The Effects of Isotonic Resistance Exercise on the Muscles of Mastication.

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DECLARATION

I hereby declare that, to the best of my knowledge, the work described herein is original and entirely the work of the author, except where due acknowledgements have been made. The work was conducted whilst the author was pursuing the degree of Master of Philosophy (Dentistry) at the Faculty of Dentistry, The University of Sydney, and carried out at the Jaw Function and Orofacial Pain Research Unit, Westmead Centre for Oral Health, under the supervision of Associate Professor Chris Peck and Professor Greg Murray. I verify that this thesis has not been submitted, wholly or in part, for any other Masters or higher degree award to any other institution or university, and that all the assistance received and sources used in the preparation of this thesis have been duly acknowledged.

Alexander Wirianski
MPhil(Dentistry) Candidate
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ABSTRACT

Temporomandibular disorders (TMD) are jaw muscle and joint problems that affect around 10% of the adult population. The most prevalent subgroup of TMD are those with a musculoskeletal origin and are largely idiopathic in nature. Physiotherapy, in the form of resistance exercise, has been successful in relieving some of the symptoms of TMD by possibly altering muscle activity. Electromyography (EMG) is frequently utilised as a means of quantifying muscle activity.

The aims of this thesis were to train individuals to use their jaw muscles differently, and to demonstrate that these changes in muscle use can occur in the short term and can be maintained over the longer term following the application of a four-week home-based isotonic resistance exercise programme

Twelve healthy, asymptomatic adult volunteers participated in this study. Each subject was asked to attend a total of three data collection sessions: an Initial Training Session (ITS); Testing Session 1 (TS1), conducted four weeks after the ITS; and Testing Session 2 (TS2), eight weeks after the ITS. The ITS was divided into four sections: Subject Preparation; Pre-Exercise Data Collection; Exercise Training; and Post-Exercise Data Collection. Each Testing Session was divided into two sections: Subject Preparation; and Data Collection.

EMG activity was recorded with bipolar surface electrodes overlying the anterior temporalis and masseter muscles bilaterally and the right anterior digastric muscle. The EMG activity of these muscles was quantified by computing the area under the normalised EMG data curve, the peak EMG activity, the time to peak EMG activity, the time to peak EMG activity in relation to movement onset, the duration of EMG activity and the relative

onset and offset of EMG activity in relation to movement onset and offset, respectively. An analysis of total activity, maximum activity, rate of activity increase and timing of activity onset and offset of the normalised EMG signal was undertaken.

Movement of the jaw was obtained with an optoelectronic system (JAWS3D) that tracked three light emitting diodes (LEDs) attached to the maxillary and mandibular teeth. JAWS3D computed the motion of the mandibular mid-incisor point relative to the maxillae and displayed it in real time to the subject to standardise movements with an LED bank.

The immediate effects of isotonic resistance exercise were tested during the ITS, where subjects were asked to perform five trials of right lateral jaw movement before and after isotonic resistance exercises, where subjects applied finger pressure at 60% of maximum voluntary contraction to the mandible against right lateral movement of the jaw.

To test the training effects of a four-week home-based isotonic resistance exercise programme, all subjects were randomised to either a Control Group or an Exercise Group following the completion of the ITS. Subjects in the Exercise Group were asked to perform the same isotonic resistance exercise as in the ITS, three times per day for four weeks, and to perform 10 repetitions of each exercise. Subjects in the Control Group were instructed to continue with their normal activities of daily living and not asked to perform any jaw exercises during the following four weeks. At the end of this four weeks' period, all subjects were asked to return for TS1, for retesting and data collection of the same movement trials as in the ITS.

To test the long-term effects of the four-week home-based isotonic resistance exercise programme, at the end of TS1, all subjects were instructed to continue with their normal activities of daily living and not asked to perform any jaw exercises during the following

four weeks. At the end of this second four weeks' period, all subjects were asked to return for TS2, for retesting and data collection of the same movement trials as in the ITS.

At the completion of the study all subjects were able to perform the same standardised right lateral movements during each testing session and between each testing session. The muscles which showed changes most consistently throughout all the tested variables were the left (contralateral) anterior temporalis, the left (contralateral) masseter and the right (ipsilateral) anterior digastric. These three muscles showed changes in three out of the four variables that had significant changes over the testing period ($p < 0.05$). The right (ipsilateral) anterior temporalis showed changes in two out of the four significant variables ($p < 0.05$). The right (ipsilateral) masseter showed no significant changes in any of the tested variables ($p > 0.05$). Some of these changes occurred after the immediate application of the resistance exercise, some occurred after four weeks of training, while some occurred four weeks after the cessation of the training period.

Following the immediate application of isotonic resistance exercise there was a reduction in the duration of EMG activity in the ipsilateral anterior temporalis, with a concomitant increase in the duration of EMG activity in the ipsilateral anterior digastric, which also showed an increase in the time taken to reach its peak EMG activity. The offset of the EMG activity of the contralateral masseter also approached the movement offset. A reduction of the duration of EMG activity may imply that other muscles of mastication may have increased their activity in order to complete the standardised jaw movement task. Along with the increased duration of EMG activity in the ipsilateral anterior digastric there may have also been changes in the lateral and/or medial pterygoids which are the primary agonists of right lateral jaw movement but were not tested in this study.

After four weeks of a home-based isotonic resistance exercise programme the Exercise Group showed less changes than the Control Group in the time taken to reach peak EMG activity in the ipsilateral anterior temporalis, the contralateral masseter and the ipsilateral anterior digastric. This was interpreted as a reduction in the variability of the measured EMG variables and the possible maintenance of a more stable motor control pattern. Changes in the variability of EMG activity and reduction of the duration of EMG activity continued after four weeks following the Exercise Group ceased the home-based isotonic resistance exercise programme

This study has shown that isotonic resistance exercise can cause changes in EMG activity of the muscles of mastication in asymptomatic individuals. Determining the mechanisms by which these changes occur will help facilitate the development and the appropriate application of rehabilitation programmes in order to reduce the impact of TMD and return symptomatic individuals to normal jaw function.

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ABBREVIATIONS

%MVC	Percentage of maximum voluntary contraction (MVC)
α	The probability of a Type I error, set at $\alpha = 0.05$
Ag-AgCl	Silver-silver chloride
ANCOVA	Analysis of co-variance
ANOVA	Analysis of variance
AUC	Area under the curve
C_1	First cervical ventral ramus
CCD	Charge coupled device camera
CN V3	Cranial nerve V3; Mandibular division of Trigeminal nerve
CN VII	Cranial nerve VII; Facial nerve
CN XII	Cranial nerve XII; Hypoglossal nerve
ED	Duration of EMG activity
EMG	Electromyographic
EPk	Peak EMG activity
ETPk	Time to peak EMG activity
ETPm	Time to peak EMG activity in relation to movement onset
FHP	Frankfort horizontal plane
FPL	Flexor pollicis longus muscle
Hz	Hertz
ITS Post	Initial Training Session, Post-Exercise Training
ITS Pre	Initial Training Session, Pre-Exercise Training
ITS	Initial Training Session
kHz	Kilo Hertz
K-S	Kolmogorov-Smirnov test of normality
LBP	Low back pain
LED	Light emitting diodes
M1	Primary motor cortex
MEP	Motor evoked potential
MIPT	Mandibular mid-incisor point
mL	Millilitres
mm	Millimetres

MSE	Mean square error
MVC	Maximum voluntary contraction
n	Sample size
p	p-value, level of statistical significance, set at $p = 0.05$
RDC/TMD	Research Diagnostic Criteria for Temporomandibular Disorders (TMD)
RET	Relative EMG offset time in relation to movement offset
ROT	Relative EMG onset time in relation to movement onset
SD	Standard deviation
sec	Seconds
SigOn90Y	90% of the normalised peak EMG activity
TENS	Transcutaneous electrical nerve stimulation
TMD	Temporomandibular disorders
TMJ	Temporomandibular joint
TMS	Transcranial magnetic stimulation
TS1	Testing Session 1
TS2	Testing Session 2
VL	Vastus lateralis muscle
VMO	Vastus medialis oblique muscle

PRESENTATIONS

Part of this thesis has been presented in the form of a Poster Presentation at the conferences listed below.

Regional Conferences

Wirianski A, Murray G and Peck C. Modifying jaw muscle activity with physiotherapy exercises. International Association for Dental Research (Australia/New Zealand Division) Annual Scientific Meeting, October 2008. Perth, WA, Australia.

Wirianski A, Murray G and Peck C. Modifying jaw muscle activity with physiotherapy exercises. Australian Physiotherapy Association NSW Branch State Symposium, October 2008. Sydney, NSW, Australia.

Local Conferences

Wirianski A, Murray G and Peck C. Modifying jaw muscle activity with physiotherapy exercises. Faculty Research Day, Faculty of Dentistry, The University of Sydney, September 2008. Westmead, NSW, Australia.

Wirianski A, Murray G and Peck C. Modifying jaw muscle activity with physiotherapy exercises. From Cell To Society 6, The University of Sydney, November 2008. Leura, NSW, Australia.

LITERATURE REVIEW

Introduction to Literature Review

The most prevalent orofacial pain condition, temporomandibular disorders (TMD), affects around 10% of the adult population. The clinical signs of TMD include pain, clicking and movement dysfunction of the temporomandibular joint (TMJ). Various treatment modalities have been proposed in an attempt to alleviate these often very debilitating symptoms.

Physiotherapy, including exercises, has been shown to be effective in the treatment of musculoskeletal pain and dysfunction in several areas of the body including the patellofemoral joint, shoulder and lumbar spine. An underlying principle in the successful treatment of these conditions has been the identification of imbalances in specific muscles or muscle groups. These imbalances have included weakness and loss of muscle bulk, changes in muscle recruitment patterns and changes in the timing of the recruitment patterns of specific muscles. Addressing these imbalances has resulted in the relief of symptoms and a return to more normal function of these areas. It has therefore been postulated that by alleviating the underlying biomechanical disturbances in individuals with musculoskeletal dysfunction there will be a relief of symptoms and a return to normal function.

One physiotherapy modality commonly used in restoring normal muscle bulk and strength is resistance exercise. Like the patellofemoral joint, shoulder and lumbar spine, the TMJ is another musculoskeletal subsystem. It is therefore plausible that appropriately applied physiotherapy modalities would also be successful in the treatment of some patients with TMD. Isokinetic resistance exercise has been applied to the TMJ for the relief of clicking,

however the mechanism behind the relief of clicking is yet to be elucidated. In order to determine which physiotherapy modality is most appropriate in improving dysfunction in particular groups of TMD patients it is first important to understand the functional anatomy of the jaws. This includes determining the characteristics of the normal movement patterns of the TMJ. It is also important to determine whether the motor control of the muscles of mastication that produces an individual's movement patterns can be influenced by physiotherapy modalities such as resistance exercise.

This thesis attempts to document the movement patterns of the TMJ in normal, asymptomatic volunteers along with the electromyographic (EMG) activity of the muscles which contribute to standardised movements. This thesis also attempts to document the effect of resistance exercise on the movement and/or EMG patterns in both the immediate timeframe and after four weeks of a home-based isotonic resistance exercise programme.

Functional Anatomy of the Masticatory System

An in-depth account of the functional anatomy of the masticatory system is beyond the scope of this thesis. A brief outline of the main relevant anatomical structures which impact on jaw movement follows. The reader is directed to the relevant references for more detail if required.

Temporomandibular Joint

The temporomandibular joint (TMJ) is a synovial joint formed by the articulation of the head of the mandible with the mandibular fossa and the articular tubercle of the temporal bone (Cunningham and Romanes, 1986, O'Rahilly, 1986) and the post-glenoid tubercle (Figure 1, O'Rahilly, 1986). An articular disc divides the joint into two compartments which have been suggested to function as an upper plane joint and a lower, smaller hinge

joint (Figure 1, Cunningham and Romanes, 1986, O'Rahilly, 1986). This structural design enables the TMJ to be capable of hinge-type movements (ginglymos) as well as gliding movements and it has therefore been classified as a ginglymodiarthroidial joint (Figure 1, Blasberg and Greenberg, 2003).

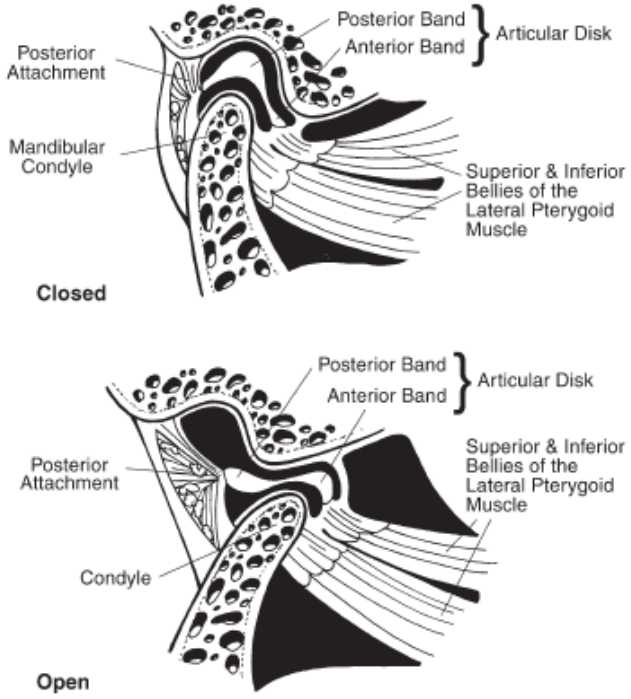

Figure 1: Schematic diagram of the lateral aspect of the right temporomandibular joint with the mouth closed (upper diagram) and mouth open (lower diagram). Note how the shape of the articular surfaces and the articular disc allow the joint to have hinge-like movements as well as gliding movements throughout its range of motion (Blasberg and Greenberg, 2003).

An articular capsule surrounds the TMJ and is attached to the margins of the articular area of the temporal bone above and the neck of the mandible below (Figure 2, Cunningham and Romanes, 1986). A lateral, triangular thickening of the articular capsule forms the lateral ligament (Figure 2, Cunningham and Romanes, 1986). The base of the lateral ligament is attached to the zygomatic process of the temporal bone and the tubercle at its

root while the apex is attached to the neck of the mandible on the lateral side (Figure 2, Cunningham and Romanes, 1986). The only true primary restraints of the TMJ are the articular capsule and its lateral ligament (Cunningham and Romanes, 1986). The sphenomandibular and stylomandibular ligaments also attach the mandible to the skull (Figure 2). The ligaments and the articular capsule provide little if any stability or strength to the TMJ with its integrity maintained primarily by the muscles of mastication (Cunningham and Romanes, 1986).

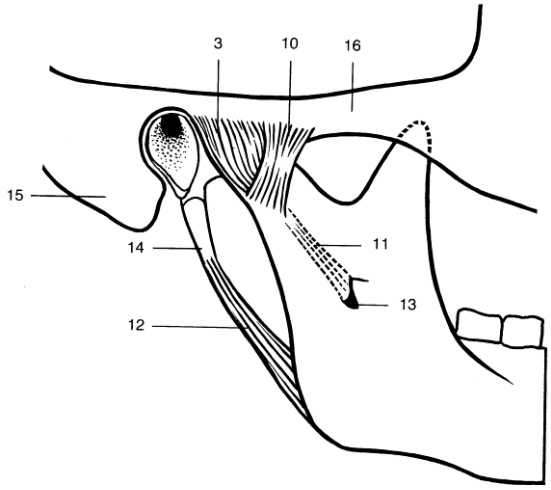

Figure 2: Schematic diagram of the lateral aspect of the right temporomandibular joint showing the relationship of the articular capsule (3) and the lateral ligament (10). Also depicted are the zygomatic arch (16), mastoid process (15), the styloid process (14) with the stylomandibular ligament (12) attached. The silhouette of the sphenomandibular ligament (11) attached to the ligular next to the mandibular foramen (13) can also be seen (Rohen and Yokochi, 1983).

Muscles of Mastication

The movements of the TMJ are primarily controlled by the coordinated actions of the four muscles of mastication, which include, masseter, temporalis, medial pterygoid and lateral pterygoid (Cunningham and Romanes, 1986, O'Rahilly, 1986). These muscles develop

from the mesoderm of the mandibular arch and are hence innervated by branches of the motor root of the mandibular division of the Trigeminal nerve (Cranial Nerve V3, Blasberg and Greenberg, 2003, Cunningham and Romanes, 1986, O'Rahilly, 1986). The muscles of mastication have a complex and diverse architecture (Schumacher, 1961) which provides multiple options for tendon pull (Hannam and McMillan, 1994) and hence TMJ movement. Theoretically the musculoskeletal system of the jaw has an infinite number of ways in which to use the muscles to perform jaw movements (Van Eijden et al., 1990). Biomechanical modelling has also shown that very similar jaw movements are possible with very different muscle activation patterns (Lobbezoo et al., 2004). Together these findings provide the jaw with a degree of mechanical redundancy. This underlies the suggestion that the muscles of mastication are capable of flexible patterns of activation depending on the functional demands placed upon them during normal movements and activities.

Masseter

The masseter muscle is a thick, quadrate muscle that arises from the inferior border of the medial aspect of the zygomatic arch (Figure 3, Cunningham and Romanes, 1986, O'Rahilly, 1986) with a complex multipennate architecture (Figure4, Brunel et al., 2003, Gaudy et al., 2000, Hannam and McMillan, 1994, Schumacher, 1961). It has been described as having two heads of origin (Cunningham and Romanes, 1986, Miller, 1991) but more commonly as having three heads of origin which divide it partially into superficial, intermediate and deep portions (Brunel et al., 2003, Gaudy et al., 2000, Hannam and McMillan, 1994, O'Rahilly, 1986).

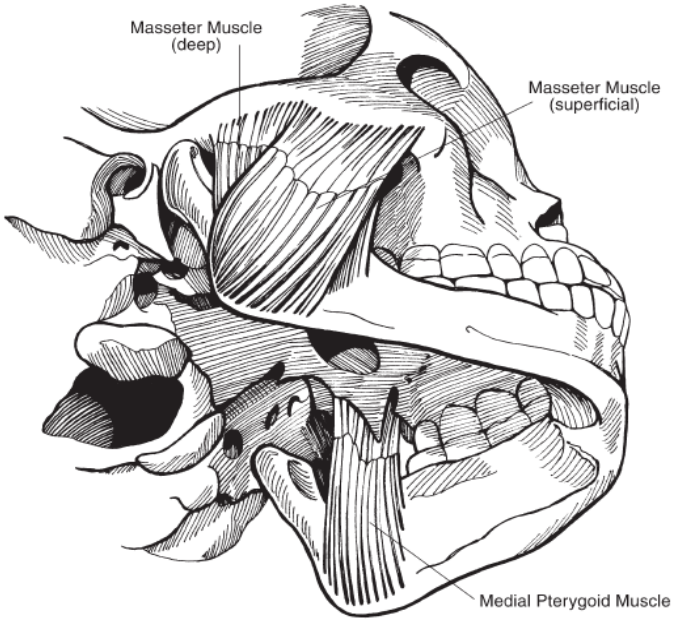

Figure 3: Schematic diagram of the oblique view of the right side of the skull depicting the superficial and deep heads of the masseter and the medial pterygoid muscles. The approximate alignment of the muscle fibres is also shown (Blasberg and Greenberg, 2003).

The superficial head of masseter travels inferiorly and posteriorly between the anterior two-thirds of the zygomatic arch and the lateral aspect of the mandible between the angle and the vertical ramus (Hannam and McMillan, 1994) at an angle of 58º (Gaudy et al., 2000) to 60º to the Frankfort horizontal plane (Brunel et al., 2003). The superficial head is made up of two alternating musculo-aponeurotic layers that further divide it into superficial and deep layers (Brunel et al., 2003). The superficial layer originates from the lateral most aponeurosis (Figure 4, Hannam and McMillan, 1994) and the angle of the mandible with its tendon inserting into the whole of the inferior border of the zygomatic bone (Brunel et al., 2003, Gaudy et al., 2000). In contrast, the deep layer originates from the zygomatic arch and its tendon inserts into the lateral aspect of the angle and vertical ramus of the mandible (Brunel et al., 2003, Gaudy et al., 2000).

The aponeurotic layer of the intermediate head of masseter arises from the central medial third and the lower border of the posterior third of the zygomatic arch (Hannam and McMillan, 1994, Miller, 1991). Along the inferior border of the zygomatic arch it is made up of strands that are separated by fleshy, thin, ribbon-like bands (Brunel et al., 2003). The muscle belly continues inferiorly to its insertion on the lateral aspect of the mandible with its fibres generally orientated at 90º to the Frankfort horizontal plane (Brunel et al., 2003). Two areas of insertion in the general area of the angle of the mandible have been described. The first is on the central part of the lateral aspect of the ascending ramus of mandible up to the level of the coronoid process (Hannam and McMillan, 1994, Miller, 1991). More recently, the insertion has been described as the inferior quarter of the lateral aspect of the mandibular ramus just above the tendinous insertion of the deep portion of the superficial masseter (Brunel et al., 2003). It is within the intermediate head of masseter where the majority of the variations in the muscle's morphology have been observed. In one-third of the dissected specimens the aponeurosis had the appearance of a continuous layer, while in edentulous specimens the muscle mass was very much reduced (Brunel et al., 2003).

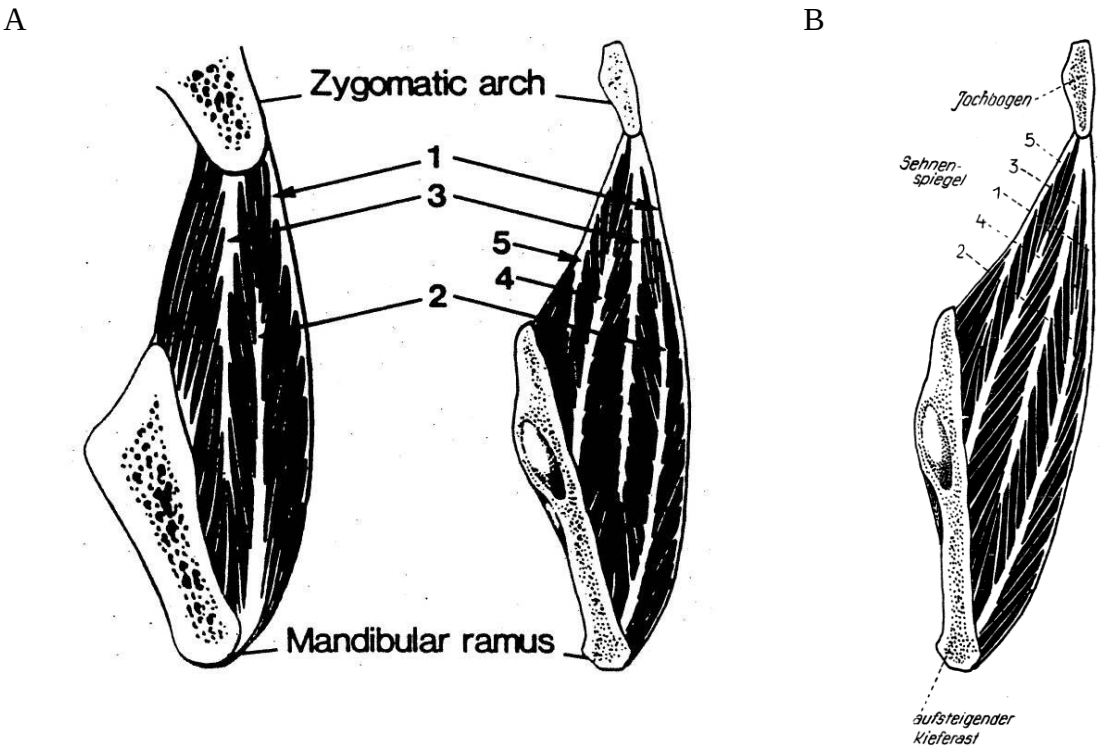

Figure 4: Schematic diagram of the coronal section through the right human masseter. A: Two reported patterns of aponeuroses (numbered 1 to 5, Hannam and McMillan, 1994). B: More detail of the pattern of pennation between the aponeuroses (Sehnenspiegel, numbered 1 to 5) showing the approximate alignment of the muscle fibres. Also labelled are the zygomatic arch (Jochbogen) and the mandibular ramus (Aufsteingender Kieferast, Schumacher, 1961).

The deep head arises from the deep aspect of the zygomatic arch and inserts into the upper part of the lateral aspect of the ascending ramus of mandible up to the coronoid process (Hannam and McMillan, 1994, Miller, 1991). It has been described as a muscular tendinous fan that is separated into an anterior portion and a posterior portion (Brunel et al., 2003, Gaudy et al., 2000, Hannam and McMillan, 1994). The fibres of the anterior portion are generally orientated at 90º-100º to the Frankfort horizontal plane and is made

up of a single layer. The posterior part is made up of three alternating muscular aponeurotic layers (Brunel et al., 2003, Hannam and McMillan, 1994).

When viewed in the frontal plane the different heads of masseter are described as being separated by thick, multileaved, internal aponeuroses aligned roughly in the parasagittal plane (Figure 4 and Figure 6, Brunel et al., 2003, Hannam and McMillan, 1994, Schumacher, 1961). Some of these aponeuroses traverse through masseter downwards from the zygomatic arch above and interleave between similar aponeuroses that traverse the muscle upwards from the mandible below to form septa in the posterior part of the masseter (Figure 4, Hannam and McMillan, 1994, Schumacher, 1961). It is these septa that provide the anchorage for most of masseter's muscle fibres (Figure 4 and Figure 6, Hannam and McMillan, 1994).

Masseter is not a muscle that consists of parallel fibres that insert at fixed pennation angles into parallel aponeuroses as is assumed by most generic muscle models (Hannam and McMillan, 1994). The multipennate arrangement of masseter's fibres can be seen when viewed coronally and from behind (Figure 4b and Figure 6, Brunel et al., 2003, Hannam and McMillan, 1994). Most of these oblique subsets of fibres insert into the adjacent interleaved aponeuroses while some insert into the bone of the zygomatic arch or the mandibular ramus (Figure 4b and Figure 6, Brunel et al., 2003, Hannam and McMillan, 1994). When viewed horizontally the muscle fibre subsets are aligned parallel and obliquely from the zygomatic arch to the mandible and also mediolaterally through the plane of view as they attach between the adjacent interleaved aponeuroses (Hannam and McMillan, 1994).

Regional differences have also been observed in terms of muscle fibre, sarcomere and tendon lengths (Hannam and McMillan, 1994, Van Eijden and Raadsheer, 1992). Muscle fibres were reported to be 35% longer anteriorly compared to posteriorly and 5% longer in the superficial layer compared to the deep layer. Although tendon lengths were reported to be similar throughout the superficial layer they were reported to be 35% longer than those of the deep layer, where they were shortest posteriorly. Sarcomere lengths were reported to be 6% longer superficially compared to the deep layer, with no differences between anterior and posterior regions (Hannam and McMillan, 1994, Van Eijden and Raadsheer, 1992).

Regional variability has also been observed in the task profiles of masseter's motor units (McMillan and Hannam, 1992, Ogawa et al., 2006). McMillan and Hannam (1992) divided the superficial and deep portions of the right masseter into quadrants and sampled single motor units in each of the eight quadrants during 13 voluntary jaw tasks. Most of the sampled motor units were observed to contribute in all of the tasks. However, motor unit firing was seen to be dependent on jaw position, the direction of effort, and the bite point along the tooth row (McMillan and Hannam, 1992). Also, the firing pattern of all the studied single motor units during each of the voluntary contractions varied between the performed tasks (McMillan and Hannam, 1992). It was therefore argued that this indicated changes in the excitatory drive leading to variations in the descending or efferent input to the trigeminal motoneurone pool dependant on which task was being performed (McMillan and Hannam, 1992).

Regional differences in motor unit function are also related to bite force direction and duration (Ogawa et al., 2006). Sixty five single motor units were sampled in the

superficial and deep heads of the right masseters of four subjects while simultaneously recording the duration of the bite force as well as its three dimensional direction. Each of the recorded motor units was found to have a specific firing range of bite force magnitude and direction as well as an optimum direction for recruitment which suggests that the contribution of motor units to the production of bite force is not uniform throughout the masseter. The differences in motor unit function between the superficial and deep heads of masseter were attributed to the structural and physiological differences between the two muscle heads (Ogawa et al., 2006).

The architectural complexity of masseter, with its three heads and the interleaved aponeuroses to which the majority of the muscle's fibres attach at varying angles of pennation, along with its task dependant efferent drive (McMillan and Hannam, 1992), would together explain the functional differences seen in EMG/muscle activation (Miller, 1991, Van Eijden et al., 1990). It would also allow for masseter to adapt appropriately during different functional tasks in order to move the jaw accurately and effectively.

Masseter is innervated by the masseteric nerve, a branch of the anterior trunk of cranial nerve V3 (CN V3), which reaches the deep surface of masseter after passing through the mandibular notch (O'Rahilly, 1986). The masseteric nerve separates the middle and deep portions of masseter and the masseteric artery separates the anterior and middle portions (Hannam and McMillan, 1994). Masseter is a powerful elevator of the mandible (Table 1) and can be palpated during clenching of the teeth (O'Rahilly, 1986). It also acts during protrusion and ipsilateral lateral movement of the mandible (Table 1).

Temporalis

The temporalis is a fan-shaped muscle that lies in the temporal fossa (Figure 5, Blasberg and Greenberg, 2003, Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, O'Rahilly, 1986). Superiorly it has a bony and a soft tissue attachment (Hannam and McMillan, 1994, O'Rahilly, 1986). The bony attachment is from the floor of the temporal fossa below the inferior temporal line, which includes the frontal, parietal, sphenoid, temporal and sometimes the zygomatic bones (O'Rahilly, 1986). The majority of the muscle fibres originate from the bony attachment with the soft tissue attachment being from the deep surface of the temporal aponeurosis or fascia (Hannam and McMillan, 1994, O'Rahilly, 1986).

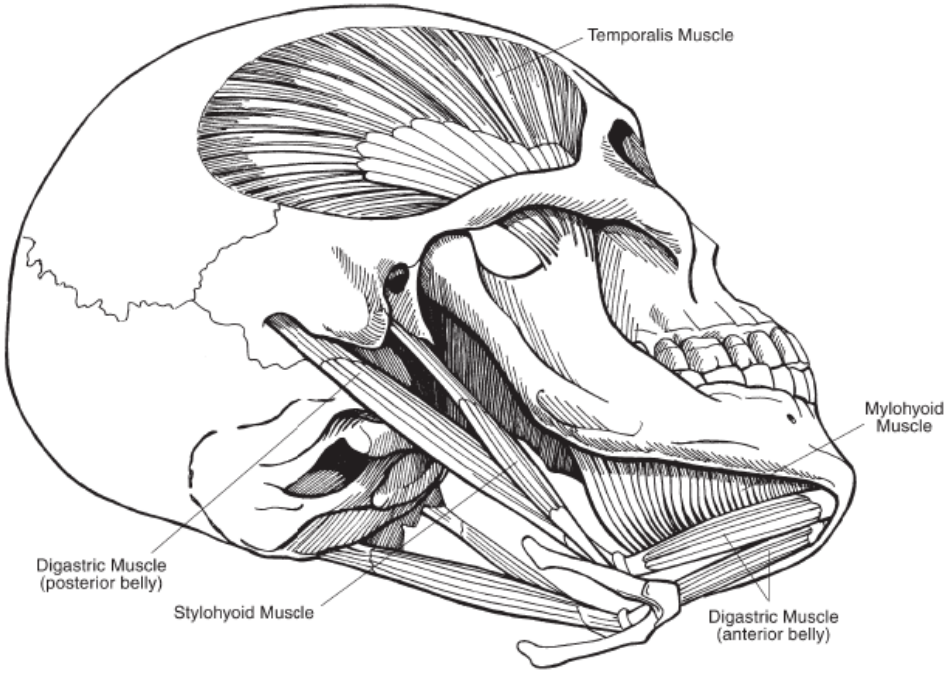

Figure 5: Schematic diagram of the oblique view of the right side of the skull depicting the superficial head of the temporalis muscle, the anterior and posterior bellies of the digastric, the mylohyoid muscle and the stylohyoid muscle. The approximate alignment of the muscle fibres is also shown (Blasberg and Greenberg, 2003).

Temporalis passes deep to the zygomatic arch (Figure 5) and has a complicated pattern of insertion into the medial surface, apex and the anterior and posterior border of the coronoid process and also the anterior border of the ramus of the mandible (Cunningham and Romanes, 1986, Hannam and McMillan, 1994, O'Rahilly, 1986). The deeper, more muscular fibres in the deep fascia attach to the medial aspect of the coronoid process and become more tendinous distally (Cunningham and Romanes, 1986, Hannam and McMillan, 1994). These deeper fibres reach down to the junction of the anterior border of the ramus and the body of the mandible, onto the mandibular crest inferiorly almost as far as the third molar (Cunningham and Romanes, 1986, Hannam and McMillan, 1994).

Temporalis has been described as having two different tendon arrangements. Firstly, a deep and superficial tendon of insertion each arising from one of its two heads respectively (O'Rahilly, 1986). Secondly, temporalis can have one central conspicuous tendon formed by the convergence of the muscle's fan-shaped collection of long fibres (Figure 6, Cunningham and Romanes, 1986, Hannam and McMillan, 1994). This single tendon extends superiorly into the muscle as an internal central aponeurosis which separates the muscle into its superficial and deep parts (Figure 6, Hannam and McMillan, 1994). In either case, and in conjunction with its two heads of origin, the temporalis has a resultant bipennate arrangement of its fibres (Figure 6), especially in the anterior region, when viewed in the frontal plane (Cunningham and Romanes, 1986, Hannam and McMillan, 1994). Fibre direction changes between the different regions of the temporalis. When viewed in the coronal plane, anterior fibres diverge to the central aponeurosis at low pennation angles, while medially the fibres fan out at different lengths and angles of pennation (Hannam and McMillan, 1994). In the parasagittal plane the fibres descend vertically in the anterior part of the muscle, becoming almost horizontal posteriorly, with

no obvious bipennate arrangement as the muscle is thin in this region (Hannam and McMillan, 1994).

