Specific Resistance Exercise Can Alter Masticatory Movement and Muscle Recruitment Patterns of the Human Jaw.

Alexander Wirianski
BSc(UNSW) BAppSc(Physio)(Hons)(Syd) MPhil(Dent)(Syd)

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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 Doctor of Philosophy 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 Professor Chris Peck and Professor Greg Murray. The research work that forms part of this PhD was completed at two collaborating institutions in Japan. The study described in Chapter 2 was completed at the Division of Aging & Geriatric Dentistry, Graduate School of Dentistry, Tohoku University, in Sendai, Japan, under the supervision of Professor Yoshinori Hattori. The study described in Chapter 3 was completed at Removable Partial Prosthodontics, Masticatory Function Rehabilitation, Graduate School, Tokyo Medical and Dental University, in Tokyo, Japan, under the supervision of Assistant Professor Ichiro Minami. I verify that this thesis has not been submitted, wholly or in part, for any other 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
PhD Candidate
11 April 2016
Dedication

On 11 March 2011 at 14:46 Japan Standard Time the Pacific Coast of Japan was struck by the 2011 Great East Japan Earthquake. Registering magnitude 9.0 on the Richter scale and a maximum 7 on the Japan Meteorological Agency scale of seismic intensity, it was the largest earthquake in Japan’s recorded history. The devastating tsunami that followed as a result essentially wiped out the majority of the east coast of the island of Honshu. 15,880 people lost their lives and 2,694 are still officially reported as missing, while a further 6,135 suffered injuries. In total, over 315,000 people were evacuated from their homes. The ensuing nuclear accident at the Fukushima Daiichi Nuclear Power Plant also required that approximately 154,000 people evacuate from the surrounding restricted areas. Despite the massive relief and reconstruction efforts nearly 230,000 people are still unable to return home.

This PhD is dedicated to the memory of those that lost their lives in the 2011 Great East Japan Earthquake and the tsunami that followed, as well as their families and all those who assisted in the massive relief efforts.

Ganbarou Sendai, ganbarou Tohoku, ganbarou Japan.

http://www.reconstruction.go.jp/english/topics/2013/03/about-us-situation-overview.html
In Memorium

In loving memory of those who passed away during the course of this project...

Callie ~ 8 November 2010

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Abstract

Temporomandibular disorders (TMD) are musculoskeletal conditions that affect the jaws and the muscles of mastication and their associated structures. Affecting up to 18% of the adult population, TMD are the most prevalent orofacial pain condition of non-dental origin and the second most common musculoskeletal condition that results in pain and disability after chronic low back pain. Limitations and/or deviations of mandibular movements are common signs and symptoms of TMD which impact on an individual’s ability to chew. Mastication is a complex, intermittent, semiautomatic, rhythmical movement of the jaws that is produced and controlled by the masticatory central pattern generator located in the brainstem and modulated by inputs and feedback from structures in both the central and peripheral nervous systems.

Therapeutic exercise is a common treatment modality in the management of many musculoskeletal conditions, including TMD, and has been shown to be associated with improvements in symptoms and function. In other joints, such as the knee, shoulder, cervical spine and lumbar spine, painful musculoskeletal conditions are often associated with biomechanical deficiencies of altered patterns of muscle activation which are thought to be the result of alterations in motor control at these joints. The prescription of therapeutic exercise addressing the biomechanical deficiencies has resulted in the restoration of normal electromyographic (EMG) activity patterns and kinematics in these joints. In particular, in recurrent low back pain patients the restoration of normal EMG activity patterns were associated with cortical reorganisation of the motor cortical representations of transversus abdominis that closely resembled those found in healthy, asymptomatic individuals.
Recent systematic reviews have reported that therapeutic exercise may be of benefit in the
management of TMD and one of their actions may be to target and produce changes in the
motor control of the muscles of mastication. This thesis presents two randomised
controlled studies which individually investigated the effects of an isometric resistance
exercise task applied to the mandible on masticatory movement patterns and the EMG
activation patterns of the muscles of mastication during chewing.

