A FUNCTIONAL STUDY OF HUMAN LATERAL PTERYGOID MUSCLE IN TEMPOROMANDIBULAR DISORDER PATIENTS

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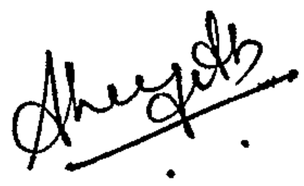
STATEMENT OF AUTHORSHIP

I declare that all the work presented in this thesis is my own, unless otherwise stated. The work of colleagues is acknowledged in general terms within the Acknowledgements and specifically within the body of the text, wherever it is appropriate.



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LIST OF ABBREVIATIONS

TMD	- Temporomandibular disorder
TMDs	- Temporomandibular disorders
TMJ	- Temporomandibular joint
TMJs	- Temporomandibular joints
LP	- Lateral pterygoid muscle
IHLP	- Inferior head of lateral pterygoid muscle
SHLP	- Superior head of lateral pterygoid muscle
EMG	- Electromyographic
VAS	- Visual analogue scale
MVC	- Maximum voluntary contraction
MPQ	- McGill pain questionnaire
MVOF	- Maximum voluntary occlusal force
SD	- Standard deviation
SMU	- Single motor unit
LEDs	- Light emitting diodes
MIPT	- Mid incisor point
CT	- Computer tomography
RMS	- Root – Mean – Square
RDC	- Research Diagnostic Criteria

ABSTRACT

Temporomandibular Disorders (TMD) comprise a number of clinical problems that involve the masticatory muscles and/or the temporomandibular joints (TMJs) and associated structures. They are the most common cause of non-dental pain in the orofacial region, and are associated with jaw joint and/or jaw-muscle pain, limitation of jaw movement and often, joint sounds. There have been some studies suggesting that TMD pain affects jaw motor activity. However most of these studies have involved experimental pain. Moreover in those studies on pain patients the jaw movements performed by the patients were not standardized. We therefore have limited information as to the effect of the chronic pain experienced by TMD patients on jaw motor activity.

The aim of this study was two-fold:

(a) to characterise jaw motor function in TMD patients performing a standardised task.

(b) to characterise the electromyographic (EMG) activity of the lateral pterygoid (LP) muscle in TMD patients to determine whether there was any evidence supporting the notions of hyperactivity or lack of coordination within the LP that have been pointed.

Six TMD patients (3 males, 3 females; age range of 31 to 52 yr) with signs and symptoms of TMJ pain, muscle pain and/or limited mandibular movement participated in this study. Electromyographic recordings were made from the inferior head of the lateral pterygoid (IHLP), masseter and anterior digastric muscles. Jaw movements were recording the movement of the mid-incisor point (MIPT, the point between the incisal edges of the lower central incisor teeth) and the palpated lateral condylar pole by using the JAWS3D

tracking system. Jaw movements consisted of standardised trials of contralateral and protrusive movements. These data were then compared qualitatively with the data already recorded from six asymptomatic subjects (age 21 – 41 yr; 3 males, 3 females) without any signs and symptoms of TMD and without any history of chronic pain or neuromuscular condition.

The results showed that there was qualitatively more variability and inconsistency in jaw tracking in the TMD patients than the asymptomatic subjects while performing similar tasks. In addition, multiple-step movements showed more variability than the single-step movements, and protrusive jaw movements were found to be more variable than the contralateral jaw movements. The TMD patients showed a higher percentage of trials with activity in masseter/digastric and IHLP muscles while they performed contralateral and protrusive jaw movements, than did the asymptomatics. Two of the six TMD patients who participated in this study showed tonic activity of IHLP at postural jaw position while none of the asymptomatic subjects showed any IHLP activity at postural jaw position. This study also suggests that the pain level increases with function and decreases with rest.

This study has provided a detailed description of some differences shown by TMD patients while performing similar jaw movement tasks in comparison with asymptomatic subjects. The data support the hypothesis that jaw motor function is altered in TMD patients. The presence of IHLP activity at postural jaw position in two out of six subjects suggests that there may be differences in IHLP activity in TMD patients in comparison with asymptomatic individuals.

INTRODUCTION

Temporomandibular Disorders (TMDs) comprise a number of clinical problems that involve the masticatory muscles and/or the temporomandibular joints (TMJs) and associated structures. Temporomandibular disorders are the most common cause of non-dental pain in the orofacial region, and are associated with jaw joint and/or jaw-muscle pain, limitation of jaw movement and often, joint sounds (Mohl 1999; Carlsson 1999). Epidemiologic surveys of TMDs have demonstrated that a considerable proportion of the population – up to 5% to 6% - experience persistent pain severe enough to demand treatment (Svensson 2001).

There have been many studies attempting to explain the cause of the pain in TMDs and the effect of the pain in TMDs. Many of these studies have infused some algesic agents like hypertonic saline into muscles, thus inducing pain (Nielsen *et al.*, 1997; Ashton-Miller *et al*, 1990; Svensson *et al*, 1998). These study designs allow the effects of the pain to be evaluated and some studies have described effects on jaw motor activity. Many studies to date have therefore been done on experimental muscle pain models (Lund *et al*, 1991; Svensson *et al*, 1995, 1997, 1998; Nielsen *et al.*, 1997; Ashton-Miller *et al*, 1990). While the cause of the pain is known in these studies since it is induced, the pain is not the same as that experienced by TMD patients who often have chronic, persistent pain. Nonetheless, it is believed that jaw motor function and chronic muscle pain are somehow

interrelated, because the cardinal symptoms of TMDs include both pain and tenderness in craniofacial muscles and restrictions and deviations in jaw movements. There have been some studies suggesting that TMD pain affects jaw motor activity, for example, mastication. However most of these studies have been done on experimental pain. Moreover in those studies on pain patients the jaw movements performed by the patients were not standardized. We therefore have limited information as to the effect of the chronic pain experienced by TMD patients on jaw motor activity.

In this study the overall aim was to provide preliminary data on the effect of chronic TMD pain on jaw motor function while the TMD patients perform a standardised motor task.

Electromyographic (EMG) Activity at Rest

Experimental Muscle Pain

A number of human experimental pain studies have been done in the past to clarify the relationship between muscle pain and surface EMG activity at rest.

The cervical-muscle EMG response to acute experimental sternocleidomastoid pain was studied by Ashton-Miller and co-workers (1990). This quantitative study revealed that the surface EMG activity of the sternocleidomastoid muscle increased significantly during a painful injection of 5-ml hypertonic saline (5%) into this muscle. But they later ascribed the changes to mimetic responses from the platysma. That is, they believed that the

changes in sternocleidomastoid EMG activity were actually due to activation of the platysma muscle as a voluntary facial response to the pain.

Stohler and co-workers (1996) conducted studies to examine whether tonic muscle pain of an intensity at least as great as reported by the majority of chronic-muscle-pain patients, caused an increase in postural EMG activity. Studies were done on 20 young volunteers: six females and 14 males, aged between 22 and 28 years. Pain was induced by computer-controlled infusion of 5% medication-grade hypertonic saline into the masseter muscle, midway between the origin and the insertion. The rate of infusion was adjusted using regularly updated pain intensity scores from a computerised visual analogue scale (VAS) with anchors marked as 'no pain' and 'most pain imaginable'. Repeated measures were taken on four experimental conditions: (1) baseline 1, (2) tonic experimental muscle pain, (3) sham pain (i.e. active recall of an experience of past pain) and (4) baseline 2. Surface electromyograms were recorded from electrodes placed over the bellies of the masseter and the anterior temporalis muscles on the right and left sides. It was found that the postural activities at all four recording sites were significantly higher from baseline 1 and 2 during tonic pain. However, postural activities during tonic pain and sham pain were not significantly different from each other. This study found significant increases in surface EMG activity of the jaw-closing muscles at rest when the jaw-closing muscles were exposed to painful infusion of hypertonic saline. But the EMG increases were not significantly different from those induced by sham pain. It was concluded that the change in EMG activity during pain was more likely because of contamination with EMG activity from mimetic muscles (Stohler 1996).

Graven-Nielsen and co-workers (1997) conducted human studies to investigate the influence of experimental muscle pain on resting, static and dynamic muscle activity. Studies were done on healthy male volunteers. In the resting and static experiments, the EMG activity and the contraction force of the tibialis anterior muscle were assessed before and after injection of 0.5 ml of hypertonic saline (5%) into the muscle. During static experiments the subjects were seated in a dental chair with the right foot strapped to a torque pedal. The torque from a dorsiflexion of the foot was recorded by a strain-gauge system. In the dynamic experiment, injections of 0.5-ml hypertonic saline (5%) were performed into either the tibialis anterior muscle or the gastrocnemius muscle, and the muscle activity and co-ordination were investigated during gait on a treadmill. No evidence was found at rest of EMG hyperactivity during muscle pain. But the static maximum voluntary contraction during muscle pain was significantly lower than the control condition in either tested muscle. During a static contraction at 80% of the pre-pain maximum voluntary contraction, muscle pain caused a significant reduction in endurance time. During dynamic contractions, muscle pain resulted in a significant decrease in EMG activity in the muscle that was agonistic to the painful muscle and a significant increase of the EMG activity of the muscle that was antagonistic to the painful muscle. So they concluded that muscle pain:

- *per se* causes no increase in EMG activity at rest,
- reduces maximal voluntary contraction,
- reduces endurance time (which was estimated by a linear regression technique) during submaximal contractions, and

- changes the co-ordination between the agonist and antagonist muscles during dynamic exercises.

The findings of this study are in accordance with the pain-adaptation model of Lund and co-workers 1991, which explains a link between activity in nociceptive afferents, a central pattern generator, and the motor function and co-ordination of the corresponding muscles.

The Pain-Adaptation Model proposes that the relative pattern of action of agonist and antagonist muscles is modified during pain. It is well established that jaw movements in chewing are generated by a central pattern generator in the brain stem. The pain adaptation model proposes that pain in skin, joints or muscles results in nociceptive information accessing the central pattern generator and, as a result, the motor command from the central pattern generator is altered. Thus, although there is a direct excitatory pathway to the α-motoneurones supplying the agonist (i.e. jaw-closing) muscles, there would be increased inhibitory firing of the excitatory interneurones and increased excitatory firing of the inhibitory interneurones to these agonist α-motoneurones. The net effect of this is to result in decreased activity in the agonist muscles. At the same time there would be an increased excitatory firing of the excitatory interneurones and a decreased inhibitory firing of the inhibitory interneurones to the α-motoneurones of the antagonist muscles (i.e. jaw openers; lateral pterygoid muscle, digastric muscle), thus resulting in an increased activity of the antagonist muscles. Thus, in pain, the increased antagonist activity and decreased agonist activity would lead to a reduction in MVC and a

reduction in the range and velocity of movement. This would help protect the injured part, avoid further damage and thus promote healing.

In 1998, Peter Svensson and co-workers conducted studies to describe the effects of experimental muscle pain on resting EMG activity in a jaw-closing muscle (masseter) and a leg muscle (tibialis anterior muscle). Ten healthy men who were not under any regular medication, in an age range of 21-26 volunteered for the study. Muscle pain was induced by intramuscular bolus injections of sterile hypertonic (5%) saline. Injections of sterile isotonic (0.9%) saline were used as control. All injections were given using a computer-controlled syringe pump. The pain intensity was scored on a 10-cm VAS, where 0 cm indicated "no pain" and 10 cm the "most intense pain". Both surface and intramuscular wire electromyograms were obtained from the resting muscles before, during and after saline injections. The EMG activity was analysed in 30-s intervals. In both muscles, a significant transient increase in EMG activity was noted 30-60 s after injection of hypertonic saline, but not after injection of isotonic saline. In contrast to the transient increase in EMG activity, the pain sensation lasted up to 600 s after the injection of hypertonic saline. So it was concluded that painful injections of hypertonic saline into the masseter and tibialis anterior muscles showed a transient increase in both intramuscular and surface EMG activity. However, the EMG increases were not correlated to the intensity or duration of pain and this suggests that human experimental muscle pain *per se* is unable to cause longer lasting muscle hyperactivity.

In summary, these human experimental pain studies show that muscle pain at a clinically relevant level and induced by infusion of hypertonic saline has little or no effect on postural EMG activity. They also reveal the limitations of surface EMG recordings used in most of these studies. A surface electrode picks up EMG activity from a wider area and hence doesn't give a reliable information on the action of a specific muscle for a specific task (Rugh *et al.* 1990).

Clinical Muscle Pain

There have been many studies indicating that there are no significant differences in postural activity between patients with TMD pain and control subjects. But recently, a few studies have showed results contrary to this.

Kapel and co-workers (1989) compared 20 TMD patients with myofascial pain to 20 non-pain control subjects who were matched with the TMD group for age and gender. Surface EMG data were recorded from the left and right frontalis, temporalis and masseter muscles. The results showed that the TMD patients had significantly elevated baseline EMG activity values for four of the six sites examined.

Glaros and co-workers (1997) tested the hypotheses that EMG activity at rest would be significantly greater for TMD patients with myofascial pain than for non-pain control subjects and that a cutoff score based on EMG values could be established to accurately separate the two groups. Fifty-four TMD patients diagnosed with myofascial pain and 54

non-pain control subjects who were matched with the TMD group for age and gender were included in this study. Silver-silver chloride surface electrodes were used to collect EMG data from right & left frontalis, temporalis and masseter muscles. Results showed that in all 6 sites, the EMG activity for the TMD group was higher than the corresponding activity for the non-pain group. But in only three sites (right frontalis, left temporalis and left masseter) however, were the differences of sufficient magnitude to be statistically significant.

There have been other studies also that have reported a small increase in postural EMG activity in TMD patients with pain (Rugh *et al.* 1987, Dolan *et al.* 1988) while some other studies even reported a small decrease in postural EMG activity (Paesani *et al.* 1994).

In his pain adaptation model article, Lund and co-workers (1991) concluded that the level of resting or postural muscle activity was no higher than normal in the five musculoskeletal pain conditions that he had described (TMD, muscle tension headache, fibromyalgia, chronic lower-back pain and post-exercise muscle soreness), with the occasional exception of the facial muscles. However, it is likely that this exception was unrelated to the source of pain and is simply a reflection of the changes in facial expression that pain often causes. So the specificity of these slightly increased surface EMG activities in the jaw-closing muscles has been questioned as it could be easily contaminated with cross-talk of EMG activity from mimetic muscles e.g. eye muscles or platysma.

In 1992, studies done by LeResche and co-workers on the effects of TMD-pain duration on facial expressions and verbal report of pain, also found that pain is reflected in the facial expressions and mimetic responses, which seems to support a potential contribution from mimetic muscles to the surface EMG recordings of jaw-closing muscles, Intramuscular EMG recordings might avoid the problem of EMG "cross talk" (Loeb and Gans, 1986; Svensson, 2001).

In summary, there is no convincing evidence in favour of an increase in postural EMG activity of the jaw-closing muscles in patients with craniofacial muscle pain. Specific studies with intramuscular EMG recordings have to be done to minimise the problem of EMG "cross talk" from facial muscles (Loeb and Gans, 1986; Svensson, 2001). However, even though there might be some increase in resting EMG activity, the absolute magnitude of such EMG changes and its pathophysiologic consequences and clinical relevance have yet to be studied in detail (Glaros *et al.*, 1997).

Electromyographic Activity During Movement

Experimental Muscle Pain

Of the many studies completed, Lund and co-workers (1991) studied the motor function at rest and during open-close movements of the mandible at a controlled rate in normal subjects before, during and after pain. In addition, resting EMG activity was recorded while the subjects remembered a previous painful experience (sham pain). Experimental

muscle pain was induced by the injection of 5% saline into the body of the left masseter muscle. After the first injection of 0.1 ml, the intensity of pain increased to an average of 5.5 on a VAS with endpoints labeled "no pain" and "pain as bad as it could be". Repeated injections of very small amounts of hypertonic saline were then begun to maintain a constant pain level. Electromyographic activity was mapped with surface electrodes placed over the masseter, temporalis and facial muscles on both sides of the head and needle electrodes were inserted near the site of infusion in some subjects. They concluded that:

- muscle pain was not a direct cause of any increased postural activity of the jaw-closing muscles; mean surface EMG activity was not significantly increased during pain in most subjects,
- surface EMG activity recorded over the frontalis region sometimes increased during pain and sham pain, but this was coincident with a change in facial expression,
- the velocity and amplitude of the repetitive opening movements decreased during pain,
- the area under the curve of the rectified and smoothed right and left masseter bursts was reduced during jaw closure in the presence of pain,
- some masseter motor units that were silent during jaw opening in the control periods became active during pain, while others increased their firing rate.

These findings were later confirmed by a series of studies by Svensson and co-workers in groups of healthy men exposed to hypertonic saline injection.