Temporalis is innervated by the deep temporal branches of the anterior trunk of the mandibular nerve (CN V3, Cunningham and Romanes, 1986, O'Rahilly, 1986). The action of the temporalis is to maintain the resting posture of the mandible and to elevate the mandible into molar occlusion (Table 1). During closing of the mouth, the posterior fibres pull the condyle of the mandible backward from the articular tubercle and into the mandibular fossa (Table 1, Cunningham and Romanes, 1986, O'Rahilly, 1986). It also acts during ipsilateral lateral movement of the mandible (Table 1).

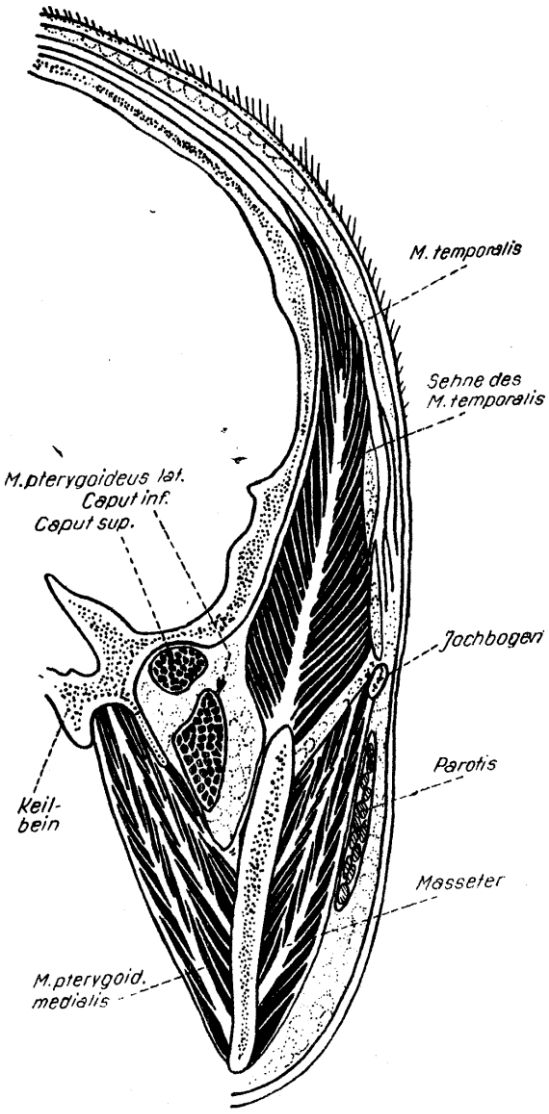

Figure 6: Schematic diagram of the coronal section through the vertical ramus of the right mandible from behind depicting the muscles of mastication. The muscles shown include: superficial and deep portions of temporalis (M. Temporalis), with its central aponeurosis or tendon (Sephne des M. Temporalis); masseter; medial pterygoid (M. Pterygoid medialis); and, the superior head (Caput sup.) and inferior head (Caput inf.) of the lateral pterygoid muscle. The approximate alignment of the muscle fibres is also shown, with the fibres of the lateral pterygoid travelling perpendicular to the plane of the page. Also labelled are the parotid gland (parotis), the zygomatic arch (Jochbogen) and the pterygoid plates (Keilbein, Schumacher, 1961).

Medial Pterygoid

The medial pterygoid is a rectangular muscle that lies on the medial aspect of the ramus of the mandible (Figure 7 and Figure 8). It possesses two heads of origin. The larger, deep head arises from the medial surface of the lateral pterygoid plate and the pyramidal process of the palatine bone. The smaller, superficial and more inferior head arises from the pyramidal process of the palatine bone and the maxillary tuberosity. These two heads embrace the inferior head of the lateral pterygoid and unite (Figure 8, Cunningham and Romanes, 1986, Hannam and McMillan, 1994). The medial pterygoid passes downward, backward and laterally to insert into a rough area between the mandibular foramen and the angle of the mandible (Figure 3, Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, O'Rahilly, 1986) with its fibres running nearly parallel to those of the superficial fibres of masseter (Cunningham and Romanes, 1986). The two heads can be considered as separate muscles (O'Rahilly, 1986).

The medial pterygoid has been described as having at least six and occasionally up to eight aponeuroses which resemble those of masseter (Figure 6 and Figure 7, Hannam and McMillan, 1994). This interleaved organisation of aponeuroses positioned closely together creates a multipennate arrangement of short muscle fibres (Figure 6 and Figure 7, Hannam and McMillan, 1994). Along with the angulation of the medial pterygoid and the orientation of its attachment sites these morphological features offer little scope to vary its angle of pull when viewed in the frontal plane (Figure 6 and Figure 7, Hannam and McMillan, 1994). Viewed in the parasagittal plane, however, the medial pterygoid has a relatively vertical origin and a wide insertion on the mandible (Hannam and McMillan, 1994). These features provide fibre angulations that are divergent enough to suggest the ability to produce different actions on the mandible (Hannam and McMillan, 1994). In

particular muscle tension could be generated along at least two principal axes, one anteriorly and directed upward, medially and forward, and the other posteriorly and directed upward, medially and more forward (Hannam and McMillan, 1994).

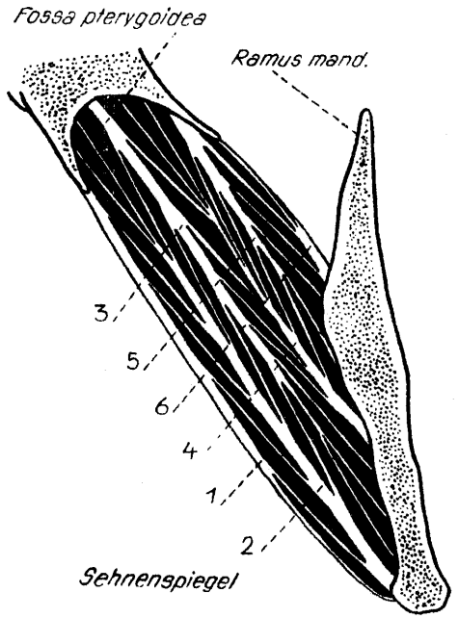

Figure 7: Schematic diagram of the coronal section through the right medial pterygoid showing the interleaved aponeuroses (Sehnenspiegel, numbered 1 through 6) resulting in the multipennate structure with short muscle fibres. Also labelled are the pterygoid fossa (Fossa pterygoidea) and the mandibular ramus (Ramus mand., Schumacher, 1961).

The medial pterygoid is innervated by a branch from the mandibular nerve (CN V3, Cunningham and Romanes, 1986, O'Rahilly, 1986). The action of medial pterygoid is summarised in Table 1. Acting as a synergist to masseter it elevates the mandible (Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, O'Rahilly, 1986). Acting together with lateral pterygoid it protrudes the mandible (Cunningham and Romanes, 1986, Hannam and McMillan, 1994, O'Rahilly, 1986) and produces contralateral

lateral movement of the mandible (Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, O'Rahilly, 1986).

Lateral Pterygoid

The lateral pterygoid occupies the infratemporal fossa and possesses two distinct heads of origin (Figure 8, Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, O'Rahilly, 1986). The smaller, superior head with its thin, flat band of fibres (Hannam and McMillan, 1994), arises from the infratemporal ridge and infratemporal surface and crest of the greater wing of sphenoid (Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, O'Rahilly, 1986). The fibres of the superior head travel posteriorly, laterally and caudally to converge on their insertions into the anterior surface of the articular capsule, the anterior margin of the articular disc and the fovea on the front of the neck of the mandible (Figure 1 and Figure 8, Hannam and McMillan, 1994, Miller, 1991, Widmalm et al., 1987). Described as being three times larger than the superior head (Miller, 1991) and having twice the cross sectional area (Hannam and McMillan, 1994) the inferior head arises from the lateral surface of the lateral pterygoid plate (Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, O'Rahilly, 1986) as well as the pyramidal process of the palatine bone and the maxillary tuberosity (Miller, 1991). The fibres of the inferior head converge posteriorly and laterally to insert into the fovea on the front of the neck of the mandible (Figure 1 and Figure 8, Miller, 1991, O'Rahilly, 1986). Together, the two heads of origin have a curved, fan-shaped attachment that sweeps through an arc of near vertical fibres in the case of the superior head to near horizontal fibres in the inferior head (Figure 8, Hannam and McMillan, 1994). This divergent alignment of its fibres allows the lateral pterygoid a varied line of pull dependent on function. This has lead some authors to

describe the two heads as being capable of acting independently (Miller, 1991, O'Rahilly, 1986).with the superior head having more biomechanical advantage to close the mandible, and the inferior head being more biomechanically efficient at lowering the mandible and for translation of the condyle (Miller, 1991).

In contrast, morphologically the two heads of lateral pterygoid have been reported to converge towards their insertions where their individual fibres become difficult to separate (Hannam and McMillan, 1994). This arrangement would explain more recent findings that the two heads of the lateral pterygoid could be considered as a system of fibres that act as one muscle which is capable of producing varying amounts of evenly graded levels of activity throughout its entire range dependent on the biomechanical demands of specific tasks (Hannam and McMillan, 1994, Murray et al., 2007, Murray et al., 2004). Having a wide arc of origin converging to a relatively small insertion of interdigitating fibres would facilitate the fibres of lateral pterygoid to act sequentially when their angle of pull coincided with the respective fibre's optimal mechanical advantage which would produce the desired movement.

Unlike the other muscles of mastication the internal architecture of lateral pterygoid is quite simple (Figure 6 and Figure 8). Comprised of long fibres with their line of action parallel to the muscle belly, the lateral pterygoid is most suited to shortening over longer distances than either masseter of medial pterygoid. This arrangement offers greater propensity for near-isotonic contractions (Hannam and McMillan, 1994).

The lateral pterygoid is innervated by a branch from the anterior trunk of the mandibular nerve (CN V3), which may arise from the masseteric or buccal nerves (Cunningham and Romanes, 1986, O'Rahilly, 1986).

Table 1 summarises the actions of the lateral pterygoid. Due in part to its attachment to the articular disc and anterior portion of the articular capsule, the lateral pterygoid is the chief protractor of the articular disc and hence the mandible by drawing the head of the mandible and the disc forwards on the articular tubercle (Cunningham and Romanes, 1986, Huang et al., 2005, Miller, 1991, Murray et al., 1999, O'Rahilly, 1986). Mouth opening is produced by the rotational pull of the lateral pterygoid and anterior belly of digastric muscles (O'Rahilly, 1986).

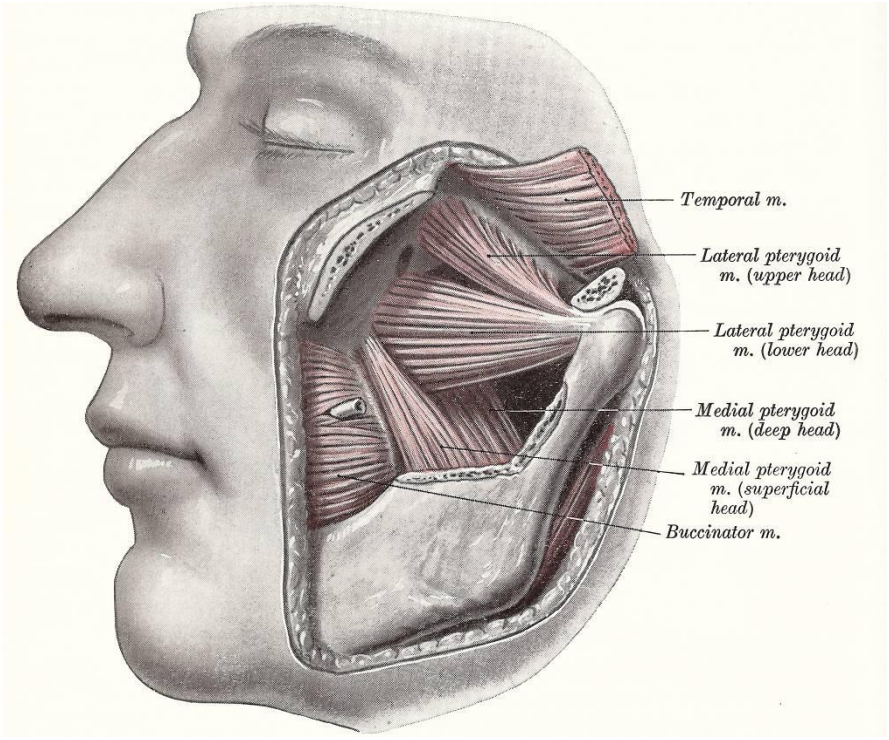

Figure 8: Drawing of the lateral view of the left side of the face with the region of the infratemporal fossa exposed, with the zygomatic arch removed and the temporalis (Temporal m.) reflected. Depicted are the superior (upper) head and the inferior (lower) head of the lateral pterygoid muscle, and the deep and superficial heads of the medial pterygoid muscle. The buccinator with the parotid duct is also drawn. The approximate alignment of the muscle fibres is also shown (Johnston et al., 1958).

The superior head of lateral pterygoid can also act to stabilise the articular disc and condylar head and even maintain them together with the joint capsule as they move anteriorly and slightly superiorly during protrusion and opening in order to maintain the position of the condyle and disc along the articular surface of the temporal bone during these movements (Miller, 1991). Acting together with medial pterygoid it also produces contralateral lateral movement of the mandible (Cunningham and Romanes, 1986, Miller, 1991, O'Rahilly, 1986). When acting unilaterally both the superior and inferior heads of the lateral pterygoid act to produce contralateral lateral movement of the jaw (Huang et al., 2005, Murray et al., 1999, Widmalm et al., 1987).

Other Muscles Acting on the TMJ

Some of the muscles of the suprahyoid group also contribute to the function of the TMJ. Although beyond the scope of this thesis it is worth noting that the infrahyoid muscles could be considered as acting indirectly on the mandible by stabilising the hyoid bone in order for the suprahyoid muscles to act effectively on the mandible.

Digastric

The digastric muscle consists of two bellies united by an intervening tendon (Figure 5, Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, O'Rahilly, 1986). The shorter anterior belly is attached to the digastric fossa on the lower border of the mandible close to the symphysis and is directed backward and downward (Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, O'Rahilly, 1986). The middle tendon is attached to the body and the greater horn of the hyoid bone by aponeurotic fibres of the cervical fascia (Hannam and McMillan, 1994, O'Rahilly, 1986) which form a fibrous loop containing a synovial sheath that is attached at the junction of the body with the greater horn of the hyoid (Cunningham and Romanes, 1986, Hannam

and McMillan, 1994). The larger posterior belly then travels upward and backward to insert in the mastoid notch of the temporal bone (Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, O'Rahilly, 1986). In contrast to the classic twin belly description of the digastric, there have also been reports of unilateral and bilateral anatomical anomalies in the anterior belly of digastric, including four separate tendons of insertion into the mandible unilaterally (Celik et al., 2002), a triangular shaped anterior belly inserting into the raphe of the mylohyoid muscle (Liquidate et al., 2007), supernumerary anterior bellies inserting into the contralateral intermediate tendons, the mylohyoid raphe and the hyoid bone (Liquidate et al., 2007) and accessory bellies arranged in a cross formation (Aktekin et al., 2003).

The two bellies of the digastric have a different neurological supply due to their differing embryological development (Cunningham and Romanes, 1986, Hannam and McMillan, 1994). The anterior belly develops from the first embryological pharyngeal arch and is therefore innervated by the mylohyoid branch of the inferior alveolar nerve, a branch of CN V3 (Cunningham and Romanes, 1986, O'Rahilly, 1986). The posterior belly develops from the second pharyngeal arch and is hence innervated by the facial nerve (Cranial Nerve VII, Cunningham and Romanes, 1986, O'Rahilly, 1986).

The actions of the anterior belly of digastric are summarised in Table 1. The anterior belly of digastric muscle assists the lateral pterygoid to rotate the condyle of the mandible in the mandibular fossa during early mouth opening by pulling the chin backward and downward while the hyoid is held stable by the infrahyoid muscles (Cunningham and Romanes, 1986, Miller, 1991, O'Rahilly, 1986). In particular the orientation of the fibres of the anterior belly of digastric are well placed to produce a vector that would open and retrude the

mandible, while the fibre orientation of the posterior belly would produce a vector that would elevate the hyoid bone (Miller, 1991). Although difficult to confirm, it has been postulated that both bellies of digastric act simultaneously for most tasks as this would be necessary to open the mandible against a fixed hyoid or alternatively elevate the hyoid (Cunningham and Romanes, 1986, Hannam and McMillan, 1994). The synchronised activity of both bellies has been confirmed with EMG studies during movement, chewing and swallowing (Hannam and McMillan, 1994, Munro, 1972, Munro, 1974) with occasional asynchronous activity also being reported (Hannam and McMillan, 1994, Munro, 1974, Widmalm et al., 1987). Having separate innervations for each belly offers the potential for differential activation of the digastric muscle (Hannam and McMillan, 1994).

Mylohyoid

The mylohyoid muscle lies above the anterior belly of digastric and has a similar development and innervation. It arises from the mylohyoid line on the internal surface of the mandible, extending from the last molar tooth almost to the symphysis menti. Most of its fibres are directed medially where they insert into a median tendinous raphe. The posterior fibres insert into the body of the hyoid bone. The two mylohyoid muscles form the muscular floor of the front of the mouth (Figure 5, O'Rahilly, 1986).

The mylohyoid muscle is innervated by the mylohyoid branch of the inferior alveolar nerve, which is a branch of CN V3 (Cunningham and Romanes, 1986, O'Rahilly, 1986).

The two mylohyoids form a muscular sling that supports the tongue. Contraction of the mylohyoids elevates the floor of the mouth making it more shallow. This elevates the tongue and, if the teeth are held in occlusion, produces an increase in pressure on the

tongue which forces it backward, as occurs during swallowing. Thus the mylohyoids force the contents of the mouth from the oropharynx to the laryngopharynx (O'Rahilly, 1986). Through their attachment to the hyoid bone, the posterior fibres of the mylohyoid muscles would also act to depress the mandible during opening while the hyoid was held stable (Table 1, Blasberg and Greenberg, 2003, O'Rahilly, 1986) and may even contribute to retrusion of the mandible (Blasberg and Greenberg, 2003).

Geniohyoid

The geniohyoid muscle is situated superior to the mylohyoid. It originates from the inferior genial tubercle behind the symphysis of the mandible and travels posteriorly to insert into the front of the body of the hyoid bone. It is in contact or fused with the muscle of the opposite side (O'Rahilly, 1986).

Geniohyoid is innervated by fibres from the first cervical ventral ramus (C_1) through the hypoglossal nerve (CN XII, Cunningham and Romanes, 1986). The geniohyoid protrudes the hyoid bone and thereby contributes to shortening the floor of the mouth. With the hyoid bone stabilised, the geniohyoid would also assist in the depression of the mandible while the hyoid was held stable (Table 1, Blasberg and Greenberg, 2003, O'Rahilly, 1986) and may even contribute to retrusion of the mandible (Blasberg and Greenberg, 2003).

Electromyography

In the laboratory setting electromyography is frequently used to assess muscle recruitment, both qualitatively and quantitatively, as it provides a measure of the electrical activity in muscles (Basmajian and Luca, 1985, Miller, 1991). Excitation of a muscle fibre by its motor neurone elicits transient changes in the membrane sodium channels causing a brief (1 to 2 milliseconds) depolarisation of the muscle fibre of around 50 to 80 millivolts (Miller, 1991). This depolarisation is then propagated as a wave of electrical activity

throughout the innervated muscle fibre (Miller, 1991) causing it to contract. The resultant potential difference can be recorded extracellularly in microvolts (Miller, 1991). Commonly, two electrodes are used to record the electromyographic (EMG) signal from each individual muscle. In this bipolar electrode arrangement the EMG signal is first detected by the electrode closest to the signal source before it reaches the second electrode. The potential difference between each electrode and a distant ground electrode is then measured. One potential difference is then subtracted from the other and amplified. This differential amplification results in amplification of the signals generated close to the electrodes and reduces spurious signals from distant sources, electronic noise and cross-talk (Fridlund and Cacioppo, 1986, Miller, 1991).

The most effective mode of quantifying the EMG activity of active muscles is to first rectify the raw EMG signal and then to further process the rectified signal with an assimilated integration in order to produce a smooth curve, the shape of which applies to the relative area at successive time points within the rectified signal (Miller, 1991). Digital Butterworth filtering can also produce a smoothed curve that is reflective of the rectified EMG signal. The Butterworth filter sacrifices rolloff steepness for monotonicity in the passband (the range of frequencies or wavelengths that can pass through a filter without being attenuated) and the stopband (the range of frequencies that are attenuated to very low levels or prevented from passing through a filter). The Butterworth filter thus provides smoothing, and is used because it suits applications that require preservation of amplitude linearity in the passband region. It is this feature that makes the Butterworth filter an ideal candidate for conditioning the EMG signal (The Mathworks Inc, 2000).

Despite its widespread use, EMG has some important limitations that need to be considered. The main factors that can affect the quality of EMG recordings include the signal-to-noise

ratio, cross-talk and distortion (Long, 2004). Signal noise is that part of the EMG signal that is not part of the wanted EMG signal and has many sources, which include, mains electricity lines, computers and their monitors, especially cathode ray tubes, the EMG testing system (Fridlund and Cacioppo, 1986) and even the electrical activity of the subject's heart (Drake and Callaghan, 2006). Movement artefact is another source of noise, either from the electrodes moving on the skin, movement of the cables connecting the electrodes to the amplifier or the subject's movements during data collection. The EMG signal is also inherently unstable as it is a quasi-random discharge of action potentials from the muscle fibres in a frequency range of several hertz to over 2000 hertz (Fridlund and Cacioppo, 1986). It is important to minimise the recording of these unwanted frequencies through the use of appropriate electrical shielding from and filtering of this interference (Fridlund and Cacioppo, 1986).

Cross-talk refers to the inadvertent sampling of electrical activity from adjacent muscles due to the inaccuracy of the electrode placement. This can be minimised through accurate and reproducible placement of the electrodes. It is suggested that bipolar electrodes should be placed parallel to the course of the muscle fibres in close proximity to the underlying muscle with minimal intervening tissue (Fridlund and Cacioppo, 1986).

Recently, it has been shown that by utilising the EMG waveform of the anterior temporalis, and masseter muscles bilaterally, it is possible to predict typical jaw movements such as left and right lateral movement and protraction 96% of the time during a simulated bruxing task (Long, 2004). Thus these muscles, which are the most accessible for recording with surface electrodes, seem to be sensitive to changes in jaw movements and would be worthy of recording when assessing if exercise training changes muscle activation strategies for typical jaw movements. The anterior digastric is also easily accessible with surface

electrodes and has a force vector that provides some opening of the jaw during lateral movements, in order to avoid accidental tooth contact during the movement (Miller, 1991).

Jaw Movements

The overall articular function of the TMJ is derived from the interaction of soft and hard tissue constraints, such as muscle activity, articular morphology and dental contact (Lobbezoo et al., 2004) and also the intrinsic elasticity or tightness of the soft tissues surrounding the TMJ. The resultant action of these constraints can be active, passive or a combination of both. Active constraints such as muscle activity produce jaw movement or can act on other soft tissues that they insert into, such as the joint capsule (Christo et al., 2005, Widmalm et al., 1987) or articular disc (Murray et al., 2004, Widmalm et al., 1987). Passive constraints such as the articular morphology, dental contact or tight soft tissues may work to guide the direction of the jaw movement in a particular functional or even parafunctional direction. The specific movements of the TMJ however are controlled more by the action of the muscles rather than by either the shape of the articular surfaces or the ligaments (Cunningham and Romanes, 1986, O'Rahilly, 1986). The chief movements of the TMJ and the chief factors responsible for each of the movements are summarised in Table 1.

Mandibular Movement	Chief Factors Responsible for Movement		
	From O'Rahilly (1986)	**From Cunningham (1986)**	**From Miller (1991)**
Depression	• Lateral Pterygoid • Digastric • Mylohyoid • Geniohyoid • Gravity	• Lateral Pterygoid • Digastric acting through a fixed hyoid bone (infrahyoid muscles)	• Inferior head of lateral pterygoid • Anterior Digastric
Elevation	• Temporalis • Masseter • Medial Pterygoid	• Temporalis • Masseter • Medial pterygoid	• Temporalis • Masseter • Medial pterygoid
Protrusion	• Lateral Pterygoid • Medial Pterygoid • Masseter	• Lateral pterygoid, and to a lesser extent and less effectively: Medial pterygoid • Superficial fibres of masseter which also prevent lateral pterygoid opening the mouth	• Inferior head of lateral pterygoid bilaterally • Masseter
Retraction	• Posterior Temporalis	• Posterior fibres of temporalis	• Posterior temporalis • Anterior digastric
Lateral movement	• Ipsilateral Temporalis Masseter • Contralateral Medial Pterygoid Lateral Pterygoid	• Grinding movements are produced by the muscles of opposite sides acting alternately	• Ipsilateral Temporalis Masseter • Contralateral Medial Pterygoid Inferior head of Lateral Pterygoid

Table 1: The chief movements of the mandible and the chief factors responsible for each of the movements (modified from (Cunningham and Romanes, 1986, Miller, 1991, O'Rahilly, 1986).

Neural Influence on Muscle Activity

Voluntary control of jaw muscles derives from the cerebral cortex. There are a number of factors that influence the motor control of muscles and hence their ability to produce forces and movement. Two in particular include, the sensory innervation of the surrounding anatomical area in which the particular muscle(s) are acting and the effect of pain on this muscular activity, and secondly, training and its effects on neural plasticity. These factors are significant to the question as to whether therapeutic jaw exercises can influence jaw muscle activity patterns.

Sensory Innervation and Pain

Using the human thumb as an example during a weight matching task completed by the flexor pollicis longus (FPL), anaesthesia of the skin of the thumb results in subjects perceiving a target weight to be significantly heavier than when not anaesthetised (Kilbreath et al., 1995). This indicates that the skin afferents play a role in modulating the force produced by the FPL during a weight matching task (Kilbreath et al., 1995).

Comparing EMG recordings of masseter and anterior temporalis muscles during maximal jaw clenching with and without anaesthesia of the upper canines showed an increase in integrated EMG activity in both the masseter and anterior temporalis following anaesthesia (Manns et al., 1991). An even greater increase in EMG activity was seen when both the upper and lower canines were anaesthetised (Manns et al., 1991). This suggested that the periodontal receptors provide negative feedback to the motoneurones of the jaw elevator muscles during maximal jaw clenching (Manns et al., 1991).

In the lumbar spine, chronic low back pain and experimentally induced muscular pain have been shown to change the temporal characteristics and amplitude of EMG activity in the

transversus abdominis muscle with these changes persisting following the resolution of the experimentally induced pain (Hodges et al., 2003, Hodges and Richardson, 1996). This single acute onset of pain was postulated as a mechanism that could potentially predispose individuals to ongoing symptoms or future exacerbation (Hodges et al., 2003).

Recently it has also been shown that using capsaicin to induce experimental pain in the tongue results in poorer performance in a novel tongue-protrusion task compared to pain-free trials of the same task (Boudreau et al., 2007). Furthermore, performance improvements over the fifteen minutes duration of the tongue-protrusion task were less during the capsaicin induced pain trials compared to the pain-free trials (Boudreau et al., 2007).

It is plausible that other sensory afferents that innervate the TMJ and other oral and facial structures also play a role in modulating the forces produced by the muscles of mastication. This could potentially affect the control of the muscles acting on the TMJ and thereby influence the function of the TMJ, especially in patients with TMD.

Neural Plasticity

Neural plasticity is the ability of the nervous system to adapt by altering its structure and function in response to motor experiences, such as skill, strength and/or endurance training. These alterations in structure and function can occur in the motor cortex (Adkins et al., 2006, Boudreau et al., 2007, Sanes and Donoghue, 2000), the spinal cord (Adkins et al., 2006, Wolpaw and Tennissen, 2001), peripheral motoneurones, in relation to motor unit firing and recruitment, and in the reflexes (Bawa, 2002). Furthermore, the acquisition and maintenance of normal motor performance as well as the dysfunctional movements associated with disease involve activity dependent plasticity at multiple sites throughout

the central nervous system, including various areas in the brain and spinal cord (Sale and MacDougall, 1981, cited in McConnell, 2002, Wolpaw and Tennissen, 2001). It is therefore plausible that exercise therapies may indeed target muscle recruitment patterns by changing parameters such as timing, duration and amplitude of muscle activity.

Inputs from the cortex and periphery are capable of causing both short-term and long-term changes in the spinal cord that affect its output (Wolpaw and Tennissen, 2001). Spinal cord plasticity induced by inputs from c-fibres contributes to the syndromes of spontaneous pain, abnormal sensitivity to both noxious and innocuous stimuli, and referred pain (Wolpaw and Tennissen, 2001) which are known to have a significant role in the development and persistence of TMD (Dworkin and Massoth, 1994, Suvinen et al., 2005).

In spinal cord injured patients with incomplete lesions, treadmill training increased walking ability, producing greater speed, strength, coordination and endurance as well as reducing their need for assistive devices (Wolpaw and Tennissen, 2001). These functional improvements were found to be specific to the task that was trained.

Long term effects of the descending inputs on the spinal cord help establish and maintain spinal cord function in a state that is most amenable to effective motor performance (Wolpaw and Tennissen, 2001). Task-specific and activity dependent plasticity is driven by descending and peripheral inputs which shape and continuously modify spinal cord function throughout life (Wolpaw and Tennissen, 2001). Training aimed at the gradual acquisition of new motor tasks causes reductions in spinal reflexes leading to a reduction in the direct peripheral influence on motoneurones which in turn lead to increased cortical control, thus allowing more precise movement (Wolpaw and Tennissen, 2001).

In the orofacial region, short-term training can lead to plasticity in the primary motor cortex (M1), particularly in the tongue (Boudreau et al., 2007). Using transcranial magnetic stimulation (TMS) and measuring motor evoked potentials (MEPs) from the tongue musculature it was found that there was a significant enhancement of the MEPs following fifteen minutes training of a novel tongue-protrusion task, with a concomitant small decrease in the tongue M1 threshold (Boudreau et al., 2007). These results suggested that a short bout of training is capable of producing rapid neuroplasticity of the tongue M1 (Boudreau et al., 2007). In addition, capsaicin induced experimental pain of the tongue interfered with these neuroplastic changes (Boudreau et al., 2007).

Temporomandibular Disorders

Temporomandibular disorders (TMD) are the most prevalent chronic orofacial pain conditions and affect approximately 10% of the adult population (Drangsholt and LeResche, 1999). Some reports of up to 12% prevalence amongst clinical cases and up to 26% prevalence in the community have also been published for the muscle disorders subgroup (Group 1) of TMD as defined by the Research Diagnostic Criteria for TMD (RDC/TMD; (Dworkin and LeResche, 1992). The clinical signs and symptoms of TMD include (but are not limited to): TMJ and/or jaw muscle pain, clicking in the area of the TMJ, and TMJ movement dysfunction. TMD have been characterised as a collective term that encompasses a number of clinical presentations that involve the TMJ and its associated structures and the muscles of mastication (Michelotti et al., 2005). TMD can be categorised into two groups (Michelotti et al., 2005):

- Specific where there is an underlying pathology such as a neoplasm, inflammation, growth disturbance or systemic disease, which results in the manifestation of the clinical signs and symptoms;

- Non-Specific which is considered a musculoskeletal disorder with an unknown aetiology.

The non-specific group of TMD can be further categorised into three main diagnostic subgroups as defined by the RDC/TMD (Dworkin and LeResche, 1992, Lobbezoo et al., 2004):

Group I: Muscle disorders.

Group II: Disc displacements.

Group III: Arthralgia, arthritis, and arthrosis.

Since TMD often have high levels of impairment of jaw function (Stegenga et al., 1993), strategies which modify motor activity to improve function need to be developed and subsequently integrated into clinical management.

Management of Temporomandibular Disorders with Physiotherapy

The management of TMD is generally directed at reducing pain and inflammation, reducing psychosocial stressors, increasing muscle strength, increasing range of motion and improving bite comfort (Dahlstrom, 1992, Stohler and Zarb, 1999) and minimizing future occurrences or exacerbations of the symptoms. Physiotherapy is a frequent component in the management of TMD patients (Glass et al., 1993, Michelotti et al.,

2005). The general objectives of physiotherapy are to reduce pain and discomfort and include altering functional parameters such as reducing muscle tone, increasing muscle strength, modifying loading patterns and improving jaw and joint function (Michelotti et al., 2005). Physiotherapy utilises various modalities in attempting to achieve these objectives, including: manual therapy, which encompasses joint manipulation, joint mobilisation and soft tissue massage; electrophysical agents, such as, ultrasound, moist heat, cryotherapy, low level laser, transcutaneous electrical nerve stimulation (TENS) and interferential; patient education of the condition and its management; and, exercise prescription (For review see Medlicott and Harris, 2006).

Dentists readily refer TMD patients for physiotherapy and utilise various physiotherapeutic modalities in their management of patients with TMD. In their study of the treatments used by General Practice and Specialist Dentists in the United States, Glass et al., (1993) found that physiotherapy modalities were commonly prescribed for patients with myofascial pain disorders. In particular, General Dentists prescribed thermal packs to 27% of patients and referred 10% of patients to Physiotherapists, while Specialist Dentists prescribed thermal packs to 28% of patients and ultrasound to 7% of patients and referred 17% of patients to Physiotherapists (Glass et al., 1993). Physiotherapy is often chosen as a treatment for dysfunctions of the orofacial region because it is relatively simple and low cost, it is reversible and non-invasive, and, it allows for easy patient education of the management program which facilitates a self-management approach (Michelotti et al., 2005).

Therapeutic Exercise for the Treatment of Musculoskeletal Conditions

The prescription of exercise is an important modality for the treatment of musculoskeletal conditions by Physiotherapists. Muscle training has been found to be specific to limb position (Doucette and Child, 1996, Sale and MacDougall, 1981, cited in McConnell, 2002) and the performed task (Adkins et al., 2006, Sanes and Donoghue, 2000). Furthermore, training has been reported to cause changes within the nervous system that may allow an individual to coordinate better the activation of muscle groups (Sale and MacDougall, 1981, cited in McConnell, 2002). It is therefore important to ensure that the prescribed exercises are task specific and address the musculoskeletal dysfunction in order to facilitate the most appropriate recovery of normal function. To this end, exercise and its effects on muscle activation patterns have been studied in various central and peripheral joints. The following is a brief summary of the effects of specific exercise programmes that have attempted to address musculoskeletal dysfunction in the knee, shoulder and the lumbar spine.

