JAW REFLEXES IN SUBJECTS WITH TEMPOROMANDIBULAR DISORDERS AND BRUXISM

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DECLARATION

This thesis is the original work of the author.

ATEF M. OKDEH

To my dearest and most precious wife,

I gratefully dedicate this work.

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LIST OF ABBREVIATIONS

μm	micrometer
μs	microsecond
cm	centimetre
EMG	Electromyography
min	minute
mm	millimetre
ms	millisecond
MVC	Maximum voluntary contraction
sec	second
SEM	Standard error of the mean
SP	Silent period
T	Threshold
TMD	Temporomandibular disorders
TMJ	Temporomandibular joint
VAS	Visual Analogue Scale

APPENDIX

PUBLICATIONS AND PRESENTATIONS TO SCIENTIFIC MEETINGS

Parts of the work presented in this thesis have been presented at scientific meetings and published or submitted for publication in scientific journals as follows:

The effect of stimulus polarity on jaw reflexes evoked by electrical stimulation across the lip. Okdeh A.M., Lyons M.F. and Cadden S.W.
Presented to the Annual Conference of the British Society for Dental Research, April, 1996, Bristol, UK
Published in *Journal of Dental Research*, 1996, **75**, No. 5, p. 1140.

Jaw reflexes evoked by stimulation across the lip.
Okdeh A.M., Lyons M.F. and Cadden S.W.
Presented to the 2nd International Congress on Orofacial Pain and Temporomandibular Disorders, May, 1996, Paris, France.
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The effect of occlusal splints on jaw reflexes evoked by electrical stimulation of the lower lip. Okdeh A.M., Lyons M.F. and Cadden S.W.
Presented to the 75th International Association for Dental Research, March, 1997, Orlando, USA
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Jaw reflexes evoked by stimulation across the lip in bruxists.
Okdeh A.M., Lyons M.F. and Cadden S.W.
Presented to the 20th Meeting of The Society of Oral Physiology, May, 1997, Lattrop, The Netherlands.

The effect of stimulus intensity and polarity on masseter muscle reflexes evoked by electrical stimulation of the lip in human subjects.
Okdeh A.M., Lyons M.F. and Cadden S.W.
Submitted for publication in the *Journal of Oral Rehabilitation.*

The effect of occlusal splints on jaw reflexes evoked by electrical stimulation of the lower lip. Okdeh A.M., Lyons M.F. and Cadden S.W.
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Masseter muscle reflexes evoked by stimulation across the lip in bruxist subjects.
Okdeh A.M., Lyons M.F. and Cadden S.W.
In preparation for publication.

SUMMARY

Jaw reflexes, particularly the inhibitory reflexes of the masseter muscles (silent periods), have attracted considerable interest from clinicians since it was claimed that this reflex is modified in patients suffering from temporomandibular dysfunction, and that this may be used for the diagnosis and monitoring of the treatment of dysfunction. It is thought that, a major function of these jaw reflexes is to reduce damage to the soft tissues in and around the mouth when a hard object is inadvertently bitten upon.

There is general agreement on the bi-phasic nature of the inhibitory responses to intra-oral stimuli in healthy subjects, but there is dispute as to the nature of responses evoked by stimulation of peri-oral structures. A different pattern of jaw reflexes may also be seen in bruxists. Furthermore, disagreement exists regarding the inhibitory reflexes in the active masseter muscle in patients with temporomandibular disorders (TMD). Thus, the pattern of jaw reflexes in dysfunction and parafunction is unclear. The overall aim of this study, therefore, was to investigate the jaw-opening reflexes in TMD patients and bruxists in order to get a better understanding of these disorders.

At the beginning, it was decided to study the reflexes evoked in the masseter muscle by electrical stimulation across the lip in healthy subjects. The aim was to find the threshold at which each of the significant responses occurred and to investigate the effect of reversing the polarity of the stimulus electrode on the pattern of reflexes. A sequence of inhibitory, excitatory, inhibitory and excitatory responses could be produced in the muscle by both polarities of stimuli. It was found that stimulation of

nerves supplying the skin outside the mouth evokes predominately long-latency jaw reflexes whereas short-latency responses can be evoked by stimulating nerves supplying oral mucosa. Furthermore, long-latency excitatory reflexes seem to be the most easily evoked by stimulation of the lip.

Another aim of the study was to investigate whether difference could be observed between bruxists and non-bruxists in response to electrical stimulation of the lip. In both groups, there were significant differences in the thresholds of the different responses. Also, there were differences between the two groups in the presence of the short-latency excitation and the long-latency inhibitory responses. These findings suggest that long-latency inhibitory responses evoked by electrical stimulation of the lip are weaker in bruxists than in non-bruxists.

Finally, jaw reflexes in TMD patients were investigated to determine whether differences could be detected in electrically-evoked inhibitory and excitatory responses. Moreover, the occlusal splint is one of the most universally accepted forms of therapy in TMD patients and it was possible that these splints might have an effect on the pattern of jaw reflexes in view of their therapeutic effect. Therefore, it was considered to investigate the effect of occlusal splint therapy on masseter muscle reflexes in these patients. It has been found that one week of occlusal splint therapy results in changes in the pattern of reflexes as well as symptomatic relief. Since the changes of the reflexes involved a relative increase in inhibitory responses, it is possible that this would reduce the use of the muscles and be, at least in part, responsible for the accompanying symptomatic relief.

CHAPTER ONE

GENERAL INTRODUCTION AND AIMS OF THE STUDY

General introduction

The reflexes of the muscles of mastication, particularly the inhibitory response (silent period) of the masseter muscle, have been the subject of considerable clinical research since the suggestion by Bessette et al (1971, 1974) that its duration may have diagnostic value. These authors showed that the silent period (SP) determined from the surface electromyogram of patients with temporomandibular disorders (TMD) was found to be significantly longer than in normal subjects. These results were later confirmed by other workers (Widmalm, 1976; McCall et al, 1978; Hussein & McCall, 1983). The results of these studies have been questioned, however, because of the large intraindividual variations and methodological problems (Hellsing & Klineberg, 1983; Lavinge et al, 1983).

The masseteric inhibitory reflex, or silent periods can be defined as a transient absolute or relative decrease of the EMG activity level following some stimulus during a sustained contraction (Steenberghe et al, 1989). The function of this reflex is thought to reduce damage to the soft tissues in and around the mouth when a hard or sharp object is inadvertently bitten upon (Lund, 1991). There are different types of stimuli that have been used to study the reflexes of the jaw muscles in human subjects, including mechanical, electrical and acoustic stimulation.

Stimulation within the mouth can produce a sequence of inhibitory, excitatory, inhibitory and excitatory responses in the masseter muscle. These reflex responses appear as a series of downward and upward-going waves in full-wave rectified and

averaged EMGs. While there is general agreement on the multiphasic nature of these responses to intra-oral stimuli (e.g. Bratzlavsky, 1972; Yemm, 1972a; Yu et al, 1973; Godaux and Desmedt, 1975; Glas et al, 1985; Carels and Steenberghe, 1986; Widmer, 1987; Cadden and Newton, 1988), there is some dispute as to the nature of responses to stimulation of peri-oral structures. Some studies have reported that a multiphasic response similar to that produced by intra-oral stimuli can be evoked by stimulation of the lips (e.g. Bratzlavsky, 1972; Godaux and Desmedt, 1975; Desmedt and Godaux, 1976; Di Francesco et al, 1986), while others have reported a simpler response consisting of a single inhibitory period and a single excitatory period which seem to be equivalent to the last two elements of the responses evoked by intra-oral stimuli (Yu et al., 1973; Cadden and Newton, 1988, 1994). Thus, these discrepancies require investigation.

A number of terms have been used to describe dysfunction of the TMJ and associated muscles. In their review of the literature, Rugh and Solberg (1976) found some consensus on three symptoms which comprise TMJ dysfunction; pain, tenderness of the muscles of mastication and TMJ and limitation of the mandibular movements. Currently, the term temporomandibular disorders (TMD), which is synonymous with the term craniomandibular disorders, is considered the preferred term (McNeill, 1993).

Differences have been found in the inhibitory reflexes of TMD patients and symptom-free subjects. Griffin and Munro (1971) reported a shorter latency and more frequent absence of the silent period (SP) in patients during the open-close-clench cycle. Other studies, on the other hand, found significantly longer durations of the inhibitory period

after chin tap, (Bessette et al, 1971; Widmalm, 1976; McCall and Hoffer, 1981), which shorten again to normal ranges after occlusal splint therapy (Bessette et al, 1971; Bailey et al, 1977; Skiba & Laskin, 1981). The SP duration increased when the condition was acute, and returned to normal when the condition was successfully treated. In the contrary, it has been found that there were no significant differences in SP duration between the patients that achieved complete, partial or no relief of the symptoms (Strychalski et al, 1984).

Furthermore, the incidence of a second silent period after mechanical or electrical tooth pulp stimulation was lower in TMD patients (De Laat et al, 1985; Sharav et al, 1982). By contrast, Hussein & McCall (1983), using electrical stimulation of the mental nerve area, consistently found two inhibitory periods in both patients and symptoms-free subjects.

The occlusal splint is one of the most universally accepted forms of therapy in patients with signs and symptoms of functional disorders of the masticatory system and nocturnal bruxism (Faulkner, 1990). Many investigations have been carried out to evaluate the treatment outcome of the occlusal appliances. Success with occlusal splint therapy has been reported in 70-90% of TMD patients (Franks, 1965; Greene & Laskin, 1972; Carraro & Caffesso, 1978; Okeson & Kemper, 1982; Okeson et al, 1983; Suvinen & Reade, 1989). Thus, it is possible that occlusal splints might have an effect on the pattern of jaw reflexes in view of their therapeutic effect.

It is generally accepted that the aetiology of temporomandibular disorders is multifactorial (Moss & Garrett, 1984; Yemm, 1985), and many authors implicate bruxism in the aetiology of TMD (Franks, 1965,b; Laskin, 1969; Christensen, 1971; Trenouth, 1979). De Laat et al (1985) investigated the reflex pattern in bruxist subjects and found that there was a strong correlation between bruxism and the occurrence of single inhibitory period in response to mechanical stimulation of the tooth. Therefore, it was of interest to investigate the masseter muscle reflexes in bruxist subjects in response to electrical stimulation.

Some difficulty arises in comparing the results from different papers not only because of the variety of stimulation methods, but also because a uniform evaluation of the different inhibitory period parameters was lacking. In some studies, the latency and duration of the inhibitory periods were determined by visual inspection of individual or a number of superimposed sweeps. In some other studies, all sweeps were full wave rectified, superimposed and averaged.

In response to mechanical stimulation of a tooth, double inhibitory periods occur less frequently than after electrical stimulation of the gingiva above the same tooth (Carels & Steenberghe, 1986). Further, there are problems with the use of mechanical stimuli in the study of inhibitory jaw reflexes; it is difficult to deliver a mechanical stimulus in a reproducible manner (Bishop et al, 1984). Moreover, a mechanical stimulus which is large enough to cause inhibition will also stimulate several receptor systems which have different reflex connections with the motor neurone under study. However, according to Turker and Miles (1989), electrical stimulation offers advantages over mechanical

stimulation in the study of inhibitory jaw reflexes; the stimulus parameters (intensity, duration, and frequency) can be readily set and kept constant; electrical stimulation does not cause vibration and hence does not stimulate the vibration sensitive receptors in muscles and in the inner ear; electrical stimulation does not change the jaw position and hence does not activate the position sensitive receptors which are situated in the jaw muscles and in the temporomandibular joint. Therefore, it was decided to use electrical stimulation to investigate jaw reflexes.

Aims of the study

The overall aim of this study was to investigate the jaw-opening reflexes in TMD patients and bruxists in order to get a better understanding of these disorders. The specific aims were:

1. To study the reflexes evoked in the masseter muscle by electrical stimulation across the lip using a range of stimulus intensities, and to find the threshold at which each of the significant responses occurred in healthy subjects.

2. To investigate the effect of reversing the polarity of the stimulus electrode on the pattern of reflexes.

3. To investigate whether difference can be observed between bruxists and non-bruxists in response to electrical stimulation of the lip.

4. To determine whether differences can be detected in the responses evoked in TMD patients and what effect, if any, an occlusal splint had on these responses.

CHAPTER TWO

REVIEW OF THE LITERATURE

2.1 Jaw reflexes

The muscles of mastication, like those elsewhere on the body, are subject to reflex controls. There are three principal reflexes which control the vertical relationship between the mandible and the maxilla and hence TMJ movements: the jaw jerk, jaw opening and jaw unloading reflexes (McKay et al, 1992).

These reflexes, particularly the inhibitory reflexes of the masseter muscles (silent periods), have attracted considerable interest from clinicians since it was claimed that this reflex is modified in patients suffering from temporomandibular dysfunction, and that this may be used for the diagnosis and monitoring of the treatment of dysfunction.

There are different types of stimuli that have been used to study the reflexes of the jaw muscles in human subjects. In this section, the reflexes in healthy subjects will be reviewed, while the reflexes in TMD patients will be reviewed later in this Chapter.

2.1.1 Jaw-jerk reflex

The principal response to a sudden downward movement of the mandible while the muscles are at rest is a stretch reflex. The jaw closing muscles (i.e. masseter and

temporalis muscles), are stretched and this evokes a short latency reflex excitation. This reflex is also referred to as a myotatic and is not seen in the jaw opening muscles (Hufschmidt & Spuler, 1962; Munro & Griffin, 1971; Yemm, 1972,b; Goldberg, 1972; Yamada & Ash, 1982; Murray & Klineberg, 1984; Hellsing & Klineberg, 1983; Kossioni & Karkazis, 1995).

The mean latency of the excitatory response ranges from 5.7 ms (Kossioni & Karkazis, 1995) to 9.9 ms (Goldberg, 1972). The mean duration ranges from 6.1 ms (Widmalm & Hedegard , 1976) to 9.4 ms (Yaeger et al, 1978). A great variability in the amplitude of the reflex has been reported within and between subjects (Munro & Griffin, 1971; Bishop et al, 1984; Kossioni & Karkazis, 1995). Most of the discrepancy can be attributed to the differences in sample selection, the various recording and analysis systems to determine the jaw jerk.

The stretch reflex helps to maintain the postural stability of the mandible (Cooker et al, 1980). This reflex also helps with the fine control of jaw/TMJ movement throughout normal function, for example, to take account of different consistencies of food (McKay et al, 1992).

The Silent Period Following Chin Tapping

If a tap on the chin is applied while subjects maintain a voluntary activity of the jaw closing muscles, the jaw jerk in the masseter muscle is followed by a period of

depressed activity, the so called silent period (SP) (e.g. Hufschmidt & Spuler, 1962; Godaux & Desmedt, 1975; Widmalm, 1976; Bailey et al, 1977; McNamara et al, 1977).

Many investigators have used mechanical chin tapping to evoke the silent period reflex. The effect of various mechanical input parameters on the silent period duration has been studied, and the results are controversial. It has been found that the duration of the masseteric EMG silent period varies inversely with the magnitude of bite force (Lavinge et al, 1983). The results confirmed previous findings in which it had been found that decreased isometric muscle force resulted in statistically significant increases in silent period duration (McNamara et al, 1977; Gale & McCall, 1980; Bernstein et al, 1981; Fung et al, 1982; However, other studies found that increasing the voluntary clenching force did not alter the duration of the SP (Bessette et al, 1973; Bailey et al, 1977; Palla et al, 1981). The methods, however, were not uniform, e.g. Bessette et al (1973) used a force transducer between the incisors while McNamara et al (1977) used a force transducer between the premolars unilaterally.

It has been found that varying the type of mechanical stimulation and altering the occlusal vertical dimension did not significantly alter the resultant silent period duration (McNamara et al, 1977). Further, Gillings (1974) found that numerous directions and differing natures of mandibular taps had no effect on the latency-inhibition behaviour of the contracting masticatory muscles.

Human variability in the measurement of masseteric silent period was quantified by Lavigne et al (1983). They investigated four sources of variability in the estimation of

the SP: variability between different observers rating the same EMG traces; variability within a single observer rating the same traces twice; bias in observers expecting a difference between traces; and variability across trials in the same normal subject. There were significant differences among observer groups, individual observers and repeated measurement of the same silent period. Further, the distance between the incisor teeth (vertical dimension) had a highly significant influence on SP duration. They concluded that the investigation of the silent period duration requires a standardisation of all the variables involved.

The influence of stimulus type and stimulus strength on the silent period in human masticatory muscles was studied by Kroon and Naeije (1984). The stimulus types used were the open-close-clench movement, the mechanical chin tap and electrical skin stimulation. They showed that the forceful open-close-clench movements produced shorter silent period durations, as others have also found (Widmalm, 1976; Furuya and Hedegard, 1981). Increasing the chin tap strength resulted in longer silent periods, and produced a clear shift of the silent period pattern from the single pattern at low chin taps to the depressed, merged and double type at higher chin taps. Moreover, one subject produced a very long silent period which confirms an observation of Hellsing and Klineberg (1983) that a long silent period can be evoked in subjects without TMJ dysfunction.

Recently, Tamura et al (1995) investigated the effect of muscle activity and the magnitude of biting force on silent period duration of the masseter muscle. The jaw jerk was evoked by tapping the chin of a subject with a reflex hammer during both clenching

teeth (10%, 50%, and maximum) and biting on a transducer (50N, 100N, and maximum). The results suggested that SP duration was positively influenced by the strength of the background activities of the muscles. With an increase of muscle activity, SP duration was significantly decreased in the normal group. This is inconsistent with the earlier results of Bessette et al (1973) and Bailey et al (1977) who reported that muscle tension does not influence the silent period. However, the results are in agreement with those of Bernstein et al (1981), McNamara et al (1977), and Fung et al (1982) who showed that the silent period decreased in healthy subjects, both while biting on the transducer and while clenching with teeth together.

Overall, one can say that wide inter- and intra-individual variability of the silent period response is due to the varying methodology, the lack of control of experimenter bias and the variability of the SP within a single subject.

2.1.2 Jaw unloading reflex

A sudden closing movement during the contraction of the elevator muscles produces an unloading reflex (Hannam et al, 1968; Lamarre & Lund, 1975). This reflex involves a cessation of activity in jaw closing muscles, together with an activation of jaw opening muscles. The reflex is evoked when a hard object which is being bitten breaks suddenly thus 'unloading' the jaw closing muscles of the resistance against which they were

working to prevent any damage to the teeth (McKay et al, 1992).

The unloading reflex is probably important in both chewing and biting. When one is biting on brittle objects such as nut shells, activity in the jaw closing muscle builds up until the object fractures. When the object between the teeth breaks suddenly, the lower jaw first closes very rapidly, but a combination of the unloading reflex and digastric stiffness end the movement (Lund et al, 1983).

2.1.3 Jaw opening reflex

The term jaw opening reflex results in either jaw opening or a cessation of jaw closing (McKay et al, 1992). This reflex results in an inhibition of the jaw closing muscles with no activation of the digastric muscles (Yemm, 1972,b). In the light of this observation, Turker and Miles (1985) suggested the term 'elevator-inhibition reflex' to describe this reflex in humans.

The jaw opening reflex can be evoked experimentally by mechanical stimulation of the teeth or the lips (Glas & Steenberghe, 1988), and it can be evoked by electrical stimulation of intra- or peri-oral sites (e.g. Yemm, 1972,a; Cadden & Newton, 1988).

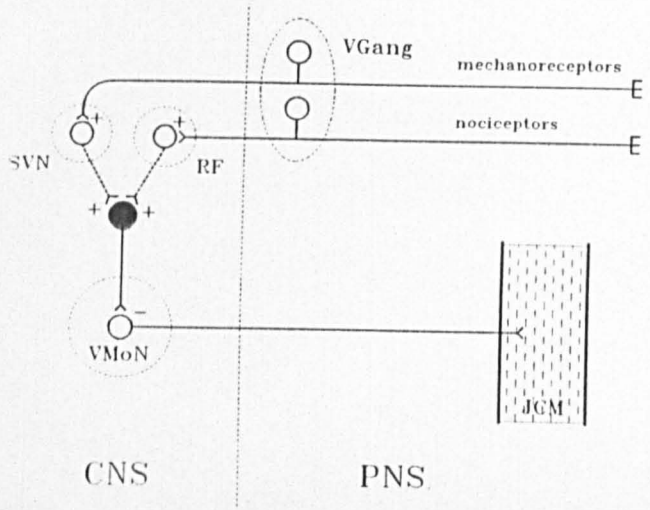
The jaw opening reflex is thought to protect the soft tissues and lips against being bitten during jaw closure as well as against being damaged due to excessive occlusal forces if the teeth encounter a hard object (Hannam & Matthews, 1968; Lund, 1991; McKay, et al, 1992).

The central pathways involved in jaw reflexs may be restricted to the brainstem. The trigeminal nerve is attached to the lateral part of the pons by a large sensory and a small motor root. The sensory root is continuous with the trigeminal ganglion which is located within the trigiminal cave in the middle cranial fossa. The cell bodies of the sensory neurons in the trigiminal nerve are situated in the trigiminal ganglion (Johnson & Moore, 1997).

The proprioceptive sensory fibres have their cell bodies located not in the sensory ganglion of the trigeminal nerve but in the mesencephalic nucleus, a central nucleus within the brainstem. Central branches of the cells in the mesencephalic nucleus connect with cell in the reticular formation from which axons pass to the motor nucleus of the trigeminal nerve to complete a reflex pathway.

The reticular formation consists of a large number of neurones synapses and runs through the brain stem from the medulla right up to the cortex. All sensory information coming into the central nervous system passes to the reticula formation. The reticular formation arouses the cerebral cortex. The activity of the reticular formation is depressed during sleep (Greene, 1978).

The pathway for the jaw opening reflexes is polysynaptic with the first synapse in either the supra trigeminal nuclei or the adjacent reticular formation. The final synapse is in the trigiminal motor nucleus which is situated in the brain stem (McKay et al, 1992). The following diagram shows the jaw-opening reflex pathway.



Schematic representation of the pathway for the jaw opening reflex in man. Note the polysynaptic connections between mechanoreceptive and nociceptive afferent nerves from mouth and face and the motorneurones. Abbreviations are: CNS: central nervous system; PNS: peripheral nervous system; JCM: jaw closinf mucle; VMoN: trigeminal motor nucleus; Vgang: trigeminal ganglion; SVN: supra trigeminal nucleus; RF: reticular formation; - inhibitory synapses; +: excitatory. (after McKay et al, 1992).

2.1.3.1 Mechanical stimulation

Orofacial function such as mastication may result in mechanical stimulation of several receptor types, i.e. spindles in the jaw elevator muscles, temporomandibular joint receptors, mechanoreceptors around the teeth in the peridontium, and acoustic receptors (Glas & Steenberghe, 1988). Further, jaw closing muscles are reflexly inhibited as a result of normal tooth contact during mastication (Ahlgren, 1969, Hannam et al, 1969). This has prompted the use of mechanical stimulation as the means of investigating the reflex control of elevator muscle activity.

During sustained clenching, weak and transient mechanical stimuli on a tooth elicit a variety of EMG reflexes in jaw-closing muscles (e.g. Hannam et al, 1969; Sessle & Schmitt, 1972; Steenberghe et al, 1981). These reflexes, denoted as the post stimulus EMG complex by Glas et al (1984), appear as a series of downward- and upward-going waves in full wave rectified and averaged EMG of the masseter muscles. They can be distinguished from chance fluctuations by a computer programme with statistical criteria (Glas & Steenberghe, 1981).

Yu et al (1973) investigated the inhibitory effects of innocuous and noxious stimulation of facial and intraoral sites on masseter muscle activity. Controlled mechanical stimulation of the upper lip in seven subjects resulted in inhibition of the electro-myographic activity of the masseter muscle. When a painful stimulus was delivered to the lip, a long-latency inhibitory period was produced, the intensity of the stimulus and the level of muscle activity maintained by the subject influenced the occurrence and duration of the inhibitory period. This confirmed the previous results of Sessle and Schmitt (1972). Bratzlavsky (1975) has also reported that mechanical taps applied to the labial area give rise to silent periods with a latency of 20 ms and duration of 40 ms.

Bjornland et al (1991) studied the effect of the tapping force on the masseteric post stimulus reflex. Up to three different inhibitory responses were demonstrated, depending on the force of the tap, with a lower threshold for the early inhibitory phase than for the late phase. It was concluded that the first inhibitory phase probably resulted from activation of periodontal mechanoreceptors, because it disappeared after infiltration of local anaesthetic around the tooth (Goldberg, 1971; Sessle & Schmitt, 1972; Aars, et al,

1984; Glas et al, 1985). With higher forces, receptors further away from the tooth, including the auditory receptors, may also contribute to the development of the reflex (Glas et al, 1988).

Sato et al (1994) investigated the reflex responses evoked by periodontal mechano-receptors in masseter muscles. Weak taps were applied to the labial surface of a central incisor tooth and these evoked inhibitory reflex responses in both masseter muscles. These reflexes were abolished when local anaesthesia was infiltrated around the stimulated tooth and noise was played into the ears through earphones. They concluded that, in the absence of auditory masking, the total reflex evoked by tooth taps is the result of the summation of the inputs from both periodontal mechanoreceptors and auditory receptors. This is in contrast with the results of Bonte & Steenberghe (1989) who observed that the reflex responses evoked by tapping an incisor tooth with a pendulum stimulator were not completely abolished by local anaesthesia, even in the presence of high-intensity auditory white noise. It is likely that the tapping force used with Bonte and Steenberghe, which was 10 times stronger than the one used by Sato et al, activated other receptors remote from the periodontal ligament.

Recently, Louca et al (1996) investigated the roles of periodontal ligament mechanoreceptors in the reflex control of human jaw closing muscles. Controlled mechanical stimuli were applied to an upper central incisor tooth in 19 subjects. Stimuli consisted of a ramp leading to a sustained plateau at an intensity of 1 N, in addition, 1 N tap stimuli were employed in some subjects. Tap stimuli generally produced a sequence of inhibition-excitation-inhibition-excitation, in agreement with other studies (e.g. Glas

et al, 1985). Applying ramp-plateau forces produced only short latency inhibitions and excitations. Tap simuli are more likely to set up vibrations which may activate receptors in the gingivae, periosteum, inner ear and temporomandibular joint. Therefore, it was concluded that periodontal ligament mechanoreceptors contribute only to shorter latency reflexes in human jaw closing muscles, with the longer latency reflexes being attributed to mechanoreceptors stimulation situated elsewhere due to vibration.

The findings of Louca et al (1996) are in contrast to the findings of Brodin et al (1993), who found that slow pushes applied to human teeth evoked, in addition to the short-latency responses, a long latency, primarily excitatory, responses in the masseter muscle, and at low stimulus intensity, there was little evidence for a preceding inhibitory response. However, the different forces applied in these studies might explain the discrepancies.

Masseter muscle reflexes in edentulous subjects wearing complete dentures have also been studied. Matthews and Yemm (1970) investigated the silent period in the masseteric EMG following tooth contact in subjects wearing full dentures. They found that the changes in muscle activity that follow tooth contact are similar to those of subjects with natural teeth. They concluded that silent periods in masticatory muscles are not necessarily dependent on stimulation of periodontal mechanoreceptors. In 1976, Nagasawa et al also studied the masseteric silent period after tooth contact in full denture wearers. The effect of anaesthesia on the silent period duration was also studied. Topical anaesthesia was applied on the mucous membrane under the upper and lower dentures. As the tapping rate was increased, the duration of the silent periods were

reduced. After topical anaesthesia, the duration of the silent period of the masseter muscle was considerably prolonged. As a result of these findings, they concluded that the origin of the silent period after the tapping of the teeth in centric occlusion is not necessarily due to superficial receptors in the mucous membranes supporting full dentures; other receptors, e.g. in muscle and joint, could be involved.

Brodin et al (1991) investigated the masseter muscle reflexes by tooth tapping prior to extraction of the remaining maxillary teeth, and compared these reflexes with those evoked by tapping on a corresponding denture tooth at intervals over a period of 4-6 months after insertion of an immediate denture. The general pattern of the masseteric reflex by mechanical stimulation of a denture was similar to that evoked by tooth tapping prior to extraction. However, the thresholds of these responses were higher with the denture in place. They suggested that the mechanoreceptors in the mucosa might take over the functional role of the periodontal mechanoreceptors for evoking the masseter reflex during tapping. Further, they suggested that the elevated threshold for masseter inhibition may cause a delayed onset of this protective reflex in denture wearers, who may thus be more prone to biting trauma than individuals with natural teeth.

2.1.3.2 Electrical stimulation

Electrical stimulation of oral mucous membrane produces no response if the muscles are relaxed (Goldberg, 1971), however, during voluntary contraction of the elevator muscles, periods of depressed activity are seen. Many investigators studied the reflexes in the masticatory muscles in response to electrical stimulation.

Yemm (1972,a) studied the response of the masseter and temporal muscles following electrical stimulation of oral mucous membrane. Electrical stimuli of 1 ms duration were applied to the incisive papilla. He observed a silent period in the electrical activity of both muscles to follow the stimulus. The silent periods appeared in both masseter and temporalis muscles, the timing was similar for both muscles. This finding was later confirmed by Godaux and Desmedt (1975) and Bratzlavsky et al (1976) who found that the two inhibitory periods occurred simultaneously in the masseter and temporalis muscles on both sides. In another study, Yemm (1972, b) confirmed the previous findings and concluded that the jaw-opening reflex following intra-oral stimulation seemed to be due mainly to inhibition of mandibular elevator muscles, since no change in the activity of the principal depressor muscles could be detected.

Bratzlavesky (1972) also studied the inhibitory reflex of the masseter and temporalis muscles in response to electrical shocks. The results showed that a painless electrical stimulus induced a suppression of the muscle activity; stronger stimulation induced a second reflex inhibition with longer latency. It was suggested that the late inhibition

seems to act in protecting the intraoral mucosa and the perioral skin from being bitten, further, he assumed that the short latency reflex inhibition is involved in the modulation of the jaw closing muscle activity during normal chewing and licking movements.

According to Yu et al (1973), innocuous electrical stimulation resulted in a period of inhibition with a latency and duration comparable to those produced by innocuous mechanical stimulation of the upper lip and incisors. At high levels of maintained muscle activity, the incidence and duration were markedly lower than those produced at lower levels of muscle activity. In agreement with the previous studies of Yemm (1972) and Bratzlavesky (1972), they found that the second inhibitory period usually occurred at noxious levels.

Carels and Steenberghe (1986) investigated the reflexes in the masseter muscle following electrical gingival stimulation. Seventy-two electrical stimuli were delivered to the gingiva overlying the root of an upper incisor. There was a similar sequence of inhibitory and excitatory periods in the left and right masseter muscles. The first inhibitory period was reported to have a latency between 10 and 17 ms and to last from 10 to 25 ms. When the second inhibitory period was observed, its latency is more variable, and values from 30 to 60 ms have been reported (Godaux and Desmedt, 1975; McGrath et al, 1981).

It has been found that the depth of the suppression is proportional to the intensity of the stimulation (Godaux and Desmedt, 1975; McGrath et al, 1981). Also it has been found that, as the stimulus intensity increases the two inhibitory periods become stronger and

tend to fuse (Godaux and Desmedt, 1975; Bratzlavsky, 1975). In addition, according to some authors, the first inhibitory period has a lower stimulus threshold than the second inhibitory period (Yemm, 1972; Yu et al, 1973; Bratzlavsky, 1975). In contradiction to these findings, Godaux and Desmedt (1975) and Desmedt and Godaux (1976) electrically stimulated the upper and lower lips and tongue and compared the threshold and form of EMG response to the subjects' reports of their sensory experiences. They found that the stimulus thresholds of the first and second inhibitory periods were found to be identical. Further, the threshold stimulus for inhibitory periods always caused slight pain. Similarly, tooth pulp stimuli were reported always to be painful by Bratzlavsky et al (1976).

However, McGrath et al (1981) carried out an experiment that was similar in design to that of Godaux and Desmedt (1975) but which gave different results. They found that the threshold for suppression of masseter muscle activity was approximately the same as each subject's sensory detection threshold and that was far below the pain threshold.