In 1995, Svensson and co-workers carried out experimental muscle pain studies on 10 healthy male subjects, who were not under any regular medications, to describe the sensory experience of saline-induced unilateral masseter muscle pain and the effect of this pain on jaw movements and jaw-muscle EMG activity during mastication. Experimental muscle pain was elicited by a bolus injection of 0.15 ml of 5% hypertonic saline into the human masseter muscle. The sensory experience was described using 10-cm VAS scores and McGill Pain Questionnaires (MPQ) in the 10 subjects. Thirteen other healthy male volunteers who were not under any regular medications participated in the study on interactions between jaw-muscle pain and mastication using kinematic recordings of the mandible and jaw-muscle electromyography. Jaw movement and EMG data were transformed into single masticatory cycles, which were averaged within subjects to produce mean masticatory cycles. Injection of 5% saline through normal skin and anaesthetised skin produced similar VAS profiles and MPQ features, with the pain consistently referring to adjacent areas. They found that:

- displacement of the mandible during painful mastication was significantly smaller in the vertical axis and in the lateral axis as compared to pre-pain values,
- the mean opening and closing velocities of the mandible were significantly reduced and the cumulated distance (the perimeter of the envelope described by the recording point (the magnet) during the mean masticatory cycle) of the jaw movement was also significantly smaller during pain,

- the agonist EMG activity during pain was significantly lower in the ipsilateral masseter muscle compared with pre-pain root-mean-square (RMS) values.

Thus painful masticatory movements have smaller amplitudes and are slower, which most likely represents a functional adaptation to experimental jaw-muscle pain (Svensson 1995). This study provided the first human data to show modulation of mastication in the presence of experimental jaw-muscle pain.

In 1997, Svensson and co-workers performed similar studies to determine the effect of bilateral experimental muscle pain on human masticatory patterns. Jaw movements and EMG recordings of the jaw-closing muscles were divided into multiple single masticatory cycles and analyzed on a cycle-by-cycle basis. Furthermore, non-painful injections of isotonic saline were included in this study as controls. Ten healthy men volunteered for the study. Bipolar surface silver electrodes were placed 10 mm apart over the central part of the masseter and anterior temporalis muscles. Sterile hypertonic saline 0.2 ml (5%) was injected over 15 s into the mid-portion of the left and right masseter muscles. On a separate day 0.2 ml isotonic saline (0.9%) was injected into the masseter muscle on one side and the effect on the masticatory patterns was examined while the subject chewed one piece of gum with three orthodontic elastics. The pain intensity was rated by the subjects on a 10-cm VAS, where the left extreme denoted "no pain" and the right extreme "worst imaginable pain". They found out that:

- injection of isotonic saline into the masseter muscle was associated with no or very low pain scores whereas bilateral injections of hypertonic saline caused

strong pain; nine subjects reported a sensation of decreased masticatory ability during pain,

- during painful mastication, the mean EMG activity of all the jaw-closing muscles in the agonist phase was significantly decreased and the mean EMG activity of all the jaw-closing muscles in the antagonist phase was significantly increased as compared with pain-free mastication,
- injection of isotonic saline caused no significant changes in EMG activity or jaw movements.

From this study it was concluded that the reduction of agonist EMG activity seems to be specific to pain, because injection of isotonic saline did not cause any significant changes, and the intense bilateral pain caused a significant increase in EMG activity of the jaw-closing muscles in the antagonist phase. This corresponds to the principle of co-contraction of jaw muscles during mastication which has been described in patients with painful TMDs by Møller and co-workers (1984) and Stohler and co-workers (1988) and could be interpreted as an effect of the pain rather than a cause of the pain.

In 1998, Svensson and co-workers conducted further studies to determine the effect of constant experimental muscle pain on jaw motor function. The motor tasks performed were mastication both ipsilateral and contralateral to the infusion side. The masticatory patterns were analysed on a cycle-by-cycle level and the influence of muscle pain on isometric jaw motor function was also studied. Twelve healthy men (age range of 18-31yr) who were not under any regular medication participated in the study. Self-fabricated bipolar surface silver electrodes were used for EMG recordings from right and

left masseter and anterior temporalis muscles. Pain was induced in the masseter muscle by tonic infusion of hypertonic saline (5%) for up to 800 seconds. Subjects continuously scored the pain intensity on a 10-cm VAS. A VAS between 3 and 5 was the target level as this is comparable to the pain levels reported by a majority of TMD patients (Svensson, 1998). Mastication ipsilateral and contralateral to the infusion side was quantitatively assessed with the use of jaw-tracking and EMG recordings of jaw-closing muscles before, during and after periods of constant muscle-pain intensity. They found that:

- for ipsilateral painful mastication, a significant reduction of jaw-closing EMG activity in the agonist phase was detected in most masticatory cycles, compared with the pre-pain and post-pain masticatory cycles,
- a smaller number of masticatory cycles with significantly reduced EMG activity was found for contralateral painful mastication, and
- the maximum voluntary occlusal force (MVOF) was significantly lower during painful clenches as compared to pre-pain and post-pain clenches.

This study showed that experimental jaw muscle pain induced by tonic infusion of hypertonic saline caused a diminished capacity of the jaw-closing muscles to work against a load, which is in accordance with a functional adaptation to muscle pain. The functional significance of this could be to restrict movement and to allow healing of an injured area.

Sessle (1999) hypothesised that these responses in both jaw opening and closing muscles can be interpreted as a "splinting" reaction, with the physiological purpose to limit jaw movements and allow rapid healing. He explains these effects on the basis of peripheral and central sensitisation. Acute injury or inflammation of TMJ or muscular tissues is likely to be associated with a peripheral sensitisation process. There are numerous free nerve endings in peripheral tissues, and these provide the peripheral basis for pain. They are activated by noxious stimulation by agents like heavy pressure, algesic chemicals and inflammatory agents, which induce impulses in the small-diameter (A-delta or C) afferent fibres with which they are associated. This neural information is conducted along nociceptive afferent fibres into the CNS, where it is processed so that the location, quality, intensity and duration of the noxious stimulus can be perceived. The nociceptors are also easily sensitised and an increased excitability of peripheral nociceptive afferents could thus account for the pain and tenderness of deep tissues when they are injured or inflammed. But central neural changes may also contribute. Sessle (1999) provides evidence that the neuronal receptive field expansion, induced by deep nociceptive afferent inputs, is one feature of central sensitisation or neuroplasticity. In addition, these receptive field changes may be accompanied by an increased responsiveness of the nociceptive neurones and a lowering of their threshold for activation by peripheral stimuli. These are thought to contribute to the tenderness, hyperalgesia and allodynia of superficial as well as deep tissues in the craniofacial region. Thus the central sensitisation process induced in the trigeminal brain-stem complex by deep nociceptive afferent inputs is also associated with increased activity in jaw-opening and jaw-closing muscles in animals. He suggests that these responses in both jaw-opening and jaw-closing muscles

can be interpreted as a "splinting" effect that counteracts excessive movement and so protects the articular or muscular tissues from further damage with the physiological purpose to limit jaw movements and allow rapid healing.

In summary, these human experimental pain studies on the effects of hypertonic saline injection into the masseter muscle have consistently shown a decrease in agonist EMG activity and a small increase in antagonist EMG activity and that the displacement of the mandible during painful mastication was significantly smaller. These studies also show that the mean opening and closing velocities of the mandible during painful mastication were significantly reduced.

Clinical Muscle Pain

There have been few studies of the effects of clinical muscle pain on EMG activity during jaw movements.

A study has been done by Mongini and co-workers (1989) on habitual mastication in TMDs. This study assessed mandibular movements and masseter EMG activity during habitual mastication in a group of patients with TMD and compared the data with those obtained from 12 normal subjects. A total of 86 TMD patients (18 men and 68 women) with main complaints of craniofacial pain, headache, TMJ clicking and restriction of mandibular movement took part in the study. All patients were asked to sit on a wooden chair in an upright position. They were given a predetermined amount of "crisp-bread"

and were asked to place it on their tongues and bring the mandible into the maximum intercuspal position. Then on a signal from the computer they started to chew normally and data was collected for 15 s. Three tests were performed for each subject. The Dysfunction index system was used to build two subgroups within the patient group: patients with severe TMJ impairment (n=21) and patients with severe muscle dysfunction (n=15). Silver-sliver chloride disk electrodes were used to collect EMG data from masseter muscles. The movement of the mid-incisor region was recorded with a Sirognathograph machine during chewing. Data on the different parameters of mastication were obtained for the individual patients, and the whole group and the two subgroups and were statistically compared together and with those from the normal subject data. They also investigated whether different dysfunction patterns could influence the masticatory performance in specific ways. Their findings were:

- in TMD patients, the normal symmetrical and balanced distribution of the chewing cycles is lost and movements were more restricted,
- there was a marked difference in the mean number of chewing cycles (% of preferred side chewing cycles ± S.D.) performed in preferred side of mastication in the normal group (57.7 ± 6), whole group (74.72 ± 14.9), muscle dysfunction group (73.7 ± 12.3) and TMJ impairment group (82.1 ± 12.4),
- in TMJ-impaired patients, movements were often repetitive and markedly deviated towards the affected side and a generalised restriction of movements was observed, and

- the EMG data showed marked alterations with an increase of masseter muscle activity during opening in some patients and an irregular distribution during closing.

Feine and co-workers (1988) evaluated the criteria used to diagnose mandibular dysfunction with a mandibular kinesiograph. Ten asymptomatic subjects and seven symptomatic subjects with reported frequent pain in the masticatory muscles during normal activity participated for the study. Kinesiographic recordings of mandibular movements were done according to the manufacturers' instructions. All the participants were asked to perform 3 tasks:

Task I

- Task Ia was designed to allow a measurement of vertical displacement, which is the distance between the opening and closing paths. Instructions were "Close your teeth together. Now open and close just once and stay closed".
- Task Ib was designed to allow a measurement of velocity. Subjects were instructed to "Open and close wide and fast".

Task II Closure from rest position to maximal intercuspation by instructing "let the jaw hang, relax and keep it steady then close the teeth together".

Task III Chewing movements. Subjects chewed one stick of gum for 20 s.

They concluded that the mandibular movements of symptomatic subjects could not be differentiated from those of the normal subjects by kinesiographic recordings. But they

reported that the average and maximum opening velocities of the mandible were lower in patients with persistent jaw muscle pain than in matched control subjects. Mean opening and closing velocities of the normal group ranged from 95 mm/s to 285 mm/s, whereas for the symptomatic group, it ranged from 89 mm/s to 191 mm/s. But they found no differences in the maximum displacement during empty open-close movements.

Møller and co-workers (1984) found that TMD-pain patients used their jaw-closing muscles significantly less during the agonist phase; but they found that the relative contraction strength was higher, most likely because of a decreased maximum voluntary occlusal force as well. They also found that TMD pain patients have a significantly longer duration of the masticatory cycle, i.e., they chew more slowly than control subjects.

Studies done by Stohler and co-workers (1985, 1988) found increased EMG activity of the jaw-closing muscles in the antagonist phase during painful mastication. These findings were in accordance with the findings of Møller and co-workers (1984).

Nielsen and co-workers (1990) conducted studies to determine the pattern of mandibular movement in TMDs. Twenty-four normal subjects (age range: 18-35 yr.) with no past history or present symptoms of TMD, with "normal facial morphology" (Nielsen *et al,* 1990) and without extreme malocclusions and 26 adult patients with muscle pain or tenderness to palpation participated for the study. Jaw-tracking was done by tracking the mandibular incisor position in three planes with either a Myotronic Kinesiograph or a

Siemens Sirognathograph instrument, while the subject was seated in an upright position. Mandibular movements in sagittal, frontal and horizontal planes were recorded while the subjects performed speech and mastication. To obtain the speech pattern, subjects were asked to say three to four complete sentences and mastication was recorded while the subjects were chewing peanuts. They observed that in normal subjects, the range of rest position was 1 to 5 mm in relation to the intercuspal position; the range of maximum excursion during speech was 30% to 36% of maximum opening; the vertical extent of excursion during mastication was 18% to 90% of the maximum vertical opening dependent on the bolus. Protrusive movements were straight forward, dividing the angle evenly between left and right laterotrusion. Laterotrusive movements were of equal length and similar to the length of protrusion. The TMD patient group with muscle pain demonstrated asymmetry in the length of laterotrusive movements. The difference between the left and right laterotrusion for each subject in the normal group was less than 2 mm whereas in the patient group a difference of 3 to 5 mm was noted. On laterotrusion, different pathways were noted when the mandible moved away from the intercuspal position and returned to this position. The extent of mandibular movement during speech and mastication were similar to that of the controls.

Kumai (1993) performed studies to determine the difference in chewing patterns between symptomatic and opposite sides in patients with unilateral TMJ and myofascial pain-dysfunction. Thirty patients (age: 12-74 yr) with unilateral TMJ and myofascial pain-dysfunction and 20 control subjects (age: 19-27 yr) participated in this study. Surface EMG was recorded bilaterally using bipolar disc electrodes over the anterior temporalis

muscle and the middle portion of the masseter, while the subjects performed tasks of maximal clenching and gum and peanut chewing. The gum-chewing integrated EMGs were transformed to a differential EMG figure to facilitate comparison of mastication on the dysfunctional side and opposite side. Variables, mainly in the gum-chewing EMG data, were statistically analysed, and were compared with those of normal subjects. The patients were classified into three types.

1. Same-side patients who chewed peanuts on the dysfunctional side.
2. Opposite-side patients who chewed peanuts by avoiding the dysfunctional side and showed excellent chewing activity on the side opposite the dysfunction.
3. Intermediate side patients who had a chewing pattern that was a combination of the first two types.

The gum-chewing EMG of normal subjects was found to be roughly symmetrical between the right and left side. Peanut EMGs in the normal subjects indicated that 60% of the subjects chewed food alternately on both sides (Kumai, 1993). The patterns of gum-chewing EMG in the TMD patients were mostly asymmetrical and quite different from those in the normal subjects and showed variation between patients and only a few ($n = 7$) chewed peanuts alternately on both sides.

Kumai (1993) found that in normal subjects, the EMG during maximal clenching in the intercuspal position demonstrated that electrical muscle activity was balanced between the paired temporalis and masseter muscles of both sides. But in the TMD patients, the rate of unilateral chewing increased and the activity in clenching was not balanced

between the paired muscles. Hence, he concluded that the TMD pain patients might avoid the dysfunctional side when chewing food and the muscle activity on that side might be lower.

Lund and colleagues (1991) drew attention to the fact that comparable findings with slower movements and less EMG activity in the agonist phase and more EMG activity in the antagonist phase could also be observed during other dynamic motor tasks, such as gait, and in other musculoskeletal pain conditions such as fibromyalgia, chronic lower back pain and muscle-tension headache. These observations are in accordance with the pain-adaptation model.

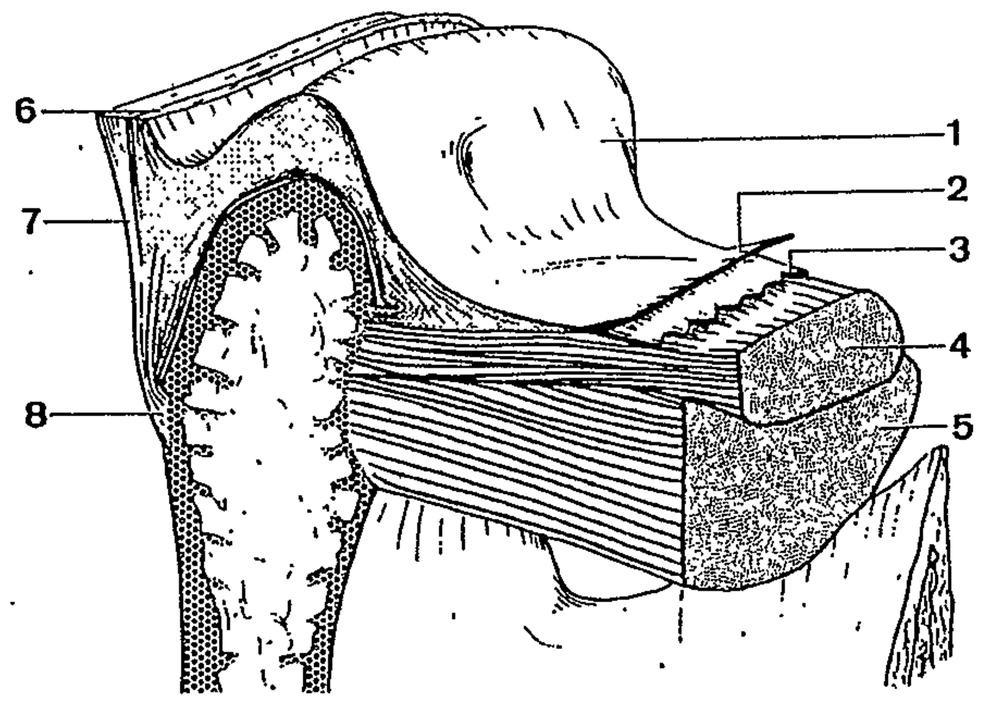
In summary, all of these previous studies show that there is a difference in EMG activity of the agonist and antagonist muscles in TMD patients with pain during movement, when compared with the asymptomatic control subjects. Most studies also show a difference in rate, symmetry and range of movement of mandible in TMD patients with pain. The findings from these experimental and clinical pain studies, suggest that dynamic motor function is changed by the presence of pain, and suggest that changed motor function is not the cause of pain.

