# A PROVOCATION STUDY OF THE EARLY DEVELOPMENT OF NECK DYSFUNCTION DUE TO UNILATERAL OCCLUSAL CONTACT

BY

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# ABSTRACT

A number of reports have stressed, with little basis in research, the importance of neck involvement in patients with temporomandibular disorders. This study examined the consequences of a seven to twelve day period of unilateral occlusal contact, in eight healthy subjects. The subjects wore occlusal splints built-up on one side. The subjects were examined on two days prior to the splint wear period, and on two days at the conclusion of the splint wear period, with respect to their head posture in the frontal plane, range of motion of the head and neck in side flexion, and the peak activity of their anterior temporalis and sternocleidomastoid muscles in several clenching and head moving tasks.

The results indicate that:

1. Unilateral posterior occlusal contact did not produce any significant signs and symptoms of TMD in the experimental period.
2. There were no significant changes in the head posture of the subjects.
3. Unilateral occlusal contact did alter the head and neck range of motion in side flexion by increasing the asymmetry in the range of motion to the two sides. Six of the subjects showed a reduced range of motion to the non-splint side which is consistent with tightness of the sternocleidomastoid on the splint side.

4. Unilateral occlusal contact reduced the EMG activity of the anterior temporalis on the non-splint side in both maximum clenching tasks and in partial clenching tasks (20 - 25% of maximum).

5. The sternocleidomastoid muscles were active in maximum clenching tasks and demonstrated activity in some subjects during partial clenching (20 - 25% of maximum). The period of unilateral occlusal contact reduced the activity of the non-contact side sternocleidomastoid.

6. In the head moving tasks, some subjects demonstrated changes in activity levels of the sternocleidomastoid muscles which may have been caused by dysfunction in the neck produced by the asymmetric occlusal contact.

The results supported the concept that asymmetric occlusal contact could produce early dysfunction of the neck muscles in a short period of time, indicating a functional relationship between the masticatory and neck muscles.

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Finally to Lesa, my wife, friend, and physiotherapy consultant, whose encouragement, advice, and love made this project possible.

## DEDICATION

To Lesa

I shall be telling this with a sigh
Somewhere ages and ages hence:
Two roads diverged in a wood, and I-
I took the one less traveled by,
And that has made all the difference.

Robert Frost

The Road Not Taken

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# REVIEW OF THE LITERATURE

## TEMPOROMANDIBULAR DISORDERS

Dysfunction of the masticatory system was first described and studied by Costen (1934, 1936). He ascribed pain and dysfunction in the region as being due to "disturbed function of the temporomandibular joint": specifically overclosure of the mandible causing condylar impingement of various structures in close proximity to the temporomandibular joint or TMJ. The anatomic basis for Costen's syndrome (as it became known) was later disproven (Sicher, 1948), but Costen had succeeded in focusing medical and dental attention on the subject of pain and dysfunction in the region.

Numerous people have examined the problem of dysfunction of the masticatory apparatus, but under a number of different terms. Many of these terms included the word "syndrome" ( Shore, 1959, Schwartz, 1968, Laskin, 1969), which implied a narrow and rigid view of the condition. Storey (1979) took issue with this view and stated: "Mandibular dysfunction is not a syndrome but a spectrum of syndromes." Increasingly, the complex and integrated nature of the masticatory apparatus has led to more comprehensive approach to dysfunction in the region, both in terms of etiology and symptomatology. This has been reflected in two recent names given the disorder: Craniomandibular Dysfunction (Gelb, 1971) and Temporomandibular Disorders (Solberg, 1982). This paper will make use of the term temporomandibular disorder, or TMD, which will subsume the large number of terms and disease entities found in the literature to describe dysfunction of the masticatory system.

A comprehensive discussion of TMD is beyond the scope of this paper, so that the interested reader is referred to De Boever (1979), Storey (1979), Perry (1981), Moss and Garret (1984), and the 1982 report edited by Laskin et al.

## TMD SYMPTOMATOLOGY

There are three major signs and symptoms which are pathognomic of TMD (Gold, 1980):

1. pain in the muscles of mastication and/or in the temporomandibular joint or in their general anatomic region
2. limitation of mandibular movements
3. temporomandibular joint sounds.

These are fundamental defects for any musculoskeletal system and have become the defacto definition of TMD. These defects can be due to dysfunction of the muscles and their supporting tissues (tendons, etc.) or of the joint and its supporting tissues (ligaments, the capsule, the disc, etc.). In a number of cases, both the joint and the muscles are involved in the dysfunction. In many acute cases dysfunction of the muscles probably predominates.

In addition to the fundamental or pathognomic signs and symptoms of TMD described, there is a large group of secondary peripheral symptoms which are related to TMD in the minds of many clinicians. A number of these peripheral symptoms are related to the ear and include impaired hearing, tinnitus, dizziness and burning sensations in the tongue. Some have questioned the relationship between these symptoms and TMD (Norris and Eakins, 1974; Koskinen et al, 1980; Brooks et al, 1980), while others have discovered evidence for such a relationship (Myrhaug, 1965, 1970; Roydhouse, 1976; and Sharav et al, 1978). Unfortunately, these secondary peripheral symptoms are poorly documented and less well reported than are the primary ones. The dental research community has largely ignored them.

## TMD Etiology

Given the broad scope and complexity of the masticatory system, it is not surprising that the etiology of TMD is equally complex. A number of theories on the etiologic processes involved have been developed to explain TMD and a number of authors have attempted to categorize these (De Boever, 1979; Zarb and Speck, 1977; and Rugh and Solberg, 1979). All of these attempts at establishing an organized view of the etiologic basis of TMD have some validity, but some are more useful than others.

The most basic and easily conceptualized organization of etiology can be expressed under the headings of structural, functional, and psychological (Rugh and Solberg, 1979). Even using these basic categories, a number of factors can be placed in two or all of them. For instance, a functional factor such as masticatory muscle spasm could be due to bruxism or parafunction brought on by a structural factor such as an occlusal anomaly, by psychological stress and anxiety, or perhaps by both factors. A system of organization is useful if it aids in the discussion and conception of a large number of entities, but one should always keep in mind that the categories cannot be rigidly defined and are often interactive.

## The Role of Occlusion

Occlusal factors have been emphasized in a number of theories on the etiology of TMD. It is believed that occlusal factors may cause TMD through three major mechanisms:

1. active mandibular guidance to avoid or lessen the impact of the occlusal discrepancy (Storey, 1975)
2. initiation of bruxism and masticatory muscle hyperactivity by occlusal interferences (Scharer, 1974)
3. unfavorable loading in the joints produced by some occlusal discrepancies or interferences (Smith, 1984).

If the occlusion does play a role in the etiology of TMD, one would expect to see a correlation between temporomandibular disorders and occlusal discrepancies in epidemiological studies. Some studies have found such a relationship (Franks, 1965; Agerberg and Carlsson, 1973; Ingerval et al, 1980), while others have not (Helkimo, 1974; De Boever and Adriaens, 1983). It is not surprising to find such diversity in these findings on the role of occlusion, when one considers the multifactorial nature of that etiology. While epidemiological studies have demonstrated an association between various factors and patients suffering from TMD (Solberg, 1982), they are crude instruments to sort out the specific role of various etiologic factors within individuals.

A number of studies have looked at the activity of muscles in patients with various occlusal discrepancies (Jarabak, 1956; Troelstrup and Moller, 1970; Solberg, Clark and Rugh, 1975; Kolprogge and Van Griethnysen, 1976; Funakoshi, Fujita and Takehana, 1976; Ingervall and Carlsson, 1982; and Mac Donald and Hannam, 1982). Compared with asymptomatic controls these researchers found various changes in muscular activity, including a loss of symmetry or balance in bilateral activity, an increase in activity at rest or a combination of the two. The basic mechanism for these differences is usually attributed to a reflex alteration in muscle activity produced by the pattern of tooth contact mediated through periodontal ligament receptors (Funakoshi, Fujita and Takehana, 1976).

Perhaps the most interesting studies examining the role of occlusion are so-called provocation studies. In these investigations, occlusal discrepancies are placed in healthy asymptomatic subjects and the various consequences of these occlusal changes are examined. A number of studies found that occlusal interferences were capable of producing both TMD symptoms and alterations in the pattern of muscular activity (Randow et al, 1976; Bakke and Moller, 1980; Dreschler et al,1973; Riise and Sheikholeslam, 1982; Sheikholeslam and Riise,1983; Rugh et al, 1984)

Not all provocation studies, however, have produced signs or symptoms of dysfunction. Plata et al (1982) found no evidence, even after one to six months, of dysfunction in six patients given a lateral deflective occlusal contact. In an unpublished continuation of this study, Barghi et al later found development of significant TMD symptoms. De Boever (1969) placed balancing side contacts in asymptomatic patients and found no significant alterations in the EMG pattern of the temporal or masseter muscles. These findings indicated that some subjects seem to be capable of adapting to occlusal anomalies with no apparent dysfunction. It is widely accepted that the subject's reaction to the occlusal discrepancy will largely determine the development of TMD symptoms.

## Extent of Muscle Involvement in TMD

As in any musculoskeletal disorder, muscles play a large role in TMD etiology and symptomatology (Schwartz, 1955; and Moller, 1981). The tendency is to focus attention on the muscles of mastication, but a number of studies have reported the apparent involvement of neck muscles as well (Roydhouse, 1976; Travell and Simons, 1983; and Bell, 1985). More fundamentally however, the integrated nature of the musculoskeletal system requires the investigation of muscles beyond those of the masticatory system proper.

The concept of the potential for more extensive muscle involvement in TMD is not a new one. Brodie (1950) stressed the importance of the bilateral symmetry of the head and neck musculature and the functional relationships between the various muscles. He believed that alterations in the tension of the muscles in one area led to alterations in the muscles of the entire system. He also believed that the masticatory and hyoid muscles play an important role in the posture and muscle balance of the head and neck by acting as major flexors of the head. Last (1955) questioned some of Brodie's conclusions; he claimed quite correctly that Brodie had overstated the importance of the masticatory and hyoid muscles, while ignoring the prevertebral and sternocleidomastoid musculature as the major flexors of the head. Last did not deny, however, the integrated nature of the head and neck musculature.

Schwartz (1955) stated that not only are the muscles of mastication involved in TMD but, so too, are the posterior cervical, trapezius, and sternocleidomastoid muscles. Perry (1957) subscribed to Brodie's theory on the balance of the muscular system of the head and neck; he accepted that an imbalance imposed on one part would produce compensations throughout the entire system. He hypothesized that an occlusal discrepancy could produce the need for compensation, and later an imbalance in the masticatory musculature, so that the affected muscles become symptomatic. The posterior cervical musculature may then be called upon to produce further stress relieving compensations which, if the initial occlusal problem was not corrected, could lead to symptoms developing in the neck musculature. He did not, therefore, believe it surprising that postcervical musculature could be associated with certain problems of occlusion.

Clinical findings appear to support the potential for the involvement of the neck musculature in TMD. Roydhouse (1976), in a review of eleven surveys of TMD patients, found that seven of these reported that cervical pain was a symptom present in at least some of the patients. It was significant that cervical pain was among the most commonly reported symptoms in the surveys studied. It might also be suspected that some of the other surveys not reporting cervical pain, may not have looked for it. Griffin et al (1975) found that, of 22 TMD patients studied, nine had symptoms in the neck, shoulder, thorax, or arms as well. They believed that these findings were significant and related to the TMD. One must avoid drawing any hard conclusions from this data, as in some cases, the cervical pain could have been an incidental finding unrelated to TMD.

Physiotherapists, who specialize in the diagnosis and treatment of musculoskeletal disorders, do believe that a significant relationship exists between the neck and masticatory musculature (Grieve, 1981; Hertling, 1983). They have the clinical impression that hyperactivity of the masticatory muscles can lead to hyperactivity of the muscles of the neck .

If such a relationship exists, its nature and etiologic basis are largely unknown. Is it primarily a structural relationship in which, as Perry (1957) stated, a disturbance in one area (the occlusion) will lead to compensation in the neck musculature due to changes in the muscles of mastication? Or is it a nociceptive relationship where pain in one area of the body will produce pain and spasm in nearby related musculature as a guarding reaction? Simons et al (1943) found that pain in the head from various sites, including the temporal muscle, was capable of eliciting increased tension and pain in the ipsilateral neck muscles. What is the mechanism for this neck muscle involvement and how quickly will the neck musculature become involved by either mechanism? These important questions have been posed for more than two decades yet they remain unanswered (Storey, 1979).

## THEORIES CONCERNING THE PERIPHERAL SYMPTOMS IN TMD

The significance of the peripheral symptoms and extensive muscle involvement in TMD has been stressed by many clinicians. One investigator who has emphasized these symptoms in the diagnosis and treatment of TMD has been Harold Gelb. In a number of publications (Gelb and Arnold, 1959; Gelb, 1971; Gelb, 1979; Gelb and Bernstein, 1983) and in the text he edited (Gelb,1985), he has stressed a more global approach to the TMD patient wherein the clinician looks beyond the masticatory apparatus for symptoms and causative factors in the dysfunction. Unfortunately the evidence is primarily clinical and anecdotal in nature. Gelb and several other individuals have generated a number of theories on TMD, but with little or no research on which to base them. For this reason, many researchers have ignored Gelb and his theories; although he receives much attention from clinicians, both inside and outside the dental profession.

In a number of published surveys (Gelb et al , 1967; Gelb and Tarte, 1975; Gelb and Bernstein, 1983) Gelb found that most of his TMD patients suffered from a number of peripheral symptoms including headache, vertigo, and pain in the muscles of the neck and back. One must be cautious in the interpretation of his data because his clinical practice is based on referrals. Therefore his patients can be expected to have had a more severe and long-standing disability than the typical TMD patient. In addition, his surveys may have progessively represented a more highly selected sample, which would have increased the apparent rate of peripheral symptoms. Nevertheless, Gelb did find that peripheral symptoms were a significant finding in many TMD patients.

More speculative is Gelb's explanation for these symptoms (Gelb, 1979; Gelb and Bernstein, 1983). He believed that in TMD patients, a number of factors led to malposition of the mandible and that many patients possessed structural anomalies of their dentitions, such as a failure of vertical development on one, or occasionally both sides. The possible causes of these anomalies were neither presented nor discussed. Gelb believed that these jaw malpositions could act as "potent stressors" on the entire body, often affecting the entire chain of musculature along the axial skeleton down into the lower limbs, producing widespread muscular symptoms and leading to both mandibular and general body posture changes. He asserted, but did not document, that in his examination of thousands of patients, he found alterations in body posture which he attributed to mandibular malposition. In one diagram illustrating these changes of the head (Gelb, 1979), the head posture appeared to be asymmetrical with a definite lateral tilt to the left.

The apparent mixture of structural and postural changes is confusing, but it seems that the majority of these changes were postural since he treated the patients with mandibular repositioning appliances and found the improvement of the total individual was "unbelievable". He presented radiographs (Gelb, 1985) showing the entire body, which demonstrated dramatic improvements in the symmetry of the skeleton in posture, but these cases were not well documented and the technique he used for the postural radiographs was not discussed. In the only documented evidence of his assertions on postural change, Gelb claimed that, of two hundred patients seen in his clinic (Gelb and Bernstein, 1983), 150 of them demonstrated an asymmetrical body posture. His theories are interesting and provocative, but they remain essentially untested and only speculative at present.

Gelb is not alone in the support of these theories however, with dentists (Tanaka, 1985; Friedman and Weisberg, 1985), physiotherapists (Hertling, 1983), osteopaths (Hruby, 1985) and chiropractors (Vernon et al, 1982) embracing the concepts in various degrees. Gelb has also modified his ideas in accordance with the views of these professional groups, yet he remains the central and most outspoken figure supporting the more general body approach to TMD.

A close colleague of Gelb is Marion Rocabado, a physiotherapist specializing in the treatment of TMD and cervical spine disorders, who lectures extensively to physiotherapists and dentists. His concepts are very similar to those of Gelb, with whom he works (Rocabado, 1981). He believes that TMD can cause symptoms in the neck, and vice versa, with hyperactivity of masticatory and neck muscles often occuring together. His evidence has also been clinical and he has not suggested any specific mechanisms for the spread of dysfunction and symptoms. He did, however, state:

> the cervical and mandibular motor and neurosensory systems are so closely tied together that a dysfunction of one system undoubtedly affects the other even if the specific mechanism is not well understood

(Rocabado, 1981).

In the physiotherapy literature these ideas have been present for some time and are now largely regarded, by most members of the profession, as having been established (Trott and Goss, 1978; Grieve, 1981; Friedman and Weisberg, 1982; Danzig and Van Dyke, 1983; and Hertling, 1983). Some in the field, have gone so far as to suggest that treatment of TMD can improve athletic performance (Eversaul, 1985), but recent investigations of this claim have found little evidence to support this highly speculative idea (Hart et al, 1981; Schubert et al, 1984).

Despite the lack of research into, and documentation of, peripheral symptoms in TMD, the clinical impressions and concepts of many musculoskeletal specialists who treat these patients, does support the relationship between many peripheral symptoms and the basic pathognomic ones. This relationship should be investigated rather than ignored.

## RELATIONSHIP BETWEEN THE HEAD AND NECK MUSCLES

### The Functional Basis

The direct evidence for a functional relationship between the neck muscles and the masticatory system is not extensive. Nevertheless, there is reason to believe that there is such a relationship, and it is surprising that it has not been studied more. Dellow (1976) discussed the "automatic synergy of cranial-cervical musculature in swallowing" and Hannam (1976) believed this functional relationship should be explored, but the cervical musculature has largely been ignored by the dental research community.

Halbert (1958) examined the activity of the posterior cervical and infrahyoid musculature principally in relation to head movement, but also in relation to some jaw movements. He discovered that the posterior cervical musculature did show some minor activity during forced clenching and during rapid mouth opening. More recently, Davies (1979) examined the activity of several muscles of the neck during various mandibular movements. Davies looked at the sternocleidomastoid, semispinalis capitis and the sternohyoid muscles, and found that both the sternocleidomastoid and the semispinalis capitis were active during all movements of the mandible. The sternocleidomastoid was particularly active during forced opening, but was also very active bilaterally during clenching. Davies concluded that:

> the rhythm and coincidence of their activity (the neck muscles examined) with mandibular muscles suggest they possess strong motor influences in common with the latter muscles.

Interestingly this relationship had been discovered sometime earlier by Campbell (1955) who had been investigating the activity of the scalene and sternocleidomastoid muscles during respiration. He found that, during the course of his experiments, the subjects demonstrated continuous activity in these muscles when a mouthpiece was held firmly in their mouth.

Despite the apparent involvement of neck muscles in TMD, very little research has been performed to investigate this phenomenon. In a study of five normal subjects Widmalm (1984) discovered activity of one of the neck extensors (obliquus capitis superior) during voluntary tooth grinding in four of the subjects. He concluded that bruxing may potentially lead to hyperactivity and pain in the neck muscles.

In a general sense, these findings should not be surprising as children, and animals in particular, show significant and rhythmic head movement during chewing. Hiiemae (1976) examined masticatory movements in mammals and concluded that for monkeys:

> cranial flexion and extension are as much a part of mastication as are mandibular elevation and depression.

The activity of the neck musculature has also been shown to influence the activity of the muscles of mastication. Funakoshi and Amano (1973) found in decerebrated and labyrinthectomized rats, that various movements of the head elicited activity in the masseter, temporal and digastric muscles. Furthermore, this affect was eliminated when the first three cervical nerves were cut. Funakoshi, Fujita and Takehana (1976) showed that the activity of masticatory muscles was affected by various movements of the head in human subjects. They also showed that this effect appeared to be altered by the presence of occlusal discrepancies. Wyke (1979) provided a possible explanation for this phenomenon in his claim to have found that mechanical receptors in the cervical spine produced reflex activity in the jaw muscles. Unfortunately he did not provide any evidence for this, so that it must remain speculative.

## The Neuro-Anatomic Basis

The neuro-anatomic basis for the relationship between the trigeminal system which innervates the masticatory apparatus and the nerves supplying the neck has been studied extensively. In their examination of the trigeminal system, Wall and Taub (1962) noted that the descending tract and nucleus of the trigeminal descended to the cervical segments. They believed that the only explanation for these afferent fibres initially travelling away from the central nervous system was for the mediation of some function of the cervical cord.

Kerr (1961) found that fibres of the spinal tract of the trigeminal nerve extended caudally into the cervical cord as far as the midpoint of the second cervical segment. Later, Kerr (1972) found that convergence of the trigeminal spinal tract and the cervical cord systems extended to the third cervical segment with a minimal trigeminal relationship to the level of C4 and no direct relationship to C5 and below. This did not rule out some connections, via interneurons, with these lower levels, but Kerr believed such connections to be weak. He held that this intimate relationship between the two systems probably indicated a functional relationship. He could not find any evidence of trigeminal fibres directly relating to the motor nuclei of VII, IX, X, XI or XII.

Green, DeGroot and Sutin (1957), however, did find that trigeminal input produced efferent volleys in these cranial nerves including XI, the accessory cranial nerve, which innervates the sternocleidomastoid and trapezius muscles. Kerr and Olafsson (1961) confirmed the convergence of trigeminal and cervical dorsal root afferents in the upper cervical cord in cats, and believed that a similar convergence was suggested by studies on humans. They proposed that this relationship between the trigeminal and the upper cervical cord could explain the spread of facial pain and participate in reflex head turning activities mediated by trigeminal input.

Much of the work investigating the anatomic relationship between the trigeminal and the upper cervical spinal cord supplying the neck, has stressed the importance of these findings in relation to craniofacial pain (Gregg, 1977). A number of other studies, however, have found evidence that the trigeminal input plays a role in the muscle activity of the neck and the movements of the head (Manni et al, 1975; Abrahams and Richmond, 1977; Abrahams et al, 1979). Abrahams (1977) concluded: "our experiments leave us in no doubt that the spinal tract of the trigeminal nerve must play a major role in the control of head movement."

Rose and Sprott (1979) suggested that the cervical motor neurons are larger, with more potential connections to other nerves, than had previously been thought. This complexity of input was confirmed by Coulter et al (1979) who found that the cervical segments controlling neck movements received extensive descending projections from the cerebral cortex, brain stem and deep cerebellar nuclei. Troiani and Petrosini (1981) suggested that trigeminal input to the vestibular system may be involved with movement and posture of the head which required the involvement of the cervical muscles.

An important feature of the functional relationship between the trigeminal system and the muscles of the neck is the trigemino-neck reflex. This reflex appeared to be the first one to arise in the developing human embryo (Humphrey, 1964). As early as 7 1/2 weeks into fetal life, stimulation of the maxillary division of the trigeminal nerve, by light stroking of the perioral region with a hair, produced contralateral flexion in the cervical region. This avoidance reflex produced contraction of the neck muscles on the contralateral side and movement of the head away from the stimulus. Shortly after the appearance of this avoidance or "negative" type of reflex, a "positive" reflex of ipsilateral flexion toward the stimulus appeared. During fetal life, the trigeminal spinal tract passed to the upper level of C4 (Humphrey, 1952). The mandibular division appeared to travel the farthest caudally, and the opthalmic division, the shortest distance caudally. It was also observed that the response to trigeminal stimulation may have included bilateral shoulder movement thought to be due to the trapezius muscle.

> (This) indicates the transmission of impulses from the spinal tract of V to both ipsilateral and coaccessory nuclei, at least, if not other ventral horn neurons

(Humphrey, 1952).

The trigemino-neck reflex has been compared to the jaw opening reflex (Sumino and Nozaki, 1977). In cats, central stimulation of the infra-orbital nerve produced a vigorous trigemino-neck reflex response (Sumino et al, 1981). Central stimulation of the inferior alveolar nerve, the lingual nerve, and the masseter nerve had a similar effect, although they demonstrated a higher threshold of stimulation. Sumino and Nozaki (1977) concluded that the neck muscles received excitatory input from intra-oral structures and facial musculature, as well as from facial skin.

## MUSCLE IN PAIN AND DYSFUNCTION

Although joint noises are usually the most common symptom reported in TMD (Helkimo, 1979), pain, often of muscular origin, is usually the reason most patients seek treatment. It has been recognized for some time that muscles play a large role in the pain and dysfunction of TMD ( Schwartz, 1955; Thomson, 1976; Kraus, 1963; Berry, 1967; Mikhail and Rosen, 1980; and Clark, 1981; and Moller, 1981), but the processes causing and accompanying these problems are not well understood. Despite being the largest single organ of the human body, accounting for %40 or more of body mass, skeletal muscle has not received the attention it should have by the medical community (Travell and Simons, 1983; and Reynolds, 1983). This lack of knowledge concerning muscle disorders has hampered the study of all musculoskeletal disorders, including TMD. The reader is referred to Carrabre (1986) for a recent review of the literature on muscle pain.