Turker and Miles (1985) investigated the effect of stimulus intensity and jaw opening on the pattern of inhibition of the jaw closing muscles in normal subjects. The results showed a characteristic, biphasic inhibitory response. Further, they found that the amount of initial muscle activity, the type and intensity of stimulus used, the jaw separation and the method of EMG analysis all affect the measurement of SP duration. In agreement of previous results of Godaux and Desmedt (1975), the inhibition consisted of 2 phases which tended to merge as the stimulus intensity increased.

Turker (1988) studied the effect of bite force and stimulus intensity on the electrically induced SP. Five different levels of bite force were used. The intensity of stimulation was determined using the sensory perception threshold for the subjects (T). Upon the results of his investigations, Turker suggested the following as a method for standardisation of silent period measurements in human masseter muscle: (a) the sensory perception threshold (T) of subjects should be used when studying and comparing the SP between individuals rather than the actual values of the electrical stimulation; (b) rectified and averaged EMG records should be used rather than single raw traces; (c) the subjects should be asked to perform 25 % of his/her maximum bite force and the bite force should be given as a feedback; (d) a stimulus intensity of about 6T should be used so that small changes in the bite force would not affect the SP duration.

Cadden and Newton (1988) studied the reflex depressions of masseter muscle activity evoked by intra- and peri-oral electrical stimulation. They found that, in all subjects, intra-oral stimulation produced two phases of depressed masseteric activity with mean latency of 14 ms and 47 ms. By contrast, the responses to the labial skin generally consisted of a single phase of depressed activity, the time of which was similar to the late phase produced by intra-oral stimuli (48 ms). These results were confirmed by the same authors in 1994, 1995 in which only the late inhibitory period was evoked in response to electrical stimulation of the upper lip. These findings are in agreement with pervious results of Yu et al (1973) but contradict those of Btatzlavsky (1972), Godaux and desmedt (1975), Desmedt and Godaux (1976), and Di Francesco et al (1986) who reported two inhibitory periods in response to peri-oral stimulation. Cadden and Newton

suggested that as the short-latency reflex is only evoked by stimulating nerves supplying intra-oral tissues, this reflex may play a role in functions such as mastication.

In summary, inhibitory reflexes in the electromyogram (EMG) of the jaw closing muscles have been elicited in many ways: (1) Mechanical stimulation: a tap is delivered by a light weight hammer, this tap is usually applied to the symphysis of the lower jaw in a downward direction (Fung et al, 1982). Tapping a tooth is also widely used (De Laat et al, 1985); (2) Electrical stimulation of intra- and peri-oral mucosa (Goldberg, 1971; Yu et al, 1973; Carels and Steenberghe, 1986) and of the tooth pulp (Bratzlavsky et al, 1976); (3) Tooth tapping (Hannam et al, 1969); and (4) The reflex responses of single motor units in jaw muscles has been studied (Miles and Turker, 1986; Miles et al, 1987; McMillan and Hannam, 1989, 1992; McMillan, 1993).

Double inhibitory periods occur less frequently after mechanical stimulation of a tooth than after electrical stimulation of the gingiva above the same tooth (Carels & Steenberghe, 1986; De Laat et al, 1988). The incidence of single or double inhibitory periods is also not the same after electrical stimulation of different sites, the second inhibitory period being predominant after facial stimulation (Widmalm & Ash, 1985, Cadden & Newton, 1988,1994), and both inhibitory periods always being present after intra oral stimulation (Godaux & Desmedt, 1975, Turker & Miles, 1985).

There are several problems with the use of mechanical stimuli in the study of inhibitory jaw reflexes. Firstly, it is difficult to deliver a mechanical stimulus in a reproducible manner (Bishop et al, 1984). Secondly, a mechanical stimulus which is large enough to

cause inhibition will also stimulate several receptor systems which have different reflex connections with the motor neurone under study. However, according to Turker and Miles (1989), electrical stimulation offers the following advantages over mechanical stimulation in the study of inhibitory jaw reflexes: (a) the stimulus parameters (intensity, duration, and frequency) can be readily set and kept constant; (b) electrical stimulation does not cause vibration and hence does not stimulate the vibration sensitive receptors in muscles and in the inner ear; (c) electrical stimulation does not change the jaw position and hence does not activate the position sensitive receptors which are situated in the jaw muscles and in the temporomandibular joint.

2.1.3.3 Acoustic stimulation

Meire-Ewert et al (1974) reported that a strong acoustic stimulus can evoke an inhibitory response in the jaw closing muscles. The response was evoked only at very high stimulus intensities. These reflexes were said to be absent in subjects who were totally deaf. However, it was not clear whether the present reflex was the result of sound entering the ear, and/or conduction of a shock wave through the bone.

The relationship between acoustic stimulation and the silent period was also studied by Kobayashi et al (1981). Twenty normal subjects were subjected to pure tone stimulation,

delivered unilaterally and bilaterally while performing a clenching exercise. The results showed no SP of the diagastric muscle for any subject, while only four subjects exhibited SP in their temporalis and masseter muscles. They concluded that larger stimulation should be used to elicit SP and they suggested, therefore, that TMJ clicking may cause SP.

2.2 Temporomandibular Disorders (TMD)

2.2.1 Introduction

The current view is that TMD is a cluster of related disorders in the masticatory system, and not one single disorder (Griffiths, 1983; Bell, 1990; McNeill, 1993). The word "syndrome", which has been used in association with TMD (Costen, 1934; Schwartz, 1955; Laskin, 1969), is inappropriate because TMD consists of several different, but related, disorders (Reynolds, 1988).

Currently, the term temporomandibular disorders (TMD) is considered the preferred term and defined as "a collective term embracing a number of clinical problems that involve the masticatory musculature, the temporomandibular joint and associated structures or both" (McNeill, 1993).

2.2.2 Incidence

According to epidemiological studies, the signs and symptoms of TMD are highly prevalent and may be present in up to 70 % of the general population (Agerberg &

Carlsson, 1973; Helkimo, 1974; Solberg et al, 1979; Schiffman et al, 1990). However, these relatively high prevalence figures do not necessarily imply the existence of a clinical TMD that is in need of treatment (Kleinknecht et al, 1987). It is estimated that about 5%-7% of the population reporting signs and symptoms of TMD seeks treatment for these symptoms (Greene & Marbach, 1982; Schiffman et al, 1989; Dworkin et al, 1990).

The prevalence of TMJ disturbances among complete denture patients has also been studied by many authors (e.g. Bergman and Carlsson, 1972; Agerberg and Carlsson, 1975; Mercado and Faulkner, 1991) who demonstrated a high prevalence of TMJ dysfunction symptoms in denture wearers. Carlsson (1976) reported that individuals with few remaining natural teeth had more symptoms of mandibular dysfunction than those with most of their natural teeth, and complete denture wearers had more symptoms than those with natural teeth. These results are also supported by Helkimo (1976).

2.2.3 Muscle Pain

Pain is the most common complaint in patients with TMD and is the most important reason for seeking treatment (Greene et al, 1969; Dworkin et al, 1990; Bell, 1990; McNaill, 1993). It arises from irritation of nociceptor systems, located in adventitial sheaths of blood vessels running between muscle fibres. This may occur physically from

mechanical trauma, or chemically as a result of muscle hyperactivity (muscle spasm) or muscle fatigue (Klineberg, 1988). Overuse, in terms of sustained activity and high level of contractions without rest periods, inhibits venous drainage from muscles leading to an accumulation of metabolites such as carbon dioxcide and lactic acid which produce chemical irritation of nociceptive afferent leading to pain (Klineberg, 1988)

2.2.4 Aetiology

Many different theories have been proposed but most authors agree on a multifactorial aetiology, with muscle hyperactivity as one of the key mechanisms.

Masticatory muscle hyperactivity theory

The explanation of masticatory muscle hyperactivity is divided into two categories: peripheral (e.g. occlusal interferences) and centrally mediated (Yemm, 1976).

Peripheral cause (Occlusal interferences)

Occlusal interferences (malocclusion) has been one of the most frequently cited causes of TMD (Moss & Garrett, 1984). It has been proposed that malocclusion of the teeth

results in mandibular displacement, usually in the posterior direction, and can cause compression of sensitive soft tissues. Continued compression of this tissue could result in pain as well as impairment of the blood supply to joint structures. This, therefore, could lead to degenerative changes (Laskin, 1969). Support to this cause (malocclusion) is provided by the finding that many TMD patients have malocclusion (Ramfjord, 1961; Mohlin et al, 1980; Graham et al, 1982) and that occlusal adjustment is often followed by clinical improvement in a high percentage (Kopp, 1979; Weinberg, 1979; Ingervall & Carlsson, 1982).

Malocclusion has also been suggested as a factor which may lead to destructive oral habits, such as bruxism, which may be related to TMD (Ramfjord, 1961; Beyron, 1969; Trenouth, 1979). This proposition has been criticised on the grounds that not all bruxists have malocclusion and that not all individuals with malocclusion are bruxists (Thompson et al, 1972; Glaros & Rao, 1977). This has led to the suggestion that muscular hyperactivity may originate centrally in the nervous system (Franks, 1965,b; Berry, 1967; Weinberg, 1974; Yemm, 1976).

Centrally mediated causes

The other important cause of masticatory muscle hyperactivity is psychological and/or physical stress (Berry, 1967; Franks, 1965; Laskin, 1969; Yemm, 1976). Two types of studies have supported this hypothesis: (a) studies of stress in TMD patients and (b)

studies which have attempted to experimentally induce stress and measure the physiological reactions of TMD patients.

A high incidence of psychophysiological disorders (stress-related symptoms) in TMD patients has been found. Berry (1969) noted an incidence rate of headache and backache which was ten times higher than that reported for normal populations. Also Gold et al (1975) found that when compared to a group of healthy controls, TMD patients reported more frequent low back and neck pain, nervous stomach, asthma and history of ulcers. However, this does not necessarily means that these disorders are psychologically-induced, it may simply be that these particular patients are susceptible to musculo-skeletal disorders.

Muscular activity has been studied in stressful situations. Kydd (1959) found that jaw muscle EMG activity increased during stress. Further, Yemm (1969) showed that patients with TMD are more likely to maintain stress-induced muscle activity. These findings were later supported by Mercuri et al (1979) who found that TMD patients exhibited significantly higher masseter and frontalis EMG activity in contrast to controls.

Other studies have found a decrease in stress-related symptoms in TMD patients after treatment. This decrease can probably be attributed to the decrease in the symptoms of TMD (De Leeuw et al, 1994). These findings support the idea that these emotional reactions are a consequence of TMD rather than a precipitator (Greene et al, 1982; Gamsa, 1990).

Elevated EMG activity levels have been found in the masticatory muscles of TMD patients (Jarabak, 1956; Perry, 1957; Lous et al, 1970; McCall et al, 1978; Sheikholeslam et al, 1982). Also pain similar to that of TMD has been induced in non-TMD subjects by sustained contraction of the masticatory muscles and this supports the notion of muscular involvement in TMD (e.g. Christensen, 1971, 1975; Scott & Lundeen, 1980).

2.2.5 Treatment

Most patients respond positively to conservative treatment. Further, long term follow-up of patients with TMD shows that over 50% of the patients have few or no symptoms after conservative treatment (Carlsson, 1985). The treatment procedures are extensively reported in the literatures, however, only the occlusal appliances (splints) will only be reviewed in this chapter.

2.2.5.1 Occlusal splints

An occlusal splint is a device made of hard or soft acrylic resin that is placed on either the upper or lower teeth. These appliances have been widely used in treatment of TMD, and the success with occlusal splint therapy has been reported in 70-90% of TMD

patients (Greene & Laskin, 1972; Roura & Clyton, 1975; Carraro & Caffesse, 1978; Dahlstrom et al, 1982; Okeson et al, 1982, 1983; Clark, 1984; Sheikhleslam et al, 1986; Tsuga et al, 1989; Suvinen & Reade, 1989; Wilkinson et al, 1992; Visser et al, 1995).

Full-coverage stabilization splints

These appliances offer the advantage that they control and maintain tooth position. They made of hard acrylic resin which can be used on either arch. However, the upper splint is more stable, more retentive and less likely to break. The splint should cover the whole arch to establish a simultaneous and even contact with the opposing teeth in the retruded jaw position. Anterior guidance should be provided by adding a ramp in the canine or anterior area to avoid any posterior contacts during lateral and protrusive excursions (Ramfjord & Ash, 1994).

The stabilization splints are mainly used to relax the masticatory muscles, decrease the parafunctional activity that often accompanies periods of stress (bruxism and clenching of teeth), redistribute occlusal forces, protect the teeth from wear, and stabilize the temporomandibular joints (Clark et al, 1979; Okeson et al, 1983; McNaill, 1985; Sheikholeslam et al, 1986).

Several types of occlusal splints are used in the treatment of TMD. However, a stabilization-type is considered to provide highly effective but reversible therapy (Clark, 1984; Ash, 1986). Tsuga et al (1989) selected 30 patients with more than two major

symptoms (TMJ pain, masticatory muscle pain, or limitation of movement) and evaluated the short-term effectiveness of stabilization occlusal splints worn during sleeping hours. They found that 87% of the patients with pain responded to this therapy and more than 50% had complete relief from pain 4 weeks after insertion of the splint. Patients with joint sounds and limitation of movement also responded but not as significantly as those with pain.

2.2.5.2 Effect of occlusal splints treatment on EMG activity

The effect of occlusal splints on EMG activity has been widely studied. Fuchs (1975) found that TMD patients without splints have higher muscular activity during sleep than healthy persons and that use of the splints reduces masticatory muscle activity to the same level of normal subjects.

Holmgren et al (1985) and Sheikhoeslam et al (1982) investigated the postural activity of the temporal and masseter muscles in TMD patients before, during and 3-6 months after insertion of a maxillary stabilization splint. A significant reduction in the muscular activity was found in 52% of patients with the splint in situ. The symptoms were also significantly reduced after treatment. However, after cessation of splint therapy the signs and symptoms returned to pre-treatment levels in 80 per cent of patients. Similar findings of decreased muscular activity after splint therapy have been reported

(Balciunas et al, 1987; Chong-Shan & Hui-Yun, 1989; Naeije & Hanson, 1991; Carr et al, 1991; Lobbezoo et al, 1993; Visser et al, 1995).

2.2.6 The EMG inhibitory period (SP) in TMD

Bessette et al (1971) studied and compared the silent period durations in the masseter muscles of TMD patients and normal subjects during maximal voluntary clenching. They noted an abnormally long silent period duration following the jaw-jerk reflex in TMD patients (mean 60 ms) compared to that seen in normal subjects (mean 24 ms). After conventional occlusal treatment with marked symptom improvement, the patients' SPs were significantly shortened and fell within the normal range. On the basis of these observations, they proposed that the duration of the masseteric silent period during a maximal voluntary clench could be a useful diagnostic tool and assist in the prognosis and treatment of TMD.

Beemsterboer et al (1976) and McNamara (1977) also analysed the EMG silent period of the temporal and masseter muscles in TMD patients before and after treatment with occlusal splints. The mean duration of the silent period was significantly longer before treatment than after treatment. Consequently, they supported the proposal that the duration of the silent period may be used as a diagnostic measurement and also as an indication of treatment effectiveness.

Further studies by Bessette and Shatkin (1979) claimed that masseteric silent periods could be used to predict the outcome of treatment for TMD patients, and their results suggested that 93 % of patients could be treated successfully with occlusal splints and occlusal adjustments. Skiba and Laskin (1981) compared the silent period in asymptomatic adults and TMD patients unilaterally in the masseter, temporal, medial and lateral pterygoid muscles. In agreement with Bessette and Shatkin (1979), all of the patients had significantly prolonged silent period in at least one masticatory muscle. After successful conservative treatment, silent period durations showed values within normal limit. Bailey et al (1977) also supported the previous reports in which the TMD group had significantly longer silent periods than the healthy group.

The effect of TMJ symptom severity on the EMG silent period was studied by McCall et al (1978) in 48 patients. The silent period of masseter and anterior temporalis muscles was evoked by a tap to the chin during maximal clenching. The mean silent period duration increased significantly with increased severity of TMJ dysfunction. The latencies were not found to differ significantly. This is also supported by Helkimo et al (1979) who found that the SP duration increased with increased severity.

The variability of the silent period duration of the masseter and temporal muscles was examined by McCall et al (1981) in TMD patients. Unilateral recordings were made of five taps during maximal clench. Considerable intra-subject variability in the duration of the SP was found and the variability increased with the symptom severity. It was recommended that multiple measurements should be averaged to obtain an accurate estimate of the SP duration. McCarrol et al (1984) investigated the SP duration in TMD

patients with varying severity of symptoms from the masticatory muscles. EMG signals were recorded from the left and right masseter and temporal muscles. They found, in contrast to McCall et al (1978, 1981) and Helkimo et al (1979), that SP did not increase with increasing severity of symptoms. Unilateral pain was, however, associated with an increased activity in the masseter muscle and SP duration on the affected side was longer compared to the unaffected side. It was concluded that TMD symptoms, whether of arthrogenic or myogenic origin, result in asymmetry of the neuromuscular response in the masseter muscle between the affected and non-affected sides.

Moini et al (1980) investigated the effect of a non-occluding (palatal) splint and occlusal bite plane splint on the latency and duration of the silent period in healthy subjects in response to a chin tap during maximal clench. The mean silent period durations did not differ among three muscles (left and right masseter and right temporalis muscles) and agreed with previous studies which compared the duration between masseter muscles (Bessette et al, 1971; Widmalm, 1976) and among masseters and anterior temporal muscles (McNamara et al, 1977; Bailey et al, 1977). This finding suggests that the duration from one side only needs to be measured. The insertion of a palatal splint did not change the silent period duration. However, the mean silent period duration with the full maxillary occlusal bite plane splint was found to be significantly longer than without a splint. This is in contrast with McNamara (1976) who used the open-close-clench method of eliciting silent periods and found that the insertion of a splint did not alter the mean duration. The difference in methods, however, may account for the difference in results.

Bernstein et al (1981) investigated the influence of voluntary muscle activity on the duration of the masseteric silent period in both asymptomatic and dysfunctional individuals. A statistically significant decrease in the silent period duration was observed as the voluntary effort was raised from 40 % of maximum force to 100 % of maximum force in both groups. This is in agreement with previous results of McNamara et al (1977) who found a decrease in duration with increasing force. The results confirmed once again that TMD patients have a longer silent period duration than healthy subjects. Fung et al (1982) also found that the duration of the silent period varied inversely with the magnitude of bite force.

Often a longer silent period is reported to be found in TMD patients. However, in the literature there is disagreement on the influence of experimental parameters on the silent period. It was reported by Bessett et al (1973), Bailey et al (1977), and Palla et al (1981), that the bite force and the strength of the tap do not influence the silent period duration. On the other hand, other studies conclude that the silent period is shorter at higher bite force levels and it is longer at stronger taps (McNamara et al, 1977; Bernstein et al, 1981; Fung et al, 1982; McCarroll et al, 1984; Kroon and Naeije, 1984; Tamura et al, 1995).

Naeije and Hansson (1986) studied the differences between myogenous and arthrogenous TMD patients. Surface EMG recordings were obtained from the right and left masseter and anterior temporalis muscles. Standardized taps were delivered on the chin of the patients. There was no statistically significant difference in either EMG

amplitude or SP duration between healthy subjects and TMD patients. However, a statistically significant difference between the two groups of patients was found. They suggested that the difference in the amplitude and silent period duration supported the distinction into a myogenous or arthrogenous origin of TMD pain.

Electrically evoked reflexes were studied by Sharav et al (1982). They examined the masseter inhibitory period, evoked by tooth pulp stimulation, in healthy subjects and patients with TMD. They found three different configurations of inhibitory periods in TMD and normal groups, (1) a single inhibitory period with a latency of 10-15 ms and a duration of 10-20 ms, (2) a double inhibitory period in which there were two depressions separated by a burst of muscle activity, the first depression had a latency and duration similar to the single period, while the second had a latency of 35-40 ms and a duration of 15-25 ms, and (3) a merged inhibitory period in which the two inhibitory periods seem to be combined, the latency of the merged inhibitory period between 8-12 ms and a duration of 60-75 ms. In contrast to healthy subjects, TMD patients had a larger proportion of single inhibitory periods and a smaller proportion of double inhibitory periods. The total duration of the double inhibitory periods was significantly shorter in TMD patients. They concluded that the reduction in the duration of the masseter inhibitory period is consistent with the hypothesis that there is hyper-excitability of jaw closing muscles in TMD.

In another study of electrically evoked reflexes, Hussien and McCall (1983) investigated the masseter muscle reflexes in TMD patients in comparison to healthy subjects. Electrical pulses were delivered to the right mental nerve area. Five pulses of 3.3 mA

intensity and 0.1 ms duration were delivered at about 1 s intervals while the subjects clenched at 100 % of maximum EMG and then at 50 % of maximum. In contrast to previous study by Sharav et al (1982), two silent periods were typically observed from each masseter muscle in both healthy and TMD patients. The early and late silent periods had shorter latencies and longer durations in the TMD group compared to the normal group.

Furthermore, Hussien and McCall reported that the durations of both early and late silent periods were longer in the TMD group at 100 % masseter muscle activity than in the healthy group, even when the healthy group clenched with only 50 % maximal EMG activity. This is in agreement with Bernstein et al (1981) who found that TMJ patients had silent period durations which were still significantly longer than healthy subjects even when the duration of the SP's in the TMD group at 100 % of maximal activity was compared with the duration in the healthy group at 40 % of maximal activity.

De Laat et al (1985) focused on the pattern of reflexes rather than the duration of the silent period. They referred to this pattern as the post-stimulus EMG complex (PSEC) and studied this in patients with TMD. The PSEC were derived by standardised mechanical stimulation of an upper central incisor during clenching at a constant level of 5 % of maximal masseteric EMG activity. Seventy-two sweeps per subject were processed by means of a computer programme. In bruxism and TMD, a single inhibitory period was found whereas in symptom free subjects, a double silent period occurred. There was no correlation to symptoms severity but there was a strong correlation between tooth-grinding habits and the occurrence of a single inhibitory period. This

pattern changed to include two inhibitory periods after occlusal splint therapy (De Laat & Steenberghe, 1985). Turker et al (1989, 1995) also studied the inhibitory periods in TMD patients and found that the occurrence of a single inhibitory period in TMD patients was predominant.

Recently, Bjornland et al (1996) studied the reflex response of the masseter muscle in patients with chronic arthritis or internal derangement of the TMJ in comparison with symptom-free subjects. Mechanical taps were delivered to the central incisor. The duration of the first inhibitory wave was significantly longer in the arthritis group than in the derangement group, and longer compared to the control group. This supports other studies which have shown that patients with TMD have a longer duration of the short latency inhibition than healthy subjects (Helkimo et al, 1979; De Laat et al, 1985; Turker et al, 1989). However, in contrast to De Laat et al (1985) and Turker et al (1989), they observed no marked differences between the three groups of subjects with regard to the occurrence of the second inhibitory wave.

In summary, the reported durations of the inhibitory period (silent period) differ considerably and this may be a consequence of methodological variations. In spite of these differences, there is a consistent finding among these reports that longer SP duration occurred in TMD patients in comparison with healthy subjects. Increased symptoms severity, is associated with increased silent period duration. Further, following successful treatment of TMD patients with occlusal stabilization splints, lengthened masseteric silent periods returned to normal. However, controversy exists regarding the occurrence of double inhibitory periods in TMD patients, as some have

reported the occurrence of only a single inhibitory period, while others have reported the occurrence of double inhibitory periods as in healthy subjects.

2.3 Bruxism

Bruxism has been defined by Nadler (1957) as the grinding or clenching of teeth at other times than for the mastication of food.

2.3.1 Prevalence

Numerous studies have investigated the prevalence of bruxism. Nadler (1957) stated that 70 to 80 % of the population suffers from bruxism. Agerberg and Carlsson (1975) indicated, from a sample of 1106 people, that 10% described grinding of the teeth and 20% clenched the teeth. Tooth grinding was most commonly seen in the 25-34 age group, while clenching was most commonly seen in the 45-54 age group. However, Helkimo (1974) showed that parafunction to be common among all age groups (over 50% in the 35-44 year old group).

Because of different methodologies, operational criteria, population samples, and definitions used in various investigations, the reported prevalence of symptoms related to bruxism varies from 15% to 90% in the adult population, and from 7% to 88% in

children (Attanasio, 1991). Although most individuals demonstrate signs of bruxism, only 5 to 20% of the general population are aware of this habit (Glaros, 1981).

2.3.2 Signs and symptoms

Pavone (1985), in reviewing the signs and symptoms of bruxism, described abnormal attrition patterns, chipping and splitting of the teeth, tooth mobility, pulpal hyperaemia, increased muscle tonus, muscle hypertrophy (particularly the masseter muscles), muscle tiredness (especially on awakening), trismus, cheek or lip or tongue biting, headache, audible sounds of bruxism, periodontal disease, occlusal disharmony especially a discrepancy between centric relation and centric occlusion as being the major signs and symptoms.

Painful masticatory musculature, especially to palpation, can be a common symptom of nocturnal bruxism and can even result in limited movement of the mandible upon awakening (Clark et al, 1981). In addition this mandibular parafunction activity may be responsible for TMJ pain, muscle tension headache pain (Glarose & Rao, 1977; Villarosa & Moss, 1985; Nilner, 1981, 1985).

2.3.3 Aetiology

Various aetiological theories have been proposed in the literature, generally these theories fall into three categories: occlusion-related (Krough-Poulson & Olsson, 1966; Graf, 1969), psychological (Rugh et al, 1984), or origin within the central nervous system (Kopp, 1982). Therefore, the aetiology of nocturnal bruxism is still controversial, and most probably it is multifactorial and overlapping (Attanasio, 1991).

Malocclusion, occlusal discrepancies, and faulty restorations are thought to be the precipitating factors for bruxist activity (Dawson, 1989). The assumption was that the patient was making a subconscious effort to eliminate occlusal interferences. However, several studies suggested that there was no direct relationship between bruxism and occlusal interferences (Kardachi et al, 1978; Ingervall et al, 1980; Rugh et al, 1984). Furthermore, occlusal adjustment did not stop bruxism (Karadchi et al, 1978; Bailey & Rugh, 1980). Also, the experimental induction of occlusal discrepancies did not elicit nocturnal bruxism (Rugh et al, 1984).

Many investigators have used a ***psycho-analytical*** approach in their studies of bruxism. Molin and Levi (1966) examined 103 bruxists and compared them with normal subjects. All participants completed a personality questionnaire. They reported that anxiety and depressive symptoms were more evident in bruxists than in the control group. Thaller et al (1967) also showed a significantly higher anxiety trait in bruxists than non bruxists.

Strother and Mitchell (1954) placed great emphasis on the role of emotional disturbances and psychic factors in the aetiology of bruxism. Franks (1965,b) studied the relationship of emotional tension, muscular hyperactivity, and bruxism to the temporomandibular symptoms. He reported that there was repeated confirmation of the relationship of bruxism to a general background of emotional disturbances and nervous tension.

On the other hand, Marbach et al (1978) investigated the relationships between facial pain, bruxism and anxiety levels. They found that the level of anxiety did not differ in patients with facial pain from controls and also there was no difference between facial pain sufferers who bruxed and those who did not. This view was also shared by Scott and Humphreys (1987) who reported that there was no conclusive evidence of psychiatric involvement with bruxism.

However, Lowenthal (1981) emphasized the relationship between stress and anxiety on the one hand and bruxism and muscle pain on the other hand. He further stated that facial pain and other problems were relieved when the psychological problems were discovered. Other workers supported the view of the association between psychological problems and bruxism (Carlsson, 1981; Dworkin and Burgess, 1987; Dettmar and L'Estrange, 1987; Lundeen et al, 1987).

The aetiology of nocturnal bruxism is thought to be a ***sleep disorder*** that is centrally mediated, and precipitated by emotional stress (Rugh & Harlan, 1988). Extensive

studies have been carried out during various stages of sleep to investigate muscular activity in bruxists. In 1964, Reding et al studied the sleep patterns of tooth grinding in 12 bruxist patients. They found that bruxism was associated with periods of rapid eye movement (REM) sleep, indicative of dreaming. Contraction of the masseter muscle occurred at rate of 20.9 per hour during REM periods as compared to 5.3 per hour during non-REM periods. Heightened activity of the masseter muscles was also observed during movement of the trunks and limbs. These findings are later supported by Powell (1965) and Robinson et al (1969) who postulated that nocturnal grinding of teeth is associated with partial arousal from sleep.

Studies have shown that the number and duration of bruxing events during sleep vary greatly, not only between different patients but also within the same patient. Okeson et al (1994) reported that the subjects averaged one bruxing event every 10.7 min of sleep. These findings are similar to Reding et al (1968) who reported one event every 13.5 min of sleep. Okeson et al (1994) also reported that bruxing events were associated with an increase in heart rate. It was found that every subject had an increase in heart rate during the bruxing event. The average increase was 16.6 % varied considerably from 6.1%-40.2%.

In another study, Clarke et al (1984) compared the forces of bruxism in sleep with those produced by maximal conscious effort. They found that the forces of clenching bruxism were generally high when compared with maximum conscious clenches. Kydd and Daley (1985) reported that a group of 10 bruxists clenched their teeth for a total mean duration of 11.4 min. per night. These clenches occurred in single episodes lasting 20-40

sec, while non-bruxers had a mean duration of 3.1 min. These findings are much less than those reported by Trenouth (1979) who revealed that a TMJ/bruxing group spent 38.7 min with the teeth together during an 8 hours period, whereas control group spent only 5.4 min with the teeth together during an 8 hours period.

2.3.5 Effect of occlusal splints on bruxism

The occlusal splint is one of the most universally accepted forms of therapy in patients with signs and symptoms of functional disorders of the masticatory system and nocturnal bruxism (Faulkner, 1990). The occlusal splint allows for optimal positioning of the condyles and discourages bruxism through elimination of the habitual dysfunctional contact patterns of the teeth (Ash and Ramfjord, 1995).

Electromyography (EMG) has been used to study the functional mechanism of the occlusal splints. Solberg et al (1975) measured nocturnal activity of the masseter muscle in 8 patients with bruxism and found that muscle activity was reduced after insertion of a full arch occlusal splint. When occlusal splints were removed, pre-treatment EMG levels returned.

Kawazoe et al (1980) found that, in subjects with normal occlusion and without signs and symptoms of functional disorders, there was no significant difference between the levels of EMG activity in the masseter muscle during maximal bite in the intercuspal

position compared to maximal clenching on occlusal splint. While in patients with occlusal interferences (premature contacts) as well as functional disorders, the level of EMG activity during maximal clenching on the occlusal splint was significantly lower than during maximal bite in the intercuspal position.

Cox et al (1982, 1983) studied the silent period of the masseter muscle in experimental bruxing with and without the use of bite splints. A jaw jerk was stimulated by tapping the mandibular symphesis with an electromagnetic hammer. All subjects showed a significant increase in silent period duration immediately following and 3 hours after a session of experimental tooth grinding. The silent period had returned to presessional levels by 3 weeks. Further, while small increases in the silent periods were observed while wearing the splints, no statistically significant increases were obtained and maximum level of muscle activity was lower than those recorded without the splints. This latter finding supports the previous work of Christensen (1981) and supports the use of occlusal splint in the treatment of bruxism.

Shan and Yun (1991) studied the effect of an occlusal splint on the EMG of the masseter muscles in 23 patients. They found that an occlusal splint is effective in balancing the activities of the two masseter muscles and that occlusal splints can decrease masseter muscle activity, thus exerting a therapeutic effect.

However, Holmgren et al (1993) studied the effect of a full-arch maxillary occlusal splint on grinding and clenching during sleep in bruxers with TMD. Active shiny facets caused by nocturnal bruxism appeared on the occlusal surface of the splints of all

patients. In 61% of the patients, wear facets were observed at every visit, (2-week interval), in each visit the splint was polished by sandpaper only to remove the shiny area of the facets. The results revealed that the occlusal splints did not stop the habit of nocturnal bruxism. This finding is in line with the earlier findings of Kydd and Daly (1985), who claimed that occlusal splints did not significantly reduce bruxing activity.

In regard to the material of occlusal splint fabrication, Okeson (1987) compared soft and hard splints, and claimed that 80 % of patients using hard splints had reduced nocturnal bruxism, while only 10 % of those using soft splints showed improvement.