In many of the previous studies, the jaw movements were not standardised. We propose that it is important to standardise jaw movements, so that we would be in a better position to understand the normal function of muscles and their function in patients with TMD. This is because in a study where the jaw movement rate and amplitude are standardised, specific EMG features (e.g. the single motor unit (SMU) task relations and firing

properties) can be related to defined dynamic features of jaw movement. This standardised methodology also gives a reliable method for performing the same tasks by different individuals and thus this facilitates comparing the same tasks done by asymptomatic subjects with those performed by symptomatic TMD patients with pain. This will provide information on the effects of chronic pain on motor function in TMDs.

The first aim of this study therefore was to characterize jaw motor function in TMD patients performing a standardised task.

The human lateral pterygoid muscle (LP) has been often implicated as playing an important role in the control of jaw movements and, by virtue of its direct insertion into the condyle and disk-capsule complex of the TMJ, in the control of TMJ function (Dubner *et al.*1978; Wilkinson 1988). The LP consists of two heads or bellies, an upper or superior head (SHLP) and a lower or inferior head (IHLP). The SHLP originates from the roof of infratemporal fossa and is inserted into the condyle and the disk-capsule complex of the TMJ, whereas the IHLP arises from the lateral surface of the lateral pterygoid plate and is inserted into the condylar neck of the mandible and the capsule (Wilkinson *et al.* 1989; Meyenberg *et al.* 1986; Heylings *et al.* 1995).



Left TMJ, medial sectional view sectioned between medial and middle third.

1. Disc
2. Attachment to the articular eminence
3. Fascia
4. SHLP
5. IHLP
6. Attachment to the petrotympanic fissure
7. Capsule, posterior wall
8. Posterior condylar attachment

Fig 1. Shows the insertion of IHLP and SHLP and its relation to the TMJ.

(Meyenberg *et al.* 1986)

In general terms, many EMG studies suggest that the IHLP plays a role in opening, protrusion and contralateral jaw movements, the SHLP plays a role in closing, retrusion, and ipsilateral jaw movements, and that there is a reciprocal relationship between the activity of SHLP and IHLP (Hannam and McMillan 1994; Hiraba *et al.* 1995 & 2000; Klineberg 1991; Miller 1991).

There is a widely accepted clinical opinion that the motor function of LP is altered in TMD patients (Okeson 1998; Klineberg 1991). This is because of the anatomical relation of the LP muscle to the TMJ. Theories of LP disturbance include muscle hyperactivity, muscle hypoactivity, poor co-ordination between the two heads of the muscle, and/or a

disturbance to the normal role of the muscle in the control or stabilization of the TMJ (Murray *et al*, 2001). But though all these theories are still unsubstantiated, some treatments rendered to TMD patients such as irreversible therapies like occlusal grinding and restorative treatments, as well as reversible therapies using occlusal splint or jaw exercises, are based on these concepts. Hence it is important to study the function of the LP in TMD patients.

Until recently, there was very little information on the normal function of the LP. In recent studies, Murray and co-workers (2001) have provided new evidence showing that one of the major functions of LP is in the fine control of horizontal jaw movements. In a study on the functional properties of single motor units (SMUs) in IHLP, Phanachet and co-workers (2001) studied the activities of 99 SMUs recorded from IHLP. All 99 SMUs were active during contralateral jaw movements with the teeth apart, and protrusive jaw movements with the teeth apart, and 81% were active during submaximal jaw-opening movements. None were active on maximal ipsilateral or retrusive jaw movements with the teeth apart or on jaw-closing / clenching in intercuspal position. Thresholds of SMUs ranged from <0.2 mm of contralateral or protrusive horizontal displacements to 61-89% of the maximum contralateral or protrusive displacement, respectively. Recruitment thresholds (mm) of some of the units were rate dependent with thresholds significantly decreasing with increasing rate of horizontal jaw movement in protrusion and contralateral movements. Then after dividing IHLP into four regions, the SMUs recorded in the superior-medial zone exhibited significantly lower mean threshold values than for the SMUs recorded in the other zones (no units were recorded in the inferior-lateral

zone). Thus this study has given preliminary evidence on functional heterogeneity with in IHLP. Taken together, these data suggest that specific regions of the IHLP are capable of selective activation in a finely controlled manner to allow the application of the appropriate force vector (magnitude and direction) to effect the required condylar movement needed for the generation and control of horizontal jaw movements. They also found that none of the SMUs were spontaneously active at postural jaw position.

With these new information we are now well placed to study the activity of the LP in TMD patients. So the second aim of this study was to characterise the EMG activity of the LP muscle in TMD patients to determine whether there is any evidence supporting the notions of hyperactivity or lack of coordination within the LP.

AIMS

The aim of this study is:

(1) to characterise jaw motor function in TMD patients performing a standardised task, and

(2) to characterise the EMG activity of the lateral pterygoid muscle in TMD patients to determine whether there was any evidence supporting the notions of hyperactivity or lack of coordination within the LP that have been pointed.

MATERIALS & METHODS

Six TMD patients presenting at the Orofacial Pain Clinic, Westmead Center for Oral Health, Westmead Hospital, volunteered for the studies. They had signs and symptoms of TMJ pain, muscle pain and/or limited mandibular movement (see Table 1). The ages ranged from 31 to 52 yr and there were 3 males and 3 females. All participants were given a good briefing of the experiment and a detailed history and clinical examination was done at the start of the study. All participants gave informed consent, and the Western Sydney Area Health Service Ethics Committee of Westmead Hospital and Human Ethics Committee of the University of Sydney approved experimental procedures.

In summary, electromyographic recordings were made from the IHLP, masseter and anterior digastric muscles and recording of the movement of the mid-incisor point (MIPT, the point between the incisal edges of the lower central incisor teeth) and palpated lateral condylar pole were made with the JAWS3D tracking system. Jaw movements consisted of standardised trials of contralateral and protrusive movements. These data were then compared qualitatively with the data already recorded from six asymptomatic subjects. All six volunteers (age 21 – 41 yr; 3 males, 3 females) were without any signs and symptoms of TMD and without any history of chronic pain or neuromuscular condition.

Subjects	TMD-1	TMD-2	TMD-3	TMD-4	TMD-5	TMD-6
Accident	Extraction	Jaw Stretch	Motor Vehicle Accident	No	No	Diving Accident
Gender	M	F	M	F	F	M
Age	31	46	43	52	49	41
Duration of Symptoms	13 months	7 yr	6 yr	10 yr	18 yr	25 yr
Pain - Continuous	Y	Y	Y	Y	Y	Y
Joint Pain	Y	Y	Y	Y	Y	Y
Joint L / R	L	L	R	L	L	L
Muscle Pain	Y	Y	No	Y	Y	Y
Muscle L/R	L	L	No	L	L - R	L
Click	Y	Y	Y	Y	Y	Y
Click L/R	L	L	R	R	L - R	L – R
Click Position on opening	35 mm	25 mm	5 mm	5mm	5 mm	5 mm
Crepitus	Y	N	Y	Y	No	No
Crepitus L/R	L	N	R	L	No	No
Dentate	Y	Y	Y	Y	Y	Y
Missing Posterior Support	No	No	Y	No	Y	No
Missing Teeth	No	Y	Y	Y	Y	Y
Occlusion	Cl 1	Cl 1	Cl 3	Cl 3	Cl 2	Cl 3
Max. Opening	45 mm	40 mm	40 mm	28 mm	46 mm	55 mm
Laterotrusion - L	5 mm	10 mm	6 mm	8 mm	8 mm	10 mm
Laterotrusion - R	5 mm	10 mm	10 mm	5 mm	8 mm	10 mm
Protrusion	5 mm	10 mm	5 mm	5 mm	8 mm	10 mm
Diagnosis						
Chronic Joint	Y	Y	Y	Y	Y	Y
Chronic Muscle	Y	No	No	Y	Y	No

Table 1. The clinical signs and symptoms of TMD patients who volunteered for this study. RDC/TMD not followed and so was difficult to explain whether the TMD was of arthrogenous, myogenous or arthrogenous/myogenous origin.

Recording of condylar and MIPT movement during standardised movements

Condylar and MIPT movements were recorded with an optoelectronic jaw tracking system (JAWS3D, Metropoly AG, Zurich, Switzerland, sampling rate = 67/s). Upper and lower impressions were taken on the first visit and custom-made maxillary and mandibular metal clutches were fabricated for the individual subjects.

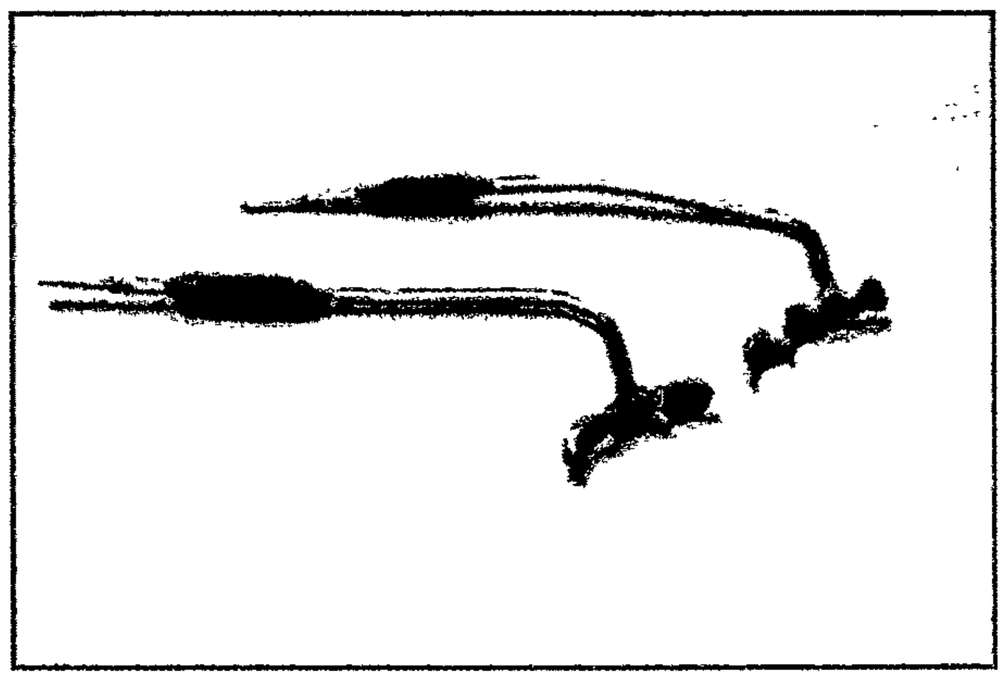


Fig 2. The custom made maxillary and mandibular clutches.

These clutches were glued to the maxillary and mandibular teeth during the experiment using super glue instant (Final Touch Products). One lightweight target frame, containing three light-emitting diodes (LEDs) arranged in a triangle, was attached to the distal ends of these clutches. The plane of each target frame was oriented parallel to the sagittal plane, and the longer arm of each target frame was oriented parallel with the Frankfort-Horizontal plane.

During the recordings, the participants sat in an upright position without head support. The MIPT and the palpated lateral condylar pole were selected as reference points. However, only the position of the subject's MIPT in the horizontal plane was displayed as a dot (termed MIPT dot) on the video screen positioned in front of the subject. The JAWS3D tracking system recorded the motion of the target frames and then, off-line, could also calculate the motion of any point subsequently selected. All jaw movements were performed with the teeth apart, and movements started from the postural jaw position. Subjects were instructed to swallow and relax their jaws with their lips lightly touching to achieve the postural jaw position (PJP).

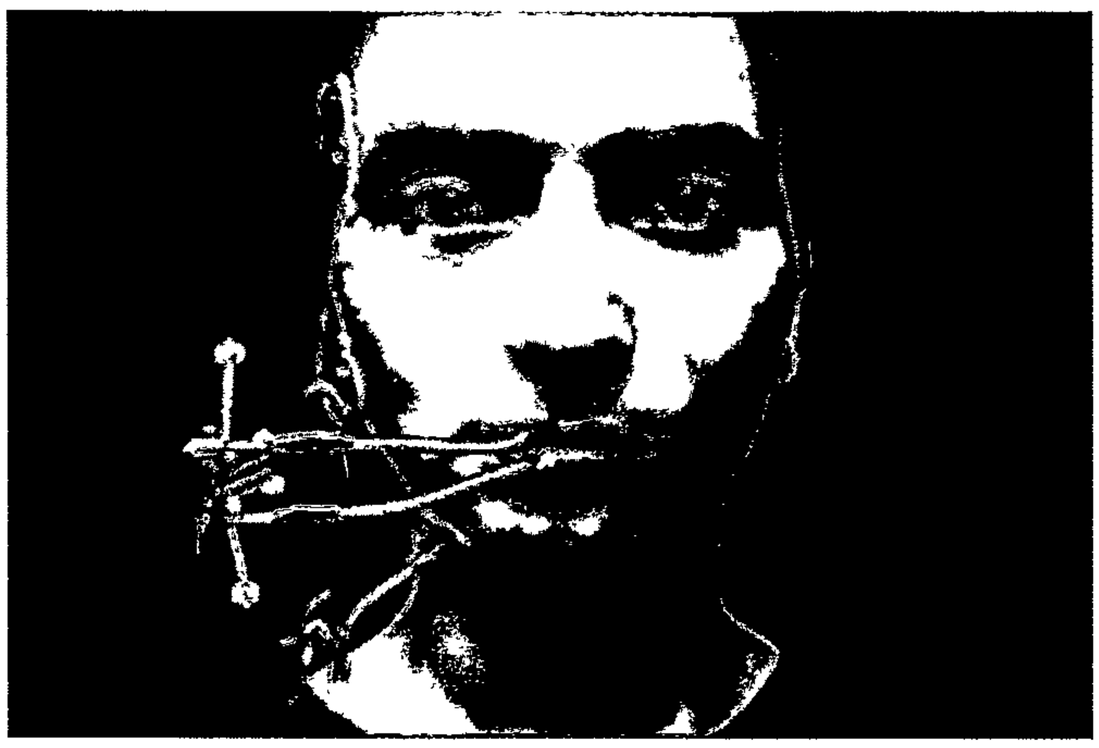


Fig 3. The participant seated in an upright position without head support and the clutches glued to the maxillary and mandibular teeth.

Jaw movements were standardised by having the participant move the position of the MIPT dot, so as to track a computer-controlled target. The target in these standardised tasks was an illuminated LED as part of a linear bank of 15 LEDs positioned over the video screen and to the side of the trajectory of the MIPT dot. The trajectory of the MIPT

dot in the horizontal plane was displayed on the video screen. The LEDs were controlled by scripts written in Spike2 software (Cambridge Electronic Design, Cambridge, UK, CED) and run on the CED system that was also used to record EMG. Only one LED was illuminated at any one time.

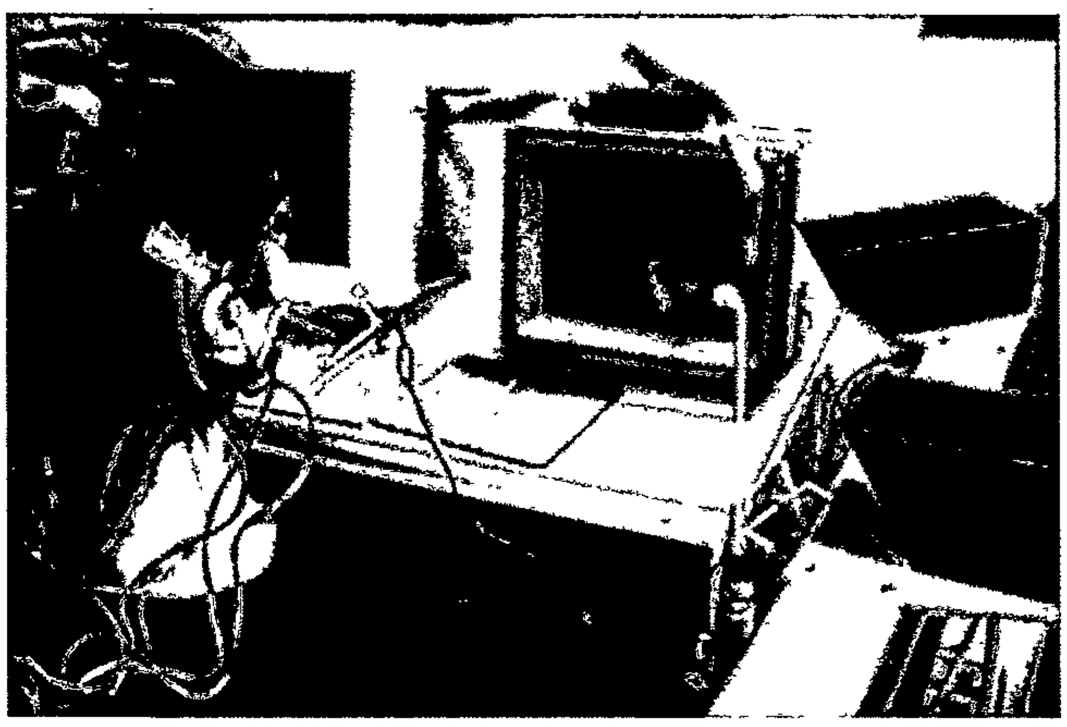


Fig 4. The Jaws3D cameras on the side and the video screen with the LED bank positioned in front of the subject.