The first study tested the hypothesis that exercise modifies mandibular masticatory
movement paths. Fourteen asymptomatic volunteer participants were randomly allocated to
either a Control group (n = 7) or an Exercise group (n = 7). Jaw movement data were
collected from each participant during five trials of chewing cooked rice until swallowing
before and after an “exercise condition”. During the “exercise condition” participants in
the Exercise group completed an isometric resistance exercise task at 30% of their
maximum voluntary contraction (MVC) against right lateral jaw movements for 15
minutes while participants in the Control group sat quietly for 15 minutes and did not
complete the exercise task. Principal component analysis (PCA) on a variance-covariance
matrix revealed that five principal components (PCs) of a total of 123 PCs explained 92.1%
of the total variance in the chewing cycles of all participants. Furthermore, in
asymptomatic individuals, isometric resistance exercise applied against a lateral jaw
movement resulted in masticatory movement paths that were significantly (p < 0.001)
more horizontally orientated in the coronal plane and more protruded in the sagittal plane.

The second study tested the hypothesis that isometric resistance exercise can change the
patterns of EMG activation of the muscles of mastication during free chewing. Surface
EMG activity was recorded from the anterior temporalis and masseter muscles bilaterally
during chewing in 20 asymptomatic volunteer adults. Participants were randomly allocated to either a Control group (n = 10) or an Exercise group (n = 10). Jaw movement and EMG activity were simultaneously collected from each participant during five trials of chewing a single pellet of gum: (i) at baseline, before the exercise task; (ii) immediately after the exercise task (isometric resistance at between 20% to 30% MVC against right lateral jaw movements); (iii) after two weeks of a home-based exercise programme; and (iv) at four-weeks follow-up. Performance of the exercise task resulted in significantly (p < 0.05) earlier onset of the EMG activity in the ipsilateral anterior temporalis and the ipsilateral masseter during the masticatory cycle.

Similar patterns of earlier EMG activation have been demonstrated in the knee, shoulder, cervical spine and in patients with recurrent low back pain after completion of specific therapeutic exercise regimens. The earlier onset of transversus abdominis in recurrent low back pain patients was also associated with reorganisation of the motor cortex representation of transversus abdominis following the completion of two weeks of a specific exercise programme. Together, the results from previous studies along with those of the two studies presented in this thesis suggest that changes in masticatory jaw movement trajectories and the temporal changes in EMG activation patterns of the muscles of mastication during chewing may reflect changes in the central motor control of mastication brought about by the application of the exercise task.

Future studies are warranted to investigate the presence of delayed muscle activation patterns along with any associated cortical changes in patients with TMD who also present with deviations in masticatory movement paths. These studies will provide further insight into the mechanism(s) associated with the motor control of the masticatory system and
how these mechanisms may be influenced by the application of therapeutic exercise regimens. This will provide clinicians with valuable information for the prescription of appropriate and effective exercise programmes as part of their management of patients with TMD.
## Contents

DECLARATION ........................................................................................................... ii  
DEDICATION ........................................................................................................ iii  
IN MEMORIUM ........................................................................................................ iv  
ACKNOWLEDGMENTS ................................................................................................. v  

ABSTRACT ................................................................................................................ xi  

CONTENTS .................................................................................................................. xv  
LIST OF FIGURES ........................................................................................................ xviii  
LIST OF TABLES .......................................................................................................... xxii  
ABBREVIATIONS ......................................................................................................... xxiii  
CONFERENCES AND PRESENTATIONS ....................................................................... xxv  
PUBLICATIONS ............................................................................................................ xxvi  

CHAPTER 1 .................................................................................................................... 1  
LITERATURE REVIEW ................................................................................................. 1  

|--------------|-----------------------------------------------|------------------------|-----------------------------|----------------------------------------------------------|-----------------------------------------------|------------------------|----------------------|-----------------|-----------------|------------------|------------------|-------------|-----------------------------|---------------|-----------------------------|-----------------------------------------------|------------------------|-------------------------|------------------|------------------|--------------------------|
CHAPTER 2 ................................................................................................................................. 57
ISOMETRIC RESISTANCE JAW EXERCISE ALTERS JAW MOVEMENT
PATTERNS DURING CHEWING................................................................................................. 58
  ABSTRACT ............................................................................................................................... 58
  INTRODUCTION ....................................................................................................................... 59
  METHODS ................................................................................................................................. 62
    Participants ............................................................................................................................. 62
    Jaw movement ....................................................................................................................... 62
    Maximum voluntary contraction (MVC) ................................................................................. 65
    Isometric resistance exercise training task ........................................................................... 66
    Data Analysis ........................................................................................................................ 67
  RESULTS .................................................................................................................................. 69
    Chewing Trials ....................................................................................................................... 69
    Principal Component Analysis (PCA) .................................................................................... 71
    Reconstruction of Jaw Displacements ............................................................................... 72
    Multivariate ANOVA of the Principal Components ............................................................ 78
    Univariate Analyses .............................................................................................................. 78
  DISCUSSION .......................................................................................................................... 84
  CONCLUSION ......................................................................................................................... 92