2.4 Muscles of mastication

The masticatory muscles performs movements and provide force for a variety of functions such as jaw posture, speech, and swallowing, mastication and clenching. The relative contribution to the closing force from each of the jaw elevator muscles is about 30-50 % for the temporalis, 35 % for the masseter, and about 20-40 % for the medial pterygoid muscle, the temporalis muscle being most predominant in slow contractions (Devlin & Wastell, 1985).

The power capability of the masseter muscle is high because of its multipennate arrangement of the fasciculi. It is bulky and rectangular, and is placed between the zygomatic arch and the ramus of the mandible on each side. The major direction of the pull of the masseter muscle is vertical with an anteromedial component, most typically for the superficial portion, whereas the deep portion also has a posterolateral component (Belser & Hannam, 1986; Eidgen, 1990).

The major contribution of the muscle takes place with the teeth in contact, and during mastication in the working side when crushing and grinding effects are required (Diaz-Tay et al, 1991). The superficial portion is most active in the working side, whereas the deep portion is more active than the superficial portion in the balancing side during unilateral chewing (Belser & Hannam, 1986).

The muscle fibres which are cylindrical or spindle-shaped cells, have considerable variation in size and length between various muscles in order to facilitate the various activity of the muscles, the main diameter in adult jaw elevator muscles ranges from 20-60 μm (Eriksson & Thornell, 1983). Between the muscle fibres is the endomysium, a thin connective-tissue matrix, with a supply of capillaries, of which the density in the jaw muscles is considered to be high and uniform (Eriksson & Thornell, 1987). Atrophy of muscle fibres, which might take place because of age may be counteracted by exercise and physical activity (Grimby & Saltin, 1983).

Muscle fibres may be classified into 3 types, these include: Type 1, low ATPase activity, long contraction times (slow-twitch), and resistance to fatigue; Type 11A, high ATPase activity, rapid contraction times (fast-twitch), and intermediate fatigability; and Type 11B, fast-twitch and high fatigability (Buchthal & Schamalbrugh, 1980). Type 1 fibres predominate, most markedly in the masseter muscle (70-80 %) (Eriksson & Thornell, 1983). This supports the finding that jaw muscles have a higher resistance to fatigue than limb muscles (Steenbergh et al, 1978; Clark & Carter, 1985). The superficial part of the temporalis muscle was comprised of 50% type 11B fibres, which indicates the ability to develop higher forces and to contract rapidly.

The functional entity of the muscles is the ***motor unit***, which consists of the motoneuron, its axon and the muscle fibres innervated by branches of the axon. All muscle fibres in a motor unit are considered to be of uniform histochemical, functional and morphologic type (Buchthal & Schmalbrugh, 1980).

The Proprioceptors are sensory receptors located in deep tissues where they signal the consequences of internal motor activity. These sense organs include muscle receptors (muscle spindles and tendon organs), temporomandibular joint receptors, and periodontal mechanoreceptors (Ash & Ramfjord, 1995).

Muscle spindles are stretch receptors providing information about the relative length and rate of length change of the muscle fibres, by contraction or relaxation of the muscle fibres, the motoneuron adjust the length of the spindles (Jami, 1992). These muscle spindles are receptors for an excitatory jaw-closing reflex (stretch reflex).

Joint receptors are found in the capsule and ligaments of the temporomandibular joint (Bakke, 1993). Some of the mechanoreceptor afferents fire almost continuously during jaw opening; others are phasic with bursts during maximal opening, lateral deviation, or with the teeth in contact, providing information about jaw displacement and velocity (Klineberg, 1988).

Periodontal mechanoreceptors are those receptors that respond to forces applied to the teeth (Johansson & Olsson, 1976; Steenberghe, 1979). These sensory receptors in the periodontal membrane and periosteum surrounding the teeth are either free axonal endings (mainly pain receptors) or organized receptors (mainly pressoreceptors) (De Laat, 1987). Periodontal pressoreceptors are direction sensitive and are affected by the rate of force application. The low-threshold pressoreceptors provides positive feedback to the jaw-closing muscles and therefore an increase of the biting force, but as bite force

reaches a certain magnitude, other periodontal pressoreceptors with higher thresholds reacting to pressure are activated and limit bite force (Lavigne et al, 1987; Ottenhoff et al, 1992).

Muscle contracts normally in response to a stimulus, natural or artificial, this contraction can be recorded by EMG as follows: the stimulus is transmitted via the motor nerve fibre and its terminal branches to the neuromuscular junction. At the neuromuscular junction acetylcholine is released, and this allows sodium ions to inter the end-plate receptors and generate a local end-plate potential. When this end-plate potential reaches a critical magnitude, the rest of the muscle fibre becomes depolarised and forms a muscle action potential which precedes the mechanical contraction. The action potential is due to sodium ions entering and potassium ions leaving the muscle (Green, 1978).

The ***conduction velocity*** in a nerve fiber is usually proportional to the size of the fibre i.e. the larger the nerve fibre diameter, the faster the conduction velocity (Ganong,1991). Nerve fibers have been classified into groups by size, rate of conduction, and function. Two different classifications are used, a numerical system (Ia, Ib, II, III, IV) and a letter system (Aα, Aβ, Aγ, Aδ, B, C).

The larger nerve fibres, Aα (I) with a diameter of 12-21 μm and with conduction velocity of 70-120 m/sec, convey afferent (i.e. sensory) information from large complex peripheral mechanoreceptors, such as muscle spindles (Ia) and tendon organs (Ib). They also provide efferent (i.e. motor) connection via motoneurons to motor end plates of skeletal muscles. Aβ fibers which are smaller in size (diameter 5-12 μm with conduction velocity of 30-70 m/sec), are responsible for conveying touch and pressure sensation (Ganong, 1991).

The nerve fibres transmitting pain sensation may be either myelinated Aδ fibres (2.5 μm diameter, conducting at 12-30m/sec) thought to be responsible for acute, sharp pain and non-myelinated C fibers (0.4-1.2 μm in diameter, conducting at only 0.5-2 m/s) thought to be responsible for dull, more diffuse pain. Aδ fibers also convey information from thermoceptors and low threshold mechanoreceptors and C fibres are thought to be purely nociceptive (Klinberg, 1991).

2.5 Measurement of pain

Pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage." (Merskey, 1979). Several assessment methods have been used to objectively measure the complex perceptual experience of pain by the patient or subject. In clinical pain assessment, the most common methods employed have been behavioural measurements, subjective pain reports (such as visual analogue scales) and word descriptors. The critical analysis of these methods in general pain assessment has been discussed by Chapman et al (1985).

2.5.1 Visual Analogue Scales (VAS)

One of the most widely used non-verbal measurement techniques is the visual analogue scale (Duncan et al, 1989). Patients describe their pain level by placing a mark on a 10 cm line at the appropriate distance between the end points (no pain and unbearable pain). VAS have been shown to be rapid, easy, and valid methods for measuring both clinical and experimental pain (Woodford & Merskey, 1972; Scott & Huskisson, 1976; Aun et al, 1986). Their excellent responsiveness (i.e. ability to detect small changes) has also been reported (Dao et al, 1994). Completion of a VAS scale may be difficult for some patients (Woodford and Merskey, 1972) because no references other than the words at the extremities are given, and people must imagine their pain in linear terms (Duncan et al, 1989). However, these scales give a more sensitive and accurate representation of pain intensity than the descriptive scales (Ohnhaus and Adler, 1975; Sriwatanakul et al, 1983).

Pressure algometer: Measurement of the pain-pressure threshold with a pressure algometer is commonly used to quantify trigger point sensitivity in the jaw muscles. When variables such as the rate of applied pressure, the size of algometer recording tip, and the degree of muscle contraction are controlled, then the recording technique is sensitive and reliable (Reeves et al, 1986).

Behavioural measurement: Objective measurement in the clinical context can be achieved by observing and scoring patients behaviour or expressions. Among commonly reported variables are: activity; measures of the amount of time spent standing or sitting; sleep patterns; food intake; and normal household activities such as meal preparation and gardening (Chapman et al, 1985). The disadvantages of this method are that it does

not quantify pain directly and the behaviours may change with illness for a variety of reasons, including pain or pain relief.

Category Scales: These are commonly used to assess both pain and pain relief following treatment. They involve pain description, e.g. none, slight, moderate, severe. It is difficult with these scales to specify the size of each category and whether the categories are of equal spacing. (Chapman et al, 1985).

***Sensory Decision Theory*:** Sensory decision theory (SDT), or signal detection theory, is a method of simultaneously measuring the sensory and the psychological aspect of pain (Christensen, 1988). This theory is concerned with the subject's ability to discriminate a stimulus above background "noise", i.e. it measures the accuracy of judgment of stimulus intensity and the error rate.

Category-Scaling Methods: A category scale with ratio properties may be used in the measurement of pain. This type of scale consists of a range of numbers (0-10), which are anchored by words or expression with a quantitative measuring (Borg, 1982).

2.6 Electromyography (EMG)

2.6.1 Introduction

Electromyography (EMG) is a sensitive tool for recording the muscle action potentials transmitted from the active fibres to the recording electrodes. This can be achieved through surface electrodes applied over the skin of the muscle, or by needle electrodes inserted into the muscle. It has been widely used since it was first introduced in dental research by the orthodontist Moyers in 1949.

Researchers and clinicians have used EMG to detect postural hyperactivity (Lous et al, 1970; Sheikoleslam et al, 1982), abnormal occlusal positions (Franks, 1965; Michler et al, 1988), functional hyperactivity (Moller et al, 1984), muscle spasm (Ramfjord, 1961) fatigue (Naeije & Hansson, 1986), muscle imbalance (Dohrmann & Laskin, 1978), and jaw reflexes (De Laat et al, 1985). In addition, records of EMG activity before and after treatment have been used to document changes in muscle function and have been cited

as proof that the treatment was successful (Ramfjord, 1961; Fuchs, 1975; McNamara 1976; Holmgren et al, 1985).

Portable EMG recording instruments have been introduced and widely used to detect diurnal and nocturnal phasic activity of the masseter muscle as a measure of bruxism (Solberg et al, 1975; Kardachi & Clarke, 1977; Rugh & Johnson, 1981).

2.6.2 The electrodes

The electrical activity produced by muscle action potentials (i.e. ionic movement within the muscle) is detected by electrodes. These electrodes may be unipolar (mono-polar configuration), in which case the electrode is placed adjacent to the muscle with other remotely placed reference electrode, or pair electrodes placed adjacent to one another, which is called bi-polar configuration.

1- Surface electrodes:

Surface electrodes record the sum of the electrical activity generated by the action potentials in the underlying muscle. These electrodes are usually silver/silver chloride disc electrodes fixed to the skin overlying the muscle by an adhesive disc or they may be

self-adhesive. They are especially applicable to surface muscles, such as masseter, which are easily accessible.

2- Needle electrodes:

Muscle activity may detected from intramuscular electrodes recording the activity from single motor units at low activity level. These electrodes are fine, silver electrodes of approximately 0.04 mm diameter, inserted in the muscle percutanously. With increasing strength of contraction, the EMG comprises summated action potentials from numerous motor units irrespective of the type of electrode (Bakke, 1993). The use of fine wire electrodes or indwelling needle electrodes has provided more precise information about the diagastric, and medial and lateral pterygoid muscles (e.g. Molin, 1973; Wood et al, 1986; Widmalm et al, 1987; McMillan and Hannam, 1989).

3- Ground electrode

In order to reduce unwanted signals or noise, it is necessary to ground or earth subjects when recording EMG. These electrodes may be placed on the ear lobe, the back of the neck or the forehead.

4- Electrode placement

Variations in inter-electrode distance make a large difference to the amount of electrical activity detected because of variations in resistance of the sub-cutanous tissues and position relative to active muscle fibres (Kramer et al, 1972). The surface electrodes are best placed approximately 2 cm apart, from the center of each electrode, and in the line with main direction of the muscle fibres (Yemm, 1977).

2.6.3 The reproducibility of the EMG

Cecere et al (1996) studied the effect of position of electrodes over the muscle belly on the reproducibility of the EMG. They found that there was no significant influence of electrode repositioning upon reproducibility, which supports the findings of Pancherz and Winnberg (1981) and Visser et al (1992). On the other hand, it has been suggested that the considerable variation that may occur between readings on different days is due to problems of electrode relocation (Frame et al, 1973).

The electrical activity of the muscle is very small, 400 microvolts over the masseter muscle at full effort (Moller, 1969). This electrical activity must therefore be amplified. In order to measure the quantity of surface EMG activity, an integrator must be used

which converts all the negative wave forms to positive and gives a continuous record of the on-going activity (Michler et al, 1988).

It has been reported that the degree of noise present in the surface EMG has an effect on analysis of the data (Buxbaum et al, 1996). It was found over a number of studies, that a software computer program which substracted the noise from actual EMG data, produced essentially noiseless EMG (Glas & Steenberghe, 1981).

End of the review of literatures

CHAPTER THREE

GENERAL METHODS AND MATERIALS

3.1 Ethical Committee Approval

Approval was obtained from the Area Dental Ethics Committee of the Greater Glasgow Health Board prior to the experiments.

3.2 Types and position of electrodes

EMG recordings were made from the masseter muscle using a pair of self-adhesive, surface electrodes (Red Dot 3M, St Paul, USA). The skin was prepared by rubbing with gauze soaked in alcohol. The electrode centres were approximately 20 mm apart, oriented along the direction of the muscle fibres. A common electrode was applied to the forehead (Figure 1).

3.3 Amplifier specifications

The signals were amplified x 5000 by an isolated amplifier and stored on video tape cassettes by means of a PCM-8 A/D video recorder adapter (Medical Systems Corp., Greenvale, NY 11548, U.S.A.) (Figure 2). The sampling frequency of the adapter was 22 kHz and the frequency response was DC-7 kHz.

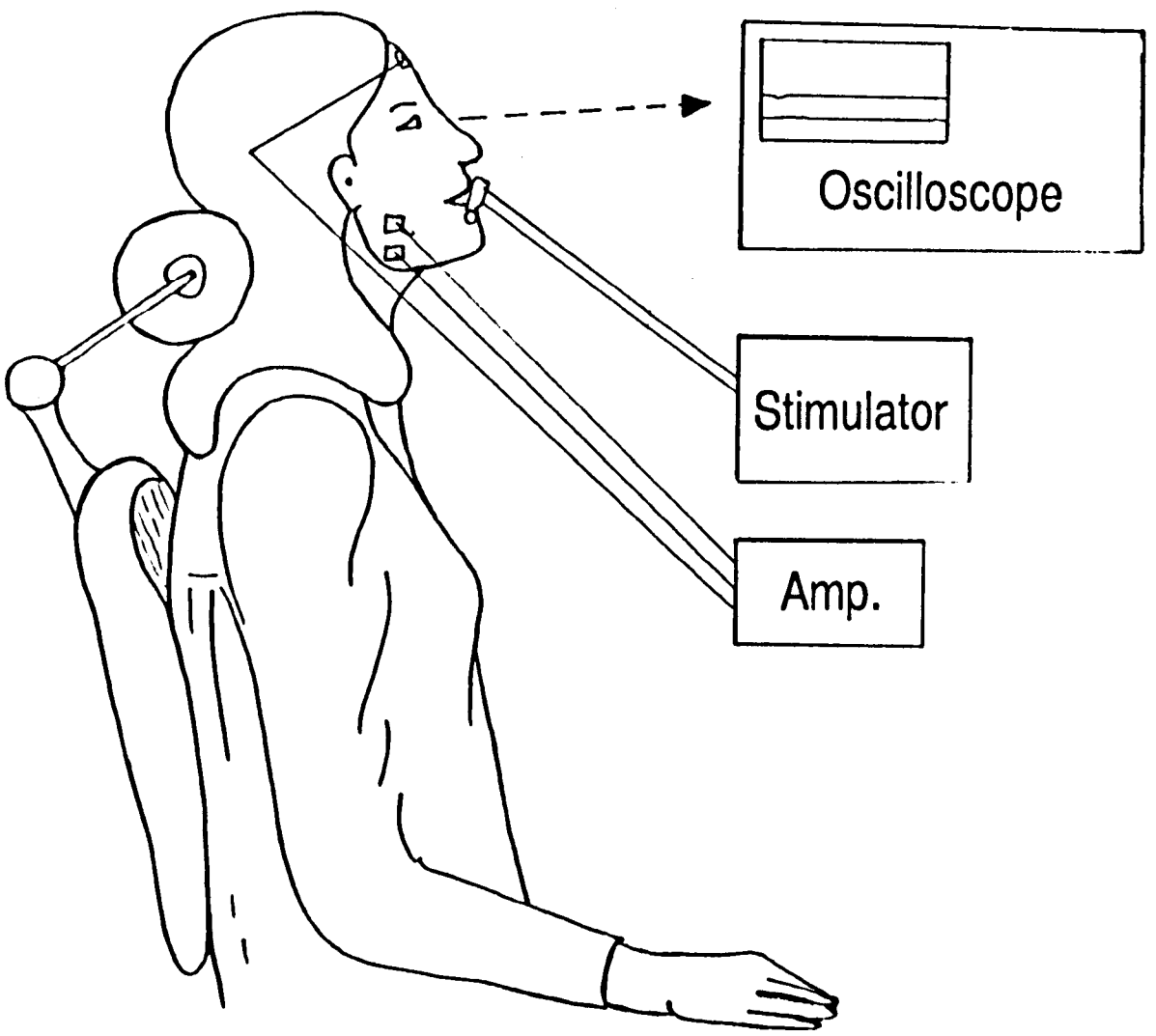


Figure (1) The experimental set-up. EMG recording were made from the masseter muscle while electrical stimuli were applied to the ipsilateral lower lip. During periods of stimulation, subjects were maintaining a 10% of masseter muscle activity with the aid of visual feedback.

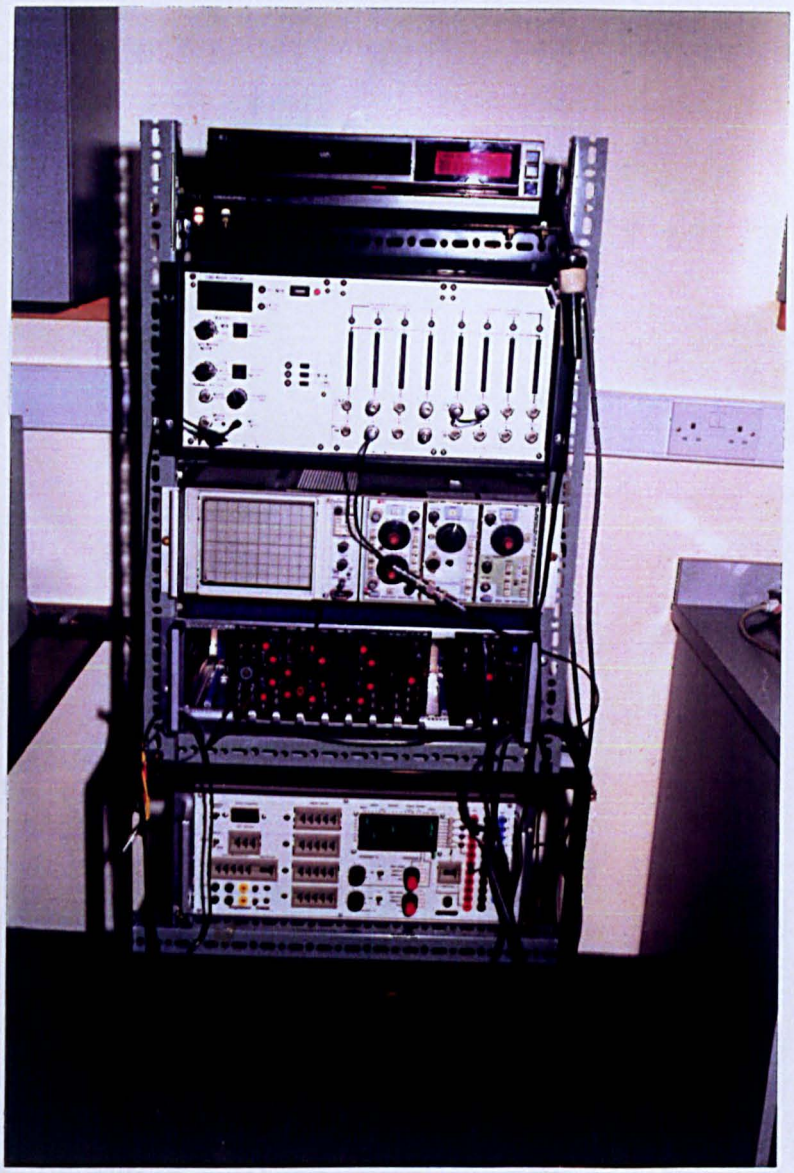


Figure (2) The EMG equipment used in the experiment.

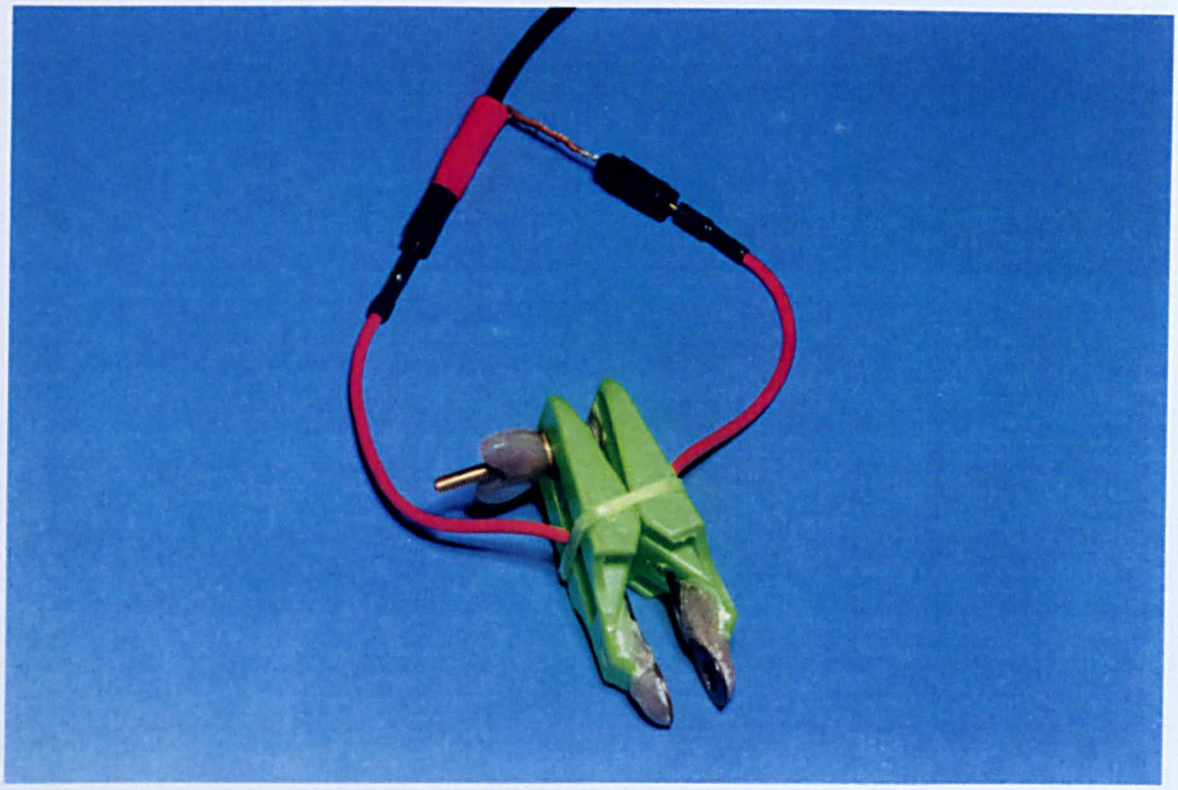
3.4 Stimulation

A special bipolar electrode was fabricated to allow electrical stimuli to be applied across the lower lip (Figure 3). Both poles were silver/silver chloride 9 mm EMG disc electrodes (SLE Ltd, Croyden, UK). These electrodes were attached to a spring holder which clipped the lower lip with light pressure. The skin of the lower lip was prepared by rubbing with gauze soaked in alcohol then applying conductive gel. The other electrode was positioned on the oral mucosa so that the stimulus current passed directly through the lip. The electrodes were always placed on the lower lip ipsilaterally to the masseter from which recordings were made.

The stimuli were applied by means of a high-voltage constant-current stimulator with an isolated output stage (Model DS7, Welwyn Garden City, Hertfordshire, England) (Figure 4). The stimuli consisted of single waves of 1 ms duration.

3.5 The experimental protocol

At the beginning of experiments, each subject was asked to produce a brief maximum clench, with visual feedback being supplied from the masseter EMG. For this purpose, the EMG was rectified and integrated with a time constant of 200 ms and displayed on an oscilloscope. After appropriate alterations to the amplification of the feedback signal, second, earthed, beam was set on the scope at a level equivalent to 10 % of the



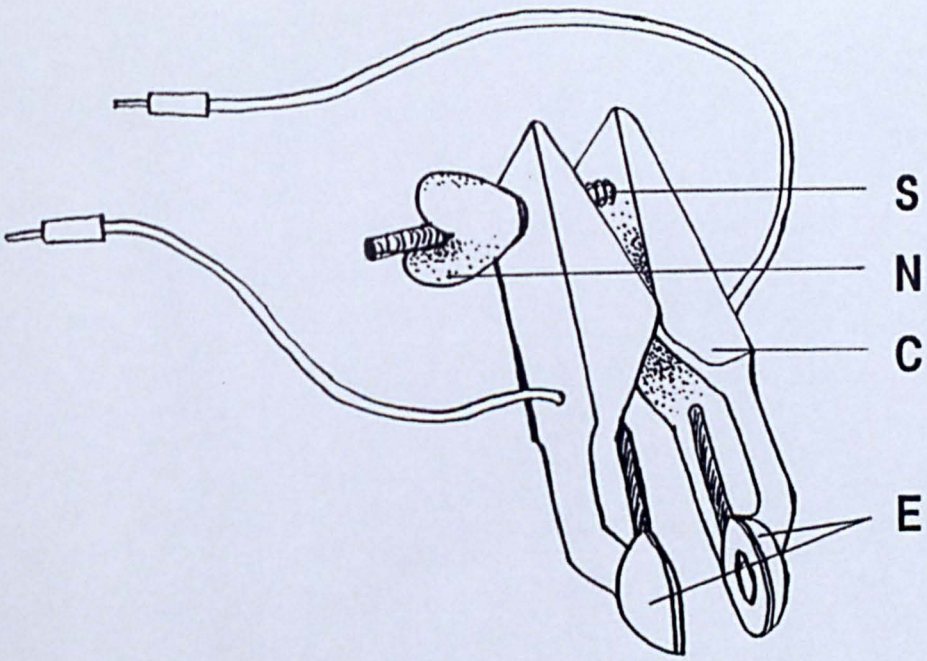


Figure (3) The stimulating device fabricated from two EMG surface disc electrodes. Abbreviations: S, spring; N: wing nut; E: silver/silver chloride electrodes.

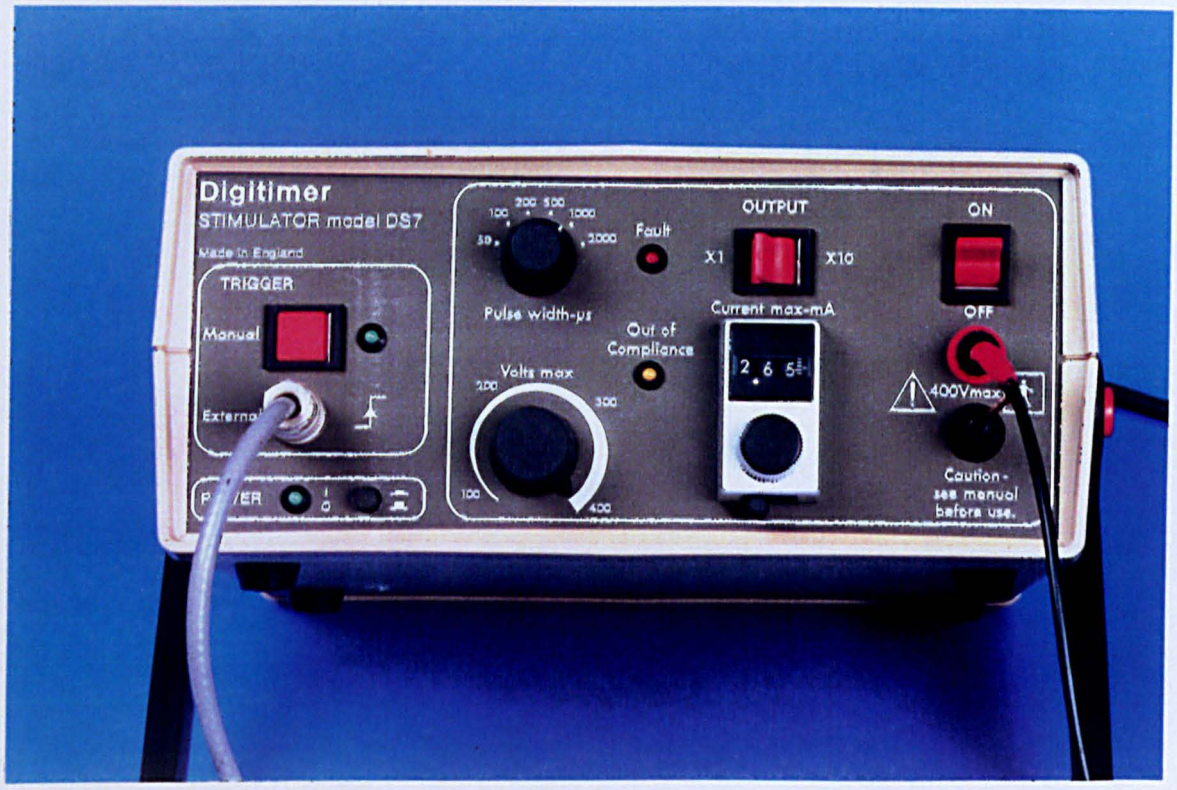


Figure (4) High voltage constant-current stimulator used in the experiment.

maximum EMG. The subjects were subsequently asked to clench to this level while stimuli were applied. This level of masseter muscle activity was adequate to allow inhibitory and excitatory reflexes to be observed yet was sufficiently low to avoid muscle fatigue.

Before applying any stimuli, eight sweeps were recorded with the subjects relaxed and eight sweeps with them biting to 10 % of maximum activity (one sweep every four seconds).

The perception threshold (T) for stimulation of the lip was determined by gradually increasing the stimulus intensity until the subject first perceived a sensation. Stimulus intensities of 1T, 1.5T, 2T, 2.5T, 3T, 5T, 6T, 7T, 8T, and 9T were subsequently applied in order to evoke the reflex responses. First the cathode was placed intra-orally, on the oral mucosa, and the anode on the skin of the lip. Then the sequence was repeated with this polarity reversed without removing the clip electrode. The triggering of the stimulator and of the recordings was achieved by a digital programmer (Digitimer D4030, Welwyn Garden City, Hertfordshire, England). For each intensity and polarity, eight stimuli were applied at four-second intervals.

3.6 Processing and analysis of EMG

The EMG signals were processed from the recordings stored on magnetic tape. The EMG was full-wave rectified and then digitised (sampling once every 500 μs) and averaged using a microcomputer (BBC with Unilab interface). The averaged records were smoothed using a 4 ms sliding window. The eight sweeps obtained with the subjects relaxed were also rectified, averaged and smoothed, and the mean level of these was considered to be the noise of the recording system. This noise level was subsequently subtracted from the other rectified and averaged EMGs .

A custom-written programme was used to calculate the mean level of activity in the 86.5 ms prior to the stimulus and then to measure the latency and magnitude fluctuations from this level in the following 165.5 ms. The terms "positive waves" and "negative waves" were used to denote such fluctuations above and below this mean pre-stimulus level, respectively. The magnitudes of these waves were expressed as integrals of time and activity (the latter being expressed as a percentage of the pre-stimulus level after the subtraction of noise).