For ease of viewing small displacements, the display on the computer screen was magnified 3X, 6X or 9X accordingly. The participant was instructed to perform a few trials of lateral or protrusive jaw movement to get accustomed to the task. The linear bank of LEDs was then oriented along the direction of movement in the horizontal plane that the MIPT dot tracked during jaw movement. The Spike2 software illuminated the LEDs in sequence and the participant was instructed to move the jaw so that the MIPT dot on the screen followed the illuminated LED as smoothly as possible. This program allowed adjustment to the rate of jaw movement by changing time-off duration between each

LED, and time-on duration of each LED, and also allowed control of the desired amount of displacement by varying the highest LED in the bank that was illuminated.

Each subject was asked to perform single step and multiple step displacements in protrusion and contralateral movements.

Single-step displacements

Each movement started with the jaw in postural position for 2 s. The participants were instructed to move the MIPT dot smoothly and track the target at the rate and magnitude of displacement controlled by the Spike2 software during protrusive and lateral excursions. Each participant was required to hold the MIPT dot as much as possible within the boundaries of the LED that was illuminated for the holding phase period of the step displacement. The jaw was then returned to the postural position again following the return targets, and this concluded the trial. The diameter of each LED was 2.8 mm. After the participant kept the jaw at postural position for 2 s, the first LED was turned off. After 200 ms the next LED was lit up for 100 ms. This cycle (200 ms off, next LED 100 ms on) was repeated until the jaw had displaced to a position requiring holding of the jaw for 5 s which was achieved by illuminating the assigned target LED for 5 s. Then the same cycle started again from the target LED back to the first LED. This entire sequence, termed as a trial, was repeated 3-6 times with a rest period of at least one minute between trials. During jaw movements, participants were required to track the target by moving the dot to any point within the diameter of an LED (e.g. centre of the boundary of the LED). Each subject was required to hold the MIPT dot as much as possible within the boundaries of the LED that was illuminated for the holding phase period of each step

displacement. For trials at slower rates of movement, the light-off duration was changed from 200 to 600 ms or 1 s. These durations corresponded with rates of movement of 6.5, 2.2 or 1.3 mm/s, respectively. The 200 ms light off duration trial was termed fast speed, 600 ms termed intermediate speed and the 1 s one as slow speed. The fastest rate of movement was about the fastest that our subjects found comfortable to perform. This range of rates of movements therefore provided a wide range of rates of displacement that was suitable for characterising associated SMU firing properties. A range of target displacements from 0.65 - 12.0 mm were possible with this methodology. The latter represented about the maximum displacement possible. Even though recordings at fast, intermediate and slow speeds were done for all subjects, only intermediate speed data were analysed for this study.

Multiple-step displacements

Participants also performed lateral and protrusive jaw movements in a step-like manner to several intermediate holding displacements and these were three-step movements and two-step movements. To achieve this, the participant tracked the target and moved the jaw to the first assigned target and maintained the jaw in that position for 3-5 s. The jaw was then moved a further small amount forward or laterally to the next assigned target and again maintained in that position for another 3-5 s. The participant then moved the jaw to the final target LED for another 3-5 s. As with the single-step displacement, each subject was required to hold the MIPT dot as much as possible within the boundaries of the LED that was illuminated for each holding phase period. To conclude the trial, the

jaw was moved back to the postural position by following a pre-set target LED sequence. In TMD participants, only two-step displacements were performed as they found it uncomfortable and painful to do three-step movements and to hold the jaw in position for that length of time. Trials for different rates of movement (6.5. 2.2 and 1.3mm/s) were repeated 3-6 times. The total experimental recording period lasted 3-4 hrs for each subject. After each trial, the participant was asked to indicate their pain level felt by them on a 100mm visual analogue scale (VAS) where, 0 mm represents "no pain" and 100 mm the "worst possible pain".

There were rest periods of 30s to 1min between each trials. But it is possible that there was some muscle fatigue. Muscle fatigue was not quantified in this study and has to be taken into consideration in future studies.

Electromyographic Recordings

EMG activity of IHLP was recorded with bipolar Teflon-coated stainless-steel wire electrodes. An intraoral approach was used for IHLP electrode placement. The method for electrode placement within IHLP (modified from Wood *et al.*, 1986) involved inserting a sterilized, pre-curved needle containing two Teflon-coated, stainless-steel fine wires through the oral mucosa above the level of the upper second molar tooth.

Fig 5. The intraoral approach used for IHLP electrode placement.

The wires were cut with sterile scissors immediately prior to placement to provide fresh cut-wire ends for recording. The needle was advanced to contact the lateral surface of the lateral pterygoid plate. The needle was then withdrawn, leaving the wires within the IHLP, and the wires were secured to the buccal surface of the upper first molar tooth with a small piece of Stomahesive wafer (ConvaTec, Victoria, Australia) and led out through the angle of the mouth. Electrode location in IHLP was verified after the recording by computer tomography (CT scan). At the end of each recording session, five to nine CT-axial slices (1-3 mm thick) were taken inferior to and parallel with the clinically approximated Frankfort horizontal plane. The Frankfort horizontal plane is the plane of best fit to four points on the skull: the lowermost border of the infraorbital rim bilaterally and the uppermost border of the bony external auditory meatus bilaterally. These CT scan data confirmed electrode location within the IHLP and showed the location within the muscle relative to the boundary of the IHLP.

Electromyographic activities of masseter and anterior digastric muscles were recorded with Duo-Trode silver/silver chloride surface electrodes, which were positioned on

clinical examination over the body of these muscles on the same side of IHLP recording. The surface of the skin was cleansed with 70% v/v isopropyl alcohol sterile swabs before the electrodes were placed.

The data-acquisition equipment was the micro1401 from Cambridge Electronic Design (Cambridge, UK) and the sampling rate was 10,000 samples/s, and the bandwidth 100 Hz to 10 KHz.

Data Analysis

The mean and standard deviation (SD) of every 50^{th} data point of MIPT movement was calculated for all subjects on each trial for each task performed to assess qualitatively the variability in the multiple trials done for the same task. The data were then transformed to coefficient of variance and this allowed a qualitative comparison for differences in jaw movement within and between groups. For each trial, the coefficient of variance was expressed as the standard deviation (SD) value divided by the corresponding mean value and was then expressed as a percentage. The coefficient of variance of similar tasks for each participant was calculated and plotted together for the two groups and compared.

The EMG recordings from the IHLP were filtered (using high pass filter), rectified and smoothed with a digital filter (Paynter filter; integration time constant, 50 ms). The filtered data was used later in qualitatively comparing the muscle activity along with the jaw tracking records.

RESULTS

Jaws3D Tracking Analysis

Qualitative Analysis

1. ***Comparison of stability of MIPT position at postural jaw position***

Figure 6 shows Jaws3D recordings of the postural jaw position of the six TMD patients (left panel) compared with the plots derived from two asymptomatic subjects; recordings of the postural jaw position were not made in the remaining four subjects. Even though recordings of postural jaw position were not performed in the four asymptomatic subjects, visual inspection during the experimental sessions showed no activity of the lateral pterygoid muscle at postural jaw position. Although there is no clear difference between TMD and asymptomatic subjects in the stability of the MIPT tracings, there is a suggestion that, compared to the two asymptomatic plots, one of the TMD plots (i.e. b) showed increased oscillatory displacement of the MIPT while at postural jaw position. The shift of the z-axis away from other two axes reflects the movement of the jaw to postural jaw position at the beginning of this trial in b.

Jaws3D recording of postural jaw position

TMD

Asymptomatic

a)

b)

c)

d)

e)

f)

g)

h)

2 mm

5 s

5 s

Fig 6 Jaws3D recordings of the postural jaw position of the six TMD patients and two asymptomatic subjects.

2. *Comparison of reproducibility of horizontal jaw tracking*

Graphical Display

In all the participants the MIPT target could be tracked during single and multiple-step displacements at different rates and magnitudes of movement. While all the asymptomatic participants fulfilled the criterion for single-step and multiple-step displacements during contralateral and protrusive jaw movements, some of the TMD patients were not able to perform all the required tasks. This could be because of the pain/discomfort they had or because of dysfunction or both. The plots derived from multiple trials of one task within a subject were compared to see the variability of jaw tracking in performing the same task. These plots were then compared with the corresponding plots for the same or similar tasks derived from asymptomatic subjects to see whether any clear differences in jaw tracking between the two groups were apparent.

A. Single-step contralateral movement.

An intermediate rate of single-step contralateral movement is shown in Figure 7. In each subject plots of the y-axis have been overlayed and compared with similar plots derived from the asymptomatic group. The horizontal line seen across each plot represents the position of the target during the holding phase.

Generally the asymptomatic group was more consistent in performing this task than the TMD group. One of the TMD patients (i.e. c) in particular was not able to perform the task as well as the other TMD or asymptomatic subjects in both the holding and moving phases, with for example, one trial moving in the wrong direction. The remaining trials overshot the target and one trial although achieving the target displacement, was not maintained at the target position for the required duration. Another TMD patient, (a), exhibited some variability in the outgoing phase of each trial. With the exception of these TMD patients (a and c), the other TMD patients appeared capable of performing the task in a manner similar to that performed by the asymptomatics.

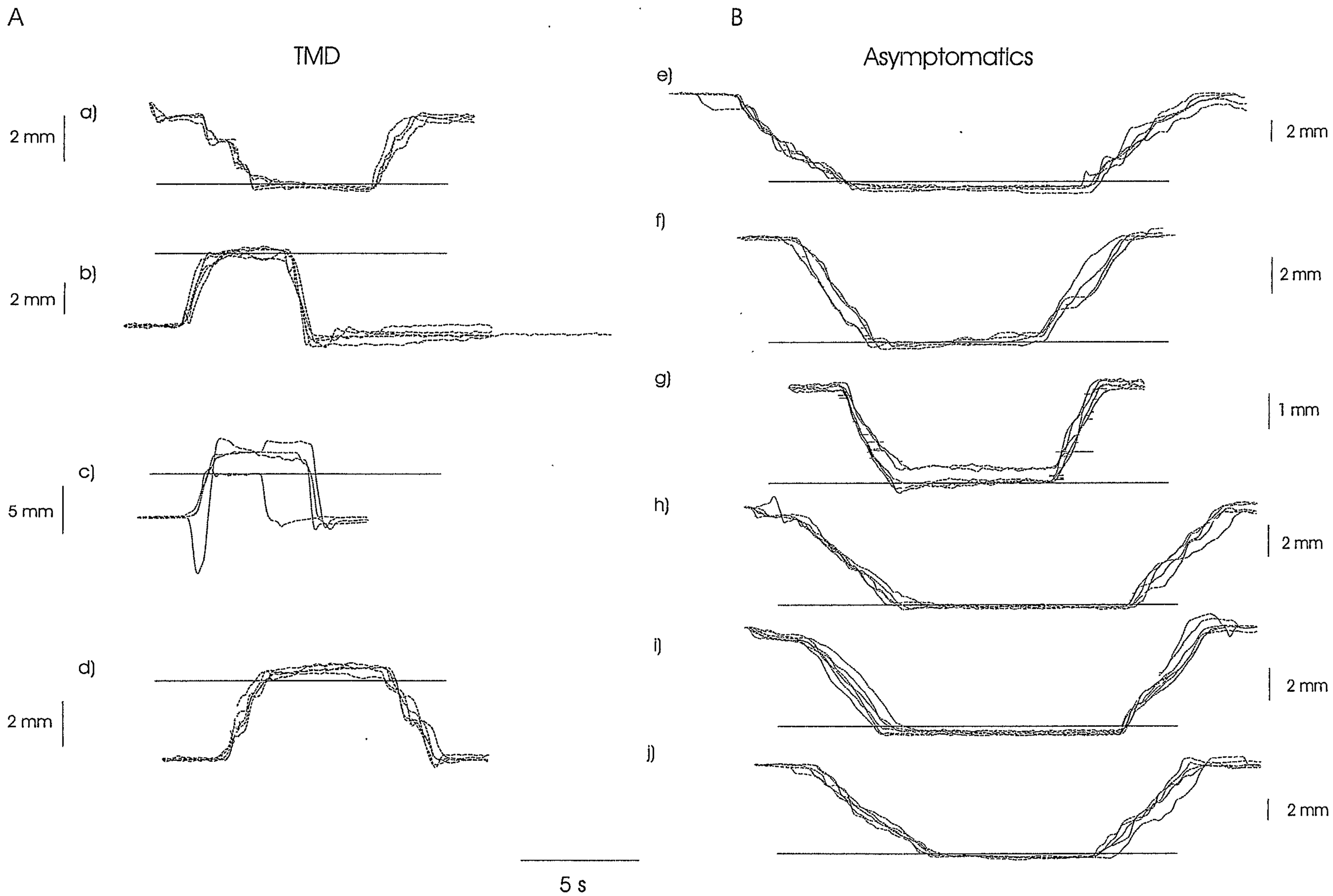


Fig 7 Jaws3D recordings displayed in y-axis (i.e. medio-lateral axis) of single-step contralateral movement performed by the TMD patients and the asymptomatic subjects.

B. Multiple-step contralateral movement

Unlike the asymptomatic group where all the participants performed a three-step displacement, all except one patient in the TMD group performed a two-step contralateral movement. This was because of the observation of their initial tracking suggested to us that TMD patients would not be able to perform the three-step movement.

Representative data from two-step and three-step contralateral tasks are shown in Figure 8A for each TMD patient and from three-step contralateral tasks for each asymptomatic subject are shown in Figure 8B. As seen in the plots, the TMD group showed more variability in the multiple trials than the asymptomatics. While all of the asymptomatics performed the three-step displacement tasks consistently without much variation, the MIPT tracings from most of the TMD subjects exhibited considerable variability from trial to trial. For example, the MIPT tracings from TMD patient (b) were very variable in both holding and dynamic phases, with one trial movement starting well ahead of the target onset and all trials overshooting the target. In another TMD patient, (d), almost all trials had a large variation on the return phase, with two trials returning too early. Most TMD patients showed a large variation both in the dynamic phases and holding phases. In general, the TMD group performed the tasks with much more variability than the asymptomatics.

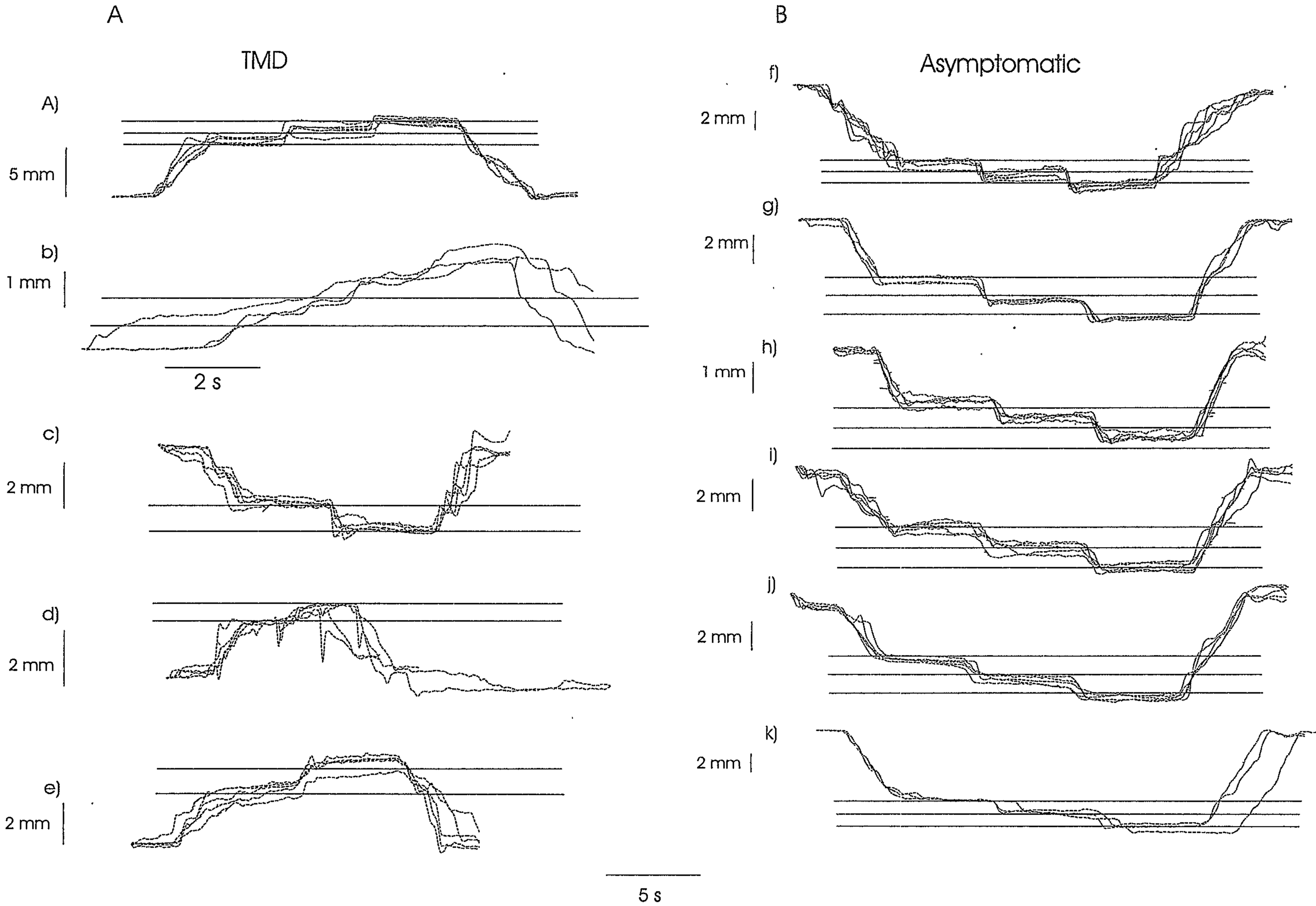


Fig 8 Jaws3D recordings in y-axis of multiple-step contralateral movements performed by the TMD patients and the asymptomatic subjects.