CHAPTER 3 .................................................................................................................................. 93
ISOMETRIC RESISTANCE JAW EXERCISE ALTERS
ELECTROMYOGRAPHIC ACTIVITY IN THE JAW MUSCLES DURING
MASTICATION. .............................................................................................................................. 94
  ABSTRACT ............................................................................................................................... 94
  INTRODUCTION ....................................................................................................................... 96
  METHODS ................................................................................................................................. 100
    Participants ............................................................................................................................. 100
    Maximum Voluntary Contraction (MVC) .............................................................................. 103
    Jaw movement ....................................................................................................................... 106
    Electromyographic (EMG) Data .......................................................................................... 110
    Synchronisation of Jaw Movement and EMG Data ............................................................ 110
    Isometric resistance exercise training task ........................................................................... 110
    Data Processing .................................................................................................................... 112
    Statistical Analyses .............................................................................................................. 125
  RESULTS .................................................................................................................................. 126
    Participants ............................................................................................................................. 126
    Preferred Chewing Side ......................................................................................................... 126
    Initial Exercise Time and Time Between Data Collection Sessions ..................................... 127
    Participants’ Adherence to the Home Exercise Programme ............................................... 128
    Tested Variables ..................................................................................................................... 129
  DISCUSSION .......................................................................................................................... 182
    Adherence with the Home Exercise Programme .................................................................. 182
    Jaw Movement Changes ..................................................................................................... 184
    Electromyographic Changes ............................................................................................... 187
    Patterns of Jaw Movement and EMG Changes ................................................................. 192
    Jaw Motor Control ................................................................................................................. 193
    Strengths of the study ............................................................................................................ 196
    Limitations ............................................................................................................................. 197
    Future Studies ....................................................................................................................... 199
  CONCLUSION ........................................................................................................................ 200
List of Figures

Figure 1.1: The muscles of mastication showing their direction of pull and their subsequent action on the mandible to produce jaw movements .......................... 26

Figure 1.2: Patterns of mandibular movement during mastication as shown in the coronal plane .............................................................................................................. 39

Figure 1.3: A classification of five masticatory muscle activity patterns in mammals based on the function of the muscles of mastication during the fast closing, power stroke and initial opening phases of chewing. Three functional muscle groups related to the direction of tendon pull are shown ........................................................................................................................................... 49

Figure 2.1: Jaw tracking and force measurement equipment used in the first study conducted at Tohoku University .................................................................................. 64

Figure 2.2: Mean number of chewing cycles per trial in both the Control group and the Exercise group as well as for all participants combined .................. 70

Figure 2.3: Scree plot of the principal component analysis (PCA) confirming that the fifth PC (PC5) is at the elbow or inflexion point ............................. 72

Figure 2.4: Graphical representations of the displacement of the jaw for each of the first five principal components that explained at least 90% of the variance of the chewing cycles in all 14 participants in the coronal plane and the sagittal plane .......................................................... 73

Figure 2.5: Graphical representations of the displacement of the jaw of the 3 principal components (PCs) that showed significant changes of the 5 PCs that explained at least 90% of the variance of the chewing cycles in the 7 Control group participants before and after the “Exercise” condition .......................................................... 76

Figure 2.6: Graphical representations of the displacement of the jaw of the 3 principal components (PCs) that showed significant changes of the 5 PCs that explained at least 90% of the variance of the chewing cycles in the 7 Exercise group participants before and after the “Exercise” condition .......................................................... 77
Figure 3.1: Flowchart depicting the experimental design with sample sizes (n) in each group ................................................................. 102