Disorders of muscle are often described in terms of tone which is a non-specific property to describe the general state of a muscle when it is not involved in active movement. It is not a precise term, but it is clinically useful and frequently used in the literature. For most purposes, it is defined as "that resistance which is felt by the examiner's hand on passively extending a muscle" (Foley, 1961), but it also includes the feeling of firmness, or turgor, of the muscle at a state of rest. Drachman (1967) stated "tone is the result of the viscous and elastic properties of the muscle and its state of contraction at rest". He further stressed that: "alterations of tone may be due to a variety of influences playing upon the muscle from many levels of the nervous system, or to intrinsic changes in the muscle itself".

Finally, tone is also used to describe how vigorously the muscle reacts, or reflexly contracts in response to various stimuli, particularly to stretch (Basmajian, 1978).

There are a number of muscle disorders involving muscular tone (Drachman, 1967). The most common type seen in musculoskeletal disorders such as TMD, involve increased muscular tone or hypertonus. This can interfere with normal movement due to the increased resistance to stretch, and is often accompanied by pain in the muscle. This increased muscular tone is a form of hyperactivity in the muscle and is a common response to pain in the muscle (Storey, 1982).

Muscle spasm is another term used to describe hyperactivity in the muscle (Rasch and Burke, 1978). Travell and Simons (1983) defined spasm as: "incresed tension with or without shortening of a muscle due to non-voluntary motor nerve activity". It has been postulated that muscle pain can produce muscle spasm which in turn exacerbates and perpetuates the pain, leading to a pain spasm cycle (Travell et al, 1942). Muscle spasm does imply increased muscular activity and increased resistance to stretch, but there are no formally defined parameters for muscle spasm such as EMG activity or decreased range of motion. As a result, the term muscle spasm means different things to different people and this confusion is reflected in the literature. For this reason, the use of the term should be avoided unless it is formally defined and this paper will not use the term except where it is used in the literature.

This concept of a self-perpetuating cycle of pain and spasm is a common clinical notion which has been tested experimentally. De Vries (1966) produced fatigue pain in subjects and found that the characteristic pain, which developed 24 to 48 hours after the overuse period, was accompanied by small, but significant, increases in muscular activity. He concluded that a tonic localized spasm of motor units was the cause of this pain of late onset. Arroyo (1966) and Cobb et al, (1975) also found evidence of increased activity in painful muscles, supporting the concept. Brucini et al (1981) examined muscular activity in periarticular muscles of patients suffering from osteoarthritis of the knee. They found increased levels of muscular activity in these patients compared to control subjects and concluded that:

> these results seem also to indicate that abnormal afferent activity from a tender muscle or other periarticular tender areas may contribute to the establishment of a vicious cycle, by maintaining sustained contraction.

Not all studies, however, confirm these findings (Kraft et al, 1968).

That the relationship between muscle pain and increased muscle activity (spasm) is not a simple and direct one, can be seen in a number of studies. Nouwen and Solinger (1979) studied the effectiveness of EMG feedback training in the treatment of low back pain and found that, with training, both the muscle activity measured by EMG and pain in the lumbar muscles declined. Some time after training however, EMG levels tended to rise toward previous levels, while pain did not. McGlynn et al (1979) similarly found that EMG feedback produced a decline in pain after muscle exercise. EMG activity was not altered until 48 hours later when it did not rise, as De Vries (1966) had found it did under normal circumstances without EMG feedback.

If it can be accepted that muscle pain can give rise to increased muscle activity in some cases, one is still confronted with the problem of what produced the muscle pain initially. It would seem likely that muscle hyperactivity is capable of producing the initial pain, much as it does during overuse in exercise (Storey, 1982). Storey stated that this pain due to sustained hyperactivity is caused by the release and buildup of endogenous pain producing substances within the muscle.

This leads to the question of what level of muscle activity will produce such pain. Moller (1981) examined this problem and believed that sustained contractions of as little as 5% (or possibly less) of the maximum contraction can lead to signs of tiredness. Moller (1981) referred to two studies (Lous et al, 1970; and Sheikholeslam et al, 1981) as "the first quantitative proof of an association between natural muscular hyperactivity and pain". Rugh (1982) examined a number of studies which investigated muscle hyperactivity similar to that seen in TMD patients including the studies of Christensen (1971, 1978, and 1979), Banasik and Laskin (1972), and Scott and Lundeen, (1980). Rugh (1982) concluded that "these studies clearly demonstrate that muscle hyperactivity can result in symptoms similar to those found in (TMD) patients."

Sheikholeslam (1985) stated: "it is generally accepted that abnormal muscle activity in terms of hyperactivity is a prime factor "in the etiology of TMD.

## FACTORS EXAMINED IN MUSCULOSKELETAL DYSFUNCTION

A variety of parameters of musculoskeletal function are examined in studies of dysfunction; the two most common and informative ones being electromyography and range of motion tests. These same techniques are used in the study of many sports (Kraus, 1977) because they are important components of function in the examination of the musculoskeletal system, both in health and disease. Some studies use high speed computerized motion pictures to examine the pattern of movements, but this method is seldom used in clinical studies of dysfunction because of the expense. Range of motion tests are a more basic and simplified study of motion designed to determine the subjects' limits to movement and hence the extent of the dysfunction. It is a useful measurement to follow the progression of dysfunction in exacerbation or resolution.

A third parameter related to the movement pattern is that of posture which is a measure of the relative position of the body parts in various repeatable poses, usually: standing, sitting or lying. It is not often used in formal studies for reasons which are not clear. It is however, frequently used in clinical assessments of individuals with musculoskeletal disorders by a variety of health care workers including dentists and physiotherapists.

### Electromyography

Probably the most powerful tool in examining the musculoskeletal system is electromyography, or EMG, which measures the electrical activity of muscles by the analysis of the amplified action potentials generated by the muscles during function or resting postures. The raw EMG signals can be filtered electronically and can be further processed by computer to integrate the signal, yielding complex data on the activity of the muscle (Soderberg and Cook, 1984). EMG has many potential uses, but in studies of muscular function, it is chiefly used to determine the timing and coordinatiom of muscle activity and the relative levels of activity of different muscles in different tasks and under different conditions.

EMG has been used to study these parameters of muscle function in the masticatory muscles during various tasks, in TMD patients (Jarabak, 1956; and Munroe, 1975) or in those given occlusal anomalies (Randow et al, 1976; Bakke and Moller, 1980; and Sheikholeslam and Riise, 1983). Generally, these studies confirmed the alteration of the pattern of masticatory muscle activity from that seen in normal controls. EMG has also been used to examine the muscle activity in various postures of the body and head (Portnoy and Morin, 1956; Poppen and Maurer, 1982; Sturgis et al, 1984). It was found that some postures of the body, or structural relationships, are associated with higher or lower muscle activity and it was shown that in some cases, minor displacements produced significant changes in activity.

At both the clinical and research levels, EMG has been used in patients with musculoskeletal disorders. In patients with headaches or backaches, it has been used as a source of biofeedback, in an attempt to teach these patients to become aware of their muscle activity and to try to lower it. Given the complexity of muscle involvement in such disorders, and our present lack of knowledge, it is not surprising that EMG biofeedback is successful for some patients, while not so for others (Carlsson et al, 1975; Hart and Cichanski, 1981 and Dahlstrom, 1984).

As powerful a measuring tool as EMG is, it does not provide an unequivocal reading of muscle activity in a subject. Beyond the technical factors relating to the signal processing, electronic equipment, and electrodes, a number of factors can alter the muscle activity and EMG signal produced (Sturgis et al, 1984). These factors include the part of the muscle or motor units sampled by the electrodes, the precise motion executed by the subject, as well as its speed and vigor; subtle differences in these factors may alter the pattern of muscular activity. The levels of muscle activity can also be altered by fatigue and dysfunction in the musculoskeletal system. If these factors are ignored, they may cause conflicting and confusing results when different studies are compared. An example of this may be seen in studies of the trapezius muscle which is commonly involved in headaches.

The trapezius is considered to be a shoulder muscle (Gray's Anatomy, 1980), but it is also capable of moving the head in lateral flexion and rotation if the shoulder is fixed. A number of studies have examined the trapezius muscle function with most concluding that it is not active in unresisted head movements (Yamshon and Bierman, 1948; Wiedenbauer and Mortensen, 1952; Freitas and Vitti, 1980; and Bull et al, 1984). In contrast however, Sturgis et al (1984) reported that the trapezius was more active in head movements than the sternocleidomastoid, which is considered to be a prime mover in head movements

(Rasch and Burke, 1978; and Gray's Anatomy, 1980). In this latter study, the subjects were told to touch their ear to their shoulder which required significant shoulder elevation as well as side flexion of the head and neck. Therefore one cannot conclude from this study that the trapezius muscle is active in head movements.

## Range of Motion

One of the most useful clinical parameters for measuring the function of the musculoskeletal system is range of motion. It is a measure of the angular distance through which a joint, or complex of joints, can be moved from a defined neutral position to the limits of movement in a given body plane. Because motion is such a vital component of musculoskeletal function, range of motion is a fundamental measure of dysfunction in any region (Cole and Tobias, 1982; Tomberlin et al, 1984; and Saunders, 1985).

Range of motion is actually a specific measure of the flexibility of a subject in a given joint system and flexibity is an important component of physical fitness (Wells, 1971). Cooper et al (1982) stated:

> As with strength, the inability to perform normal ROM (range of motion) exercises at a specific joint may indicate severe restrictions on normal functioning and may prevent performance of some common necessary movements.

In the treatment of musculoskeletal problems, increasing, or restoring, range of motion in a joint system is often a primary goal and a measure of success (Gould and Davies, 1985) Dentists use range of motion to assess masticatory function; restricted mandibular movements are pathognomic of TMD (Gold, 1980).

In the clinical setting, range of motion is not simply a single measurement at each joint; rather, it includes such factors as: pain during movement, ease of movement, and the sensation of the quality of limitation present. This latter factor may include a sudden unyeilding block or a gradual sensation of increasing resistance leading to a restriction in movement (Barak et al, 1985). There are also two kinds of range of motion tests performed: active, wherein the patient moves the joint through its range with his own muscles, possibly under the guidance of the clinician; and passive, wherein the clinician both guides and produces the movement himself to the limit of motion. This information is analyzed to determine the tissues and pathological processes involved in the restrictions to movement.

Wells and Luttgens (1976) listed the three major factors, or tissues, determining a joint's range of motion as being:

1. shape of the articular surfaces and capsular structures
2. restraining effect of the ligaments
3. muscles.

One can see that the first two factors will remain stable over time with only the possibility of gradual change, except in the case of acute trauma. Muscle, however, is a very labile tissue which can be altered to affect the range of motion at a joint within several hours or days (Gossman et al, 1982).

It is reasonable to believe that muscles must play a large role in limiting the range of motion in acute disorders not involving major trauma to the joint or ligaments (Korr, 1975). This is a common clinical idea, but it is not well studied or documented. Two treatment modalities directed toward treating the muscles, massage and muscle stretch, have been studied (Crossman et al, 1984; and Odeen, 1981, respectively) and found to produce increased range of motion in patients.

There are a number of guides to muscle testing which describe the techniques for range of motion testing and the average measurement for each joint system in the three planes of motion (American Academy of Orthopedic Surgeons, 1965; Russe, 1972; Gerhardt and Russe, 1975; Hoppenfield, 1976). The figures given for the average range of motion are more accurately described as being figures for healthy individuals with no pathology; thus the figures given should be considered more ideal than normal.

There are no definitive rules to determine when a range of motion measurement indicates dysfunction; the accompanying findings and the patient himself will help to determine the presence and degree of dysfunction. Often the best guide is to compare the measurements for each side of the body, which should be similar if there is no dysfunction (Lehmkuhl and Smith, 1983). The measurement of range of motion is of limited value by itself, but must be interpreted in relation to the other findings of dysfunction and the pattern of alteration, in the range, over time. For this reason, the range of motion measurement is particularly valuable to determine the progression and resolution of musculoskeletal dysfunction.

The importance of range of motion for the vertebral column and neck is as great as it is for other joints, although the situation is more complicated because these are multi-joint systems. Nevertheless, range of motion is an important parameter to measure in studying dysfunction of the neck (Jackson, 1977; Maitland and Brewerton, 1977; Maitland, 1979; Manus-Garlinghous and Bloom, 1979; and Gould, 1985). There have been few recent studies of neck range of motion, but there have been several in the past few decades (Leighton, 1956a, 1956b; Buck et al, 1959; Kotke and Mundale, 1959; Lysell, 1961; Ferlic, 1962). These studies produced few important results except to show the normal range of measurements in several populations, illustrating the variability between individuals and that range of motion tended to decline with age. The average figures discovered in these studies were similar to those found in the muscle testing books listed above, but the inter-subject variability demonstrated the need to assess each subject as an individual, rather than comparing them to a published standard. Jackson (1977) and Gould (1985) stressed the need of comparing the range of motion of the neck in the two directions; balance in the muscles of each side of the neck is important.

## Posture

A third factor to be investigated in the study of the musculoskeletal system and its dysfunction is that of posture. Of the three factors discussed, it is the least frequently used in research studies, although it is commonly listed as an important part of the examination of musculoskeletal problems (Manus-Garlinghous and Bloom, 1979; Kessler, 1983; and Saunders, 1985), including TMD (Douglas, 1982).

The standard posture is really an ideal posture rather than an average posture; just as a normal class I occlusion is an ideal one. Kendall and McCreary (1982), in a text on muscle function and testing, described the standard posture as being a matter of skeletal alignment. In the frontal view, the ideal posture is characterized by balance or symmetry. The ideal posture in the lateral view involves a balance of normal postural curves, but is impossible to describe concisely, and the reader, if interested, is encouraged to consult the Kendall and McCreary text. Ideal postural alignment is rare in adults. For instance, the unilateral dominance of a hand or handedness, leads to a postural asymmetry in many cases, consisting of a lower shoulder on the dominant side, hips higher and deviated to the dominant side, with the spine tending to be deviated to the opposite side.

It is a widely held belief by many in the health care field that faulty posture can lead to, or perpetuate, muscular symptoms of pain, fatigue, and strain (Lowman and Young, 1960; Kendall and McCreary, 1982; Hartley, 1983; Espinoza, 1983), but there is little or no research to defend this common clinically accepted idea. It has been suggested by some, that faulty posture is more often the result of muscle symptoms and disease rather than its cause (Joseph, 1960). One does see faulty posture of a long-standing nature accompanied by weakness and chronic shortening of the muscles, but it is virtually impossible to determine which preceeded and caused the other.

It is also generally accepted that faulty skeletal alignment due to poor posture will produce, over a long period of time, osteoarthritic changes in the joints (Wells and Luttgens, 1976), but there is little direct evidence for this. This process can take decades, but may arise from the increased stress placed on various joint surfaces by the faulty skeletal alignment. As supporting evidence, one can cite the fact that, by the age of 40 years, most people will show the signs, if not the symptoms, of cervical spondylosis or osteoarthritic changes of the cervical vertebra (Maitland, 1982) and a forward head posture is considered to be one of the most common postural faults (Wells and Luttgen, 1976). This may be a normal process of aging which is accelerated by the additional stresses of poor posture, so that it may also be considered a pathological process.

An interesting book dealing with posture, among other concepts, is The Alexander Technique by Barlow (1982). The technique involves an approach to the musculoskeletal system which stresses "that there are certain ways of using your body which are better than others" and if these are ignored, dysfunction and degeneration will result. The book stressed the importance of correct posture and patterns of movement, placing particular emphasis on the head and neck, as the author believed that misuse of the entire body often begins in that region.

One specific structural and postural abnormality which has been cited as a potential cause of symptoms such as backache and leg pain, is the "short leg syndrome" (Nichols, 1960). The abnormal posture in this case is actually due to a structural problem where the legs are of unequal length; generally a 1/2 inch discrepancy is deemed to be significant. Nichols found in his study that in a control sample, 7% showed a significantly shorter leg, while in a group of patients with low bachache, the percentage with such a discrepancy rose to 22%. The situation is complex, with a number of other factors involved such as angulation of the sacrum with respect to the pelvis and angulation of the lumbar spine (Travell and Simons, 1983). It is clear that no postural fault exists in isolation without the involvement of related structures.

Gelb (1979) has suggested that the postural abnormalities caused by a short leg can extend up to the mandible in some cases and he claimed good success with the treatment of the short leg and the TMD symptoms with a leg lift in such patients. As in patients with occlusal discrepancies, patients with the structural problem of a short leg may remain asymptomatic until some additional stress on the system overwhelms their capacity to adapt.

It would seem that postural problems may be related to pathological changes in muscles and joints, but this is not well documented. To study this relationship, would involve a number of very long and involved longitudinal studies, but this would be a very fruitful area of research. The question remains whether or not postural abnormalities are the cause or the effect of those pathological changes.

## Summary

These three factors of electromyography, range of motion, and posture are not the only important parameters in musculoskeletal dysfunction, but they are the ones most commonly used in research and clinical practice. Although they are often considered separately, these factors are always related in some way. The interaction of the various findings is an important part of the disorder, as dysfunction in one parameter, is often accompanied by dysfunction revealed in another. For instance, lack of flexibility, or a decreased range of motion, is often related to a persistent faulty alignment or posture (Kendall et al, 1952; and Cooper et al, 1982). Each may represent a different manifestation of the underlying dysfunction, but results must be interpreted in the context of all the findings to develop a complete understanding of the disorder.

## THE INVESTIGATION OF HEAD POSTURE IN THE DENTAL LITERATURE

Kudler et al (1952) believed in studying the dental apparatus in its broadest sense including the posture of the head, the bones and their articulations in the head and neck, and the muscles of the neck. They discussed the interactive nature of these structures, and hence their importance to dentists. While the posture of the head has not been completely ignored in the dental literature, it has perhaps not received the attention that it deserves. Fortunately, this situation seems to be improving, with a new interest in head posture developing as dental researchers and clinicians look beyond the immediate area of the oral cavity (Mohl, 1977; Vig et al, 1980; Daly et al, 1982; and Darling et al, 1984).

The posture of the head has been related to the morphology of the head and face by some, in suggesting that individuals with retrognathic mandibles will extend their heads to make their chins appear more prominent while persons with prognathic mandibles will tend to flex the head to reduce the prominence of their chin. This hypothesis has been questioned in the literature (Cleall, Alexander and McIntyre, 1966; and Lundstrom, 1982) and supported (Marcotte, 1981), but further research is required.

The best research into skull morphology and the natural posture of the head has been carried out by Solow and Tallgren (1971a, 1971b, 1976 and 1977). In this series of experiments, they developed a protocol for taking lateral cephalometric films in the patient's natural head position with an acceptable degree of error. Their most significant finding was that the posture of the head and the vertebral column was correlated to the vertical relationships in the facial skeleton and the angulation of the cranial base, rather than antero-posterior facial skeleton relationships. They found that a great deal of the variation in the posture of the head is expressed as variations in the angulation and curvature of the vertebral column rather than simply changes in the angulation of the head to an external reference of the true vertical. This latter measure of head posture is, therefore, only one aspect of the total picture, although most studies look at this factor exclusively due to the non-feasibility of taking a large number of cephalometric films for a research project.

The posture of the head, neck, and related structures has been examined from the standpoint of the important functions for which the region is responsible, including mastication, deglutition, and respiration (Bench, 1963; Vig et al, 1980), as well as to the relationship of the mandible, hyoid bone, and tongue (McCarthy, 1980). It has been shown that changes in head posture will produce changes in the resting position of the mandible (Posselt, 1952; Brill et al, 1959; Preiskel,1965; and Dombardy, 1966). It has also been suggested that changes in head posture could produce alterations in craniofacial morphology and may produce the typical craniofacial structures seen in mouth-breathers with excessive adenoid tissue (Solow and Kreiborg, 1977).

The effect of head posture on occlusal contacts has also been investigated. McLean et al (1973) looked at the pattern of occlusal contact due to graded changes in the tilt of the entire body in the sagital plane, altering the posture of the head in relation to gravity. They found that in voluntary closure there was no alteration in the occlusal contacts, but that electrically stimulated jaw closure did produce more distal tooth contact as the body approached the supine position. Hairston and Blanton (1983) found that as the body was reclined from the upright to the supine position, the activity of a number of masticatory and related muscles was altered. They suggested that the maintenance of a patent airway was the major determinant of these muscular changes. Funakoshi, Fujita, and Takehana (1976) demonstrated that the masticatory muscles showed altered activity, in response to various movements of the head, which could not be explained by the need for airway maintenance. Others (Brenman and Amsterdam, 1963; Robinson, 1966; Mohl, 1976; and Winnberg and Pancherz, 1983) have discussed the possible significance of head posture on masticatory muscle function and occlusion, but could draw no firm conclusions.

The subject of head posture has recently received more attention in the dental literature, but this attention has almost entirely focused on the posture of the head in the mid-sagital plane. Only Gelb (1979, 1985) and Rocabado (1981) have discussed the importance of head posture as viewed from the frontal plane.

## STUDIES OF MUSCULOSKELETAL DYSFUNCTION

Unfortunately, dysfunction in the musculoskeletal system has not been extensively studied or documented, and its treatment remains largely based on clinical experience and judgement. Nevertheless, there are studies examining dysfunction in the axial skeleton which use two of the major parameters of musculoskeletal function previously discussed: electromyography and range of motion.

Back pain and dysfunction has probably received the greatest attention in the literature. Wolf et al (1979) looked at range of motion and EMG activity in the low backs of normal subjects during normal function and movement to develop normative data, to which back pain patients could be compared. In most studies, the resting EMG levels have been examined because it was thought that muscle tonus at rest would reveal the presence of pain and dysfunction. Wolf et al (1979) believed that EMG activity during movement may be more important in cases of dysfunction. Jayasinghe et al (1978) looked at postural fatigue in low back pain and found that backache patients showed large increases in EMG activity while standing, whereas control subjects showed a decline in levels. They believed that fatigue of back muscles may play a role in backache. Moreover, the backache patients showed clear differences in left and right back muscle activity and this finding was particularly marked in two subjects who had a recurrence of pain within two months of the investigation.

Kravitz et al (1981) looked at EMG levels in back muscles of back pain patients compared to controls, and found that resting levels were similar in the two groups. They found, however, that when the subjects were told to tense another set of muscles, the activity of the paralumbar muscles increased in the back pain patients, whereas the normal group exhibited no such increase. Collins et al (1982) found that chronic back pain patients actually demonstrated lower paraspinal EMG activity levels during various movements and resting postures, when compared to a matched group of controls. Under stress, however, the chronic back patients showed a greater reaction in the muscles than the controls.

A particularly interesting study (Sherman, 1985) examined left and right paraspinal activity in three groups of subjects: those with no history of back pain; those with a history of episodic back pain but none at the time of the study; and chronic low back pain patients of various diagnoses. He looked at EMG activity in the muscles in several different resting postures and movements, and found that all chronic back patients showed at least one posture or movement with a high EMG level. The particular affected movement or posture however, did not appear related to the diagnostic subgroup. This study was significant because it examined each subject individually, rather than pooling the results. It demonstrated the uniqueness of EMG abnormalities in dysfunction and pain, which can be lost or obscured if the patient results are pooled. This may explain some of the conflicting findings concerning EMG activity in patients with musculoskeletal disorders.

There are few studies of the neck region, but Jacobs and Felton (1969) found that feedback was successful in reducing muscular activity in neck injury patients. Peat (1976) used both EMG and electrogoniometry measuring joint motion to study normal and abnormal movements in the shoulder complex. Nordermar (1981) tested the use of transcutaneaous nerve stimulation in the treatment of acute neck pain and found that range of motion was significantly improved in all three planes of head motion.

Unfortunately, of the relatively few studies of musculoskeltal dysfunction carried out recently, most are either epidemiological or retrospective in nature. In these studies, subjects are often pooled into groups with individual differences being obscured. The subjects are often chronic patients who could be expected to have more complex patterns of symptoms and dysfunction, further complicating their examination. The only prospective studies carried out in musculoskeletal disorders are the provocation studies examining the role of occlusion in TMD. These studies, however, do not look beyond the immediate oral region for involvement of more distant, but related structures.

# Chapter II

# MATERIAL AND METHODS

## SAMPLE

The study was carried out on eight subjects; all but one of whom were dental students. The subjects were young (average age of 22 years, with a range of 20 to 28 years) and healthy, and included five males and three females. Prior to the study, the subjects were given a comprehensive examination and history (see Appendix A and B) which was designed to screen out any potential subject having significant musculoskeletal dysfunction (particularly TMD, neck problem or chronic headache) and to identify any factors which may determine the course of the subject's reaction to the experimental procedure.