C. Single-step protrusion.

The tracings of MIPT movement during single-step protrusion from trial to trial for each of the TMD patients exhibited more variability qualitatively than the corresponding traces for the asymptomatic group (Figure 9). One of the TMD patients, (b), showed more variation in the dynamic return phase after the holding phase and did not return to the PJP baseline for the duration of the trace. Two trials fell short of the target. In another TMD patient (a) except for one trial all other trials overshot the target by several mm. Another patient (d) exhibited more variability with one trial starting ahead of the target and another one falling short of the target during the holding phase. The MIPT tracings from another subject (c) also showed considerable variation both during the holding and dynamic phases for all the trials performed. In general, the TMD group exhibited more variability within and between trials than the asymptomatic group. None of the TMD subjects showed a level of consistency in the dynamic or holding phases comparable to that seen for most of the asymptomatics. One of the asymptomatic subjects (i) showed more variability during the holding phase with few trials overshooting the target. But most of the asymptomatic subjects were able to perform well in the holding phase though there appeared to be more variability in the dynamic phase when compared to the contralateral task in some of the subjects.

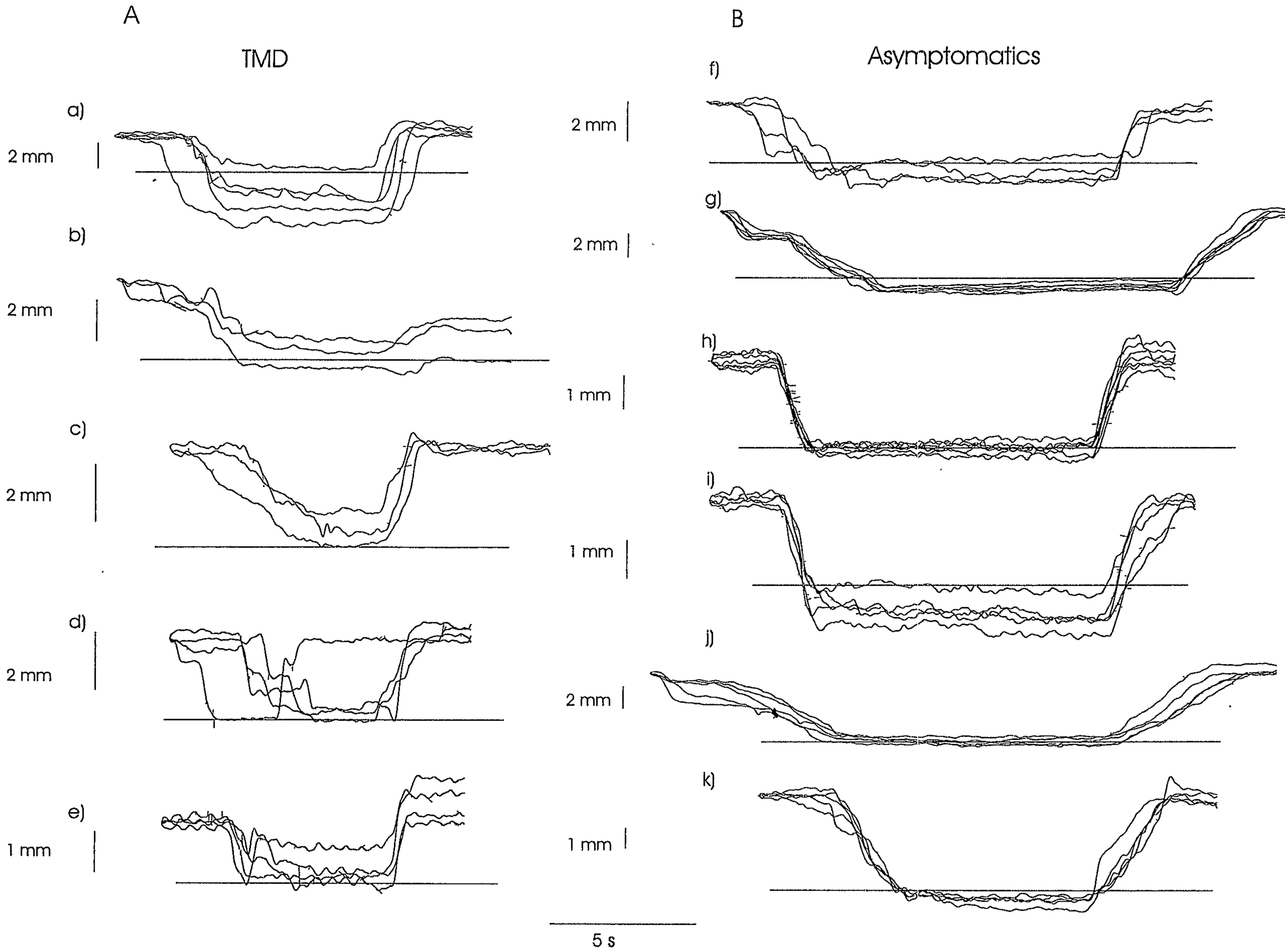


Fig 9 Jaws3D recordings of x-axis (i.e. Antero-posterior) for single-step protrusive movement performed by the TMD patients and the asymptomatic subjects.

D. Multiple-step protrusion

Representative data from two-step and one three-step protrusive tasks are shown in Figure 10A for each TMD patient and for three-step protrusive tasks for each asymptomatic subject in Figure 10B. The tracings of MIPT movement from trial to trial for each of the TMD patients exhibited more variability qualitatively than the corresponding traces for the asymptomatic group. For example, the MIPT tracings of one of the TMD patients (c) were variable in the dynamic phase after the holding phase in all the trials and with one trial even moving in the wrong direction. In another patient (d), in one trial, the initial MIPT movement occurred at the beginning of the trial and well ahead of the target.

As noticed in the plot, the MIPT tracking was highly variable in the TMD group than the asymptomatic group. The TMD patients had more variability both in moving the MIPT dot in a step-by-step manner along with the illuminated LEDs and also in holding the MIPT dot as much as possible within the boundaries of the illuminated LED for the holding phase period and finally to return to the postural position. The variability in the holding phase is illustrated very clearly in patient (b). This variability was noticed in all of the TMD patients except one (a).

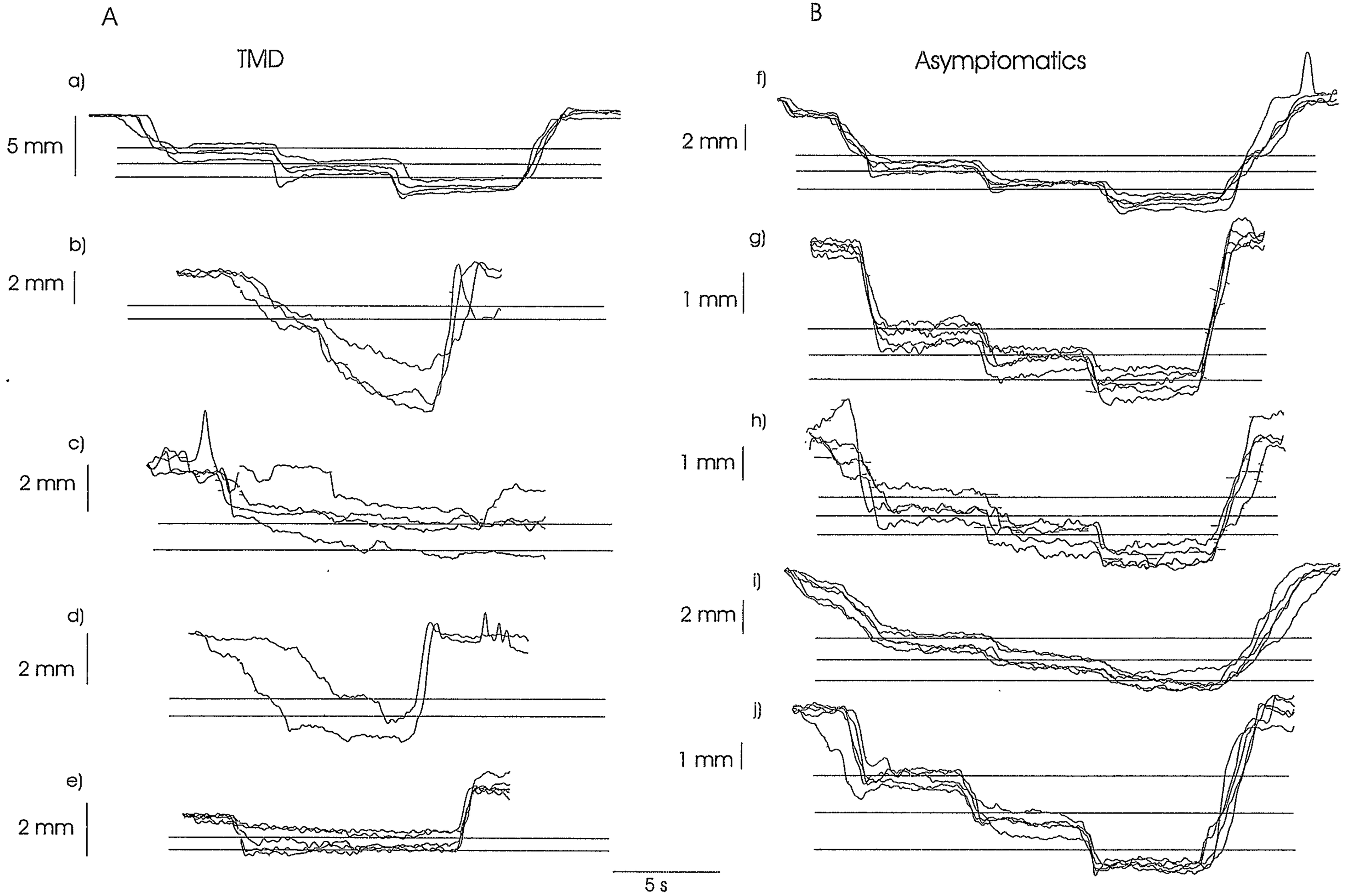


Fig 10 Jaws3D recordings in X-axis of multiple-step protrusive movement performed by the TMD patients and the asymptomatic subjects.

QUANTITATIVE ANALYSIS

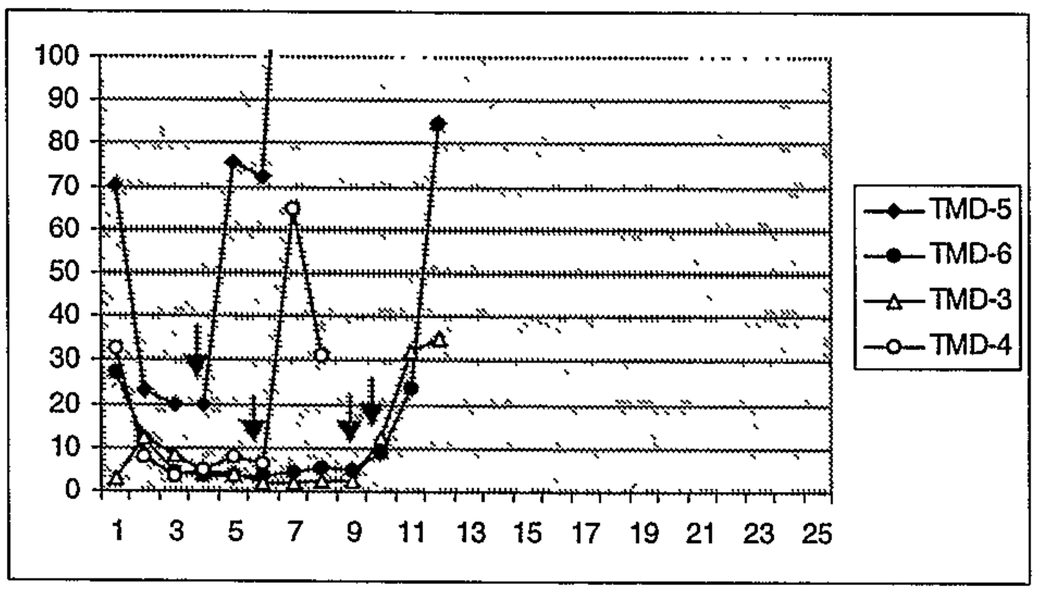
Coefficient of Variance

The coefficient of variance of similar tasks for each participant was calculated and plotted together for the two groups and compared. The comparison plots (Figures 11-14) show that on the whole, there is a noticeable visual difference between asymptomatics and TMD patients.

In general there seems to be more variance in the protrusive movements compared to the contralateral movements. Another noticeable factor was that more variance was noted in multiple-step movement than in single-step movements. Because of the small sample size, no statistical analysis comparing the asymptomatic and TMD groups was performed.

The coefficients of variance for the single-step contralateral tasks are plotted for four TMD patients and six asymptomatic individuals in Figures 11A and B, respectively. The short vertical arrows denote the ends of the holding phases in different subjects. Vertical arrows at the onset of holding phases have been omitted for clarity. Three of the TMD patients exhibited coefficients of variance comparable to those seen in the asymptomatic group, that is, all <10%. Only one TMD patients' coefficient of variance (filled diamond) exceeded 10% during a holding phase. Three of the TMD patients also exhibited a larger set of coefficients of variance values during the dynamic return phases than for any of the asymptomatic individuals.

A. TMD



B. Asymptomatic

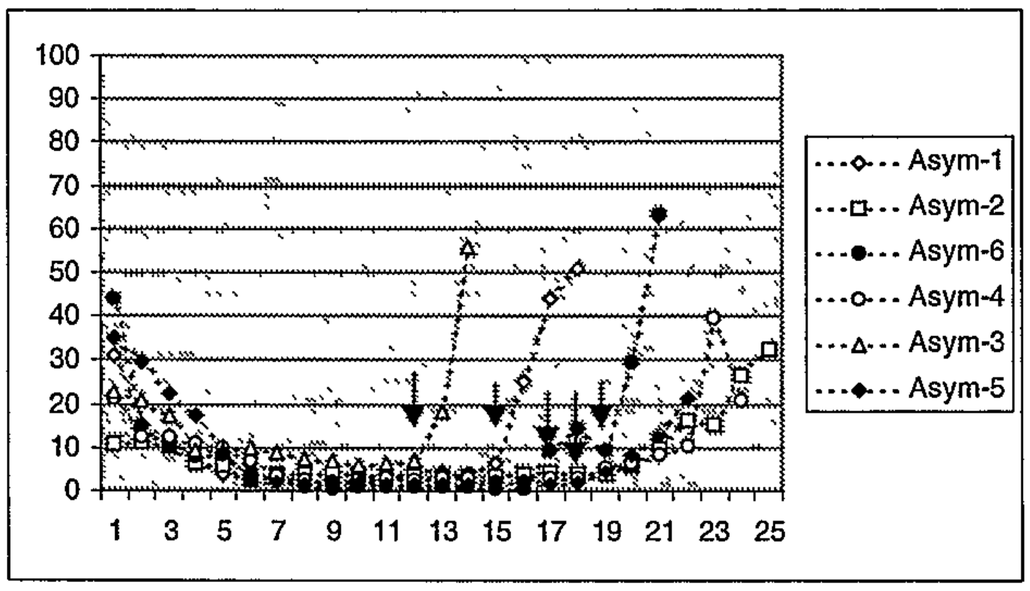
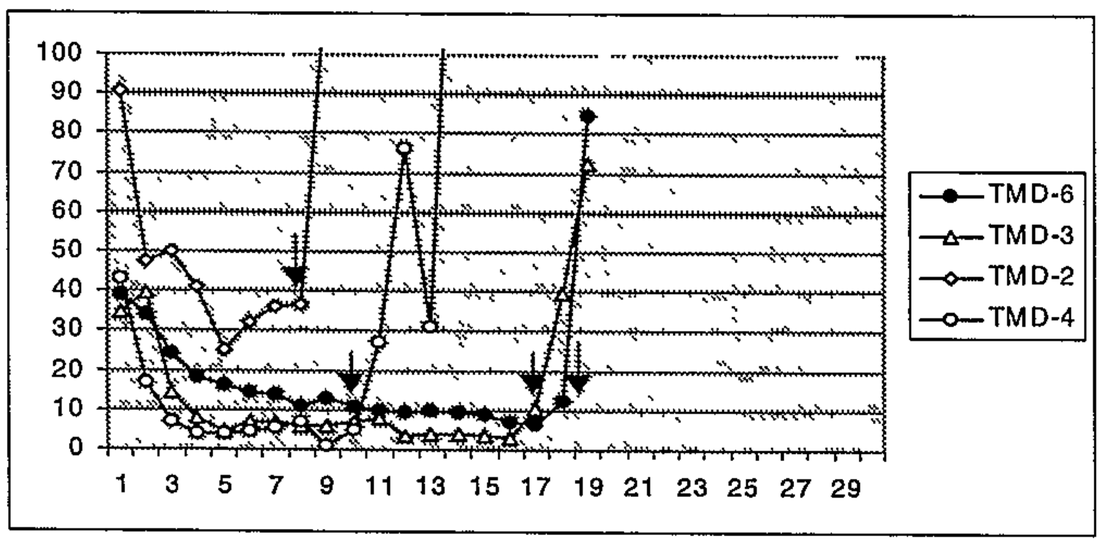


Figure 11 Contralateral Task – Single step

For the multiple-step contralateral movement, there was more variability in the TMD group than when the TMD group performed the single-step movement. Figure 12 show the coefficients of variance for the two-step contralateral task plotted for four TMD patients and the three-step contralateral task plotted for the six asymptomatic subjects, respectively. Two of the TMD patients exhibited coefficients of variance comparable to those seen in the asymptomatic group, i.e. all <10%. Two TMD patients' coefficient of variance (filled circle & unfilled circle) exceeded 10% during a holding phase, while the coefficient of variance values for all of the asymptomatics was consistently below 10% except for one asymptomatic that just exceeded 10%. As the end of the holding phases for all of the asymptomatic subjects ended approximately at the same time, only one arrow is showed to indicate the end of these holding phases. All of the TMD patients exhibited a larger set of coefficients of variance values during the dynamic return phases

while only one subject of the asymptomatic group (filled circle) showed an increased value.