Figure 3.2: Acrylic faceplate and its positioning during the maximum voluntary contraction (MVC) and resistance exercise trials ........................................... 105

Figure 3.3: Force biofeedback during the maximum voluntary contraction (MVC) and resistance exercise tasks ................................................................. 106

Figure 3.4: Oblique view of a participant showing the position of the EMG electrodes and the 3SPACE® FASTRAK® system .............................................. 109

Figure 3.5: Synchronised raw jaw opening displacement and electromyographic data from a typical chewing sequence from one participant ......................... 115

Figure 3.6: Synchronised jaw opening displacement and processed electromyographic data from a typical chewing sequence from one participant. The sixth to tenth chewing trials were used for data analysis as shown approximately between the vertical blue lines .............. 116

Figure 3.7: The synchronised movement and Butterworth filtered electromyographic data from a chewing cycle of a typical chewing trial from one participant ........................................................................... 117

Figure 3.8: Example of representative output graphs following the automated detection of peaks, onsets and offsets of the synchronised jaw opening displacement and processed rectified electromyographic data from the left anterior temporalis and left masseter from a typical chewing sequence from one participant ..................................................... 119

Figure 3.9: Mean number of exercise sets performed by all participants in the Exercise group .............................................................................................................. 129

Figure 3.10: Jaw movement duration related variables across all data collection sessions in the Control group and the Exercise group, including: Mean jaw movement duration; Mean chew cycle duration; Mean jaw movement open time; and Mean jaw movement close time ....................... 131
Figure 3.11: Median jaw movement duration as a percentage of chew cycle duration across all data collection sessions in the Control group and the Exercise group ................................................................. 135

Figure 3.12: Jaw movement displacement and velocity related variables across all data collection sessions in the Control group and the Exercise group, including: Mean jaw movement displacement; Mean jaw movement opening velocity; Median jaw movement closing velocity; and Mean chewing velocity ........................................................................................................ 137

Figure 3.13: Mean chewing frequency across all data collection sessions in the Control group and the Exercise group ............................................................................................................. 141

Figure 3.14: EMG duration of the tested muscles across all data collection sessions in the Control group and the Exercise group. The tested muscles included the ipsilateral anterior temporalis, contralateral anterior temporalis, ipsilateral masseter and contralateral masseter .......... 143

Figure 3.15: EMG Onset Relative to Jaw Movement Onset of the tested muscles across all data collection sessions in the Control group and the Exercise group. The tested muscles included the ipsilateral anterior temporalis, contralateral anterior temporalis, ipsilateral masseter and contralateral masseter ........................................................................................................ 146

Figure 3.16: Time to Peak EMG Activity Relative to Jaw Movement Onset of the tested muscles across all data collection sessions in the Control group and the Exercise group. The tested muscles included the ipsilateral anterior temporalis, contralateral anterior temporalis, ipsilateral masseter and contralateral masseter ................................................................. 151

Figure 3.17: Median EMG Offset Relative to Jaw Movement Onset of the tested muscles across all data collection sessions in the Control group and the Exercise group. The tested muscles included the ipsilateral anterior temporalis, contralateral anterior temporalis, ipsilateral masseter and contralateral masseter ........................................................................................................ 157
Figure 3.18: Median EMG Offset Relative to Jaw Movement Offset of the ipsilateral anterior temporalis, contralateral anterior temporalis, ipsilateral masseter and contralateral masseter across all data collection sessions in the Control group and the Exercise group ............... 162

Figure 3.19: Relative EMG Onset as a Percentage of Chew Duration of the tested muscles across all data collection sessions in the Control group and the Exercise group. The tested muscles included the ipsilateral anterior temporalis, contralateral anterior temporalis, ipsilateral masseter and contralateral masseter ............................................................... 167

Figure 3.20: Relative EMG Onset as a Percentage of Chew Cycle Duration of the tested muscles across all data collection sessions in the Control group and the Exercise group. The tested muscles included the ipsilateral anterior temporalis, contralateral anterior temporalis, ipsilateral masseter and contralateral masseter ......................................................... 172

Figure 3.21: Median Relative EMG Offset as a Percentage of Chew Cycle Duration of the ipsilateral anterior temporalis, contralateral anterior temporalis, ipsilateral masseter and contralateral masseter across all data collection sessions in the Control group and the Exercise group ....... 177