A further 8 sweeps at 10 % of maximum (with no stimulus) were recorded in order to determine the largest fluctuations which occurred by chance. The procedure was to set the pre-sample delay of the 10% record at 0.040 ms and analyse the post-stimulus period. Then the same record was re-analysed after moving the pre-sample delay forward by 155 ms (i.e. 155.040 ms, 310.040 ms, 465.040 ms etc.). This was repeated 20 times so that 20 of the largest fluctuations were obtained. In line with a study by (Louca et al, 1996), the positive and negative waves were considered to be significant excitations and inhibitions respectively only when their integrals exceeded the second largest corresponding values (i.e. the 95th percentile). These recordings were also smoothed averages of eight full-wave rectified sweeps. Again in line with Louca et al. (1996), negative waves were deemed to be short- or long-latency inhibitions depending on whether their latencies were less or greater than 30 ms respectively. A division between short- and long-latency excitations was made at 55 ms. Figure (5) shows the analysis of the signals from one subject at a given stimulus intensity.

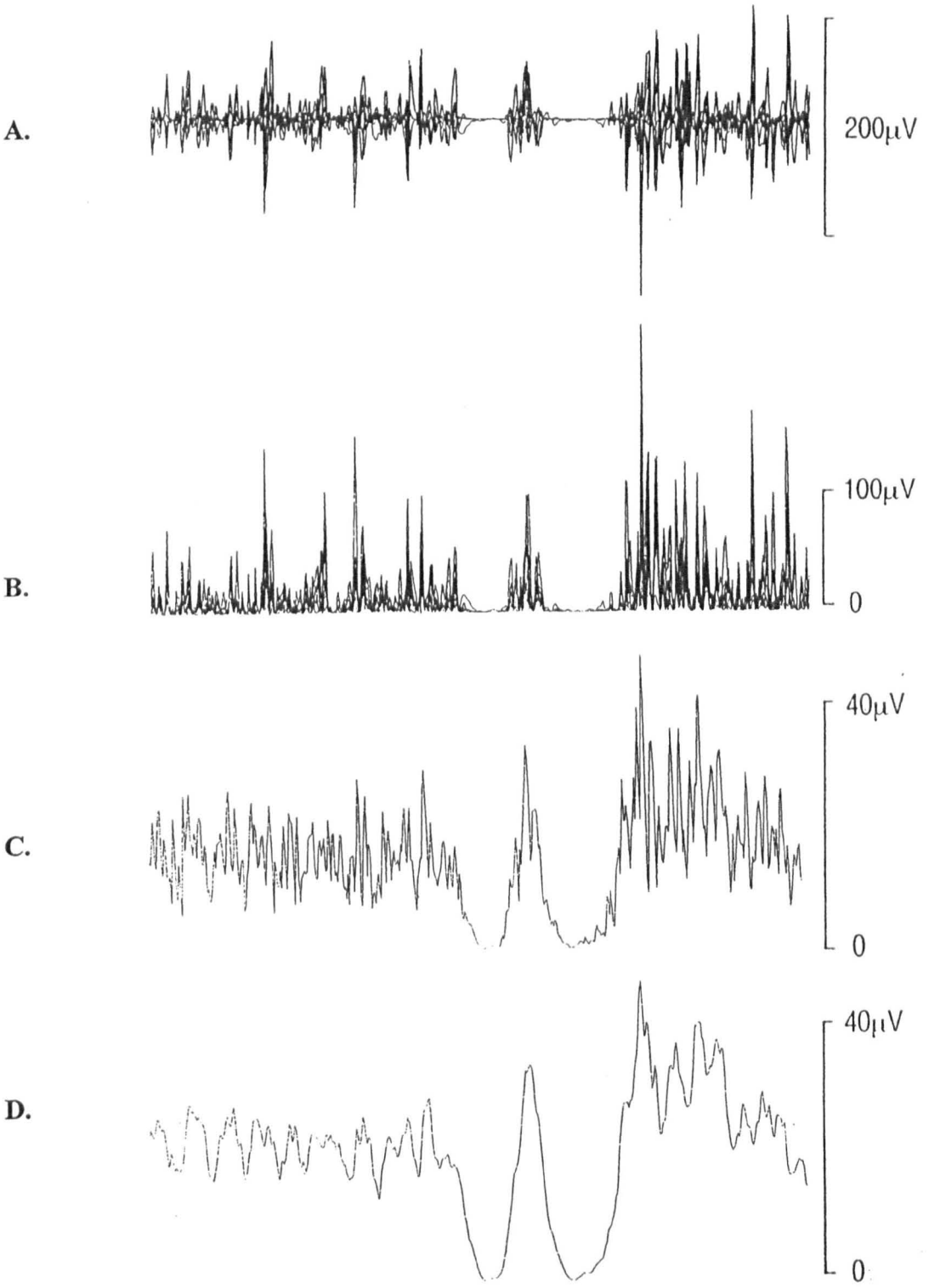


Figure (5) The sequence of signal analysis. A. 8 superimposed sweeps of the raw signal; B. rectified signal; C. averaged rectified signal; D. smoothed averaged rectified signal from which all measurements were taken.

3.7 Statistical analysis

In this study, like many other medical studies, the number of observations is small and there is doubt about the consistency of variance and the existence of normally distributed data. Therefore, non-parametric tests (distribution-free tests), which are considered more powerful than parametric tests in such studies, were used. These tests applied to the distribution of values, not to the averages, and are based on ranking the data.

The following tests have been used in this study:

Friedman test:

This is a non-parametric Analysis of Variance and was used to find out whether significant differences existed between the 4 responses in each configuration (e.g. cathode inside or cathode outside).

Wilcoxon Signed Ranks test:

This is a non-parametric (paired) test for multicomparisons. It is a non-parametric alternative to the paired *t* test. It was used to test for differences in the responses in each category (e.g. 1st inhibition vs 2nd inhibition with cathode inside). It was also used to test for the absolute comparison of the responses (i.e. to compare 1st inhibition before and after treatment).

Bonferroni correction:

If the number of comparisons which were taken can be counted, then Bonferroni correction suggests that the P-value for each comparison should be multiplied by the number of comparisons undertaken to ensure the significance of the results. This test was used as a follow up test after the multi-comparison Wilcoxon Signed Rank test.

Wilcoxon Rank Sum test:

This is a non-parametric (un-paired) test, often called Mann-Whitney test. This test is based on examining the sum of the ranks in each group and is an alternative to the two sample *t* test. It was used to compare the latencies and durations of the responses and compare the magnitude of the 1st and 2nd inhibitions and the 1st and 2nd excitations.

Fisher's Exact Test:

This is a test for the comparison of proportions in two independent samples. This test was used to compare the ratios of occurrence of significant responses in different configurations (e.g. compare the ratio of occurrence of 1st inhibition before and after treatment).

The level of significance was taken as $P<0.05$ in all cases.

CHAPTER FOUR

MASSETER MUSCLE REFLEXES IN HEALTHY SUBJECTS

Summary

The aim of this study was to investigate whether reported differences in the patterns of jaw reflexes which can be evoked by electrical stimulation of the lip might be related to the intensity or polarity of the stimuli. EMG recordings were made from a masseter muscle in 14 subjects and constant-current stimuli were applied through bipolar electrodes clipped across the lower lip. Stimuli at multiples of perception threshold were applied while the subjects sustained a level of masseter activity equivalent to 10 % of their maximum. The EMGs were analysed following rectification, averaging and smoothing.

A sequence of inhibitory, excitatory, inhibitory and excitatory responses could be produced in the muscle by both polarities of stimuli. The latencies of these four responses were generally in the ranges 10-20 ms, 25-40 ms, 40-55 ms and 80-100 ms respectively. These latencies, particularly for the last two responses, tended to decrease at higher intensities of stimulation. The threshold for the long-latency inhibition was significantly lower than that for the short-latency inhibition when the cathode was outside the mouth but not when it was inside the mouth. In addition, it was found that the long-latency excitation had the lowest threshold of the four responses regardless of stimulus polarity. Since nerves are excited particularly around a cathode, we conclude that stimulation of nerves supplying the skin outside the mouth evokes predominately long-latency jaw reflexes whereas shorter latency responses can be evoked by stimulating nerves supplying oral mucosa. Furthermore, long latency excitatory reflexes seem to be the most easily evoked by stimulation of the lip.

4.1 Introduction

It is well established that stimulation within the mouth can produce a sequence of inhibitory, excitatory, inhibitory and excitatory responses in the masseter muscle. These reflex responses, called the post-stimulus electromyographic complex (PSEC) by Glas et al (1984), appear as a series of downward and upward-going waves in full-wave rectified and averaged EMGs. While there is general agreement on the multiphasic nature of these responses to intra-oral stimuli (e.g. Bratzlavsky, 1972; Yemm, 1972a; Yu et al, 1973; Godaux and Desmedt, 1975; Glas et al, 1985; Carels and Steenberghe, 1986; Widmer, 1987; Cadden and Newton, 1988), there is some dispute as to the nature of responses to stimulation of peri-oral structures. Some studies have reported that a multiphasic response similar to that produced by intra-oral stimuli can be evoked by stimulation of the lips (e.g. Bratzlavsky, 1972; Godaux and Desmedt, 1975; Desmedt and Godaux, 1976; Di Francesco et al, 1986), while others have reported a simpler response consisting of a single inhibitory period and a single excitatory period which seem to be equivalent to the last two elements of the responses evoked by intra-oral stimuli (Yu et al, 1973; Cadden and Newton, 1988, 1994).

One explanation for this apparent discrepancy is that depending on the precise stimulation regime, some intra-oral nerves may have been excited by the peri-oral stimuli thus producing the typical intra-orally evoked responses (Cadden and Newton, 1988). If a sufficiently high stimulus intensity is used, the same response will be evoked whether the stimulus is applied intra- or extra-orally. In order to investigate this possibility, we have now studied the reflexes evoked in the masseter muscle by electrical stimulation across the lip using a range of stimulus intensities and also both

polarities of electrode. As nerves are stimulated predominantly close to a cathode, changing polarity will allow the preferential excitation of intra- or peri-oral nerves (with the cathode inside or outside the mouth respectively).

4.2 Materials and Methods

Subjects

There were 14 volunteers aged 20-43 years (mean 29), ten males and four females, included in the study. All the subjects had sound natural dentitions, no history of TMD or bruxism and gave informed consent. The subjects were relaxed and seated upright in a dental chair throughout the experiments (Figure 6).

The experimental set up and data analysis were described earlier in Chapter 3.

4.3 Results

All 14 subjects showed masseter muscle reflexes (inhibitions and excitations) in response to the electrical stimuli. However, the patterns of these reflexes were different depending on the stimulus intensity and polarity of stimulation.

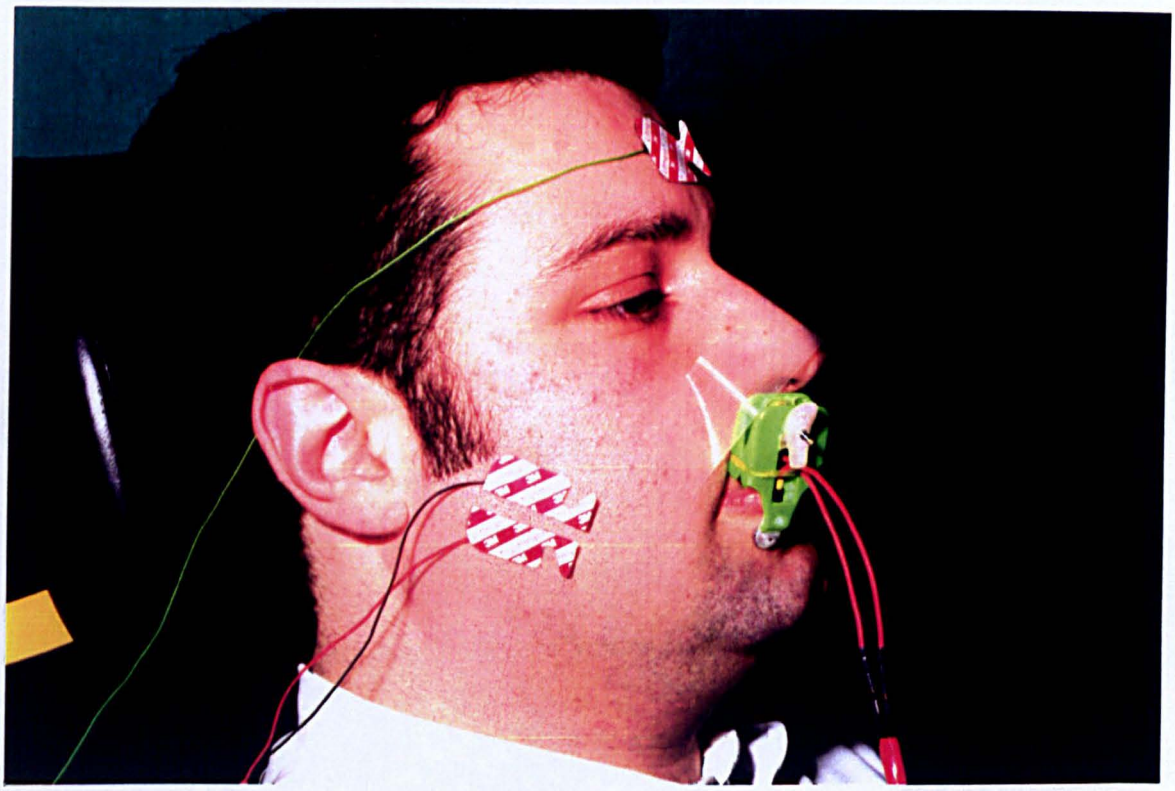


Figure (6) One of the subjects with the electrodes in place. The clip electrode was clipped over the ipsilateral lower lip.

Thresholds of responses

The reflex thresholds for the four responses (1st inhibition, 1st excitation, 2nd inhibition, 2nd excitation) for both polarities of stimulation are presented in Table 1. The significant responses for cathode inside and cathode outside the lip were summarised in Figures 7 and 8 respectively.

When the cathode was inside the mouth, there were significant differences between the thresholds of the different responses (Friedman test, $P = 0.04$). However multiple comparisons (Wilcoxon Signed Ranks Tests with a Bonferroni correction of six) revealed that the only individual significant ($P < 0.05$) difference was that the 2nd excitation had a lower threshold than the 1st inhibition. In addition there was a trend, albeit not statistically significant, for the threshold of the 2nd excitation to be lower than those of the 1st excitation and 2nd inhibition.

By contrast, when the cathode was outside the mouth, the differences were more marked (Friedman test, $P = 0.00001$). In this case, multiple comparisons showed that the 2nd excitation had a significantly lower threshold than any of the other three responses ($P < 0.02$). In addition, the 2nd inhibition had a significantly lower threshold than the 1st inhibition ($P = 0.006$). There were no other significant differences between the thresholds. The results of the threshold comparisons are listed in Table 2.

Subject	Cathode inside				Cathode outside			
	1st inhib.	1st excit.	2nd inhib.	2nd excit.	1st inhib.	1st excit.	2nd inhib.	2nd excit.
MM	6	*	5	5	*	1.5	6	1
MJ	9	*	3	2.5	*	*	2.5	1
HU	7	2	5	5	*	5	5	5
AB	5	5	5	5	7	6	5	5
FA	7	*	5	3	8	*	5	5
JO	7	2	5	1	*	2	5	1.5
AS	5	2	3	3	5	2.5	3	2
AT	5	*	3	7	5	1	6	1
MU	8	*	6	6	*	*	6	3
IS	5	3	5	3	*	*	5	3
NF	5	2	8	5	6	8	6	3
KF	9	1	5	1	*	*	5	3
LF	5	*	*	2	7	*	5	2
RF	5	5	7	3	8	7	6	2
Median	5.5	5	5	3	9	7.5	5	2.5

Table (1) Thresholds of significant responses for all volunteers.

(*) denotes a non-significant response. The non-significant responses were given a value of 10 during statistical analysis.

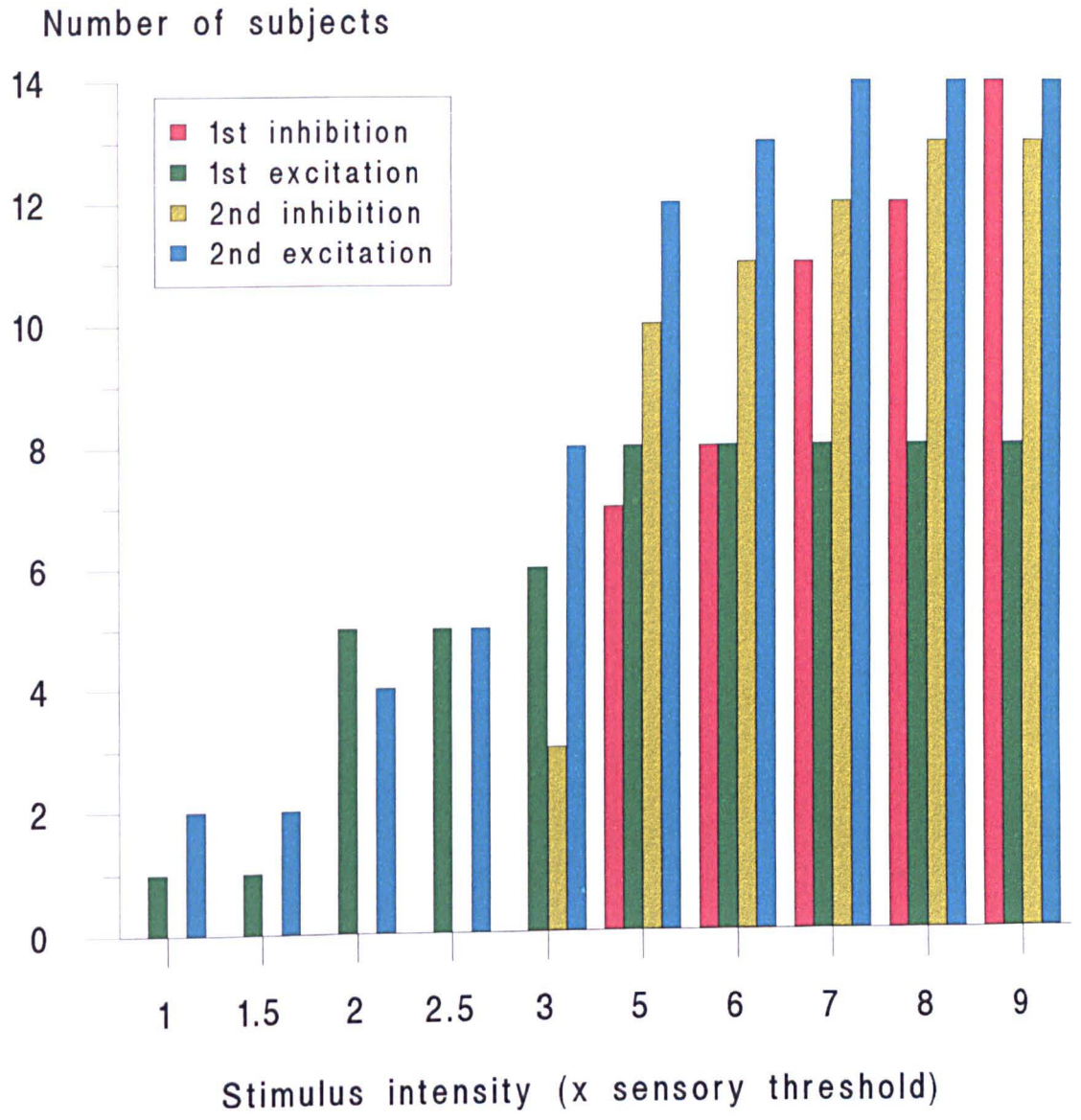


Figure (7) Cumulative frequency bar graphs showing the number of subjects who gave a significant inhibitory or excitatory response at or below each stimulus intensity with the cathode **inside** the mouth.

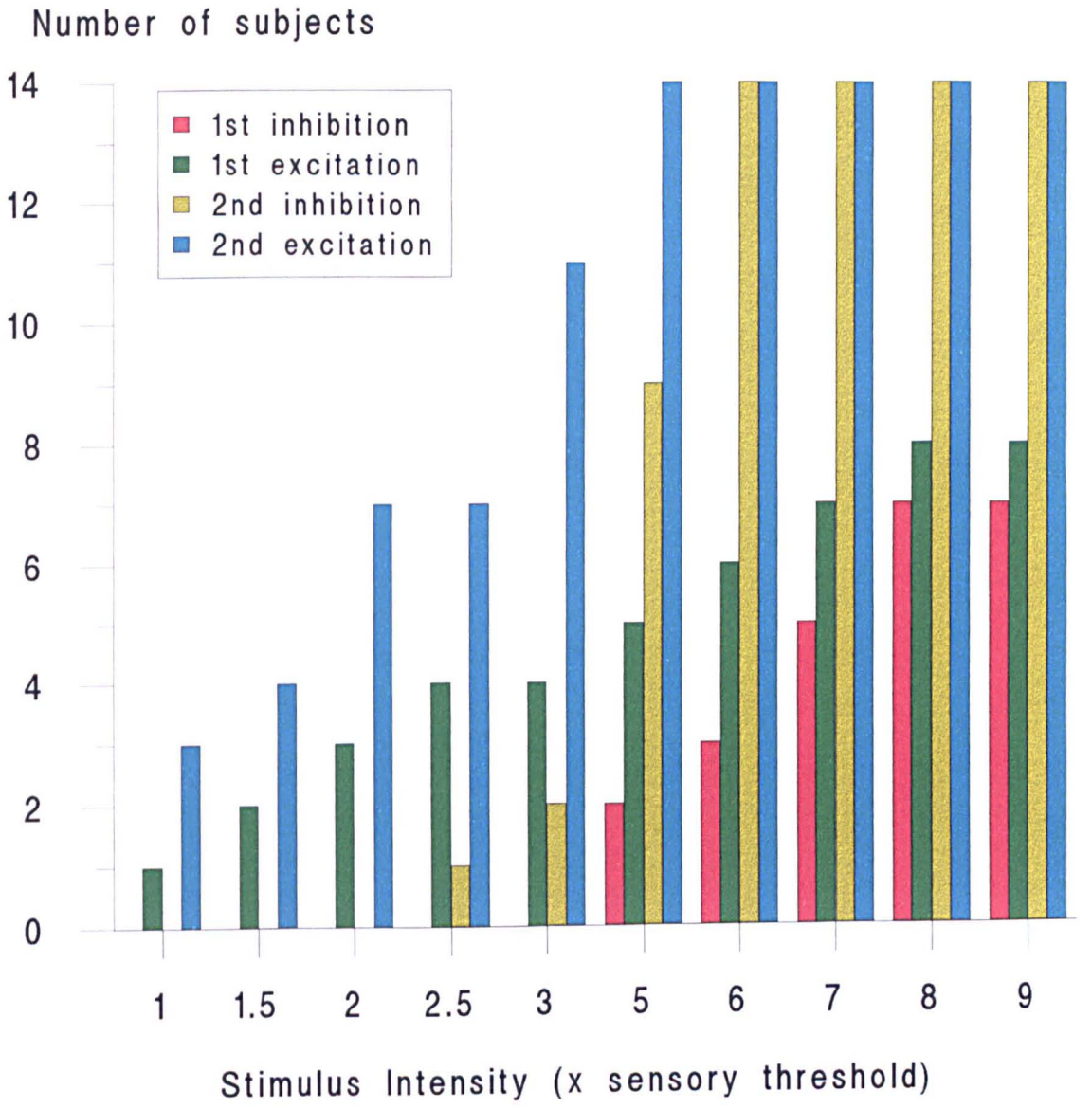


Figure (8) Cumulative frequency bar graphs showing the number of subjects who gave a significant inhibitory or excitatory response at or below each stimulus intensity with the cathode **outside** the mouth.

	Cathode inside P value	Cathode outside P value
1st inhib. Vs 1st excit.	0.724	0.139
1st inhib Vs 2nd inhib	0.722	0.001
1st inhib. Vs 2nd excit	0.006	0.001
1st excit. Vs 2nd inhib	0.556	0.115
1st excit. Vs 2nd excit	0.045	0.002
2nd inhib Vs 2nd excit	0.050	0.003

Table (2) A comparison of threshold values for the four separate responses produced by each stimulus polarity. Results are not Bonferroni corrected.

Latencies and durations of inhibitory responses

In all 14 subjects, a short-latency inhibitory response occurred with the cathode inside the mouth. However such a response was less common with the cathode outside, occurring in only seven of the 14 subjects. By contrast, long-latency inhibitions occurred in 13 subjects with the cathode inside and in 14 with the cathode outside. Figures 9 and 10 show the difference in the pattern of the inhibitory responses between the different stimulus polarities.

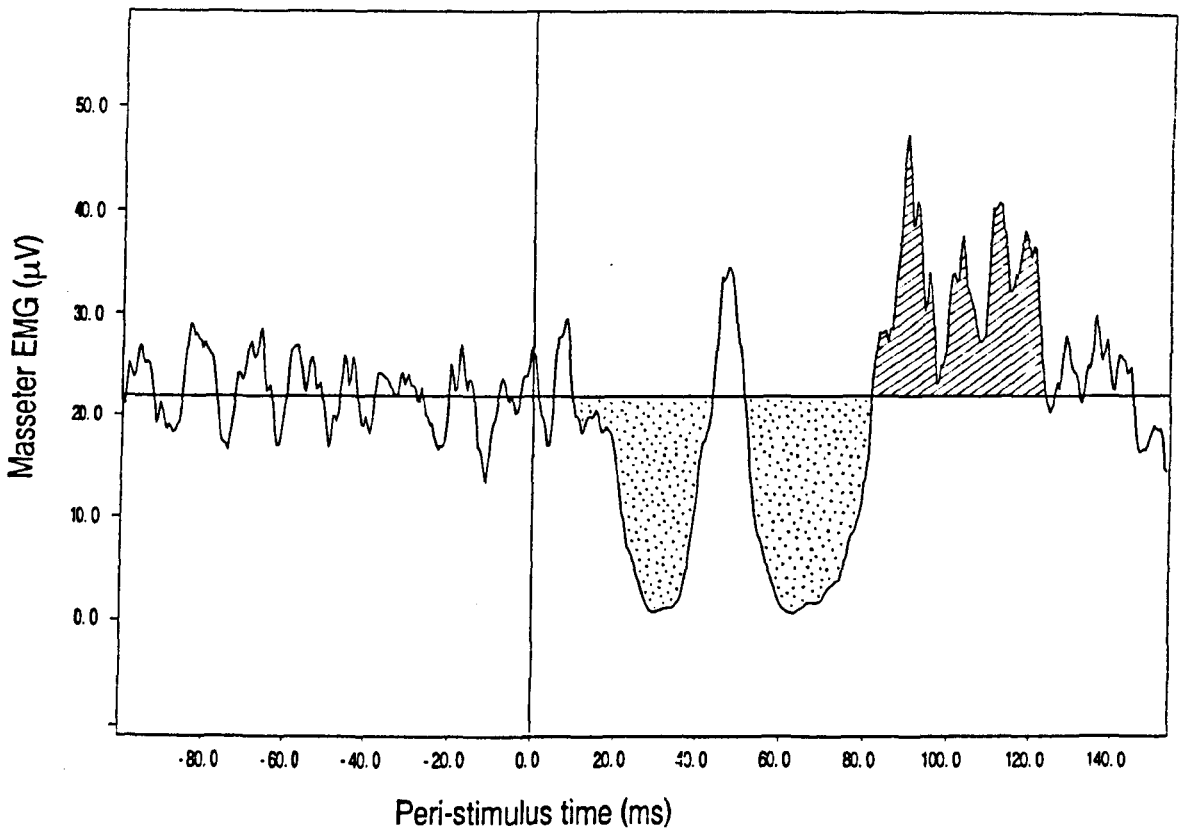


Figure (9) Averaged EMG showing the reflexes in an active masseter muscle in response to a 7T stimulus applied to the lower lip with the **cathode inside** the lip. The horizontal line shows the mean level of activity in the pre-stimulus period, the stippled areas shows the inhibitory reflexes and the lined areas show the excitations. The vertical line indicates the timing of the stimulus. Note the bi-phasic inhibitory period.

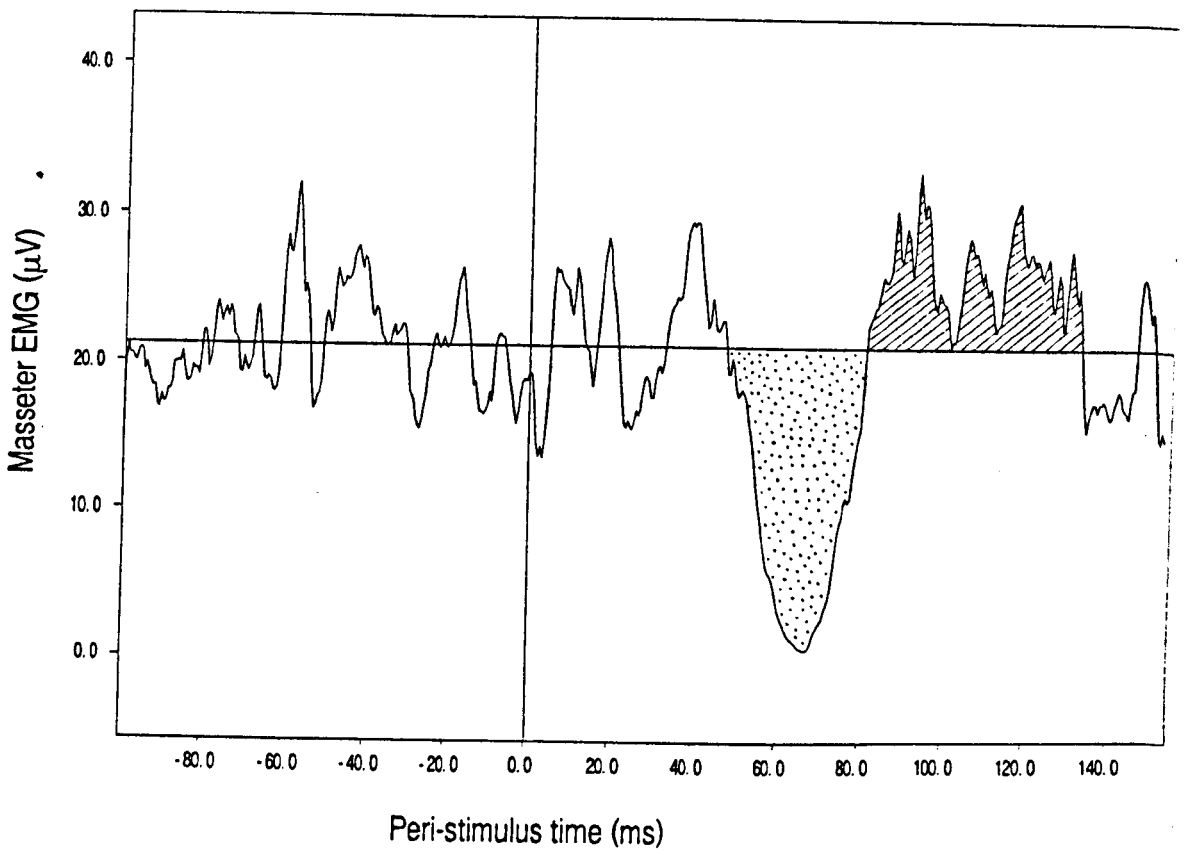


Figure (10) Averaged EMG showing the reflexes in an active masseter muscle in response to a 7T stimulus applied to the lower lip with the **cathode outside** the lip in the same subject of Figure (9). The horizontal line shows the mean level of activity in the pre-stimulus period, the stippled areas shows the inhibitory reflexes and the lined areas show the excitations. The vertical line indicates the timing of the stimulus. Note the difference in the pattern of the responses, with two inhibitory waves occuring with the cathode inside but just one when the cathode outside.

The latencies and durations of the inhibitory responses for both polarities are summarised in Tables 3 and 4. Note that with more intense stimuli, the latencies decreased and the durations increased, particularly for the longer latency responses (cf. the minimum latencies and maximum duration with those obtained with threshold stimuli).

Magnitude of inhibitory responses:

Pooled data of the inhibitory waves showed that the magnitude of short-latency inhibitory periods at 9T was significantly greater than the mean control level with cathode inside. While the long-latency inhibitory period was significantly greater than the mean control level at 5T, 6T, 7T, 8T and 9T (Figure 11).