Vastus Medialis Oblique and Patelofemoral Joint Pain

Many factors contribute to the normal, pain free functioning of the patellofemoral joint, including: intrinsic and extrinsic structural factors of the femur, patella and hip; soft tissue tightness in the structures surrounding the knee; muscle imbalances, and; poor foot biomechanics (McConnell, 2002). In terms of the contribution of the muscles around the knee joint, the role of the vastus medialis oblique (VMO) muscle is to maintain the centred position of the patella in the femoral trochlea during active knee extension (McConnell, 2002). In patients with patellofemoral pain it has been postulated that a weakness or recruitment dysfunction of the VMO leads to maltracking of the patella in the femoral

trochlea. In the majority of patients with patellofemoral pain there is a delayed onset of the EMG activity of the VMO compared to the vastus lateralis (VL) muscle in 67% of subjects when going up stairs and in 79% of subjects when going down stairs (Cowan et al., 2001). Delayed onset of the VMO in relation to VL could predispose the patella to be displaced laterally, increasing the patellofemoral contact pressures between the lateral facet of the patella and the medial aspect of the lateral femoral condyle, and thereby causing pain.

Addressing the weakness and recruitment dysfunction of the VMO has been shown to improve the symptoms of patellofemoral pain and return the patient to normal function. In a study of 65 volunteers diagnosed with Patellofemoral Pain Syndrome, 35 subjects were randomly allocated to a Physiotherapy Treatment Group and 30 were allocated to a Placebo Group. Standardised treatment protocols were applied once per week for six weeks. The active treatment consisted of progressive functional retraining of the VMO with dual channel EMG biofeedback along with strengthening the gluteal muscles and taping the patella. The Placebo Group received sham patella taping, inoperative ultrasound and light application of a non-therapeutic gel. Subjects with patellofemoral pain who underwent physiotherapy intervention for six weeks, showed a resolution of the delay in the firing of VMO relative to VL with concomitant improvement in pain and function compared to the placebo group (Cowan et al., 2002).

As in the patellofemoral joint, specific movements of the TMJ are controlled by the combined action of the muscles (Cunningham and Romanes, 1986, O'Rahilly, 1986). It is therefore plausible that a change in the onset of firing of particular muscles in the TMJ may predispose individuals to movement dysfunction of the jaw or even symptoms of TMD.

The Rotator Cuff and Shoulder Pain and Dysfunction

The functional requirement of the shoulder complex is to maintain maximum mobility for the upper limb (Ginn et al., 1997). This is achieved at some expense of passive joint stability. In most joints the passive restraining structures, including the ligaments and joint capsule, maintain the congruency of the joint and prevent excessive joint movement, which together with the action of the muscles facilitate appropriate joint function. In order to fulfil the requirement throughout its large range of motion, the passive restraining structures of the shoulder facilitate mobility more than they provide joint stability (Ginn et al., 1997). It is therefore left to the muscles around the shoulder joint, in particular the rotator cuff muscle group, to provide functional or dynamic joint stability while maintaining mobility throughout the full active range of motion of the shoulder (Ginn et al., 1997, Saha, 1971). The rotator cuff muscle group, which includes the supraspinatus, subscapularis, infraspinatus and teres minor, originate from the scapula and blend together with the joint capsule before inserting into the humerus (Ginn et al., 1997). During movements of the shoulder they provide a medial and inferior force to the humeral head which centres it in the relatively small glenoid fossa (Ginn et al., 1997, Saha, 1971, Schenkman and Cartaya, 1987). In order to adequately maintain the stability of the shoulder during movement, the rotator cuff muscles must work in coordination with each other as well as with the muscles that provide movement of the humerus and scapula (Ginn et al., 1997, Schenkman and Cartaya, 1987)

Individualised, specific exercise aimed at restoring the normal stabilising function of the rotator cuff has been shown to improve pain, movement and functional impairment in patients with mechanical shoulder pain (Ginn and Cohen, 2004, Ginn and Cohen, 2005, Ginn et al., 1997). In a randomised controlled clinical trial, 71 volunteers attending a

hospital Physiotherapy Outpatients service were randomised to either a Treatment Group, which received an individualised, specific exercise program of stretching and strengthening for four weeks, which targeted specific rotator cuff dysfunction as assessed by the treating Physiotherapist, or a Control Group, that received no treatment for the same length of time while on a waiting list (Ginn et al., 1997). Following four weeks of treatment 66 subjects were reassessed. Subjects in the treatment group had a reduction in the Self-rated Disability Score, Self-rated Improvement Score and pain intensity on a visual analogue scale along with an increase in pain-free active range of motion of the shoulder compared to the waiting list controls (Ginn et al., 1997). Furthermore, while waiting for their treatment to commence, a higher proportion of subjects in the control group experienced a worsening of their symptoms compared to the treatment group, who started their treatment immediately. In particular, 50% of subjects in the control group reported worse functional disability scores and 32% showed a decrease in pain-free abduction range of motion of more than 10º compared to the treatment group, of which only 11% of subjects showed a decline in both of these outcome measures (Ginn et al., 1997). These results indicated that over a four week period an individualised physiotherapy programme aimed at reducing shoulder joint pain and dysfunction by restoring normal shoulder muscle function can produce improvements in joint symptoms, joint range of motion and independence with daily personal care (Ginn et al., 1997). Also, in light of the higher proportion of deterioration on some outcome measures in the waiting list control group, it was argued that delaying access to appropriate treatment for shoulder dysfunction may result in an increase in functional disability (Ginn et al., 1997).

Similar to the action of the rotator cuff in the shoulder, the integrity of the TMJ is predominantly maintained by its musculature which produces movement of the joint,

primarily by the action of the muscles of mastication, and which also act to control the movement of the joint (Cunningham and Romanes, 1986, O'Rahilly, 1986). Therefore, any dysfunction in the control of the muscles of mastication could alter jaw movements which may lead to impaired jaw function, which is commonplace in TMD (Stegenga et al., 1993). Prolonged parafunctional jaw movements may predispose an individual to joint or muscle pathology and the development of TMD. Furthermore, the timely application of an individualised treatment regimen could possibly reduce jaw disability and improve function.

Lumbar Spine

Patients with unilateral acute or subacute onset of their first episode of low back pain (LBP) show evidence of reduced cross-sectional area of the multifidus muscle on the ipsilateral side at the level which corresponded to symptoms on manual palpation (Hides et al., 1994). It was postulated that this rapid onset (range of length of symptoms of 1 to 66 days) of muscle wasting could be due to two reasons. Firstly, to inhibition of the muscle from perceived pain via a long-loop reflex that prevents movement in order to protect the structures at the level of pathology. Secondly, muscle spasm, as suggested by a more rounder shape of the multifidus muscle, may reduce the circulation to that muscle resulting in local metabolic effects contributing to the muscle wasting (Hides et al., 1994). Pathologic changes in the multifidus muscle, especially moth-eaten Type 1 fibres, have also been reported in patients with poor functional outcomes following lumbar disc surgery (Rantanen et al., 1993, Sihvonen et al., 1993).

Furthermore, following spontaneous recovery of LBP symptoms there is no spontaneous recovery in the cross sectional area of the wasted multifidus muscle (Hides et al., 1996). Subjects that received a specific exercise program, which facilitated active, isometric

multifidus contraction in co-contraction with the deep abdominal muscles, had a more rapid and more complete recovery in the cross-sectional area of multifidus at the four-week follow-up (Hides et al., 1996).

Along with the reduced cross-sectional area of the multifidus, patients with a history of chronic LBP with minimal or no symptoms at the time of testing also demonstrated a delayed onset of transversus abdominis contraction during an arm movement task compared to asymptomatic matched controls (Hodges and Richardson, 1996). These results provide evidence of the preparatory nature of the central control of muscle contraction in order to maintain trunk alignment against the reactive forces generated by arm movement tasks that tend to produce flexion of the spine (Hodges and Richardson, 1996). It was postulated that in patients with chronic LBP there may be a dysfunction of the central motor control of these preparatory muscle contractions that may lead to an increased risk to the persistent or recurrent nature of their symptoms (Hodges and Richardson, 1996).

In addition to the chronic LBP model described above, experimentally induced pain in the longissimus muscle at the level of the fourth lumbar vertebra (L4) results in changes in the onset and amplitude of EMG activity of the paraspinal and abdominal muscles (Hodges et al., 2003). Hypertonic saline was injected into the longissimus muscle at L4 which induced an acute onset of pain in that area which was maintained for the duration of arm movement tasks. The most consistent finding was that in the presence of experimentally induced pain the EMG activity of the transversus abdominis was reduced in amplitude or delayed compared to the non-painful control arm movement trials (Hodges et al., 2003). Furthermore, these EMG changes persisted after the resolution of the pain, and in one

subject the response of transversus abdominis did not recover within one hour of the resolution of symptoms (Hodges et al., 2003). This finding suggested that minimal exposure to a painful experience may have long-term sequelae (Hodges et al., 2003) and may be an explanation for the recurrent nature of chronic painful conditions in the lumbar spine, and perhaps other musculoskeletal subsystems such as the TMJ.

Exercise and TMD

As mentioned earlier, non-specific TMD are considered to be musculoskeletal conditions (Michelotti et al., 2005) with the majority being muscular in origin (Lobbezoo et al., 2004). It is therefore plausible that non-specific TMD should be amenable to treatments that address musculoskeletal dysfunction and/or pain. The prescription of resistance exercises is a common physiotherapy strategy used to improve jaw function and coordination of movements (Au and Klineberg, 1993, Feine and Lund, 1997, Rocabado and Iglarsh, 1991) and to reduce or eliminate TMJ clicking (Au and Klineberg, 1993) and also bruxism (Quinn, 1995). Therapeutic exercises have also been utilised as part of the postoperative management of patients that have undergone TMJ arthroscopy (Bertolucci, 1992a) and arthroplasty (Bertolucci, 1992b, Bertolucci et al., 1989) and following surgical repair or restructuring of the TMJ (Rocabado, 1989, Quinn, 1995, Oh et al., 2002).

The prescription of therapeutic exercise for the treatment of non-specific TMD has been reported to be effective (Au and Klineberg, 1993, Feine and Lund, 1997, Katsoulis and Richter, 2008, Medlicott and Harris, 2006) although not necessarily more effective than any other form of conservative management (Dahlstrom, 1992, Stohler and Zarb, 1999). In particular, the effectiveness of exercise therapy was investigated in 20 patients with internal derangement of the TMJ (Nicolakis et al., 2001) and in 20 patients with myofascial pain dysfunction (Nicolakis et al., 2002). In both studies each patient received at least five

individualised treatment sessions lasting 30 minutes each. The treatment sessions consisted of a mix of manual therapy and exercise components. The manual therapy included massage of the painful muscles, stretches, manual joint distraction, disc/condyle mobilisations and relaxation. The exercise component included guided opening and closing movements, correction of body posture and gentle isometric tension exercises against resistance (Nicolakis et al., 2001, Nicolakis et al., 2002). At the completion of the treatment regimen six of the twenty patients with myofascial pain syndrome reported no pain and seven reported no impairment (Nicolakis et al., 2002). This rose to 16 patients reporting no pain and 13 patients reporting no impairment at the six months follow up (Nicolakis et al., 2002). Of the 20 patients with internal derangement of the TMJ four reported no pain and three reported no impairment at the completion of the treatment regimen (Nicolakis et al., 2001). At the six months follow up seven patients reported no pain and no impairment respectively (Nicolakis et al., 2001). From these results the authors of these studies concluded that exercise therapy can significantly reduce jaw pain and improve impairment in patients suffering from myofascial pain syndrome and internal derangement of the TMJ. Although promising, such conclusions should be viewed with caution given the prescribed resistance exercises were only one part of a multifaceted approach to the treatment of these two conditions. It is therefore difficult to determine the contribution of the prescribed exercises to the resolution of symptoms.

Few studies have investigated the effect of physiotherapy modalities on the EMG activity of the muscles of mastication. Mobilisation of the TMJ has been shown to reduce the EMG activity of the masseter muscle during rest and clenching (Taylor et al., 1994). In this randomised, cross-over design study 15 patients with pain in the TMJ and masticatory muscles and limited jaw opening were treated with both a distraction mobilisation

technique of the TMJ and a sham treatment of barely perceptible superficial massage. Compared with the sham treatment there was a significant decrease in masseter EMG activity during rest and clenching along with a significant increase in mandibular opening and lateral movement following TMJ mobilisation (Taylor et al., 1994). Two randomised control trials have investigated the effectiveness of EMG biofeedback in reducing EMG activity in patients with myofascial pain dysfunction syndrome (Dalen et al., 1986, Dohrmann and Laskin, 1978). In one of these trials 24 patients with myofascial pain dysfunction syndrome were randomly allocated to either a biofeedback treatment group or a sham biofeedback group (Dohrmann and Laskin, 1978). When compared to sham biofeedback the use of EMG biofeedback for twelve sessions over six weeks was shown to reduce the EMG activity in the masseter muscle (Dohrmann and Laskin, 1978). In another study 19 subjects with myofascial pain dysfunction syndrome were randomly assigned to either a control group or an experimental group (Dalen et al., 1986), The ten subjects in the experimental group were instructed to reduce the level of activity in the masseter and frontalis muscles with the use of EMG biofeedback during two biofeedback sessions per week over four weeks (Dalen et al., 1986). The experimental group was also asked to reduce the muscle tension in these target muscles at home by visualising the laboratory setup between the biofeedback sessions (Dalen et al., 1986). After completion of the four week biofeedback training there was a significant reduction in the EMG activity levels of the masseter and frontalis muscles, with the reduction being maintained in the frontalis muscle after three and six months (Dalen et al., 1986). These studies reported significant changes in EMG activity in the masseter and frontalis muscles following the application of mobilisation of the TMJ and EMG biofeedback. However, the direct effects of exercise on the EMG activity of the muscles of mastication is yet to be investigated.

A recent systematic review of 30 studies investigating the efficacy of physiotherapy modalities in the treatment of TMD concluded that therapeutic exercise may be effective in its management (Medlicott and Harris, 2006). However it was also noted that these results should be viewed with caution due to the lack of consensus in defining TMD, inclusion and exclusion criteria, and the use of reliable and valid outcome measures amongst the 30 studies reviewed (Medlicott and Harris, 2006). The fact that many studies utilise a combination of treatment modalities including exercise prescription as a part of the overall treatment regimen also makes it difficult to accurately assess the efficacy of exercise for the treatment of TMD.

To date there remains a paucity of literature that has attempted to elucidate either the underlying aetiology of non-specific TMD or the mechanisms that result in the relief of symptoms and/or signs (Michelotti et al., 2005, Stohler and Zarb, 1999). Furthermore, even though the presenting signs and symptoms may be similar in different patients, the aetiology may vary between individuals (Dahlstrom, 1992). Without the knowledge of the aetiology it is at best difficult, if not impossible to treat non-specific TMD with a causal therapy (Michelotti et al., 2005). In order to provide appropriate treatment for non-specific TMD of a muscular origin, it is important to determine the normal functioning of the muscles that control the mandible. This concept has been illustrated in patients with other musculoskeletal disorders, as described in the knee, shoulder and lumbar spine, above. Determining the normal function of a particular musculoskeletal subsystem, such as the knee, shoulder, lumbar spine or TMJ, allows for an accurate comparison to be made in symptomatic individuals, which facilitates both an accurate diagnosis and an appropriate management plan.

The mechanisms whereby therapeutic exercise is effective in some TMD patients is also unclear. The patellofemoral joint and lumbar spine literature indicate that physiotherapy can produce specific changes in timing and patterning of EMG activity. However, we have no information as to whether there are any effects of therapeutic exercises on jaw muscle activity patterns. An understanding of these exercise-induced EMG effects could significantly enhance management if specific exercises can be tailored for specific TMD symptoms.

Knowledge of the appropriate duration of an exercise programme could also enhance the efficacy of the management of TMD. Traditionally, therapeutic exercise has been prescribed for a duration of four to six weeks. In the patellofemoral joint, changes in the relative timing of EMG activity in the VMO muscle compared to the VL muscle have been described after six weeks of a specific exercise programme (Cowan et al., 2002). Completing four weeks of a specific exercise programme resulted in improvements in disability and function scores as well as an improvement in pain-free active range of motion of the shoulder (Ginn et al., 1997). In the lumbar spine, a more complete recovery in multifidus muscle cross-sectional area has been described after four weeks in patients recovering from low back pain (Hides et al., 1996). In the orofacial region, neural plasticity has been observed in the primary motor cortex of the tongue following fifteen-minutes training of a novel tongue protrusion task (Boudreau et al., 2007). From these findings it is clear that there is a wide range of exercise programme durations that result in effective treatment outcomes and changes in the neuromuscular system. It is therefore important to determine the appropriate length of time necessary for an exercise programme to elicit changes in the desired neuromuscular system. It is clear that immediate changes occur in the orofacial region following short-term exercise training of the tongue

(Boudreau et al., 2007). What remains unclear, however, is whether similar short-term changes occur after training the muscles of mastication. Furthermore, if these changes do occur in the muscles of mastication, it is unknown whether they are maintained over a longer period of time or whether a longer-term exercise programme will help to maintain or even enhance these changes.

Statement of the Problem

The preceding discussion briefly illustrated the effects of pain and dysfunction on the control of muscles in the knee, shoulder and lumbar spine. It has also described how an appropriate exercise programme can return normal function and reduce symptoms in these musculoskeletal subsystems. In the TMJ, therapeutic exercise has also been shown to effectively reduce the symptoms of TMD and aid in post-surgical recovery. Unlike the other musculoskeletal subsystems discussed there is a paucity of literature that demonstrates the effects of resistance exercise on the EMG activity of the muscles of mastication. Furthermore, biomechanical modelling of the human masticatory system has demonstrated that different muscle activation strategies are capable of producing the same lateral jaw movement as measured at the incisor point and condyles (Lobbezoo et al., 2004), suggesting a level of redundancy in the muscles that control jaw movements. It is plausible therefore that given this anatomical redundancy (Lobbezoo et al., 2004), the human masticatory system could be capable of recruiting different muscles to produce the same jaw movements dependent on the functional need of the system at the time or as a result of perturbations such as the application of resistance exercise. Therefore this thesis attempts to investigate the effects of isotonic resistance exercise training on jaw muscles of asymptomatic adult volunteers. Specifically, the aims of this thesis are:

1. To train individuals to use their jaw muscles differently while completing a standardised jaw movement task.

2. To demonstrate that these changes in muscle use occur in the short term and are maintained in the longer term.

To this end, the specific hypotheses of this thesis are that:

1. Jaw muscle recruitment strategies can undergo immediate changes following isotonic resistance exercise training.

2. Four weeks of a home-based isotonic resistance exercise programme produces EMG changes in the muscles of mastication.

3. The effects of a four-week home-based isotonic resistance exercise programme produce longer lasting changes in the EMG activity of the muscles of mastication.

In the longer term, these findings could be utilised to plan specific exercise regimens for TMD patients.

METHODS

Twelve asymptomatic adults were recruited from staff, students and patients at the Westmead Centre for Oral Health and the Faculty of Dentistry, The University of Sydney. This sample size was chosen as we believed it would provide sufficient diversity with respect to muscle activation strategies and structure on which we could test our hypotheses. Subjects provided informed consent to participate in the study. Ethics approval was obtained from the Sydney West Area Health Service Human Research Ethics Committee (Westmead Campus) and The University of Sydney Human Research Ethics Committee.

Subjects were included in the study if they were between the ages of 18 and 65 years, were able to understand the study and were willing and able to attend three recording sessions over eight weeks and perform at-home jaw exercises. Those with a past or current history of pain or dysfunction (e.g. temporomandibular disorders) in and around the orofacial region were excluded.

The flow of the experiment is outlined in Figure 9 and the protocols of each of the data collection sessions are described in Table 2. Each subject was asked to attend a total of three data collection sessions: an Initial Training Session (ITS); Testing Session 1 (TS1), conducted four weeks after the ITS; and Testing Session 2 (TS2), eight weeks after the ITS (Figure 9 and Table 2). The ITS was divided into four sections: Subject Preparation; Pre-Exercise Data Collection (ITS Pre); Exercise Training; and Post-Exercise Data Collection (ITS Post, Table 2). Each Testing Session was divided into two sections: Subject Preparation; and Data Collection (Table 2). All data collection was conducted at the Jaw Function and Orofacial Pain Research Unit, Westmead Centre for Oral Health.

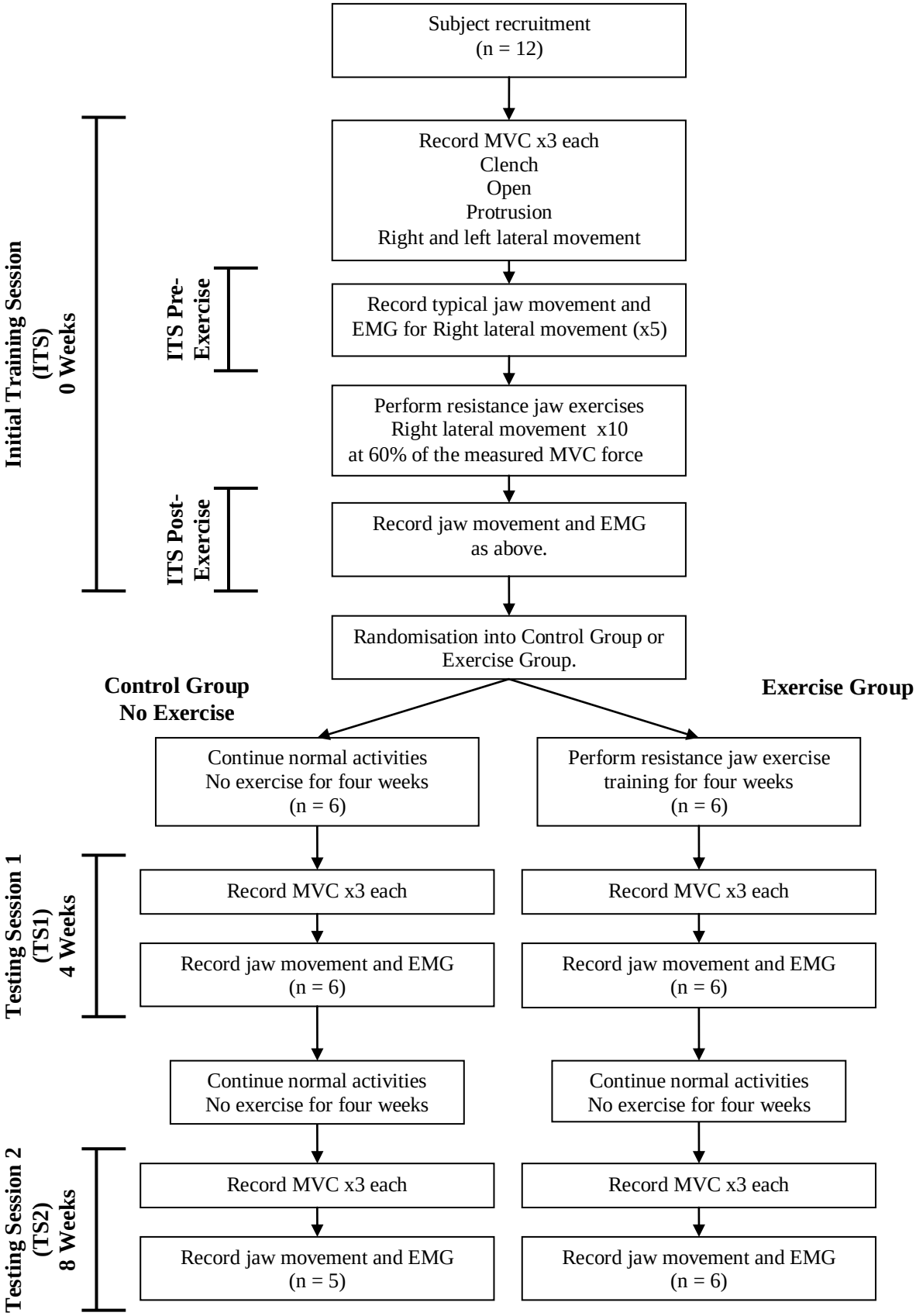

Figure 9**:** Flowchart depicting experimental design with sample sizes in each group. MVC = maximum voluntary contraction, EMG = electromyographic activity.

Initial Training Session (ITS) 0 Weeks	Testing Session 1 (TS1) 4 Weeks	Testing Session 2 (TS2) 8 Weeks
Subject Preparation		
Fabrication of clutches		
Placement of electrodes	Placement of electrodes	Placement of electrodes
Attachment of clutches	Attachment of clutches fabricated during the ITS	Attachment of clutches fabricated during the ITS
Connection of electrodes	Connection of electrodes	Connection of electrodes
Subject seated in data collection chair	Subject seated in data collection chair	Subject seated in data collection chair
Start up and test data collection equipment and software	Start up and test data collection equipment and software	Start up and test data collection equipment and software
Pre-Exercise Data Collection (ITS Pre)	**Data Collection**	
Rest Trial	Rest Trial	Rest Trial
MVC x 3 for each movement Clench Open Protrusion Left lateral Right lateral	MVC x 3 for each movement Clench Open Protrusion Left lateral Right lateral	MVC x 3 for each movement Clench Open Protrusion Left lateral Right lateral
Movement trials Open/close Protrusion Left lateral Right lateral	Movement trials Open/close Protrusion Left lateral Right lateral	Movement trials Open/close Protrusion Left lateral Right lateral
	Rest Trial	Rest Trial
	MVC x 3 as above	MVC x 3 as above
	Remove clutches and electrodes	Remove clutches and electrodes
Exercise Training		
10 repetitions of isotonic resisted right lateral movements at 60% MVC		
Post-Exercise Data Collection (ITS Post)		
Repeat movement trials as in Pre-Exercise data collection		
Rest Trial		
MVC x 3 as above		
Remove clutches and electrodes		

Table 2: This experiment comprised three data collection sessions (columns) spaced 4 weeks apart. Each column shows the data collection session divided into its sections and the order with which each section was completed (MVC = Maximum voluntary contraction, ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Initial Training Session (ITS)

In this session, we tested the hypothesis *that jaw muscle recruitment strategies can undergo immediate changes following isotonic resistance exercise training*. The ITS was divided into four sections (Table 2): Subject Preparation; ITS Pre; Exercise Training; and ITS Post.

Subject Preparation

Electromyographic Activity

To detect electromyographic (EMG) activity, bipolar silver-silver chloride (Ag-AgCl) surface electrodes (Duo-Trode, Myotronics, Washington, USA) were placed bilaterally over the middle portions of the anterior temporalis and masseter muscles, and over the right anterior digastric (Figure 10). The electrodes were aligned parallel to the direction of the alignment of the muscle's fibres. Duo-Trode bipolar electrodes have pre-jelled contact surfaces that are 12.5 mm in diameter and spaced 19 mm between centres. Prior to the placement of the electrodes the skin was wiped with 70% isopropyl alcohol (Alcowipe, Promedica, Australia) to clean the area and reduce electrical resistance. To maximise the conduction of EMG activity, a small amount (1-2 mL) of electrode conducting gel (Sigma Gel, Medtronic Denmark) was placed on the electrode's surface beneath the conducting foam using a hypodermic syringe.

To ensure reproducibility of electrode placement between data collection sessions, their location was recorded as follows. A mark was placed on the subject's skin with a non-permanent marker pen at the central point between the two poles of each electrode. The infraorbital notch or foramen was palpated bilaterally by the researcher (AW) and a reference line was drawn bilaterally between the tragus of the ear and the infraorbital notch or foramen along this clinically-determined Frankfort horizontal plane (FHP, Figure 10).

The vertical and horizontal positions of the central point of the electrodes in relation to the FHP were noted. The horizontal measurement was referenced from the tip of the tragus along the FHP, with positive measurements being anterior to the tragus. Vertical measurements were taken perpendicular to the FHP, with positive measurements being superior to the FHP. These recorded horizontal and vertical measurements of the centre of the placement of the electrodes in relation to the FHP provided a two-dimensional coordinate that could be marked on the skin prior to the placement of the electrodes in the subsequent two data collection sessions, TS1 and TS2.

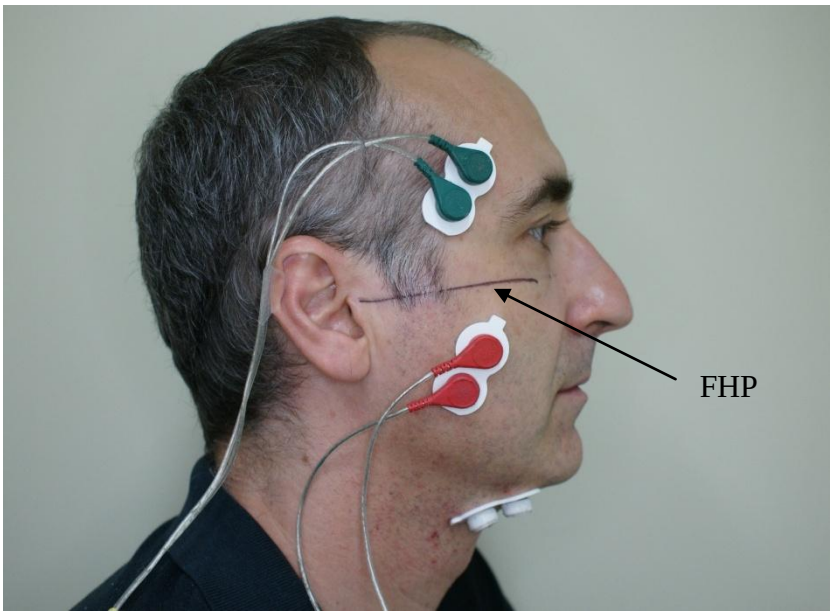

Figure 10: Right lateral view of the face showing the placement of the bipolar electromyographic (EMG) electrodes in relation to the Frankfort horizontal plane (FHP). The electrodes over the anterior temporalis (green electrode pair) and masseter (red electrode pair) were placed bilaterally. The electrodes over the digastric (white electrode pair) were placed predominantly over the right side of the submandibular area.

Jaw Movement

Jaw movement was recorded at 67.3 samples/sec and in six degrees of freedom utilising an optoelectronic system (JAWS3D, Metropoly, Switzerland). In this system, the movement of two target frames is detected by charge coupled device cameras (Figure 11a). Each target frame consists of three non-collinear light emitting diodes (LEDs; Figure 11b).

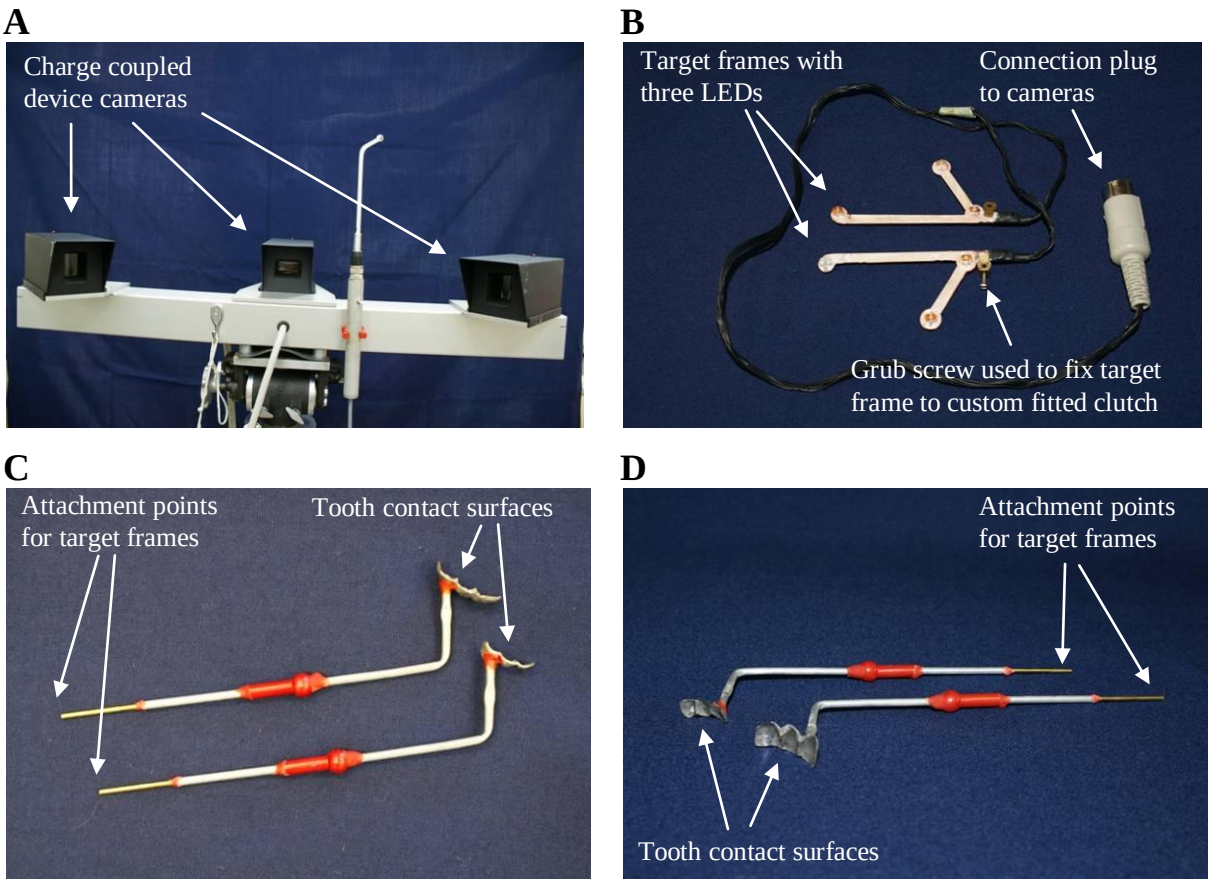

Figure 11: JAWS3D equipment used to detect jaw movements. A: JAWS3D charge coupled device cameras fixed to an adjustable stand. B: The two target frames used, showing cabling and connector and the grub screw used to attach the target frame to the custom fitted clutches. C and D: Two views of the custom fitted clutches showing the tooth contact areas to which the Duralay was applied and the attachment points for the target frames.

The target frames were attached to the mandibular and maxillary teeth via lightweight titanium/acrylic clutches custom fitted to each subject's teeth (Figure 11c and d and Figure 12).