All subjects had a Class I molar and cuspid relationship and demonstrated either no discrepancy between centric relation or retruded contact position and centric occlusion or intercuspal position, or a small (less than 1 mm.) anterior slide of the mandible from centric relation to centric occlusion. No subject demonstrated any lateral deviation of the mandible from centric relation to centric occlusion. Five subjects had full natural dentitions exclusive of third molars. One subject was congenitally missing one lateral incisor; one subject was missing four second bicuspids after they were extracted as part of orthodontic treatment. One subject was missing a lower first molar extracted in childhood due to carious breakdown, but the second molar had erupted into the space left by the extracted tooth.

None of the subjects showed evidence or reported significant signs or symptoms of musculoskeletal dysfunction in the masticatory apparatus, neck or shoulder region. Most of the subjects reported occasional clicking of one or both of the TMJ's on wide opening, but this was not associated with pain or locking of the joints so was not considered a contraindication to their inclusion in the study. Most of the subjects reported that they suffered from occasional headaches usually associated with stress, but they did not occur frequently and never lasted more than a day.

Only subject number 8 reported any history of symptoms from the lower back. He believed this was due to a minor injury suffered six years ago. As a result, this subject occasionally (approximately once per month) experienced a dull ache in the lower back which normally lasted no longer than one day and never for more than two days. This finding was not considered serious enough to warrant exclusion from the study because occasional lower back pain is a very common ailment. It also did not seem severe in this subject due to its episodic occurence and rapid resolution with no involvement of the upper back or neck region.

## EQUIPMENT AND METHODOLOGY

### EMG Equipment

The electrical activity of the 3 bilateral muscles were recorded with bipolar silver/silver chloride surface electrodes 0.4 cm in diameter (Beckman Instruments, Inc. Schiller Park, IL 60176, Catalog No. 650950) connected to a pair of twisted conductors surrounded by a grounded shield. The EMG signal was amplified at an overall voltage gain of 4000. The input impedance of the amplifier was 20 megaohms and the common mode impedance was 10 megaohms. The band width extended from 22 hertz (Hz) to in excess of 2.3 KHz. The common mode rejection ratio was from 80 to 100 db at 60 Hz. The signal amplifier was designed and built by Mr. Arthur Quanbury in the electronic shop at the Biomedical Engineering Research Department of the Rehabilitation Centre for Children, Winnipeg. The raw EMG signal was processed by means of full wave rectification and first order low pass filtering with a 3db cut off frequency at 10 Hz to form a linear envelope processed signal. A linear envelope signal closely follows the amplitude of the peaks of the raw EMG and is a suitable method for recording peak activity (Winter, 1983). This processed signal was recorded by a series 2500 eight channel Honeywell penwriter (Model 1508). The sensitivity of the EMG recording apparatus extended from 0.5 to 200 volts/cm

## EMG ELECTRODE PLACEMENT

Three paired muscles were examined in this study: the upper trapezius, the sternocleidomastoids, and the anterior temporalis. The upper trapezius muscle presented several difficulties: as with recording the activity of other thoracic muscles, EKG artifacts were amplified and recorded in addition to the skeletal muscular activity. These artifacts could not be eliminated and made the measurement of low level muscular activity difficult and potentially misleading. As the upper trapezius muscle is not normally active in head movements (Freitas and Vitti, 1980 and Bull et al, 1984), the muscular activity levels were expected to be low. It was hoped that the activity of the upper trapezius muscles would be high enough after the period of splint wear for their peak activity to be measured accurately. When the data was analysed at the conclusion of the study, however, this did not turn out to be the case, so the activity of the trapezius muscles was not included in the results of the study.

The placement of the electrodes over the bilateral anterior temporal muscles was modified after the technique of Ahlgren et al (1973). To position the electrodes, a ruler was placed resting on the superior attachment of the ear in line with the outer canthus of the eye and a mark was made on the skin 4 cm dorsal to the outer canthus of the eye. A mark was then placed 3 cm superior to the first mark on a line perpendicular to the temporal reference line formed by the lower edge of the horizontal ruler, and another mark was made 6 cm superior to the temporal reference line along the same perpendicular line. The two electrodes were placed immediately inferior to the two upper marks so that they were 2.5 and 5.5 cm above the temporal reference line (see Figure 1). In all cases this placed the electrodes for the paired anterior temporal muscles well over the body of the muscle.

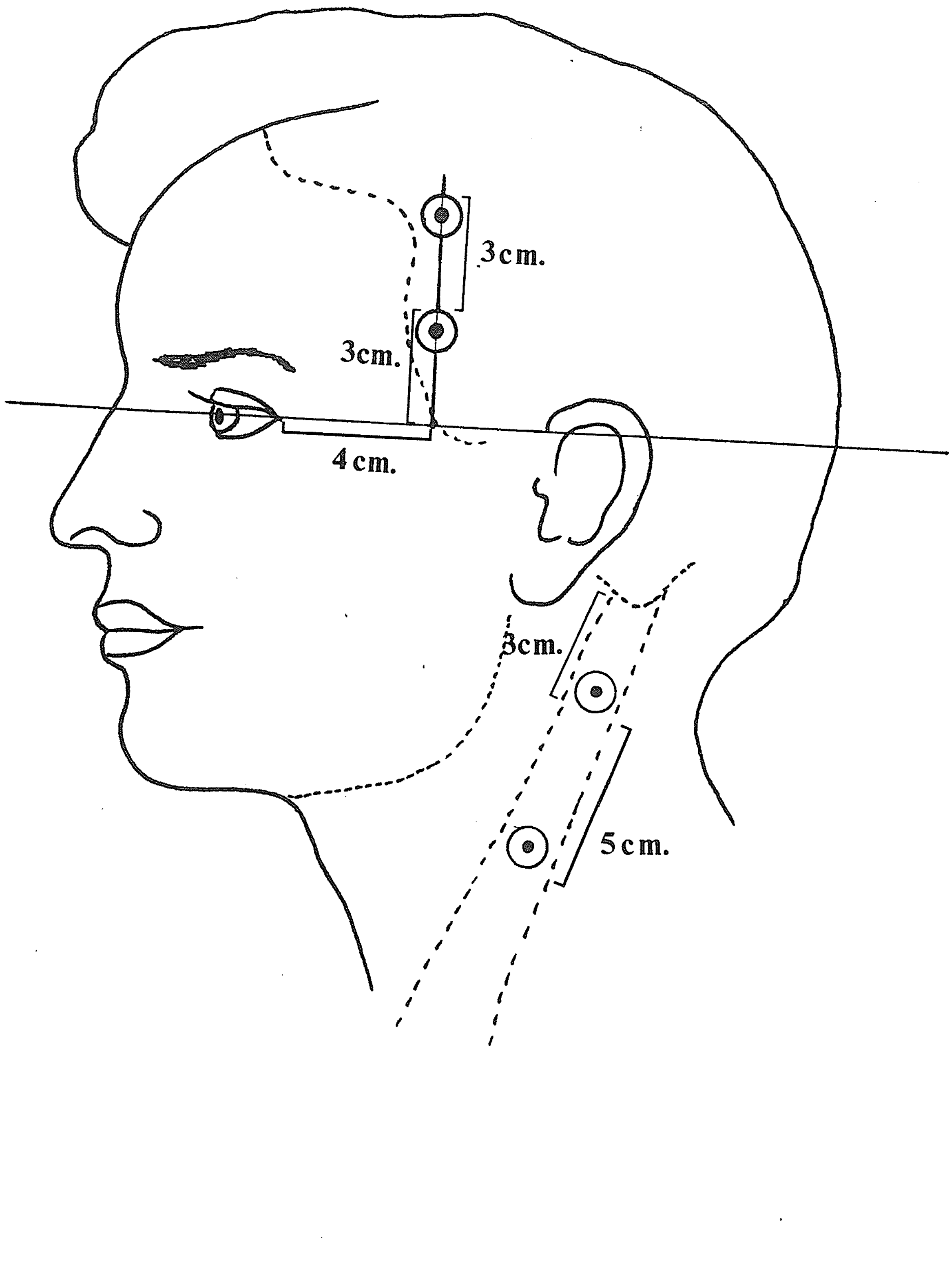
The placement of the electrodes over the paired sternocleidomastoid muscles was modified after the techniques of Lippold (1967) and Poppen and Maurer (1982). The subject was directed to turn his head slightly to the opposite side of electrode placement to bring the sternocleidomastoid muscle into a vertical line. A ruler was centered over the bulk of the muscle and two marks were made on the skin, 3 cm and 8 cm inferior to the mastoid process. An electrode was affixed immediately superior to each of these marks so that the electrodes were placed along the bulk of the muscle 2.5 and 7.5 cm inferior to the mastoid process (see Figure 1).

Prior to electrode placement, the skin at the site was cleansed with an alcohol soaked cotton gauze sponge and abraded with the rough sawcut end of a 9 mm diameter wooden dowel. The electrodes containing electrode paste (Beckman Instruments Inc., Schiller Park, IL 60176, Catalog No. 201210) were then affixed with double sided adhesive collars (Sensor Medics, Anaheim, CA, 92806, Catalog No. 650454). The electrode resistances were always less than 100 Kohm and usually less than 30 Kohm. A ground electrode was placed over the ulnar styloid process of the right hand.

Figure 1: Electrode Placement

Reference lines and electrode placements for the anterior temporalis and sternocleidomastoid muscles

Figure. 1



## HEAD POSTURE MEASUREMENT

The resting posture of the head in the frontal plane was recorded by photographing the subjects as they stood before a grid fixed to the wall. This grid contained lines spaced 10 cm apart aligned perpendicular and parallel to a vertical plumb line. The subjects stood in their stocking feet, on a fixed mark on the floor. A 35 mm single lens reflex camera (Minolta Model XG7) was fixed in a bracket on the wall opposite the grid at a distance of 3.7 M from the grid and 1.5 M from the floor. The camera was fitted with a Kiron 28 mm lens with a 3X teleconverter (Vivitar) producing an effective focal length of 74 mm. A flash unit (Model 17B, Braun Corp.) was used in addition to the ambient light for an exposure of 1/60th of a second. The film used was Fujichrome 100 ASA color transparency film (Fuji Photo Film Co. Ltd.).

## NECK RANGE OF MOTION EXAMINATON

The active range of motion of the head and neck in lateral flexion was measured according to the protocol outlined by Gerhardt and Russe (1975). An international standard goniometer (Rajowalt Co., Atwood Indiana) was used for all measurements. The axis of the goniometer was placed over the spinous process of the C7 vertebra with the fixed arm aligned with the spinous processes of the thoracic vertebra, and the movable goniometer arm was set at 180 degrees to the fixed arm. The subjects were told to hold their heads in a natural resting posture or upright position, the movable arm of the goniometer was then placed against the head and held there by the examiner's free hand. The subjects were then told to side flex their heads to the left without any rotation as far as they could comfortably move, hold for a few seconds, and return to the neutral position. During the movement, the examiner held the fixed reference arm of the goniometer against the thoracic spinous processes and the movable arm to the head which moved in an arc with the lateral flexion of the head and neck. The arc of the movable arm's path was recorded to the nearest degree. The subjects were told to move only their head and neck, keeping the shoulders and back stable, and that they were to perform the movement with the examiner's hand gently touching their heads to ensure that rotation of the head did not occur during the movement. The range of motion was measured to the left in this way 5 consecutive times followed by the measurement of the range of lateral head flexion to the right five times. The goniometer position on the subject was maintained for the measurements in both directions and all measurements were taken by the same examiner with the subject seated in the same chair. After the measurements were taken, the subjects were asked to judge whether or not they felt that it was more difficult or easier to side flex their head in one direction. If they did feel a difference between the two sides, they were asked to describe what they felt was the cause of this asymmetrical feel to their neck movement.

## SPLINT CONSTRUCTION

The splints were constructed on casts made from alginate impressions (Jeltrate-L.D. Caulk Co.) taken at the time of the initial examination of the subject. The impressions were poured in dental stone (Coe Cal-Coe Laboratories). A wax bite occlusal registration was taken at the same time to register the subject's centric occlusion. The interdental freeway space of each subject was also measured at this time after the technique described by Boucher, Hickey, and Zarb (1978). To determine their rest vertical dimensions the subjects sat upright in a dental chair with their heads unsupported and were told to relax their jaws, swallow, and relax again. With the lips lightly in contact, each subject was asked to hum and the distance between two marks placed on the nose and the chin were measured. The subjects were then told to bring their teeth together and the distance between the same two marks was measured again. The difference between the rest vertical dimension and the dental contact position was determined to be the interdental freeway space. In all subjects, this distance was found to be between 2 to 4 mm. The splints were constructed and adjusted so that the subjects' teeth were opened approximately 3 mm. beyond their freeway space in the anterior region.

The casts were mounted in a Galetti articulator for the initial construction of the full coverage splint. All splints were constructed on the lower arch. Initially, Coe-soft resilient denture liner (Coe Laboratories Inc.) was flowed around the undercuts and embrasures of the teeth, to act as a splint liner on non-occlusal tooth surfaces, for subject comfort and to aid in retention. Lang's Jet Acrylic (Lang Dental Mfg. Co. Inc.) was made into a doughy mix and formed around the teeth of the lower arch. The cast with the unset acrylic was then placed in an Acri-Dense Pneumatic Curing Unit (Coe Laboratories Inc.) at 30 P.S.I., allowing the acrylic to polymerize for at least 30 minutes.

Subjects were randomly assigned to one of two groups:

1. right side splint built-up
2. left side splint built-up.

All subjects were right-handed, so that one half of the subjects wore the splint buildup on their dominant side (referred to as the dominant group) and one half of the subjects wore the splint buildup on the non-dominant side (referred to as the non-dominant group).

The splints were trimmed to approximately 2 to 3 mm from the teeth in all areas except the posterior occlusal surface on the side where the splint was to be built up. On this side, the splint was trimmed on the articulator so that the upper posterior teeth and cuspids contacted the splint on the buildup side at an opening of approximately 4 to 6 mm beyond the interdental freeway space in the anterior region. The balance of the splint adjustment was performed in the subjects' mouths using articulating paper (Articodent-Union Broach Co. Inc.) to indicate occlusal contact with the splint. During this final adjustment, the splints were modified so that they contacted the upper posterior teeth and cuspids on the buildup side on at least one cusp tip (usually more), at a vertical opening of 2 to 4 mm beyond the interdental freeway space or rest vertical dimension in the anterior region. The splints were adjusted to have a flat occlusal table on the built-up side which allowed the subjects freedom in jaw position while maintaining the same occlusal contact on only the posterior and cuspid teeth on the built-up side. The built-up side of the splint will be referred to as the splint side for each subject although the splint is actually a full coverage one with unilateral occlusal coverage.

## EXPERIMENTAL PROTOCOL

After a subject was determined to be suitable for inclusion in the study, the risks were explained and the subject was asked to sign a consent form (see Appendix C) acknowledging these risks. The risks of participating in the study (as outlined in the consent form) were judged to be minimal and the subjects were told that they could withdraw from the study at any time. The experimental procedures and protocol were fully explained to the subjects prior to the experiment, however, the true nature of the study was not revealed until the completion of the study. This was done to preclude any of the subjects' expectations affecting their reactions to the splint. Based on data obtained in pilot studies, it was explained to the subjects that some minor discomfort, and perhaps headaches, might occur during the initial two days of the experimental period of splint wear, but that these symptoms should be minor. It was also stressed that such reactions to splint wear were not the area of investigation of the study and that the subjects were free to take any measure such as analgesics to treat them. Of the eight subjects, only one (subject no.3, a third year dental student) had any clinical dental experience which would include knowledge of TMD and related dysfunctions. It was therefore not expected that the subjects would have any significant or consistent expectations regarding the effect of the unilateral splint wear on the neck. All the subjects were told, that apart from the possible minor initial discomfort, they should not expect to become aware of any other effects of the splint wear.

All the subjects were measured for the three parameters used in this study (head posture, range of motion, and EMG) during examination sessions on two separate days during the control period of the 20 days prior to the delivery of the splint to the subject. The subjects were again measured for the same three parameters on two of the days of the experimental, or splint wear period. The subjects were all to be measured on day 5 and day 7 of the experimental period, but this was not always possible due to the subjects' schedules. As a result, subject no.'s 4, 5, and 6 were tested on day 6 and day 7, whereas subject no.8 was tested on day 5 and day 12 of the experimental period and subject no. 1 was tested on day 7 and 10. In all subjects the first and second days of testing in the control period are referred to as control day 1 and control day 2, whereas the first and second days of testing during the experimental period of splint wear are referred to as experiment day 1 and experiment day 2. On experiment day 1 and 2 the subjects were asked if they had experienced any symptoms of pain or discomfort in the TMJ region such as headache, joint sounds, periarticular pain, or painful and restricted mandibular movements. On the last day of the experimental period, the subjects' muscles in the head and neck region were palpated as they had been in the initial examination to determine the existance of any muscle pain or tenderness.

On the day of splint delivery, the splint was adjusted in the mouth as described in the previous section. The mandibular movements of the subjects were checked and recorded to ensure that these were not affected by the splint wear. The neck range of motion was also tested and recorded to serve as another control measurement. The subjects were instructed to wear the splint at all times (including during sleep), except when eating and cleaning their teeth.

## EXAMINATION SESSION PROTOCOL

All data recording was done in the same room of the Preventive Dental Science Department of the University of Manitoba. Prior to the recordings the EMG equipment was turned on and allowed to warm up for at least one half hour. Before the subject was examined, the EMG apparatus was calibrated with a 1 millivolt d.c. signal generator prior to every recording. All examination sessions were performed in the same order of data collection. The only differences occurred between the control and examination sessions because several additional tasks, with the splint being worn, were tested during the EMG recording in the experimental sessions.

At each examination session the subjects were initially asked to remove their shoes for the head posture photographs. They stood on a marked section of the floor with the grid directly behind them. They were instructed to look straight ahead at a piece of tape placed on the wall above the camera at their individual eye level. The subjects were encouraged to relax and in aid of this, told to shrug their shoulders several times. They were then asked to laterally side flex their heads gently back and forth with a decreasing amplitude until they found a comfortable position of the head. At that point the picture was taken and the process was repeated for another picture.

The subjects were then seated in a chair for the measurement of their neck range of motion. This was done according to the protocol described in the earlier section with the range measured to the left five times consecutively followed by measuring it to the right five times.

The subjects then removed their shirts (female subjects wearing a garment off the shoulders) and the EMG electrodes were affixed to the skin according to the protocol described earlier. All records were made at a paper speed of 5 mm per second.

The subjects were then asked to perform the following tasks:

1. Clench fully in the centric occlusion or maximum intercuspation position for five seconds with no splint in the mouth. This task was recorded three times. The task is referred to as a tonic clench.
2. With the teeth in contact, clench fully in the maximum intercuspation position, for a brief interval and relax immediately. This task is referred to as a phasic clench. The task was recorded five times.
3. Clench in the maximum intercuspation position at approximately 20 to 25% of full clenching for five seconds. This moderate clench was determined by the subjects' observation of their own muscular output with a voltmeter (impedance of 20,000 ohm/volt) connected to the amplified output of the anterior temporalis muscle on the splint side. This task was recorded three times. The task is referred to as a partial clench.
4. Side flex their heads to the splint side to the end of the range, hold for one second and return their heads to the neutral position. This task was recorded five times.

5. Side flex their heads to the non-splint side to the end of the range, hold for one second and return their heads to the neutral position. This task was recorded five times.
6. Side flex their heads to the right to the end of the range, hold for one second, return their heads to the neutral position and without stopping, continue tilting their heads to the left to the end of the range, hold for one second and return to the neutral position. Again without stopping at the neutral position, the subjects were instructed to continue side flexing their heads again to the right and so on. The subjects continuously side flexed their heads laterally to the right and to the left until they had moved their heads 5 times in each direction whereupon they stopped at the neutral position. This task is referred to as repetitive bilateral side flexion of the head.
7. Isometrically flex their heads maximally against the resistance of the examiner's right hand. This task was recorded three times and was designed to obtain a maximum contraction of the sternocleidomastoid muscles.

Each of these tasks was performed in the same order in all subjects at all examination sessions, with one exception. Subject no.4 did not perform task no.3 without the splint during the experimental period recording examinations. It was felt at the time that it was not necessary or informative, but it was performed in all other subjects.

In addition to the 7 tasks described, the three clenching tasks (task no.'s 1, 2, and 3) were also performed with the splint in the mouth for the experimental examination period. These tasks were performed in the same way and for the same number of repetitions with the splint in the mouth. These tasks are designated task no.'s 1s, 2s, and 3s, indicating the same tasks as no.'s 1, 2, and 3 respectively, except that the subject had the splint in his mouth. On all experimental days of testing, each clench task was performed initially without the splint, followed by the same task with the splint inserted.

The moderate clenching task (task no.3) was included because after pilot testing, it was believed that a moderate clench of 20 to 25% of maximum resembled the clench which, in their experience, pilot subjects often applied to the splint.

The tasks were described to the subjects each time before they were performed and, for the initial examination session, the subjects were allowed to practice them several times if necessary. Each individual repetition of a task was separated by an approximate 15 second rest period with a 2 minute rest period between each different type of task.

## MEASUREMENT OF HEAD POSTURE RECORDINGS

The color transparencies from the head posture photographs were measured by rear-projecting them onto a semi-opaque ground glass screen. The slide projector and the glass screen were mounted on a wooden frame at a distance of approximately 2.5 meters from each other to standardize the enlargement of the images. A sheet of clear acetate with a single pencil line was used to find a best fit line to describe the eye structures. The angulation of this line to one of the grid lines was recorded to indicate the angulation of the head posture in the frontal plane to the nearest half degree. The same reference line was used on the grid for all measurements and all measurements were made by the same examiner. The sign convention was such that the angulation of the eye line was given a positive sign if the head was tipped down toward the splint side and negative if the head was tipped down toward the other side. A measurement of 0 degrees indicated that the eye line was perpendicular to a line of true vertical to gravity.

## QUANTIFICATION OF EMG RECORDINGS

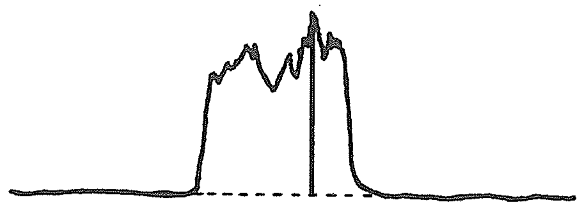
All EMG recordings were made of the linear envelope signal which follows the amplitude of the peaks of the raw EMG (Winter, 1983). All EMG values were converted to a percentage of the daily session maximum for each individual muscle. This permits the comparison of EMG activity on different days and between different subjects (Miller, 1985). The maximum activity for each muscle was determined by averaging the three maximum peaks obtained in the maximum tonic clenching task for the anterior temporal muscles and for the maximum resisted head flexion task for the sternocleidomastoid muscles. For the clench and hold tasks (task no.'s 1 and 1s, maximum clenching and, 3 and 3s, partial clenching), three measurements were taken in the middle part of the recording at one second intervals for each of the 3 biting procedures per task (see Figure 2). These 9 values for each muscle and for each task were averaged and expressed as a percentage of the session maximum. For these same tasks, the activity of the sternocleidomastoid muscles was measured in the same way. For the phasic clench tasks (task no.'s 2 and 2s), the peak value for each of the five biting procedures was measured, averaged, and expressed as a percentage of the session maximum for both the anterior temporal and sternocleidomastoid muscles.