With the cathode outside, the magnitude of the short-latency inhibitory period at any stimulus intensity was not significantly greater than the mean control level. However, the magnitude of the long-latency inhibitory periods was greater than the mean control, as with cathode inside, at 5T, 6T, 7T, 8T and 9T (Figure 12).

Responses	N	Latency				Duration			
		Threshold		Minimum		Threshold		Maximum	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
1st inhibition	14	15.9	1.3	14.1	1.0	21.8	1.1	24.3	1.6
1st excitation	8	36.0	1.6	35.9	1.6	25.1	2.6	25.7	2.2
2nd inhibition	13	50.3	2.4	44.6	1.6	37.1	3.4	47.2	4.4
2nd excitation	14	94.8	4.3	86.3	4.3	32.5	3.5	48.2	4.6

Table 3 Latencies and durations (ms) of significant responses obtained with the cathode inside the lip. "N" represents the number of subjects showing a particular response at any stimulus intensity. Data given in columns labelled 'Threshold' represent responses obtained with the lowest intensity of stimulation which evoked that response in each subject. In addition, data are given for the minimum latency and maximum duration obtained at any stimulus intensity, but excluding the durations of fused inhibitory responses.

		Latency				Duration			
Responses	N	Threshold		Minimum		Threshold		Maximum	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
1st inhibition	7	16.3	0.9	13.5	1.4	18.8	1.1	23.7	1.6
1st excitation	8	29.8	1.9	29.6	1.9	17.7	1.1	17.7	1.1
2nd inhibition	14	49.7	1.9	42.3	1.5	38.9	3.8	51.6	4.3
2nd excitation	14	96.5	3.1	83.3	2.5	32.9	3.0	53.9	3.7

Table (4) Latencies and durations (ms) of significant responses obtained with the cathode outside the lip. "N" represents the number of subjects showing a particular response at any stimulus intensity. Data given in columns labelled 'Threshold' represent responses obtained with the lowest intensity of stimulation which evoked that response in each subject. In addition, data are given for the minimum latency and maximum duration obtained at any stimulus intensity, but excluding the durations of fused inhibitory responses.

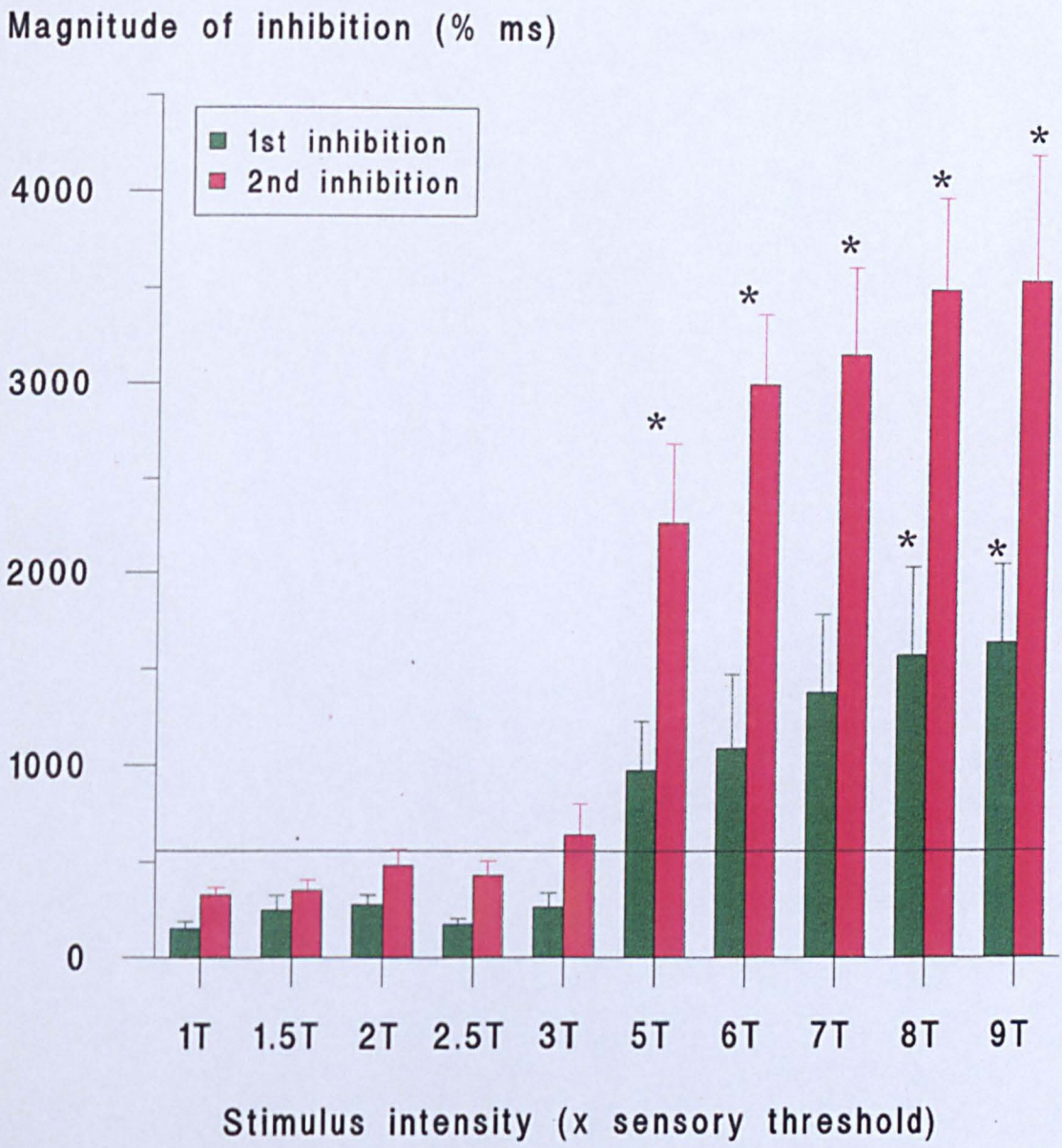


Figure (11) Pooled data showing the magnitude of the 1st and 2nd inhibitions with cathode **inside** the mouth. The solid horizontal line represents the mean size of the largest downgoing waves in the control records. Asterisks indicate values which are significantly greater than the mean control level.

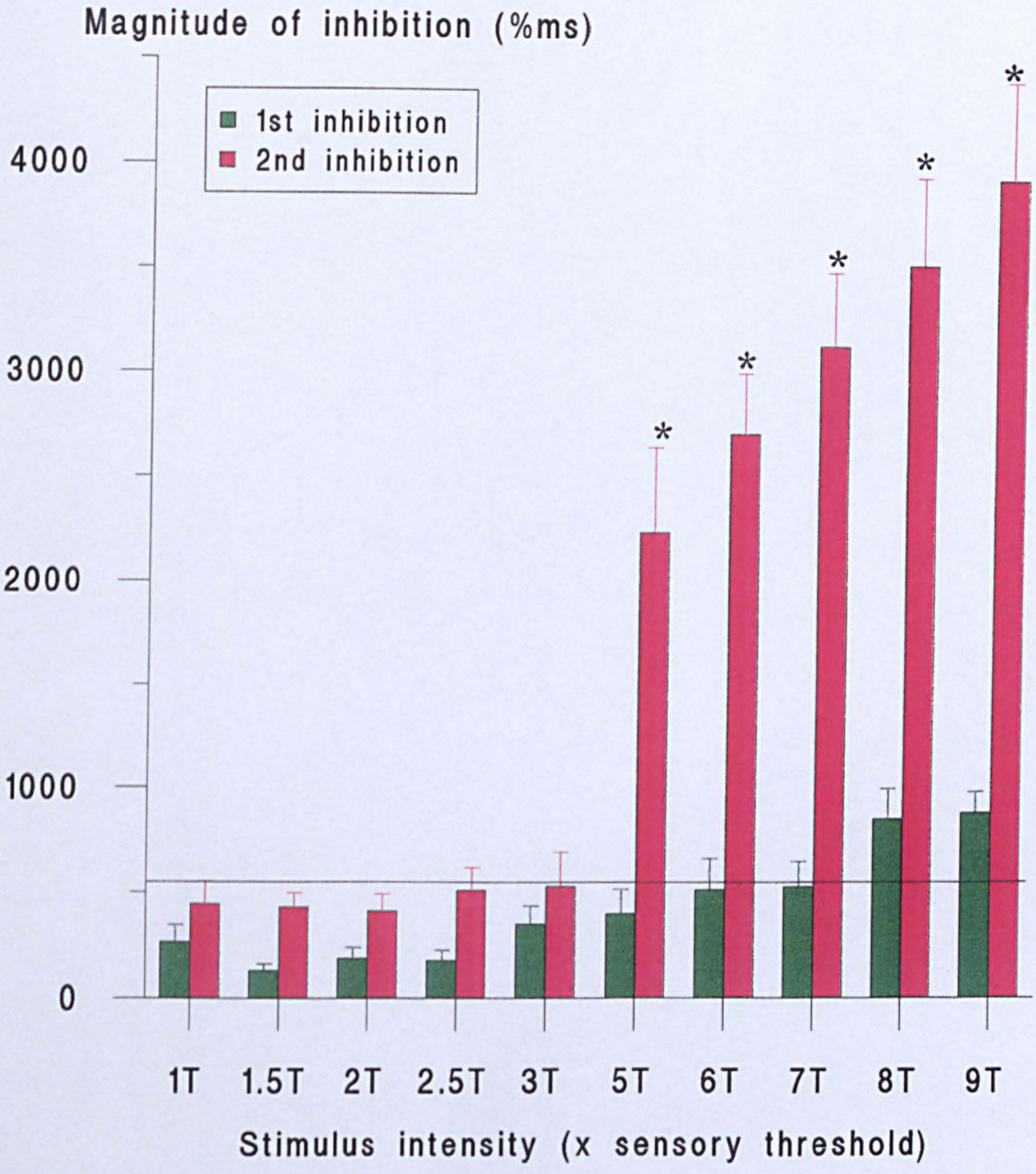


Figure (12) Pooled data showing the magnitude of the 1st and 2nd inhibitions with cathode **outside** the mouth. The solid horizontal line represents the mean size of the largest downgoing waves in the control records. Asterisks indicate values which are significantly greater than the mean control level.

Fused inhibitory responses

In four subjects, when stimulus intensities of 7T and above were employed with the cathode inside the mouth, the two inhibitory responses fused to produce a single inhibitory wave (minimum latency, 14.4 ± 3.3 ms; maximum duration, 93.4 ± 7.5 ms). When the cathode was outside the mouth, such fused inhibitory responses occurred in only two subjects (minimum latency, 18.3 ± 5.3 ms; maximum duration, 92.8 ± 3.8 ms). Interestingly, in the four subjects who showed a fused inhibition with the cathode inside, there was a biphasic inhibition at the same stimulus intensity when the stimulus polarity was reversed (Figures 13 & 14).

Excitatory responses

Excitatory responses were seen both with and without a preceding inhibition, but the latter occurred only at low stimulus intensities. The latencies and durations of these responses are summarised in Tables 3 and 4.

Excitatory responses not preceded by an inhibition

With the cathode inside, six subjects showed short-latency excitatory responses and eight showed long-latency excitatory responses which were not preceded by an

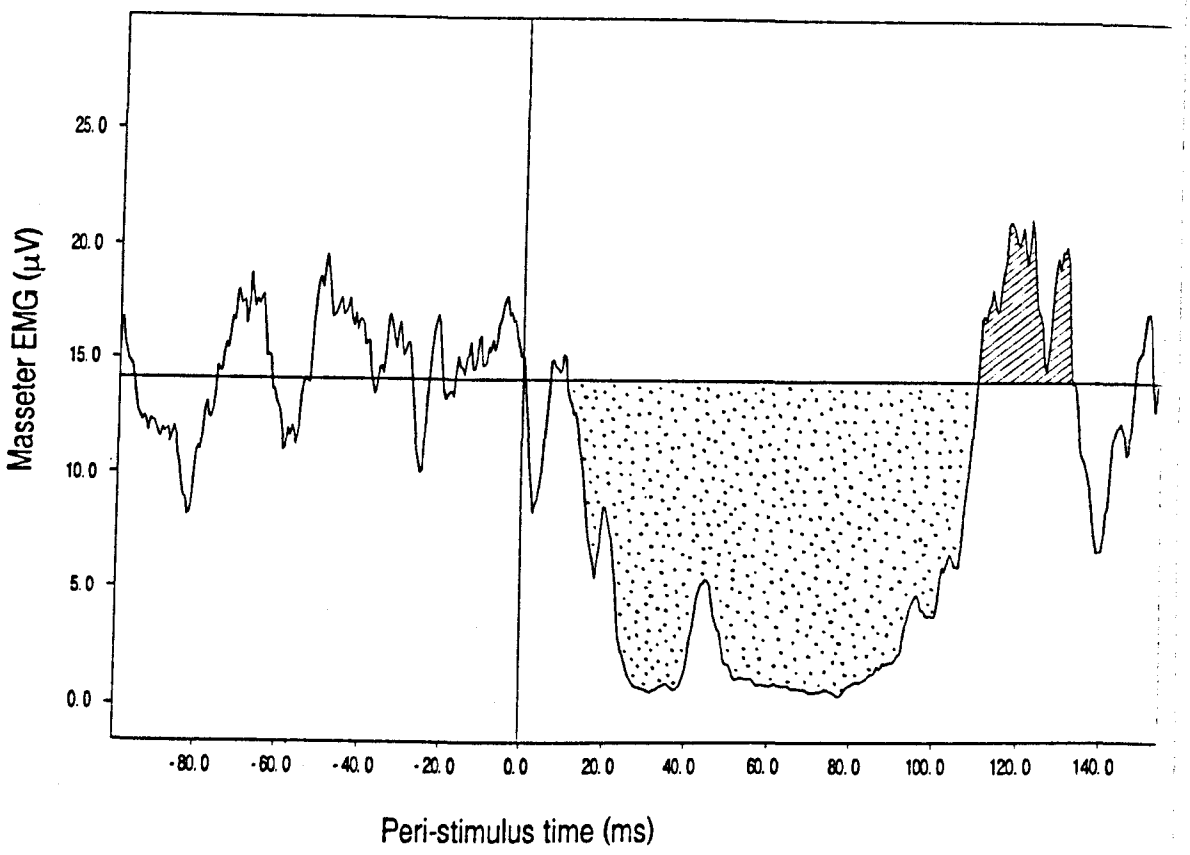


Figure (13) Averaged EMG showing inhibitory reflexes in the active masseter muscle in response to a 8T stimulus applied to the lower lip with the **cathode inside**. The horizontal line shows the mean level of activity in the pre-stimulus period, the stippled areas shows the inhibitory reflexes and the lined areas show the excitations. The vertical line indicates the timing of the stimulus. Note the fused inhibitory response.

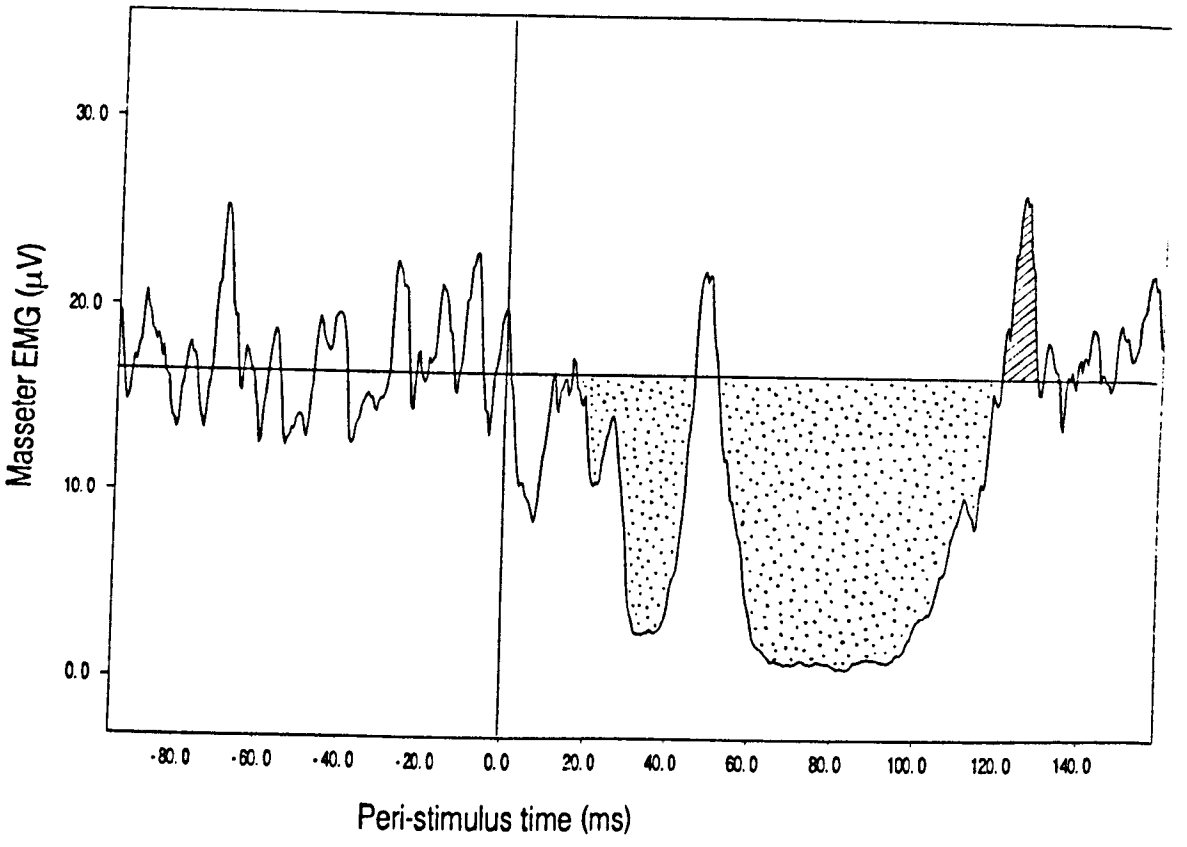


Figure (14) Averaged EMG showing inhibitory reflexes in the active masseter muscle in response to a 8T stimulus applied to the lower lip of the same subject in Figure (13) but with the **cathode outside**. The horizontal line shows the mean level of activity in the pre-stimulus period, the stippled areas shows the inhibitory reflexes and the lined areas show the excitations. The vertical line indicates the timing of the stimulus. Note the difference in the pattern of inhibition between a fused inhibitory response with cathode inside (Figure 13) and a bi-phasic inhibitory period with cathode outside as in this Figure (see text).

inhibition. The corresponding figures with the cathode outside, were seven and 11 subjects. These excitatory responses tended to occur at low stimulus intensities (1-2.5T). The latencies and durations of these responses are summarised in Table 5.

Responses	N	Latency (ms)		Duration (ms)	
		Mean	SEM	Mean	SEM
1st excit (cathode inside)	6	32.08	1.98	21.04	2.32
2nd excit (cathode inside)	8	98.91	3.27	28.90	3.06
1st excit (cathode outside)	7	26.39	2.30	19.28	2.73
2nd excit (cathode outside)	11	86.81	2.21	30.33	3.32

Table (5) Latencies and durations of the excitatory responses not preceded by an inhibition.

Excitatory responses preceded by an inhibition

With both stimulus polarities, it was found that every long-latency inhibition was followed by a long-latency excitation, however, this was not true for short-latency inhibitions and excitations. There was no significant difference between the latencies of excitations preceded by an inhibition and those not preceded by an inhibition.

Magnitude of excitatory responses

None of the short-latency excitatory waves were greater than the mean control level for both polarities. However, the magnitude of the long-latency excitatory waves was greater than the mean control level at 5T and above with the cathode inside and at 6T and above with the cathode outside the lip (Figure 15).

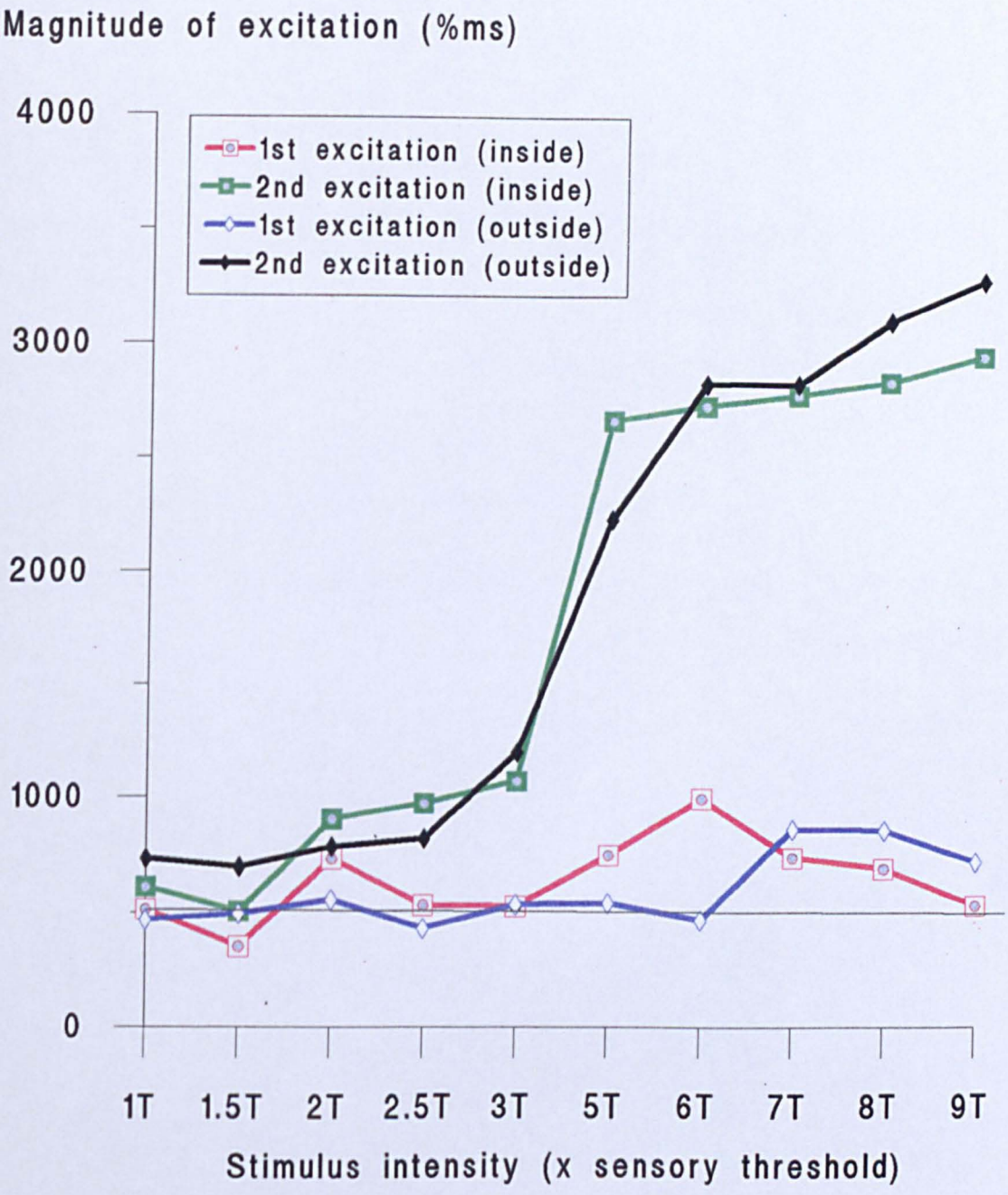


Figure (15) Pooled data showing the magnitude of the 1st and 2nd excitations for both polarities (see text). The horizontal line represents the mean size of the largest upgoing waves in the control records.

4.4 Discussion

Electrical stimulation across the lip, with both stimulus polarities, elicited inhibitory and excitatory responses in the masseter muscle. This is in agreement with many previous studies which have demonstrated that electrical stimulation in or around the mouth can produce such reflexes (e.g. Yemm, 1972 a; Bratzlavsky, 1972; Yu et al, 1973; Godaux and Desmedt, 1975; Desmedt and Godaux, 1976; Di Francesco et al, 1986; Cadden and Newton, 1988; Brodin and Türker, 1994). The latencies of the short-latency and long-latency inhibitions were similar to those observed by others (e.g. Turker & Miles, 1985; Cadden and Newton, 1988). As the stimulus intensity increased, there was a tendency for one phase of inhibition to merge with another, forming one long fused response; this has also been found by others (e.g. Godaux and Desmedt, 1975; Cadden and Newton, 1988; Brodin and Türker, 1994).

There were two principal new findings in the present study. First it was found that if the stimulus polarity was reversed, there was a consistent difference between the patterns of reflex thresholds. Second, it was found that with both stimulation polarities, the threshold of the 2nd excitation tended to be lower than those for the other three responses.

The difference in the pattern of reflex thresholds with stimulation polarity was such that the early inhibitory response had a similar threshold to that of the late inhibition when the cathode was to the inside but a significantly higher threshold when the cathode was to the outside. Since nerves are excited preferentially around the cathode, this finding

supports the previous studies of Yu et al (1973) and Cadden and Newton (1988). These authors reported that, with moderate stimulation intensities, the first inhibition can be produced only by stimulation inside the mouth but that the second inhibition can be produced by intra- or peri-oral stimulation. The fact that higher intensities of stimulation with the cathode outside the mouth did produce the earlier inhibition is most likely explained in terms of the stimulus spreading from the cathode and exciting nerves which terminate inside the mouth (Cadden and Newton, 1988). The absence of any difference between the thresholds for the two inhibitions when the cathode is inside, is in agreement with a previous study in which both electrodes were placed on the mucosa of the lip (Godaux and Desmedt, 1975). By contrast, other studies in which intra-oral stimuli have been applied to structures such as the gingivae, alveolar mucosa or hard palate (Yu et al, 1973; Cadden and Newton, 1988), have suggested that the first inhibitory response has a lower threshold than the second. This may reflect regional differences within the mouth and further suggest that long latency responses are, relatively, stronger from the lip region.

The other new finding was that the threshold of the long-latency excitation was significantly lower than those of any other response with the cathode outside and lower than the threshold of the short-latency inhibition with the cathode inside. This, arguably more than any previous finding, emphasises that the long-latency excitation is not just a consequence of the preceding inhibition but is a response to the stimuli in its own right (see e.g. Glas et al, 1985; Cadden et al, 1996). Furthermore, there is little or no possibility that the 2nd excitation could be a voluntary response rather than a reflex in view of its latency. It had a shorter latency (mean 94.8 ms with cathode inside and 96.5 with cathode outside) than the shortest reported reaction time. Yemm (1972b) reported

that the shortest reaction time in his subjects was 120 ms, although Glas et al (1984) observed a reaction time of 106 ms with mechanical stimulation and Brodin et al (1993) recorded 100 ms with both mechanical and electrical stimulation.

By contrast, there was no evidence to support the contention of Brodin and Türker (1994) that the first excitatory response has a lower threshold than either of the inhibitory responses evoked by electrical stimulation of the lip. Indeed in the present study, there were no significant differences with either polarity of stimulation, between the threshold of the first excitation and those of the inhibitions.

As has been discussed previously (for review see Lund 1991), these reflexes may have functional significance during mastication. The inhibitory responses have long been thought to be protective reflexes which may prevent biting trauma (Bratzlavsky, 1972; Yu et al, 1973; Cadden and Newton, 1988). Putative roles for the excitatory responses are more difficult to envisage. There is evidence that similar excitatory responses evoked by mechanical stimulation of teeth, may have roles to play in load compensation during mastication to take account of different consistencies of food (Ottenhoff et al, 1992). However such a role is less easily ascribed to excitatory responses evoked by stimulation of the lip as in the present study.

CHAPTER FIVE

MASSETER MUSCLE REFLEXES IN BRUXIST SUBJECTS.

Summary

It has been reported that a different pattern of jaw reflexes may be evoked by mechanical stimulation of the teeth in bruxists (De Laat et al, 1985). The aim of this study was to investigate whether such differences also exist in response to electrical stimulation of the lip. EMG recordings were made from a masseter muscle in 17 bruxists and 17 age- and sex-matched, non-bruxist controls. With activity in the muscle sustained at around 10 % of maximum, stimuli were applied through bipolar electrodes clipped over the lower lip with the cathode intra-orally. The presence of reflex responses was determined critically as described by Louca et al (1996). Friedman's ANOVA and Wilcoxon tests (with Bonferroni correction as appropriate) were used to analyse the 4 standard responses (short and long latency inhibitions and excitations). P values<0.05 were taken to be significant.

In both groups, there were significant differences in the thresholds for the different responses. These differences were more marked in the bruxists where both the short-latency inhibition and long-latency excitation had significantly lower thresholds than the long-latency inhibition. There were differences between the two groups in the presence of the short-latency excitation and the long-latency inhibitory responses. The short-latency excitation were seen in 88% of bruxists but only 59% of controls. By contrast, the long-latency inhibition was seen in only 65% of bruxists but 94% of controls. Furthermore, when present, the long-latency inhibition had a significantly longer latency

in the bruxists than in the controls (54.5±2.8 ms compared to 44.5±1.6 ms, P=0.003). These findings suggest that, as with responses to mechanical stimulation of teeth, longer latency inhibitory responses evoked by electrical stimulation of the lip are weaker in bruxists than in non-bruxists.

5.1 Introduction

There is general agreement on the bi-phasic pattern of the inhibitory response in healthy subjects (Bratzlavsky, 1972; Yemm, 1972; Yu et al, 1973; Godaux and Desmedt, 1975; Turker & Miles, 1985; Carels and van Steenberghe, 1986; De Laat et al, 1985; Cadden & Newton, 1988, 1994; Okdeh et al, 1996).

The incidence of a second silent period after mechanical or electrical tooth pulp stimulation was lower in TMD patients (De Laat et al., 1985; Sharav et al, 1982;). Whereas, Hussein & McCall (1983) consistently found two inhibitory periods in TMD patients. Thus, the pattern of responses in dysfunction and parafunction is unclear.

It is generally accepted that the aetiology of temporomandibular disorders is multifactorial (Moss & Garrett, 1984; Yemm, 1985), and many authors (Kraus, 1966; Franks, 1965; Laskin, 1969; Christensen, 1971; Trenouth, 1979) have implicated

bruxism in the aetiology of TMD. The only study has been carried out on masseter muscle reflexes in bruxist subjects found that there was a strong correlation between bruxism and the occurance of single inhibitory period in response to mechanical stimulation of the tooth (De Laat et al, 1985). The aim of this study was to investigate whether such difference can be observed in bruxists in response to electrical stimulation of the lip.

5.2 Materials and methods

Subjects

Seventeen bruxists and seventeen age- and sex-matched non-bruxist controls participated in the study. Ages ranged from 21 to 45 years (mean 27±1.5). Bruxism was diagnosed on the criteria proposed by Franks (1965) which were: (1) the patients or their spouses were aware of grinding or clenching their teeth, (2) the jaw muscles felt tired in the morning, (3) the presence of wear facets on antagonist teeth with mucosal ridging adjacent to the occlusal plane.

The method of recording and analysing the signals is described earlier in Chapter 3.

5.3 Results

Threshold analysis

In both groups, there were significant differences in the thresholds of the four different responses. A non-parametric ANOVA (Friedman test) revealed that the differences in the thresholds of the responses was significant in both bruxists (P = 0.002) and non-bruxist (P = 0.034). The reflex threshold for all responses for both bruxist and non-bruxist are presented in Table (6).

Monophasic short-latency inhibitory periods were typically observed in bruxists (Figure 16). Wilcoxon Signed Rank tests for multiple comparisons, followed by a Bonferroni correction of 6, revealed that the threshold of the short-latency inhibitory period was significantly lower than the threshold of the long-latency inhibitory period (P = 0.036). Also the long-latency excitatory period had a significantly lower threshold than the long-latency inhibitory period (P = 0.012). The results of the threshold comparison are listed in Table (7). In the non-bruxist group the only significant difference was that the long-latency excitatory period had a significantly lower threshold than the short-latency inhibitory period (P = 0.024). There were no significant differences in the thresholds of the short- and long- latency inhibitions, suggesting that a bi-phasic inhibitory period was typical in the non-bruxist group (Figure 17).