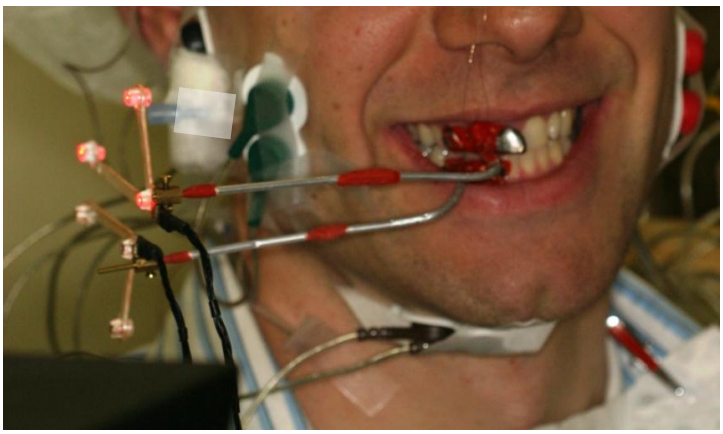

Figure 12: Oblique view of the face of a subject showing the custom fitted lightweight clutches placed on the anterior teeth of the right maxilla and mandible.

During jaw movement, the movements of the target frames were detected by the charge coupled device cameras and processed by a personal computer to display mandibular movement in relation to the position of the maxillae in real time on a computer display (Mesqui and Palla, 1985). The software allowed the preselection of any point of interest on the mandible and would compute and display the movement of that point in real time (Figure 13).

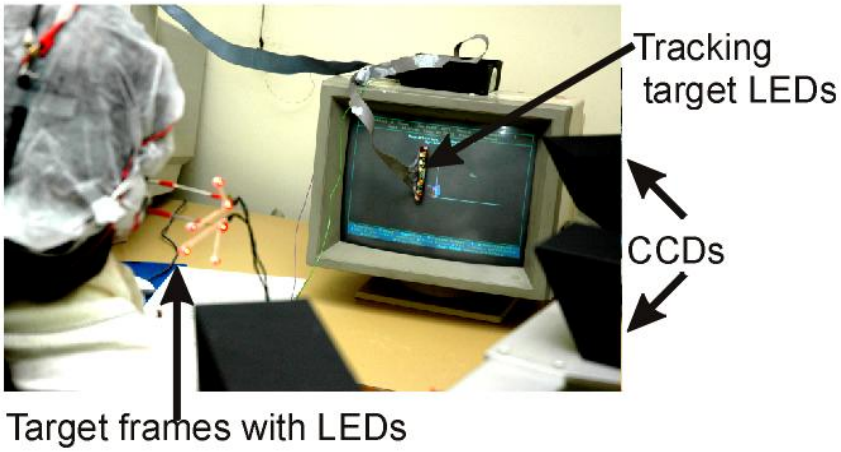

Figure 13: Setup of the JAWS3D movement tracking system showing the target frames secured to the lightweight clutches, the charge coupled device cameras (CCDs) used to detect the movement of the target frames and the visual display of the mandibular mid-incisor point (MIPT). The bank of 15 tracking target light emitting diodes (LEDs) used to standardise the movement trials is also shown.

The lightweight clutches were custom fitted for each subject by one of two qualified Dentists experienced in the fitting of these appliances (CP and/or GM). For each subject the clutches were placed across three to four suitable teeth on the right mandible (from teeth 11 to 15) and the right maxilla (from teeth 41 to 45, Figure 12). An acrylic compound (Duralay, Reliance Dental Mfg. Co., Worth, IL, USA) was used to accurately mould the clutch onto the labial/buccal surfaces of the subject's teeth. The Duralay was mixed, according to the manufacturer's instructions, and placed onto the tooth contact surface of each lightweight clutch (Figure 11c and d). While the Duralay was still soft the clutch was positioned against the appropriate teeth and held in place until the Duralay hardened. This produced a solid impression of the subject's teeth that was fixed to the lightweight clutch. The solid impression ensured that the clutch was rigidly connected to the teeth for the duration of each of the experimental sessions and also allowed for

reproducible placement of the clutches during all three data collection sessions. If required, the acrylic impression was trimmed to remove any sharp edges and/or excess in order to ensure a comfortable fit for the subject without interference of the subject's tooth contacts. Once the acrylic impression was complete the lightweight clutch was secured to the appropriate teeth with cyanoacrylate adhesive.

Then, the target frame consisting of three LEDs arranged in a triangular pattern (Figure 11b) was fixed to the end of each clutch by grub screws (Figure 11b and Figure 12). The target frames were aligned parallel to the clinically-determined FHP. The mandibular mid-incisor point (MIPT), defined as the point mid-way between the mesial edges of the mandibular central incisors, was chosen as the mandibular point of interest and its movement in relation to the position of the maxilla was computed and displayed in real time on a computer display (Figure 13).

This study collected movement and EMG data for four jaw movement tasks: open/close; protrusion/retrusion; left lateral movement and return; and, right lateral movement and return. The principal movement task of interest for this thesis, however, was right lateral movement and return only. The dominant plane of movement for lateral displacements of the MIPT is the horizontal plane. The Cartesian coordinate system of JAWS3D defines movement in the horizontal plane to be in the X-Y-plane with the X-axis aligned with the antero-posterior axis of the Frankfort p\horizontal plane and the Y-axis aligned perpendicular (i.e. lateral) to this (Metroply AG, 1990). The positioning of the target frame of LEDs resulted in movements in the right direction to have positive Y-displacement values (Metroply AG, 1990). The data from the movements in the Y-direction were used for the calculation of all the measured variables.

Maximum Voluntary Contraction

The maximum voluntary contraction (MVC) of a muscle is the peak force produced by that muscle as it contracts while pushing or pulling against an immovable object. The EMG signals of the MVC of each of the recorded jaw muscles were used to normalise the EMG data for all tasks. MVC was estimated during maximum isometric contraction of the jaw muscles, during maximum tooth clenching for the masseter and temporalis muscles and during attempted mouth opening against resistance (with approximately 15mm incisor separation) for the digastric muscle. To estimate the MVC for jaw closing subjects were asked to bite down as hard as they could onto cotton rolls placed between their right and left molar teeth. This MVC data was determined at the beginning of the data collection session prior to the commencement of the movement trials (three MVC trials), and at the end of the data collection session (a further three MVC trials). All muscle activity produced during the movement trials was subsequently normalised to the maximum values.

The maximum force exerted during right lateral excursion against resistance was determined in order to calculate and standardise the force that was applied during the resistance exercise training section of the ITS (see Isotonic Resistance Exercise Training section, page 61 below). In this isometric task, force data was collected by asking subjects to place a thin pressure sensor (Flexiforce, Tekscan, South Boston, MA, USA; Figure 14) between the skin on the side of the lower jaw adjacent to the canine root tip and one finger of the hand that applied the resistance to the various jaw movements (Figure 14c). This sensor is a thin polyester strip which contains an electrical circuit interrupted by resistive ink. This ink's resistance decreases with increasing force and consequently changes the circuit's voltage.

The Flexiforce sensor was connected to a proprietary data acquisition and processing unit (Economical Load and Force System ELF, version 3.4, Tekscan, South Boston, MA, USA). The system's overall error is +/-5% and sampling rate is 200Hz (Tekscan, South Boston, MA, USA). This software package collected the sensor data and converted it to force data and also displayed the data in real time to provide the subject with visual feedback of the amount of force they produced during each isometric MVC trial (Figure 14d). As well as the visual feedback subjects were given verbal encouragement during each MVC trial from the experimenter (AW) to push as hard as they could against their finger without causing pain or discomfort.

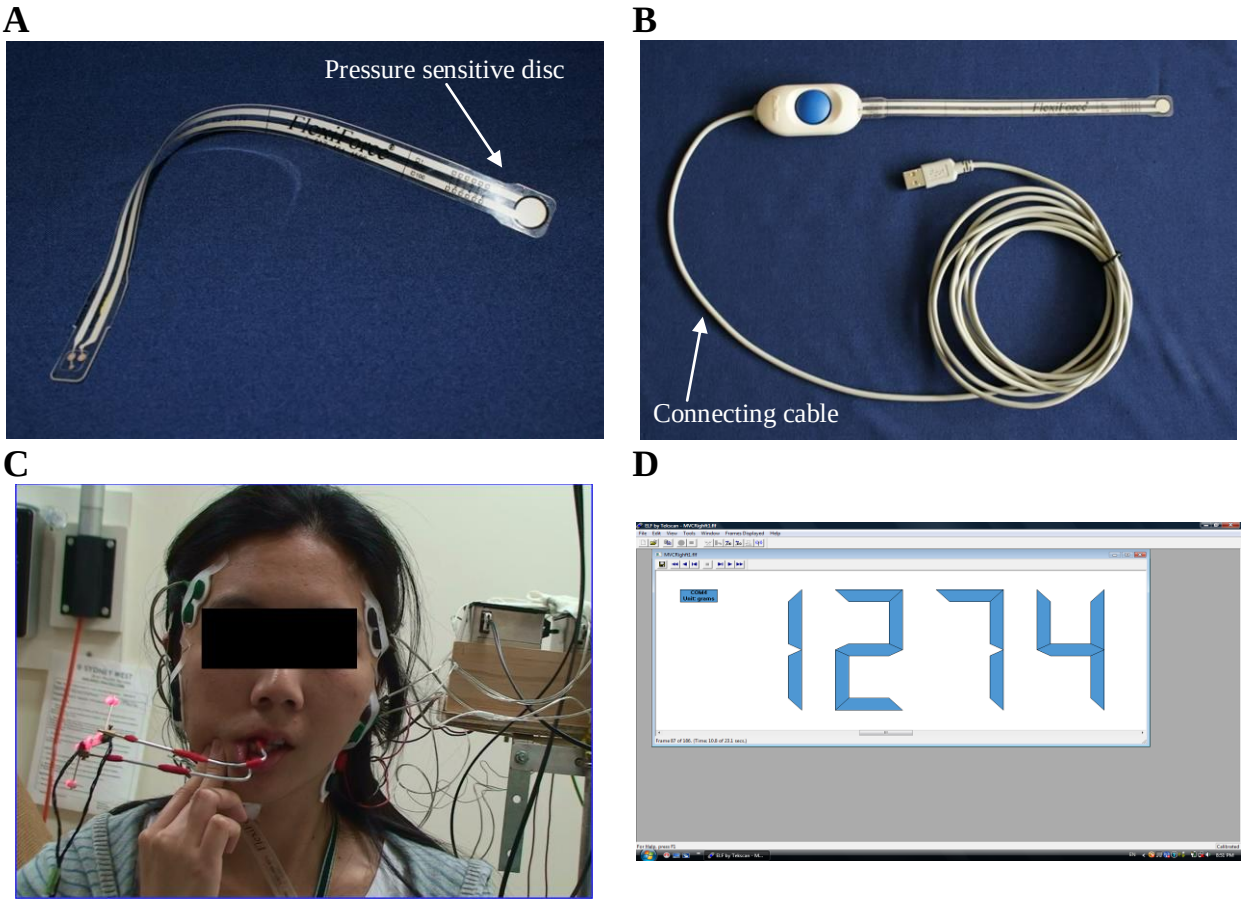

Figure 14: Setup of the force measurement system. A and B: Flexiforce sensor showing the pressure sensitive disc and cabling connecting the sensor to the computer. C: Anterior view of the experimental setup showing the position of the surface electrodes and lightweight clutches with target frames attached. The subject is holding the Flexiforce sensor against the skin overlying the right mandible while applying a lateral force against the finger of the right hand. D: Screenshot of the Economical Load and Force system software (ELF, version 3.4, Tekscan, South Boston, MA, USA) depicting force measurement from the Flexiforce sensor during measurement of a maximal voluntary contraction (MVC).

Pre-Exercise Data Collection (ITS Pre)

Each subject performed five trials of open-close, protrusion, left laterotrusion and right laterotrusion. For each movement trial the position of the mandibular MIPT was computed and displayed on the computer monitor (Figure 13). A bank of 15 LEDs arranged in a straight line was positioned on the computer monitor in line with the movement and programmed so that the LEDs activate sequentially in time with the subject's typical movement (Figure 13). The number of LEDs that were illuminated during the movement trials was determined prior to the start of each movement's set of five trials by asking the subject to comfortably move to maximum excursion. This number of LEDs was programmed into the software that activated the bank of LEDs (Spike2 version 3.14 scripts, Cambridge Electronic Design, England). In this way, each subject tracked a visual target to ensure subsequent movements were standardised because subjects were required to match their jaw movements to predetermined displacements, durations and directions during each movement trial.

The raw EMG signal was amplified and filtered by an isolated bioelectric amplifier (DB4-XB1 Fibre Optic, World Precision Instruments Inc., Sarasota, FL, USA). The raw EMG signal was filtered using the following settings: low pass at 7 kHz; high pass at 70 Hz, and notch filter at 50 Hz. The filtered signal was then digitised (Micro 1401, Cambridge Electronic Design, England) and digitally sampled at 2 KHz using Spike2 data acquisition software (version 3.14, Cambridge Electronic Design, England). The raw EMG data for each trial was saved on a personal computer during each data collection session in order for the data to be processed and analysed offline after the completion of the data collection session.

Isotonic Resistance Exercise Training

During the resistance exercise training portion of the ITS, each subject was instructed to perform right lateral movements against a standardised force of resistance (60% of MVC). Subjects were asked to hold one finger of their right hand against the right mandible to oppose the direction of movement in the same manner as in the MVC determination trials (see Maximum Voluntary Contraction section, page 57, and Figure 14c above). The same Flexiforce sensor as that used during the MVC determination trials was used to measure the resistance force and provide the subject with visual feedback of their applied force during the duration of each trial (Figure 14d). The Flexiforce sensor was placed between the skin of the face and the finger of the right hand that applied the resistance to the jaw movement. Subjects were instructed to apply the training force (60% of MVC), as indicated by the ELF Software (Figure 14d), and maintain this resistance force during the outward and return phase of right lateral movement without holding the resistance force at their maximum lateral excursion (isotonic resistance task). Subjects completed ten repetitions of this isotonic resistance task. Each repetition took approximately five seconds to complete with a five to ten seconds rest interval between each movement.

The direction of the resistance exercise task was chosen as right laterotrusion because it is a relatively novel task during normal activities of daily living and would be a direction of jaw movement that would be less likely to be influenced by normal jaw function. Unlike other jaw movements such as open and close, right laterotrusion would not be subject to a training effect on a normal daily basis. Therefore the effects of the home based exercise regimen could be assessed directly with minimal confounding effects from the subjects' normal use of their jaws. Furthermore, with this asymmetric task it would be expected that

left and right muscle activity would be quite different providing additional opportunities for muscle activity changes with training.

Post-Exercise Data Collection (ITS Post)

At the end of the resistance exercise training portion of the ITS, each subject was asked to repeat the same movement trials for each movement as they performed during the initial movement trials (ITS Pre). Each subject performed five trials each of open-close, protrusion, left lateral and right lateral. The same procedures were used as during the initial movement trials (ITS Pre). Raw EMG activity data and movement data were collected and saved for offline processing, analysis and comparison with the pre-exercise data collection trials.

If the movements were not similar, the subject underwent further exercise training and the movement was again compared. If after three training sessions, the subject was unable to perform the same movements as pre-training, then that subject's experiment was terminated.

Interval between training and testing sessions

On completion of the ITS, subjects were randomised into one of two groups: the Exercise Group; or the Control Group. The randomisation process was double blinded by having 12 pieces of paper each with either the word "Exercise" or the words "No Exercise" printed on them. Each of the 12 pieces of paper were then folded and placed inside a sealed envelope which was placed into a box with all the other sealed envelopes. Subjects were asked to select one of the 12 envelopes from the box and hand it to a researcher. The subject was then allocated to the group printed on the piece of paper.

Subjects allocated to the Exercise Group were asked to perform the same isotonic resistance exercise as in the ITS, three times per day for four weeks, and to perform 10 repetitions of each exercise. In order to measure compliance with the home based exercise programme subjects were asked to fill out an exercise diary during this period. The experimenter (AW) also made contact with each subject on a weekly basis to ensure that subjects had no problems with the home based exercise programme. At the end of the four weeks' exercise period, the subjects in the Exercise Group were asked to return for retesting and data collection of the same movement trials as in the ITS (Testing Session 1, TS1).

Subjects allocated to the Control Group were not asked to perform any jaw exercises during the following four weeks. They were simply instructed to continue with their normal activities of daily living. At the end of this four weeks' period, the subjects in the Control Group were asked to return for retesting and data collection of the same movement trials as in the ITS (Testing Session 1, TS1).

On completion of the data collection of TS1, both Exercise Group and Control Group subjects were not asked to perform any jaw exercises for a further four weeks. They were instructed to continue with their normal activities of daily living during this period and return for retesting and data collection at the end of a further four weeks (Testing Session 2, TS2).

Testing sessions (TS1 and TS2)

In these sessions, we tested the hypotheses *that four weeks of a home-based isotonic resistance exercise programme produces EMG changes in the muscles of mastication, and that the effects of a four-week home-based isotonic resistance exercise programme produce longer lasting changes in the EMG activity of the muscles of mastication*.

Following the ITS subjects returned for recording of jaw movements and muscle activity after four weeks (TS1) and after eight weeks (TS2). In both TS1 and TS2, EMG electrodes were placed in the same positions as in the ITS by utilising the recorded measurements of the initial electrode placements in relation to the clinically-determined FHP obtained at the ITS. The same customised lightweight clutches that were fabricated in the ITS, were reattached in an identical fashion to that in the ITS using cyanoacrylate adhesive, and the LED target frames were fixed to the lightweight clutches in line with the clinically determined FHP. Subjects were asked to repeat each of the MVC trials (clench, open, protrusion, left lateral and right lateral) in the same manner as during the ITS. Subjects were then asked to repeat each of the four movements (open-close, protrusion, left lateral movement, and right lateral movement) five times, and these movements and the corresponding EMG activity were compared to those recordings performed during the ITS (see Statistical Analyses section, page 72).

Data Processing

Raw EMG and movement data files were processed using customised programs developed with Agilent VEE Pro 6.0 (Agilent Technologies, Santa Clara, California, USA). Raw EMG data collected with Spike2 for each trial was exported into text files. Data from each EMG electrode channel, representing an individual muscle, was then extracted and saved

as an individual text file. The extracted channel EMG data were then digitally rectified and Butterworth filtered at 1 Hz with an order of 3 and stored separately. Each Butterworth filtered EMG data file was then normalised to the Butterworth filtered MVC EMG data of the corresponding muscle during the same movement (see Appendix 1for flowchart of data processing with Agilent VEE Pro 6.0).

The Butterworth filter was used to produce a smoothed representation of the changes in the raw EMG data (Figure 15). This filter sacrifices rolloff steepness for monotonicity in the passband (the range of frequencies or wavelengths that can pass through a filter without being attenuated) and the stopband (the range of frequencies that are attenuated to very low levels or prevented from passing through a filter) to produce the smoothing of the raw signal (The Mathworks Inc, 2000). The Butterworth filter suits applications that require preservation of amplitude linearity in the passband region which makes it ideal for conditioning the raw EMG signal while revealing frequency components likely to be reflected in changes in jaw movements (Long, 2004, The Mathworks Inc, 2000).

Movement data files collected by JAWS3D were converted to text files and stored. Each file contained the three-dimensional coordinates of the mandibular MIPT as it moved along three mutually orthogonal axes: anteroposterior (X-direction), lateral (Y-direction) and vertical (Z-direction). These coordinates of the movement of the mandibular MIPT were extracted and saved as an individual file for further analysis.

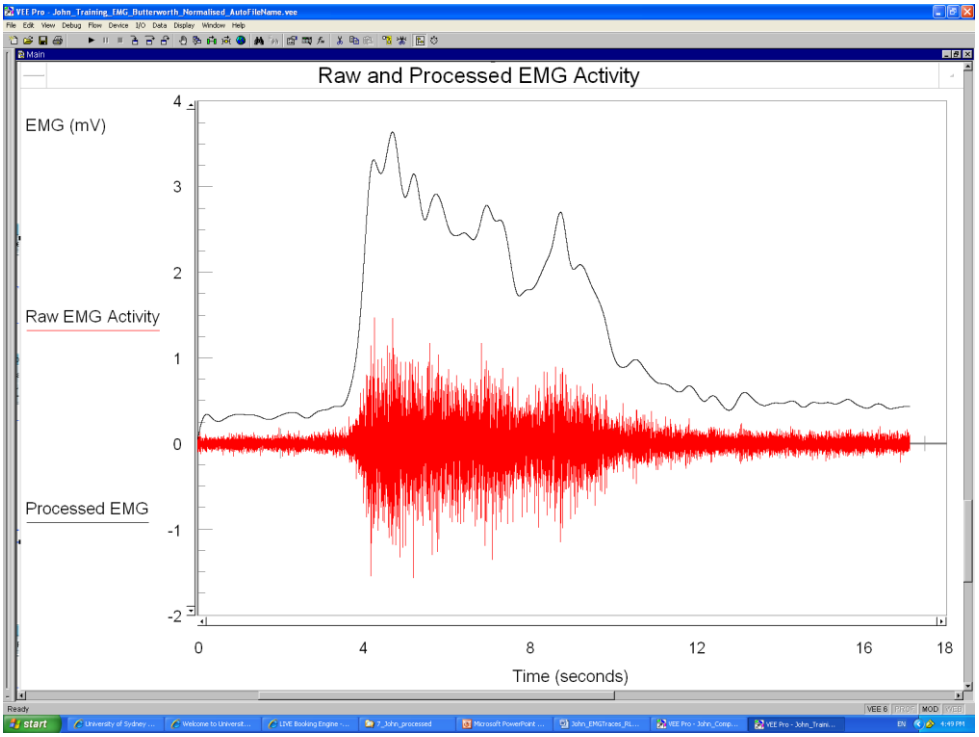

Figure 15: Screenshot of the customised Agilent VEE Pro 6.0 program depicting the raw EMG signal trace (red) and a processed EMG signal trace superimposed (black curve). The raw EMG signal was digitally rectified and then Butterworth filtered to produce the single smoothed trace.

Calculated variables of interest

The variables of interest were calculated from the normalised EMG data files. Each normalised EMG file was passed through another customised program developed with Agilent VEE Pro 6.0 (Appendix 1). This customised program calculated the variables of interest as per the definitions below. The values were then stored in a text file which was used to export the values into the statistical analysis package. The variables of the movement trials which were analysed are listed below. In order to calculate the variables of interest in relation to the EMG activity, the following four points of reference on the movement and Butterworth filtered EMG activity traces had to be determined: movement

onset; movement offset; EMG activity onset; and, EMG activity offset. These four reference points are defined first followed by the definitions of the calculated variables.

Reference points

Movement onset

The onset of movement was defined as the time point at which the right lateral movement of the mandibular MIPT commenced (Figure 16). This was determined using the Y-direction movement file which was displayed on a computer screen using the customised program developed with Agilent VEE Pro 6.0 (Appendix 1). The data in each movement file was plotted on a X-Y scatterplot which had two moveable cursors programmed onto the graph. The moveable cursors could be placed at any point along the plotted movement curve. The first cursor was positioned along the plotted curve at the first data point that deviated upwards (in the positive Y-direction) from the initial baseline level. The customised Agilent VEE Pro 6.0 program then extracted the X-coordinate value of the cursor position, which was the time point of the onset of the movement trial, and stored this value in a text file for analysis.

Movement offset

The offset of movement was defined as the time point at which the right lateral movement of the mandibular MIPT returned to the starting position (Figure 16). This was determined in the same way as that for the movement onset. For the movement offset the second moveable cursor was positioned along the plotted curve at the first data point that returned to the initial baseline level. The X-coordinate value of the cursor position, which was the time point of the offset of the movement trial, was extracted and stored in a text file for analysis.

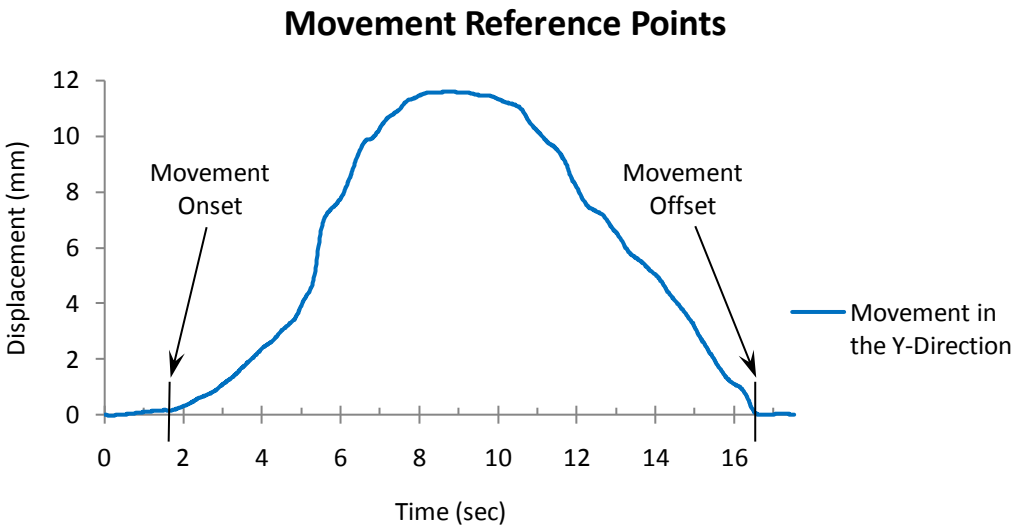

Figure 16: The movement reference points as taken from a typical movement trace from one movement trial from one subject. Movement onset was the time point at which the first data point deviated upwards from the initial baseline level. Movement offset was the first data point that returned to the initial baseline level

EMG activity onset

The onset of the EMG activity was defined as the time point at which the normalised EMG data file deviated by two standard deviations from the mean of the initial baseline resting level (Figure 17). The normalised EMG data file was displayed on a computer screen using a program developed with Agilent VEE Pro 6.0 (Appendix 1). The data in each normalised EMG data file was plotted on a X-Y scatterplot which had two moveable cursors programmed onto the graph, similar to those for the determination of the movement onset and offset. First the baseline was determined by placing two moveable cursors approximately 0.25 – 0.30 seconds apart on the plotted normalised EMG data file. The cursors were placed along a relatively flat area of the plotted curve during the initial rest phase of the movement trial. At a sampling rate of 2000 Hz this produced a sample size of 500 – 600 data points from which to calculate an average baseline or resting EMG

value. The EMG value equivalent to the mean resting EMG value plus two standard deviations was calculated. The time point at which the normalised EMG data file reached this EMG value was denoted the EMG activity onset time and recorded.

EMG activity offset

The offset of the EMG activity was defined as the time point at which the normalised EMG data file returned to the EMG onset value as calculated above (Figure 17).

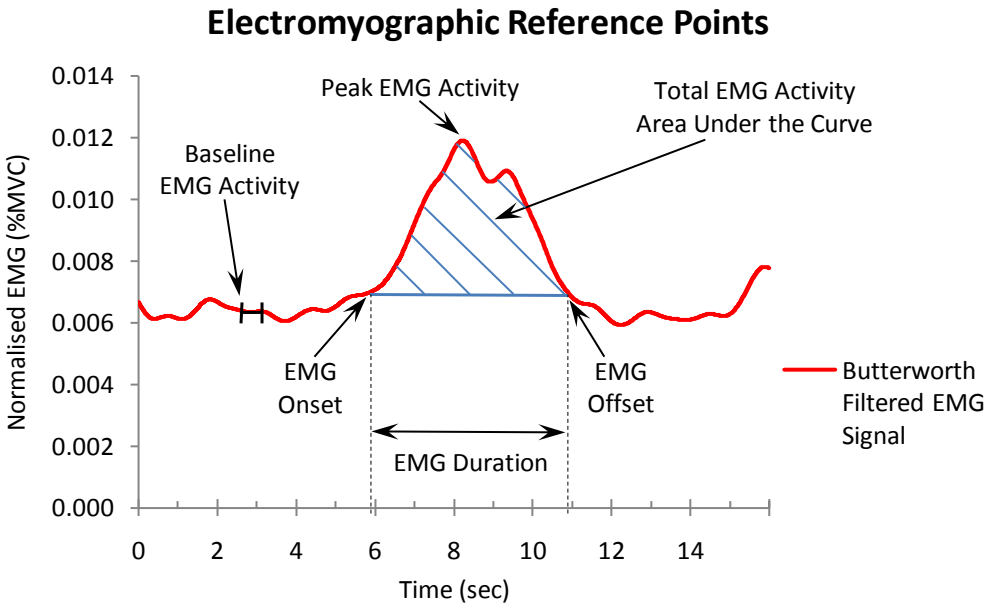

Figure 17: An example of a single rectified and Butterworth filtered EMG trace depicting some of the reference points and calculated variables of the normalised EMG signal that were used as measures of the activity of the tested muscles.

Ensuring similar pre- and post-training movements

Mean Square Error (MSE)

Mean Square Error (MSE) was used to determine the variability of the movement of the mandibular MIPT compared to an optimal movement pattern (Equation 1).

$$MSE = \frac{\sum (y_i - \hat{y}_i)^2}{n-2} \quad \text{..... Equation 1}$$

The optimal movement pattern was determined from the Spike2 software scripts used to sequence the LED illumination for standardising the subject's jaw movement during each of the movement trials. These LEDs were turned on sequentially during each movement trial and controlled by software scripts embedded in Spike2. By plotting the time course of the position of each LED as determined by the script, it was possible to produce a curve of the optimal movement pattern for each movement (Figure 19). The raw movement data was then compared to the theoretical optimal movement pattern by calculating the MSE.

Comparing pre-and post-training electromyographic (EMG) activity

The normalised EMG signals were compared from trial to trial and between different data collection sessions (ITS Pre, ITS Post, TS1 and TS2). An analysis of total activity, maximum activity, rate of activity increase and timing of activity onset and offset of the normalised EMG signal was undertaken. The variables of the EMG signal which were analysed included:

EMG Activity Characteristics

Area under the curve (AUC)

The area under the curve (AUC) was used as a measure of the total EMG activity of the muscle during the movement task. The AUC was calculated as the integral of the rectified

and Butterworth filtered EMG data file between the EMG Onset and the EMG Offset (Figure 17 and Equation 2):

$$AUC = \int_{EMG\ Onset}^{EMG\ Offset} (Normalised\ EMG\ curve\ above\ baseline + 2SD)$$

..... Equation 2

Peak EMG activity (EPk)

The peak EMG activity was the maximum value of the rectified and Butterworth filtered EMG data file between the point of EMG activity onset and the point of EMG activity offset (Figure 17).

Temporal EMG Characteristics/Variables

Time to peak EMG activity (ETPk)

The time to peak EMG activity was defined as the time taken from the onset of the EMG activity to the peak EMG activity of the rectified and Butterworth filtered EMG data file (Figure 17 and Equation 3).

$$ETPk = Time\ of\ Peak\ EMG\ Activity - EMG\ Onset\ Time$$

..... Equation 3

Time to peak EMG activity in relation to movement onset (ETPm)

The time to peak EMG activity in relation to the onset of the movement was defined as the time taken to reach the EPk (Figure 17) relative to the onset of jaw movement (Figure 16 and Equation 4). Positive values indicated that the peak EMG activity occurred after the movement onset.

$$ETPm = ETPk - Movement\ Onset\ Time$$

..... Equation 4

Duration of EMG activity (ED)

The duration of the EMG activity was defined as the time between the onset of EMG activity and the offset of EMG activity (Figure 17 and Equation 5).

$$ED = EMG\ Offset\ Time - EMG\ Onset\ Time$$ ..... Equation 5

Relative onset time (ROT)

The relative onset time of the EMG activity was defined as the time interval between the movement onset time (Figure 16) and the EMG activity onset time (Figure 17). Positive values indicated that the EMG activity onset occurred after the onset of jaw movement (Equation 6).

$$ROT = EMG\ Onset\ Time - Movement\ Onset\ Time$$ ..... Equation 6

Relative offset time (RET)

The relative offset time of the EMG activity was defined as the time interval between the movement offset time (Figure 16) and the EMG activity offset time (Figure 17). Positive values indicated that the EMG activity offset occurred after the offset of jaw movement (Equation 7).

$$RET = EMG\ Offset\ Time - Movement\ Offset\ Time$$ ..... Equation 7

Statistical Analyses

Statistical analyses were conducted using SPSS for Windows (version 15: SPSS Inc, Chicago, Illinois, USA). Each subject's data for each of the calculated variables for each movement trial was recorded. The mean value of all the movement trials was then calculated for each variable (Mean of Trials). Statistical analyses were then conducted on these Mean of Trials values for each of the tested variables from each subject. All the

Mean of Trials values were initially tested to determine if they were normally distributed for each condition using the Kolmogorov-Smirnov (K-S) Test of Normality. If the results of the K-S test showed the data to be significantly different ($p < 0.05$) from the normal distribution then non-parametric tests were utilised to test the null hypotheses. To test for differences between the Control Group and the Exercise Group the Mann-Whitney test, two-tailed, was used. The Friedman test was used to test for significant differences within subjects. For all the non-parametric statistical tests the α-value was set at 0.05 and significance was accepted at $p < 0.05$. In the situations where non-parametric tests were used the median and standard error (SE) were reported. If the K-S test was non-significant ($p > 0.05$) then the data was considered to be normally distributed and an analysis of variance (ANOVA) was used with planned contrasts. The planned contrasts related to the hypotheses and hence attempted to answer the experimental questions as outlined in Table 3. In the situations where an ANOVA was used the mean and standard deviation (SD) were reported.

Hypothesis	Experimental Question	Planned Contrast SPSS Syntax
1. Resistance exercise produces immediate EMG changes in the muscle.	Are there differences in the EMG activity between ITS Pre and ITS Post?	1 -1 0 0
2. After four weeks of a home-based exercise programme, resistance exercise produces EMG changes in the muscle.	Are there differences in EMG activity between ITS Pre and TS1?	-1 0 1 0
3. The effects of a four-weeks home-based exercise programme produce longer lasting changes in the EMG activity of the muscle.	Are there differences in EMG activity between TS1 and TS2?	0 0 -1 1

Table 3: List of hypotheses and experimental questions with the corresponding SPSS syntax for the planned contrasts used in the statistical analyses (EMG = electromyographic).