A. TMD



B. Asymptomatic

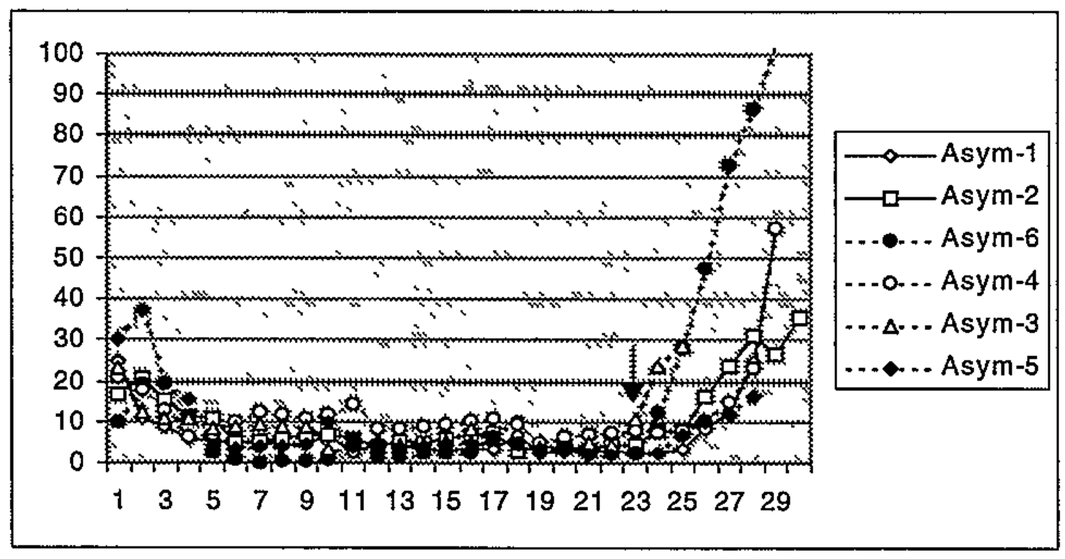
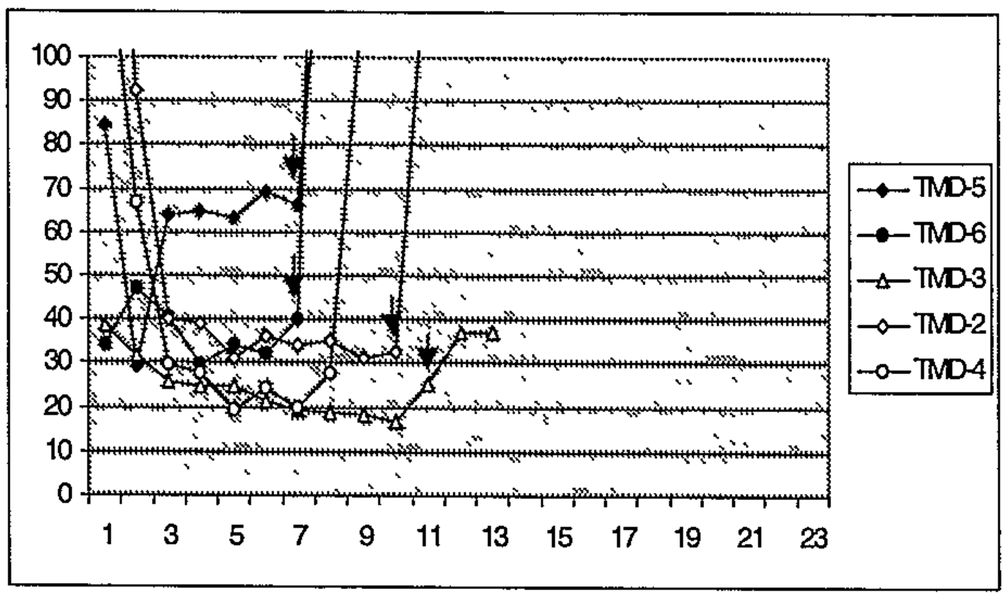


Figure 12 Contralateral Task – Multiple step

The coefficients of variance for the single-step protrusive task are plotted for five TMD subjects and six asymptomatic individuals in Figure 13A and B, respectively. All of the TMD patients exhibited coefficients of variance higher (i.e. >20%) than the asymptomatic group during the holding phase. The coefficient of variance values for all the subjects in the asymptomatic group were below 20% for the holding phase. All of the TMD patients except one (unfilled triangle) exhibited a larger set of coefficient of variance values, (i.e. >100%) during the dynamic return phases which was much higher than the corresponding values for the asymptomatic group (<100%).

A. TMD



B. Asymptomatic

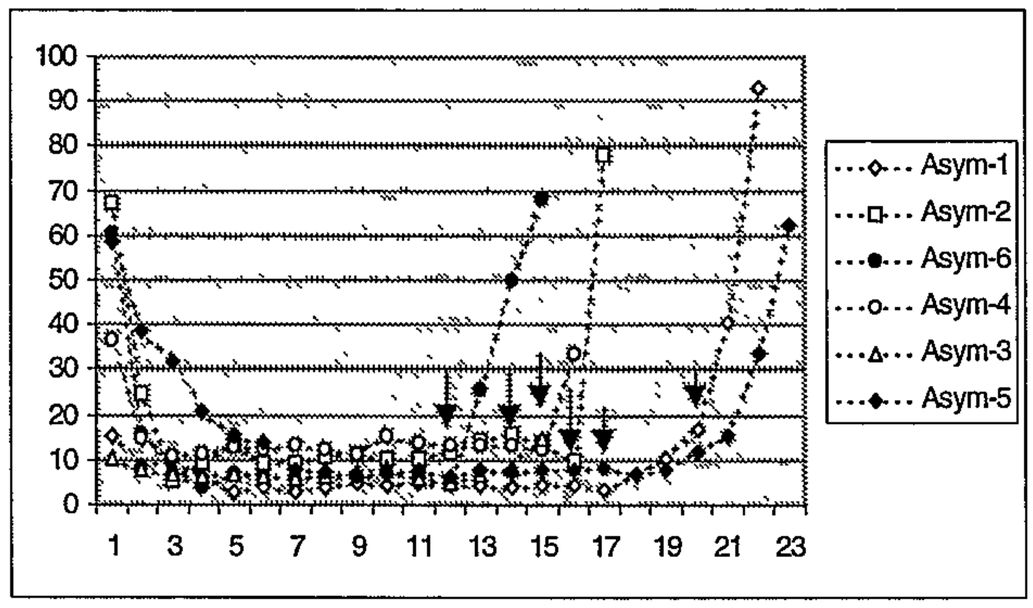
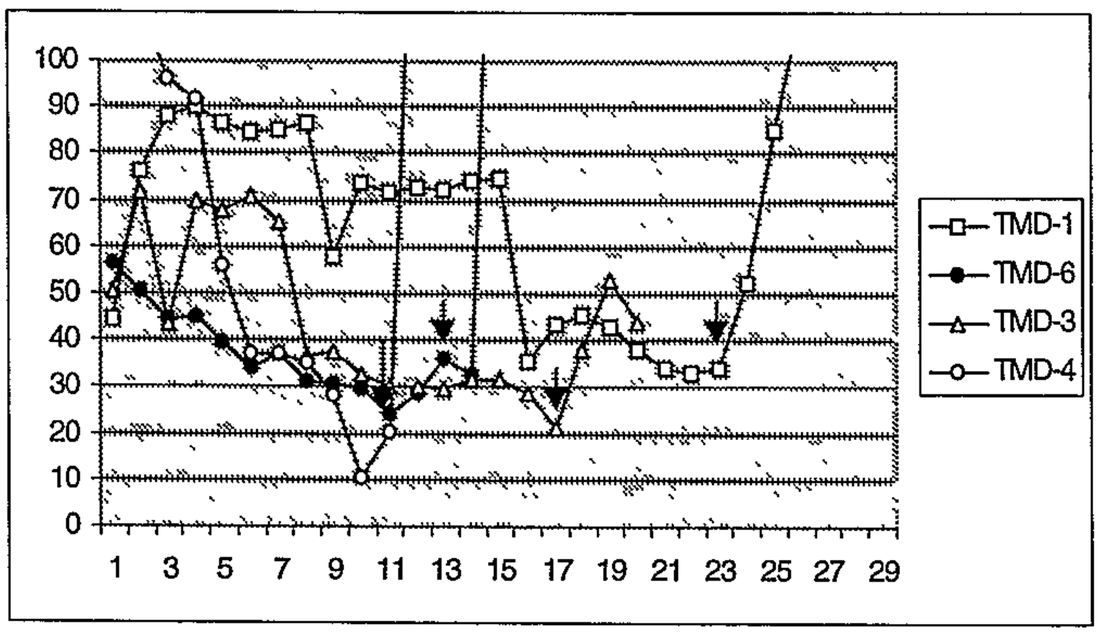


Figure 13 Protrusive Task – Single step

In the multiple-step protrusive movements a similar finding was noted as for the single-step movements. Figure 14 shows the coefficients of variance for the two-step protrusive task plotted for four TMD patients (A) and the three-step protrusive task plotted for the six asymptomatic subjects (B). As the movements for all of the asymptomatic subjects except one (filled diamond) ended approximately during the same time, only one arrow is showed to indicate the end of the holding phases. None of the TMD patients exhibited coefficient of variance values comparable to those seen in the asymptomatic group during a holding phase, that is all coefficients of variance values were <20% except one (unfilled square in the asymptomatic group). For one TMD patient (unfilled circle), two values were recorded below 20%, however this wasn't a consistent feature for this patient. All of the TMD patients exhibited a larger set of coefficient of variance values during the dynamic return phases, that is all values >100%, except one (unfilled triangle) which was

~ 50%. The coefficients of variance values for the dynamic return phase for all the participants in the asymptomatic group were < 70%.

A. TMD



B. Asymptomatic

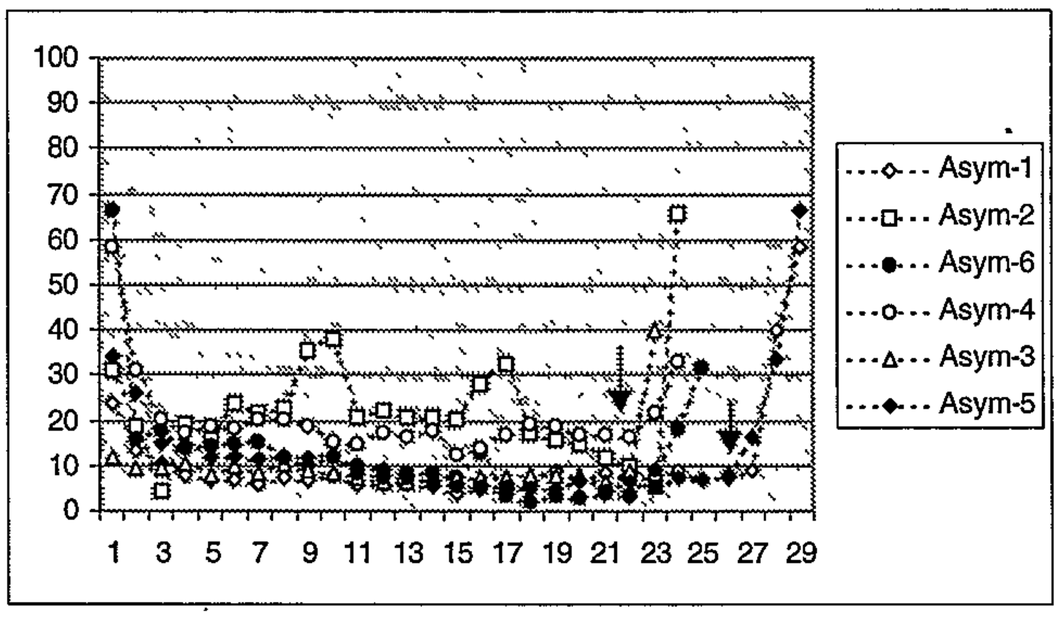


Figure 14 Protrusive Task – Multiple step

In summary, the TMD group appeared to exhibit more variability in performing similar tasks compared to the asymptomatics.

EMG Analysis

Table 2 illustrates all the different types of movements attempted by the TMD patients and whether the IHLP was active for those movements. The IHLP exhibited activity in all the TMD patients during contralateral and protrusive movements.

Patient	Side of Movement	CONTRALATERAL			PROTRUSION			Postural Jaw Position
		1 Step	2 Step	3 Step	1 Step	2 Step	3 Step	
TMD-1	Right side	Active	NT	Active	Active	Active	Active	Active
TMD-2	Right side	NT	Active	NT	Active	Active	NT	Not Active
TMD-3	Left side	Active	Active	NT	Active	Active	NT	Active
TMD-4	Right side	Active	Active	NT	Active	Active	NT	Not Active
TMD-5	Right side	Active	NT	NT	Active	NT	NT	Not Active
TMD-6	Right side	Active	Active	NT	Active	Active	NT	Not Active
NT - Not Tested								

Table 2. Movements performed by TMD patients

Subject	Side of Movement	CONTRALATERAL			PROTRUSION			Postural Jaw Position
		1 Step	2 step	3 step	1 step	2 step	3 step	
Asym-1	Left side	Active	NT	Active	Active	NT	Active	Not Active
Asym-2	Left side	Active	NT	Active	Active	NT	Active	Not Active
Asym-3	Left side	Active	NT	Active	Active	NT	Active	Not Active
Asym-4	Left side	Active	NT	Active	Active	NT	Active	Not Active
Asym-5	Left side	Active	NT	Active	Active	NT	Active	Not Active
Asym-6	Left side	Active	Active	Active	Active	NT	Active	Not Active
NT - Not Tested								

Table 3. Movements performed by Asymptomatic subjects

Table 3 shows the different types of the tasks performed by the asymptomatic group. In summary, IHLP was active during all the contralateral and protrusive tasks performed by the different subjects. It also clearly shows that for the two postural jaw position recordings, IHLP was found to be not active.

A. *EMG activity at Postural Jaw Position*

As can be noticed from Table 2, two of the six TMD patients exhibited tonic IHLP activity during postural jaw position, whereas IHLP activity at postural jaw position was totally absent in all the asymptomatic recordings.

Figure 15 shows IHLP activity of one TMD patient at postural jaw position. In this figure the EMG activity is plotted with the Jaws3D recording of the postural jaw position which shows the displacement of the MIPT during recording. A 100 ms part of the trial is enlarged to show the single motor unit activity. At least two single motor units can be identified.