Figure 3.22: The kinematic and electromyographic (EMG) features of a typical single chewing cycle expressed as a percentage of the chewing cycle duration ............................................................................................................ 181
List of Tables

Table 1.1: The movements of the mandible and the primary factors contributing to each of the movements .......................................................... 37

Table 2.1: Summary of the number of chewing cycles in each experimental group that were analysed using principal component analysis. Also shown are the mean number of chewing cycles and standard deviation, as well as the minimum and maximum number of chewing cycles in each group .......................................................... 69

Table 2.2: Extract from the PASW Principal Component Analysis showing the Total Variance explained by the first five principal components (PCs) of the chewing cycles of all 14 participants (taken from a total of 123 components). The Cumulative % column shows that the first five PCs accounted for 92.1% of the total variance in the chewing cycles .......... 71

Table 2.3: Summary of the significant changes in principal component (PC) scores of the first five PCs and their respective effects on the mandibular masticatory movement paths ..................................................... 82

Table 3.1: Demographic breakdown of the 20 participants recruited into the second study conducted at Tokyo Medical and Dental University .......... 126

Table 3.2: Preferred chewing side of the 20 participants recruited into the second study conducted at Tokyo Medical and Dental University .......... 127

Table 3.3: Mean (SD) times between data collection sections for both the Control group and the Exercise group .......................................................... 127
Abbreviations

%MVC ..........Percentage of maximum voluntary contraction (MVC).

α ......................The probability of making a Type I error, set at α = 0.05.

Ag-AgCl ..........Silver-silver chloride.

ANCOVA ........Analysis of covariance.

ANOVA ..........Analysis of variance.

β ......................The probability of making a Type II error, set at α = 0.2.

DC/TMD ..........Diagnostic Criteria for Temporomandibular Disorders (TMD).

EFD ..............Elliptic Fourier descriptors.

EMG ..............Electromyographic.

FHP ..............Frankfort horizontal plane.

gm ..............Grams.

Hz ..............Hertz.

IP ..............The intercuspal position.

LBP ..............Low back pain.

MANOVA ..........Multivariate analysis of variance.

Max ..............Maximum value.

Min ..............Minimum value.

MIPT ............Mid-incisor point.

µm ..............Micrometres.

mm ..............Millimetres.

ms ..............Milliseconds.

µV ..............Microvolts.

mV ..............Millivolts.

MVC ..............Maximum voluntary contraction.
n .................. Sample size.

p .................. p-value, level of statistical significance, set at p = 0.05, unless otherwise stated.

PC .................. Principal component.

PCA ............... Principal component analysis.

RDC/TMD ........ Research Diagnostic Criteria for Temporomandibular Disorders (TMD).

RMS ............... Root mean square.

s .................. Seconds.

S1 .................. Data collection session 1.

S1-Baseline ...... First section of Data collection session 1 (S1) during which baseline jaw movement and EMG data were collected.

S1-Post .......... Third section of Data collection session 1 (S1) during which jaw movement and EMG data collection was repeated a second time approximately 15 minutes after S1-Baseline.

S2 .................. Data collection session 2, two weeks after Data collection session 1 (S1).

S3 .................. Data collection session 3, four weeks after Data collection session 1 (S1).

SD .................. Standard deviation.

TMD ................ Temporomandibular disorders.

TMDU ............. Tokyo Medical and Dental University.

TMJ ............... Temporomandibular joint.

VL ................. Vastus lateralis muscle.

VMO ............... Vastus medialis oblique muscle.
Conferences and Presentations

Parts of this thesis have been presented in the form of either Oral or Poster Presentations at the following conferences attended during the completion of this PhD:


Publications

The following works have been published during the completion of my PhD candidature.

No part of the data reported in any of these publications has been presented in the main body of this thesis. Any reference(s) to these publications has been duly cited in the text.


I assisted in editing the final manuscript of the above publication.


I co-designed the above study with the co-authors and assisted with data collection.


I assisted in editing the final manuscript of the above publication.