With three levels of the planned contrasts the Bonferroni correction was used to determine the critical p-value ($p_{critical}$) for statistical significance of the contrasts tested as illustrated in Equation 8.

$$p_{ctitical} = \frac{\alpha}{3} = \frac{0.05}{3} = 0.0167 \quad \text{..... Equation 8}$$

RESULTS

Subjects

Table 4 summarises the demographic details of the twelve volunteers that participated in this study. Eleven participants completed all three data collection sessions, namely the Initial Training Session (ITS), Testing Session 1 (TS1) and Testing Session 2 (TS2), with one subject unable to attend TS2. Seven participants were female and five were male. The average age of all participants was 26.33 years (SD = 2.95, range 20.87 – 31.84 years). A total of six subjects were randomised into the Exercise Group (four females and two males) and six subjects were randomised into the Control Group (three females and three males). For all participants the average time between the ITS and TS1 was 30.75 days (SD = 8.04, range 19 – 48 days), while the average time between TS1 and TS2 was 29.91 days (SD = 5.84, range 20 – 42 days).

Subject 11 did not attend the final testing session (Table 4) and therefore both the movement and electromyographic (EMG) data from this subject were omitted from the analyses. This resulted in the analyses being carried out on a sample size of n = 5 for the control group and n = 6 for the exercise group except in the following case. The data relating to the digastric muscle for Subject 2 was of poor quality due to technical issues during data collection and processing. This made it impossible to process and calculate the tested variables for the digastric muscle of Subject 2. This data was omitted from the analyses which resulted in a sample size of n = 5 for the exercise group when testing the digastric muscle. For statistical analyses of the other four muscles the sample size was n = 6 for the exercise group.

Appendix 2 shows a graphical representation of the means and standard deviations of each variable across all testing sessions for each muscle. With seven variables calculated for each of the five muscles tested, a total of 35 variables were analysed. Of the seven calculated variables, four showed significant changes in at least one of the tested muscles, namely Time to Peak EMG activity (ETPk), Duration of EMG activity (ED), Time to Peak EMG activity with relation to Movement Onset (ETPm) and Relative Offset Time (RET). Across all the muscles this resulted in a total of eleven variables showing significant changes out of the possible 35 that were analysed. The relevant (significant) findings are described in the following text.

Subject ID	Gender	Group	Age At ITS (Years)	Time Between ITS and TS1 (Days)	Time Between TS1 and TS2 (Days)
1	Female	Exercise	30.72	28	29
2	Male	Exercise	26.62	28	27
3	Female	Exercise	24.97	29	28
4	Female	Exercise	28.00	36	35
5	Male	Control	24.61	29	42
6	Female	Control	24.84	48	28
7	Male	Exercise	26.33	34	27
8	Female	Control	31.84	28	30
9	Female	Exercise	27.59	28	20
10	Female	Control	24.90	21	27
11	Male	Control	24.69	41	DNA TS2
12	Male	Control	20.87	19	36
Total	**12**	**Mean**	**26.33**	**30.75**	**29.91**
Females	7	SD	2.95	8.04	5.84
Males	5	Minimum	20.87	19	20
		Maximum	31.84	48	42

Total in Control Group	**6**	**Total in Exercise Group**	**6**
Females	3	Females	4
Males	3	Males	2

Table 4: Subject details showing information pertaining to individual subjects as well as the various group break downs (ITS = Initial Training Session; TS1 = Testing Session 1; TS2 = Testing Session 2; SD = Standard Deviation; DNA = Did Not Attend).

Right Lateral Jaw Movements

An example of the graphical representation of the processed data of a typical right lateral jaw movement is illustrated in Figure 18. This displays the outgoing phase and return phase displacements in the X-, Y- and Z-directions of the mandibular mid-incisor point (MIPT) from the resting position during a single right lateral movement. The data from the Y-direction (Figure 18, red line) was used for the calculation of all the measured variables (see Jaw Movement section of Methods, page 53 for details).

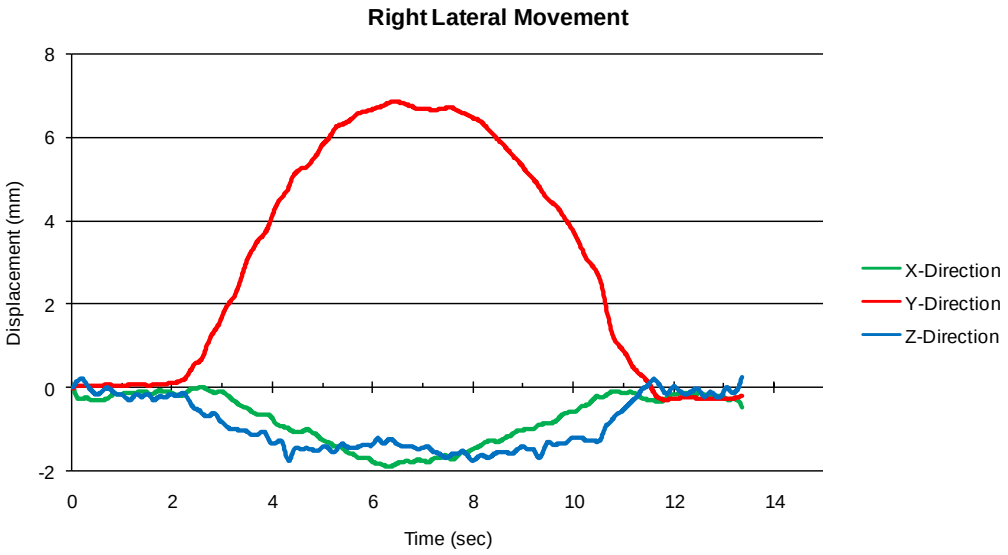

Figure 18: Graphical representation of the raw movement data from JAWS3D depicting the three directions of a typical movement pattern of the mandibular mid-incisor point (MIPT). This data was taken from one subject during the Initial Training Session. Antero-posterior movement occurred in the X-direction (green line) with anterior movements being positive, lateral movement occurred in the Y-direction (red line) with right sided movement being positive, and vertical movement occurred in the Z-direction (blue line) with superior movements being positive.

Mean Square Error (MSE)

Figure 19 shows the raw movement data traces (red lines) of all the movement trials for right lateral movement from one subject during one of the data collection sessions. Superimposed on these red movement traces is the trace of the optimal movement pattern (blue line, Figure 19) as determined by the sequential lighting of the bank of LEDs used to control the subject's movement pattern in terms of displacement and duration.

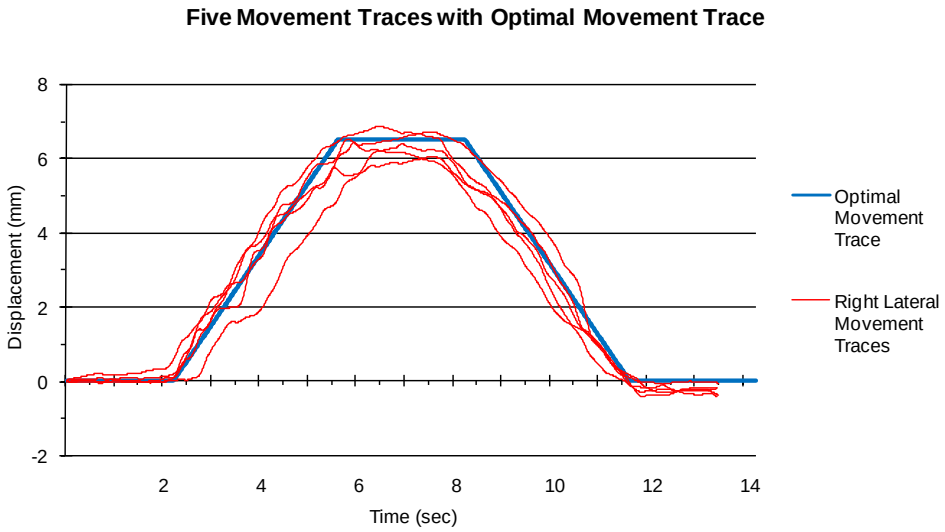

Figure 19: Graphical representation of five right lateral movement trials from one subject during Testing Session 1 (TS1, red lines). Superimposed on these movement trials is the optimal movement trace (blue line) which was calculated from the Spike2 scripts (see Methods, Mean Square Error (MSE) section, page 70 for details).

The mean MSE (+/-SD) is illustrated in Figure 20. The Kolmogorov-Smirnov (K-S) test for normality showed that the data in three out of the possible eight conditions were significantly different from the normal distribution (ITS Pre Control Group: D(6) = 0.365, p = 0.012; ITS Post Control Group: D(6) = 0.349, p = 0.021; and, TS2 Exercise Group: D(6) = 0.375, p = 0.008). With the data not being normally distributed, non-parametric tests were used. The Mann-Whitney test showed no significant difference in the MSE of the right lateral jaw movements between the Exercise Group and the Control Group across all testing sessions (U = 17.00, p = 0.87 2 tailed, for ITS Pre; U = 18.00, p = 1.00 2 tailed, for ITS Post; U = 12.00, p = 0.34 2 tailed, for TS1; U = 8.00, p = 0.20 2 tailed, for TS2). The Friedman test showed no significant differences within subjects across the testing sessions (Chi-square $\chi^2_F = 4.2$, p = 0.241). Therefore, there were no significant differences between the movement trials completed by individual subjects at each of the testing

sessions. There were also no significant differences in the movement trials between the Control Group and the Exercise group.

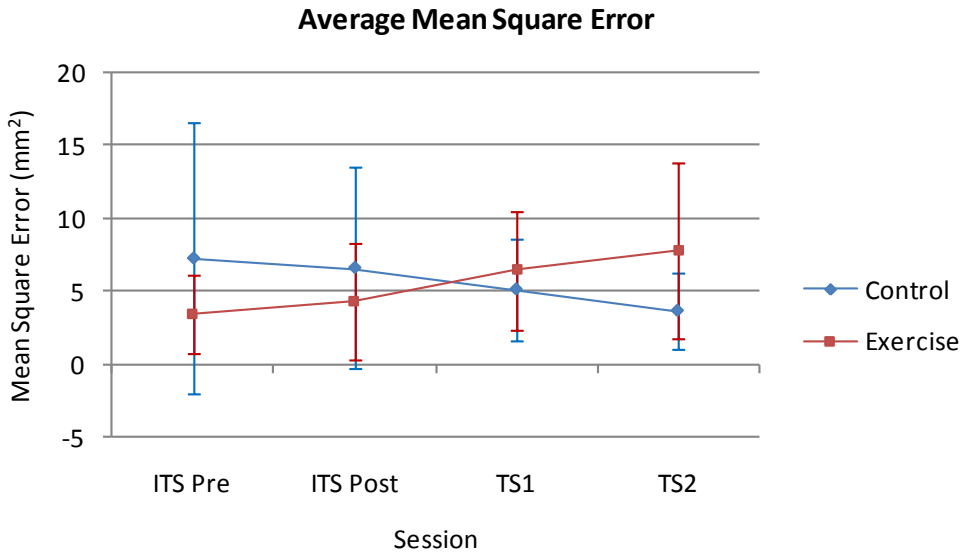

Figure 20: Average mean square error (MSE +/- SD) from control subjects (blue diamonds) and exercise subjects (red squares) across all data collection sessions for right lateral movement. There was no significant difference in the MSE within subjects or between subjects over the four data collection sessions (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Figure 21 shows a representative example from one subject comparing the movement trials during ITS Pre (red traces) with the movement trials during ITS Post (blue traces), TS1 (green traces) and TS2 (yellow traces). Qualitatively there is little change in the right lateral movement traces between the four data collection sessions. All movement trials from all subjects were retained for the analysis of the EMG activity data except for the two

cases where the data were omitted as explained above (ie Subject 2 and Subject 11, see Subjects section, page 75, for details).

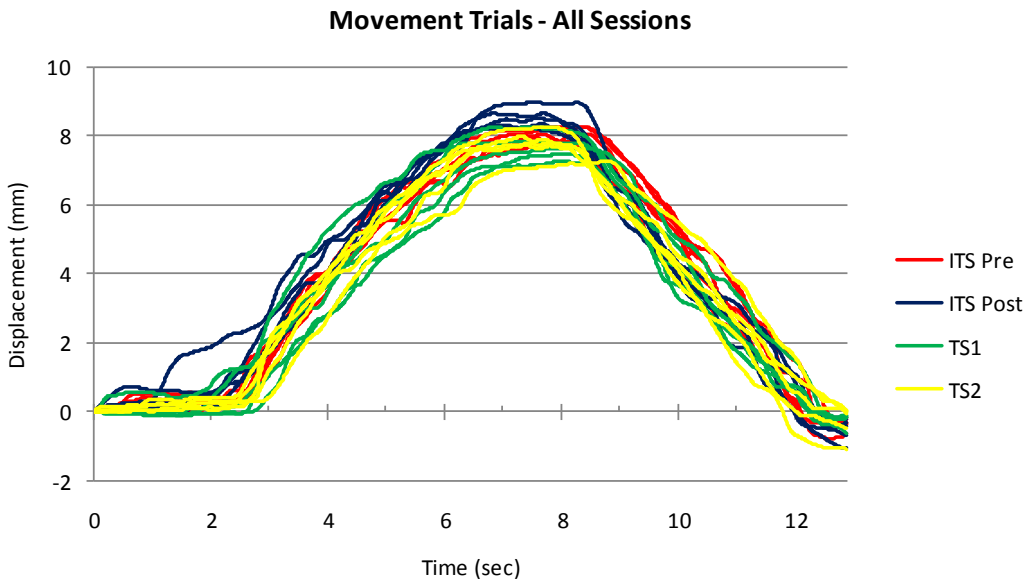

Figure 21: A representative example of the right lateral movement trials during each data collection session from one subject. Qualitatively there was little difference in the right lateral movement traces between the four data collection sessions (ITS Pre = pre-exercise data collection during the Initial Training Session, ITS Post = post-exercise data collection during the Initial Training Session, TS1 = Testing Session 1 four weeks after the ITS, TS2 = Testing Session 2 eight weeks after the ITS).

Electromyography

Seven variables were calculated for each of the five muscles tested giving a total of 35 variables that were analysed. Of the seven calculated variables, four showed significant changes in at least one of the tested muscles, namely Time to Peak EMG activity (ETPk), Duration of EMG activity (ED), Time to Peak EMG activity relative to Movement Onset (ETPm) and EMG Offset Time relative to Movement Offset (RET). Across all the

muscles this resulted in a total of eleven variables showing significant changes out of the possible 35 that were analysed. The results are described below in terms of overall muscle activity and also in terms of the relative activation of the muscles in relation to right lateral jaw movement.

Muscle Activity

Total Muscle Activity

The area under the curve (AUC) of the normalised, Butterworth-filtered EMG activity for each of the tested muscles was used as a measure of the total electrical activity in the muscle during the movement task. The K-S test for normality showed that the data in at least one condition for each muscle were significantly different from the normal distribution ($p < 0.05$). For all the tested muscles the Mann-Whitney test showed no significant difference in the AUC for the right lateral jaw movements between the exercise group and the control group across all testing sessions ($p > 0.05$). The Friedman test showed no significant differences within subjects across the testing sessions ($p > 0.05$).

Peak Muscle Activity (EPk)

Table 5 shows the mean EPk values of all subjects as a percentage of maximal voluntary contraction (%MVC) for each of the five tested muscles during the right lateral movement trials during each of the four testing sessions. The breakdown of the mean EPk values for each experimental group can be seen in Appendix 3 (Table 6). The maximum mean EPk was 17.00% of MVC (SD = 28.60) as seen in the right anterior temporalis during ITS post and the minimum mean EPk was 4.26% of MVC (SD = 3.15) as seen in the left anterior temporalis during TS1. The EPk was less than 10% of MVC in 14 out of 20 possible muscle-testing session combinations (70%) and less than 5% of MVC in seven out of 20 possible muscle-testing session combinations (35%).

Muscle	Testing Session	Mean Peak EMG (%MVC)	SD	n
Right Anterior Temporalis	ITS Pre	11.31	15.61	11
	ITS Post	17.00	28.60	11
	TS1	8.83	11.69	11
	TS2	8.41	7.33	11
Left Anterior Temporalis	ITS Pre	6.45	6.00	11
	ITS Post	6.08	6.92	11
	TS1	4.26	3.15	11
	TS2	4.59	3.67	11
Right Masseter	ITS Pre	4.47	2.67	11
	ITS Post	7.11	7.70	11
	TS1	4.63	4.91	11
	TS2	4.73	4.17	11
Left Masseter	ITS Pre	4.98	4.20	11
	ITS Post	5.65	4.26	11
	TS1	4.35	3.65	11
	TS2	5.22	6.11	11
Digastric	ITS Pre	12.57	6.14	10
	ITS Post	14.44	8.37	10
	TS1	16.01	9.27	10
	TS2	12.53	7.53	10

Table 5: Mean peak electromyographic (EMG) activity of all subjects as a percentage of maximal voluntary contraction (%MVC) for each of the five tested muscles during the right lateral movement trials (SD = standard deviation; n = sample size; ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

The K-S test of normality showed that the mean EPk data were not significantly different from the normal distribution in three of the tested muscles: right masseter; left masseter; and right digastric. ANOVA revealed no significant differences in the EPk following the application of isotonic resistance exercise in any of these muscles ($p > 0.05$). Non-

parametric tests conducted on the data from right anterior temporalis and left anterior temporalis also revealed no significant difference ($p > 0.05$) in the EPk following the application of isotonic resistance exercise.

Time-to-Peak Muscle Activity (ETPk)

The K-S test of normality showed that the mean ETPk data were not significantly different from the normal distribution for both the control and exercise groups across all testing sessions for all the tested muscles ($p > 0.05$ for all conditions). Therefore, ANOVA was used to analyse the mean data for ETPk. The time-to-peak muscle EMG activity showed significant changes in three out of five muscles tested: left anterior temporalis; left masseter; and, digastric. No significant changes were seen in the right anterior temporalis or the right masseter ($p > 0.05$). Figure 22 illustrates the significant changes in ETPk in the other three muscles. The significant changes are also described below.

Left anterior temporalis

Figure 22a illustrates the changes in the ETPk of the left anterior temporalis. ANOVA revealed a significant difference in within-subjects effects across the data collection sessions ($F_{3,27} = 3.889$, $p = 0.020$). The tests of within-subjects contrasts showed a significant difference between TS1 and TS2 ($F_{1,9} = 13.997$, $p = 0.005$). The Control Group showed a reduction in the mean ETPk of left anterior temporalis over the four weeks from 5.25 sec (SD = 0.94) at TS1 to 3.57 sec (SD = 1.58) at TS2. The Exercise Group also showed a reduction in the mean ETPk of left anterior temporalis over the same period from 4.64 sec (SD = 2.57) at TS1 to 2.56 sec (SD = 1.18) at TS2.

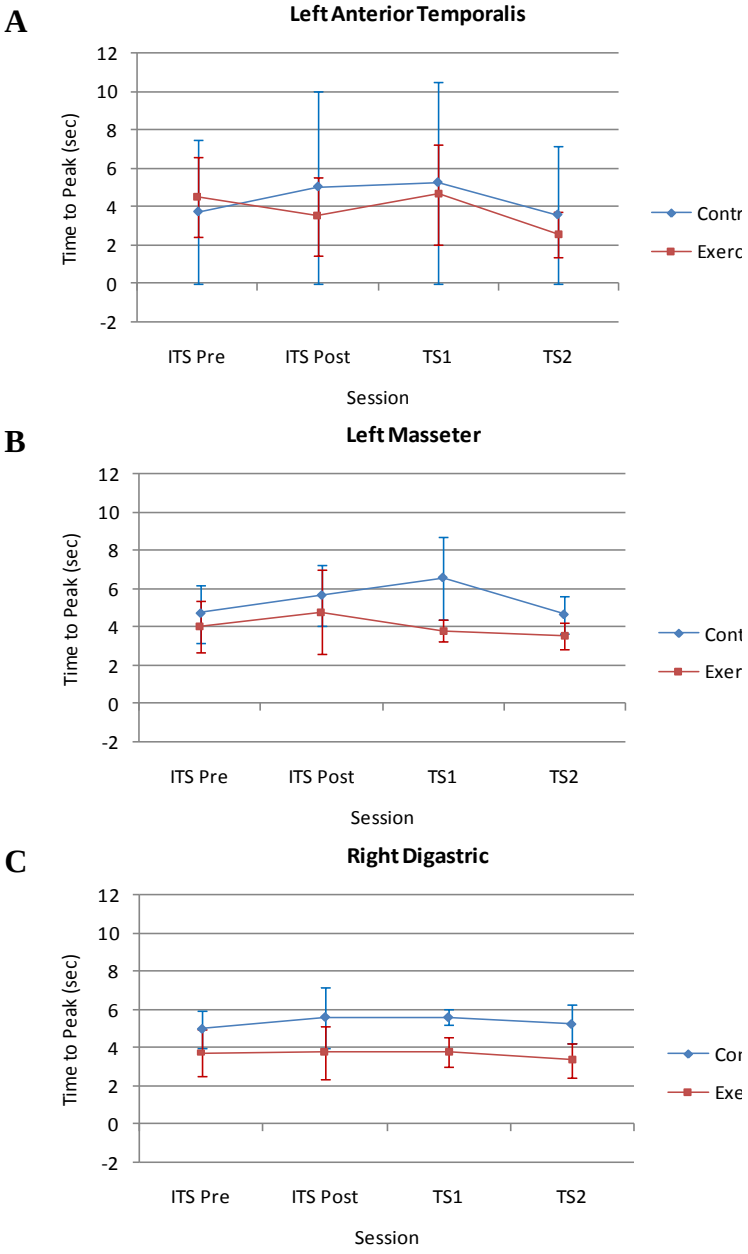

Figure 22: Mean (+/- SD) time to peak muscle activity (ETPk) values of the muscles that showed significant changes. A: Left anterior temporalis. B: Left masseter. C: Right digastric. (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Left masseter

Figure 22b illustrates the changes in the ETPk of the left masseter. ANOVA revealed a significant difference in the between-subjects effects between groups ($F_{1,9} = 5.415$, $p = 0.045$). Analysis of covariance (ANCOVA), taking account of the differences between the Exercise Group and the Control Group as a covariate, showed a significant difference in the ETPk between the two groups at TS1. At TS1 the Control group had a significantly higher mean ETPk (Corrected mean = 6.23, SD = 2.10) than that of the Exercise Group (Corrected mean = 3.79, SD = 0.56; $F_{2,9} = 4.52$. $p = 0.04$).

Digastric

Figure 22c illustrates the changes in the ETPk of the digastric. ANOVA revealed a significant difference in the between-subjects effects between groups ($F_{1,8} = 12.236$, $p = 0.008$). ANCOVA, taking account of the differences between the Exercise Group and the Control Group as a covariate, revealed that the mean ETPk of the Exercise Group (Corrected mean = 3.76, SD = 1.41) was significantly lower than that of the Control Group (Corrected mean = 5.49, SD = 1.45) at ITS Post ($F_{2,8} = 10.424$, $p = 0.006$). During TS1 the mean ETPk of the Exercise Group (Corrected mean = 3.78, SD = 0.75) was also significantly lower than that of the Control Group (Corrected mean = 5.72, SD = 0.48; $F_{2,8} = 13.628$, $p = 0.003$).

Duration of EMG Activity (ED)

The K-S test for normality showed that the data in at least one condition were significantly different from the normal distribution ($p < 0.05$) for each of the following muscles: right anterior temporalis, right masseter, and left masseter. Non-parametric tests were used to test for significant differences in these muscles. ANOVA was used to test for significant differences in the left anterior temporalis and right anterior digastric.

The ED showed significant changes in three out of five muscles tested. No significant changes were seen in the right masseter or the digastric ($p > 0.05$). Figure 23 illustrates the significant changes in ED in the other three muscles.

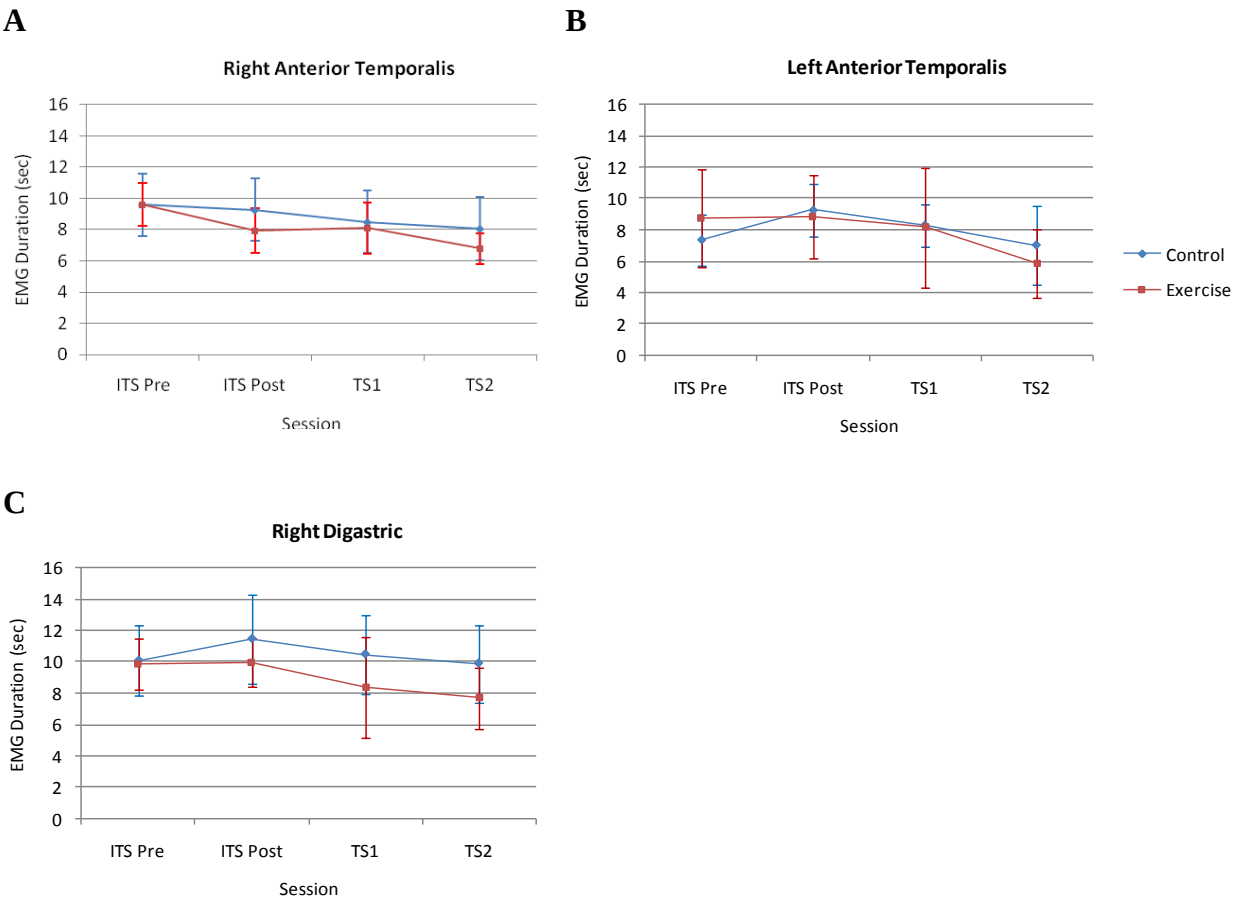

Figure 23: Muscles that showed significant differences in the duration of EMG activity (ED). A: Median ED (+/- SE) of right anterior temporalis. B: Mean ED (+/- SD) of left anterior temporalis. C: Mean ED (+/- SD) of digastric. (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Right anterior temporalis

Figure 23a illustrates the changes in the ED of the right anterior temporalis. The Mann-Whitney test showed no significant difference between the ED of the right anterior temporalis of the Exercise Group and the Control Group across all testing sessions ($p > 0.05$). The Friedman test showed a significant difference within subjects across the testing sessions (Chi-square $\chi^2_F = 10.964$, $p = 0.012$). The median ED of the right anterior temporalis in the Control Group was 9.59 sec (SE = 0.60) at ITS Pre which decreased slightly to 9.26 sec (SE = 1.00) at ITS Post. The median ED continued to decrease over the subsequent eight weeks being 8.49 sec (SE = 0.51) at TS1 and 8.06 sec (SE = 0.94) at TS2. The median ED of right anterior temporalis in the Exercise Group was 9.59 sec (SE = 1.38) at ITS Pre which decreased to 7.92 sec (SE = 1.43) at ITS Post. After four weeks of home-based isotonic resistance exercise the median ED was 8.09 sec (SE = 1.63) at TS1. This reduced to a median ED of 6.79 sec (SE = 0.97) after four weeks following the cessations of the home-based exercise programme.

Left anterior temporalis

Figure 23b illustrates the changes in the ED of the left anterior temporalis. ANOVA revealed a significant difference in the within-subjects effects across the data collection sessions ($F_{3,27} = 4.823$, $p = 0.008$). The tests of within-subjects contrasts showed a significant difference between TS1 and TS2 ($F_{1,9} = 37.261$, $p = 0.007$). The Control Group showed a reduction in the mean ED of left anterior temporalis over the four weeks between TS1 (8.31 sec, SD = 1.36) and TS2 (7.02 sec, SD = 2.54). The Exercise Group showed a greater reduction in the mean ED of left anterior temporalis over the four weeks between TS1 (8.18 sec, SD = 3.82) and TS2 (5.88 sec, SD = 2.21).

Digastric

Figure 23c illustrates the changes in the ED of the digastric. ANOVA revealed a significant difference in the within-subjects effects across the data collection sessions ($F_{3,27}$ = 3.516, p = 0.030). The tests of within-subjects contrasts showed a significant difference between ITS Pre and ITS Post ($F_{1,8}$ = 9.244, p = 0.016). There was an increase in the mean ED in the Control Group following the initial application of isotonic resistance exercise from 10.07 sec (SD = 2.25) at ITS Pre to 11.47 sec (SD = 2.82) at ITS Post. The Exercise Group showed no increase in the mean ED from ITS Pre (9.85 sec, SD = 1.62) to ITS Post (9.93 sec, SD = 1.53).

Muscle Activity in Relation to Jaw Movement

Time to Peak EMG Activity in Relation to Movement Onset (ETPm)

The K-S test for normality showed that the data in at least one condition were significantly different from the normal distribution ($p < 0.05$) for the left masseter and the right digastric. Non-parametric tests were used to test for significant differences in these muscles. ANOVA was used to test for significant differences in the right anterior temporalis, left anterior temporalis and right master. Four of the tested muscles showed significant changes in the ETPm ($p < 0.05$). No significant changes were seen in the right masseter ($p > 0.05$).

Right anterior temporalis

Figure 24a illustrates the changes in the ETPm of the right anterior temporalis. In the tests for within-subjects effects ANOVA revealed a significant difference in the testing session by group interaction ($F_{3,27}$ = 3.738, p = 0.023). The tests of within-subjects contrasts showed a significant difference between ITS Pre and TS1 ($F_{1,9}$ = 11.108, p = 0.009). Subjects in the Exercise Group had an initial ETPm of 6.21 sec (SD = 1.47) at ITS Pre

which decreased to 5.57 sec (SD = 1.37) at TS1 after four weeks of isotonic resistance exercise training. Subjects in the Control Group had an initial ETPm of 5.72 sec (SD = 1.72) at ITS Pre which increased to 7.19 sec (SD = 1.77) at TS1 after four weeks of continuing with their normal activities of daily living and not undertaking isotonic resistance exercises of the jaw.

Left anterior temporalis

Figure 24b illustrates the changes in the ETPm of the left anterior temporalis. ANOVA revealed a significant difference in within-subjects effects across the data collection sessions ($F_{3,27} = 3.343$, $p = 0.034$). Tests for within-subjects contrasts revealed a significant difference between TS1 and TS2 ($F_{1,9} = 14.063$, $p = 0.005$). Subjects in the Control Group showed a reduction in the mean ETPm from 7.01 sec (SD = 2.03) at TS1 to a mean ETPm of 5.97 sec (SD = 2.02) during TS2. Subjects in the Exercise Group showed a reduction in the mean ETPm from 6.96 sec (SD = 2.85) in TS1 to a mean ETPm of 4.77 sec (SD = 1.31) during TS2.

Left masseter

Figure 24c illustrates the changes in the ETPm of the left masseter. The Friedman test showed no significant difference within subjects across the testing sessions (Chi-square $\chi^2_F = 4.855$, $p = 0.183$). The Mann-Whitney test showed a significant difference in the ETPm of the left masseter between the groups at TS1 ($U = 4.00$, $p = 0.025$, 2 tailed). The median ETPm of left masseter in the Control Group at TS1 was 9.75 sec (SE = 0.96). This rose from a median ETPm at ITS Pre of 7.86 sec (SE = 0.75) and subsequently fell to a median ETPm of 6.63 sec (SE = 1.12) at TS2. The median ETPm of the Exercise Group at TS1 was 5.85 sec (SE = 0.56). This fell slightly from a median ETPm of 6.17 sec (SE = 0.95) at ITS Post and continued to fall to a median ETPm of 5.04 sec (SE = 0.47) at TS2.