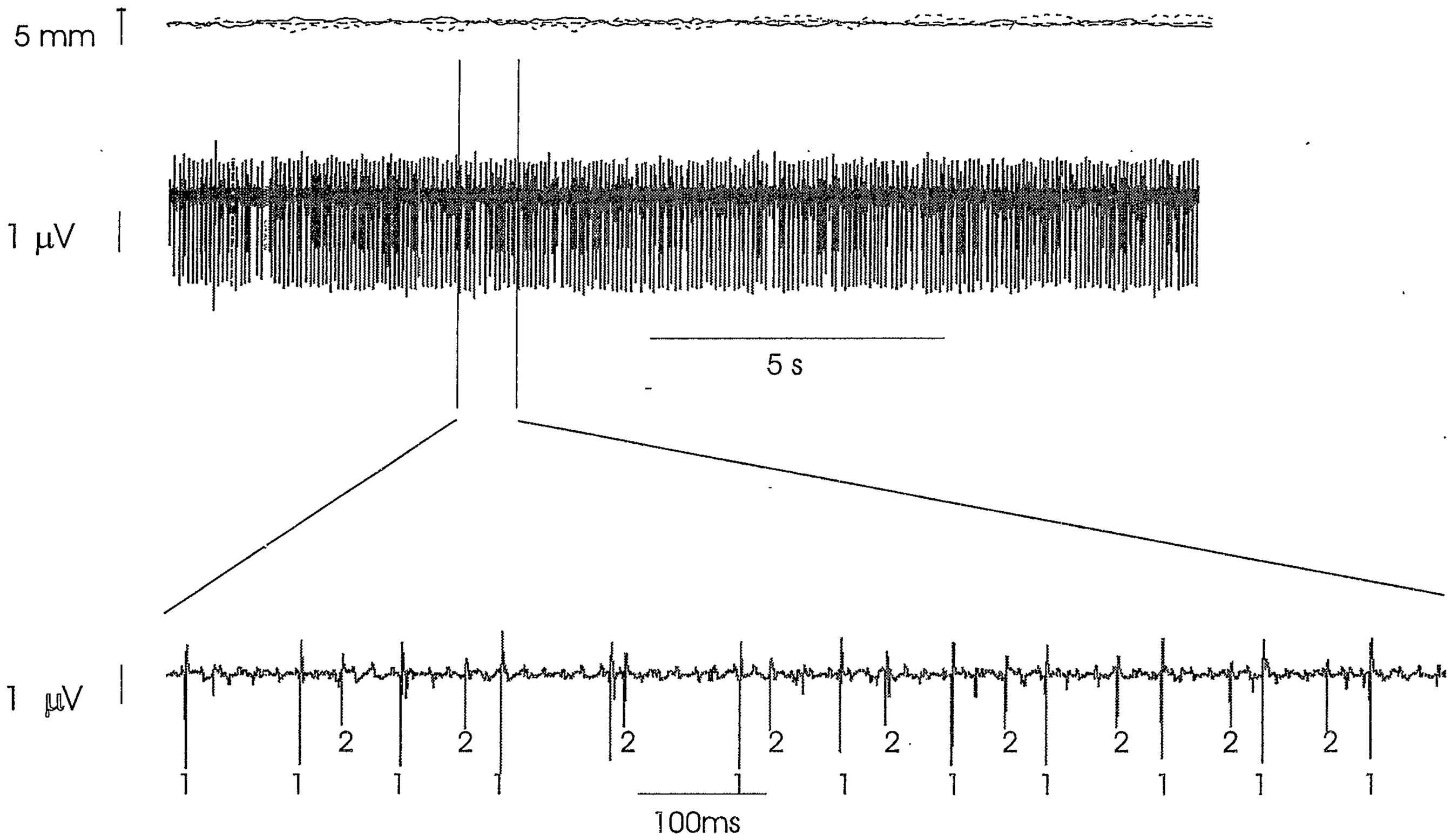


Fig 15 Shows IHLP activity at postural jaw position recorded from a TMD patient.
The two single motor units are numbered 1 & 2 respectively

B. ***EMG activity while performing tasks.***

As Tables 2 and 3 illustrate, IHLP was active in all the contralateral and protrusive movements done by both TMD and asymptomatic subjects. In all experiments, we also performed surface EMG recordings of masseter and anterior digastric muscles. Both masseter and anterior digastric muscle activity was observed in both groups for some trials.

Table 4 shows the number of trials in which EMG activity was observed in the digastric and masseter and as a proportion of the total number of trials in which EMG recordings from these muscles were made. The percentages show that for all types of movements, the TMD group showed a higher percentage of trials in which EMG activity was observed in the masseter and digastric in comparison with asymptomatics.

Group	**Digastric**				**Masseter**			
	Protrusion	%	Contralateral	%	Protrusion	%	Contralateral	%
Asymptomatics	76/175	43.4%	61/174	35.05%	36/175	20.5%	1/174	0.57%
TMD	68/123	55.28%	81/132	61.36%	42/123	34.14%	29/132	21.96%

Table 4. Comparison table showing activity of digastric and masseter in asymptomatic and TMD groups. Numerator – trials showing activity; Denominator – total number of trials performed.

Figures 16 & 17 show the EMG activity of the digastric muscle and IHLP seen in two TMD patients while performing two different tasks. Figure 16 shows the digastric activity in a TMD patient in the three trials performed for two-step contralateral movement. The activity of IHLP and the Jaws3D tracking is also plotted.

Figure 17 shows the digastric activity in a TMD patient while performing single-step protrusive movement. The figure also compares the digastric activity along with the IHLP EMG data and the Jaws3D data.

Figure 18 shows masseter activity in one TMD patient while performing single-step protrusion. Similar to the previous plots this plot also compares the EMG activity of IHLP and the Jaws3D plots for the multiple trials performed for the same task by the same participant.

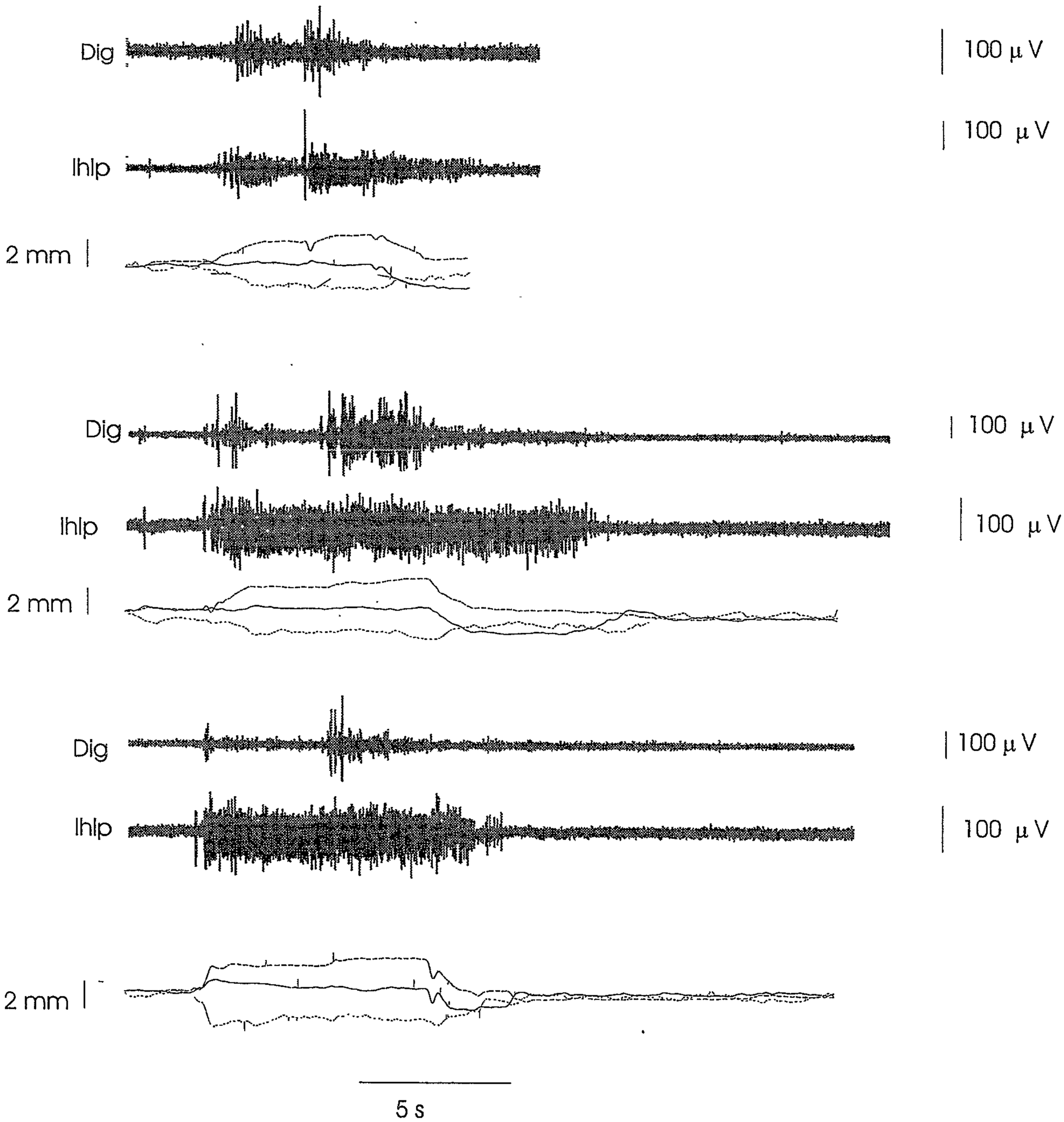


Fig 16 Shows digastric activity in a TMD patient in the three trials performed for two-step contralateral movement.

Dig : Digastric Muscle.

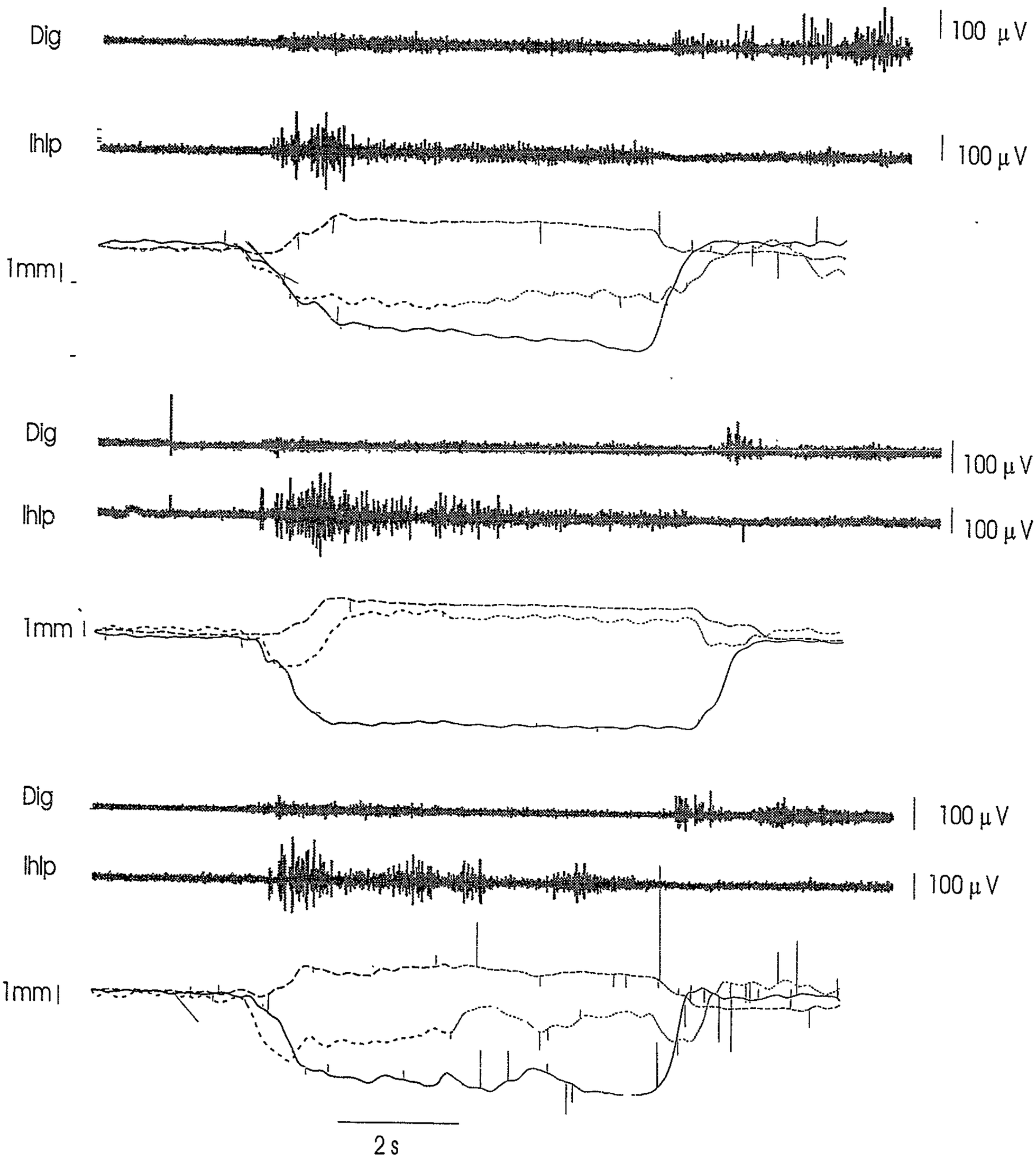


Fig 17 shows the digastric activity in a TMD patient while performing single-step protrusive movement.