The publication above reports the results of the study completed as part of my MPhil candidature at The University of Sydney (2008-2009). With the assistance of the co-authors I co-designed this study, co-wrote and submitted the ethics and grant applications, arranged and completed the data collection, extracted, processed, analysed and interpreted the data and wrote the drafts of the manuscript.

In addition to the statements above, in cases where I am not the corresponding author of a published item, permission to include the published material has been granted by the corresponding author.

Alexander Wirianski 26 August 2016

As supervisors for the candidature upon which this thesis is based, we can confirm that the authorship attribution statements above are correct.

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Chapter 1

Literature Review

Introduction

Temporomandibular disorders (TMD) are musculoskeletal conditions that affect the jaws and the muscles of mastication as well as their associated structures (Manfredini and Nardini, 2010, Leeuw et al., 2008, Lund, 2001). Temporomandibular disorders are the most prevalent orofacial pain condition of non-dental origin (Manfredini and Nardini, 2010) and, after chronic low back pain, they are the second most common musculoskeletal condition that results in pain and disability (National Institute of Dental and Craniofacial Research, 2014). They are reported to affect up to 10% to 18% of the adult population (Le Resche, 1997, Von Korff et al., 1988) with up to 15% of sufferers perceiving a need to seek treatment (De Kanter et al., 1992). Various treatment modalities, including therapeutic exercise, have been proposed for the management of TMD (Medlicott and Harris, 2006, McNeely et al., 2006, List and Axelsson, 2010). Unfortunately, due to the lack of consensus on defining TMD, inclusion and exclusion criteria, and the use of reliable and valid outcome measures (Medlicott and Harris, 2006) and that, many of the reviewed studies utilised the prescription of exercises in combination with other physiotherapy modalities, it is difficult to assess the efficacy of the specific exercise component alone in the management of TMD.

Limitations and/or deviations of mandibular movements are common signs and symptoms of TMD (Schiffman et al., 2014, Laskin, 1969, Solberg, 1983, Bell, 1983) which may impact on an individual’s ability to chew. Mastication, or chewing, is a complex, intermittent rhythmical movement of the jaws that prepares a food bolus for swallowing.
Masticatory movement patterns are produced and controlled by the masticatory central pattern generator and modulated by inputs and feedback from structures in both the central and peripheral nervous systems (Lund and Kolta, 2006, Avivi-Arber et al., 2011, Rossignol et al., 1988, Lund and Enomoto, 1988).

The temporomandibular joint (TMJ) is one of many joints that comprise the human musculoskeletal system. In other joints, such as the knee, shoulder, cervical spine and lumbar spine, painful musculoskeletal conditions are often associated with biomechanical deficiencies of altered patterns of muscle activation (Cowan et al., 2001, Hodges and Richardson, 1996, Jull et al., 2009, Wadsworth and Bullock-Saxton, 1997) which are thought to be the result of alterations in motor control at these joints (Tsao et al., 2010, Tsao and Hodges, 2007, Worsley et al., 2013). It is plausible therefore that TMD may also affect the motor control of the muscles of mastication as they act on the TMJs to produce masticatory movements.

The prescription of therapeutic exercise has been shown to be associated with improvements in symptoms and function as well as restoration of normal electromyographic (EMG) activity patterns and kinematics across a range of joints in the body (Cowan et al., 2003, Ginn and Cohen, 2005, O'Leary et al., 2007, Tsao et al., 2010). Recent systematic reviews have also reported that therapeutic exercise may be of benefit in the management of TMD (Medlicott and Harris, 2006, McNeely et al., 2006, List and Axelsson, 2010). One of the actions of therapeutic exercise may be to target and produce changes in the motor control of the muscles of mastication, similar to those reported in the tongue (Boudreau et al., 2007) and low back musculature (Tsao et al., 2008). Currently
there is limited understanding of how therapeutic exercises work in the jaw motor system or exactly how they affect masticatory motor control. The overall aim of this thesis therefore, is to investigate the effects of isometric resistance exercise training on the muscles of mastication during the functional task of chewing with the view of furthering our understanding of the motor control of the masticatory system. An understanding of the mechanisms that underlie normal motor control of the masticatory system and applying that knowledge appropriately to resolving deficits associated with altered jaw movement patterns may provide effective strategies for the management of TMD.