Subject	Non-Bruxist Subjects				Bruxist Subjects				Subject
	1st inhib	1st excit.	2nd inhib.	2nd excit.	1st inhib.	1st excit.	2nd inhib.	2nd excit.	
MM	6	*	5	5	5	2	*	5	NF
MJ	9	*	3	2.5	6	*	*	7	GL
HU	7	2	5	5	3	2.5	5	1.5	SM
AB	5	5	5	5	3	*	5	3	CH
FA	7	*	5	3	6	2	*	5	KR
JO	7	2	5	1	6	5	*	*	RH
AS	5	2	3	3	2	6	8	6	AL
AT	5	*	3	7	5	5	8	6	FR
MU	8	*	6	6	6	5	8	7	ME
IS	5	3	5	3	6	2.5	5	1.5	KH
NF	5	2	8	5	7	8	5	1.5	CC
KF	9	1	5	1	2	2	2	2	SA
LF	5	*	*	2	8	3	8	6	SN
RF	5	5	7	2	7	3	6	7	MM
SS	5	6	5	6	6	2	6	2	DC
CS	5	5	3	5	8	2	*	*	PA
CB	7	*	5	1	7	3	*	5	ES
Median	5.0	5.0	5.0	3.0	6.0	3.0	8.0	5.0	Median

Table (6) Thresholds of significant responses for all volunteers. (*) denotes a non-significant response. The non-significant responses were given a value of 10 during statistical analysis.

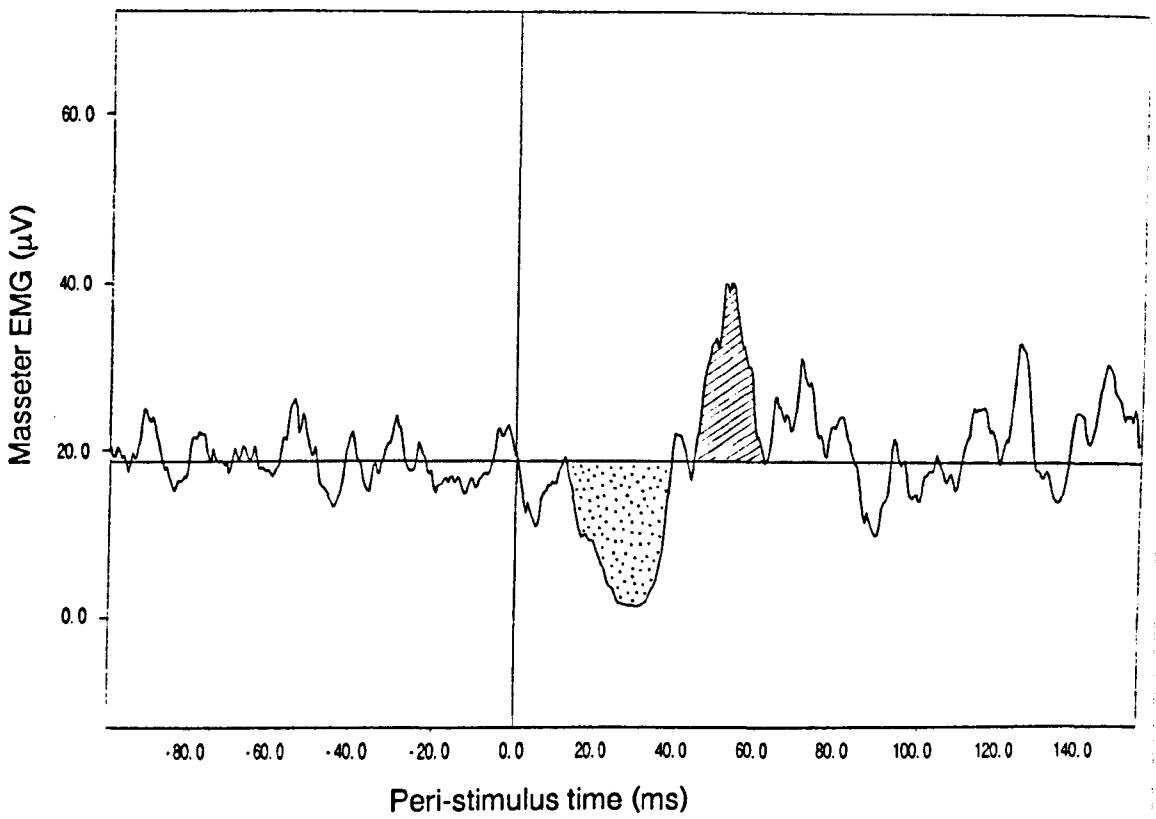


Figure (16) An example of the averaged EMG showing inhibitory reflexes in an active masseter muscle in response to a 7T stimulus applied to the lower lip in a bruxist subject. The horizontal line shows the mean level of activity in the pre-stimulus period, the stippled area shows the inhibitory reflex and the lined areas show the excitations. The vertical line indicates the timing of the stimulus. Note the monophasic short-latency inhibition.

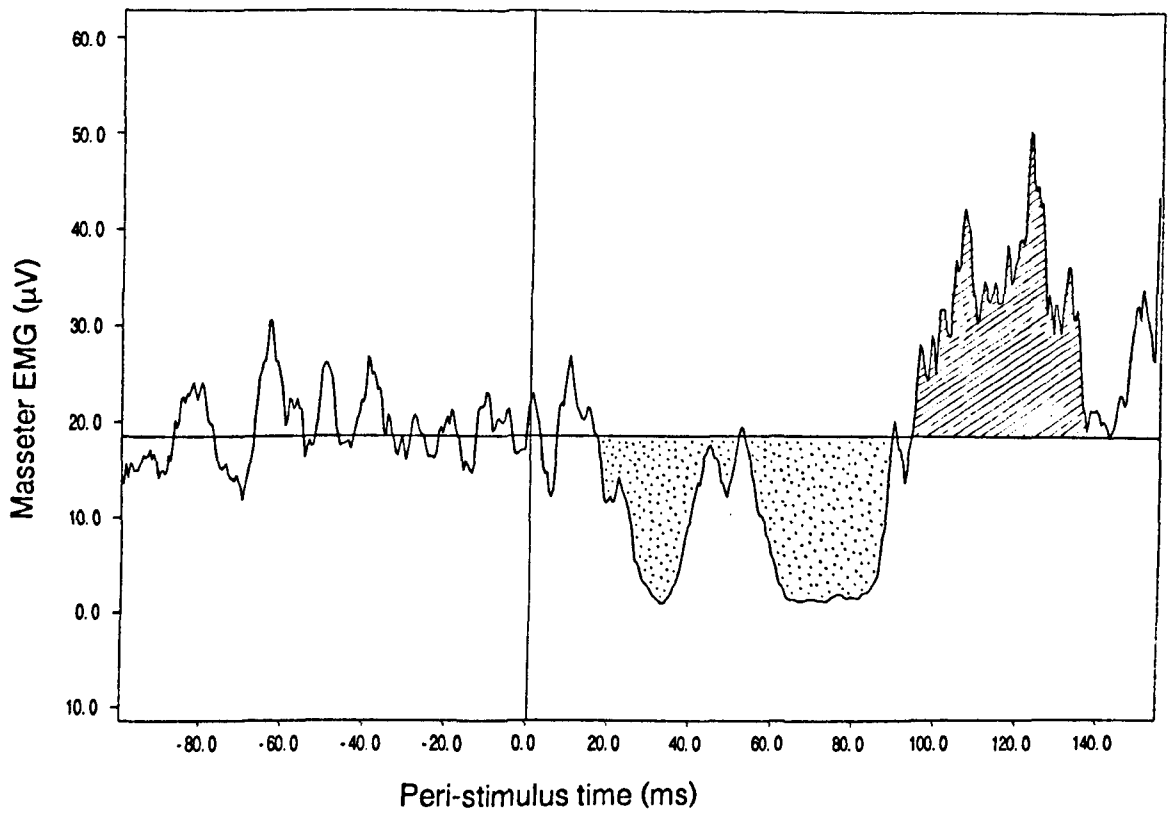


Figure (17) Averaged EMG showing the reflexes in an active masseter muscle in response to a 7T stimulus applied to the lower lip in non-bruxist subject. The horizontal line shows the mean level of activity in the pre-stimulus period, the stippled areas show the inhibitory reflexes and the lined areas show the excitations. The vertical line indicates the timing of the stimulus. Note the difference in the pattern of the responses, two inhibitory periods occurring in non-bruxist but just one in bruxist subject.

	Non-Bruxist P value	Bruxist P value
1st inhib. Vs 1st excit.	0.975	0.201
1st inhib Vs 2nd inhib	0.167	0.006
1st inhib. Vs 2nd excit	0.004	0.463
1st excit. Vs 2nd inhib	0.379	0.012
1st excit. Vs 2nd excit	0.028	0.331
2nd inhib Vs 2nd excit	0.060	0.002

Table (7) A comparison of threshold values for the four separate responses produced for bruxist and non-bruxist subjects. Results not Bonferroni corrected.

There were also differences between the two groups in the presence of the first excitatory and second inhibitory responses; the first excitation was seen in 88% of bruxists but only 59% of non-bruxist. By contrast, the second inhibition was seen in only 65% of bruxists but 94% of non-bruxist. Fisher's Exact Test, for comparing the ratios of the presence of responses in both groups, revealed that there was a significant difference between the ratios of bruxists and non-bruxist in the presence of the long-latency inhibitory period (P = 0.039), indicating that the long-latency inhibitory period occurred more frequently in the non-bruxist subjects. Figures 81 and 19 show the number of subjects who produced significant inhibitory and excitatory responses in both non-bruxist and bruxists groups respectively.

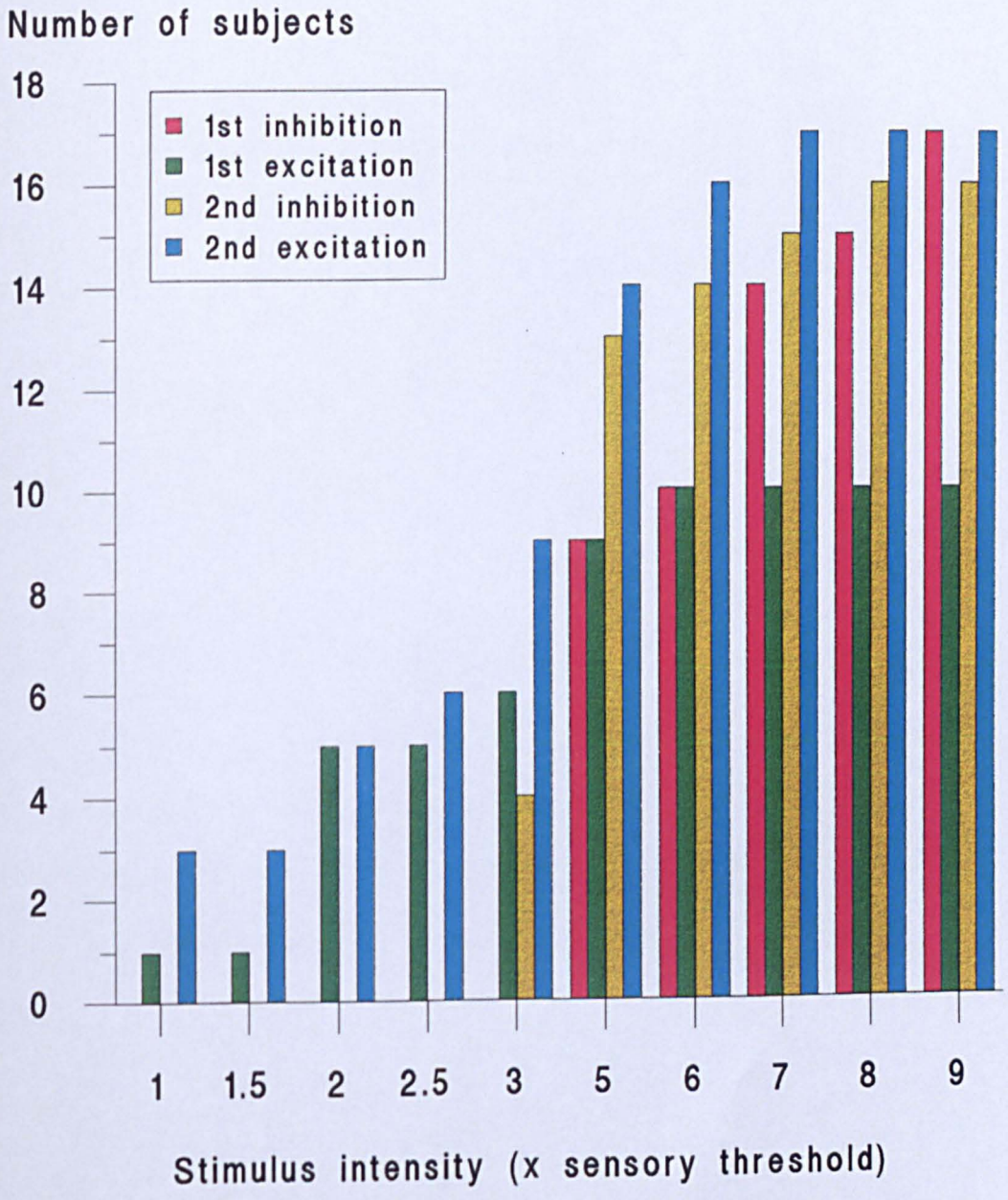


Figure (18) Cumulative frequency bar graphs showing the number of **non-bruxist** subjects who gave a significant inhibitory or excitatory response at or below each stimulus intensity.

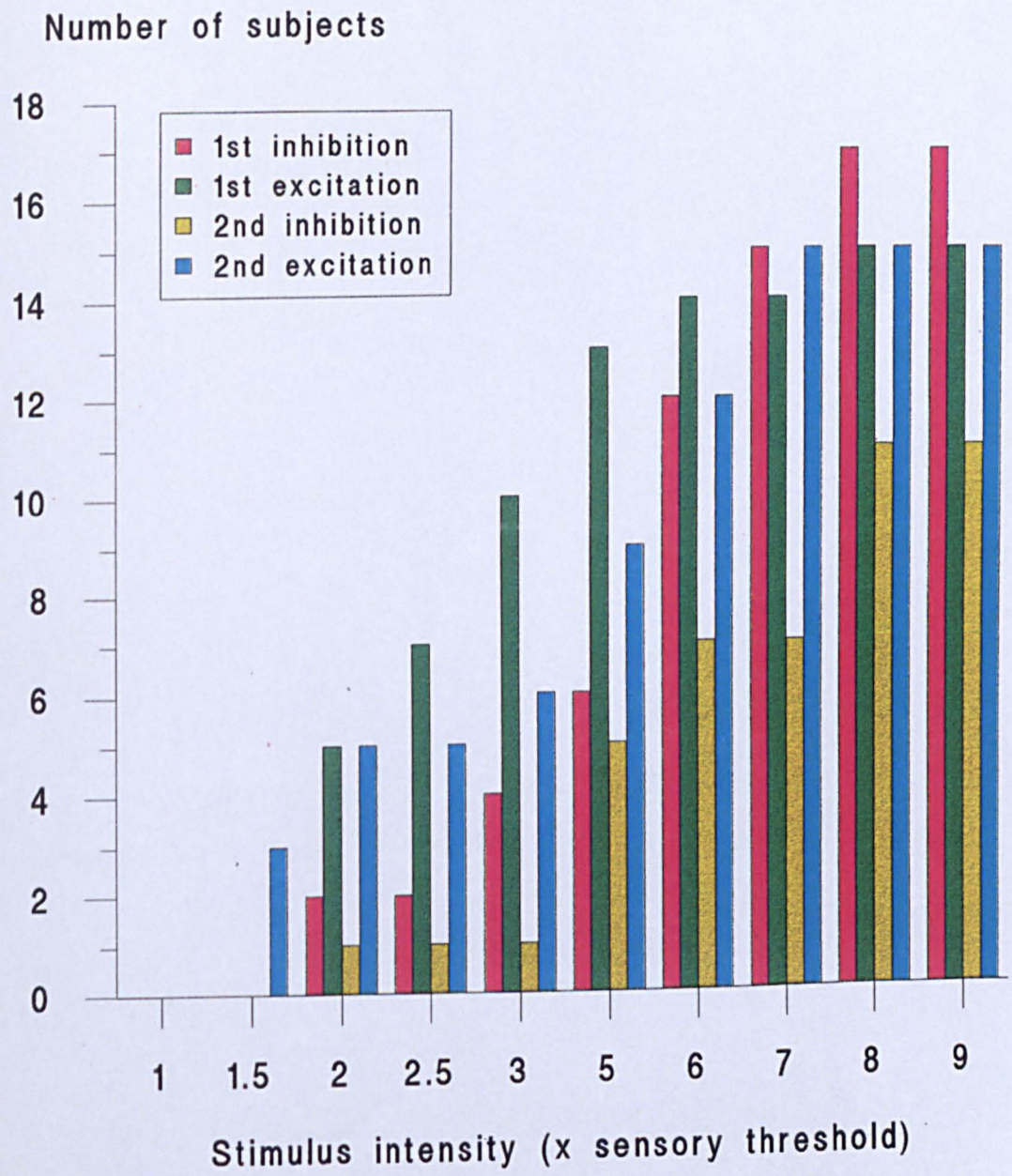


Figure (19) Cumulative frequency bar graphs showing the number of **bruxist** subjects who gave a significant inhibitory or excitatory response at or below each stimulus intensity.

Latencies and durations of inhibitory and excitatory responses

The mean latencies and durations of all responses for both non-bruxist and bruxists are summarised in Tables 8 and 9 respectively. It was found that the long-latency inhibitory period had a significantly longer latency in the bruxists than in the controls (54.5 ± 2.8 ms compared to 44.5 ± 1.6 ms, P = 0.003, Wilcoxon Rank Sum test). Further, the duration of the short inhibitory period was significantly longer in bruxists than in non-bruxist (41.8 ± 3.3 compared to 25.3 ± 1.6, P = 0.005).

Magnitude of inhibitory responses

Pooled data of the inhibitory waves showed that, in bruxist, the magnitude of short-latency inhibitory periods at 6T, 7T, 8T, and 9T was significantly greater than the mean control level. While the long-latency inhibitory period was not significantly greater than the mean control level at any stimulus intensity (Figure 20).

Magnitude of excitatory responses

The short-latency excitatory waves were significantly greater than the mean control level only at 6T. However, the magnitude of the long-latency excitatory waves was greater than the mean control level at 6T, 7T, 8T and 9T (Figure 21).

Response	N	Latency				Duration			
		Threshold		Minimum		Threshold		Maximum	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
1st inhibition	17	14.93	1.21	13.18	1.05	21.39	0.89	25.29	1.60
1st excitation	10	36.22	1.26	35.89	1.22	22.39	2.65	23.00	2.05
2nd inhibition	16	48.38	3.87	44.56	1.63	37.72	3.23	47.37	3.81
2nd excitation	17	96.79	3.69	89.06	3.92	31.85	3.10	49.26	3.91

Table (8) Latencies and durations (ms) of significant responses from non-bruxist subjects. "N" represents the number of subjects showing a particular response at any stimulus intensity. Data given in columns labelled "Threshold" represent responses obtained with the lowest intensity of stimulation which evoked that response in each subject. In addition, data are given for the minimum latency and maximum duration obtained at any stimulus intensity.

Response	N	Latency				Duration			
		Threshold		Minimum		Threshold		Maximum	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
1st inhibition	17	12.85	0.95	9.91	0.82	29.91	2.92	41.74	3.35
1st excitation	15	38.17	1.85	33.5	1.32	26.1	2.01	35.80	2.86
2nd inhibition	11	59.71	3.35	54.46	2.76	33.75	2.37	41.71	3.20
2nd excitation	15	87.83	4.26	81.10	3.61	30.87	2.34	47.33	3.40

Table (9) Latencies and durations (ms) of significant responses obtained in bruxist subjects. "N" represents the number of subjects showing a particular response at any stimulus intensity. Data given in columns labelled "Threshold" represent responses obtained with the lowest intensity of stimulation which evoked that response in each subject. In addition, data are given for the minimum latency and maximum duration obtained at any stimulus intensity.

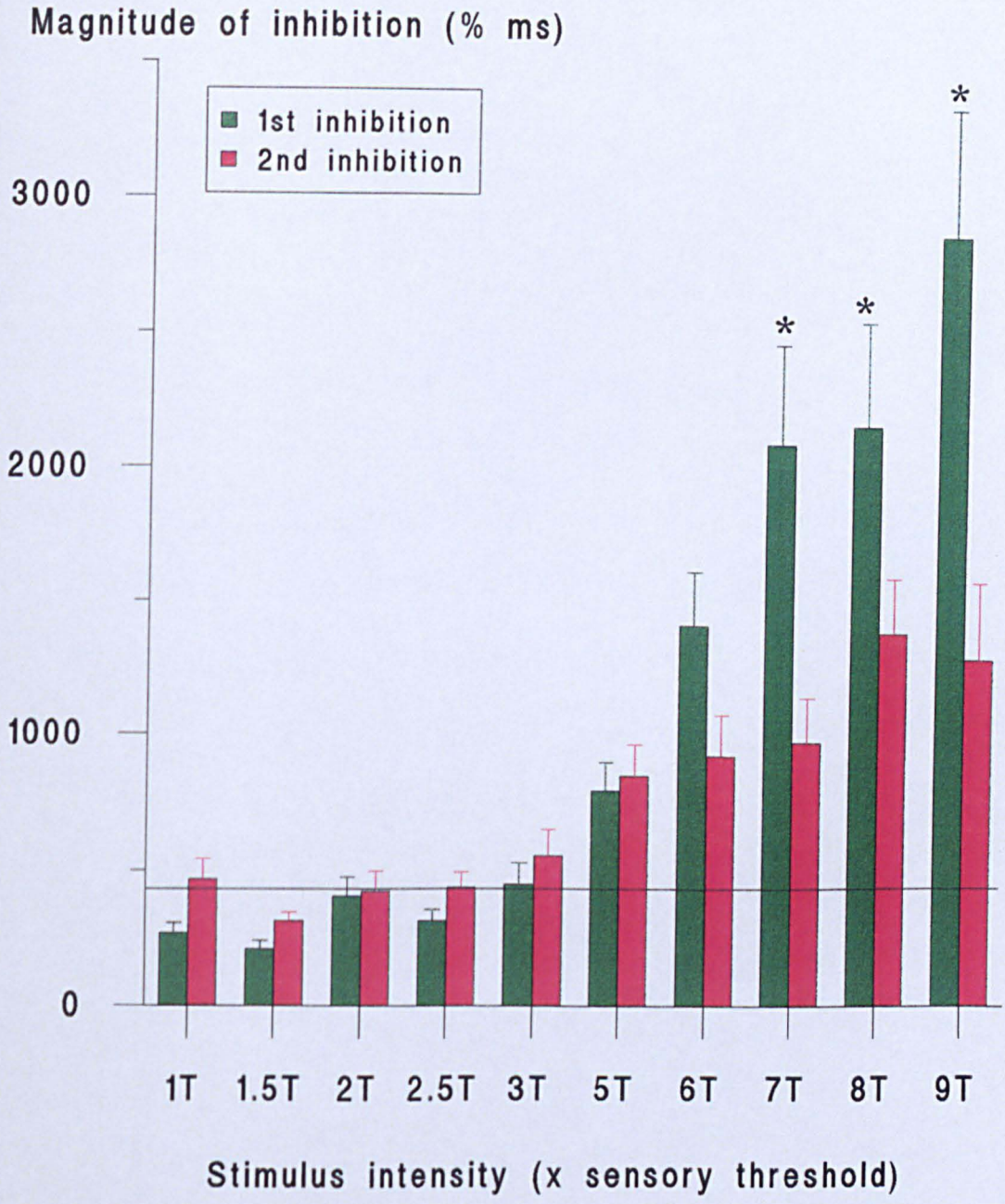


Figure (20) Pooled data from 17 **bruxist** subjects showing the magnitude of the 1st and 2nd inhibitions. The solid horizontal line represents the mean size of the largest downgoing waves in the control records. Asterisks indicate values which are significantly greater than the mean control level.

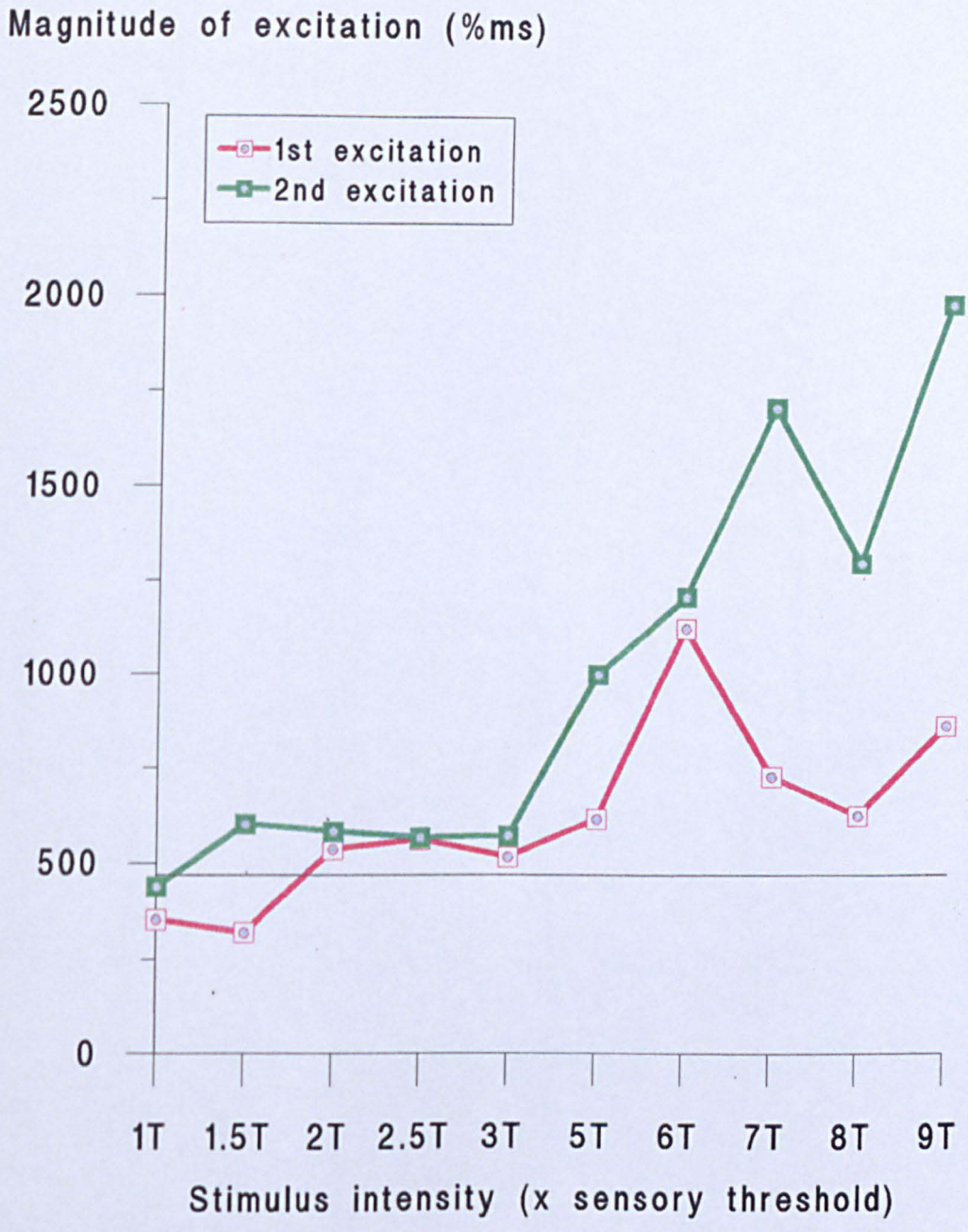


Figure (21) Pooled data from 17 **bruxist** subjects showing the magnitude of the 1st and 2nd excitations. The solid horizontal line represents the mean size of the largest upgoing waves in the control records.

5.4 Discussion

Electrical stimulation of the mucous membrane of the lower lip elicited inhibitory and excitatory responses in the active masseter muscle in both control and bruxists subjects but the pattern of responses was different. A bi-phasic inhibitory period was observed in the healthy subjects, in agreement with other reports (e.g. Yu, et al, 1973; Cadden & Newton, 1988), The latencies of both the short-latency and long-latency inhibitions were similar to those observed by others (e.g. Turker & Miles, 1985; Cadden & Newton, 1988).

In bruxists, a mono-phasic inhibitory period was predominant. De Laat et al (1985) also found that mono-phasic inhibitory periods predominated in response to mechanical stimulation of the teeth in bruxists. Furthermore, the long-latency inhibitory period, when present, had a significantly longer latency in the bruxists than in controls ($P = 0.003$); this is to be expected because generally the weaker a response, the longer its latency. The significant difference in the ratio of occurance of the long-latency inhibitory period indicated that this response occurred more frequently in the control group.

The other interesting finding is that the short-latency excitation was absent in 7 of the 17 controls but in only 2 of the 17 bruxists; this might indicate either that (a) the short-

latency excitation, when presents, tends to mask the following inhibition and/or (b) the long-latency inhibition, when present, tends to obscure the preceeding excitation.

Muscular activity has been found to be increased in bruxists (Ramfjord, 1961; Fuchs 1975), and that might explain the occurrence of the single inhibitory period in bruxist subjects. This muscular hyperactivity was found to decrease immediately following the insertion of an occlusal splint both in bruxist subjects (Solberg et al, 1975) and in subjects who undertook experimental tooth clenching (Christensen, 1980; Cox et al, 1983).

The other important finding was that the duration of the short-latency inhibitory period was significantly longer in bruxists than in controls ($P = 0.005$). To the knowledge of the author, there is no report on the duration of inhibitory responses in bruxists. However, experimental bruxism, which has shown to produce symptoms similar to TMD (Christensen, 1971), prolongs the inhibitory period to a similar extent reported to occur in TMD patients (Cox et al, 1982). The hyperexcitability of the muscle and an increased excitability of Golgi tendon organs might contribute to the lengthening of the inhibitory period in TMD patients (Bessette et al, 1971). Therfore, the elongation of the inhibitory period in bruxist subjects and after experimental bruxism might support the hypothesis that muscle hyperactivity plays an important role in the aetiology of TMD.

CHAPTER SIX

MASSETER MUSCLE REFLEXES IN TMD PATIENTS

Summary

It has been reported that inhibitory reflexes in the active masseter muscle are different in patients with temporomandibular disorders (TMD). The aim of the present study was to investigate the effect of occlusal splint therapy on masseter muscle reflexes in these patients. EMG recordings were made from an active masseter muscle in 14 patients with TMD, both before and one week after wearing a full coverage stabilisation splint. Stimuli were applied through bipolar electrodes clipped over the lower lip with the cathode placed intra-orally against the mucous membrane. The thresholds of the 4 standard responses (short and long latency inhibitions and excitations) were compared using Wilcoxon's Signed Ranks tests with a Bonferroni correction. Before treatment the threshold of the short latency inhibition was significantly lower than that of the long latency inhibition ($P<0.05$). This difference was not found after treatment. Furthermore, the long latency excitatory period had a significantly lower threshold than the other three responses before treatment but not after treatment. A comparison of each response before treatment with those of a healthy group from a previous experiment revealed that the long latency inhibition had a significantly lower threshold in the healthy group (Wilcoxon Rank Sum, $P = 0.0012$, Bonferroni Correction applied). While there was no significant difference between the responses of the two groups after treatment ($P=0.057$). Treatment also resulted in a significant reduction in the severity of the symptoms of TMD ($P<0.005$). Thus even one week of occlusal splint therapy results in changes in the pattern of reflexes as well as symptomatic relief. Since the changes of the reflexes involved a relative increase in inhibitory responses, it is possible that this would

reduce the use of the muscles and be, at least in part, responsible for the accompanying symptomatic relief.