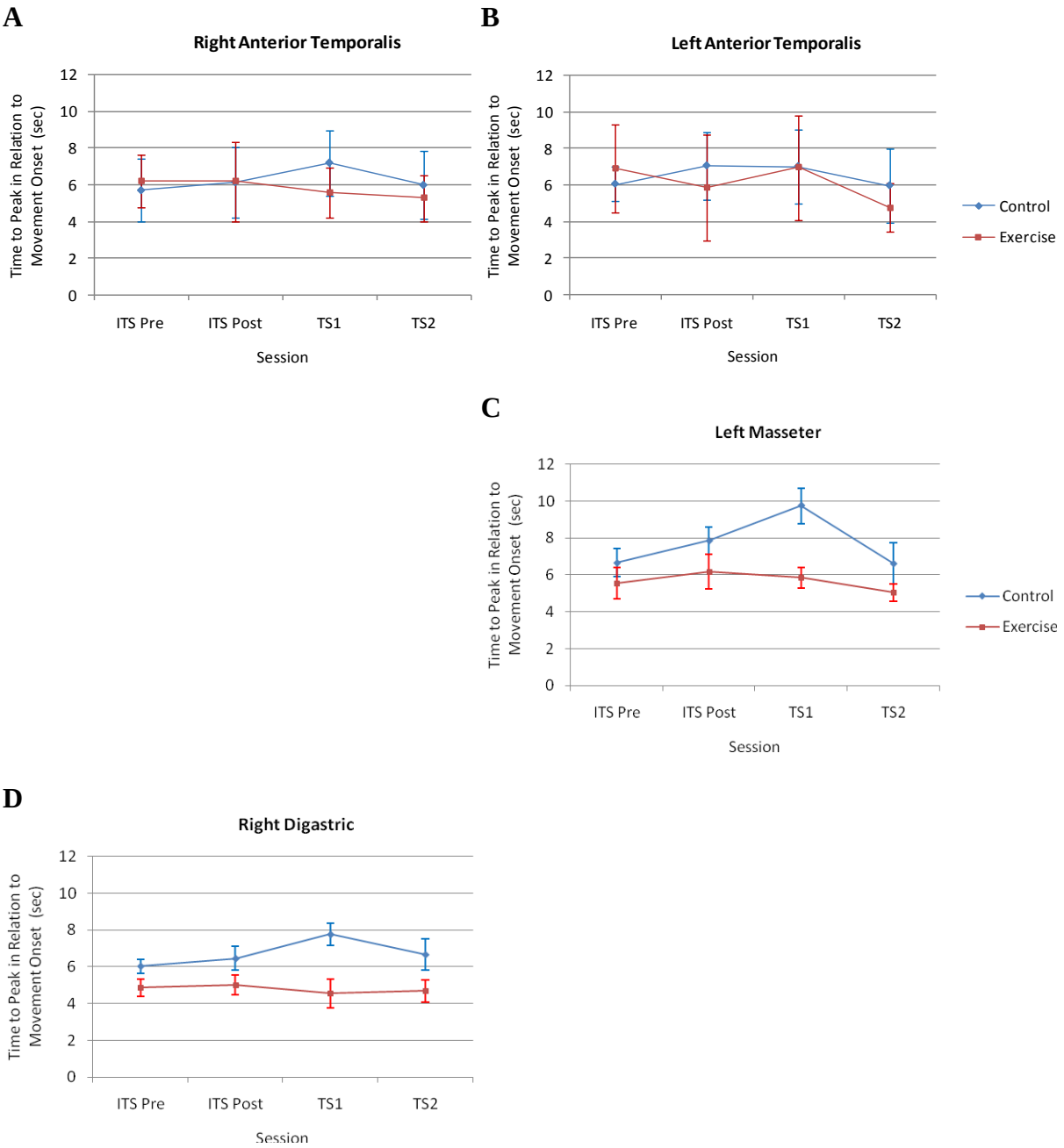

Figure 24: Muscles that showed significant differences in the time to peak muscle activity in relation to the onset of right lateral jaw movement (ETPm). A: Mean ETPm (+/- SD) of right anterior temporalis. B: Mean ETPm (+/- SD) of left anterior temporalis. C: Median ETPm (+/- SE) of left masseter. D: Median ETPm (+/- SE) of right digastric. (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Digastric

Figure 24d illustrates the changes in the ETPm of the digastric. The Mann-Whitney test showed a significant difference between the Control Group and the Exercise Group at ITS Pre (U = 1.00, p = 0.01, 2 tailed) and at ITS Post (U = 4.00, p = 0.045, 2 tailed). The Friedman test showed no significant difference within subjects across the testing sessions (Chi-square $\chi^2_F = 0.36$, p = 0.948). ANCOVA, adjusting for the difference between the Control Group and the Exercise Group revealed a significant difference in the ETPm of the digastric at ITS Post and TS1. The corrected means for the Control Group were 6.71 sec (SD = 1.58) at ITS Post and increasing to 7.38 sec (SD = 1.44) at TS1. The corrected means for the Exercise Group were 4.83 sec (SD = 1.21) at ITS Post and decreasing to 4.48 sec (SD = 0.24) at TS1.

Relative Onset of Muscle Activity (ROT)

The K-S test for normality showed that the data in at least one condition were significantly different from the normal distribution ($p < 0.05$) for the right masseter, left masseter and right digastric. Non-parametric tests were used to test for significant differences in these muscles. ANOVA was used to test for significant differences in the right anterior temporalis and the left anterior temporalis. There were no significant differences in the ROT following the application of isotonic resistance exercise in any of the muscles tested ($p > 0.05$).

Relative Offset of Muscle Activity (RET)

The K-S test for normality showed that the data in at least one condition were significantly different from the normal distribution ($p < 0.05$) for the right anterior temporalis. Non-parametric tests were used to test for significant differences in the right anterior temporalis. ANOVA was used to test for significant differences in the left anterior temporalis, right

masseter, left masseter and digastric muscles. No significant changes were seen in the right anterior temporalis, left anterior temporalis, the right masseter or the digastric ($p > 0.05$). Figure 25 illustrates the significant changes in RET in the left masseter.

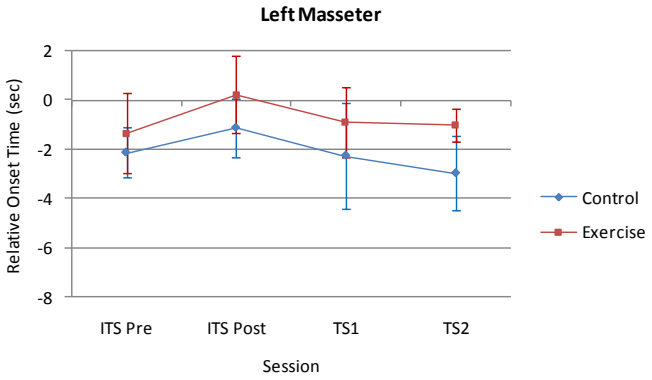

Figure 25: Mean (+/- SD) relative offset time of electromyographic (EMG) activity in relation to the offset of right lateral jaw movement (RET) of the left masseter. Negative values indicate the offset of the EMG activity preceded the movement offset. (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Left masseter

ANOVA revealed a significant difference in the within-subjects effects across the data collection sessions ($F_{3,27} = 3.915$, $p = 0.019$). In the Control Group the EMG offset of left masseter always preceded the end of the return phase of the right lateral movement. At ITS Pre, the EMG offset preceded the end of the movement by a mean of 2.14 sec (SD = 1.03) which reduced to a mean of 1.12 sec (SD = 1.17) at ITS Post and returned to near pre-exercise levels to a mean of 2.26 sec (SD = 2.14) at TS1 and to a mean of 2.98 sec (SD = 1.52) at TS2. In the Exercise Group the EMG offset preceded the end of the return phase of the movement by a mean of 1.35 sec (SD = 1.62) at ITS Pre. Following the initial

isotonic resistance exercise the EMG offset succeeded the end of the movement by a mean of 0.23 sec (SD = 1.58). After four weeks of isotonic resistance exercise the EMG offset again preceded the end of the movement by a mean of 0.91 sec (SD = 1.44) at TS1 and after a further four weeks of no exercise it preceded the end of the movement by a mean of 1.03 sec (SD = 0.68) at TS2.

DISCUSSION

Overview

This thesis investigated the effects of isotonic resistance exercise training on the jaw muscles of asymptomatic adult volunteers. Specifically, the aims of this thesis were to train individuals to use their jaw muscles differently, and to demonstrate that these changes in muscle use can occur in the short term and can be maintained over the longer term following the application of a four-week home-based exercise programme.

Physiotherapy in the form of specific individualised exercise programmes has been used effectively in the treatment of musculoskeletal pain and dysfunction in many areas of the body including the patellofemoral joint (Cowan et al., 2002, Cowan et al., 2001, McConnell, 2002), shoulder (Ginn and Cohen, 2004, Ginn and Cohen, 2005, Ginn et al., 1997) and the lumbar spine (Hides et al., 1996). The underlying principle in the successful treatment of these conditions has been the identification of imbalances in specific muscles or muscle groups. These imbalances have included weakness and loss of muscle bulk (Hides et al., 1994), changes in recruitment patterns (Hides et al., 1996) and changes in the timing of the recruitment patterns of specific muscles (Cowan et al., 2001, Hodges and Richardson, 1996). Addressing these imbalances has resulted in the relief of symptoms and a return to more normal function of these areas (Cowan et al., 2002, Ginn et al., 1997, Hides et al., 1996). It is therefore postulated that by alleviating the underlying biomechanical disturbances in individuals with musculoskeletal dysfunction there will be a relief of symptoms and a return to normal function.

Although exercise has been beneficial in the relief of pain and dysfunction associated with temporomandibular disorders (TMD, Au and Klineberg, 1993, Medlicott and Harris,

2006), the mechanisms underlying the effectiveness of exercise in the treatment of TMD is largely unknown. In order to determine which physiotherapy modality is most appropriate in improving dysfunction in patients with TMD of a musculoskeletal origin it is first important to determine the characteristics of the normal movement patterns of the temporomandibular joint (TMJ). It is also important to determine whether these movement patterns can be changed or otherwise influenced by physiotherapy modalities such as resistance exercises. If these movement patterns can be altered in a reproducible way in asymptomatic individuals it is hoped that these modalities could be applied to plan specific exercise regimens for appropriate TMD patients in an attempt to alleviate their symptoms in the short term and perhaps also reduce the incidence of recurrence.

Significant Findings

This study has demonstrated changes in some of the tested electromyographic (EMG) parameters in the muscles of mastication of asymptomatic individuals at each of the three testing sessions. Some of the variables showed immediate changes following the application of isotonic resistance exercise. Some variables showed changes after the four-week home-based exercise programme, demonstrating a training effect. Some of the variables showed longer-term changes after four weeks following the completion of the home-based isotonic resistance exercise programme. The results of this study need to be interpreted in the context of having been conducted with asymptomatic volunteers. Therefore it is possible to assume that the muscles and their corresponding motor control systems are functioning normally and in an appropriately efficient manner in these individuals. In this case there may not be a need for these muscles to respond to an exercise stimulus. If changes do occur they may be quite small and difficult to detect.

The major findings of this study are discussed below, firstly in terms of the movement characteristics and secondly, in terms of the electromyographic (EMG) characteristics.

Movement Characteristics

Reproducibility of Movement Trials

In this study subjects were able to reproducibly track a target bank of light emitting diodes (LEDs). This is evidenced by the fact that there were no statistically significant changes in the mean square error (MSE) of the movement trials either during each testing session or between each testing session (Figure 20). This implies that the right lateral jaw movement trials were the same within each testing session and between the different testing sessions. Therefore any changes in the EMG activity are most likely due to the effects of the intervention, which was the application of isotonic resistance exercise at 60% of maximal voluntary contraction (MVC) for right lateral movement of the jaw.

Electromyographic Characteristics

The EMG characteristics can be divided into the activity characteristics of the muscle during the movement task and the temporal characteristics of the EMG activity in relation to either, the EMG onset and offset, or in relation to the movement onset and offset. The muscles which showed changes most consistently throughout all the tested variables were the left (contralateral) anterior temporalis, the left (contralateral) masseter and the right (ipsilateral) anterior digastric. These three muscles showed changes in three out of the four variables that had significant changes over the testing period. The right (ipsilateral) anterior temporalis showed changes in two out of the four significant variables. The right (ipsilateral) masseter showed no significant changes in any of the tested variables.

EMG Activity Characteristics

Total Muscle Activity

The area under the curve (AUC) of the normalised, Butterworth-filtered EMG activity for each of the tested muscles was used as a measure of the total muscle activity during the movement task. The application of isotonic resistance exercise resulted in no significant changes in the total EMG activity during right lateral jaw movements in any of the tested muscles either immediately following the application of the isotonic resistance exercise or after four weeks of home-based training. (Appendix 2, Figure 28)

Maximum Muscle Activity

Peak EMG activity was used as a measure of the maximum level of muscle activity generated during the movement task. The application of isotonic resistance exercise resulted in no significant changes in the maximum muscle activity during right lateral jaw movements in any of the tested muscles either immediately following the application of the isotonic resistance exercise or after four weeks of home-based training. (Appendix 2, Figure 29)

Interpretation of the EMG Activity Characteristics

The results that there were no changes in either the total EMG activity or the maximum EMG activity following the application of isotonic resistance exercise could be explained by the fact that free jaw movements, such as the right lateral movement task performed in this study, produce small amounts of EMG activity in the tested muscles. On average the maximum peak EMG activity in this study was 17% of maximum voluntary contraction (MVC) with 14 out of a total of 20 (70%) of the possible muscle-testing session combinations recording a mean maximum peak activity of less than 10% MVC (Table 5). Qualitatively these values appear to be lower than other measures of peak muscle activity

(Long, 2004). In particular, a recent study investigating the use of surface EMG of the bilateral anterior temporalis and masseter muscles in pattern recognition during simulated bruxing tasks used 90% of the normalised peak EMG activity (SigOn90Y) as a measure of EMG amplitude (Long, 2004). In the reproducibility portion of this study a subject was asked to perform fast and slow grinding tasks 15 times each against an appliance in right lateral, left lateral and protrusive directions on five consecutive days. Qualitative review of the results (see Figure 10 in Long, 2004) shows that the mean SigOn90Y values for each of the four muscles were greater than 10% MVC in 103 out of 120 (86%) of the possible muscle-day-task combinations. When looking at just the right lateral movement data the mean SigOn90Y values for each muscle were greater than 10% MVC in 32 out of 40 (80%) of the possible muscle-day-task combinations and greater than 20% MVC in 16 out of 40 (40%) of the possible muscle-day-task combinations. This higher EMG activity is probably due to the fact that a simulated bruxing task against an appliance was used during the movement tasks (Long, 2004). Applying a clenching force increases the relative EMG activity of the masseter and temporalis muscles given that their primary role is to close the jaw (Basmajian and Luca, 1985, Miller, 1991). It is possible therefore, that due to the overall small size of the EMG activity during the free right lateral movement tasks in this study, any changes that may have occurred over time or in response to the exercise stimulus may have been too small to be recorded. Furthermore, if they were recorded, they may have been too small to be statistically significant. Future studies could investigate the effects of exercise on movement during a bruxing task.

The muscles tested in this study were the anterior temporalis and masseter bilaterally and the right anterior digastric. These muscles were chosen due to their ease of access in a clinical setting. Surface EMG electrodes are easily placed in a reproducible position on the

skin overlying the temporalis, masseter and anterior digastric muscles. These muscles contribute to producing a lateral force vector that would result in lateral movement of the jaw (Figure 26 and Table 1, Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991). It has also been shown that 96% of jaw movement can be predicted by sampling the EMG activity of temporalis and masseter (Long, 2004). It is well accepted, however, that these muscles are perhaps not the primary agonists in lateral movements of the jaw (Huang et al., 2005, Miller, 1991). Based on the alignment of their muscle fibres and their contributing mediolateral vectors of pull, the contralateral inferior head of the lateral pterygoid and the contralateral medial pterygoid have been described as the primary agonists of lateral movements of the jaw (Huang et al., 2005, Miller, 1991, Murray et al., 1999, Widmalm et al., 1987).

The muscles tested in this study may play more of a supporting role for the primary agonists by maintaining the vertical position of the mandible during lateral movements. It has been suggested that during lateral movements of the jaw, the suprahyoid muscle complex, which includes the anterior belly of digastric, produces an opening vector in order to avoid the normal dental interference (Miller, 1991). This opening action of the suprahyoid muscles is counterbalanced by the masseter acting bilaterally during right lateral movements (Miller, 1991). In contrast the ipsilateral temporalis provides an additional lateral vector along with the pterygoids and a closing vector assisting the masseter while the contralateral temporalis is minimally active and usually not recruited (Figure 26, Miller, 1991). Having more of a supportive, counterbalancing role during lateral jaw movements would explain why the tested muscles demonstrated no change in either the total muscle activity or the maximum muscle activity following the application of resistance exercise against right lateral movement. Following exercise training it would

be expected that the majority of the changes in the EMG characteristics would occur in the primary agonists which would more likely be predominantly involved in producing the right lateral movement during the application of the resistance force.

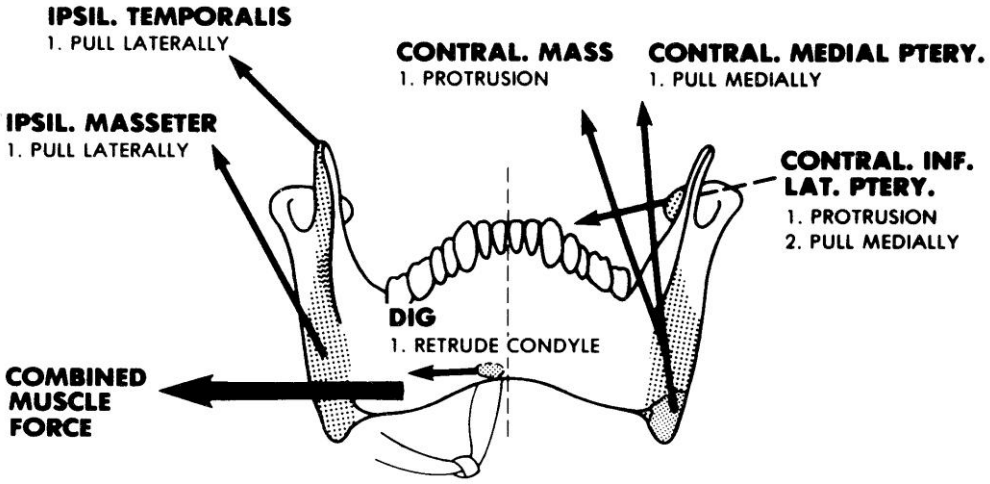

Figure 26: Force vectors of the muscles of mastication involved in producing a resultant right lateral movement of the mandible as depicted by the thickest vector line (Combined Muscle Force, Ipsil = Ipsilateral, Contral = Contralateral, Mass = Masseter, Ptery = Pterygoid, Inf = Inferior, Lat = Lateral, Dig = Digastric, Miller, 1991).

It is plausible that there may have been changes in the EMG activity of the primary agonists with only small changes in the muscles that assist in the movement as seen in the results of the current study. Analysing the EMG activity patterns of the primary agonists will help confirm these views. The tested muscles were chosen for practical clinical purposes as they are easily accessible for the reproducible placement of surface EMG electrodes. The lateral perygoid and the medial pterygoid are both technically difficult to access in order to measure their EMG activity, especially in the clinical setting. The procedures require intramuscular measurement and radiographic verification of the placement of the intramuscular EMG electrodes, which is currently predominantly limited

to research situations (Bhutada et al., 2007a, Bhutada et al., 2007b, Huang et al., 2005, Orfanos et al., 1996, Salame et al., 2007).

Temporal EMG Characteristics

The discussion of the changes in the Temporal EMG Characteristics has been divided into three sections: the Immediate Effects, the Training Effects, and the Longer-Term Effects. Each of these effects will be discussed separately.

Immediate Effects

The immediate effects are defined as the significant changes in the calculated variables that occurred following the initial application of isotonic resistance exercise during the Initial Training Session (ITS), that is between the pre-exercise data collection section of the ITS (ITS Pre) and the post-exercise data collection section of the ITS (ITS Post, Figure 9 and Table 2). These changes are described below followed by an interpretation of the results.

Time-to-Peak Muscle Activity (ETPk)

Right Digastric

Following the initial application of resistance exercise, the right anterior digastric of subjects in the Control Group took 1.73 sec longer on average than that of the subjects in the Exercise Group to reach its peak EMG activity (Figure 22c). Compared to pre-exercise the ETPk of the right anterior digastric of the Control Group increased by an average of 1.62 sec while the ETPk of the right anterior digastric of Exercise Group showed no significant change.

Duration of EMG Activity (ED)

Right anterior temporalis

Following the initial application of isotonic resistance exercise, subjects in both the Exercise Group and the Control Group showed a reduction in the ED of the right anterior temporalis by an average of 1.67 sec and 0.33 sec respectively (Figure 23a). Therefore in order to produce the same right lateral jaw movement the right anterior temporalis was required to be active for a shorter length of time following the application of isotonic resistance exercise against right lateral jaw movement.

Right Digastric

Following the initial application of isotonic resistance exercise, subjects in the Control Group showed an increase of 1.40 sec in the ED of the right digastric resulting in the right anterior digastric of subjects in the Control Group being active for 1.54 sec longer on average than those in the Exercise Group (Figure 23c). The increase in the ED of the right digastric in the Control Group returned to the pre-exercise level after four weeks of no exercise.

Time to Peak EMG Activity in Relation to Movement Onset (ETPm)

Right Digastric

Following the initial application of resistance exercise, the right anterior digastric of subjects in the Control Group took 1.88 sec longer on average than that of the subjects in the Exercise Group to reach its peak EMG activity following the onset of right lateral jaw movement (Figure 24d). Compared to pre-exercise the ETPm of the Control Group increased by an average of 0.39 sec and the Exercise Group increased by an average of 0.21 sec.

Relative Offset of Muscle Activity (RET)

Left Masseter

The application of isotonic resistance exercise produced significant changes in the RET of the left masseter immediately after the application of the resistance exercise (Figure 25). Initially, the EMG offset of the left masseter preceded the end of the return phase of the right lateral movement by an average of 2.14 sec in the Control Group and by an average of 1.35 sec in the Exercise Group. Application of isotonic resistance exercise resulted in the EMG offset of left masseter occurring after the completion of the return phase of the right lateral movement by an average of 0.23 sec in the Exercise Group. In the Control Group, which also undertook the initial exercise training, the EMG offset approached the movement offset but still preceded it by an average of 1.12 sec.

Interpretation of the Immediate Effects on the Temporal EMG Characteristics

A reduction in the duration of muscle activity during a movement task implies that other muscles must increase either their level of activity or the duration of their activity in order to complete the movement task. In musculoskeletal systems that have many muscles that contribute to joint movement, as in the case of the jaw, it is possible for other muscles with a vector of pull that contributes to the movement direction, to be recruited and increase their activity when necessary in order to fulfil the task requirements. This may explain the reduction in the ED of the ipsilateral anterior temporalis with a concomitant increase in the ipsilateral anterior digastric. Both these muscles have a vector of pull that contributes to ipsilateral jaw movement (Figure 26).

Recruitment of other muscles with a vector of pull capable of producing a lateral jaw movement may also explain the increase in the time taken for the ipsilateral anterior digastric to reach its peak EMG activity as seen by the increase in its ETPk and ETPm.

Since the ipsilateral anterior temporalis has reduced the duration of its EMG activity it is plausible that the ipsilateral anterior digastric has needed to increase its duration of activity later in the movement cycle as well as taking longer to reach its peak in order to produce the same lateral movement in the absence of EMG activity from the ipsilateral anterior temporalis. The ipsilateral anterior digastric may be compensating for the reduced activity of the ipsilateral anterior temporalis.

The immediate application of isotonic exercise also resulted in the RET of the contralateral masseter to become more positive. This means that the offset of the EMG acticvity approached the offset of the return phase of the movement. This may have been necessary in order for the contralateral masseter to assist the ipsilateral digastric as a result of the decrease in the duration of EMG activity in the ipsilateral anterior temporalis. It is also plausible that with the decrease in the duration of EMG activity in the ipsilateral anterior temporalis the contralateral masseter would have to be active later in the movement cycle in order to maintain the vertical position of the mandible during the lateral movement. It would counterbalance the increased activity of the ipsilateral anterior digastric later in the movement cycle. Future studies may elucidate this mechanism.

Similar relative EMG activity timing changes have been reported previously as a result of pain in the patellofemoral joint (Cowan et al., 2001) and the lumbar spine (Hodges and Richardson, 1996). Use of the experimental pain model in asymptomatic individuals has also resulted in EMG timing changes in the lumbar spine which persist after the resolution of that pain for up to one hour (Hodges et al., 2003). In the jaw, experimentally induced pain in the unilateral masseter of asymptomatic subjects has been reported to result in considerable variability between individuals in the effects on movement parameters (Sae-

Lee et al., 2008b) and the EMG activity of the masseter bilaterally and of the right posterior temporalis, anterior digastric, and inferior head of lateral pterygoid muscles (Sae-Lee et al., 2008a). The effects of isotonic resistance exercise on the variability of the tested EMG parameters is discussed in the Training Effects section below (see page 107).

The results of the current study are consistent with the view of previous biomechanical modelling studies which have determined that the human masticatory system has a level of redundancy (Lobbezoo et al., 2004). From a functional perspective a level of redundancy would serve a useful purpose in allowing the jaw to complete movement tasks regardless of which specific muscle was available to complete that task at any given time. Having varying numbers of muscles and varying motor control patterns available for the production of jaw movements allows for an element of flexibility in the production of jaw movements in order to fulfil the varying normal daily functional demands required of the human masticatory system.

Following the application of isotonic resistance exercise the ipsilateral digastric showed differences between the Exercise Group and the Control Group in three separate tested variables: ETPk, ETPm and ED. These differences remained after taking account of the initial differences between the two groups of subjects by use of an ANCOVA. All twelve subjects undertook the initial resistance exercise training prior to being randomly allocated to either the Exercise Group or the Control Group. Therefore any immediate effects of resistance exercise on the ETPk, ETPm or ED would be expected to have been similar across the two groups. The fact that there were differences between the two groups may indicate an intrinsic difference between the two groups that was not accounted for by the randomisation process. One explanation of this finding could be anatomical variations in

the morphology of the anterior digastric muscle (Aktekin et al., 2003, Celik et al., 2002, Liquidate et al., 2007). Given the varying types of anomalies reported in the anterior belly of digastric, it is plausible that individuals could have varying vectors of pull produced by their muscle. This functional difference between the anterior digastric muscles of individuals could lead to differences in the EMG activity detected by the surface electrodes. It is also important to note that the surface electrodes placed over the right anterior digastric could also detect the EMG activity of the other muscles in the floor of the mouth, including the mylohyoid, geniohyoid, the contralateral anterior digastric and possibly the muscles of the tongue. The amount of crosstalk detected by the surface EMG electrodes could also have been different between individuals and therefore could have contributed to the differences between the two groups in these variables. The randomisation process, however, should have balanced these potential anatomical and crosstalk differences across both groups.

Training Effects

The training effects are defined as the significant changes in the calculated variables that occurred following the four-week home-based isotonic resistance exercise programme, that is between ITS Pre and Testing Session 1 (TS1, Figure 9 and Table 2). These changes are described below followed by an interpretation of the results/findings.

Time-to-Peak Muscle Activity (ETPk)

Left Masseter

The application of isotonic resistance exercise produced no changes in the ETPk of left masseter after four weeks of a home-based exercise programme (Figure 22b). However, not continuing with a home-based exercise programme after the ITS resulted in an increase in the ETPk of the left masseter over the first four weeks following the ITS, which then

returned to pre-exercise levels after a further four weeks at TS2. Four weeks of isotonic resistance exercise appears to maintain the time taken for the left masseter to reach its peak EMG activity at pre-exercise levels which at TS1 are significantly lower than if no exercise was undertaken over the same period of time.

Right Digastric

The application of isotonic resistance exercise produced no changes in the ETPk of the right digastric after four weeks of a home-based exercise programme in the Exercise Group (Figure 22c). However, not continuing with a home-based exercise programme after the ITS resulted in a slight increase in the ETPk of the right anterior digastric over the first four weeks following the ITS from an average of 5.49 sec at ITS Pre to an average of 5.72 sec at TS1. This accounted for the Control Group taking an average of 1.94 sec longer than the Exercise Group to reach the peak EMG activity of the right anterior digastric at TS1 compared to taking an average of 1.73 sec to reach its peak EMG activity at ITS Pre. Four weeks of isotonic resistance exercise appears to maintain the time taken for the right digastric to reach its peak EMG activity at pre-exercise levels which at TS1 are significantly lower than if no exercise was undertaken over the same period of time.

Duration of EMG Activity (ED)

Right Anterior Temporalis

The application of isotonic resistance exercise produced no changes in the ED of right anterior temporalis after four weeks of a home-based exercise programme (Figure 23a). There were however differences within subjects across the four data collection sessions. Subjects in both the Exercise Group and the Control Group showed a reduction in the ED of right anterior temporalis over the eight weeks of the study. Therefore in order to

produce the same right lateral jaw movement between the testing sessions the right anterior temporalis was required to be active for a shorter length of time.

Time to Peak EMG Activity in Relation to Movement Onset (ETPm)

Right Anterior Temporalis

Four weeks of isotonic resistance exercise training resulted in the EMG activity of the right anterior temporalis reaching its peak 0.64 sec earlier on average following the onset of right lateral jaw movement Figure 24a). In contrast, not undertaking an isotonic resistance exercise programme after the initial application of resistance exercise resulted in an increase in the time taken for the right anterior temporalis to reach its peak EMG activity by an average of 1.47 sec after four weeks. This delay in the EMG activity of the right anterior temporalis to reach its peak in the Control Group returned to the pre-exercise length of time after a further four weeks.

Left Masseter

The application of isotonic resistance exercise produced no changes in the ETPm of the left masseter after four weeks of a home-based exercise programme (Figure 24c). In contrast, not continuing with a home-based exercise programme resulted in the left masseter taking an average of 3.9 sec longer to reach its peak EMG activity following the onset of right lateral jaw movement compared to the Exercise Group. This increase in the time taken for left masseter to reach its peak EMG activity returned to pre-exercise levels after a further four weeks of no exercise. Continuing with a home-based exercise programme for four weeks maintains the time to peak muscle activity following the movement onset at the pre-exercise level. Not continuing with a home-based exercise programme however leads to the left masseter reaching its peak EMG activity later following the movement onset.

Right Digastric

The application of isotonic resistance exercise produced no changes in the ETPm of the right digastric after four weeks of a home-based exercise programme (Figure 24d). However, not continuing with a home-based exercise programme after the ITS resulted in an increase in the ETPm of the right digastric by an average of 2.90 sec over the first four weeks following the ITS which then returns to pre-exercise levels after a further four weeks. Four weeks of isotonic resistance exercise appears to maintain the time taken for the right digastric to reach its peak EMG activity following the onset of right lateral jaw movement at pre-exercise levels which at TS1 are significantly lower than if no exercise was undertaken over the same period of time.

Relative Offset of Muscle Activity (RET)

Left Masseter

Four weeks of a home-based isotonic resistance exercise programme resulted in the EMG offset of the left masseter again preceding the end of the movement by an average of 0.91 sec which was similar to the average pre-exercise value of 1.35 sec (Figure 25). Over the same four weeks period the Control Group also showed a return of the RET such that the EMG offset of the left masseter preceded the movement offset by an average of 2.26 sec which was also similar to the pre-exercise value of an average of 2.14 sec.

Interpretation of the Training Effects on the Temporal EMG Characteristics

For exercise training to be effective it must be of a sufficiently long enough duration to elicit changes in the training muscle and/or the muscle's motor control system. Individualised rehabilitation programmes that have utilised exercise as one of their physiotherapy treatment modalities have been shown to be effective in the knee after six weeks (Crossley et al., 2005), the shoulder after four weeks (Ginn et al., 1997) and the

lumbar spine after four weeks (Hides et al., 1996). In the TMJ, isokinetic exercise has been shown to reduce clicking after a four-week home-based exercise programme (Au and Klineberg, 1993). In a recent systematic review it was found that a variety of exercise treatment durations, ranging between three weeks to six months, have been utilised in the treatment of TMD of a myofascial or muscular origin (For review see Medlicott and Harris, 2006). In the orofacial region plastic changes have also been shown to occur after 15 minutes of a novel tongue protrusion task (Boudreau et al., 2007).

In the current study the Initial Training Session (ITS) consisted of ten repetitions of the isotonic resistance exercise which took around 1.5 to 2 minutes to complete. This timeframe may be too short for any changes in the neuromuscular system to become evident or detectible with our equipment. The four-week home-based exercise programme, however, compares favourably with exercise regimens for other musculoskeletal subsystems and the TMJ. It is therefore plausible that the neuromuscular control system of the jaw is somehow different from that of other systems such as those that control the knee, shoulder or lumbar spine. The human masticatory system may require more training time to elicit clinically relevant effects. Investigation of the effect of varying dosages of exercise may be beneficial in determining an appropriate time frame for the application of exercise programmes in the relief of TMD.

The majority of the significant changes over the four weeks period during which the Exercise Group undertook their home-based exercise programme occurred in the Control Group. In contrast the Exercise Group tended to maintain the value of the majority of the tested variables at pre-exercise levels. This tends to suggest that there is a level of variability in the available motor control patterns in the human masticatory system for the

production of the same right lateral jaw movement under circumstances where individuals continue with their activities of daily living and do not undertake resistance exercise of the jaw. Furthermore, four weeks of isotonic resistance exercise tends to reduce this variability in the recruitment pattern of the muscles of mastication as seen by the maintenance of the values of the tested variables at pre-exercise levels.

These findings are also consistent with the view that training causes changes within the motor control system which may allow an individual to better coordinate the activation of muscle groups (Sale and MacDougall, 1981, cited in McConnell, 2002). The acquisition and maintenance of motor performance involves activity dependent plasticity at multiple sites throughout the central nervous system (Sale and MacDougall, 1981, cited in McConnell, 2002, Wolpaw and Tennissen, 2001). Furthermore, in the orofacial region, short bouts of training that result in improved performance in a novel tongue protrusion task are capable of producing rapid neuroplasticity of the primary motor cortex of the tongue (Boudreau et al., 2007). In the current study subjects completed four weeks of home-based exercise training resulting in a reduction in the variability of the tested EMG parameters. This reduced variability could infer an improvement in the performance of the right lateral movement task, either in terms of accuracy or efficiency of the motor control system. Task performance in terms of accuracy was controlled in this study by asking subjects to track a target bank of LEDs and evidenced by the fact there were no significant differences in the MSE of the movement trials within and between testing sessions (Figure 20). Performance in terms of improved efficiencies in the motor control system of the jaw was not measured in this study. The use of transcranial magnetic stimulation similar to that used during a novel tongue protrusion task (Boudreau et al., 2007) may help elucidate the mechanisms behind the reduced variability seen in the current study.

Experimentally induced pain in the tongue results in poorer performance in a novel tongue-protrusion task compared to pain-free trials of the same task (Boudreau et al., 2007). Furthermore, performance improvements over the fifteen minutes duration of the tongue-protrusion task are less during capsaicin induced pain trials compared to the pain-free trials (Boudreau et al., 2007). Experimentally induced pain in the unilateral masseter of asymptomatic subjects also results in considerable variability between individuals in the effects on movement parameters (Sae-Lee et al., 2008b) and the EMG activity of the masseter bilaterally and right posterior temporalis, anterior digastric, and inferior head of lateral pterygoid muscles (Sae-Lee et al., 2008a). In the current study there was a reduced variability in the recruitment pattern of some of the tested muscles of mastication following the application of isotonic resistance exercise. It is plausible therefore that since a four-weeks home-based exercise programme results in a reduction in the variability of some of the tested EMG variables this may lead to a reduction in pain in some individuals, especially in those whose pain is of a muscular origin. Future studies combining the experimental pain model, transcranial magnetic stimulation and intramuscular sampling of the EMG activity of the medial and lateral pterygoids may help determine the mechanisms behind these changes in motor recruitment variability.