Figure 2: Quantification of EMG for Clenching Tasks 1, 1s, 2, 2s, 3, and 3s

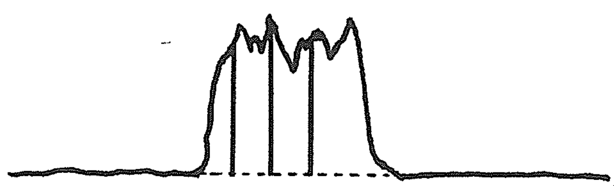
vertical lines indicate the position and height of the measurements made for each task

data was converted to a % of the average maximum value for the examination day

Figure. 2

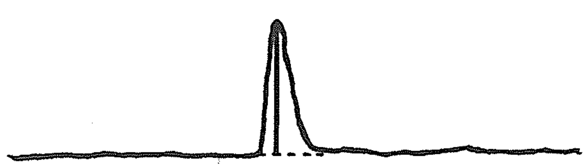


Reference Maximum Value

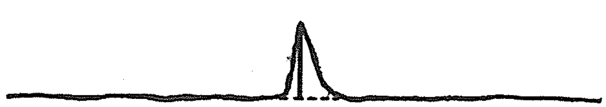


Anterior Temporalis
Maximum or Partial Values in Static Clench

Sternocleidomastoid
Maximum or Partial Values in Static Clench



Anterior Temporalis
Maximum Phasic Clench



Sternocleidomastoid
Maximum Phasic Clench

For the unilateral side flexing tasks (task no.'s 4 and 5), the ipsilateral sternocleidomastoid was initially active as a prime mover to bring the head to the end of the range. After the peak amplitude of the ipsilateral sternocleidomastoid was reached, the activity declined as the head returned to the neutral position. Early in this return phase, the contralateral sternocleidomastoid was active, reaching a lower peak activity (see Figure 3). The pattern of activity is the same for the bilateral continuous side flexing task (task no. 6) (see Figure 4). For all side flexing tasks, the peak EMG level reached by the prime mover sternocleidomastoid was measured for each of the five ipsilateral head tilts and expressed as a percentage of the session maximum (from task no.7). For these same head moving tasks, the peak activity of the contralateral sternocleidomastoid involved in the return phase of the head movement was measured for the 5 head movements and again expressed as a percentage of the session maximum.

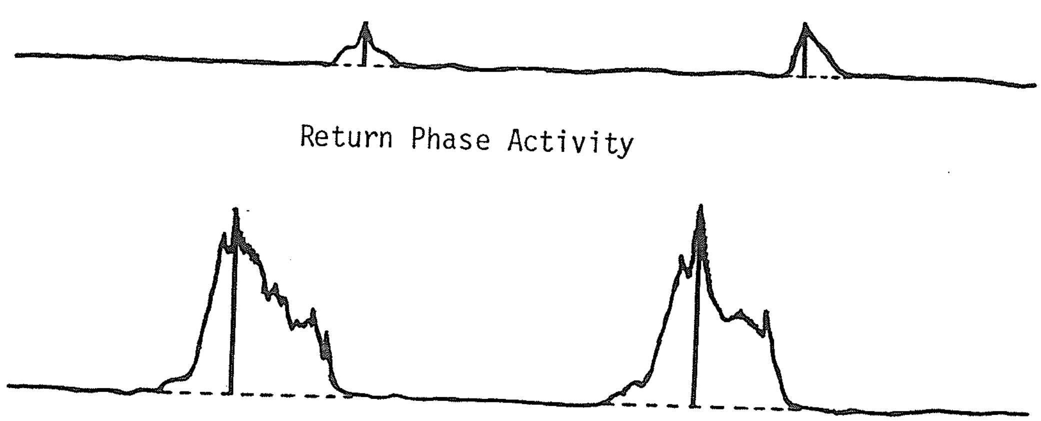
Figure 3: Quantification of EMG of Sternocleidomastoid Muscles for Head Moving Tasks 4 and 5

vertical lines indicate the position and the height of the measurements made for each task

data was converted to a % of the average maximum value for that measurement day

reference value (maximum activity level) was measured the same way as for clenching tasks

Figure. 3



Return Phase Activity

Prime Mover Activity

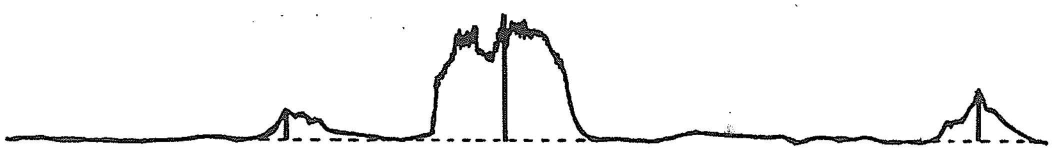
Figure 4: Quantification of EMG of the Sternocleidomastoid Muscles for Head Moving Task 6

vertical lines indicate the position and height of the measurements made for each task

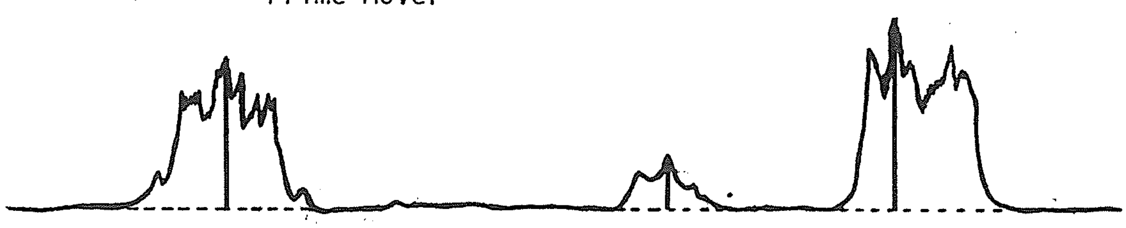
data was converted to a % of the average maximum value for that measurement day

reference value (maximum activity level) was measured the same way as for clenching tasks

Figure. 4



Sternocleidomastoid
Alternating Activity as Return Muscle and
Prime Mover



Sternocleidomastoid
Alternating Activity as Prime Mover and
Return Muscle

## ERROR STUDY

The pooled standard deviation for the error of each measurement of the head posture and the range of motion was calculated, as was the pooled standard deviation for the error of the activity level of each muscle (anterior temporalis and sternocleidomastoid muscles) for each task. From these measurements, the maximum error at the 99 % probability level was calculated for each measurement using the t value for the appropriate degrees of freedom. This figure was then divided by the square root of the number of measurements taken each day to arrive at the maximum error (at the 99 % probability level) for each day's measurement for each muscle, for each task, for each subject. This means that 99 % of the day's measurements for each task, and for each subject, should not exceed the standard error. These maximum errors are shown in Table 1 .

TABLE 1

Maximum Standard Errors for Each Day's Measurement for Each Subject

| Test | | Maximum Standard Error at 99 % level |
|---|---|---|
| Head Posture | | 1.5 degrees |
| Range of Motion Difference | | 1.4 degrees |
| EMG - Muscle Activity Levels | | |
| Task 1 - Anterior Temporalis | | 11.8 % |
| Sternocleidomastoid | | 0.9 % |
| Task 1s- Anterior Temporalis | | 12.4 % |
| Sternocleidomastoid | | 0.8 % |
| Task 2 - Anterior Temporalis | | 12.7 % |
| Sternocleidomastoid | | 2.7 % |
| Task 2s- Anterior Temporalis | | 9.4 % |
| Sternocleidomastoid | | 2.6 % |
| Task 3 - Anterior Temporalis | | 4.5 % |
| Sternocleidomastoid | | 0.3 % |
| Task 3s- Anterior Temporalis | | 2.8 % |
| Sternocleidomastoid | | 0.4 % |
| Task 4 - Sternocleidomastoid | | 9.8 % |
| Task 5 - Sternocleidomastoid | | 8.3 % |
| Task 6 -Sternocleidomastoid | - Function 1 | 11.4 % |
| | Function 2 | 3.8 % |

measurements of standard error of the muscle activity levels are expressed as a percentage of the daily maximum, not as a percentage of the average value for that task

## STATISTICAL ANALYSIS

A mixed analysis of variance was used to assess the effect of the factors and their interactions on the measurements of head posture, range of motion, and EMG activity levels in the various tasks.

For the head posture and the range of motion tests, the effect of the splint wear and the dominance (splint on dominant side, or on non-dominant side), as well as the interactions between the two factors were tested. A student's t-test was also used to assess, in both the head posture and the range of motion tests, whether or not the measurements for the control period, and the two experimental days were significantly different from an ideal (or normal) value for each test. This ideal measurement would represent a level head posture in the frontal plane and a symmetric range of motion to the two sides in head side flexion.

For the EMG tasks, the significance of the dominance (splint on dominant side, or on non-dominant side), the muscle tested (or muscle function as in repetitive bilateral side flexion), and the side of the muscle were tested, as well as their interactions, to determine the significance of their effect on the changes in the activity levels of each muscle, for each side, for each task. The EMG measurements from the control days and the experimental days were each averaged to arrive at a control period activity level and an experimental period average activity level. This was done because there were no clear trends in the activity level changes between the two experimental days and the differences in the time periods between the two experimental days (due to the subjects' availability for testing) made any trend less significant. The changes in muscle activity levels were expressed as a percentage of the activity levels measured in the control period for tasks 1, 2, 3, 4, 5, and 6. In tasks 1s, 2s, and 3s, the clenching tasks while the splint was worn, the changes in muscle activity levels were expressed as a percentage of the activity levels measured in the experimental period without the splint being worn by the subject (tasks 1, 2, and 3, respectively). The significance of these changes in muscle activity levels were then assessed using a Student's t-test of statistical significance.

# Chapter III
# RESULTS

## SUBJECTIVE REACTIONS TO SPLINT WEAR

All subjects were able to wear their splint for the duration of the experimental period with no significant problems. Three subjects did experience a headache on the first day of splint wear, but these headaches were not severe and were treated with aspirin or resolved spontaneously. One of these subjects (subject no. 5) also reported a neck ache on the splint side which occurred on the third day of the experimental period of splint wear. This pain lasted for several hours, but resolved with aspirin and did not reoccur. All subjects experienced some minor tooth discomfort at some point due to the splint, but this was never severe. All subjects were aware of minor alterations in their occlusions when they ate without the splint in their mouths during the experimental period. This rapidly resolved several days after the end of the experimental period. No subjects developed any signs or symptoms of TMD, including reductions in their range of mandibular movements during the experimental period.

During control testing of the neck range of motion, when the subjects were asked if they found it easier to laterally flex their head to one side, only three subjects indicated any difference between the two sides on one of the control days. On the other control day, they felt both sides were equal in the side flexion movement. All other subjects felt that their side flexion movements were the same on both control days. During the splint-wear period, seven out of eight subjects felt increased tension on the splint side on at least one of the experimental days, and of these, six felt that this limited their range of motion in side flexion toward the non-splint side. Subject no. 2 felt increased tension on the splint side on both experimental days, but he felt this increased tension limited his range in side flexing to that same splint side. Subject no. 6 was the only one not to have felt increased tension on the splint side during the experimental period.

Throughout the neck range of motion testing, all subjects felt that it was muscular resistance and not pain or discomfort which restricted their range of motion in side flexion. One subject (no. 8) noted, without being questioned, increased tension in his entire neck during the experimental period apart from that noticed during the range of motion testing. Subject no. 5 experienced pain in the muscles of the neck (particularly the splint side) during the head and neck movements performed during the EMG testing on the experimental days, but this pain resolved with aspirin. This same subject also demonstrated some muscular tenderness and sensitivity to palpation in the masseters (splint side being slightly more tender), as well as the left and right upper trapezius muscles, and the posterior cervical muscles (non-splint side being slightly more tender than the splint side). No other subject demonstrated any overt tenderness or pain to palpation of the muscles of the head and neck.

## HEAD POSTURE

A mixed analysis of variance was used to measure the effects of the period of splint wear, the side of the splint buildup or dominance, and the interaction of the two factors. Table 2 indicates that none of these three factors had a significant effect on the head posture of the subjects. Table 3 shows that both groups consistently had a head posture which was within 1 degree (on average) of upright (with the horizontal line through the eyes, perpendicular to the vertical).

TABLE 2

Significance of Main Effects and Interactions on Head Posture

| Source of variance | Level of Significance |
|---|---|
| Dominance | NS |
| Period of splint wear | NS |
| Dominance by period of splint wear | NS |

NS = not significant

TABLE 3

Average Head Posture of Dominant and Non-dominant Groups

| | Control period | Experimental day 1 | Experimental day 1 |
|---|---|---|---|
| Dominant Group | +1.0 NS | +0.6 NS | +0.5 NS |
| Non-dominant Group | -0.8 NS | -0.9 NS | +0.2 NS |

positive value indicates head posture tilted down to non-splint side
negative value indicates head posture tilted down to splint side
NS = not significant
all measurements in degrees
standard error= 0.44 in group 1
standard error= 0.38 in group 2

## RANGE OF MOTION

The head and neck range of motion for each day of testing consisted of two measurements: an average range of motion in side flexion to the splint side, and to the non-splint side (both measurements in degrees). These two figures were subtracted to obtain a single measurement which represented the difference between the range of motion to the two sides. The sign of the measurement indicated the side to which the range of motion in side flexion was greater; a positive sign indicated that the range of side flexion was greater to the non-splint side of the subject, whereas a negative sign indicated a greater range to the splint side. This single measurement of the difference between the range of motion to the two sides, or the symmetry of the range of motion, was used for all analyses of the range of motion.

A mixed analysis of variance was used to measure the effects of the period of splint wear, the side of the splint buildup or dominance, and the interaction of the two factors on the head and neck range of motion. Table 4 indicates that none of the three factors had a significant effect on the head and neck range of motion of the subjects. Table 5 shows the average range of motion differences for the control period and the two experimental days. Both experimental days showed a slight trend toward a greater range of motion in side flexion to the splint side, but neither experimental day showed a significantly asymmetrical range toward the splint side.

TABLE 4

Significance of Main Effects and Interactions on the Range of Motion in Side Flexion

| Source of variance | Level of Significance |
|---|---|
| Dominance | NS |
| Splint wear period | NS |
| Dominance by splint wear period | NS |

NS = not significant

When the individual subjects' range of motion measurements are examined over the control period and the two experimental days, a pattern of change does appear to emerge (see Figure 5). All but two sub-

TABLE 5

Differences Between Side Flexion Ranges of Motion to the Splint Side and the Non-Splint Side for the Control Period and Experimental Days

| | Average of Control Period | Experimental Day 1 | Experimental Day 2 |
|---|---|---|---|
| Dominant Group | -0.1 NS | -3.2 NS | -1.6 NS |
| Non-dominant Group | +2.1 NS | -0.8 NS | -2.5 NS |

positive value indicates greater range in side flexion to non-splint side
negative value indicates greater range in side flexion to splint side
NS = not significant
all measurements in degrees
negative values indicate a greater range in side flexion to the splint side
positive values indicate a greater range in side flexion to the non-splint side
standard error = 2.4 for all measurements

jects (no.'s 2 and 5) showed a trend towards a greater range in side flexion to the splint side on the first experimental day. Of these six subjects, three (subject no.'s 1, 4, and 8) maintained or increased this trend on the second day of experimental testing. The other three subjects (no.'s 3, 6, and 7) demonstrated a reduced tendency of a greater range of side flexion to the splint side on the second experimental day.

The two remaining subjects (no.'s 2 and 5) demonstrated the opposite change in their range of motion in side flexion. Subject no. 2 showed an increased range of motion toward the non-splint side on both experimental days. Subject no. 5 showed this trend on the first day of experimental testing, but on the second experimental day she demonstrated the opposite trend with a greater range of side flexion to the splint side (as had been seen in the other 6 subjects). Due to the lack of a consistent trend in terms of the direction of change in the range of motion measurements, the data was again analysed with the mixed analysis of variance. In this case however, the signs of the measurements were ignored so that the measurements of the range of motion simply indicated the magnitude of the differences or asymmetries in the side flexion range of motion to the two sides and not the side to which the range was greater. Table 6 indicates that the splint-wear period did have a significant effect on the symmetry of the neck range of motion in side flexion while the other factors did not. Table 7 indicates that both dominance groups demonstrated a significantly asymmetrical range of motion on the first experimental day. On the second day of the experimental testing, both groups showed a reduced, although still significant, trend toward an asymmetrical range of motion.

TABLE 6

Significance of Main Effects and Interactions on the Absolute Value of the Range of Motion Differences in the Control Period and on the Experimental Days

| Source of variance | Level of Significance |
|---|---|
| Dominance | NS |
| Splint wear period | ** |
| Dominance by splint wear period | NS |

NS = not significant
* = $p < .10$
** = $p < .05$
*** = $P < .01$

Figure 5: Differences in Head and Neck Range of Motion in Side Flexion for Subjects in Control Period and on Experimental Days

positive values indicate a greater range of motion in side flexion to the splint side

negative values indicate a greater range of motion in side flexion to the splint side

Figure 5

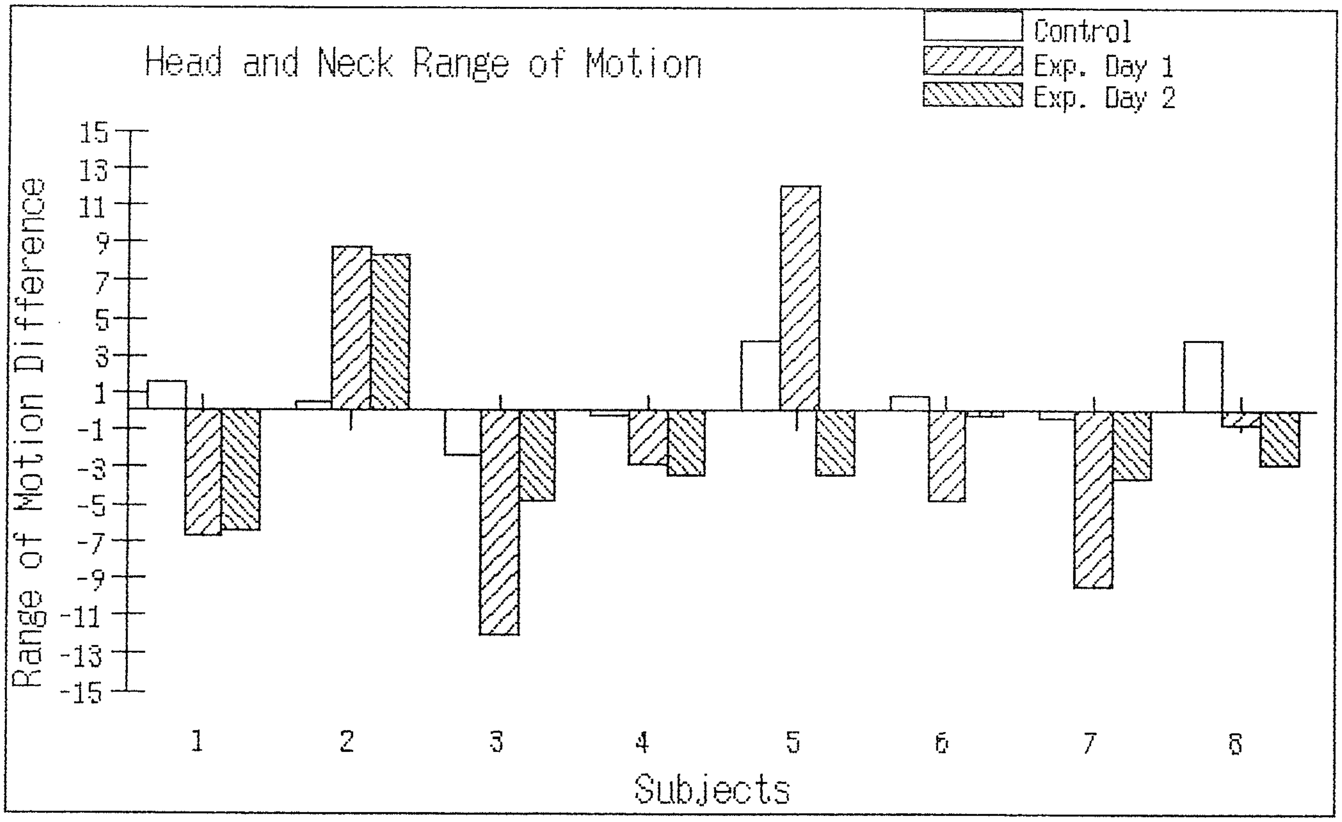

Head and Neck Range of Motion
Control
Exp. Day 1
Exp. Day 2
Range of Motion Difference
15
13
11
9
7
5
3
1
-1
-3
-5
-7
-9
-11
-13
-15
1
2
3
4
5
6
7
8
Subjects

TABLE 7

Absolute Differences Between Side Flexion Range of Motion to Splint Side and Non-Splint Side in the Control Period and in the Experimental Days

| | Average of Control Period | Experimental Day 1 | Experimental Day 2 |
|---|---|---|---|
| Dominant Group | 1.1 NS | 7.5 *** | 5.8 *** |
| Non-dominant Group | 2.3 NS | 6.8 *** | 2.5 * |

NS = not significant
* = $p < .10$
** = $p < .05$
*** = $P < .01$
standard error = 1.3 for all measurements

## ELECTROMYOGRAPHY

### Task 1 - Maximum Static Clench (No Splint)

For task 1, the change in the level of muscle activity of each muscle of each side, due to the period of splint wear was recorded. A mixed analysis of variance was used to assess the effect of the muscles (anterior temporalis or sternocleidomastoid), the side (splint side or non-splint side), dominance (dominant group, with the splint buildup on dominant side; non-dominant group, with the splint buildup on non-dominant side), and the interaction of these factors.

Table 8 shows that the factors of muscle, side, and muscle by side demonstrated a significant effect on the change in muscle activity at the 10% level of statistical significance. The effect of the muscles and side were both very close to reaching significance at the 5 % level (F=5.964 and F=5.974, respectively - the relevant F value for 5 % significance is 5.984).

In examining the percentage change in each muscle (table 9), one can see that the splint and non-splint side anterior temporalis muscles did not show a significant change in activity levels in maximum clench due to the period of splint wear. The non-splint side sternocleidomastoid, however, exhibited a significant decline in activity for both dominance groups. The splint side sternocleidomastoid showed a similar significant decline in activity in the non-dominant group. In the dominant group , however, no significant change in muscle activity of the splint side sternocleidomastoid was recorded.

TABLE 8

Significance of Main Effects and Interactions on the Changes in Muscle Activities in Maximum Static Clench (No Splint)

| Source of variance | Level of Significance |
|---|---|
| Dominance | NS |
| Muscle | * |
| Dominance by muscle | NS |
| Side | * |
| Dominance by side | NS |
| Muscle by side | * |
| Dominance by muscle by side | NS |

NS = not significant
* = $p < .10$

TABLE 9

% Change in Muscle Activity Levels Due to Splint Wear Period in Maximum Static Clench (No Splint)

| | Non-splint Side | Splint Side |
|---|---|---|
| Anterior Temporalis Muscles | -3.5 NS | -0.2 NS |
| Sternocleido-mastoid Muscles | -57.1 *** | Dominant Group +0.8 NS |
| | | Non-dominant Group -43.1 *** |

positive value indicates increase in muscle activity
negative value indicates decrease in muscle activity
NS = not significant
* = $p < .10$
** = $p < .05$
*** = $P < .01$
in cases where the change in muscle activity was not significantly different for a muscle in the 2 dominance groups, the average change for the muscle is recorded
in cases where the change in muscle activity was significantly different for a muscle in the 2 dominance groups, the average change in activity for each group is recorded
dominant group = splint buildup on dominant side of subject
non-dominant group = splint buildup on non-dominant side of subject
standard error = 7.554 for muscles averaged for 2 groups
standard error = 10.682 for muscles of individual groups

Task 1s - Maximum Static Clench With the Splint

For task 1s, the change in the level of muscle activity (for each muscle, for each side) due to the subjects' occluding on the splint during the maximum static clench was recorded. A mixed analysis of variance was used to assess the effect of the muscles (anterior temporalis or sternocleidomastoid), side (splint side or non-splint side), dominance groups (dominant group or non-dominant group), and the interaction of these factors. Table 10 indicates that the muscles were a significant source of variation at the 10% level of significance and that the muscle by splint interaction was a significant source of variation at the 5% level of statistical significance. No other factor was a significant source of variation.

When the percentage changes of the muscles are examined (see Table 11), it can be seen that only the non-splint side anterior temporalis muscle showed a significant decline in activity in the two groups of subjects due to their occluding on the splint in the maximum static clench during the experimental days of testing. It can also be seen that there were no other significant changes in the levels of muscle activity.

TABLE 10

Significance of Main Effects and Interactions on the Changes in Muscle Activity Levels in Maximum Static Clench With the Splint

| Source of variance | Level of Significance |
|---|---|
| Dominance | NS |
| Muscle | * |
| Dominance by muscle | NS |
| Side | NS |
| Dominance by side | NS |
| Muscle by side | ** |
| Dominance by muscle by side | NS |

NS = not significant
* = $p < .10$
** = $p < .05$
*** = $P < .01$

TABLE 11

% Change in Muscle Activity Levels Due to Occlusion on Splint in Maximum Static Clench With the Splint

| | Non-splint Side | Splint Side |
|---|---|---|
| Anterior Temporalis Muscles | -44.4 *** | -3.3 NS |
| Sternocleido-mastoid Muscles | +12.9 NS | +4.1 NS |

positive value indicates increase in muscle activity
negative value indicates decrease in muscle activity
NS = not significant
* = p < .10
** = p < .05
*** = P < .01
in cases where the change in muscle activity was not significantly different for a muscle in the 2 dominance groups, the average change for the muscle is recorded
in cases where the change in muscle activity was significantly different for a muscle in the 2 dominance groups, the average change in activity for each group is recorded
standard error = 9.980

<u>Task 2 - Maximum Phasic Clench (No Splint)</u>

For task 2, or maximum phasic clench, the change in muscle activity due to the period of splint wear was recorded for each muscle, for each side. A mixed analysis of variance was used to assess the effects of dominance, muscle, and side, as well as the effect of the interaction of those factors.