6.1 Introduction

Differences have been found in the inhibitory reflexes of TMD patients and symptom-free subjects. Griffin and Munro (1971) reported a shorter latency and more frequent absence of the silent period (SP) in patients during the open-close-clench cycle. Other studies found significantly longer durations of the inhibitory period after chin tap, (Bessette et al, 1971; Widmalm, 1976; McCall and Hoffer, 1981), which shorten again to normal ranges after occlusal splint therapy (Bessette et a, 1971; Baily et al, 1977; Bessette and Shatkin, 1979; Skiba & Laskin, 1981). The SP duration increased when the condition was acute, and returned to normal when the condition was successfully treated. These authors claimed that the duration of the SP was of diagnostic value in the detection of myofascial pain and the evaluation of treatment. The results of these studies have been questioned, however, because of the large intraindividual variations and methodological problems (Hellsing & Klineberg, 1983; Lavinge et al, 1983). Further, it has been also found that there were no significant differences in SP duration between the patients that achieved complete, partial or no relief of the symptoms (Strychalski et al, 1984).

Two inhibitory periods in the surface EMG after mechanical stimulation of a single tooth have been found in symptom-free subjects, while only a single inhibitory period tended to occur in patients with muscle pain (De Laat et al, 1985). This pattern changed to include two inhibitory periods after occlusal splint treatment (De Laat & Steenberghe, 1985).

More single inhibitory periods have been found by others when using electrical tooth-pulp stimulation in TMD patients, while double or merged inhibitions were present in the symptom-free group (Sharav et al, 1982). By contrast, Hussein & McCall (1983), using electrical stimulation of the mental nerve area, consistently found two inhibitory periods in both patients and symptoms-free subjects, but the latencies were shorter and the durations longer in the patients. Intra-subject and inter-subject variation in the latencies of the early and late inhibitions has been found to be small but differences have been occasionally observed in the duration.

In response to electrical stimulation of the lower lip in healthy subjects, it has been found, from the previous study (Chapter 4), that two inhibitory periods were usually evoked. However, in bruxist subjects a single short latency inhibitory period was predominant (Chapter 5).

The aims of the present study were to determine whether systematic differences could be detected in electrically-evoked inhibitory and excitatory responses in TMD patients and what effect, if any, an occlusal splint had on these responses.

6.2 Materials and methods

Subjects

Experiments were performed on 14 volunteer myogenous TMD patients (10 women and 4 men; aged 18-60 years), all of whom gave informed consent. The patients were chosen carefully and comprised of a group complaining of muscle pain and tenderness, with some limitation of opening in three subjects. There were no signs of internal derangement or other temporomandibular joint problems.

Visual analogue scale

At the begining of each visit patients were asked to record the intensity of pain on that particular day on a visual analogue scale. The scale used was a straight undivided line 100 mm in length (Figure 22), the ends of which were marked by a statement indicating the extreme limits of pain sensation to be measured, that is, "no pain" at the far left and "extreme pain" at the far right. The pain score for each visit was obtained by measuring the distance in millimetres from the far left end.

A full-arch maxillary stabilization splint was used. The splint was made of heat-polymerized acrylic resin and had a flat occlusal surface with occlusal contact in centric relation for all opposing teeth, with canine guidance anteriorly and laterally.

No Pain **Extreme Pain**

Figure (22) The Visual Analogue Scale used in this study.

The average interocclusal thickness of the splint was approximately 1.5 mm at the posterior teeth (Figure 23). The splint was adjusted and used by each patient during sleeping hours for one week. Figure 24 shows one patient with the splint in situ.

EMG recordings were made from the masseter muscle on the symptomatic side. The clip electrode was placed over the lip with the cathode being against the mucous membrane. The methods for evoking and recording the jaw reflexes were similar to those employed in previous studies.

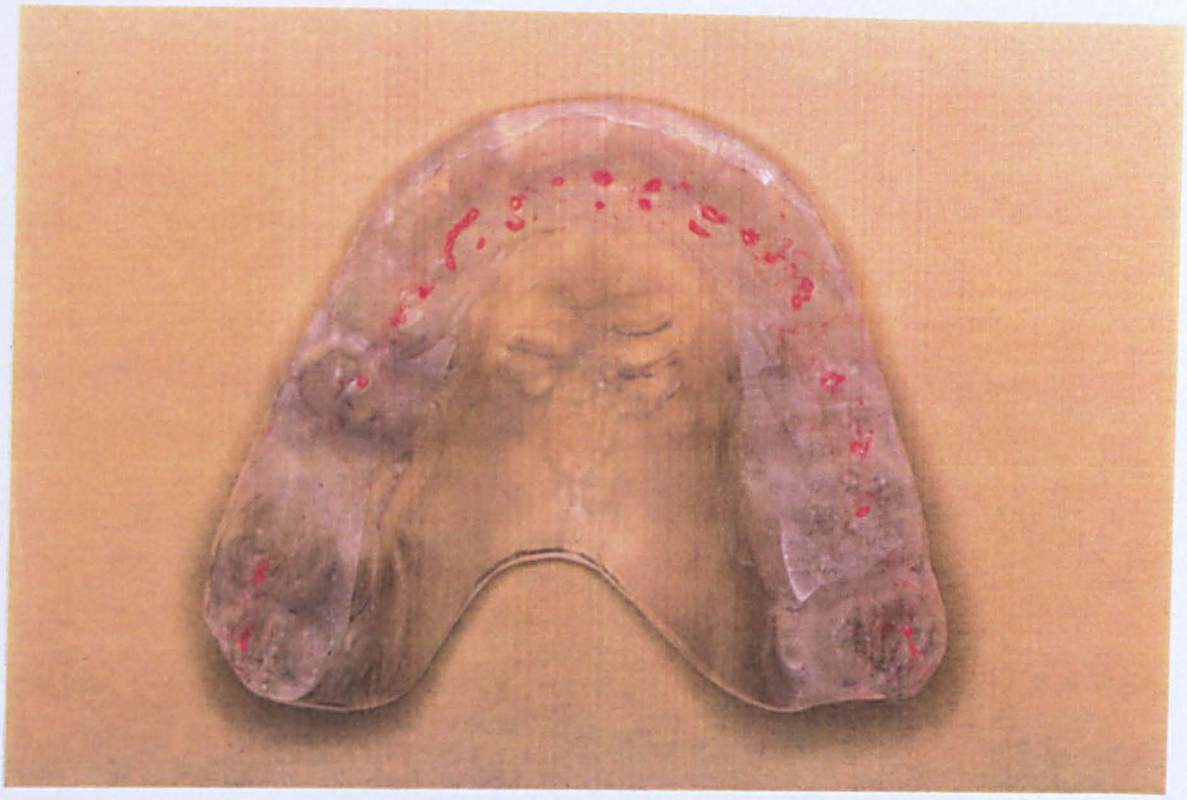


Figure (23) Maxillary stabilization splint. Note the contact marks (blue) distributed evenly over the flat surface of the splint.

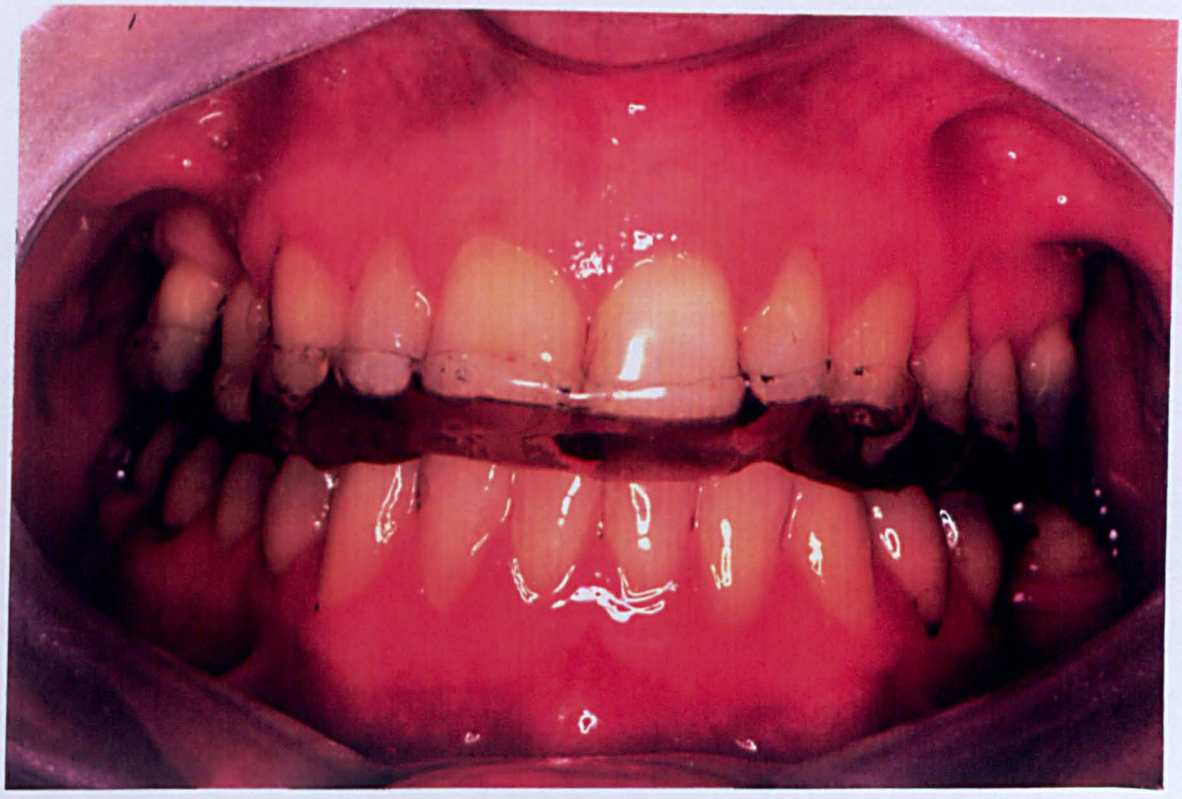


Figure (24) Patient with the splint in situ. Note the even contacts with the patient closed in centric relation.

6.3 Results

Severity of symptoms

At the end of one week of treatment all but one of the patients reported that their pain was considerably reduced. The mean VAS score before treatment was 5.59 ± 0.6 mm and after treatment was 2.04 ± 0.55 mm and this reduction was significant ($P<0.005$). There was also a reduction in muscle tenderness to palpation. Three patients who had limitation of mouth opening before treatment were able to open normally after treatment.

Threshold analysis

All 14 patients produced masseter muscle reflexes in response to electrical stimulation of the lower lip. The thresholds of these four responses were significantly different before treatment (Friedman test, $P = 0.001$), while after treatment there was no significant difference ($P = 0.091$). The reflex threshold for all responses before and after treatment is presented in Table (10).

Before treatment:

Monophasic short-latency inhibitory periods were typically observed before treatment (Figure 25). Analyses of the thresholds of the responses revealed that the threshold for

the short-latency inhibitory period was significantly lower than the threshold for the long-latency inhibitory period. Furthermore, the long-latency excitatory period had a significantly lower threshold than the short-latency excitation and long-latency inhibition but not the short-latency inhibition. The results of the threshold comparisons are listed in Table (11).

After treatment:

After treatment, there were changes in the pattern of the responses (26), most notably in the thresholds of the responses. There was now no significant difference between the thresholds of the short- and long-latency inhibitions with bi-phasic inhibitory periods being observed more frequently. Also there was no significant difference between the thresholds of the long-latency excitations and the other three responses.

Absolute comparison between each response before and after treatment revealed that the only significant difference in the threshold response was that the long-latency inhibition had a significantly lower threshold after treatment. Figures 27 and 28 shows the number of subjects who produced significant inhibitory and excitatory responses before and after treatment respectively.

subject	Before Treatment				After Treatment			
	1st inhib.	1st excit.	2nd inhib.	2nd excit.	1st inhib.	1st excit.	2nd inhib.	2nd excit.
MK	7	*	*	3	9	*	7	1
SN	6	6	9	5	6	*	6	6
JH	5	*	6	*	5	*	*	1
AS	2.5	*	*	2	5	*	7	3
LF	5	*	7	8	8	*	5	*
MC	*	3	7	3	8	1	8	1
AN	3	*	8	2	5	*	5	*
RW	8	*	8	2	9	7	9	7
MH	8	*	*	2	6	*	6	2.5
MB	5	3	*	3	5	*	5	*
JM	5	*	9	1.5	5	1.5	5	6
AH	5	2	9	2.5	*	1	*	1
HM	7	*	8	6	6	*	5	5
MS	2.5	*	5	3	3	7	3	2.5
Median	5	10	8.5	3	6	10	6	4

Table (10) Thresholds of significant responses for TMD patients before and after treatment. (*) denotes a non-significant response. The non-significant responses were given a value of 10 during statistical analysis.

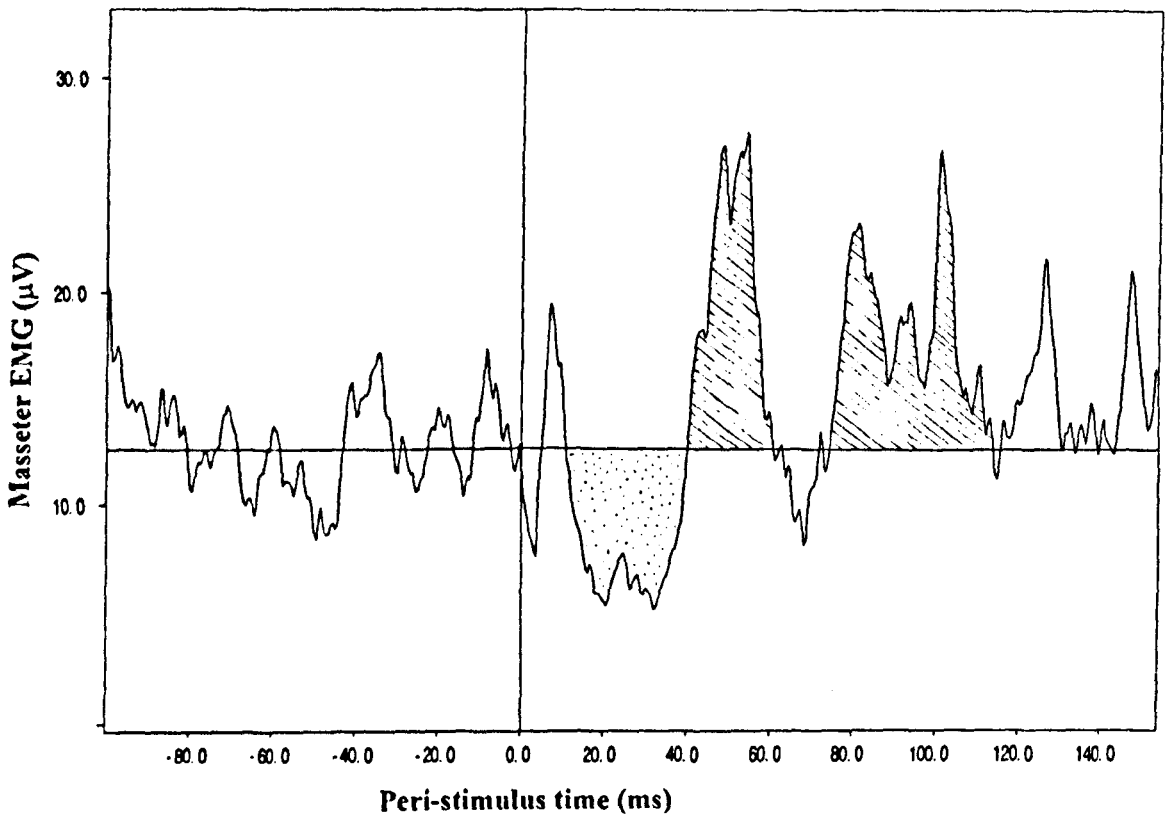


Figure (25) Averaged EMG showing inhibitory reflexes in an active masseter muscle in response to a 8T stimulus applied to the lower lip in one patient before treatment. The horizontal line shows the mean level of pre-stimulus activity, the stippled area shows the inhibitory reflex and the lined areas show the excitations. The vertical line indicates the timing of the stimulus. Note the mono-phasic inhibitory period.

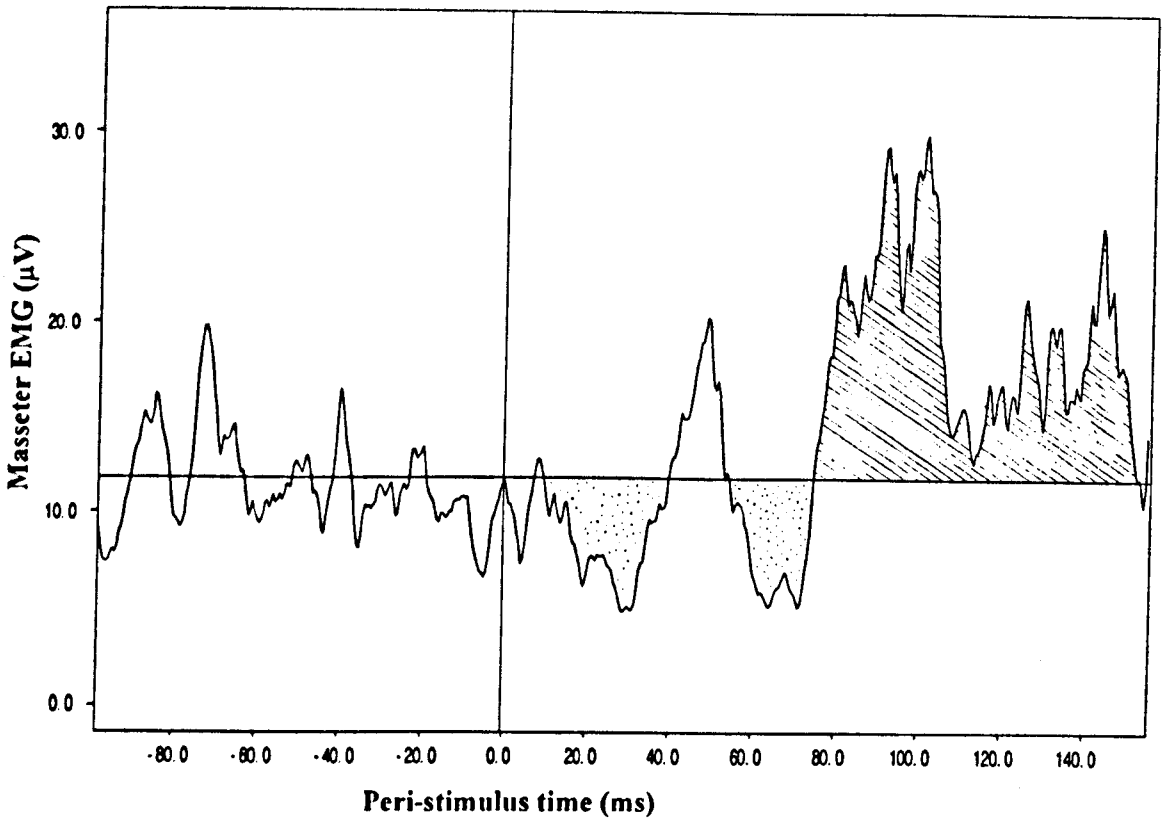


Figure (26) Averaged EMG showing inhibitory reflexes in an active masseter muscle in response to a 8T stimulus applied to the lower lip in the same patient of Figure 25, but after treatment. The horizontal line shows the mean level of pre-stimulus activity, the stippled areas show the inhibitory reflexes and the lined areas show the excitations. The vertical line indicates the timing of the stimulus. Note the difference in the pattern of responses, with one inhibitory period before treatment and two after treatment.

	Before treatment P value	After treatment P value
1st inhib Vs 1st excit	0.055	0.24
1st inhib Vs 2nd inhib	0.008	1.00
1st inhib Vs 2nd excit	0.048	0.19
1st excit Vs 2nd inhib	0.96	0.29
1st excit Vs 2nd excit	0.005	0.03
2nd inhib Vs 2nd excit	0.004	0.22

Table (11) A comparison of threshold values for the four separate responses produced before and after treatment. Results not Bonferroni corrected.

Latencies and durations of inhibitory responses:

The mean latencies and durations of the inhibitory responses before and after treatment are shown in Tables 12 and 13 respectively. It can be noted that the short-latency inhibitory period occurred in 13 patients before and after treatment, while the long-latency inhibitory periods occurred in 10 patients before and in 12 patients after treatment. The latencies of the inhibitory responses before treatment had a tendency to be shorter than the latencies after treatment, but this difference failed to reach the significance level.

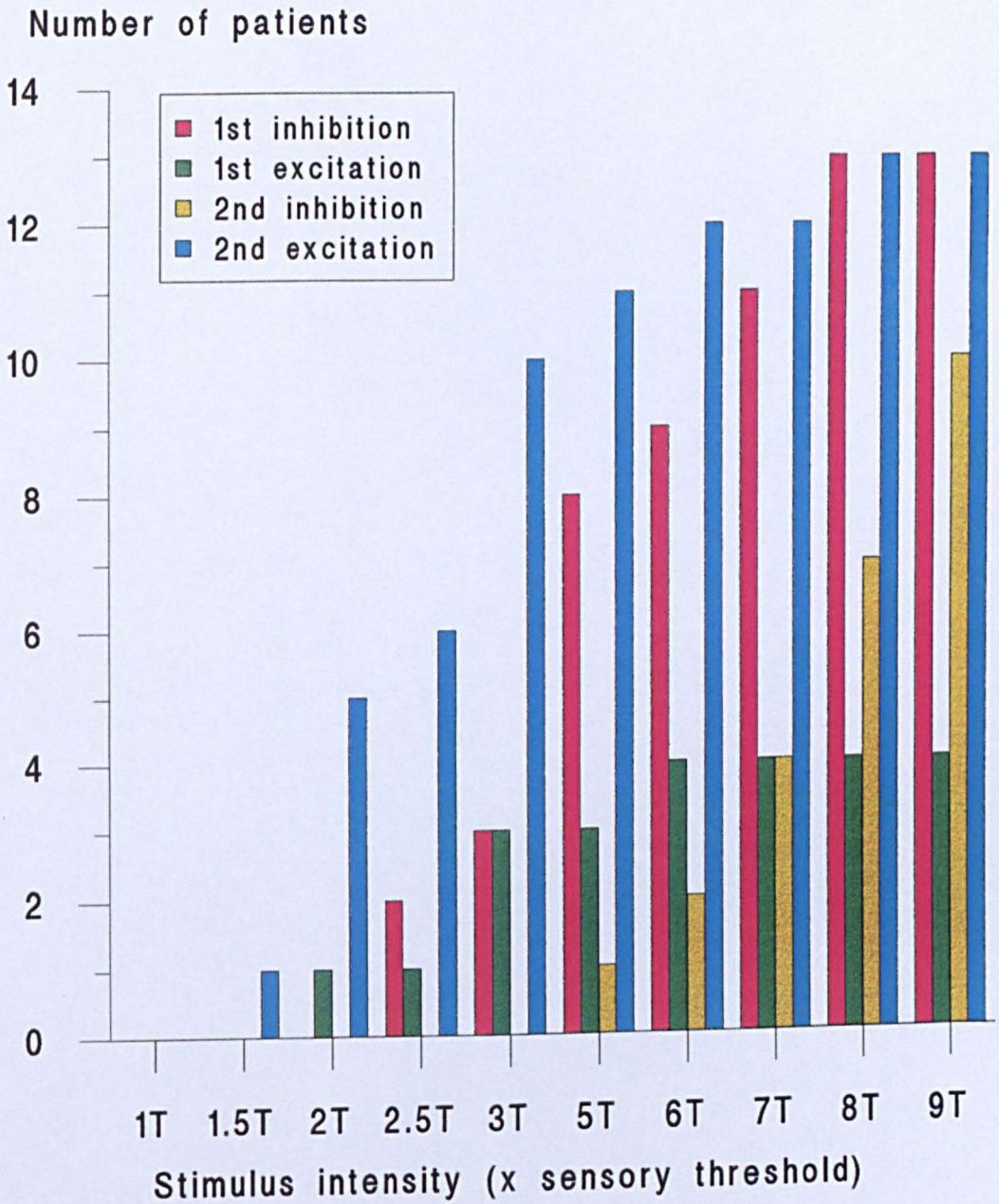


Figure (27) Cumulative frequency bar graphs showing the number of subjects who gave a significant inhibitory or excitatory response at or below each stimulus intensity **before** treatment.

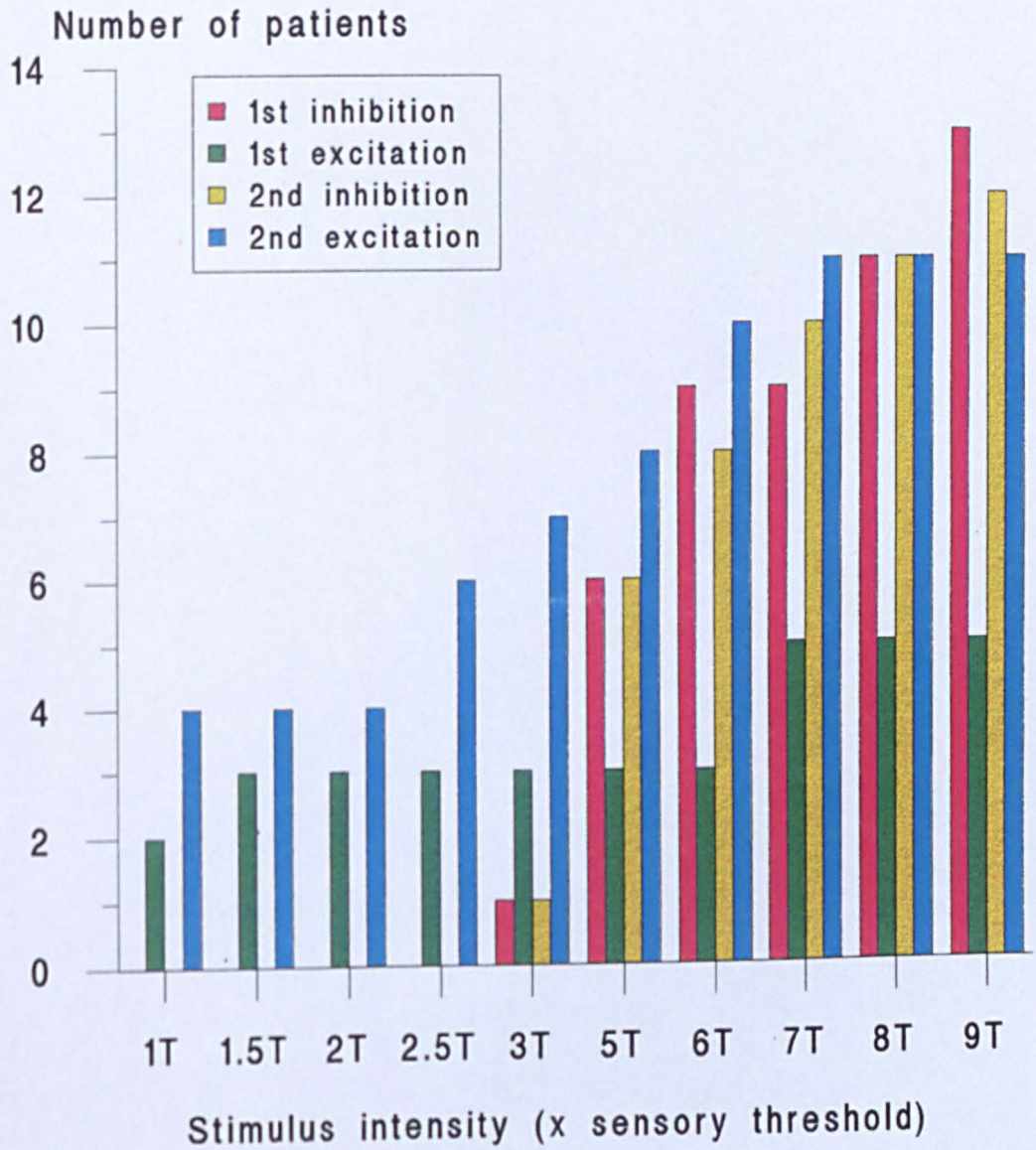


Figure (28) Cumulative frequency bar graphs showing the number of subjects who gave a significant inhibitory or excitatory response at or below each stimulus intensity **after** treatment.

Response	N	Latency				Duration			
		Threshold		Minimum		Threshold		Maximum	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
1st inhibition	13	11.12	1.08	13.31	3.37	24.12	2.04	37.5	3.02
1st excitation	4	39.0	2.50	37.5	4.0	24.25	4.25	27	7.0
2nd inhibition	10	53.15	1.90	52.05	2.13	44.90	2.95	44.90	3.30
2nd excitation	13	94.38	3.80	89.90	4.29	38.75	6.49	54.40	7.31

Table (12) Latencies and durations (ms) of significant responses before treatment. "N" represents the number of subjects showing a particular response at any stimulus intensity. Data given in columns labelled "Threshold" represent responses obtained with the lowest intensity of stimulation which evoked that response in each subject. In addition, data are given for the minimum latency and maximum duration obtained at any stimulus intensity.

Response	N	Latency				Duration			
		Threshold		Minimum		Threshold		Maximum	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
1st inhibition	13	13.91	1.63	11.50	1.32	27.45	1.98	35.06	3.45
1st excitation	5	39.6	3.26	38.80	3.84	27.0	5.92	27.0	5.95
2nd inhibition	12	58.0	2.98	52.55	1.97	32.0	3.30	47.75	4.93
2nd excitation	11	101.5	7.35	97.82	4.86	40.27	5.98	49.55	5.59

Table (13) Latencies and durations (ms) of significant responses after treatment. "N" represents the number of subjects showing a particular response at any stimulus intensity. Data given in columns labelled "Threshold" represent responses obtained with the lowest intensity of stimulation which evoked that response in each subject. In addition, data are given for the minimum latency and maximum duration obtained at any stimulus intensity.

Magnitude of inhibitory responses:

Pooled data from all the patients before treatment showed that the magnitude of short-latency inhibitory periods at 7T, 8T and 9T was significantly greater than the mean control level. The long-latency inhibitory period was significantly greater than the mean control level only at 9T (Figure 29). After treatment the magnitudes of the short and long-latency inhibitory periods were significantly greater than the mean control level only at 8T and 9T (Figure 30).

Excitatory responses

Excitatory responses, as in previous experiments, were seen both with and without a preceding inhibition, but the latter occurred only at low stimulus intensities. The latencies and durations of these responses are summarised in Tables 12 and 13.

Excitatory responses not preceded by an inhibition

Before treatment, one subject showed short-latency excitatory responses and five showed long-latency excitatory responses which were not preceded by an inhibition. After treatment two subjects showed short-latency excitatory responses and three showed long-latency excitatory responses not preceded by an inhibition. The latencies and durations of these responses are summarised in Table 14.