Another reason for the variability seen in the EMG activity of the tested muscles could be the reported complex and varied anatomical architectures of the tested muscles (Aktekin et al., 2003, Brunel et al., 2003, Celik et al., 2002, Gaudy et al., 2000, Hannam and McMillan, 1994, Liquidate et al., 2007, Schumacher, 1961). The variations in the internal and external architectures of the tested muscles could result in different vectors of muscle pull during standardised mandibular movements between individuals. This in turn could

lead to different levels of EMG activity required to produce similar movements between our subjects which would explain the large variability demonstrated in our results.

Longer-Term Effects

The longer-term effects are defined as the significant changes in the calculated variables that occurred after a further four-weeks period following the completion of the four-weeks home-based isotonic resistance exercise programme, that is between TS1 and Testing Session 2 (TS2, Figure 9 and Table 2). These changes are described below followed by an interpretation of the results.

Time-to-Peak Muscle Activity (ETPk)

Left Anterior Temporalis

The application of isotonic resistance exercise produced no changes in the ETPk of left anterior temporalis either immediately after the application of the resistance exercise or after four weeks of a home-based exercise programme (Figure 22a). There was however a significant decrease in the ETPk of the left anterior temporalis in both the Control Group and the Exercise Group from TS1 to TS2. The reduction of the ETPk of the Control Group was from an average of 5.25 sec at TS1 to 3.57 sec at TS2 which was similar to the average pre-exercise level of 3.73 sec. Although not statistically significant the Control Group did show a slight increase in the ETPk of left anterior temporalis from ITS Pre to ITS Post and a further slight increase in ETPk after four weeks at TS1, (Figure 22a). This may have lead to the ETPk becoming high enough at TS1 for the reduction back to the pre-exercise level at TS2 to become statistically significant. In the Exercise Group however, the reduction in the ETPk at TS2 was below that at ITS Pre with the left lateral anterior temporalis reaching its peak muscle activity 2.08 sec earlier on average than that at TS1 and an average of 1.95 sec earlier than during ITS Pre.

Duration of EMG Activity (ED)

Right Anterior Temporalis

Subjects in both the Exercise Group and the Control Group showed a reduction in the ED of the right anterior temporalis over the eight weeks of the study (Figure 23a). The ED of the right anterior temporalis in the Control Group fell by an average of 0.43 sec between TS1 to TS2 and by an average of 1.30 sec between TS1 and TS2 in the Exercise Group. Therefore in order to produce the same right lateral jaw movement between the testing sessions the right anterior temporalis was required to be active for even shorter lengths of time than those seen at the previous testing sessions.

Left Anterior Temporalis

There was a significant decrease in the ED of the left anterior temporalis in both the Control Group and Exercise Group from TS1 to TS2 (Figure 23b). The Exercise Group also showed a significantly greater reduction in the ED of the left anterior temporalis compared to the Control Group over the four weeks period after completion of the home-based exercise programme. Therefore in order to produce the same right lateral jaw movement between the testing sessions the left anterior temporalis was required to be active for a shorter length of time.

Time to Peak EMG Activity in Relation to Movement Onset (ETPm)

Left Anterior Temporalis

Four weeks following the completion of a four-week home-based exercise programme, there was a greater reduction in the ETPm of the left anterior temporalis in the Exercise Group compared to not exercising for the same period of time (Figure 24b). A reduction in the ETPm indicates that it takes less time for the muscle to reach its peak EMG activity

after commencement of the right lateral jaw movement. In other words the muscle reaches its peak activity earlier in the movement cycle. This may be a result of the reduced time of the duration of EMG activity in the left anterior temporalis which has produced a shift in the time at which the muscle reaches its peak activity following the movement onset.

Relative Offset of Muscle Activity (RET)

Left Masseter

The RET of the left masseter in both the Control Group and the Exercise Group became more negative at TS2 compared to the values at TS1 (Figure 25). In the Control Group the relative offset of the EMG activity of the left masseter preceded the movement offset at TS2 by an average of 0.72 sec more than that at TS1 and by 0.84 sec more than that at ITS Pre. In the Exercise Group however the change in the RET was not as great following the cessation of the home exercise programme. In the Exercise Group, the relative offset of the EMG activity preceded the movement offset at TS2 by an average of 0.12 sec more than that at TS1 and by 0.32 sec more than that at ITS Pre.

Interpretation of the Longer-Term Effects on the Temporal EMG Characteristics

Changes continued to occur in four variables across three muscles during the final four-week testing period of this study. As mentioned earlier the ED of the ipsilateral anterior temporalis continued to fall over the final testing period. The contralateral anterior temporalis also recorded a reduction in its duration of EMG activity during this period. The shorter duration of EMG activity in the ipsilateral and contralateral anterior temporalis might indicate that other muscles of mastication might be taking on a greater role in producing the movement task. No other tested muscles recorded an increase in the duration of their EMG activity. This implies that there might have been a change in the duration of EMG activity in muscles that were not tested, such as the medial and lateral

pterygoids. The medial and lateral pterygoids have been reported as being primary agonists to contralateral movement of the mandible (Huang et al., 2005, Miller, 1991, Murray et al., 1999, Widmalm et al., 1987). Future studies should investigate the role of the primary agonists in order to determine their contribution to the movement task following the application of resistance exercise.

Interestingly the contralateral anterior temporalis showed changes for the first time at the end of the eight-weeks study period, recording a lowering of its duration of EMG activity as well as its peak EMG activity occurring earlier in the movement cycle in relation to both the EMG activity onset as well as the movement onset. These two latter findings may have occurred as a result of the decrease in the duration of the EMG activity. If the muscle is active for a shorter period of time it stands to reason that its peak activity would occur earlier in the movement cycle, given its EMG onset remained unchanged. This might also indicate an increased contribution from other muscles that were not tested in this study in order for the same movement task to be completed.

The contralateral anterior temporalis of the Exercise Group demonstrated changes in the ETPk and ETPm. The ETPk fell below pre-exercise levels and the ETPm demonstrated a greater reduction compared to that in the Control Group. This finding could demonstrate the ability of resistance exercise to maintain the ETPk and the ETPm of the contralateral anterior temporalis at pre-exercise levels. Removal of the exercise may have resulted in an increase in the variability of these EMG parameters as seen in the Control Group. The implication of the differences in the variability between the Control Group and the Exercise Group as a result of undertaking a home-based exercise programme has been discussed in the Training Effects section above (see page 107). To determine the clinical

relevance of this finding it would be necessary to test this result in patients with TMD or in subjects with experimentally induced pain to determine if this time delay occurs in symptomatic individuals.

The EMG offset of the contralateral masseter preceded the offset of the return phase of the right lateral movement in both the Control Group and the Exercise Group with the Control group preceding the movement offset by more than it did during the pre-exercise data collection session. In contrast, the relative offset of the EMG activity of the contralateral masseter in the Exercise Group, preceded the movement offset by a similar amount to that at the completion of the exercise programme and also by a similar amount to that of the pre-exercise value. In both the Control Group and the Exercise Group the relative offset of the EMG activity of the contralateral masseter moved closer to the movement offset following the immediate application of isotonic resistance exercise, with the relative offset of the EMG activity of the Exercise Group occurring 0.23 sec after the movement offset. The RET then returned to near pre-exercise levels in both groups after the Exercise Group completed the four-week home-based exercise programme. However, subjects who undertook a four-week home-based isotonic resistance exercise program appeared to be able to better maintain the contralateral masseter RET values closer to the pre-exercise values after cessation of the training programme. Subjects who didn't undertake a four-weeks home-based isotonic resistance exercise program appeared to show greater variability in the RET of the left masseter in the four weeks following the cessation of the home-based exercise programme. The implication of the differences in the variability between the Control Group and the Exercise Group as a result of undertaking a home-based exercise programme has been discussed in the Training Effects section above (see page 107).

In three of the tested variables there were changes in both the Exercise Group and the Control Group, namely the ED of the ipsilateral anterior temporalis, which occurred across all the testing sessions, as well as the ETPk of the contralateral anterior temporalis and the RET of the contralateral masseter. This finding could be the result of a time effect whereby changes in these variables occurred in these muscles as a result of subjects being a part of the data collection process. However, this did not occur in all of the parameters or muscles that were tested. Therefore it may be that these muscles respond differently from the other tested muscles in relation to these variables following the application of the isotonic resistance exercise during lateral jaw movement. Furthermore, the ipsilateral anterior temporalis demonstrated these continued changes after the initial application of the isotonic resistance exercise. This may suggest that the application of isotonic resistance exercise has more longer lasting effects on the temporal EMG characteristics of the ipsilateral anterior temporalis with no added benefit being gained from continuing a home-based exercise programme for a further four weeks. Further investigation of these variables may determine whether the changes reported in this study were due to a time effect or a training effect.

Study Limitations

This study has shown that isotonic resistance exercise can cause changes in some EMG parameters of the tested muscles of mastication in asymptomatic individuals. These changes have occurred in the immediate term, straight after the application of isotonic resistance exercise, and after four weeks of a home-based isotonic resistance exercise programme, and even after four weeks following the completion of the home-based exercise programme. However, the application of isotonic resistance exercise did not result in changes in all of the EMG parameters, nor did it result in changes in all of the

tested muscles. Several methodological aspects of this study could have contributed to this result. It could be that the EMG activity during the free right lateral movement task was small in the tested muscles. It is also plausible that the EMG activity was recorded from muscles that were not directly involved in the generation of the primary force vector for right lateral movement. Also, the training regimen may have been too short to elicit any changes in either the muscle itself or its motor control centre. These methodological aspects have already been discussed in the relevant sections above in relation to the immediate effects, training effects or the longer-term effects of the isotonic resistance exercise.

Clinical Relevance

This study has demonstrated that isotonic resistance exercise can cause changes in the EMG activity of the muscles of mastication in asymptomatic individuals. Some of these changes occurred after the immediate application of the resistance exercise, some occurred after four weeks of training, while some occurred four weeks after the cessation of the training period. Therefore it is possible to retrain the human masticatory system in such a way as to change the EMG activity of some of the muscles of mastication. In some cases isotonic resistance exercise has resulted in less inherent variability in some of the measures of the EMG activity that were analysed. It has been argued that this reduction in variability of the tested EMG parameters is an indication of the utilisation of a more stable motor recruitment pattern in the production of the movement task. This may imply that the application of isotonic resistance exercise resulted in an improvement in the motor performance of the human masticatory system when performing controlled movement tasks. Determining the mechanisms by which these changes occur will help facilitate the development and the appropriate application of rehabilitation programmes in order to

reduce the impact of TMD and return symptomatic individuals to more normal jaw function. If such rehabilitation programmes can be developed then clinicians will have access to a simple, safe and relatively inexpensive treatment modality that can be prescribed to improve the functional outcomes of people with TMD of a muscular origin.

Future Directions

Future studies could help elucidate the mechanisms underlying the changes demonstrated in the current study. Initially it would be prudent to investigate the effects of isotonic resistance exercise on different movements of the jaw. The current study has only reported the data from one movement direction following the application of the exercise regimen, namely right, or ipsilateral, lateral movement. Data from four movement directions were collected during the three testing sessions. Analysing the data from contralateral (left) lateral movement as well as open/close and protrusion would demonstrate any changes to the EMG activity of the tested muscles during these jaw movements. This would help to determine the EMG responses of these muscles to the application of isotonic resistance exercise. If these responses prove to be reproducible then this will help facilitate the development of appropriate rehabilitation programmes for patients with TMD of a muscular origin.

It may also be worthwhile to investigate the effects of exercise on the EMG activity of the muscles of mastication during movement in combination with a simulated bruxing task. This would increase the EMG activity of the jaw closing muscles, including the temporalis and masseter bilaterally. This in turn might make it easier to discern changes in the EMG activity following the application of resistance exercise. It will also illustrate the changes

produced by resistance exercise during a simulated bruxing task and thereby may help develop treatment strategies to reduce the effects of bruxism on the TMJ and the dentition.

Future studies should also investigate the role of the primary agonists of lateral jaw movements in order to determine their contribution to the movement task following the application of resistance exercise. Combining the experimental pain model, transcranial magnetic stimulation and intramuscular sampling of the EMG activity of the medial and lateral pterygoids will facilitate this goal as well as helping to determine the mechanisms behind the changes in motor recruitment variability seen in the current study.

CONCLUSION

Temporomandibular disorders (TMD) are the most prevalent of the orofacial conditions in adults. The most prevalent subgroup of TMD are those with a musculoskeletal origin and are largely idiopathic in nature. Therapeutic exercise is a long established treatment modality of musculoskeletal conditions. In particular, specific individualised exercise programmes have been shown to reduce pain and improve function in the knee, shoulder and lumbar spine and also the temporomandibular joint (TMJ). There is however a paucity of literature that has investigated the mechanisms by which exercise may affect the neuromuscular systems that control jaw movement and function. This study has shown that isotonic resistance exercise can cause changes in EMG activity of the muscles of mastication in asymptomatic individuals. Some of these changes occurred after the immediate application of the resistance exercise, some occurred after four weeks of training, while some occurred four weeks after the cessation of the training period. Determining the mechanisms by which these changes occur will help facilitate the development and the appropriate application of rehabilitation programmes in order to reduce the impact of TMD and return symptomatic individuals to normal jaw function.

REFERENCES

ADKINS, D. L., BOYCHUK, J., REMPLE, M. S. & KLEIM, J. A. (2006) Motor training induces experience-specific patterns of plasticity across motor cortex and spinal cord. *Journal of Applied Physiology,* 101**,** 1776-82.

AKTEKIN, M., KURTOGLU, Z. & OZTURK, A. H. (2003) A bilateral and symmetrical variation of the anterior belly of the digastric muscle. *Acta Medica Okayama,* 57**,** 205-7.

AU, A. R. & KLINEBERG, I. J. (1993) Isokinetic exercise management of temporomandibular joint clicking in young adults. *Journal of Prosthetic Dentistry,* 70**,** 33-9.

BASMAJIAN, J. V. & LUCA, C. J. D. (1985) *Muscles alive: their functions revealed by electromyography,* Baltimore, Md., Williams & Wilkins.

BAWA, P. (2002) Neural control of motor output: can training change it? *Exercise & Sport Sciences Reviews,* 30**,** 59-63.

BERTOLUCCI, L. E. (1992a) Physical therapy post-arthroscopic TMJ management (update). *Cranio,* 10**,** 130-7.

BERTOLUCCI, L. E. (1992b) Postoperative physical therapy in temporomandibular joint arthroplasty. *Cranio,* 10**,** 211-20.

BERTOLUCCI, L. E., URIELL, P. & SWAFFER, C. (1989) Postoperative physical therapy in temporomandibular joint arthroplasty. *Cranio,* 7**,** 214-22.

BHUTADA, M. K., PHANACHET, I., WHITTLE, T., PECK, C. C. & MURRAY, G. M. (2007a) Activity of superior head of human lateral pterygoid increases with increases in contralateral and protrusive jaw displacement. *European Journal of Oral Sciences,* 115**,** 257-64.

BHUTADA, M. K., PHANACHET, I., WHITTLE, T., WANIGARATNE, K., PECK, C. C. & MURRAY, G. M. (2007b) Threshold properties of single motor units in superior head of human lateral pterygoid muscle. *Archives of Oral Biology,* 52**,** 552-61.

BLASBERG, B. & GREENBERG, M. S. (2003) Temporomandibular Disorders (Ch10). IN GREENBERG, M. S. & GLICK, M. (Eds.) *Burket's oral medicine: diagnosis and treatment.* 10th ed. Hamilton, Ont., BC Decker.

BOUDREAU, S., ROMANIELLO, A., WANG, K., SVENSSON, P., SESSLE, B. J. & ARENDT-NIELSEN, L. (2007) The effects of intra-oral pain on motor cortex neuroplasticity associated with short-term novel tongue-protrusion training in humans.[see comment]. *Pain,* 132**,** 169-78.

BRUNEL, G., EL-HADDIOUI, A., BRAVETTI, P., ZOUAOUI, A. & GAUDY, J. F. (2003) General organization of the human intra-masseteric aponeuroses: changes with ageing. *Surgical & Radiologic Anatomy,* 25**,** 270-283.

CELIK, H. H., ALDUR, M. M., OZDEMIR, B. & AKSIT, M. D. (2002) Abnormal digastric muscle with unilateral quadrification of the anterior belly. *Clinical Anatomy,* 15**,** 32-4.

CHRISTO, J. E., BENNETT, S., WILKINSON, T. M. & TOWNSEND, G. C. (2005) Discal attachments of the human temporomandibular joint. *Australian Dental Journal,* 50**,** 152-60.

COWAN, S. M., BENNELL, K. L., CROSSLEY, K. M., HODGES, P. W. & MCCONNELL, J. (2002) Physical therapy alters recruitment of the vasti in patellofemoral pain syndrome. *Medicine & Science in Sports & Exercise,* 34**,** 1879-85.

COWAN, S. M., BENNELL, K. L., HODGES, P. W., CROSSLEY, K. M. & MCCONNELL, J. (2001) Delayed onset of electromyographic activity of vastus medialis obliquus relative to vastus lateralis in subjects with patellofemoral pain syndrome. *Archives of Physical Medicine & Rehabilitation,* 82**,** 183-9.

CROSSLEY, K. M., COWAN, S. M., MCCONNELL, J. & BENNELL, K. L. (2005) Physical therapy improves knee flexion during stair ambulation in patellofemoral pain. *Medicine & Science in Sports & Exercise,* 37**,** 176-83.

CUNNINGHAM, D. J. & ROMANES, G. J. (1986) *Cunningham's manual of practical anatomy,* Oxford ; New York, Oxford University Press.

DAHLSTROM, L. (1992) Conservative treatment methods in craniomandibular disorder. *Swedish Dental Journal,* 16**,** 217-30.

DALEN, K., ELLERTSEN, B., ESPELID, I. & GRONNINGSAETER, A. G. (1986) EMG feedback in the treatment of myofascial pain dysfunction syndrome. *Acta Odontologica Scandinavica,* 44**,** 279-84.

DOHRMANN, R. J. & LASKIN, D. M. (1978) An evaluation of electromyographic biofeedback in the treatment of myofascial pain-dysfunction syndrome. *Journal of the American Dental Association,* 96**,** 656-62.

DOUCETTE, S. A. & CHILD, D. D. (1996) The effect of open and closed chain exercise and knee joint position on patellar tracking in lateral patellar compression syndrome. *Journal of Orthopaedic & Sports Physical Therapy,* 23**,** 104-10.

DRAKE, J. D. M. & CALLAGHAN, J. P. (2006) Elimination of electrocardiogram contamination from electromyogram signals: An evaluation of currently used removal techniques. *Journal of Electromyography & Kinesiology,* 16**,** 175-87.

DRANGSHOLT, M. & LERESCHE, L. (1999) Temporomandibular disorder pain. IN CROMBIE, I., CROFT, P., LINTON, S., LERESCHE, L. & KORFF, M. V. (Eds.) *Epidemiology of Pain.* Seattle, IASP Press.

DWORKIN, S. F. & LERESCHE, L. (1992) Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *Journal of Craniomandibular Disorders,* 6**,** 301-55.

DWORKIN, S. F. & MASSOTH, D. L. (1994) Temporomandibular disorders and chronic pain: disease or illness? *Journal of Prosthetic Dentistry,* 72**,** 29-38.

FEINE, J. S. & LUND, J. P. (1997) An assessment of the efficacy of physical therapy and physical modalities for the control of chronic musculoskeletal pain. *Pain,* 71**,** 5-23.

FRIDLUND, A. J. & CACIOPPO, J. T. (1986) Guidelines for human electromyographic research. *Psychophysiology,* 23**,** 567-89.

GAUDY, J. F., ZOUAOUI, A., BRAVETTI, P., CHARRIER, J. L. & GUETTAF, A. (2000) Functional organization of the human masseter muscle. *Surgical & Radiologic Anatomy,* 22**,** 181-190.

GINN, K. A. & COHEN, M. L. (2004) Conservative treatment for shoulder pain: prognostic indicators of outcome. *Archives of Physical Medicine & Rehabilitation,* 85**,** 1231-5.

GINN, K. A. & COHEN, M. L. (2005) Exercise therapy for shoulder pain aimed at restoring neuromuscular control: a randomized comparative clinical trial. *Journal of Rehabilitation Medicine,* 37**,** 115-22.

GINN, K. A., HERBERT, R. D., KHOUW, W. & LEE, R. (1997) A randomized, controlled clinical trial of a treatment for shoulder pain. *Physical Therapy,* 77**,** 802-9; discussion 810-1.

GLASS, E. G., GLAROS, A. G. & MCGLYNN, F. D. (1993) Myofascial pain dysfunction: treatments used by ADA members. *Cranio,* 11**,** 25-9.

HANNAM, A. G. & MCMILLAN, A. S. (1994) Internal organization in the human jaw muscles. *Critical Reviews in Oral Biology & Medicine,* 5**,** 55-89.

HIDES, J. A., STOKES, M. J. P., SAIDE, M., JULL, G. A. & COOPER, D. H. (1994) Evidence of Lumbar Multifidus Muscle Wasting Ipsilateral to Symptoms in Patients with Acute/Subacute Low Back Pain. *Spine,* 19(2)**,** 165-172.

HIDES, J. A. P., RICHARDSON, C. A. P. & JULL, G. A. M. (1996) Multifidus Muscle Recovery Is Not Automatic After Resolution of Acute, First-Episode Low Back Pain. *Spine,* 21**,** 2763-2769.

HODGES, P. W., MOSELEY, G. L., GABRIELSSON, A. & GANDEVIA, S. C. (2003) Experimental muscle pain changes feedforward postural responses of the trunk muscles. *Experimental Brain Research,* 151**,** 262-71.

HODGES, P. W. & RICHARDSON, C. A. (1996) Inefficient Muscular Stabilization of the Lumbar Spine Associated With Low Back Pain: A Motor Control Evaluation of Transversus Abdominis. *Spine,* 21**,** 2640-2650.

HUANG, B. Y., WHITTLE, T. & MURRAY, G. M. (2005) Activity of inferior head of human lateral pterygoid muscle during standardized lateral jaw movements. *Archives of Oral Biology,* 50**,** 49-64.

JOHNSTON, T. B., DAVIES, D. V., GRAY, H. & DAVIES, F. (Eds.) (1958) *Gray's anatomy, descriptive and applied,* London, Longmans, Green & Co.

KATSOULIS, J. & RICHTER, M. (2008) [Efficacy of specific physiotherapy for temporomandibular joint dysfunction of muscular origin]. *Revue de Stomatologie et de Chirurgie Maxillo-Faciale,* 109**,** 9-14.

KILBREATH, S. L., GANDEVIA, S. C., WIRIANSKI, A. & HEWITT, B. (1995) Human flexor pollicis longus: role of peripheral inputs in weight-matching. *Neuroscience Letters,* 201**,** 203-6.

LIQUIDATE, B. M., BARROS, M. D., ALVES, A. L. & PEREIRA, C. S. B. (2007) Anatomical Study of the Digastric Muscle: Variations in the Anterior Belly. *International Journal of Morphology,* 25**,** 797-800.

LOBBEZOO, F., DRANGSHOLT, M., PECK, C., SATO, H., KOPP, S. & SVENSSON, P. (2004) Topical review: new insights into the pathology and diagnosis of disorders of the temporomandibular joint. *Journal of Orofacial Pain,* 18**,** 181-91.

LONG, C. L. (2004) Pattern recognition using surface electromyography of the anterior temporalis and masseter muscles. *Department of Oral Biology, The Faculty of Graduate Studies.* Vancouver, BC, Canada, The University of British Columbia.

MANNS, A. E., GARCIA, C., MIRALLES, R., BULL, R. & ROCABADO, M. (1991) Blocking of periodontal afferents with anesthesia and its influence on elevator EMG activity. *Cranio,* 9**,** 212-9.

MCCONNELL, J. (2002) The physical therapist's approach to patellofemoral disorders. *Clinics in Sports Medicine,* 21**,** 363-87.

MCMILLAN, A. S. & HANNAM, A. G. (1992) Task-related behavior of motor units in different regions of the human masseter muscle. *Archives of Oral Biology,* 37**,** 849-57.

MEDLICOTT, M. S. & HARRIS, S. R. (2006) A systematic review of the effectiveness of exercise, manual therapy, electrotherapy, relaxation training, and biofeedback in the management of temporomandibular disorder.[see comment]. *Physical Therapy,* 86**,** 955-73.

MESQUI, F. & PALLA, S. (1985) Real-time non-invasive recording and display of functional jaw movements. *Journal of Oral Rehabilitation,* 12**,** 541-542.

METROPLY AG (1990) JAWS 3D User's Manual. 1.00 ed. Zurich, Switzerland, Metroply AG.

MICHELOTTI, A., DE WIJER, A., STEENKS, M. & FARELLA, M. (2005) Home-exercise regimes for the management of non-specific temporomandibular disorders. *Journal of Oral Rehabilitation,* 32**,** 779-85.

MILLER, A. J. (1991) *Craniomandibular muscles: their role in function and form,* Boca Raton, CRC Press.

MUNRO, R. R. (1972) Coordination of activity of the two bellies of the digastric muscle in basic jaw movements. *Journal of Dental Research,* 51**,** 1663-7.

MUNRO, R. R. (1974) Activity of the digastric muscle in swallowing and chewing. *Journal of Dental Research,* 53**,** 530-7.

MURRAY, G. M., BHUTADA, M., PECK, C. C., PHANACHET, I., SAE-LEE, D. & WHITTLE, T. (2007) The human lateral pterygoid muscle. *Archives of Oral Biology,* 52**,** 377-80.

MURRAY, G. M., ORFANOS, T., CHAN, J. Y., WANIGARATNE, K. & KLINEBERG, I. J. (1999) Electromyographic activity of the human lateral pterygoid muscle during contralateral and protrusive jaw movements. *Archives of Oral Biology,* 44**,** 269-85.

MURRAY, G. M., PHANACHET, I., UCHIDA, S. & WHITTLE, T. (2004) The human lateral pterygoid muscle: a review of some experimental aspects and possible clinical relevance. *Australian Dental Journal,* 49**,** 2-8.

NICOLAKIS, P., ERDOGMUS, B., KOPF, A., EBENBICHLER, G., KOLLMITZER, J., PIEHSLINGER, E. & FIALKA-MOSER, V. (2001) Effectiveness of exercise therapy in patients with internal derangement of the temporomandibular joint. *Journal of Oral Rehabilitation,* 28**,** 1158-64.

NICOLAKIS, P., ERDOGMUS, B., KOPF, A., NICOLAKIS, M., PIEHSLINGER, E. & FIALKA-MOSER, V. (2002) Effectiveness of exercise therapy in patients with myofascial pain dysfunction syndrome. *Journal of Oral Rehabilitation,* 29**,** 362-8.

O'RAHILLY, R. (1986) *Gardner-Gray-O'Rahilly Anatomy: A regional study of human structure,* Philadelphia, Saunders.

OGAWA, T., KAWATA, T., TSUBOI, A., HATTORI, Y., WATANABE, M. & SASAKI, K. (2006) Functional properties and regional differences of human masseter motor units related to three-dimensional bite force. *Journal of Oral Rehabilitation,* 33**,** 729-40.

OH, D. W., KIM, K. S. & LEE, G. W. (2002) The effect of physiotherapy on post-temporomandibular joint surgery patients. *Journal of Oral Rehabilitation,* 29**,** 441-6.

ORFANOS, T., SARINNAPHAKORN, L., MURRAY, G. M. & KLINEBERG, I. J. (1996) Placement and verification of recording electrodes in the superior head of the human lateral pterygoid muscle. *Archives of Oral Biology,* 41**,** 493-503.

QUINN, J. H. (1995) Mandibular exercises to control bruxism and deviation problems. *Cranio,* 13**,** 30-4.

RANTANEN, J., HURME, M., FALCK, B., ALARANTA, H., NYKVIST, F., LEHTO, M., EINOLA, S. & KALIMO, H. (1993) The lumbar multifidus muscle five years after surgery for a lumbar intervertebral disc herniation. *Spine,* 18**,** 568-574.

ROCABADO, M. (1989) Physical therapy for the postsurgical TMJ patient. *Journal of Craniomandibular Disorders,* 3**,** 75-82.

ROCABADO, M. & IGLARSH, Z. A. (1991) *Musculoskeletal approach to maxillofacial pain,* Philadelphia, Lippincott.

ROHEN, J. W. & YOKOCHI, C. (1983) *Color atlas of anatomy : a photographic study of the human body,* New York, Igaku-Shoin.

SAE-LEE, D., WHITTLE, T., FORTE, A. R. C., PECK, C. C., BYTH, K., SESSLE, B. J. & MURRAY, G. M. (2008a) Effects of experimental pain on jaw muscle activity during goal-directed jaw movements in humans. *Experimental Brain Research,* 189**,** 451-62.

SAE-LEE, D., WHITTLE, T., PECK, C. C., FORTE, A. R. C., KLINEBERG, I. J. & MURRAY, G. M. (2008b) Experimental jaw-muscle pain has a differential effect on different jaw movement tasks. *Journal of Orofacial Pain,* 22**,** 15-29.

SAHA, A. K. (1971) Dynamic stability of the glenohumeral joint. *Acta Orthopaedica Scandinavica,* 42**,** 491-505.

SALAME, T. H., PECK, C. C. & MURRAY, G. M. (2007) A new method for lateral pterygoid electromyographic electrode placement. *Journal of Prosthetic Dentistry,* 98**,** 224-31.

SANES, J. N. & DONOGHUE, J. P. (2000) Plasticity and primary motor cortex. *Annual Review of Neuroscience,* 23**,** 393-415.

SCHENKMAN, M. & CARTAYA, V. R. D. (1987) Kinesiology of the Shoulder Complex. *The Journal of Orthopaedic and Sports Physical Therapy,* 8**,** 438-450.

SCHUMACHER, G. H. (1961) *Funktionelle Morphologie der Kaumuskulatur,* Jena, Veb Gustav Fischer Verlag.

SIHVONEN, T., HERNO, A., PALJARVI, L., AIRAKSINEN, O., PARTANEN, J. & TAPANINAHO, A. (1993) Local Denervation Atrophy of Paraspinal Muscles in Postoperative Failed Back Syndrome. *Spine,* 18**,** 575-581.

STEGENGA, B., DE BONT, L. G., DE LEEUW, R. & BOERING, G. (1993) Assessment of mandibular function impairment associated with temporomandibular joint osteoarthrosis and internal derangement. *Journal of Orofacial Pain,* 7**,** 183-95.

STOHLER, C. S. & ZARB, G. A. (1999) On the management of temporomandibular disorders: a plea for a low-tech, high-prudence therapeutic approach. *Journal of Orofacial Pain,* 13**,** 255-61.

SUVINEN, T. I., READE, P. C., KEMPPAINEN, P., KONONEN, M. & DWORKIN, S. F. (2005) Review of aetiological concepts of temporomandibular pain disorders: towards a biopsychosocial model for integration of physical disorder factors with psychological and psychosocial illness impact factors. *European Journal of Pain,* 9**,** 613-33.

TAYLOR, M., SUVINEN, T. & READE, P. (1994) The effect of Grade IV distraction mobilisation on patients with temporomandibular pain-dysfunction disorder. *Physiotherapy Theory and Practice: An International Journal of Physiotherapy,* 10**,** 129 - 136.

THE MATHWORKS INC (2000) MATLAB Script Signal Processing Toolbox. IN AGILENT TECHNOLOGIES INC (Ed.) *VEE Pro 6.0.* USA, The Mathworks Inc.

VAN EIJDEN, T. M., BRUGMAN, P., WEIJS, W. A. & OOSTING, J. (1990) Coactivation of jaw muscles: recruitment order and level as a function of bite force direction and magnitude. *Journal of Biomechanics,* 23**,** 475-85.

VAN EIJDEN, T. M. & RAADSHEER, M. C. (1992) Heterogeneity of fiber and sarcomere length in the human masseter muscle. *Anatomical Record,* 232**,** 78-84.

WIDMALM, S. E., LILLIE, J. H. & ASH, M. M., JR. (1987) Anatomical and electromyographic studies of the lateral pterygoid muscle. *Journal of Oral Rehabilitation,* 14**,** 429-46.

WOLPAW, J. R. & TENNISSEN, A. M. (2001) Activity-dependent spinal cord plasticity in health and disease. *Annual Review of Neuroscience,* 24**,** 807-43.