Table 12 indicates that only dominance had a significant effect on the variation; no other factor had a significant effect.

Table 13 examines the percentage changes in muscle activity levels due to the splint wear period. Both sternocleidomastoid muscles showed significant declines in activity levels in the non-dominant group only. No other muscles demonstrated significant changes.

TABLE 12

Significance of Main Effects and Interactions on the Changes in Muscle Activities in Maximum Phasic Clench (No Splint)

| Source of variance | Level of Significance |
|---|---|
| Dominance | * |
| Muscle | NS |
| Dominance by muscle | NS |
| Side | NS |
| Dominance by side | NS |
| Muscle by side | NS |
| Dominance by muscle by side | NS |

NS=not significant
*=p < .10

TABLE 13

% Change in Muscle Activity Due to Splint Wear Period in Maximum Phasic Clench (No Splint)

| | Non-splint Side | Splint Side |
|---|---|---|
| Anterior Temporalis Muscles | +15.6 NS | +4.8 NS |
| Sternocleido-mastoid Muscles | Dominant Group<br>- 4.0 NS | Dominant Group<br>+12.6 NS |
| | Non-dominant Group<br>-56.4 *** | Non-dominant Group<br>-45.0 ** |

positive value indicates increase in muscle activity
negative value indicates decrease in muscle activity
NS = not significant
* = $p < .10$
** = $p < .05$
*** = $P < .01$
in cases where the change in muscle activity was not significantly different for a muscle in the 2 dominance groups, the average change for the muscle is recorded
in cases where the change in muscle activity was significantly different for a muscle in the 2 dominance groups, the average change in activity for each group is recorded
dominant group = splint buildup on dominant side of subject
non-dominant group = splint buildup on non-dominant side of subject
standard error = 7.554 for muscles averaged for the 2 groups
standard error = 10.682 for muscles of individual groups

## Task 2s - Maximum Phasic Clench With the Splint

For task 2s, the change in muscle activity levels due to the subjects' occluding on the splint, was recorded for each muscle, for each side. A mixed analysis of variance was used to assess the effects of dominance, muscle, and side, as well as the effect of the interactions of those factors.

Table 14 indicates that the muscle and the side had a significant effect on the change in muscle activity at the 10% level of significance. There were no other significant factors affecting the changes in muscle activity.

Table 15 displays the percentage changes in muscle activity levels due to the subjects' occluding on the splint. It indicates that only the non-splint side anterior temporalis showed a significant decline in activity levels. All other muscles displayed statistically similar activity levels.

TABLE 14

Significance of Main Effects and Interactions on the Changes in Muscle Activity Levels in Maximum Phasic Clench With the Splint

| Source of variance | Level of Significance |
|---|---|
| Dominance | NS |
| Muscle | * |
| Dominance by muscle | NS |
| Side | * |
| Dominance by side | NS |
| Muscle by side | NS |
| Dominance by muscle by side | NS |

NS = not significant
* = $p < .10$

TABLE 15

% Change in Muscle Activity Levels Due to Occlusion on Splint in Maximum Phasic Clench With the Splint

| | Non-splint Side | Splint Side |
|---|---|---|
| Anterior Temporalis Muscles | -64.1 *** | -10.4 NS |
| Sternocleido-mastoid Muscles | -21.4 NS | -23.5 NS |

negative value indicates decrease in muscle activity
NS = not significant
* = $p < .10$
** = $p < .05$
*** = $P < .01$
in cases where the change in muscle activity was not significantly different for a muscle in the 2 dominance groups, the average change for the muscle is recorded
in cases where the change in muscle activity was significantly different for a muscle in the 2 dominance groups, the average change in activity for each group is recorded
standard error = 16.787

## Task 3 - Partial Clench (No Splint)

For task 3, the change in muscle activity due to the period of splint wear was recorded for each muscle, for each side. A mixed analysis of variance was used to assess the effects of dominance, muscle, and side, as well as the effect of the interaction of these factors.

Table 16 indicates that none of the factors or interactions had a significant effect on the changes in muscle activity levels. Table 17 indicates that while some muscles, such as the non-splint side anterior temporalis showed a large change in muscle activity levels, no muscles displayed a significant change due to the splint wear period. The reason for this lack of significance is the large standard error.

TABLE 16

Significance of Main Effects and Interactions on the Changes in Muscle Activity Levels in Partial Clench (No Splint)

| Source of variance | Level of Significance |
|---|---|
| Dominance | NS |
| Muscle | NS |
| Dominance by muscle | NS |
| Side | NS |
| Dominance by side | NS |
| Muscle by side | NS |
| Dominance by muscle by side | NS |

NS = not significant

TABLE 17

% Change in Muscle Activity Levels Due to Splint Wear Period in Partial Clench (No Splint)

| | Non-splint Side | Splint Side |
|---|---|---|
| Anterior Temporalis Muscles | +67.9 NS | +22.0 NS |
| Sternocleido-mastoid Muscles | -4.2 NS | -12.6 NS |

positive value indicates increase in muscle activity
negative value indicates decrease in muscle activity
NS = not significant
in cases where the change in muscle activity was not significantly different for a muscle in the 2 dominance groups, the average change for the muscle is recorded
in cases where the change in muscle activity was significantly different for a muscle in the 2 dominance groups, the average change in activity for each group is recorded
standard error = 43.538

## Task 3s - Partial Clench With Splint

For task 3s, the change in muscle activity levels due to the subjects' occluding on the splint was recorded for each muscle for each side. A mixed analysis of variance showed that of the sources of variation tested, the factors of muscle and side both demonstrated a statistically significant effect on the change in muscle activity levels (see

Table 18). Table 19 displays the percentage changes in muscle activity levels due to the subjects' occluding on the splint. The non-splint side anterior temporalis muscle showed a significant decline in both dominance groups. The splint side sternocleidomastoid showed no change in activity levels in the dominant group, but the non-dominant group showed a statistically significant increase in the muscle's activity levels. The other muscles showed no significant changes in activity levels.

TABLE 18

Significance of Main Effects and Interactions on the Changes in Muscle Activities in Partial Clench With the Splint

| Source of variance | Level of Significance |
|---|---|
| Dominance | NS |
| Muscle | ** |
| Dominance by muscle | NS |
| Side | ** |
| Dominance by side | NS |
| Muscle by side | NS |
| Dominance by muscle by side | NS |

NS = not significant
* = $p < .10$
** = $p < .05$
*** = $P < .01$

TABLE 19

% Changes in Muscle Activity Due to Occlusion on Splint in Partial Clench With the Splint

| | Non-splint Side | Splint Side |
|---|---|---|
| Anterior Temporalis Muscles | -78.7 ** | +4.2 NS |
| Sternocleido-mastoid Muscles | 0.0 NS | Dominant Group 0.0 NS |
| | | Non-dominant Group +120.8 ** |

positive value indicates increase in muscle activity
negative value indicates decrease in muscle activity
NS = not significant
* = p < .10
** = p < .05
*** = P < .01
in cases where the change in muscle activity was not significantly different for a muscle in the 2 dominance groups, the average change for the muscle is recorded
in cases where the change in muscle activity was significantly different for a muscle in the 2 dominance groups, the average change in activity for each group is recorded
dominant group = splint buildup on dominant side of subject
non-dominant group = splint buildup on non-dominant side of subject
standard error = 25.594 for muscles averaged for the two groups
standard error = 39.095 for muscles of individual groups

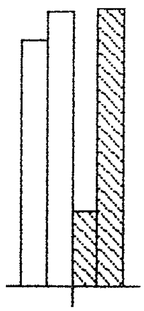
One must be cautious in the interpretation of the change in the activity levels of the sternocleidomastoid muscle in the partial clenching tasks because of the very low levels of muscle activity present.

Figure 6 demonstrates these low levels of activity in displaying the activity levels of the individual subjects for the initial levels in the control period and those recorded with the splint inserted in the experimental period. There were no consistent changes in the activity levels of the sternocleidomastoid muscles. Three subjects (no.'s 2, 3, and 7) showed no sternocleidomastoid activity at any time during the partial clench. The other subjects showed low average levels of activity: usually less than 1% of maximum levels and never more than 2.5%. Subject no.'s 1 and 8 showed a trend toward an increase on the non-splint side and a decline on the splint side. Subject no.'s 4, 5, and 6 demonstrated a different pattern. Subject no.'s 4 and 5 showed a decline in the non-splint side sternocleidomastoid activity levels to zero. Subject no.'s 4, 5, and 6 demonstrated an increase in the splint side sternocleidomastoid levels. In the case of subjects 4 and 6, the splint side muscle showed activity in the partial clench with the splint inserted when there was no sternocleidomastoid muscle activity on that side in the control period. All subjects demonstrated a large decline in the actvity of the non-splint side anterior temporalis with the splint in the mouth. There was some variation in the pattern of that change.

All subjects demonstrated a large decline in the activity of the non-splint side anterior temporalis with the splint in the mouth. Figure 6 displays the activity levels of the anterior temporalis mus-

Figure 6: Activity of the Sternocleidomastoid and Anterior Temporalis Muscles in Partial Clenching

For each muscle, for each subject:



Column 1 represents the average activity of the non-splint side muscle on the 2 control days

Column 2 represents the average activity of the splint side muscle on the 2 control days

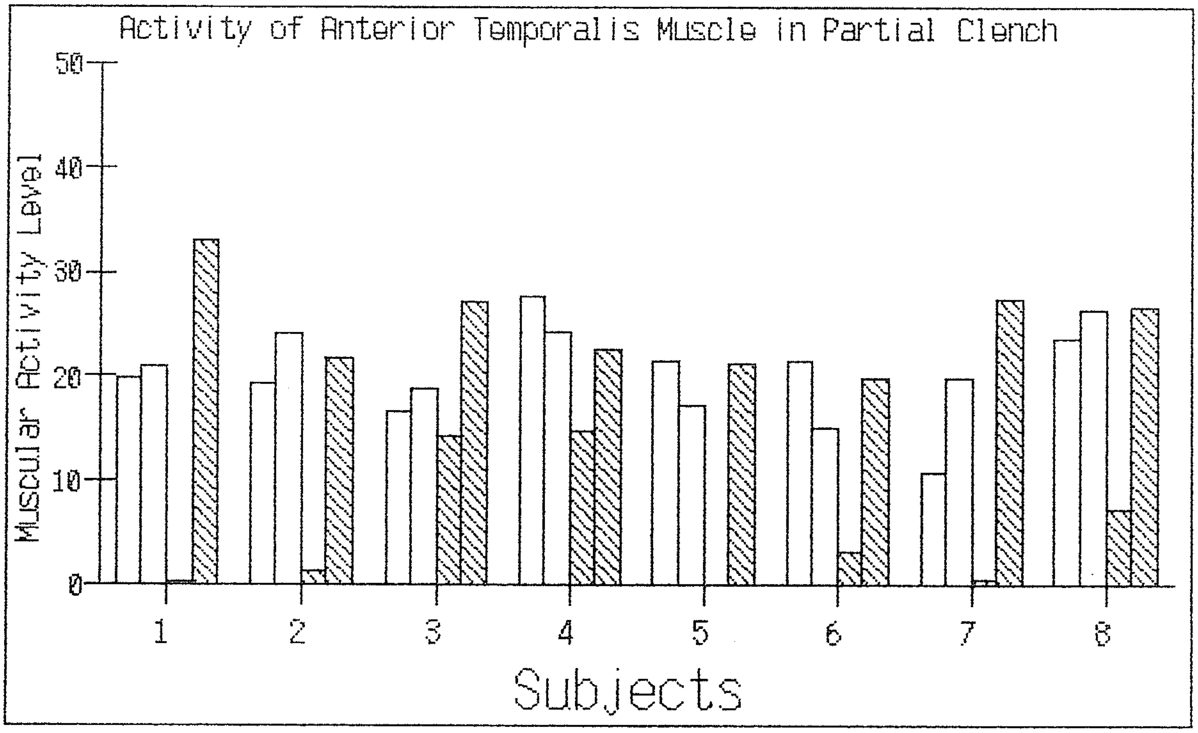
Column 3 represents the average activity of the non-splint side muscle on the 2 experimental days

Column 4 represents the average activity of the splint side muscle on the 2 experimental days

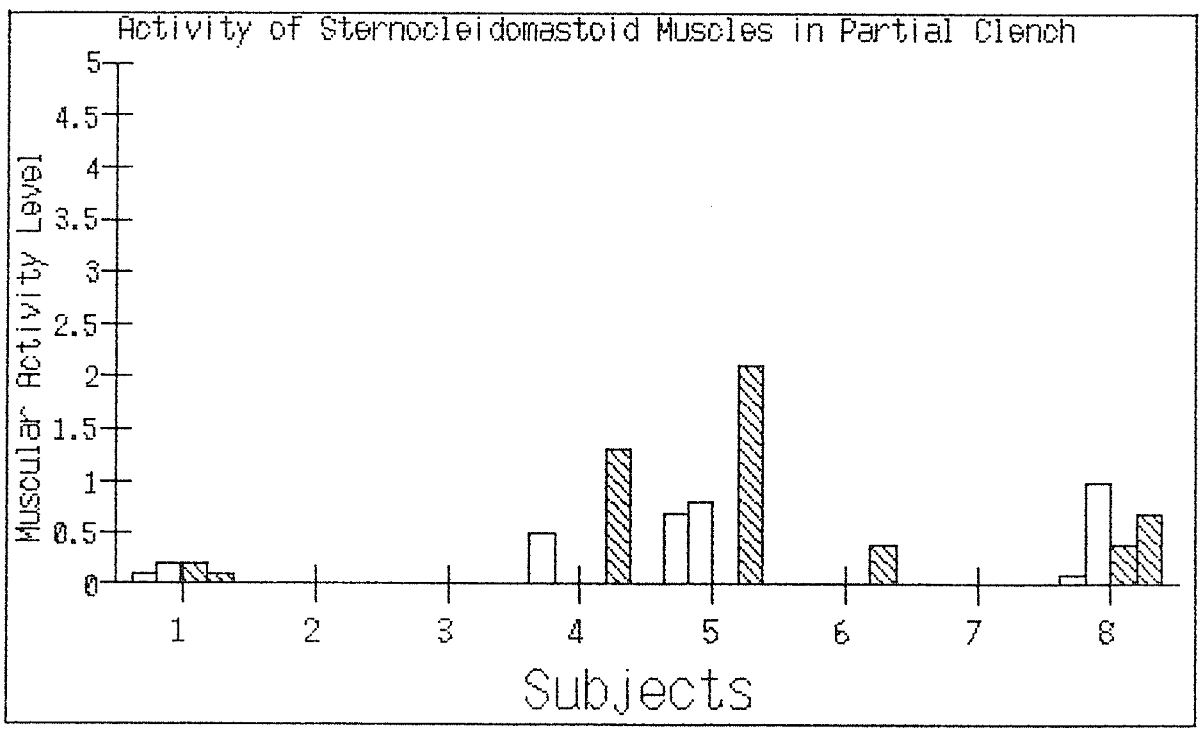
The vertical axis of the graphs indicates the muscular activity levels as a % of the daily maximum of that muscle

Columns with no bar indicate that no muscle activity was recorded for that muscle on those days

Figure 6



Activity of Anterior Temporalis Muscle in Partial Clench
Muscular Activity Level
50
40
30
20
10
0
1
2
3
4
5
6
7
8
Subjects

Activity of Sternocleidomastoid Muscles in Partial Clench
Muscular Activity Level
5
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0
1
2
3
4
5
6
7
8
Subjects

cles of the individual subjects from the initial levels in the control period to those recorded with the splint inserted in the experimental period. There was some individual variation in the pattern of change in the subjects. On the experimental days subject no.'s 1, 2, 5, and 7 showed a very low level of non-splint side anterior temporalis activity (approaching zero). Subject no.'s 6 and 8 showed a somewhat higher, although still very low level of activity. Subject no.'s 3 and 4 showed a higher level of non-splint side activity in the anterior temporalis, although the activity levels of the non-splint side were lower than the control values and lower than the splint side muscle.

## Task 4 - Side Flexion of the Head to the Splint Side

For task 4, the change in muscle activity levels due to the splint wear period was recorded for both sides of the sternocleidomastoid muscle. The splint side muscle was active as a prime mover in the side flexing of the head to the end of the range, whereas the non-splint side muscle was active in the return phase of the head movement back to the upright or neutral position. A mixed analysis of variance was used to assess the effect of the side and the dominance, as well as the interaction of the two factors. Table 20 indicates that none of the factors had a significant effect on the change in muscle activity levels due to the splint wear period. Table 21 illustrates the percentage changes in sternocleidomastoid activity levels. It can be seen that only the non-splint side muscle in the non-dominant group showed a significant increase in its activity level. The other muscles and group showed no statistically significant changes in the activity levels due to the splint wear period.

TABLE 20

Significance of Main Effects and Interactions on the Changes in Muscle Activity Levels in Side Flexion of the Head to the Splint Side

| Source of variance | Level of Significance |
| --- | --- |
| Dominance | NS |
| Side | NS |
| Dominance by side | NS |

NS = not significant

TABLE 21

% Change in Muscle Activity Levels Due to Splint Wear Period in Side Flexion of the Head to the Splint Side

| | Non-splint side Sternocleidomastoid Muscle | Splint side Sternocleidomastoid Muscle |
|---|---|---|
| Dominant Group | -3.1 NS | -10.0 NS |
| Non-dominant Group | +81.2 *** | +15.2 NS |

positive value indicates increase in muscle activity
negative value indicates decrease in muscle activity
NS = not significant
* = $p < .10$
** = $p < .05$
*** = $P < .01$
standard error = 19.295

## Task 5 - Side Flexion of the Head to the Splint Side

For task 5, the change in muscle activity levels due to the splint wear period was recorded for each side. A mixed analysis of variance was used to assess the effect of the side and the splint side group, as well as the interaction of the factors. Table 22 indicates that none of these factors had a significant effect on the changes in muscle activity levels due to the splint wear period. Table 23 shows the percentage changes in the, muscle activity levels due to the splint wear period. The non-splint side sternocleidomastoid muscle was active as a prime mover in the head movement, while the splint side muscle was active in the return phase. It can be seen that neither muscle demonstrated a significant change in activity levels in either splint side group.

TABLE 22

Significance of Main Effects and Interactions on the Changes in Muscle Activity Levels in Side Flexion of the Head to the Non-splint Side

| Source of variance | Level of Significance |
|---|---|
| Dominance | NS |
| Side | NS |
| Dominance by side | NS |

NS = not significant

TABLE 23

% Change in Muscle Activity Levels Due to Splint Wear Period in Side Flexion of the Head to the Non-Splint Side

| | Non-splint side Sternocleidomastoid Muscle | Splint side Sternocleidomastoid Muscle |
|---|---|---|
| Dominant Group | +16.0 NS | +6.6 NS |
| Non-dominant Group | -17.3 NS | -15.1 NS |

positive value indicates increase in muscle activity
negative value indicates decrease in muscle activity
NS = not significant
standard error = 20.863

Task 6 - Repetitive Bilateral Side Flexion of the Head

For task 6, the change in muscle activity due to the splint wear period was recorded for both sides of the sternocleidomastoid muscle and for both functions of the muscle. During the task (which is really a combination of the the two previous head moving tasks), both sternocleidomastoid muscles are active as prime movers and in the return phases of the movements.

A mixed analysis of variance was used to analyse the effect of the dominance, function of the muscle, side, and the interactions of these factors. Table 24 shows that only the dominance by side interaction had a significant effect on the change in muscle activity (at the 5% level of significance).

Table 25 illustrates the percentage changes in muscle activity due to the splint wear period. The splint side sternocleidomastoid showed no significant change for either function. The non-splint side sternocleidomastoid showed no increase for either function in the non-dominant group. In the dominant group, however, the non-splint side sternocleidomastoid showed significant increases for both functions: as a prime mover and in the return phase of the side flexion.

The changes in muscle activity levels are summarized in table 26 and table 27. Only the significant changes in muscle activity are shown; muscles not undergoing a significant change are marked as having no change. In cases where both dominance groups show similar changes, only the average figure for the muscle is displayed. In cases where the two dominance groups do demonstrate significant differences from each other, the activity levels change for each group is displayed.

TABLE 24

Significance of Main Effects and Interactions on the Changes in Muscle Activity Levels in Repetitive Bilateral Side Flexion of the Head

| Source of variance | Level of Significance |
|---|---|
| Dominance | NS |
| Muscle function | NS |
| Dominance by muscle function | NS |
| Side | NS |
| Dominance by side | ** |
| Muscle function by side | NS |
| Dominance by muscle function by side | NS |

NS = not significant
* = $p < .10$
** = $p < .05$
*** = $P < .01$

TABLE 25

% Change in Muscle Activity Levels Due to Splint Wear Period in Repetitive Bilateral Side Flexion of the Head

| | Non-splint Side | Splint Side |
|---|---|---|
| Sternocleido- mastoid Muscle as a Prime Mover | Dominant Group +79.3 *** | +16.1 NS |
| | Non-dominant Group -18.4 NS | |
| Sternocleido- mastoid Muscle in Return Phase | Dominant Group +79.5 *** | +13.6 NS |
| | Non-Dominant Group -4.1 NS | |

positive value indicates increase in muscle activity
negative value indicates decrease in muscle activity
NS = not significant
* = p < .10
** = p < .05
*** = P < .01
in cases where the change in muscle activity was not significantly different for a muscle in the 2 dominance groups, the average change for the muscle is recorded
in cases where the change in muscle activity was significantly different for a muscle in the 2 dominance groups, the average change in activity for each group is recorded
dominant group = splint buildup on dominant side of subject
non-dominant group = splint buildup on non-dominant side of subject
standard error = 12.791 for muscles averaged for 2 groups
standard error = 18.089 for muscles of individual groups

TABLE 26

Summary of the Changes in Muscle Activity Levels in Clenching Tasks

| Task | Anterior Temporalis Non-Splint Side | Anterior Temporalis Splint Side | Sternocleidomastoid Non-Splint Side | Sternocleidomastoid Splint Side |
|---|---|---|---|---|
| 1 - Maximum Tonic Clench (No Splint) | NSC | NSC | -57.8% *** | Dominant Group NSC<br>Non-Dominant Group -43.1% *** |
| 1s - Maximum Tonic Clench With Splint | -44.4% *** | NSC | NSC | NSC |
| 2 - Maximum Phasic Clench (No Splint) | NSC | NSC | Dominant Group NSC<br>Non-Dominant Group -56.4% *** | Dominant Group NSC<br>Non-Dominant Group -45.0% *** |
| 2s - Maximum Phasic Clench With Splint | -64.1% *** | NSC | NSC | NSC |
| 3 - Partial Clench (No SPlint) | NSC | NSC | NSC | NSC |
| 3s - Partial Clench With Splint | -78.7% ** | NSC | NSC | Dominant Group NSC<br>Non-Dominant Group +120.8% ** |

positive value indicates increase in muscle activity
negative value indicates decrease in muscle activity
NSC = no significant change
** = $p < .05$
*** = $P < .01$
in cases where the change in muscle activity was not significantly different for a muscle in the 2 dominance groups, the average change for the muscle is recorded
in cases where the change in muscle activity was significantly differ-

ent for a muscle in the 2 dominance groups, the average change in activity for each group is recorded
dominant group = splint buildup on dominant side of subject
non-dominant group = splint buildup on non-dominant side of subject

TABLE 27

Summary of the Changes in Muscle Activity Levels in Head Moving Tasks

| Task | Anterior Temporalis Non-Splint Side | Anterior Temporalis Splint Side | Sternocleidomastoid Non-Splint Side | Sternocleidomastoid Splint Side |
|---|---|---|---|---|
| 4 - Head Flex To Splint Side | NSC | NSC | Dominant Group NSC<br>--------<br>Non-Dominant Group +81.2% *** | NSC |
| 5 - Head Flex To Non-Splint Side | NSC | NSC | NSC | NSC |
| 6 - Repetitive Bilateral Side Flexion | Dominant Group +79.3% ***<br>--------<br>Non-Dominant Group NSC | NSC | Dominant Group +79.5% ***<br>--------<br>Non-Dominant Group NSC | NSC |

positive value indicates increase in muscle activity
NSC = no significant change
*** = $P < .01$
in cases where the change in muscle activity was significantly different for a muscle in the 2 dominance groups, the average change in activity for each group is recorded
dominant group = splint buildup on dominant side of subject
non-dominant group = splint buildup on non-dominant side of subject

# Chapter IV

## DISCUSSION

The objective of this study has been to assess the effect of a one week period of unilateral occlusal contact on the following factors:

1. The deviation of the natural resting head posture in the frontal plane.
2. The difference between the range of motion in side flexion of the head to the splint and the non-splint side.
3. The EMG activity levels of the sternocleidomastoid and anterior temporalis muscles during the performance of several clenching and head moving tasks.