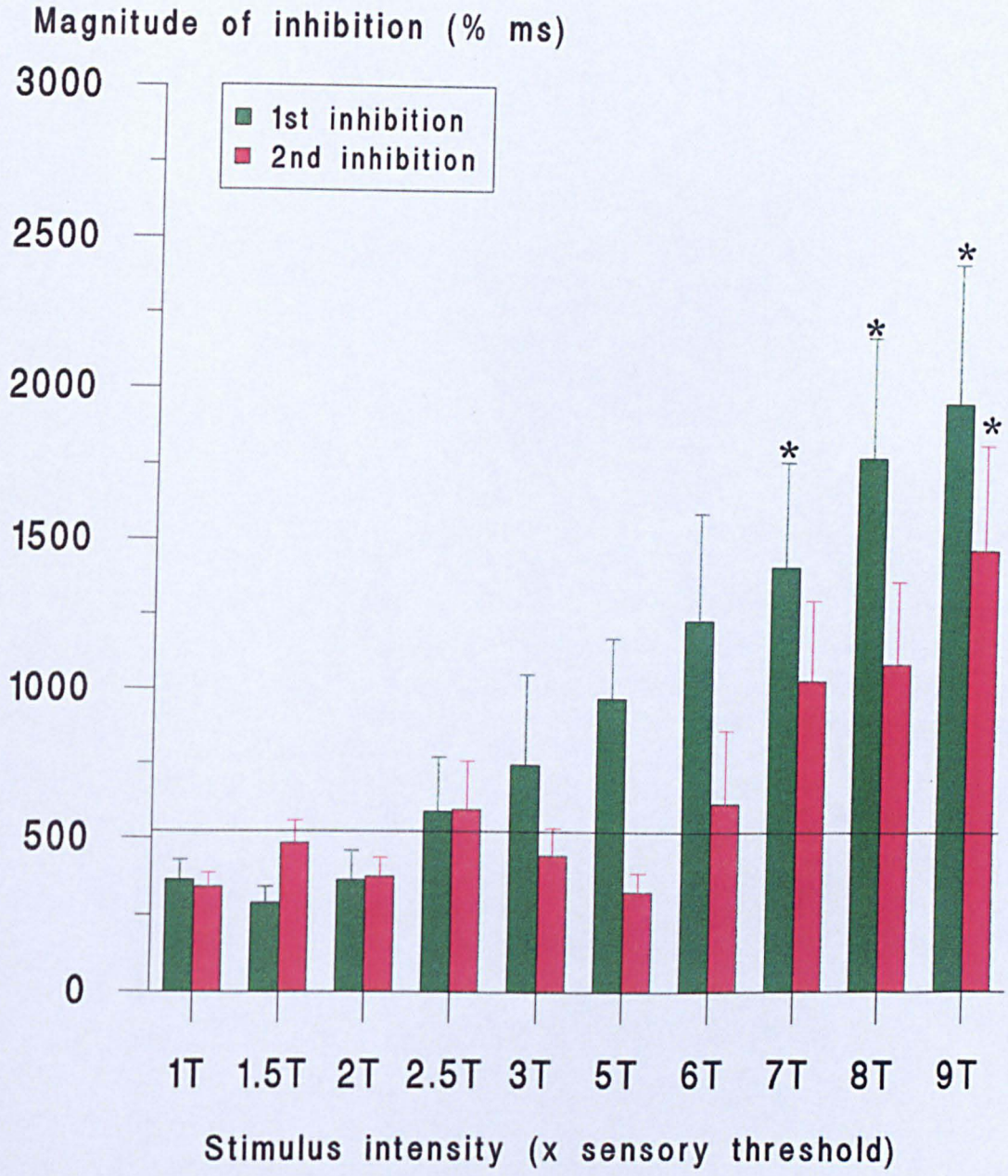


Figure (29) Pooled data showing the magnitude of the 1st and 2nd inhibitions **before** treatment. The solid horizontal line represents the mean size of the largest downgoing waves in the control records. Asterisks indicate values which are significantly greater than the mean control level.

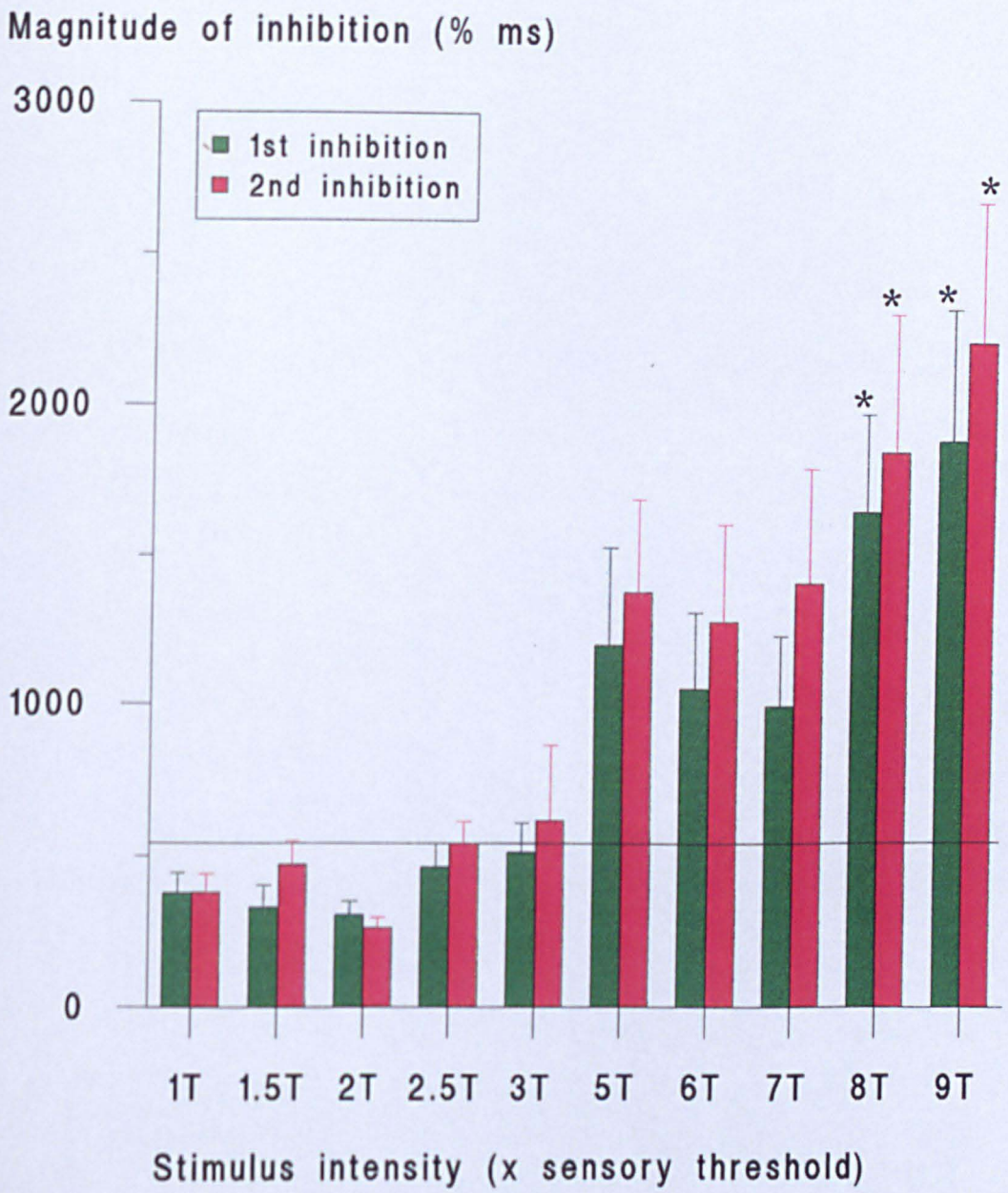


Figure (30) Pooled data showing the magnitude of the 1st and 2nd inhibitions **after** treatment. The solid horizontal line represents the mean size of the largest downgoing waves in the control records. Asterisks indicate values which are significantly greater than the mean control level.

Responses	N	Latency (ms)		Duration (ms)	
		Mean	SEM	Mean	SEM
1st excit (Before treatment)	1	23.83	-	25.33	-
2nd excit (Before treatment)	5	97.81	3.27	27.59	2.81
1st excit (After treatment)	2	37.50	2.50	21.83	1.75
2nd excit (After treatment)	3	99.51	2.65	34.71	2.26

Table (14) Latencies and durations of the excitatory responses not preceded by an inhibition.

Magnitude of excitatory responses

None of the short-latency excitatory waves were significantly greater than the mean control level, either before or after treatment. It was only at 9T that the magnitude of the long-latency excitatory waves was significantly larger than the mean control level before treatment, despite the fact that they had a significantly lower threshold than the short-latency responses. However, after treatment the magnitude was greater than the mean control group at 7,8 and 9T (Figure 31).

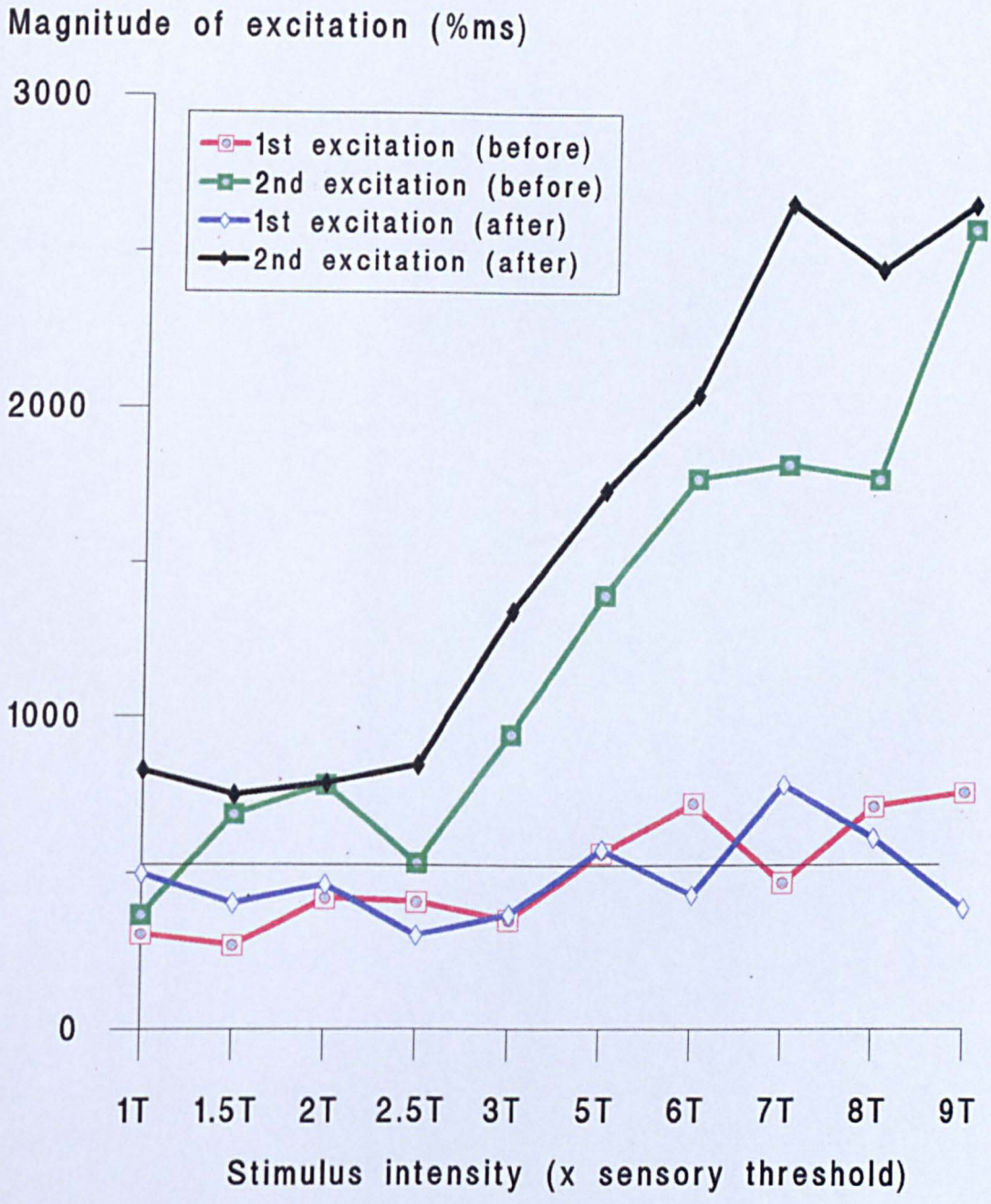


Figure (31) Pooled data showing the magnitude of the 1st and 2nd inhibitions **before** and **after** treatment (see text). The solid horizontal line represents the mean size of the largest upgoing waves in the control records.

Comparison between TMD patients and controls before and after treatment:

Absolute comparison between each response in the patients before treatment with the control group revealed that the long-latency inhibition in controls had significantly lower threshold than in TMD patients (P=0.0002, Wilcoxon Rank Sum test). This difference did not exist after treatment (P=0.057). Values not Bonferroni corrected. There was no difference with regard to other responses. Fisher's Exact test revealed no significant differences in the ratio of occurrence of the responses between controls and TMD patients, neither before treatment nor after treatment.

Comparison between TMD patients and bruxists before and after the treatment:

Absolute comparison of each response in the TMD patients before treatment with the bruxist group revealed that the short-latency excitation had a significantly lower threshold in the bruxists than the TMD patients (P=0.0025, Wilcoxon Rank Sum test). After treatment, the difference was still significant (P=0.032), but not as significant as it was before treatment. Values not Bonferoni corrected. There was no difference with regard to other responses.

Fisher's Exact test revealed that there was a significant difference in the ratio of occurrence of the short-latency excitation between bruxists and TMD patients before treatment (P=0.0009) which means that it occurred more frequently in the bruxists. After treatment the difference was still significant (P=0.003) but again not as significant as it was before treatment. There was no difference with regard to other responses.

6.4 Discussion

The symptom of TMD that prompts most patients to seek treatment is pain (Bell, 1990; McNeill, 1990; Dworkin & LeResche, 1992), therefore it was considered important to measure the patients' subjective perception of their pain before and after treatment to assess the clinical effect of this treatment. VAS is widely used and has been shown to be a rapid, easy, and valid method for measuring both clinical and experimental pain (Woodforde & Merskey, 1972; Scott & Huskisson, 1976; Aun et al, 1986).

Most patients reported that their pain decreased and the VAS data showed that the reduction in pain was significant ($P < 0.005$). These results are in agreement with other reports of the high rate of clinical success achieved with splints used alone or combined with other therapies (Zarb &Thompson, 1970; Greene & Laskin, 1972; Carraro & Caffesse, 1978; Dahlstrom et al, 1982; Okeson et al, 1982, 1983; Clark, 1984; Moss & Garrett, 1984; Sheikhleslam et al, 1986; Tsuga et al, 1989; Suvinen & Reade, 1989; Wilkinson et al, 1992). This finding also confirms the clinical impression that a well-made stabilisation splint has a significant effect on symptoms after one week.

Double inhibitory periods from the masseter muscle in response to electrical stimulation of the oral mucosa in asymptomatic subjects have been reported (e.g. Yemm, 1972; Yu et al, 1973; Cadden and Newton, 1988; Okdeh et al, 1996). In contrast to healthy subjects, the most obvious difference observed in TMD patients was that the short-latency inhibitory period appeared to be more prominent (i.e. had a significantly lower threshold) than the long-latency inhibitory period. Sharav et al, (1982), using electrical

tooth-pulp stimulation, also found more single inhibitory periods in patients, while double or merged inhibitions were present in the symptom-free group. Another study by De Laat et al, (1985) reported that mechanical stimulation of teeth consistently evoked only the short-latency inhibitory period in TMD patients. In contrast, Hussien & McCall (1982) consistently found two inhibitory periods in both TMD patients and symptom-free subjects in response to electrical stimulation of the mental nerve area. The reason for this discrepancy in the pattern of reflexes might be that the results presented in this study and those of Sharav et al, (1982) and De Laat et al, (1985) were obtained by stimulating intra-oral nerve fibres while Hussien and McCall (1982) stimulated peri-oral nerves.

Two inhibitory periods were predominant after treatment; the stimulus that normally evokes a single inhibitory period before treatment might produce a double inhibitory period after treatment (Figure 26). As the biting level was controlled by visual feedback in this experiment, the occurrence of the double inhibitory period cannot be explained as being due to the decrease in muscle hyperactivity induced by the splint as has been suggested by others (Griffin & Munro, 1971; Vissere et al, 1995). However, De Laat & Steenbergh (1985) also reported that in TMD patients the pattern of responses to tooth taps changed to a double inhibitory period after successful occlusal treatment.

This finding of an extra inhibitory period after treatment might be consistent with an hypothesis that there is hyperexcitability of jaw closing muscles in TMD patients before treatment and that part of the success of the treatment results from a reflexly mediated decrease in excitability of the central motoneurone pool. The extra inhibitory periods

after treatment may result in reduction in the hyperactivity of the muscles and hence a reduction in pain. This increased inhibition is possibly a defence mechanism to protect the muscle from overloading (Widmalm, 1976).

One week after treatment the relation between the long-latency excitation to the other responses had changed, but not to the pattern which has been reported in healthy subjects (Okdeh et al, 1996). The long-latency excitation had a significantly lower threshold than the other responses before treatment, but after treatment there was no significant difference in thresholds between the 4 responses (short and long latency excitations and inhibitions). This relationship after treatment was different to the relationship in healthy subjects, in which the long-latency excitation had a significantly lower threshold than the other responses. Although this change in the relationship between the long-latency excitation and the other responses after the treatment may be surprising, a diminished importance of excitatory responses could contribute further to a useful decreased excitability in the motorneurone pool. To the best of the author's knowledge there is no other report of such a difference in the relationship of the excitatory periods to the other responses.

Although most of the previous investigations of masseter muscle inhibitory periods in TMD patients have emphasized the latency and duration of the inhibitory period, it was found that the pattern of both inhibitions and excitations is different. Moreover, the reflex pattern of the inhibitory waves returns to normal after treatment, while the excitatory waves have a different (i.e. not normal) pattern.

CHAPTER SEVEN

GENERAL DISCUSSION AND CONCLUSIONS

7.1 GENERAL DISCUSSION

The transient decrease of the EMG activity level (inhibitory reflexes) following stimulation of the peripheral receptors has been extensively reported by many others who have used a variety of stimulation techniques such as electrical, mechanical and acoustic. It has been demonstrated in this study that electrical stimulation in or around the mouth can produce both inhibitory and excitatory reflexes. This is in agreement with previous studies in which electrical shocks to the lip have been used to evoke jaw reflexes (e.g. Yemm, 1972 a; Bratzlavsky, 1972; Yu et al, 1973; Godaux and Desmedt, 1975; Desmedt and Godaux, 1976; Di Francisco et al, 1986; Cadden and Newton, 1988; Brodin and Türker, 1994). Further, the latencies of the short- and long-latency inhibitions were similar to those observed by others (e.g. Turker & Miles, 1985; Cadden and Newton, 1988).

There was a consistent difference between the patterns of reflex thresholds when the stimulus polarity was reversed. The difference was such that the short-latency inhibitory response had a similar threshold to that of the long-latency inhibition when the cathode was applied to the inside of the lip ($P= 0.72$) but a significantly higher threshold when the cathode was outside the lip ($P= 0.001$). This finding supports the previous studies of Yu et al (1973) and Cadden and Newton (1988), in which the first inhibition can be produced only by stimulation inside the mouth but that the second inhibition can be produced by intra- or peri-oral stimulation.

It has been found that the short-latency inhibitory response has a lower threshold than the long-latency response when intra-oral stimuli have been applied to structures such as the gingivae, alveolar mucosa or hard palate (Yu et al, 1973; Cadden and Newton, 1988). However, in the present study, there was no difference between the thresholds for the two inhibitions when the cathode was inside. This is in agreement with a previous study in which both electrodes were placed on the mucosa of the lip, and always elicit two successive inhibitory periods with identical stimulus thresholds (Godaux and Desmedt, 1975). This may suggest that long latency responses are, relatively, stronger from the lip region.

Contrary to earlier findings (Bratzlavesky, 1972; Yu et al, 1973; Godaux and Desmedt, 1975), the threshold of the long-latency inhibitory response was below the pain threshold, and the volunteers described it as uncomfortable only at 8T and 9T. This is in accord with Grath et al (1981) who found that the threshold for suppression of masseter muscle activity was far below the pain threshold. Other experiments in which the oral mucosa was stimulated electrically also reported that the threshold of the reflex response was below the pain threshold (e.g. Cadden & Newton, 1988; Turker, 1988; Brodin et al, 1994; McMillan, 1994). Moreover, as the stimulus intensity increased, there was a tendency for one phase of inhibition to merge with another, forming one long fused response; this has also been found by others (e.g. Godaux and Desmedt, 1975; Cadden and Newton, 1988; Brodin and Türker, 1994).

The clenching level, in this study was 10% of the maximum EMG activity. This was chosen because it was enough to elicit masseter muscle reflexes, and yet muscle fatigue

was avoided. This level was easily maintained by the subjects. The level of background activity has been found to be an important determinant of the pattern of the reflex response (Glas et al, 1984).

In the other part of the study, the inhibitory response patterns in bruxist subjects were markedly different, with long-latency inhibition more prominent in normal subjects and short-latency inhibition more persistent in bruxist subjects. De Laat et al (1985) also reported that there was a strong correlation between tooth-grinding habits and the occurrence of a single inhibitory period. The other important finding is that the duration of the short-latency inhibitory period was significantly longer in bruxists than in controls ($P = 0.005$). Furthermore, the long-latency inhibitory period, when present, had a significantly longer latency in the bruxists than in controls ($P = 0.003$); this is to be expected because generally the weaker a response, the longer its latency.

In TMD patients, controversy exists regarding the pattern of inhibitory periods as some have reported the occurrence of only a single inhibitory period (Sharav et al, 1982; De Laat et al, 1985; Turker et al, 1989, 1994), while others have reported the occurrence of double inhibitory periods, as in healthy subjects (Hussien and McCall, 1982; Bjorland et al, 1996). However, in this study, the most obvious difference observed between TMD patients and healthy subjects was that, in TMD patients, as in bruxist, the short-latency inhibitory period appeared to be more prominent (i.e. had a significantly lower threshold) than the long-latency inhibitory period. The differences in the methodology used by different workers might be the reason for the discrepancy in the pattern of inhibitory responses.

Since many studies have reported elevated EMG activity levels, in the masticatory muscles of TMD patients and bruxists (Jarabak, 1956; Ramfjord, 1961; Lous et al, 1970; Fuchs 1975; McCall et al, 1978), the similarity in the pattern of inhibitory periods in TMD patients and bruxists subjects was expected. There was no significant differences in the threshold of the inhibitory responses, nor in the ratio of occurrence of these responses between the two groups. The other important point is that, in contrary to TMD patients, the bruxist subjects were asymptomatic at the time of the experiment which might indicate that the increased hyperactivity and not pain was the reason for the occurrence of the single inhibitory period in both TMD patients and bruxist subjects.

The increased muscular hyperactivity was found to be decreased immediately following the insertion of an occlusal splint in bruxist subjects (Solberg et al. 1975), and in experimental tooth clenching (Christensen, 1980; Cox et al., 1983). Further, in TMD patients, two inhibitory periods were predominant after occlusal splint treatment; the stimulus that normally evokes a single inhibitory period before treatment might produce a double inhibitory period after treatment. The extra inhibitory periods after treatment may result in reduction in the hyperactivity of the muscles and hence a reduction in pain. This increased inhibition is possibly a defence mechanism to protect the muscle from overloading (Widmalm, 1975).

The other important response in the pattern of jaw reflexes, in this study, was the excitatory responses. The new finding was that, in healthy subjects, the threshold of the long-latency excitation was significantly lower than those of any other responses with

the cathode outside and lower than the threshold of the short-latency inhibition with the cathode inside in healthy subjects. Furthermore, in contrast to Brodin and Turker (1994), there were no significant differences between the threshold of the first excitation and those of the inhibitions with either polarity of stimulation.

In TMD patients, one week after treatment the relation between the long-latency excitation to the other responses had changed. The long-latency excitation had a significantly lower threshold than the other responses before treatment, but not after treatment. This relationship after treatment was different to the relationship in healthy subjects, in which the long-latency excitation had a significantly lower threshold than the other responses. A diminished importance of excitatory responses could contribute to a useful decreased excitability in the motoneuron pool.

The other interesting finding in this study was that the short-latency excitation was the only response which significantly differed between the TMD and bruxist groups, and there was no significant difference in the other responses between the two groups. In bruxist subjects the short-latency excitation had a significantly lower threshold than in the TMD patients before treatment ($P=0.0025$) and also it occurred more frequently in the bruxists ($P=0.0009$). This predominant response in bruxist is surprising and hard to explain.

Goldberg (1971) reported that high intensity (near pain threshold) electrical stimulation delivered to the gingiva, evoked a brief, short latency excitatory reflex (latency approximately 7 ms). Clark and Goldberg (1983) also reported the presence of an

excitatory reflex. On the other hand, Yemm, 1972, Bratzlavesky (1972), Yu et al (1973), Godaux and Desmedt (1975), Carels and Steenberghe (1986), and the present study, observed no short-latency excitatory response in the masseter muscle. It might be that the excitatory action from these receptors is so small that indeed higher stimulus intensities with a larger area for stimulation must be used to evoke this response, as suggested by Clark and Goldberg (1983).

The jaw-reflexes may have functional significance during mastication. The inhibitory responses have long been thought to be protective reflexes which may prevent biting trauma (Bratzlavsky, 1972; Yu et al, 1973; Cadden and Newton, 1988). Bratzlavesky (1972) further suggested that the short-latency reflex inhibition is involved in the modulation of the jaw closing muscle activity during normal chewing and licking movements. The excitatory responses that were observed in this study might be analogous to the excitatory responses evoked by mechanical stimulation of teeth, and could be a part of a reflex that might be responsible for positioning and crushing the food.

7.2 CONCLUSIONS

From this study, the following conclusions were reached:

1. Stimulation of perioral skin preferentially produces long-latency jaw reflexes, while intra-oral stimulation produces the short- and long-latency jaw reflexes.

2. The principal response to stimulation of either oral mucosa or peri-oral skin may be a long-latency excitatory response.

3. In bruxist subjects, the short-latency inhibitory responses were more prominent (had significantly lower threshold) than the long-latency inhibitory responses.

4. The pattern of jaw reflexes in TMD patients was also different from healthy subjects, in which the short-latency inhibitory responses were more prominent than the long latency inhibitory period.

5. The pattern of the masseter reflexes was different in TMD patients before treatment (mono-phasic inhibitory period) compared to after treatment (bi-phasic inhibitory periods).

6. It appears that treatment can alter the relationship between the threshold of the long-latency excitatory responses and those of the other responses.

7. After one week of wearing an occlusal splint at night, there was a significant reduction in the severity of symptoms in *TMD patients.*

Finally, although most of the previous investigations of masseter muscle inhibitory periods in TMD patients have emphasised the latency and duration of the inhibitory period, it was found in this study, that the pattern of both inhibitions and excitations is different from healthy subjects. Further, the role of the excitatory responses in TMD patients, specially after treatment, needs further investigation.

APPENDIX A.

The effect of stimulus polarity on jaw reflexes evoked by electrical stimulation across the lip. A. M. Okdeh*[1], M. F. Lyons[1] and S.W. Cadden[2] ([1]Dental School, University of Glasgow; [2]Dental School, University of Dundee, UK)
Journal of Dental Research, 1996, **75** : P. 1140.

It is often reported that electrical perioral, like intraoral, stimuli can produce a sequence of inhibitory, excitatory, inhibitory and excitatory responses in jaw closing muscles; however, other studies have shown that perioral stimuli may produce only the last two of theses responses. The aim of this study was to investigate whether this discrepancy might be related to the polarity of the stimuli. EMG recordings were made from a masseter muscle in 14 subjects. Constant current stimuli were applied through bipolar electrodes clipped over the lower lip. Perception thresholds were determined for both electrode polarities and stimuli set at multiples of this were then applied while the subjects maintained the EMG level around 10% of maximum with the aid of visual feedback. The presence or absence of responses in EMG was determined as previously described (Louca C, et al, J. Physiol. 475; 5P-6P, 1994). With both electrode polarities, it was possible to produce all 4 responses but the threshold for the 2nd excitatory response was lower than those for some or all of the other responses (Wilcoxon Signed Ranks test, $P<0.05$). Furthermore, when the cathode was outside the lip, the 2nd inhibitory response had a significantly lower threshold than the 1st inhibition ($P= 0.02$) although there was no significant difference between these thresholds when the cathode was inside the lip. Since one would expect nerves to be excited particularly around the cathode, these findings support the view that stimulation of perioral nerves preferentially produces long latency jaw reflexes. In addition, it appears that a late excitatory response in jaw closing muscles may be the principal response to stimulation of labial skin or mucosa.

Jaw reflexes evoked by stimulation across the lip. A. M. Okdeh*[1], M. F. Lyons[1] and S.W. Cadden[2] ([1]Dental School, University of Glasgow; [2]Dental School, University of Dundee, UK).
Journal of Orofacial Pain, 1996, 10 : P. 175.

Electrical stimulation of intra-oral sites may produce a sequence of inhibitory, excitatory, inhibitory and excitatory responses in jaw closing muscles. However, there is disagreement about the pattern of responses evoked by stimulation of peri-oral sites, with some studies showing only the long-latency responses similar to the last two evoked by intra-oral stimuli. The aim of this study was to investigate the responses obtained by stimulation across the lip, with different stimulus polarities and varying stimulus intensities. EMG recordings were made from a masseter muscle in 14 subjects and constant current stimuli were applied through bipolar electrodes clipped over the lower lip. The presence or absence of responses in EMG was determined as previously described (Louca C, et al, J. Physiol. 475; 5P-6P, 1994). All 4 responses as described above could be obtained irrespective of stimulus polarity. However, at some stimulus intensities (5-9 times sensory threshold) there were significant differences between the sizes of the responses which were produces: most notably the short latency inhibition was larger when the cathode was inside the lip than when it was outside. However, there was no such difference with respect to the longer latency responses. Since nerves are excited preferentially around a cathode, theses results supports the hypothesis that long latency jaw reflexes are predominant ones to stimulation of peri-oral nerves (Cadden & Newton, Archs oral Biol. 33: 863-869. 1988).

The effect of occlusal splints on jaw reflexes evoked by electrical stimulation of the lower lip. A. M. Okdeh[*1], M. F. Lyons[1] and S.W. Cadden[2] ([1]Dental School, University of Glasgow; [2]Dental School, University of Dundee, UK)
Journal of Dental Research, 1996, **75** : P. 1140.

It has been reported that inhibitory reflexes in the active masseter muscle are different in patients with temporomandibular disorders (TMD). The aim of the present study was to investigate the effect of occlusal splint therapy on masseter muscle reflexes in these patients. EMG recordings were made from an active masseter muscle in 14 patients with TMD, both before and one week after wearing a full coverage stabilisation splint. Stimuli were applied through bipolar electrodes clipped over the lower lip with the cathode placed intra-orally against the mucous membrane. The presence or absence of responses in EMG was determined as previously described (Louca C, et al, J. Physiol. 475; 5P-6P, 1994). The thresholds of the 4 standard responses (short and long latency inhibitions and excitations) were compared using Wilcoxon's Signed Ranks tests with a Bonferroni correction. The severity of symptoms were also recorded before and after treatment using visual analogue scales. Before treatment the threshold of the short latency inhibition was significantly lower than that of the long latency inhibition ($P<0.04$). This difference was not found after treatment. Furthermore, the long latency excitatory period had a significantly lower threshold than the other three responses before treatment but not after treatment. The severity of the symptoms was significantly lower after treatment ($P<0.005$). Thus the pattern of the inhibitory responses was different before treatment compare to after treatment. In addition, it appears that the treatment can alter the relationship between the threshold of the long latency excitatory responses and those of the other responses.

Jaw Reflexes Evoked by Stimulation Across the Lip in Bruxists. A. M. Okdeh, M. F. Lyons and S.W. Cadden*, The Dental Schools, Universities of Glasgow and Dundee, UK). To be presented at the 20th Meetings of The Society of Oral Physiology.

It has been reported that a different pattern of jaw reflexes may be evoked by mechanical stimulation of the teeth in bruxists (De Laat et al, 1985). The aim of this study was to investigate whether such differences also exist in response to electrical stimulation of the lip. EMG recordings were made from a masseter muscle in 17 bruxists and 17 age- and sex-matched, non-bruxist controls. With activity in the muscle sustained at around 10 % of maximum, stimuli were applied through bipolar electrodes clipped over the lower lip with the cathode intra-orally. The presence of reflex responses was determined critically as described by Louca et al (1996). Friedman's ANOVA and Wilcoxon tests (with Bonferroni correction as appropriate) were used to analyse the 4 standard responses (short and long latency inhibitions and excitations). P values<0.05 were taken to be significant.

In both groups, there were significant differences in the thresholds for the different responses. These differences were more marked in the bruxists where both the short-latency inhibition and long-latency excitation had significantly lower thresholds than the long-latency inhibition. There were differences between the two groups in the presence of the short-latency excitation and the long-latency inhibitory responses. The short-latency excitation were seen in 88% of bruxists but only 59% of controls. By contrast, the long-latency inhibition was seen in only 65% of bruxists but 94% of controls. Furthermore, when present, the long-latency inhibition had a significantly longer latency in the bruxists than in the controls (54.5±2.8 ms compared to 44.5±1.6 ms, P=0.003). These findings suggest that, as with responses to mechanical stimulation of teeth, longer latency inhibitory responses evoked by electrical stimulation of the lip are weaker in bruxists than in non-bruxists.

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