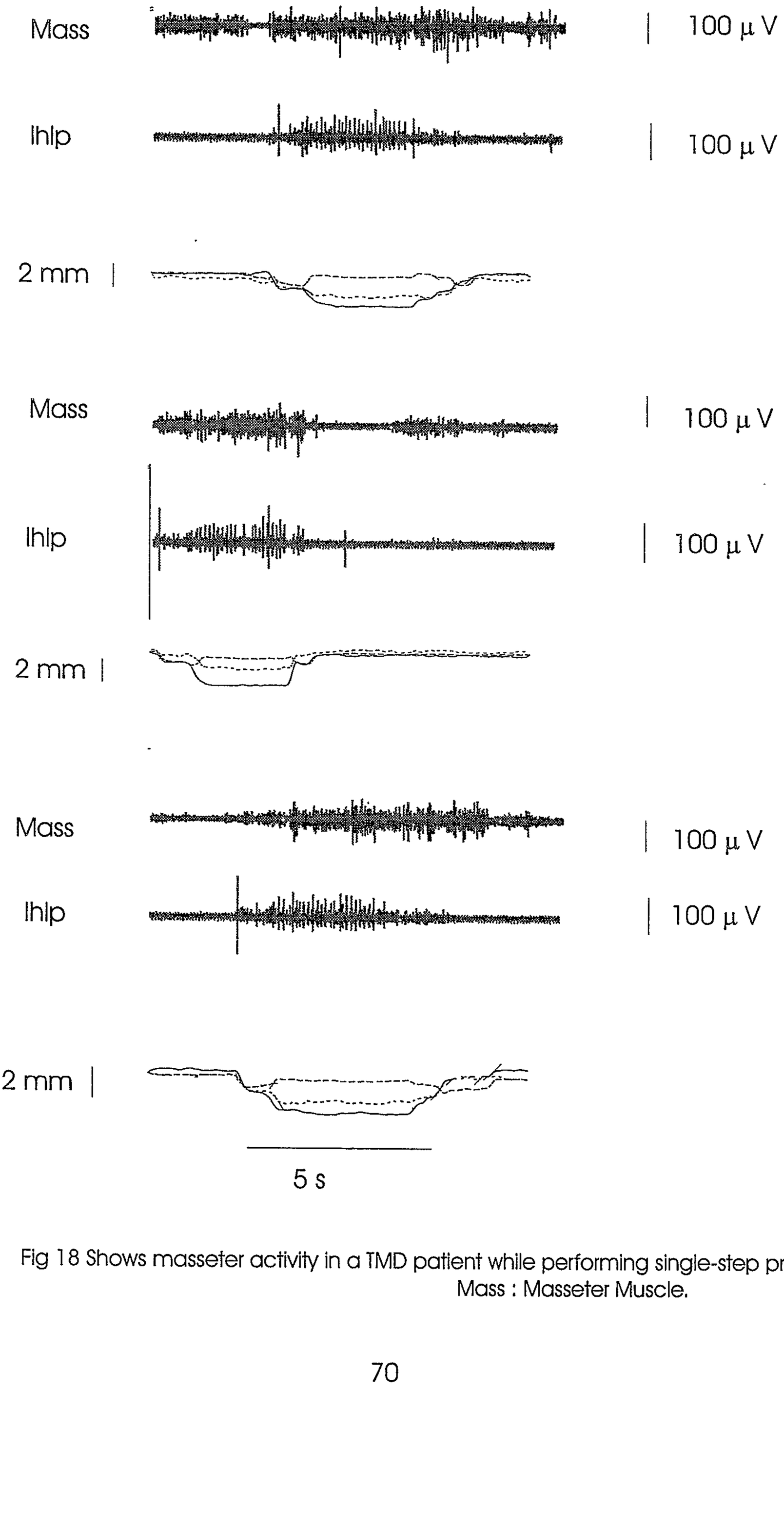


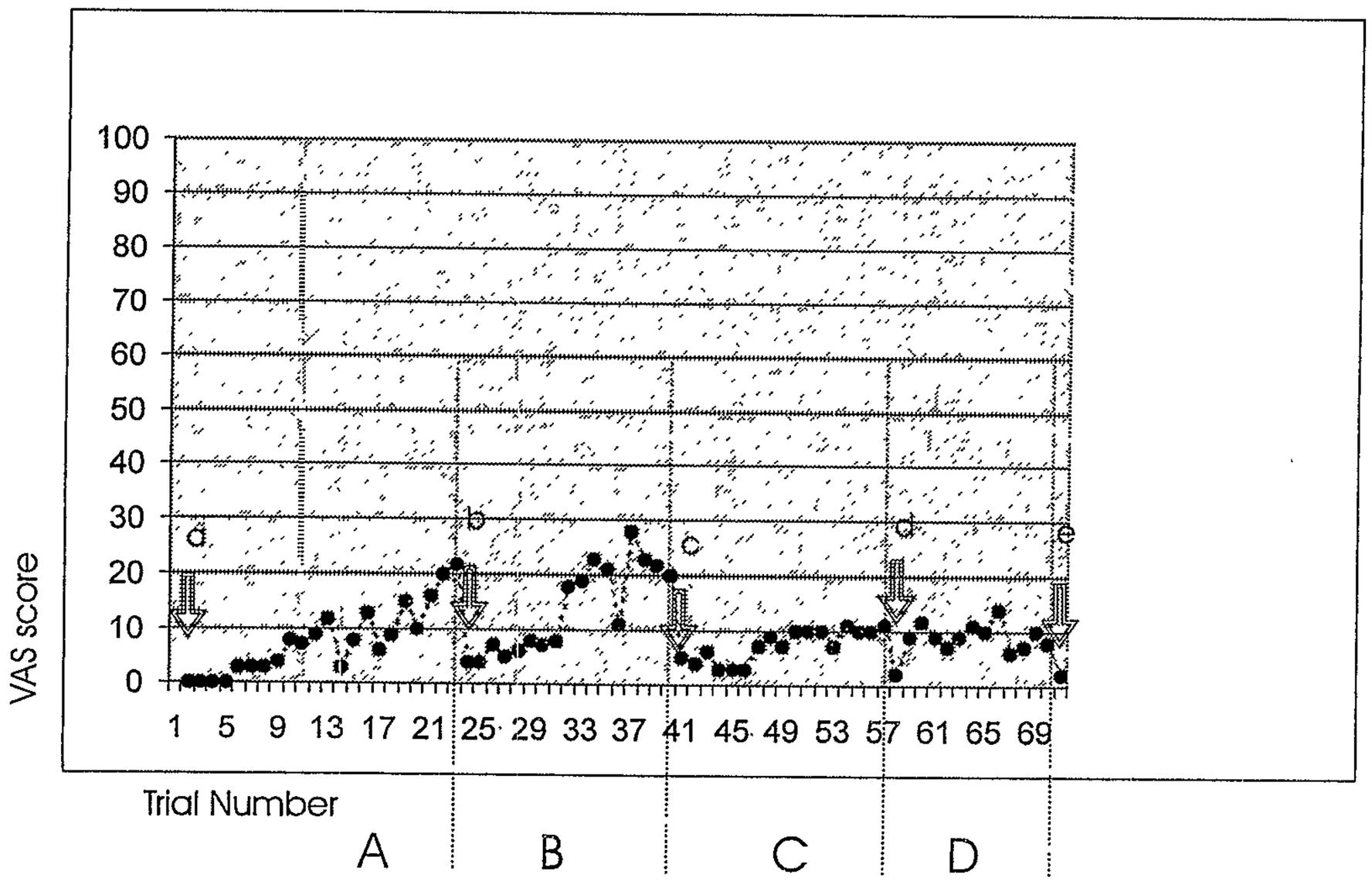
Fig 18 Shows masseter activity in a TMD patient while performing single-step protrusion.
Mass : Masseter Muscle.

Pain Score Analysis

All TMD patients were asked to score their pain levels on a 100-mm VAS scale after each trial. Of the six TMD patients only one scored consistently from the start of the recording session till the end on a trial-by-trial basis. Those VAS scores were then plotted against the trial numbers in Figure 19.

Figure 19 shows the VAS profile for one TMD patient and it shows that there was a gradual increase in the pain level as the experiment progressed until a rest period occurred. The arrows in the plots show the rest periods between groups of trials and the corresponding fall of VAS scores. The vertical lines in the figure show the end of one particular task and the beginning of another set of trials. Generally there was a gradual increase in VAS score from the start of each trial in a group of one task towards the end.

Shorter rest periods of approximately 30 s to one minute was taken between individual trials. At rest period "a", which indicated the start of the recording session, there was a time period of two minutes, at "b" the rest period was six minutes between recordings, at "c" the rest period was five minutes, "d" was a postural jaw position recording of 15 s after a one minute interval from the previous trial, and "e" was the final trial recorded, a postural jaw position recording of 15 s after a time period of one minute from the previous recording. Whenever there was a rest period there was a fall in VAS scores noticed for this patient.



A: Two step contralateral movement
B: Single step contralateral movement
C: Two step protrusion
D: Single step protrusion

⇩ Rest Periods

Fig 19 Shows the VAS profile of one TMD patient. The gradual increase in the pain level as the experiment progressed until a rest period occurred can be noticed.

DISCUSSION

This study aimed to determine whether there were any qualitative differences in jaw tracking performed by a group of TMD patients in comparison with an asymptomatic group and to describe qualitatively the EMG activity of the LP muscle in TMD patients to determine whether there was any evidence supporting the notion of hyperactivity within the LP in TMD patients. In general, the results showed that there was qualitatively more variability and inconsistency in jaw tracking shown by the TMD patients than the asymptomatic subjects while performing similar tasks. In addition, multiple-step movements showed more variability than the single-step movements, and protrusive jaw movements were more variable than the contralateral jaw movements.

The TMD patients showed a higher percentage of trials with activity in masseter/digastric and IHLP muscles while they performed contralateral and protrusive jaw movements, than did the asymptomatics. Two of the six TMD patients who participated in this study showed tonic activity of IHLP at postural jaw position while none of the asymptomatic subjects showed any IHLP activity at postural jaw position. The data from this study also suggest that the pain level increases with function and decreases with rest.

These data are consistent with the emerging notion that pain has distinct effects on motor function and that increased motor activity exacerbates pain (Stohler 1999, Svensson *et al* 2001). Stohler (2001) concluded that *pain has direct effects on motor function that*

include changes in facial expression and body posture, along with a tendency to avoid movements or to execute them more slowly.

This study indicates that pain alters motor function. Initial observations were that the TMD patients with pain showed a tendency to limit or avoid the range of movements. While performing the multiple-step tasks, TMD patients were asked to perform two-step movements, unlike asymptomatics who performed three-step tasks, because the TMD patients reported increased discomfort and pain when the trials were longer and they also had difficulties in holding the jaw in a particular position for other than short periods of time (e.g. ~2 s). So by decreasing the multiple-step trials by one step and by decreasing the total amount of time in the holding phases, the range of movement and total length of the trials was reduced. These initial qualitative observations about the difficulties the TMD patients experienced in performance of these tasks, support the notion that pain and discomfort alters normal motor activity. As the results of this study show, the TMD group also showed more variability when compared with the asymptomatic group in performing similar tasks. This increased variability was noticed even though the trial duration was shorter in the TMD patients. It is therefore likely that the TMD patients may have performed the tasks with even more variability with longer duration trials and with three-step trials.

Previous studies support the notion that maximum muscle output during forceful concentric muscle work is reduced in the presence of pain (Stohler, 1999). In another study on fibromyalgia patients, Backman and co-workers (1988) concluded that

fibromyalgia patients reach only 64% of mean control maximum handgrip. High *et al* (1988) recorded the maximum bite force before and after third-molar extractions and found that the maximum bite force was reduced in pain following third-molar extraction when compared with measurements taken prior to the surgical intervention.

Besides the maximum force output, reductions of functional muscle activities have also been reported in pain situations, resulting in a decrease of force or speed of movement during the concentric muscle work (Svensson 1996). Our study showed that the accuracy and the consistency in tracking is generally lacking in TMD patients. Whether pain alters the speed of movement is not clear, but the ability to track a target is more variable in TMD patients with pain in comparison with asymptomatic individuals. More specifically, the range and pattern of movements and the fine control in the movements were altered in TMD patients with pain.

Effects of TMD pain on masseter and digastric EMG

The results showed that 34% of TMD patients exhibited masseter activity on protrusion compared to 20% of asymptomatics, while 21% exhibited masseter activity on contralateral movement compared to 0.5% of asymptomatics. These significant differences between the two groups could be explained on the basis of the pain adaptation model (Lund *et al.* 1991), which proposes that the relative pattern of action of agonist and antagonist muscles is modified during pain. In the presence of deep pain, the antagonist muscles appear to become activated in an attempt to limit activity of the agonist muscles. So that means that in pain patients, the increased antagonist muscle activity (eg. digastric)

would help to protect the injured part, avoid further damage and pain and would help to promote healing by limiting the range and rate of movement. This finding also is consistent with Sessle's (1999) view that the responses in the agonist and the antagonist muscles could be interpreted as a "splinting" effect that counteracts excessive movement and so protects the articular or muscular tissues from further damage with the physiological purpose to limit jaw movements and allow rapid healing. Okeson terms this phenomenon as "co-contraction" (Bell's Orofacial Pains, Fifth Edition. 1995).

The splinting effect, which protects the jaw and muscles from further damage in TMD patients with pain might be a reason or contributing factor in the variability, inconsistency and inaccuracy in tracking shown by TMD patients. Okeson (1995) states that some pains are a result of a protective mechanism called co-contraction. Co-contraction may result from an alteration in sensory or proprioceptive input and also may result as a response to deep pain or even the threat of injury or an increase in emotional stress (see Moulton 1966, 1968). Protective co-contraction is a CNS response to injury or threat of injury. In the presence of an injury or threat of injury, the normal sequencing of muscle activity seems to be altered to protect the threatened part from further injury. In the presence of altered sensory input or pain, antagonistic muscle groups seem to fire during movement in an attempt to protect the injured part.

In this study, evidence for co-contraction of the agonist and antagonist muscles was seen more frequently in the TMD patient group than in the asymptomatics. The co-contraction seen in the asymptomatics could be attributed to the psychological status of the subjects

during the experiment. As Moulton (1966, 1968) explained, the increase in emotional stress could be the cause of the co-contraction in asymptomatic subjects. In future studies, subjects could score via their visual analogue scale pain levels, discomfort and their anxiety levels during the trial and after each trial. This might provide an opportunity to address these issues in a better way.

Effects of TMD pain on jaw movement features

In previous experimental pain studies conducted by Lund *et al* (1991), motor function at rest and during open-close movements of the mandible was assessed at a controlled rate in pain free subjects before, during and after experimental pain induced by infusion of 5% saline into the body of the left masseter muscle. They found that the velocity and amplitude of the repetitive opening movements decreased during pain and that the area under the curve of the rectified and smoothed masseter bursts were reduced during jaw closure in the presence of pain. Experimental muscle pain studies performed by Svensson and others (1995) by injection of 5% hypertonic saline into the masseter muscle found that displacement of the mandible during painful mastication was smaller in the vertical and lateral axes. They also found that the mean opening and closing velocities of the mandible and the total displacement of jaw movement were significantly reduced during pain. They also noticed that the agonist EMG activity during pain was significantly lower in the ipsilateral masseter muscle. Svensson and co-workers in another study (1997) to determine the effect of bilateral experimental muscle pain on human masticatory patterns, concluded that the mean EMG activity of all the jaw-closing muscles in the agonist phase was significantly decreased and the mean EMG activity of all the jaw-closing muscles in

the antagonist phase was significantly increased as compared with pain-free mastication. The maximum voluntary occlusal force has also been shown to be significantly lowered during painful clenches compared to the pre-pain and post-pain clenches (Svensson *et al.* 1997, 1998, 2001).

A few studies have also been conducted in TMD patients. In a study conducted by Mongini and others (1989) to assess the mandibular movements and masseter EMG activity during habitual mastication in TMD patients, it was found that in TMD patients, the normal symmetrical and balanced distribution of the chewing cycles was lost and the movements were more restricted. That is the chewing cycles showed marked irregularities such as sudden changes of direction, movement stoppage and/or re-opening during closing. They also noticed that the EMG data showed marked alterations with an increase in masseter muscle (antagonist) activity during opening in some patients. In another study, Feine and co-workers (1988) found that the average and maximum opening velocities of the mandible were lower in patients with persistent jaw muscle pain.

All these previous studies point to the notion that pain alters motor function. The data from the present study are also consistent with this hypothesis that pain alters jaw motor function. The increased variability in jaw tracking shown by the chronic TMD patients with pain when compared to asymptomatic subjects while performing similar standardised tasks, clearly suggest that not only is motor function altered by pain but, further the fine jaw motor control is compromised in TMD patients.

Activity of the IHLP in TMD pain patients

The action of the IHLP muscle in horizontal jaw movement seems to be affected in TMD patients with pain. In a single motor unit study of IHLP carried out while asymptomatic subjects performed standardised tasks, IHLP was shown to be active during contralateral, protrusive and submaximal opening jaw movements (Phanachet *et al* 2001). They recorded 99 single motor units from 22 recordings. All 99 single motor units were active during contralateral jaw movements and protrusive jaw movements with the teeth apart and 81% were found to be active on sub-maximal jaw-opening movements. Uchida and others (2001) conducted studies to determine the EMG activity of the IHLP during performance of a standardised horizontal isometric jaw task. They found that the IHLP plays an important role in the generation and fine control of contralaterally directed horizontal isometric forces such as are required in mastication and parafunctional activities. Many other studies have also concluded that the IHLP is active in protrusive, contralateral and jaw-opening movements (Moyers 1950; Mahan *et al* 1983; Widmalm *et al* 1987; Murray *et al* 1999).

In the present study, intramuscular EMG recordings of the IHLP showed that the muscle was active in all contralateral and protrusive jaw movements performed by both TMD and asymptomatic groups. But at the postural jaw position, IHLP was found to be inactive in all asymptomatic subjects. Two of the six TMD subjects in the present study showed tonic activity of IHLP at postural jaw position. Other surface EMG studies have

reported a small increase in postural EMG activity of the jaw-closing muscles in TMD patients with pain (Rugh *et al* 1987; Dolan *et al* 1988). We also found evidence for increased masseter EMG activity in TMD patients. However, these findings are not entirely consistent with other experimental pain studies done in asymptomatics. A number of experimental pain studies (Stohler *et al* 1996; Graven-Nielsen *et al* 1997; Svensson *et al* 1998) concluded that muscle pain at a clinically relevant level, and induced by infusion of hypertonic saline had little to no effect on postural EMG activity. But all these studies were based on experimentally induced pain and surface EMG recordings and given the complexity of TMD pain, it may be that the effects on EMGs of TMD pains may not be the same as those derived from experimentally induced pain. This could be due to the complexity of TMD pain as compared to the simpler nature of experimentally induced pain.

Lund and co-workers (1991) studied five musculoskeletal pain conditions – TMD, muscle tension headache, fibromyalgia, chronic lower-back pain and post-exercise muscle soreness - and found that the level of resting or postural muscle activity was no higher than normal in all conditions. But they found an occasional exception in an increase in EMG activity in some facial muscles and concluded that this increase was a reflection of changes in facial expression that was caused by pain. The IHLP activity found at postural jaw position suggests the possibility of increased activity in the muscle. This may be because the IHLP was attempting to position the jaw to a new rest position, which may be less painful. So, depending on the new postural positioning of the jaw required for minimising pain, certain muscles could be expected to be more active. If the

least painful postural jaw position was lateral to the normal postural jaw position, then the contralateral IHLP could be expected to become more active. This could be the reason for the increased activity seen in some patients.

Our study also demonstrated that the normal role of IHLP as shown in asymptomatics, that is consistent activity in both contralateral and protrusive movement, was also apparent in TMD patients. However, more variability in jaw tracking was noticed within the TMD group while performing protrusive movements than seen in the contralateral movement. This could be because of a lack of co-ordination of function between bilateral IHLP activity in TMD patients. Unlike contralateral movements, a co-ordinated bilateral IHLP activity is needed while performing protrusive movements. The coordination in muscle function might be altered in TMD patients. Clinically, lateral displacements may be more likely to evoke intra-capsular problems, than the symmetrical movement of protrusion (I.J.Klineberg, personal communication). But at this stage it is unclear how this clinical observation relates to our findings.

FUTURE STUDIES

Future studies should address more TMD patients with clearly identified TMD conditions and compared with similar studies in age-and-gender matched control subjects. Patients selected for future studies should be classified as TMD based on RDC/TMD criteria. Further, patient groups of chronic joint pain and chronic muscle pain could be studied separately to determine if there would be any difference in motor activity and function between the two groups.

As mentioned earlier, in future studies, VAS scores could be recorded separately for pain, discomfort and the anxiety level of the subjects after each trial. This would give a good base to attribute the differences noticed in EMG activity and/or jaw movement to pain, discomfort, dysfunction and/or anxiety level of the patients at that particular time during each trial.

Bilateral studies of IHLP could also be done in future as explained by Reid *et al.* (1994) that clinical jaw muscle pain is often bilateral. That may also shed some light onto the coordinated action of the same muscles of both sides.

Further detailed single motor unit studies are needed on LP in TMD patients to study whether there are differences in EMG activity between the TMD group and asymptomatics while performing similar standardised tasks for the same time duration. This is especially important as the LP has been implicated as playing an important role in

TMDs (Hiraba *et al* 2000; Lund 2000; Okeson 1998). Therefore it would be valuable to have baseline information on the functional properties of single motor units from the LP during standardised tasks in TMD patients and to compare them with similar data collected from matched control asymptomatic subjects. The data in this study would provide a basis for the design of future studies of the possible involvement of the LP in TMD.

CONCLUSION

This study has provided a detailed description of some differences shown by TMD patients while performing similar jaw movement tasks in comparison with asymptomatic subjects. The data support the hypothesis that jaw motor function is altered in TMD patients. We found that IHLP is active at postural jaw position in two out of six subjects and this suggests that there may be differences in IHLP activity in TMD patients.

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