The subjects did not display any signs and symptoms of TMD or neck pain and dysfunction prior to the experimental period. They were tested with respect to the above factors before the period of splint wear and at the end of the splint wear period, so that the subjects served as their own controls. In addition, the subjects' subjective reactions to the alterations in their occlusions were recorded. Data was analyzed using a mixed analysis of variance as well as a Student's t-test to determine the significance of the changes due to the period of splint wear. The results of the factors studied will be discussed separately, but will be related to each other whenever appropriate.

## HEAD POSTURE

There were no significant changes in the head posture of the subjects, in the frontal plane, due to the splint wear period. Both splint side groups demonstrated a head posture which was consistently within 1 degree (on average) of upright (with the horizontal line through the eyes, perpendicular to the vertical).

These findings are not surprising, given the important role of the head in the basic orientation system of the body. This perceptual system operates through a complex interaction among the vestibular, visual, and kinesthetic mechanisms (Bartley, 1970), two of which are found in the head. The neck muscles do appear to play an important role in the kinesthetic mechanisms of the basic orientation system (Cohen, 1961; Abrahams, 1972; Abrahams, 1977; Richmond and Abrahams, 1979; Manzoni et al, 1979; Barnes and Forbat, 1979; Bakker and Richmond, 1980; Boyle and Pompeiano, 1981; Bizzi, 1981).

It is clear however, that the orientation system must furnish the individual with an internal frame of reference which is aligned with the external frame of reference including the gravitational vertical. It would seem likely therefore, that the system would be resistant to forces which might upset that internal frame of reference. The head posture in the sagital plane shows greater variability; this may be due to the priority of airway maintenance which would require plasticity in the control of head posture in the sagital plane, but not the frontal plane. Bartley (1970) stated that any two of the three mechanisms involved in the orientation system can maintain an adequate posture. Interestingly, the results of the pilot study indicated that the head posture changes were no greater if the subjects' eyes were closed during testing.

Schneider and Bartley (1962) examined the effect of asymmetrical tension in the neck muscles on one side due to torque on the head in the frontal plane, achieved by adding weights to one side of the head. This required the subjects to increase the activity and tension of the neck muscles on the contralateral side in order to maintain a level head posture. They found that the unilaterally increased tension caused the subjects' visual perception of the vertical to be altered in the direction opposite the side of the increased tension. As the tension in the neck muscles increased, so too did the deviation of the subjects' visual vertical, indicating that neck tension can potentially alter the subject's perception of the visual vertical and perhaps lead to an altered head posture conforming to this altered perception of the vertical. Two subsequent studies (Bizzi et al, 1976; and Bizzi et al, 1978) have provided further evidence that tension in the neck muscles can alter the position of the head (in monkeys). The short period of splint wear in the present study may not have been sufficient to produce a significant alteration in the tension of the neck muscles.

A related phenomenon is that of dizziness which has been associated with TMD as a secondary peripheral symptom. The mechanism for the development of dizziness in TMD has not been established, but Myrhaug (1969 and 1970), an otologist, found a high incidence of balance problems in a large group of TMD patients he studied. He related this to hyperactivity and spasm of the tensor tympani muscle which is innervated by the trigeminal nerve. There is however, little evidence to support this.

A more plausible explanation is that pain and dysfunction of the neck muscles can produce dizziness. The large number of studies emphasizing the importance of neck muscles in the kinesthetic mechanisms of orientation lend evidence to this suggestion. Ingarashi et al (1969) and Manzoni et al (1979) found that neck lesions can produce significant defects in posture and locomotion in monkeys. In humans, Travell and Weeks (1955) and Travell (1981) have each reported a case in which dizziness was related to dysfunction in the sternocleidomastoid muscle. Sharav et al (1978) examined TMD patients and found that dizziness was significantly correlated with pain in at least one sternocleidomastoid muscle. The potential for dizziness caused by neck muscle lesions is widely supported (Gray, 1956; Cope and Ryan, 1959; Sandler, 1967; and Jongkees, 1969). Brookes et al (1978) concluded after their intensive study that:

> it is possible for the chronic neck aches, often coincidental in the temporomandibular dysfunction syndrome, to act as an additional factor in association with underlying autonomic instabiliy to cause labyrinthine dysfunction and aural symptoms.

It has also been suggested that the use of occlusal splints can improve athletic performance (Eversaul, 1985). Recent studies have shown that occlusal splints do not increase the strength of various muscles in atheletes (Hart et al, 1981; and Schubert et al, 1984).

It is interesting to speculate that neck muscle dysfunction may be the mechanism at work to explain the improvement in athletic performance which some people claim with occlusal splints. Given the importance of the neck muscles in balance and orientation, dysfunctions of those muscles could adversely affect the performance of athletes by upsetting their sense of equilibrium, perhaps in very subtle, but significant ways. Keele (1981) stressed the importance of reflexes, and particularly the tonic neck reflex, in muscle synergies. He suggested these reflexes may be important for strength and coordination. Cooper et al (1982) held that neck reflexes are important in sports. Gowitzke and Milner (1980) stated: "with the possible exception of the spindle reflexes, neck reflexes are probably the most important single reflex mechanism used in sports skills". If the occlusion can adversely affect the neck muscles it opens up a number of avenues of investigation. It indicates that one should perhaps be measuring neck function in athletes as well as coordination and balance before and after an occlusal splint has been worn, rather than simply measuring strength. It also suggests that normal subjects with no occlusal abnormality or neck dysfunction would not benefit from an occlusal splint.

## RANGE OF MOTION

The splint wear period did produce a significant amount of asymmetry in the side flexion range of motion to the two sides in both dominance groups. This change was generally in the direction of a greater range in side flexing to the splint side, except in two subjects where the range was greater toward the non-splint side on at least one experimental day. Furthermore, some subjects demonstrated that this asymmetrical range was reduced on the second experimental day.

Although a number of tissues can potentially affect the range of motion of a joint system (Wells and Luttgens, 1976), it would seem reasonable to conclude that the significant changes in the range of motion seen in this study were due to changes in the muscles of the neck. The only other possible factor causing such a rapid change in range would be pain limiting the movement. No subject however, complained of pain during the range of motion testing and all believed that it was a structural element in the neck which limited the range to one side.

Gossman et al (1982) stated that movement dysfunction can be caused by both structural and reflex mediated length changes in muscle. They stressed that muscle is a very mutable tissue which displays more changes when it is shortened including anatomical, biochemical, and physiological changes. They suggested that clinical evidence indicated that changes in muscle length could occur in hours or days. The potential speed and magnitude of this change was demonstrated in the present study by one subject whose range of motion difference changed 15 degrees in a 24 hour period during the experimental period.

Partridge and Benton (1981) stated that muscles demonstrate "a memorylike retention of effects of mechanical history". So that if a muscle is held at a stationary length and stimulated moderately, it will become stiff, at least in the short-term. Moore and Hutton (1980) stated that "a muscle is initially more resistant to change in length after a static contraction". This could explain the development of the asymmetrical range of motion seen in the subjects in this study. Unilateral activity of a neck muscle or group of muscles may produce stiffness in those muscles leading to a decreased range when the subject is asked to perform a movement requiring those muscles to be stretched. In side flexion a number of muscles on the side contralateral to the movement must be stretched to permit the full range of movement. These muscles include the sternocleidomastoid and the scalene muscles; both can limit the range of motion in side flexion if they are tight (Gould, 1985).

The subjects' feelings of unilateral neck tension correlated well with the range of motion measurements. Seven out of the eight subjects felt increased tension on the splint side of the neck on at least one of the experimental days and six of these subjects felt that this increased tension limited their range of motion in side flexing to the opposite, non-splint side. Subject no.2 believed that the increased tension he felt, limited his side flexion to the same side (measurements confirmed this). The reason for this reaction is not clear, but it could very well have been due to involvement of other neck muscles, of which the subject was not directly aware. The only subject not to have felt increased tension on the splint side was subject no.6, whose range of motion was only significantly asymmetrical on the first experimental day. This may have indicated that this subject's neck muscles were not affected to the same extent as those of the other subjects.

Gardner-Medwin and Walton (1974) pointed out, although they did not cite references, that muscles can demonstrate contractures wherein one finds a rearrangement of collagen fibrils. Initially this is thought to be reversible by repeated stretching, but it may develop into an actual fibrosis which is permanent. Odeen (1981) and Lewit and Simons

(1984) demonstrated that muscle stretching increased the range of motion and decreased associated pain. In the present study, both the EMG and the range of motion testing produced streching of the neck muscles because the subjects were instructed to side flex their heads to the end of the range. This may have stretched out most of the muscular tightness in the subjects who showed a decreased asymmetry in the range of motion on the second experimental day. The other subjects, not showing the improvement in range of motion symmetry, may have showed a greater asymmetry in their range had they not been subjected to significant muscle stretching on the first experimental day.

The biological significance of the asymmetrical range of motion is unknown, but was probably minimal for the subjects in the present study due to its short duration. If the condition had remained for some time, the consequences may have been more significant. Crossman et al (1984) suggested that a reduced range of motion can put stress on joints with the potential of producing dysfunction. In theory, muscle dysfunction may give rise to joint dysfunction (Rose and Rothstein, 1982). Janda (1982) stressed dysfunction of the entire motor system which often includes tightness and imbalances in muscle tension and strength as an important part of the condition, demanding early treatment. Grieve (1979) believed that "asymmetrical tethering" of the neck due to tightened muscles could be a significant problem, although this was often neglected. Johnston et al (1985) studied the kinematics of cervical function and concluded that asymmetrical cervical side flexion "appeared to be an early indicator of a measurable impairment of cervical function". Cooper et al (1982) stressed the importance of range of motion and made the point that decreased range of motion and faulty postural alignment were related.

## EMG-CLENCHING TASKS

In the clenching tasks, it is important to note that the effect of two factors were being studied:

1. the subjects' wearing of the unilateral splints over the course of the experimental period
2. the subjects' performances of the specific tasks while actually wearing the unilateral splints.

During tasks 1, 2, and 3, therefore, the subjects were not wearing the splints while the tasks were being performed. Thus, only the effect of the splint wear period over the experimental period was being tested. During tasks 1s, 2s, and 3s, on the other hand, the subjects were occluding on the splints at the time of the EMG recording. These activity levels recorded in these tasks were then compared to the activity levels recorded in the same tasks performed at the same recording session, but without the splint in the mouth (tasks 1, 2, and 3). Tasks 1s, 2s, and 3s therefore, only tested the change in the subjects' activity levels due to their occluding on the splint during the same experimental recording session.

In both task 1 and 2 , or the maximum static and phasic clenching task without the splint, neither splint side group showed a significant change in the activity of the anterior temporalis muscles. The sternocleidomastoid muscle activity declined in in all cases except the splint side muscle in the dominant group.

Only one other study has examined the activity of the sternocleidomastoid muscles during clenching tasks (Davies, 1979) and this study did not examine levels of activity. It only demonstrated that the sternocleidomastoid muscles were active in various mandibular tasks, including clenching. If the sternocleidomastoid muscle is functionally related to the muscles of mastication as Davies (1979) suggested, then one can only speculate that the period of splint-wear altered this relationship in some way.

In tasks 1s and 2s, the effect of the subjects' occluding on the splint in both a phasic and tonic maximum clench was tested during the experimental period of splint wear. Occlusion on the unilateral splint did not lead to any changes in activity levels of the sterno-cleidomastoid muscles despite a significant decline in the non-splint side anterior temporalis. It would seem that the activity of the sternocleidomastoid in clenching was not directly related to the simultaneous activity of the anterior temporalis.

For task 3, the partial clenching task, no muscles showed a significant change in activity levels due to the splint wear period. The non-splint side anterior temporalis increased by more than 65%, but the very large standard error rendered this change insignificant. The trend toward an increase in activity of this muscle was probably due to the tendency of the upper teeth on the non-splint side to erupt slightly. This produced minor alterations in the subjects' occlusion with the splint removed. All subjects were aware of these changes, but they did not last for more than several days after the end of the study. These changes in occlusion did not produce any changes in the activity levels of the sternocleidomastoid muscles in either dominance group.

In task 3s, the effect of the subjects' occluding on the splint was tested during the partial clench task. The anterior temporalis on the non-splint side demonstrated a significant decline of almost 80%. The splint side anterior temporalis was the muscle monitored by the subjects during clenching so that it was not expected to show any significant changes in activity levels.

The changes (or lack thereof) in the activity levels of the sternocleidomastoid muscles must be interpreted carefully because, in a number of cases, subjects displayed very little or no sternocleidomastoid muscle activity in the partial clenching tasks. This does not mean that no activity was present, but that the EMG equipment did not detect any activity above the noise floor of the equipment. DeVries (1965) emphasized that very sensitive equipment is required to detect very low levels of muscle activity.

The use of the partial clenching task was designed to test the subjects' responses similar to what they might have been doing with the splint during the experimental period. It was discovered during the pilot study that, with the splint inserted, subjects alternated between relaxing and clenching their mandibular muscles. They rarely found themselves clenching on the splint with a maximum effort; more often, they clenched on the splint moderately with an anterior temporalis activity level which was measured to be approximately 20 to 25% of maximum. Different subjects however, may very well have clenched on the splint with different amounts of force at different times.

This partial clenching task, therefore, was only an estimate of the subjects' reaction to the splint.

Of the five subjects demonstrating any sternocleidomastoid activity during the partial clench task, subjects no.'s 4, 5, and 6 demonstrated the most interesting reactions to the splint. With the splint in the mouth, each of these subjects either abolished the activity of the non-splint side sternocleidomastoid muscle or produced activity in the splint side muscle where there was none without the splint.

The large differences in the sternocleidomastoid activity levels during the moderate clenching tasks may be related to different thresholds in subjects. Some subjects may show significant sternocleidomastoid activity at a partial clench of 10% of maximum, whereas others may not show any activity until 40% of maximum. Moreover, the threshold for sternocleidomastoid activity may be different for the two sides in the same subject.

It is interesting to speculate that the subjects all may have demonstrated the changes in sternocleidomastoid activity levels seen in subject no.'s 4, 5, and 6, at the clenching levels they habitually achieved during the splint wear period. This remains highly speculative until further testing can be performed on the activity levels of the sternocleidomastoid muscles at different levels of partial clenching, and of the levels of partial clenching achieved by subjects during a period of unilateral splint wear.

Another significant factor of the subjects' reaction to the splint would have been the amount of time each subject occluded (or bruxed) on the splint. This factor is also unknown, but probably had a significant effect on the muscular reaction to the splint wear period.

The reaction of the anterior temporalis to the splint was expected and was, in fact, the purpose of the unilateral splint used. Specifically, the unilateral splint was intended to produce an asymmetry in mandibular elevator activity, with decreased activity on the non-splint side, while maintaining activity on the splint side. Bakke and Moller (1980) found that on the side of an occlusal interference, the activity levels of the masseter and temporalis muscles were unchanged, whereas those on the non-interference side declined significantly. Bakke and Moller (1980) examined interferences of less than 1 mm., but Moller (1975) and Rasmussen and Moller (1975) found similar results for unilateral onlays with vertical openings similar to those used in the present experiment.

The primary reason for this asymmetrical elavator activity is believed to be due to the absence of contact on the non-splint or non-interference side. Lund and Lamarre (1973) demonstrated that, at least under some conditions, the periodontal receptors are stimulated, producing positive feedback to achieve normal levels of mandibular elevator activity. Lund and Lamarre (1973) also showed that the mandibular elevator response to the feedback was not normally under the subject's conscious control. The subjects in the present study did not have any opportunity to develop control of this feedback reaction. It is reasonable therefore, to believe that the asymmetrical elevator activity during the partial clench task was an accurate indication of the subjects' reactions to the splint over the entire experimental period and not a learned or willed response.

Sheikholeslam and Riise (1983) also suggested that the temporomandibular joint receptors may modify the activity of the mandibular elevators. This could also have been responsible for the changes in mandibular activity seen in the present situation. If the subject was to have equal bilateral activity of the mandibular elevators with the unilateral occlusal contact, the longer lever arm on the non-splint side would tend to unseat the condyle on the splint side. In this situation, it would not be unexpected that the temporomandibular joint receptors would sense this unseating of the condyle and inhibit the activity of the elevators on the non-splint side. This is only speculative, however.

Wood and Tobias (1984) also used a unilateral splint to test the reaction of the mandibular elevators to an alteration in occlusal contact. They found no alteration in activity levels of the elevators on either side, but their subjects did not wear their splints for any significant length of time as they did in the present study.

MacDonald and Hannam (1982) concluded that the patterns of muscle activity "were consistently dependant upon the position and number of occlusal stops employed". They found different patterns of activity depending on whether the contact was on the working side or the balancing side of the jaw. It was for this reason, that the present experiment used a unilateral splint rather than a single interference. In the latter case, subjects may shift their jaw to one side or the other, to obtain a more comfortable occlusion. The side to which the jaw is deviated may then affect the subject's reaction to the interference. It was hoped that a unilateral splint, with flat occlusal contact, would not produce any lateral deviation of the mandible and so obviate any working-side versus balancing-side effects. It was found in the pilot studies, and later confirmed in the study, that subjects did seem to occlude on the splint with no lateral deviations. This finding agreed well with Smith (1985) who believed that the condyles are more resistant to stress when the mandible is in the midline.

## EMG - HEAD MOVING TASKS

For tasks 4 and 5, simple unilateral side flexion, there were no significant changes in activity levels of the sternocleidomastoid except in the subjects wearing the splint on the non-dominant side who demonstrated a significnt increase in the activity of the non-splint side sternocleidomastoid muscle.

For task 6, in the continuous bilateral side flexion, the splint-side sternocleidomastoid muscles showed no significant changes for either splint-side group during activity as a prime mover or during the return phase of the movement. The non-splint side muscle also showed no change in activity levels in the non-dominant group during either activity. The non-splint side muscle in the dominant group, however, did demonstrate a significant increase in activity levels both during function as a prime mover and in the return phase of the head movement.

The interpretation of these results is made difficult by the fact that no one has studied the activity of the sternocleidomastoid muscle during head movements of subjects with acute changes in their range of motion. Sturgis et al (1983) did examine the activity of several muscles, including the sternocleidomastoid, in various dynamic tasks. Their purpose was to compare a group of subjects suffering from chronic headaches to a group of headache-free controls. They did not take into account the range of motion of the subjects, however, and the head movements included a significant amount of shoulder movement so that the results are not comparable to the present study. Wolf et al (1979) examined low back range of motion and EMG activity levels in a large group of control subjects in an attempt to develop a normative data base to which patients with dysfunction could be compared. Unfortunately, they did not attempt to correlate the two factors.

The subject of low back pain has attracted much more research than that of neck pain and dysfunction. Unfortunately, Sherman (1985) found the literature examining EMG activity in patients with low back pain to be confusing and contradictory. In his study, Sherman (1985) stressed the importance of individual patterns of EMG activity associated with pain and dysfunction in the lower back. He found that in the chronic patient, there was no single predictable pattern of EMG abnormality. Moreover, the EMG activity pattern existing in a patient did not seem to correlate with any factor such as the severity of the pain or dysfunction, or its etiology.

The present study remains very different from all other studies looking at dysfunction in the axial skeleton because it is a prospective study examining the early stages of dysfunction in a healthy subject. All other studies have examined patients with chronic conditions, of a long standing and complex nature. One would expect that a group of patients with the identical initial injury or stress factor would show more similar patterns of dysfunction in terms of EMG, range of motion, and other factors. With time however, the progression of these patients' dysfunctions will diverge as numerous factors such as age, lifestyle, pain tolerance, joint dysfunction, posture, anatomical factors, treatments, and others, interact to alter the pattern of dysfunction.

In the interpretation of the results of the present study, the most consistent and interesting finding is that the dominant group showed significantly higher sternocleidomastoid activity levels on the non-splint side for both activities in side flexing in task 6: as a prime mover and in the return phase of the movment. A tight (resistant to stretch) sternocleidomastoid muscle can limit side flexion (Gould, 1985) so that the muscle of the opposite side involved in performing the movement, might reach higher levels of activity to overcome the increased resistance. A tight splint side sternocleidomastoid would also explain the decreased range in side flexion to the non-splint side which was found in all subjects, and in the dominant group in particular. One would have expected a similar higher activity level of the non-splint side in the non-dominant group, but their range of motion was not affected to the same extent as the dominant group. Furthermore two members of the non-dominant group showed that the tightness they had in the splint side muscle was easily stretched out and was not therefore, as severe.

Again one is presented with differences in response to the splint based on the relationship between the splint buildup side and the side of dominance of the subject. Interestingly, one subject in the non-dominant group showed the same pattern of increase in the activity level of the non-splint side as was found in the dominant group. This subject did not display a less asymmetrical range on the second experimental day indicating that he may have been affected to a greater extent than the others in the non-dominant group.

Janda (1986) commented that the interpretation of these findings, based on the suggestion that the sternocleidomastoid on the splint side had a greater resistance to stretch, was only speculative. He stressed the complexity of the biomechanics of the neck which precludes a simple antagonistic relationship between contralateral muscles.

Unfortunately, the pattern of activity level changes in the sternocleidomastoids in the repetitive side flexing task was not seen in the other simple side flexing tasks. This lends evidence to the assertion that the pattern of dysfunction in the present study was probably more complex than simply the increased resistance to stretch of a single neck muscle. The non-dominant group did demonstrate an increased level of activity in the non-splint side sternocleidomastoid in side-flexing to the splint side during the return phase of the movement; a finding which would be consistent with increased resistance to stretch in the splint side muscle. One would also have expected the non-splint side muscle to be affected in side flexing to the non-splint side, but there was no significant change. The lack of any significant changes in the dominant group in the unilateral side flexing tasks demonstrates that these tasks are not as similar to the repetitive bilateral side flexing task as they appear.

The side flexing tasks may have differed in the degree to which the subjects attempted to reach the end-point of their range of motion. Ideally one would measure the subjects' movements with an electrogoniometer, simultaneously with the EMG recordings. One can only assume that the subjects were side flexing to the same point as they did when their range of motion was tested with the goniometer prior to the EMG recording. This would seem a reasonable assumption considering the low standard errors for the range of motion measurements and that all subjects were given the same instructions in each case.

Another complicating factor was that, while a subject may not have side flexed a different amount in different tasks, the subject may have expended more or less effort in one task over another. This does not preclude comparisons, but it does mean that tasks which appear to be very similar may be quite different from the standpoint of EMG activity.

In the repetitive side flexing task, where the muscles were being alternately contracted and then stretched, one could speculate that the initial contraction in the affected splint-side muscle may have produced a lingering facilitation in that muscle. This may have increased the stiffness and resistance to stretch when the muscle was immediately called upon to relax. It must be stressed that stiffness in a muscle does not always reduce the range of motion, but rather it may simply make the muscle more resistant to stretch within the same range (Johnston et al, 1985). In the simple unilateral side flexing tasks, the muscle which was contralateral to the movement (the muscle which will be stretched by the movement) was not actively contracted prior to its being stretched. This may have produced a lower stiffness within its range of motion than in the previous case.

## MECHANISM HYPOTHESIS

The significant changes seen in the present sample were consistent with the widely held clinical concept that mandibular dysfunction can produce dysfunction in the muscles of the neck. The results do not conclusively prove such a relationship exists because of the small sample, the short time period of the study, and the fact that the provocation of the unilateral splint did not produce frank mandibular dysfunction. Nevertheless, the unilateral splint did appear to produce significantly asymmetrical mandibular elevator activity and this is often found in cases of temporomandibular dysfunction (Bakke and Moller, 1979). It is also potentially dangerous and unethical to produce a frank case of TMD in an asymptomatic subject. Even if one does this however, one is studying an acute case which may be different from the chronic case (which describes most of the clinical cases seeking treatment). There are advantages to studying the early, acute case (as outlined earlier), but one cannot assume it is identical to the chronic case. Moreover, the reaction to frank TMD pain and dysfunction may cause neck dysfunction simply as a splinting reaction to the head pain, whereas the present results are more likely due to a functional relationship. In addition, the present study is significant in demonstrating that the evidence of neck dysfunction appeared after less than one week of the occlusal disturbance.