Appendix 1: Data Processing Flowchart of the VEE Pro 6 Program

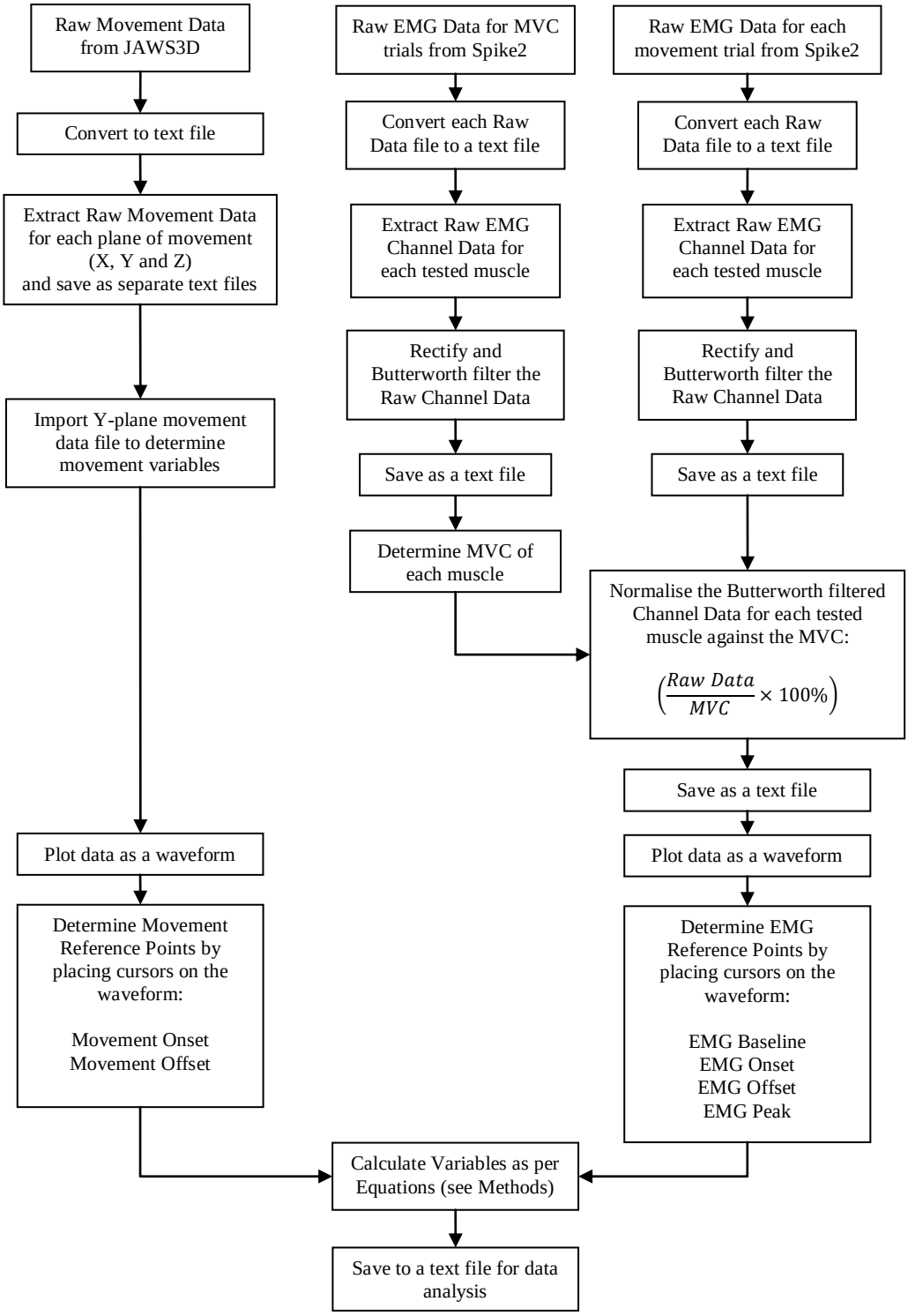

Figure 27: Flowchart depicting the flow of data through the developed Agilent VEE Pro 6 program. Raw data is acquired from the sampling hardware and software, relevant data is extracted and stored as text files for further processing. Variables are then calculated and stored for analysis.

Appendix 2: Graphical Representation of the Changes in the Electromyographic (EMG) Variables

The figures on the following pages show a graphical representation of the results for each tested variable. The results for each tested muscle is represented separately under each of the variable headings. Where the data was not normally distributed non-parametric tests were used for the analysis and the results depict the median (+/- SE) as described in the caption. Where the data was normally distributed analysis of variance (ANOVA) was used for the analysis and the results depict the mean (+/- SD) as described in the caption.

Area Under the Curve (AUC)

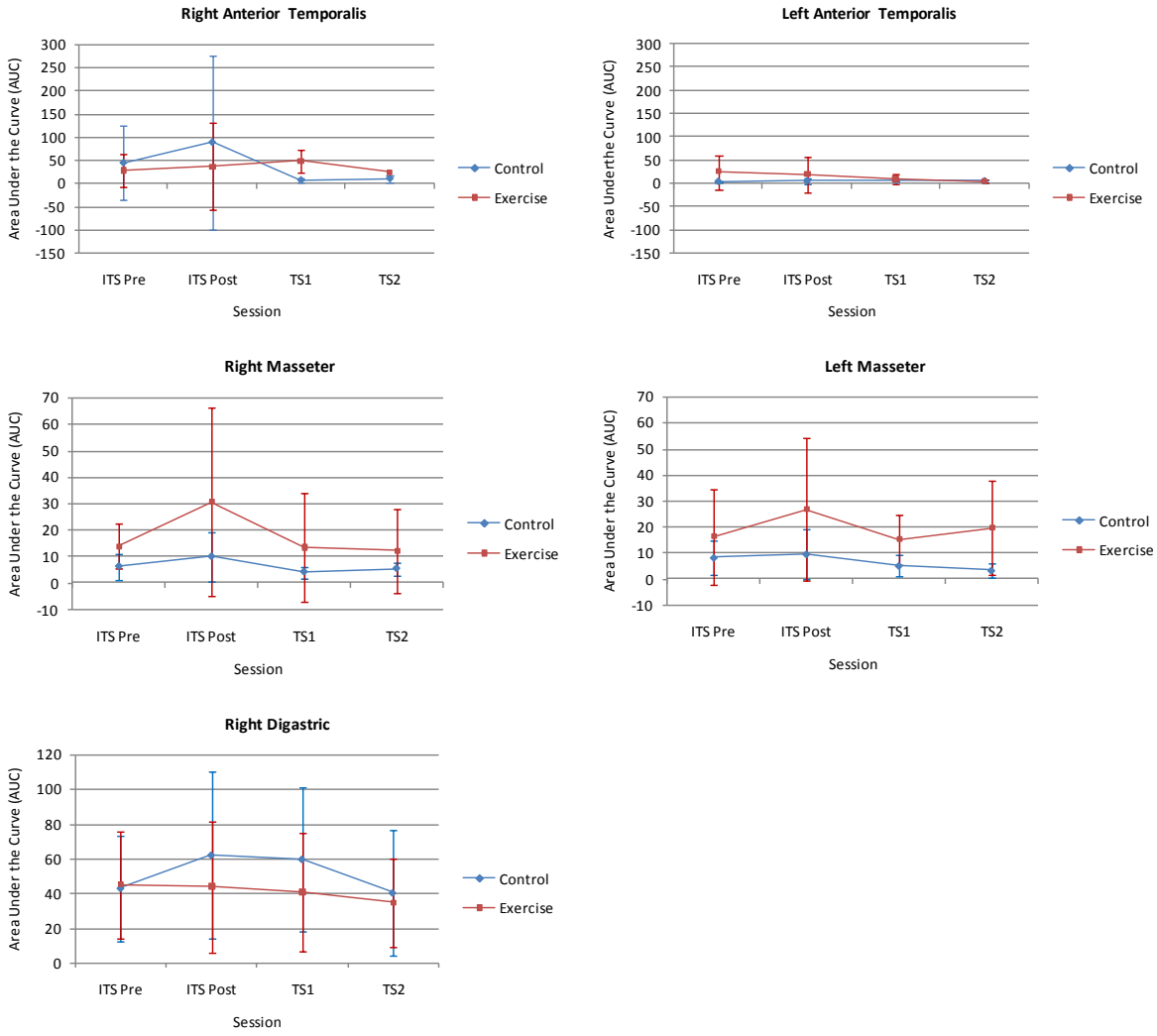

Figure 28: Mean (+/- SD) values of the area under the curve (AUC) of the normalised electromyographic (EMG) data of all the tested muscles (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Peak EMG Activity (EPk)

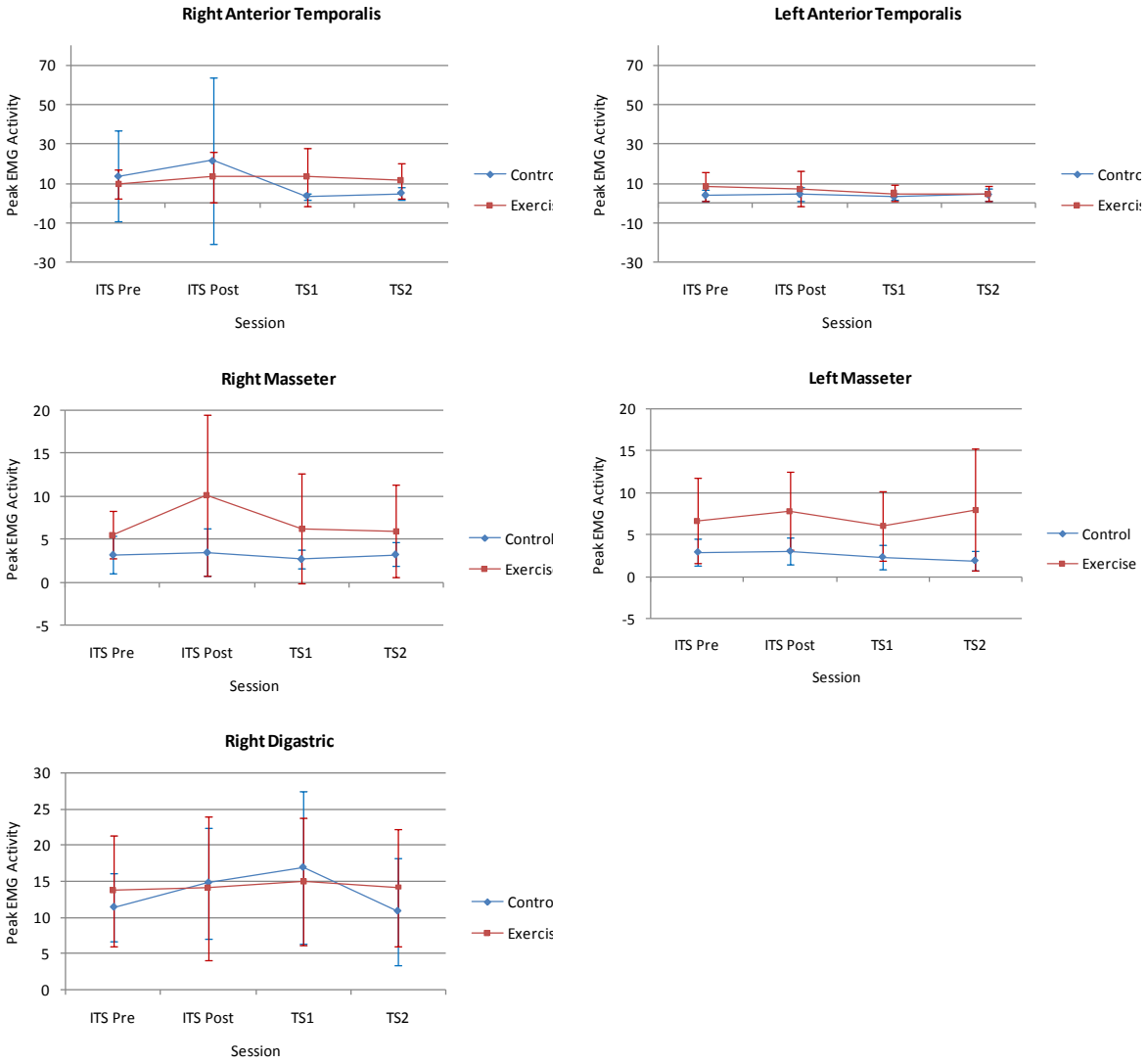

Figure 29: Mean (+/- SD) values of the peak electromyographic (EMG) activity (EPk) of all the tested muscles (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Time to Peak EMG Activity (ETPk)

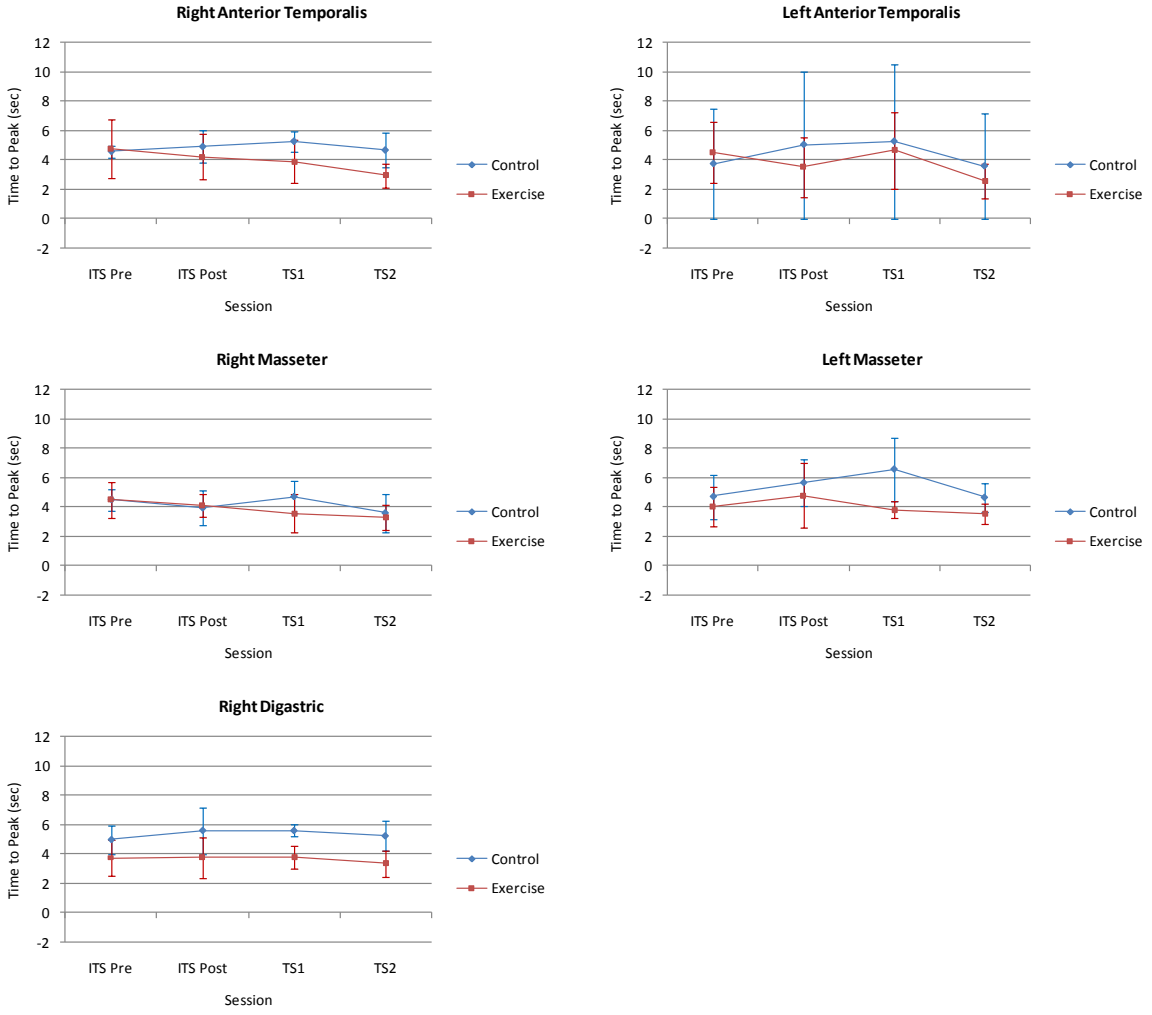

Figure 30: Mean (+/- SD) values of the time to peak muscle activity (ETPk) of all the tested muscles (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Duration of EMG Activity (ED)

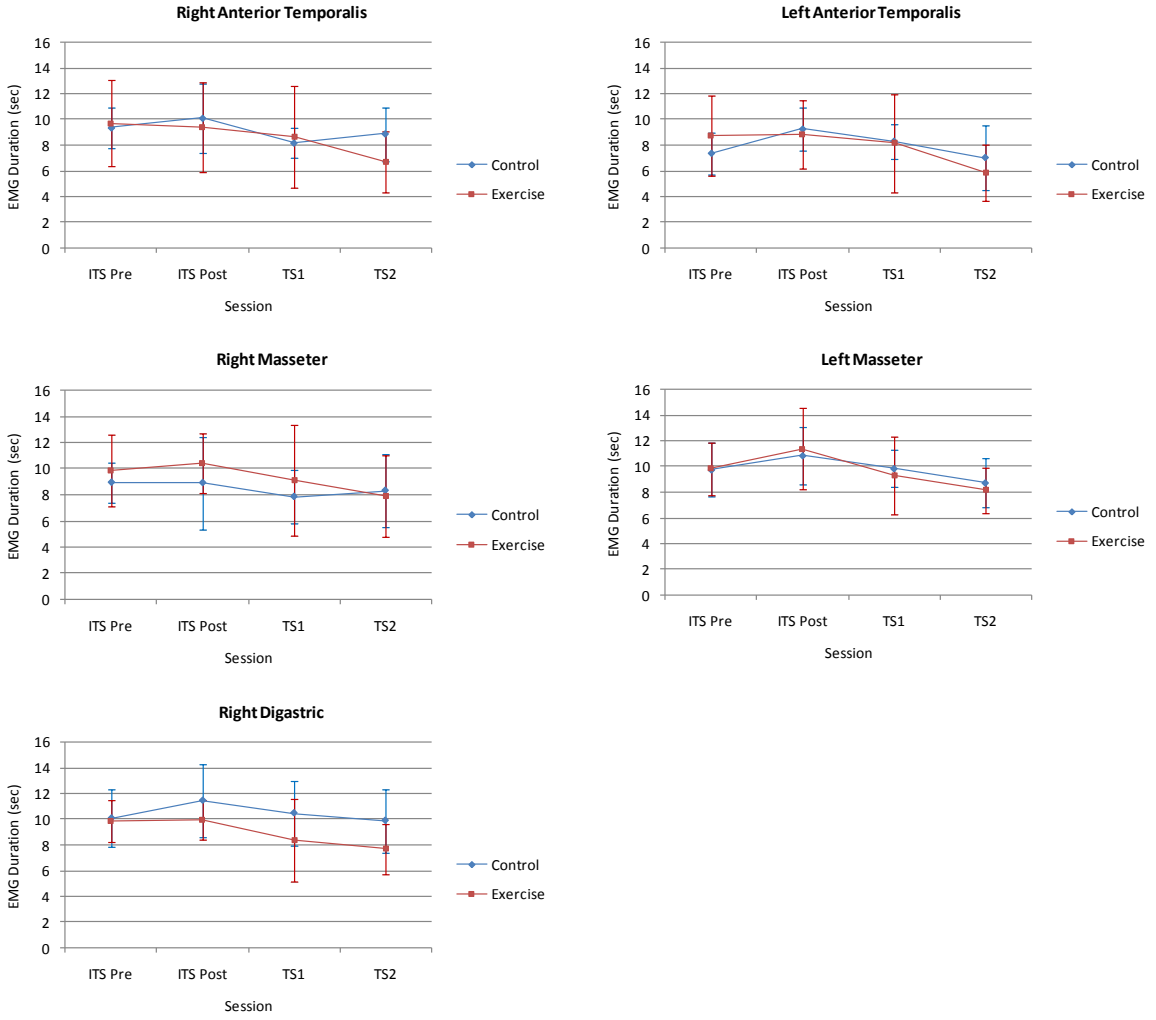

Figure 31: Values of the duration of EMG activity (ED) of all the tested muscles. The mean ED (+/- SD) is depicted for the left anterior temporalis, right masseter, left masseter and right anterior digastric muscles. The median ED (+/- SE) is depicted for the right anterior temporalis muscle (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Time to Peak EMG Activity in Relation to Movement Onset (ETPm)

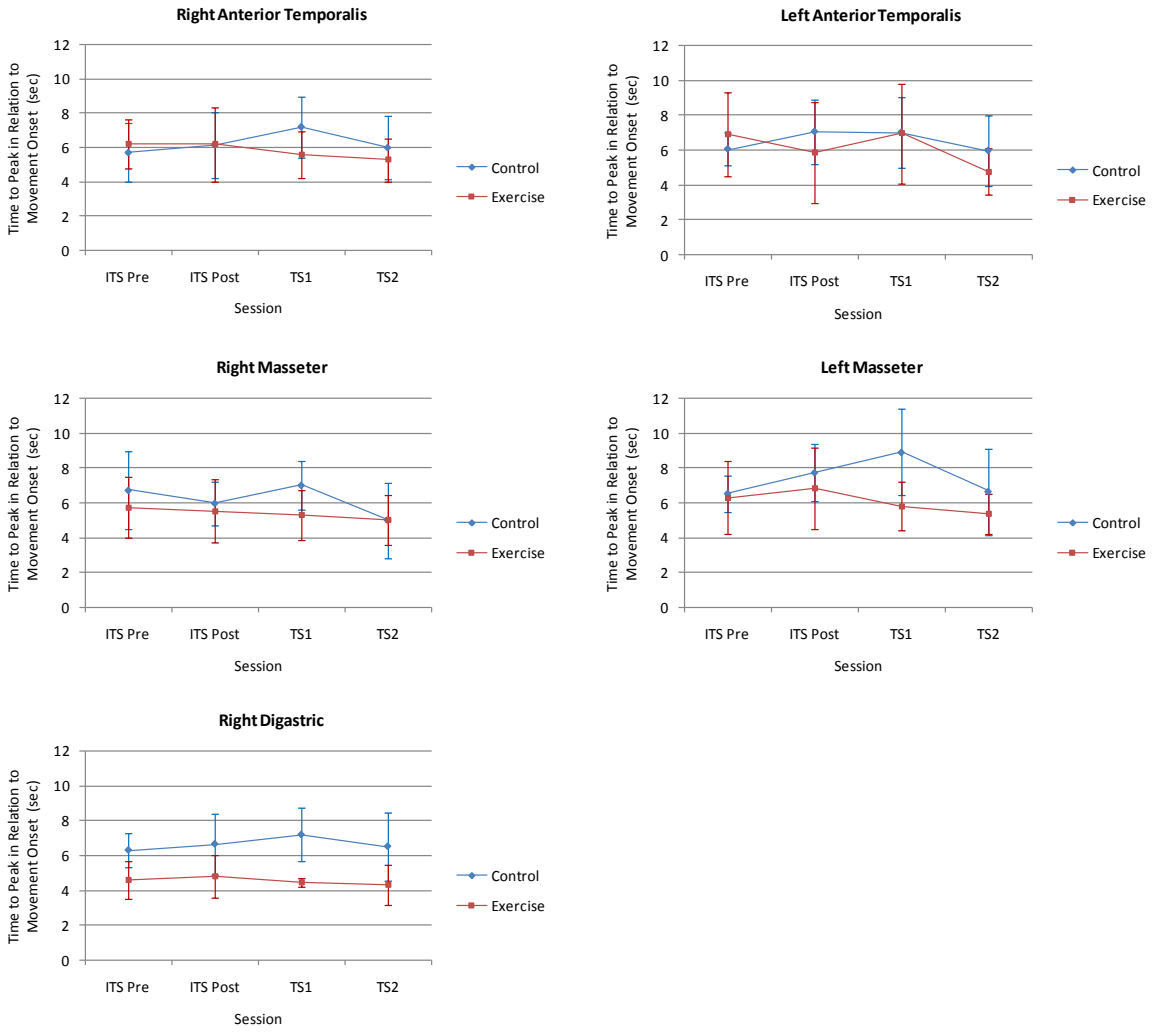

Figure 32: Values of the time to peak EMG activity in relation to movement onset (ETPm) of all the tested muscles. The mean ETPm (+/- SD) is depicted for the right anterior temporalis, left anterior temporalis and right masseter muscles. The median ETPm (+/- SE) is depicted for the left masseter and right anterior digastric muscles (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Relative Onset of EMG Activity (ROT)

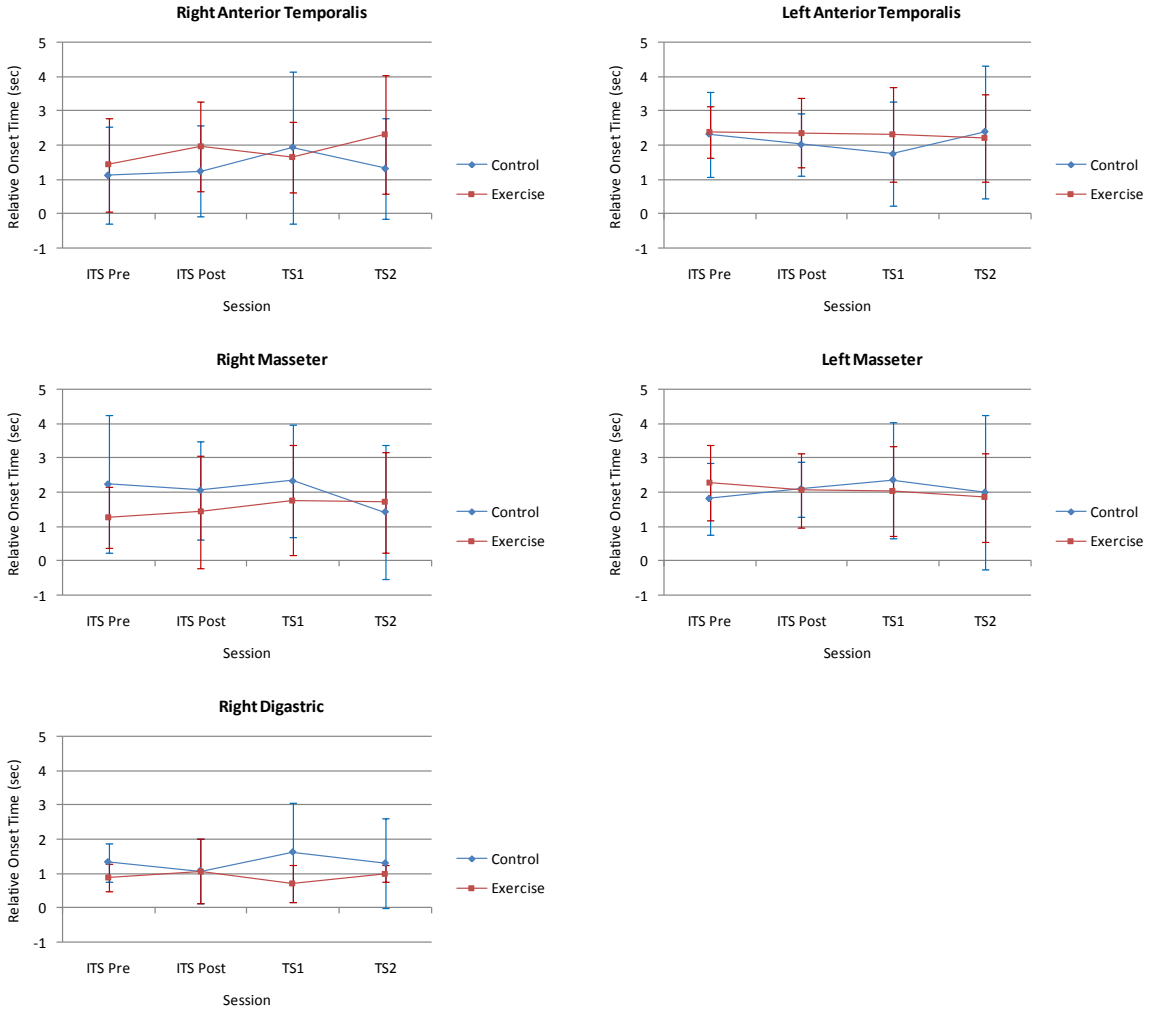

Figure 33: Mean (+/- SD) values of the relative onset of electromyographic (EMG) activity in relation to the onset of right lateral jaw movement (ROT) of all the tested muscles. Negative values indicate the onset of the EMG activity preceded the movement onset (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Relative Offset of EMG Activity (RET)

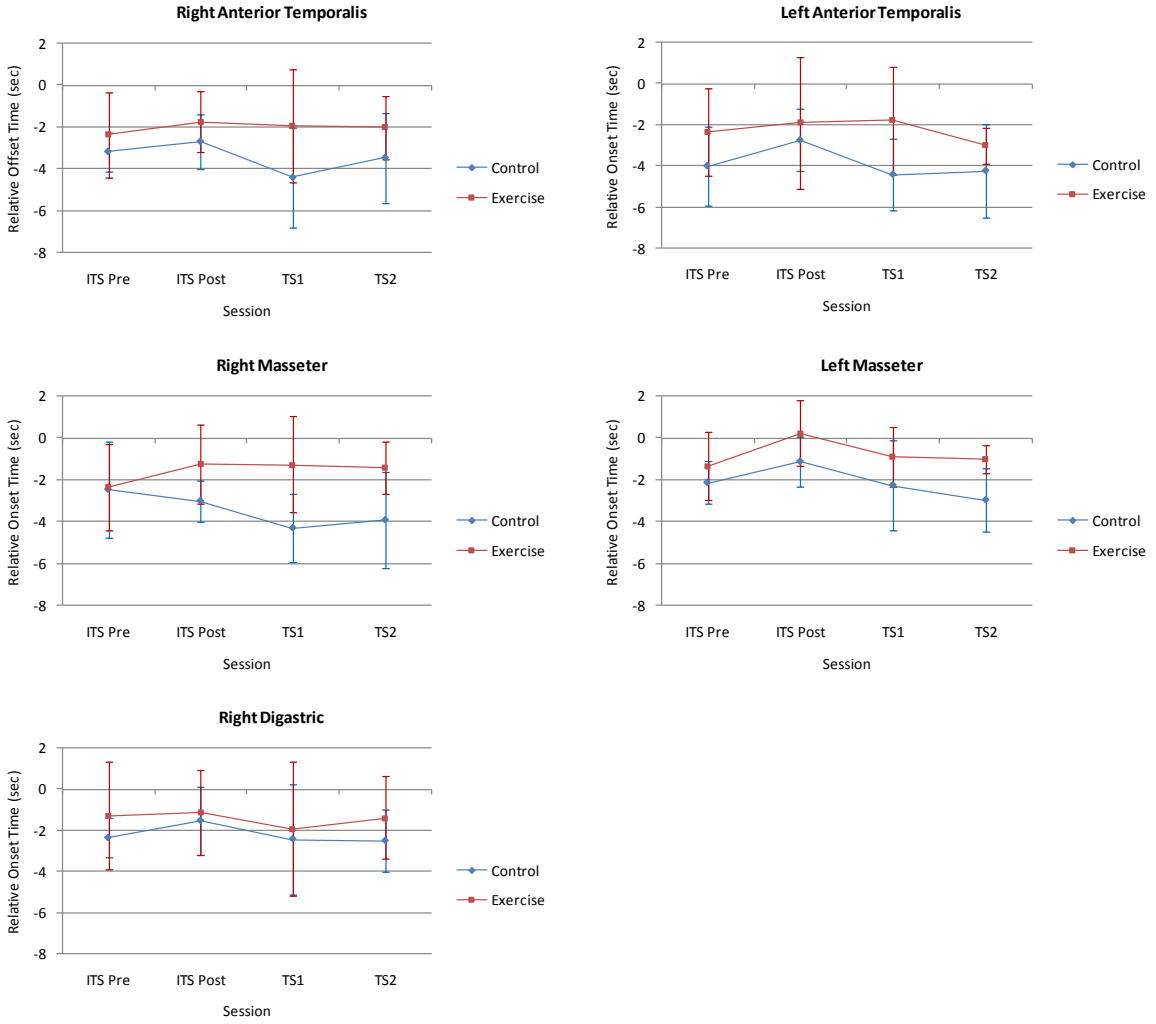

Figure 34: Mean (+/- SD) values of the relative offset time of electromyographic (EMG) activity in relation to the offset of right lateral jaw movement (RET) of all the tested muscles. Negative values indicate the offset of the EMG activity preceded the movement offset. (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2).

Appendix 3: Mean Peak EMG Values

Table 6: The table on the following page shows the Mean Peak EMG activity of each of the tested muscles as a percentage of maximum voluntary contraction (%MVC) for each experimental group (Control or Exercise) during each data collection session. The Total Mean Peak EMG activity of each of the tested muscles as a percentage of maximum voluntary contraction (%MVC) for each data collection session is also shown (ITS Pre = Initial Training Session, Pre-Exercise Training; ITS Post = Initial Training Session, Post-Exercise Training; TS1 = Testing Session 1; TS2 = Testing Session 2; SD = standard deviation; n = sample size).

Muscle	Testing Session	Group	Mean Peak (%MVC)	SD	n
Right Anterior Temporalis	ITS Pre	Control	13.60	23.04	5
		Exercise	9.41	7.29	6
		Total	**11.31**	**15.61**	**11**
	ITS Post	Control	21.68	42.29	5
		Exercise	13.10	12.83	6
		Total	**17.00**	**28.60**	**11**
	TS1	Control	3.37	1.72	5
		Exercise	13.38	14.70	6
		Total	**8.83**	**11.69**	**11**
	TS2	Control	4.91	2.96	5
		Exercise	11.33	8.83	6
		Total	**8.41**	**7.33**	**11**
Left Anterior Temporalis	ITS Pre	Control	4.01	2.96	5
		Exercise	8.49	7.36	6
		Total	**6.45**	**6.00**	**11**
	ITS Post	Control	4.53	3.57	5
		Exercise	7.37	9.02	6
		Total	**6.08**	**6.92**	**11**
	TS1	Control	3.29	1.62	5
		Exercise	5.07	4.00	6
		Total	**4.26**	**3.15**	**11**
	TS2	Control	4.33	3.36	5
		Exercise	4.80	3.68	6
		Total	**4.59**	**3.67**	**11**
Right Masseter	ITS Pre	Control	3.18	2.15	5
		Exercise	5.54	2.74	6
		Total	**4.47**	**2.67**	**11**
	ITS Post	Control	3.45	2.76	5
		Exercise	10.16	9.38	6
		Total	**7.11**	**7.70**	**11**
	TS1	Control	2.72	1.04	5
		Exercise	6.23	6.38	6
		Total	**4.63**	**4.91**	**11**
	TS2	Control	3.23	1.38	5
		Exercise	5.97	5.40	6
		Total	**4.73**	**4.17**	**11**
Left Masseter	ITS Pre	Control	2.93	1.59	5
		Exercise	6.69	5.05	6
		Total	**4.98**	**4.20**	**11**
	ITS Post	Control	3.04	1.64	5
		Exercise	7.82	4.65	6
		Total	**5.65**	**4.26**	**11**
	TS1	Control	2.34	1.45	5
		Exercise	6.03	4.18	6
		Total	**4.35**	**3.65**	**11**
	TS2	Control	1.90	1.11	5
		Exercise	7.99	7.30	6
		Total	**5.22**	**6.11**	**11**
Digastric	ITS Pre	Control	11.40	4.87	5
		Exercise	13.74	7.73	5
		Total	12.57	**6.14**	**10**
	ITS Post	Control	14.79	7.70	5
		Exercise	14.08	9.91	5
		Total	**14.44**	**8.37**	**10**
	TS1	Control	16.93	10.66	5
		Exercise	15.08	8.82	5
		Total	**16.01**	**9.27**	**10**
	TS2	Control	10.85	7.39	5
		Exercise	14.21	8.11	5
		Total	**12.53**	**7.53**	**10**