, grinding etc.?

Y N 2- Do you have any discomfort, pain or stiffness in any movement of your mouth and jaw?

Y N 3- Do you have at least 28 natural teeth in your mouth?
If not, which ones? _____

Y N 4- Do you have missing back teeth? How many? _____

Y N 5- Does your bite feel comfortable, does it close equally?

Y N 6- Do you grind or clench your teeth?

Y N 7- Have you had any major dental work other than filings for cavities?
If positive, describe: _____
_____
_____

Y N 8- Have you ever had any orthodontic work done on your teeth? (braces, retainers) and/or do you wear any mouth piece, splint, bite plate or appliance ?
If positive, describe it with its goal and effect:
_____
_____
_____
_____

Y N 9- Have ever had extensive dental crowns and bridges?

Y N 10- Do you wear a removable partial denture?

Y N 11- Have you ever been hospitalized for a neck of a head injury?

Y N 12- Do you have any current head, neck or jaw discomfort? (i.e. cervical pains, frequent headaches, etc.)

Y N 13- Have you ever been diagnosed as having any neuro-muscular disorder? (i.e. muscular dysthrophy, multiple sclerosis, facial palsy, etc.)

Y N 14- Are you presently taking any prescription or non-prescription medications?
If positive, describe it with its goal and effect:
_____
_____

Y N 15- Have you ever been treated for problems of your jaw joints or for facial problems?
If positive, please describe: _____
_____
_____
_____

Y N 16- Do you, or any member of your family, suffer from arthritis?

Y N 17- Do you have any other health problem?
If positive, describe: _____
_____
_____
_____

Y N 18- Are you allergic to any topical anesthetics or analgesics, especially prilocaine and lidocaine?
If positive, describe: _____
_____

SUBJECT CODE: _____

EMLA post-test questionnaire

The purpose of this questionnaire is to gather information on the analgesic cream used before the needle insertion.

1- How did you feel during the application of the cream and dressing?

..... fine
..... discomfort
..... itchy/cold/hot
..... sensitive
..... other

describe:.....

2- Did you have any specific sensations during the analgesic period? (while the cream was on your cheek)

..... no
..... yes, numbness
..... yes, itchy, discomfort
..... yes, sensitive
..... other,

describe: .....

3- what did you fell on insertion of the needles?

..... no pain
..... slight pain
..... moderate pain
..... severe pain

Please score your pain on this scale:

0.....10

0: no sensation
10: worst imaginable pain

4- How did you fell during the testing period? (while you were doing your jaw movements).

..... no pain
..... slight pain
..... moderate pain
..... severe pain

Please score your pain on this scale:

0.....10

0: no sensation
10: worst imaginable pain

5- How did you feel when we pulled the wires out ?

..... no pain
..... slight pain
..... moderate pain
..... severe pain

Please score your pain on this scale:

0.....10

0: no sensation
10: worst imaginable pain

Any other comments?

PRINT NAME : .....

EMLA post-test questionnaire

TO BE FILLED BY THE EXAMINERS

A- Oral comments during insertion procedures:

B- Facial expression during insertion procedures:

C- Was repetitive insertion trail necessary Yes..... No.....

D- Local reactions observed:

E- Analgesic time .....minutes.

F- Testing time .....minutes.

G- 48 hours reaction by phone:

NAME Of SUBJECT .....

UNIVERSITÉ D'OTTAWA
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FACULTÉ DES SCIENCES DE LA SANTÉ
FACULTY OF HEALTH SCIENCES

July 29 1992

Miss Chantal Lafreniere
School of Human Kinetics
Montpetit Hall
University of Ottawa
INTRA

RE: Your project entitled: "Temporomandibular Joint Dysfunction : The Role of the Lateral Pterygoid Muscles".

Dear Chantal,

It is my pleasure to inform you that the Faculty of Health Sciences, Human Research Ethics Committee, after study of the documentation provided, concluded that your project met the appropriate standards of ethical acceptability and falls within Category 1A.

I hereby attach a copy of the certificate of clearance granted by the University Human Research Ethics Committee and the original has been sent to them as well.

This certificate is valid for a period of one year from the time of issuance. I would also like to remind you that, in accordance with the policies of the UHREC, it is your responsibility to notify the Committee of any major changes in this project.

On behalf of the Committee, I wish you success in your project.

Sincerely,

Marie A Loyer

Marie-des-Anges Loyer, Ph.D
Chair
Human Research Ethics Committee

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CERTIFICATION OF INSTITUTIONAL HUMAN RESEARCH ETHICS COMMITTEE
FACULTY OF HEALTH SCIENCES

This is to certify that the Institutional Human Research Ethics Review Committee of the Faculty of Health Sciences has examined the research proposal by Chantal Lafreniere for the project entitled "Temporomandibular Joint Dysfunction : The Role of the Lateral Pterygoid Muscles" and concludes that, in all respects, in the proposed research protocol meets the appropriate standards of ethical acceptability, at a Category 1A level.

MEMBERS OF THE COMMITTEE

Name (Optional)	*Position held*	*Department of discipline*
Richard Bouchard	*Student*	*Human Kinetics*
Anne Carswell	*Professor*	*Programme of Occupational Therapy*
Jean Harvey	*Vice-Dean*	*Faculty of Health Sciences*
Marie Loyer	*Chair*	*Human Research Ethics Committee & School of Nursing*
Joan McComas	*Professor*	*Programme of Physiotherapy*
Jacqueline Neatby	*Member-at-Large*	
James Thoden	*Professor*	*Human Kinetics*

SIGNATURE

29-07-92 Marie des Anges Loyer
Date *Committee Chairperson - Marie des Anges Loyer, Ph.D*

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