The provocation of the short-term wear of a unilateral splint does appear then, to be a reasonable model to study the effects of asymmetrical mandibular elevator activity on the system. Given this, what is the mechanism by which this asymmetrical mandibular activity produces the mild, but significant neck dysfunction seen in this study?

The fundamental cause of neck involvement in TMD would seem to lie in the intimate functional and anatomic relationship between the head and neck muscles, and regional structures in general. There is ample evidence that the muscles of the head and neck (and particularly the anterior temporalis and sternocleidomastoid) are related in the case of both migraine and tension headaches (Simons et al, 1943; Bakal and Kaganov, 1977; Olesen, 1978; Lous and Olesen, 1982; and Hudzinski, 1983). This relationship could be due to a splinting type of reaction in one set of muscles as a response to pain in a nearby region, as Simons et al (1943) found. There is a significant body of evidence relating headaches and temporomandibular dysfunction (Magnusson and Carlsson, 1978; Magnusson and Carlsson, 1980; Magnusson, 1980; Reik and Hale, 1981; and Magnusson, 1982). While this splinting reaction to pain almost certainly occurs to produce neck involvement, the functional relationship between the masticatory system and the neck muscles also appears to be an important factor in the development of neck dysfunction.

The present study indicates the existence of this strong functional relationship between mandibular function and the neck muscles, because all of the subjects displayed some changes in their neck muscles due to the splint wear period, yet none of them experienced sigificant pain lasting more than 8 hours. Jimenez (1986) suggested that the activities of the muscles of mastication are modified by a complex integrated input from a number of sources including receptors in the periodontal ligament, temporomandibular joint, periosteum, and muscles. This study suggests that an equally complex input from the oral region may be stimulating and modifying the activity of the sternocleidomastoid muscle and probably other neck muscles as well.

The reasons behind this possible relationship are open to conjecture, but it is reasonable to believe that the neck muscles are active in mandibular tasks in order to stabilize the head. Smith (1984), in his model of the muscles of mastication and temporomandibular joint loading, found that the system is not well suited to resolve lateral forces on the teeth and the mandible. This problem could be solved by closing the mandible firmly to 'lock' the occluding teeth together, and the mandible to the skull. The neck muscles could then actively resist the lateral forces developed by the mandibular movement or activity. One can readily observe this mechanism at work when a dog tears at an object with its teeth, or when a human rips a piece of toffee or licorice with his teeth.

These situations are, however, quite rare (particularly in the case of humans), so one is left to wonder why there would be relatively low, but significant activity in normal vertical clenching tasks such as the one used in the study (and presumably during the entire period of splint wear). It may be that in any number of mandibular tasks, including clenching, the neck muscles including the sternocleidomastoid, are stimulated to a low activity level to prepare them for the possibility of being called upon to act as significant stabilizers of the mandible and the head.

Marsden et al (1978) found that more distant muscles react with short latencies to load changes in prime movers. They originally believed that this reaction was simply due to stretch reflexes in the prime mover muscles, but the very short latency periods suggested to them that the effect was centrally driven by the afferent input to the prime mover. The more distant muscles anticipate the effect of the load changes upon the prime mover and tend to minimize them. They further suggested that the organization of responses must be very complex. Tanji and Evarts (1976) found activity in the motor cortex which preceeded the intended movement. They found that there was directional specificity in the activity of motor cortex units, which was related to the details of the impending muscular contraction.

It is also of interest to note that it has been suggested that orofacial dyskinesia which is a neurological disorder involving excessive uncoordinated movements of the face, jaw, tongue, and neck, may be related in some patients to occlusal imbalances (Sutcher et al, 1973; Farrar, 1976; and Sutcher and Sugar, 1982). These clinicians have treated such patients with occlusal therapy and have observed significant improvements in their condition.

The steps in the development of the dysfunction seen in the present study would seem to be as follows:

1. The unilateral splint produced primarily unilateral activity and stimulation of the ipsilateral elevators.

2. The asymmetrical occlusal table could have caused a significant amount of parafunction in the subjects. There is no direct evidence for this, although it is a common clinical concept that an occlusal interference or discrepancy can produce abnormal masticatory function. Most subjects themselves were aware of bruxing on the splint, but the important factor of how often and with what force they bruxed was a significant, yet unknown, factor.
3. The primarily unilateral activity of the ipsilateral mandibular muscles produced an abnormal amount of ipsilateral activity of the sternocleidomastoid muscle.
4. The sternocleidomastoid muscle activity would generally have been of a low level, but it could have caused shortening of the muscle as it would have constituted contraction at a relatively fixed length. This is based on the reasonable assumption that the subjects would not generally move their heads during the clenching. This may have been particularly true when the subject was asleep. One could speculate that the subjects' muscles were most affected by the stiffening process at this time. The muscles of the neck may also be particlarly prone to tightness because in most people, they are rarely moved through a full range of motion during the course of a normal day. Evidence for this is afforded by the fact that three of the subjects showed a significant improvement in their range of motion after the stretching of the first experimental day. It was also observed in the pilot studies that if the range of motion in side flexion was checked daily, the subjects did not show

the same magnitude of range of motion changes due to this brief, yet full stretch of the muscles.

One problem with this step in the development of the dysfunction is that several subjects did not display measurable sternocleidomastoid activity during the partial clenching task with the splint. This could simply be due to the lack of high sensitivity of the EMG equipment used. It must also be remembered that the EMG surface electrodes do sample a wide area of the muscle, but there could be small, but significant areas of activity which are not detected due to electrode placement. The lack of measurable activity could also mean that while there was no muscle activity at that level of clench, the subject may have clenched during the experimental period with more force during the splint wear period, than was measured in the partial clenching task.

5. The increased tightness and resistance to stretch of the ipsilateral splint-side sternocleidomastoid could have produced the reduced range in side flexion to the non-splint side seen in 6 of the subjects. It could also have produced the sensation of tightness in side flexing to that side seen in seven of the subjects. This tightness could have led to the increased activity seen in the non-splint side sternocleidomastoid for the dominant group during the repetitive side flexion task.

The changes which occurred in the sternocleidomastoid activity levels during the clenching tasks without the splint, but after the splint wear period, are difficult to explain. At the present level of knowledge, one can only speculate that the integrated stimulating input from the oral region to the muscle or the muscle's response to that input were simply, or collectively, altered during the period of splint wear.

These changes, however, led to an additional finding of the present study which was the difference in response between the groups wearing the splint buildup on the dominant and non-dominant sides. Laterality, or motor dominance of one side, is a well established phenomenon based on asymmetrical function in the brain and central nervous system (Kinsbourne, 1978). The motor implications of this dominance have not been extensively studied except where they concern the performance of skilled tasks (which are more easily performed by the dominant side). Handedness and footedness are well established and they are generally the same side within an individual, although this is not always the case (Nachson et al, 1983). It has also been established that many individuals demonstrate facedness or dominance of one side of the face (Chaurasia and Goswami, 1975). Generally, the dominant side of the face is contralateral to the dominant side of the rest of the body which is not surprising as the facial nerve supplying the face consists of uncrossed fibres, whereas the cervical cord motor fibres supplying the rest of the body are generally crossed fibres (Barr, 1974).

No one has specifically examined the relative dominance of the two sides of the axial skeleton. If there is a dominant side, one would expect it to generally coincide with the dominance of the limbs, as the fibres to the neck muscles (including the sternocleidomastoid) are primarily crossed. One can therefore only speculate, that the muscles of the dominant side of the neck are more responsive to input from sources such as the masticatory system. This would correlate with the findings in the present study where, during the clenching tasks without the splint in the mouth, the splint-side sternocleidomastoid did not show the decline in activity levels due to the splint wear period in the dominant group as was found in the non-dominant group. Higher activity of the splint-side sternocleidomastoid in the dominant group, over the splint-wear period, would also explain the increased tension implied by the range of motion and side flexion EMG findings.

Unfortunately, the problem of dysfunction in the musculoskeletal system has not received the attention it deserves. The subject of dysfunction in the axial skeleton has received even less attention than other problems. There have been few epidemiological studies focusing on back and neck problems, and often the cause of dysfunction cannot be determined (Kelsey, 1982). In North America, the osteopathic profession has carried out research in the field and has specialized in its treatment, yet it is somewhat outside of traditional medicine and its literature is not readily accessible. Many physiotherapists assess and treat patients very successfully using osteopathic concepts, but in most cases the theory behind that treatment has not been validated.

In Europe, the situation is better as the field of physical medicine is larger and more active than it is in North America, although the complexity of dysfunction in the musculoskeletal system is still an impediment to research. In the field of physical medicine, Janda has done much to generate a theoretical framework for the understanding of musculoskeletal dysfunction supported by both clinical findings and treatment, as well as research. He has stressed that dysfunction always involves the entire motor system including the central nervous system, muscles, and joints (Janda, 1980, 1982a, 1982b, and 1985). He has further suggested that altered proprioception and afferent activity of joint receptors often produces the dysfunction seen in patients. According to Janda, this acute joint dysfunction often arises due to changes in muscle length and biomechaninics (both reflex and anatomical).

Janda (1986) believed that the results of the present study indicated the possible involvement of numerous structures in the neck. The initial changes were probably in the muscles of the neck (including, but not restricted to the sternocleidomastoid), but that the muscle changes may have produced changes in joint relationships, particularly around the C1 and C2 vertebra, where one would expect compensations to maintain a level head posture. Janda suggested that the complexity of the dysfunction is indicated by the different changes in activity levels of the sternocleidomastoid muscles in the head moving tasks which were similar, but slightly different. Janda (1986) suggested that this indicated the results of this study cannot be simply explained by the increased stiffness of the sternocleidomastoid muscle, but rather other muscles and joints were probably involved.

An attempt was made to correlate the different findings in the subjects with habits such as the tendency to chew on one side or to sleep on one side. No correlations could be found, but often a subject will not be aware of a habit so that this finding does not rule out the importance of such habits in the development of dysfunction.

The present study indicates, therefore, that asymmetric masticatory muscle activity can produce evidence of early neck dysfunction. It would seem then, that this does lend evidence to the suggestion that an occlusal discrepancy and the resulting masticatory hyperactivity often seen in TMD may be associated with the development of dysfunction in the cervical region (Trott and Goss, 1978; Danzig and Van Dyke, 1983, Friedman and Weisberg, 1985; and Passero et al, 1985). At the very least, it indicates the potential for involvement of the sternocleidomastoid muscles. Because these muscles alone cannot stabilize the head (Kapandji, 1974), one would hypothesize that other neck muscles may also be involved.

Dysfunction in the neck due to masticatory muscle hyperactivity may not always produce frank symptoms which prompt patients to seek treatment. It may, however, produce functional impairment of the neck which would set the patient up for an acute episode of pain and dysfunction when an additional stress is placed on the subject. Many of the conclusions and hypotheses in this study are highly speculative, reflecting the relative lack of knowledge in the field of clinical physical medicine. This study does suggest that the area of cervical involvement in temporomandibular disorders deserves further investigation.

# Chapter V

## SUMMARY AND CONCLUSIONS

The reaction of the neck and the sternocleidomastoid muscle in particular, to a seven to twelve day period of unilateral occlusal contact was studied in eight healthy subjects. The subjects wore a lower occlusal splint built-up on only one side to achieve unilateral posterior occlusal contact in centric occlusion and in lateral excursions. They wore the splint at all times, apart from meals. The subjects were divided into two groups: those wearing the splint buildup on their dominant side, and those wearing the splint buildup on their non-dominant side (referred to as dominant and non-dominant groups). The unilateral vertical buildup was designed to avoid lateral deflections of the mandible on closure. The subjects demonstrated no signs or symptoms of temporomandibular dysfunction, or neck pain or dysfunction prior to the study, and they served as their own controls. The subjects were tested on two days prior to the splint wear period, and on two days at the conclusion of the splint wear period. On each day of testing, the subjects were tested with respect to their natural resting head posture in the frontal plane, their range of motion in side flexion of the head and neck, and the peak EMG activity of the paired anterior temporalis and sternocleidomastoid muscles during several clenching and side flexing tasks. Data was analysed using a Mixed Analysis of Variance and a Student's t-test to determine if the changes due to the splint wear period were statistically significant.

Unilateral posterior ccclusal contact did not produce any significant signs or symptoms of TMD in the subjects over the period of splint wear. All subjects tolerated the splint with no significant problems apart from a single, mild period of head, or neck pain reported in several subjects lasting no more than eight hours.

The period of unilateral occlusal contact did not produce any significant changes in the head posture of the subjects in the frontal plane. All subjects were found to consistently hold their heads very close to the upright position with the interoccular line perpendicular to the gravitational vertical.

The unilateral occlusal contact did alter the head and neck range of motion in side flexion by increasing the asymmetry in the range of motion to the two sides. This effect was greater in the dominant group. In six of the eight subjects, the range of motion was relatively less in side flexion to the non-splint side which is consistent with increased resistance to stretch of the sternocleidomastoid on the splint side. Two subjects showed the opposite direction of change in their range of motion in side flexion so that the direction of change was not significant for the subjects as a whole. The reaction of the these two subjects with respect to range of motion illustrated that even in the early stages of dysfunction, some subjects will react differently. The alteration in symmetry of side flexion range of motion was significantly improved in some subjects by the stretching of the tightened muscles during the range of motion and EMG testing.

Unilateral occlusal contact reduced the EMG activity of the anterior temporalis on the non-splint side in both maximum clenching tasks and in partial clenching tasks (20 - 25% of maximum). The sternocleidomastoid muscles were active in maximum clenching tasks in all subjects and demonstrated activity in some subjects during partial clenching (20 - 25% of maximum). The period of unilateral occlusal contact reduced the activity of the non-contact side sternocleidomastoid muscle in all subjects. The activity of the contact side muscle was also reduced in the non-dominant group. In the dominant group however, the activity of the contact side sternocleidomastoid during maximum clenching tasks was not altered.

Repetitive side flexing from side to side for several cycles demonstrated higher peak activity levels of the non-splint side sternocleidomastoid (subjects wearing the splint buildup on their dominant side). This is consistent with an increased resistance to stretch in the splint-side sternocleidomastoid. Only one subject of the the non-dominant group demonstrated this increase in activity of the non-splint side sternocleidomastoid in the repetitive head moving task.

The period of unilateral occlusal contact, lasting approximately one week, did seem to produce changes in the neck which were consistent with increased tension in the sternocleidomastoid on the contact or splint side of the subjects. This was particularly true of subjects with the occlusal contact on the dominant side of their body. It was suggested that the unilateral occlusal contact may have produced largely unilateral parafunctional occlusal activity in the subjects which produced unilateral stimulation of the sternocleidomastoids on the side ipsilateral to the occlusal contact. It was further suggested that this stimulation of the muscle at a relatively fixed length produced the increase in stiffness and resistance to stretch seen in the subjects. The early development of these signs indicate the potential for involvement in TMD involving masticatory muscle hyperactivity, of the sternocleidomastoid and probably other neck neck muscles as well. It also demonstrates a functional relationship between the masticatory and neck muscles.

## FUTURE RESEARCH RECOMMENDATIONS

The results of the present study indicate that the sternocleidomastoid muscle can be adversely affected by unilateral occlusal contact leading to the early stages of dysfunction in the neck. There remain however, numerous questions which should be investigated by further research.

1. The period of unilateral occlusal contact was quite brief, yet significant changes were discovered in the sternocleidomastoid and the neck. The lack of significant TMD symptoms and the lack of complications in the subjects studied, suggest that a longer period of splint wear may be necessary. A splint with unilateral occlusal contact cannot be worn indefinitely because of the eruption of the unopposed teeth. Eating without the splint will slow the development of these changes. Repetition of this study over a one month period of splint wear would appear feasible with close monitoring. It would also be of value to examine a larger sample of subjects in a future study.

2. In addition to the sternocleidomastoid muscles, future studies should also examine changes in the scalene muscles which function similarly to the sternocleidomastoids, as well as the posterior cervical muscles such as the splenius capitis, semi-spinalis capitis, erector spinae, and the upper trapezius muscles. The present study attempted to measure the latter, but EKG artifact made this impossible. The use of a computer to eliminate this artifact should be considered. It would also be useful to use more complex transformations of the raw EMG data by the latest state of the art methods. For instance, power spectral analysis might reveal fatigue in the muscles due to the hyperactivity of unilateral contact. It would also be useful to examine the resting activity of these muscles using more sensitive equipment than that used in the present study. Head and neck movements such as rotation should also be tested. It would be valuable to design an electrogoniometer or use a film camera to record the head movements during the EMG recording and to correlate them with the muscle activity.

3. The amount of time and force with which the subject occludes on the unilateral splint must have a significant bearing on the development of dysfunction. It would be useful, therefore, to design a splint which could record these two factors for storage and later retrieval.

4. It was suggested in the study that the muscles of the neck including the sternocleidomastoid, may have excitatory input from a number of receptors in the masticatory system such as muscle spindles and tendon organs, as well as receptors in the joints, periodontal ligament, and periosteum. The relative importance

of these could be tested by using local anesthetics to block their input and observing the activity of the neck muscles. It would also be desirable to observe the activity of the neck muscles in different subjects at different levels of partial clench.

5. The present study examined the natural head posture of the subjects in the frontal plane during quiet standing. Under this situation, the subject might find it easy to correct for any tendency of postural deviation. One could examine the head posture of subjects performing a standardized task such as viewing a short film. In this way, a more realistic picture of how the subject holds his head might be developed and may show more changes.

6. Postural adaptations would probably involve the shoulders and possibly the entire skeleton, so that a photographic protocol should be developed to assess this as well.

7. If unilateral masticatory activity produces unilateral sternocleidomastoid activity, one would expect that subjects who brux might demonstrate bilateral sternocleidomastoid activity producing a forward head posture. This too could be measured by a photographic technique.

8. The hypothesis that neck dysfunction produced by occlusal dysfunction may lead to problems in balance and distorted neck reflexes could be tested in subjects by using tests of balance such as those used by Nashner et al (1983). Possibly related to this dysfunction of orientation, changes in athletic performance could be tested by assessing performance in an obsta-

cie course before and after the wearing of a unilateral occlusal splint. An obstacle course would also be a more comprehensive test of the effectiveness of treatment splints in athletes rather than tests of strength.

9. An epidemiological study should be used to determine the neck function of all types of TMD patients, but particularly in those with unilateral contact or prematurities.
10. In addition to the occlusal provocation of a unilateral splint, the effect of single tooth prematurities involving mandibular deviations should also be studied in relation to neck dysfunction.

In addition to these dentally related studies, the use of a unilateral occlusal splint would be a valuable method to study the early development of dysfunction in the neck. No other study has examined this problem and the development of such a model would allow the study of the early changes in muscle activity and joint function in the neck. One could also look for involvement of the thoracic and lower back.

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# Appendix A

## HISTORY

Patient Name

1. GENERAL HEALTH

   a. Have you ever had the following?

      sinus infection   ear infection   arthritis   loose joints

   b. Do you have frequent headaches?

      How often do they occur?

      What area of the head?

      How long do they last?

      Does anything seem to precipitate them?

      Do you suffer from migraines?

   c. Have you ever had a severe blow to the head or had a whiplash type neck injury?

   d. Have you ever experienced any tingling, numbness or weakness in the upper or lower extremities?

   e. Do you presently take any medication?

   f. Describe any other current non-dental physical problems or recent medical care you have received.

2. PAIN SYMPTOMS

   a. Do you have any pain or discomfort in the head, neck, shoulders or back? frequent stiff necks or shoulders? frequent headaches?

      Describe the character of the pain:

      sharp, dull, aching, deep, superficial, burning, spreading, pulsating?

Is the pain constant, intermittent?

For how long does the pain last?

Does the pain start gradually, suddenly?

Does the pain stop graduallly, suddenly?

What time of day is the pain most severe?

How often do you have the pain?

What is the longest period you have gone with out the pain?

What medication, if any, do you take for the pain?

Does any other factor appear to increase or decrease the pain?

Have you found any position of the jaw or head which relieves or eliminates the pain?

b. Do any of the following normal activities cause pain?

Yawning, chewing, swallowing, speaking, moving head, moving neck, moving shoulders, moving arms, moving trunk?

3. ORAL FUNCTION

a. Does your jaw opening seem normal?

b. Does your mandible ever lock open on wide opening?

c. Do you ever have any of these sounds in your TMJ's?

| | | | | | |
|---|---|---|---|---|---|
| Grating | R | L | Clicking | R | L |
| Snapping | R | L | Popping | R | L |

How often are these sounds present?

In what position is your mandible when they occur?

d. Do you ever have any pain around your TMJ's or ears?

e. Have you noticed any change in your occlusion?

f. Does your occlusion seem normal? Are you comfortable with it?

g. Do you have any problems swallowing.

4. RELATED FACTORS

a. Have you noticed any change in your hearing?

b. Do you ever have tinnitus (ringing) in your ears?
or a feeling of fullness pressure, or blockage?

c. Do you ever experience dizzyness? Fainting?
Nausea? Drop attacks?

d. Are you aware of grinding or clenching your teeth
during the night or day?

e. Do you frequently chew gum, fingernails, pencils or other objects?

f. Do your masticatory muscles ever become tired?

g. Do you play a musical instrument such as
a reed instrument, flute, or violin?

h. Do you frequently shoot guns?

i. Do you lie on your stomach when you sleep?

j. Have you recently had extensive dental work,
oral surgery, or a general anesthetic?

k. Have you ever been treated by an orthodontist?

l. Have you had your occlusion altered by a dentist?

m. Have you ever been treated for temporomandibular
dysfunction, or felt you might be suffering from it?

n. Do you believe you are under particular stress?

## Appendix B

## EXAMINATION

1. FACIAL SYMMETRY

   -appears grossly normal?

2. PALPATION OF THE TMJ's - Pain

| | | | | |
|---|---|---|---|---|
| a. TMJ palpation on lateral aspect (teeth in rest position) | | right | left | none |
| b. TMJ palpation on lateral aspect (teeth clenched) | | right | left | none |
| c. TMJ palpation of posterior apsect | | right | left | none |
| d. During right lateral movement | | right | left | none |
| e. During left lateral movement | | right | left | none |
| f. Right condyle translation | smooth | grating | asynchronous | |
| g. Left condyle translation | smooth | grating | asynchronous | |

3. PALPATION OF THE MUSCLES - Pain

| | | | |
|---|---|---|---|
| a. Masseter | right | left | none |
| b. Medial Pterygoid | right | left | none |
| c. Lateral Pterygoid (pain on forced) protrusion | right | left | none |
| d. Anterior Temporalis | right | left | none |
| e. Middle Temporalis | right | left | none |
| f. Posterior Temporalis | right | left | none |
| g. Digastric, anterior belly | right | left | none |
| h. Digastric, posterior belly | right | left | none |
| i. Sternocleidomastoid | right | left | none |

j. Trapezius, upper portion (neck) right left none

k. Trapezius, middle portion (shoulder) right left none

l. Splenius Capitis right left none

4. MANDIBULAR MOVEMENTS

a. Maximum opening without pain mm.

b. Maximum movement to right with teeth in contact mm.

c. Maximum movement to left with teeth in contact mm.

d. Maximum mandibular protrusion mm.

e. Relationship of dental midlines in rest position

f. Relationship of dental midlines inintercuspal position

g. Freeway space mm.

h. Mandibular opening path in both frontal and lateral view

i. Mandibular closing path in both frontal and lateral view

j. TM joint sounds:side, charachter and timing

k. Condylar movement pattern, left and right.

5. OCCLUSION

a. Tooth contact in centric relation

b. Slide from CR to CO, direction and distance

c. Pattern of contact in centric occlusion

d. Tooth contact in right lateral excursion:

working side:

balancing side

e. Tooth contact in left lateral excursion:

working side:

balancing side

f. Tooth contact in protrusion:

g. Presence of wear facets indicative of bruxism or clenching

h. Position of mandible accounting for wear facets

i. Fremitus of anterior teeth on closure

j. Angle occlusion class

k. Vertical overbite overjet

l. Preferred side of chewing

6. ORTHOPEDICS

a. General body posture

Axial Extension Kyphotic Lateral Deviation

b. Eyes grossly level?

c. Shoulders appear grossly level?

d. Hips appear grossly level?

e. Spine and back appear grossly straight?

f. Head posture from frontal view; equidistant from shoulders?

g. Head posture from lateral view

flexion extension forward

h. Range of motion of head in lateral side bending

Grossly normal range?

Pain present at end of range?

Grossly equal in both directions?

Does subject feel range is equal in both directions of lateral flexion?

Is the end-feel similar in both directions?

i. Range of motion of head from lateral view grossly normal in both extension and flexion?

j. Range of motion of head in rotation

Grossly normal range?

Pain present at end of range?

Grossly equal in both directions?

Does subject feel range is equal in

both directions of rotation?

Is the end-feel similar in both directions?

k. Is there any pain in resisted isometric neck motions from the neutral position?

Lateral flexion?

Flexion extension

Rotation

l. Freeway space mm

## Appendix C

## CONSENT FORM

It has been explained to me by Dr. J. W. Campbell that a study is being performed to assess changes due to an asymmetric alteration in vertical jaw relationships. I understand that some minor discomfort in the region of the temporomandibular joint and muscles of mastication may occur during the experimental period, but that any signs and symptoms should resolve promptly. I understand there is a remote possibility that some of the symptoms will persist beyond the experimental period. I further understand that in the unlikely event that I do experience such persistent symptoms, I will recieve treatment at the University of Manitoba Orthodontic Clinic until their resolution.

I consent to the taking of the following records from me with the understanding that this involves no risk.

1. Impressions for models

2. Bite registration records

3. Examination and history to be taken prior to, during, and subsequent to the experimental period.

4. Surface electromyography

5. Frontal facial photography

6. Goniometric measurement of head and neck mobility

It has been explained to me that I am free to withdraw from this study at any time with no penalty.

## Appendix D

### TABLE OF THE HEAD POSTURE OF INDIVIDUAL SUBJECTS

| Subject Numbers | Control Period Average | Experimental Day 1 | Experimental Day 2 |
|---|---|---|---|
| 1 | +0.9 | +0.3 | +0.3 |
| 2 | +1.8 | 0.0 | +0.5 |
| 3 | +0.4 | +1.5 | +0.8 |
| 4 | -0.4 | no data | -0.3 |
| 5 | -2.5 | -1.3 | -2.0 |
| 6 | -0.8 | -1.5 | 0.0 |
| 7 | +0.9 | +0.8 | +3.3 |
| 8 | -0.8 | -1.5 | -0.5 |

all measurements in degrees
positive number indicates head is tipped down toward the non-splint side
negative number indicates head is tipped down toward th splint side

## Appendix E

### TABLE OF THE DIFFERENCES IN HEAD AND NECK RANGE OF MOTION IN INDIVIDUAL SUBJECTS

| Subject Numbers | Control Days Average | Experimental Day 1 | Experimental Day 2 |
|---|---|---|---|
| 1 | +1.5 | -6.6 | -6.4 |
| 2 | +0.5 | +8.8 | +8.4 |
| 3 | -2.3 | -12.0 | -4.8 |
| 4 | -0.2 | -2.8 | -3.4 |
| 5 | +3.8 | +12.0 | -3.4 |
| 6 | +0.9 | -4.8 | -0.2 |
| 7 | -0.4 | -9.4 | -3.6 |
| 8 | +3.9 | -0.8 | -2.8 |

all measurements are in degrees
positive values indicate a range in side flexion greater to the non-splint side
negative values indicate a range in side flexion greater to the splint side

## Appendix F

### TABLE OF MUSCLE ACTIVITY FOR SUBJECT NUMBER 1

| Task | Day | Non-splint side Anterior Temporalis | Splint side Anterior Temporalis | Non-splint side Sternocleido-mastoid | Splint side Sternocleido-mastoid |
|---|---|---|---|---|---|
| 1 | con1 | 84.6 | 77.0 | 2.7 | 0.7 |
| | con2 | 74.1 | 79.1 | 2.6 | 3.8 |
| | exp1 | 90.0 | 93.2 | 1.3 | 1.4 |
| | exp2 | 78.1 | 73.4 | 0.3 | 0.2 |
| 1s | exp1 | 71.0 | 147.1 | 2.3 | 1.7 |
| | exp2 | 81.8 | 136.8 | 1.8 | 1.5 |
| 2 | con1 | 65.4 | 64.7 | 1.8 | 0.7 |
| | con2 | 78.0 | 68.4 | 3.2 | 3.0 |
| | exp1 | 92.0 | 122.1 | 2.5 | 2.1 |
| | exp2 | 100.3 | 117.0 | 1.8 | 1.0 |
| 2s | exp1 | 40.0 | 134.1 | 1.7 | 1.4 |
| | exp2 | 42.7 | 100.5 | 1.0 | 1.1 |
| 3 | con1 | 21.0 | 22.1 | nil | nil |
| | con2 | 18.7 | 19.9 | 0.2 | 0.5 |
| | exp1 | 54.8 | 29.7 | 0.2 | nil |
| | exp2 | 36.3 | 33.3 | nil | nil |
| 3s | exp1 | nil | 29.7 | 0.2 | nil |
| | exp2 | 0.6 | 36.5 | 0.2 | 0.2 |
| 4 | con1 | | | 3.1 | 82.9 |
| | con2 | | | 0.6 | 51.0 |
| | exp1 | | | 0.9 | 58.1 |
| | exp2 | | | 0.7 | 60.0 |
| 5 | con1 | | | 49.0 | 3.5 |
| | con2 | | | 51.6 | 2.5 |
| | exp1 | | | 65.0 | 0.9 |
| | exp2 | | | 53.4 | 1.3 |
| 6 | | Non-splint side SCM-Function1 | Splint side SCM-Function1 | Non-splint side SCM-Function2 | Splint side SCM-Function2 |
| | con1 | 47.4 | 47.2 | nil | 1.9 |
| | con2 | 47.7 | 54.3 | nil | 1.3 |
| | exp1 | 68.5 | 49.5 | 0.3 | 1.3 |
| | exp2 | 84.6 | 57.6 | 0.2 | 0.4 |

all measurements represent the % of the maximum activity on that day

con1=control day 1, con2=control day2, exp1=experimental day 1, and exp2=experimental day 2

## Appendix G

### TABLE OF MUSCLE ACTIVITY FOR SUBJECT NUMBER 2

| Task | Day | Non-splint side Anterior Temporalis | Splint side Anterior Temporalis | Non-splint side Sternocleido-mastoid | Splint side Sternocleido-mastoid |
|---|---|---|---|---|---|
| 1 | con1 | 75.5 | 73.7 | 4.9 | 1.1 |
| | con2 | 83.4 | 77.5 | 7.7 | 6.1 |
| | exp1 | 86.5 | 87.8 | 4.5 | 7.0 |
| | exp2 | 84.6 | 85.2 | 3.7 | 5.1 |
| 1s | exp1 | 4.1 | 51.1 | 1.5 | 1.5 |
| | exp2 | 17.9 | 78.0 | 4.1 | 2.8 |
| 2 | con1 | 80.0 | 92.8 | 7.3 | 2.2 |
| | con2 | 75.3 | 73.9 | 6.7 | 3.8 |
| | exp1 | 69.9 | 68.8 | 2.7 | 5.1 |
| | exp2 | 72.2 | 70.9 | 1.2 | 1.6 |
| 2s | exp1 | 1.4 | 35.7 | 1.2 | 1.2 |
| | exp2 | 7.2 | 58.6 | 2.2 | 1.6 |
| 3 | con1 | 19.1 | 28.5 | 0.3 | nil |
| | con2 | 19.8 | 19.5 | nil | nil |
| | exp1 | 26.3 | 22.7 | nil | nil |
| | exp2 | 25.7 | 17.2 | nil | nil |
| 3s | exp1 | 1.7 | 24.8 | nil | nil |
| | exp2 | 0.7 | 18.7 | nil | nil |
| 4 | con1 | | | 9.6 | 12.4 |
| | con2 | | | 6.1 | 6.3 |
| | exp1 | | | 5.5 | 7.1 |
| | exp2 | | | 7.3 | 3.9 |
| 5 | con1 | | | 13.8 | 4.1 |
| | con2 | | | 20.5 | 6.5 |
| | exp1 | | | 20.3 | 4.6 |
| | exp2 | | | 19.8 | 4.0 |
| 6 | | Non-splint side /SCM-Function1 | /Splint side /SCM-Function1 | /Non-splint side /SCM-Function2 | /Splint side /SCM-Function2 |
| | con1 | 8.8 | 5.1 | 9.8 | 2.8 |
| | con2 | 8.9 | 4.3 | 5.8 | 7.6 |
| | exp1 | 14.8 | 7.1 | 6.3 | 3.4 |
| | exp2 | 12.1 | 7.0 | 8.6 | 5.3 |

all measurements represent the % of the maximum activity on that day

con1=control day 1, con2=control day2, exp1=experimental day 1, and exp2=experimental day 2

## Appendix H

### TABLE OF MUSCLE ACTIVITY FOR SUBJECT NUMBER 3

| Task | Day | Non-splint side Anterior Temporalis | Splint side Anterior Temporalis | Non-splint side Sternocleido-mastoid | Splint side Sternocleido-mastoid |
|---|---|---|---|---|---|
| 1 | con1 | 88.9 | 84.2 | 0.5 | nil |
| | con2 | 88.0 | 90.1 | 0.4 | 1.3 |
| | exp1 | 86.8 | 87.4 | nil | 0.2 |
| | exp2 | 85.7 | 85.2 | 0.2 | 0.2 |
| 1s | exp1 | 57.5 | 75.1 | nil | nil |
| | exp2 | 32.5 | 47.3 | 0.2 | 0.5 |
| 2 | con1 | 86.2 | 75.3 | 1.0 | nil |
| | con2 | 76.4 | 76.5 | 0.2 | 2.2 |
| | exp1 | 104.3 | 100.7 | 0.6 | 0.2 |
| | exp2 | 93.2 | 89.7 | 0.5 | 1.1 |
| 2s | exp1 | 40.2 | 60.9 | 0.7 | 0.6 |
| | exp2 | 40.5 | 56.4 | 0.4 | 1.1 |
| 3 | con1 | 18.7 | 20.2 | nil | nil |
| | con2 | 14.4 | 17.3 | nil | nil |
| | exp1 | 11.7 | 25.6 | nil | nil |
| | exp2 | 24.8 | 27.2 | nil | nil |
| 3s | exp1 | 4.1 | 25.2 | nil | nil |
| | exp2 | 24.3 | 28.8 | nil | nil |
| 4 | con1 | | | 4.1 | 14.9 |
| | con2 | | | 3.3 | 19.1 |
| | exp1 | | | 9.0 | 29.8 |
| | exp2 | | | 2.8 | 16.8 |
| 5 | con1 | | | 18.2 | 2.1 |
| | con2 | | | 15.5 | 3.0 |
| | exp1 | | | 18.2 | 7.9 |
| | exp2 | | | 18.9 | 1.7 |
| 6 | | Non-splint side SCM-Function1 | /Splint side /SCM-Function1 | /Non-splint side /SCM-Function2 | /Splint side /SCM-Function2 |
| | con1 | 21.6 | 32.5 | 2.4 | 1.4 |
| | con2 | 11.0 | 25.5 | 1.3 | 2.1 |
| | exp1 | 24.2 | 30.0 | 7.8 | 5.3 |
| | exp2 | 24.3 | 32.6 | 4.1 | 4.4 |

all measurements represent the % of the maximum activity on that day

con1=control day 1, con2=control day2, exp1=experimental day 1, and exp2=experimental day 2

## Appendix I

### TABLE OF MUSCLE ACTIVITY FOR SUBJECT NUMBER 4

| Task | Day | Non-splint side Anterior Temporalis | Splint side Anterior Temporalis | Non-splint side Sternocleido-mastoid | Splint side Sternocleido-mastoid |
|---|---|---|---|---|---|
| 1 | con1 | 75.8 | 84.8 | 3.5 | 2.6 |
| | con2 | 84.6 | 79.1 | 7.2 | 4.6 |
| | exp1 | 75.4 | 86.9 | 4.3 | 7.0 |
| | exp2 | 73.9 | 77.7 | 3.7 | 5.2 |
| 1s | exp1 | 33.9 | 50.0 | 0.7 | 2.5 |
| | exp2 | 39.0 | 58.0 | 2.1 | 3.9 |
| 2 | con1 | 57.6 | 72.7 | 2.0 | 4.5 |
| | con2 | 85.6 | 58.9 | 5.9 | 2.9 |
| | exp1 | 65.2 | 74.9 | 7.5 | 8.7 |
| | exp2 | 71.5 | 57.3 | 5.8 | 5.3 |
| 2s | exp1 | 33.8 | 42.3 | 1.8 | 3.1 |
| | exp2 | 32.8 | 45.0 | 4.1 | 4.4 |
| 3 | con1 | 22.7 | 23.5 | 0.2 | nil |
| | con2 | 32.5 | 25.1 | 0.5 | nil |
| | exp1 | no readings taken | | | |
| | exp2 | no readings taken | | | |
| 3s | exp1 | 19.6 | 25.0 | nil | 1.3 |
| | exp2 | 9.7 | 20.2 | nil | 1.2 |
| 4 | con1 | | | 1.3 | 4.2 |
| | con2 | | | 19.1 | 4.1 |
| | exp1 | | | 11.0 | 1.1 |
| | exp2 | | | 8.0 | 5.3 |
| 5 | con1 | | | 4.6 | 0.6 |
| | con2 | | | 8.8 | 9.8 |
| | exp1 | | | 8.0 | 7.5 |
| | exp2 | | | 7.8 | 5.5 |
| 6 | | Non-splint side SCM-Function1 | /Splint side /SCM-Function1 | /Non-splint side /SCM-Function2 | /Splint side /SCM-Function2 |
| 6 | con1 | 4.7 | 2.6 | 6.4 | 2.8 |
| | con2 | 4.3 | 4.7 | 10.0 | 15.5 |
| | exp1 | 10.3 | 3.5 | 8.9 | 7.6 |
| | exp2 | 12.6 | 4.6 | 8.4 | 3.3 |

all measurements represent the % of the maximum activity on that day

con1=control day 1, con2=control day2, exp1=experimental day 1, and exp2=experimental day 2

## Appendix J

### TABLE OF MUSCLE ACTIVITY FOR SUBJECT NUMBER 5

af 7970

| Task | Day | Non-splint side Anterior Temporalis | Splint side Anterior Temporalis | Non-splint side Sternocleido-mastoid | Splint side Sternocleido-mastoid |
|---|---|---|---|---|---|
| 1 | con1 | 75.8 | 73.4 | 7.5 | 11.0 |
| | con2 | 81.4 | 69.1 | 1.5 | 3.8 |
| | exp1 | 83.7 | 71.2 | nil | 5.1 |
| | exp2 | 73.6 | 65.9 | 0.2 | 0.6 |
| 1s | exp1 | 43.9 | 100.5 | 0.2 | 4.3 |
| | exp2 | 40.8 | 105.4 | nil | 3.4 |
| 2 | con1 | 102.4 | 100.6 | 12.2 | 14.6 |
| | con2 | 91.9 | 93.8 | 5.5 | 8.6 |
| | exp1 | 115.8 | 49.6 | 2.7 | 9.5 |
| | exp2 | 84.7 | 30.4 | 2.7 | 6.8 |
| 2s | exp1 | 22.1 | 87.1 | 2.5 | 9.8 |
| | exp2 | 26.0 | 102.2 | 7.3 | 13.0 |
| 3 | con1 | 25.5 | 19.1 | 1.4 | 1.5 |
| | con2 | 17.7 | 15.5 | nil | nil |
| | exp1 | 33.4 | 21.5 | nil | 1.5 |
| | exp2 | 66.8 | 20.0 | nil | 0.2 |
| 3s | exp1 | nil | 23.7 | nil | 2.5 |
| | exp2 | nil | 18.9 | nil | 1.7 |
| 4 | con1 | | | 9.3 | 30.4 |
| | con2 | | | 3.1 | 20.7 |
| | exp1 | | | 1.6 | 18.9 |
| | exp2 | | | 18.2 | 55.9 |
| 5 | con1 | | | 31.4 | 19.3 |
| | con2 | | | 24.1 | 26.2 |
| | exp1 | | | 6.3 | 18.9 |
| | exp2 | | | 31.4 | 28.8 |

| Task | Day | Non-splint side SCM-Function1 | Splint side SCM-Function1 | Non-splint side SCM-Function2 | Splint side SCM-Function2 |
|---|---|---|---|---|---|
| 6 | con1 | 26.0 | 37.0 | 11.6 | 12.4 |
| | con2 | 41.5 | 25.7 | 10.6 | 17.6 |
| | exp1 | 6.8 | 21.1 | 3.0 | 21.8 |
| | exp2 | 36.4 | 57.1 | 20.0 | 32.2 |

all measurements represent the % of the maximum activity on that day

con1=control day 1, con2=control day2, exp1=experimental day 1, and exp2=experimental day 2

## Appendix K

### TABLE OF MUSCLE ACTIVITY FOR SUBJECT NUMBER 6

| Task | Day | Non-splint side Anterior Temporalis | Splint side Anterior Temporalis | Non-splint side Sternocleido-mastoid | Splint side Sternocleido-mastoid |
|---|---|---|---|---|---|
| 1 | con1 | 83.8 | 82.0 | 2.7 | 2.4 |
| | con2 | 84.4 | 87.3 | 3.9 | 4.0 |
| | exp1 | 70.7 | 71.7 | 1.7 | 2.3 |
| | exp2 | 79.5 | 76.5 | 3.0 | 3.6 |
| 1s | exp1 | 33.8 | 45.4 | 1.3 | 2.2 |
| | exp2 | 38.9 | 56.0 | 2.0 | 3.3 |
| 2 | con1 | 87.2 | 95.1 | 5.1 | 5.4 |
| | con2 | 87.9 | 73.0 | 15.8 | 16.9 |
| | exp1 | 108.0 | 94.5 | 5.8 | 7.1 |
| | exp2 | 115.0 | 108.0 | 5.0 | 5.3 |
| 2s | exp1 | 39.5 | 56.8 | 2.9 | 5.0 |
| | exp2 | 46.8 | 54.4 | 2.4 | 6.1 |
| 3 | con1 | 22.8 | 19.5 | nil | nil |
| | con2 | 20.4 | 10.9 | nil | nil |
| | exp1 | 8.4 | 16.4 | nil | nil |
| | exp2 | 22.5 | 19.5 | nil | nil |
| 3s | exp1 | 4.7 | 20.0 | nil | nil |
| | exp2 | 1.7 | 19.7 | nil | 0.8 |
| 4 | con1 | | | 13.7 | 28.4 |
| | con2 | | | 13.6 | 66.7 |
| | exp1 | | | 6.4 | 16.5 |
| | exp2 | | | 4.9 | 18.7 |
| 5 | con1 | | | 44.9 | 7.9 |
| | con2 | | | 30.7 | 15.0 |
| | exp1 | | | 39.8 | 8.1 |
| | exp2 | | | 25.6 | 8.9 |
| 6 | | Non-splint side SCM-Function1 | /Splint side SCM-Function1 | /Non-splint side SCM-Function2 | /Splint side SCM-Function2 |
| 6 | con1 | 45.6 | 31.4 | 7.8 | 7.9 |
| | con2 | 73.1 | 80.3 | 6.1 | 9.1 |
| | exp1 | 22.1 | 25.0 | 3.5 | 7.9 |
| | exp2 | 26.7 | 21.6 | 4.7 | 10.6 |

all measurements represent the % of the maximum activity on that day

con1=control day 1, con2=control day2, exp1=experimental day 1, and exp2=experimental day 2

## Appendix L

### TABLE OF MUSCLE ACTIVITY FOR SUBJECT NUMBER 7

| Task | Day | Non-splint side Anterior Temporalis | Splint side Anterior Temporalis | Non-splint side Sternocleido-mastoid | Splint side Sternocleido-mastoid |
|---|---|---|---|---|---|
| 1 | con1 | 80.3 | 74.6 | 1.2 | 1.5 |
| | con2 | 85.1 | 85.4 | 1.2 | 1.6 |
| | exp1 | 84.7 | 86.0 | 0.4 | 0.9 |
| | exp2 | 74.7 | 81.2 | 0.2 | 0.7 |
| 1s | exp1 | 52.3 | 93.0 | 0.2 | 0.9 |
| | exp2 | 40.3 | 78.6 | 0.2 | 0.9 |
| 2 | con1 | 109.1 | 117.0 | 2.5 | 2.5 |
| | con2 | 104.4 | 106.0 | 1.7 | 2.0 |
| | exp1 | 108.0 | 105.3 | 0.8 | 1.0 |
| | exp2 | 96.0 | 90.7 | 0.3 | 0.8 |
| 2s | exp1 | 42.0 | 101.3 | 0.4 | 0.9 |
| | exp2 | 39.3 | 99.7 | nil | 0.6 |
| 3 | con1 | 11.5 | 20.1 | nil | nil |
| | con2 | 10.0 | 19.6 | nil | nil |
| | exp1 | 41.3 | 27.7 | nil | 0.2 |
| | exp2 | 35.7 | 24.9 | nil | 0.3 |
| 3s | exp1 | 1.0 | 32.0 | nil | nil |
| | exp2 | nil | 22.7 | nil | nil |
| 4 | con1 | | | 2.3 | 11.3 |
| | con2 | | | 0.5 | 12.7 |
| | exp1 | | | 2.3 | 12.7 |
| | exp2 | | | 2.3 | 6.3 |
| 5 | con1 | | | 5.7 | 0.2 |
| | con2 | | | 2.9 | 0.9 |
| | exp1 | | | 2.5 | 0.8 |
| | exp2 | | | 2.0 | 0.3 |
| 6 | | Non-splint side SCM-Function1 | /Splint side /SCM-Function1 | /Non-splint side /SCM-Function2 | /Splint side /SCM-Function2 |
| 6 | con1 | 11.1 | 15.3 | 2.0 | 1.2 |
| | con2 | 2.7 | 16.3 | 0.5 | 2.4 |
| | exp1 | 2.6 | 15.7 | 1.3 | 1.3 |
| | exp2 | 7.3 | 11.3 | 2.5 | 1.5 |

all measurements represent the % of the maximum activity on that day

con1=control day 1, con2=control day2, exp1=experimental day 1, and exp2=experimental day 2

## Appendix M

### TABLE OF MUSCLE ACTIVITY FOR SUBJECT NUMBER 8

| Task | Day | Non-splint side Anterior Temporalis | Splint side Anterior Temporalis | Non-splint side Sternocleido-mastoid | Splint side Sternocleido-mastoid |
|---|---|---|---|---|---|
| 1 | con1 | 82.4 | 85.4 | 1.7 | 3.9 |
| | con2 | 82.4 | 91.0 | 3.8 | 5.4 |
| | exp1 | 64.7 | 77.2 | 1.1 | 1.8 |
| | exp2 | 61.2 | 78.9 | 1.2 | 2.4 |
| 1s | exp1 | 36.3 | 50.6 | 1.7 | 2.0 |
| | exp2 | 69.9 | 61.2 | 2.8 | 2.4 |
| 2 | con1 | 105.5 | 96.9 | 2.9 | 7.5 |
| | con2 | 100.2 | 115.7 | 5.5 | 7.4 |
| | exp1 | 120.0 | 110.8 | 1.3 | 4.0 |
| | exp2 | 201.4 | 110.1 | 4.0 | 4.2 |
| 2s | exp1 | 46.9 | 65.3 | 1.6 | 2.6 |
| | exp2 | 68.8 | 48.0 | 2.1 | 1.8 |
| 3 | con1 | 10.8 | 18.9 | 0.2 | nil |
| | con2 | 36.2 | 34.0 | 0.2 | 1.6 |
| | exp1 | 15.3 | 29.8 | 0.2 | 0.2 |
| | exp2 | 22.5 | 26.7 | 0.6 | 0.2 |
| 3s | exp1 | 13.8 | 31.7 | 0.8 | 0.9 |
| | exp2 | 0.6 | 21.6 | nil | 0.5 |
| 4 | con1 | | | 1.3 | 18.0 |
| | con2 | | | 0.2 | 5.9 |
| | exp1 | | | 1.7 | 20.4 |
| | exp2 | | | 4.1 | 29.2 |
| 5 | con1 | | | 25.2 | 1.4 |
| | con2 | | | 28.8 | 3.2 |
| | exp1 | | | 34.8 | 1.6 |
| | exp2 | | | 31.5 | 1.2 |

| Task | Day | Non-splint side SCM-Function1 | Splint side SCM-Function1 | Non-splint side SCM-Function2 | Splint side SCM-Function2 |
|---|---|---|---|---|---|
| 6 | con1 | 26.0 | 11.8 | 2.5 | 0.9 |
| | con2 | 31.9 | 12.3 | 2.0 | 2.2 |
| | exp1 | 38.1 | 28.9 | 2.2 | 0.7 |
| | exp2 | 48.3 | 24.1 | 1.3 | 1.4 |

all measurements represent the % of the maximum activity on that day

con1=control day 1, con2=control day2, exp1=experimental day 1, and exp2=experimental day 2