This literature review provides a discussion of the possible mechanisms underlying the efficacy of therapeutic exercise in the management of common painful musculoskeletal conditions as well as the effects of various therapeutic exercise regimens on motor control. This is followed by an overview of our current understanding of TMD and the use of therapeutic exercise in its management. The functional anatomy of the TMJ and the muscles of mastication are also briefly discussed. To provide an overview of the methodologies used in this thesis an outline of the collection of EMG activity and jaw movement data is provided. As with other common musculoskeletal pain conditions, therapeutic exercise is also utilised in the management of TMD and may have similar effects on the motor control of the masticatory system as those demonstrated in other musculoskeletal systems. The motor control of mastication and orofacial function is therefore briefly discussed. A summary of the problem under investigation is then provided and the aims and hypotheses of this thesis are presented. Finally, to help place the studies presented herein into a relevant context a brief outline of the clinical relevance is provided.
Physiotherapy and Musculoskeletal Motor Control

The prescription of specific exercises is a common treatment modality for the management of musculoskeletal conditions. Muscle training has been reported to be specific to limb position (Doucette and Child, 1996, McConnell, 2002, Sale and MacDougall, 1981), movement pattern, velocity of joint movement, the type of muscle contraction utilised in the joint movement and the force of muscle contraction (Sale and MacDougall, 1981) as well as the performed task (Adkins et al., 2006, Sanes and Donoghue, 2000).

Improvements in performance are thought to result from neural and muscle adaptations as a response to specific strength training that allow for improvements in the coordination of the activation patterns of muscle groups (McConnell, 2002, Sale and MacDougall, 1981). In addition, compared to performing an unskilled reaching task, training of a skilled reaching task has been shown to induce greater plastic changes in the rat motor cortex as evidenced by an expansion in the movement representation of the wrist and digit (Kleim et al., 2002, Kleim et al., 1998) as well as an increased number of synapses for each neurone in these cortical areas (Kleim et al., 2002).

Many musculoskeletal pain conditions have been reported to be associated with alterations in the activation patterns of the muscles that control particular joints including the knee (Cowan et al., 2002, Cowan et al., 2001), shoulder (Cools et al., 2003, Wadsworth and Bullock-Saxton, 1997), cervical spine (Falla et al., 2004b, Falla et al., 2004a, Jull et al., 2004) and lumbar spine (Hodges and Richardson, 1996, O'Sullivan et al., 1998, Tsao and Hodges, 2008, Tsao and Hodges, 2007). Furthermore, training responses to specific exercise regimens aimed at retraining the altered muscle activation patterns to a normal state of coordination have been demonstrated to be beneficial in reducing pain and disability and improving function in the knee (Cowan et al., 2002, Cowan et al., 2003,
Crossley et al., 2002), shoulder (Ginn and Cohen, 2004, Ginn and Cohen, 2005, Ginn et al., 1997), cervical spine (Falla et al., 2006, Jull et al., 2009) and lumbar spine (Hides et al., 1996) and these will now be briefly discussed.

**Knee**

The knee complex comprises the tibiofemoral joint, which is a combination hinge and pivot joint, and the gliding patellofemoral joint (Flandry and Hommel, 2011, Oatis, 2009). These two articulations share the same supporting structures but also exhibit unique anatomical features and motions (Oatis, 2009) that contribute to the normal function of the lower limb. In particular, the normal function of the tibiofemoral joint is reliant on the appropriate motion of the patellofemoral joint (Oatis, 2009). The patella is a triangular shaped sesamoid bone that articulates with the femoral condyles (Flandry and Hommel, 2011, Fox et al., 2012, Oatis, 2009). The articular facets lie on the posterior aspect of the patella which are divided by a central ridge that runs from its proximal base to the distal apex (Flandry and Hommel, 2011, Fox et al., 2012, Oatis, 2009). The central ridge glides superiorly and inferiorly in the trochlear notch, a reciprocal sulcus formed between the medial and lateral femoral condyles on the anterior surface of the distal femur (Fox et al., 2012, Oatis, 2009). Through its position in the patella tendon the main role of the patella is to increase the mechanical advantage of the quadriceps muscle group while completing functional tasks involving concentric and eccentric knee extension and flexion (Fox et al., 2012, Schindler and Scott, 2011). The quadriceps femoris muscle group comprises the vastus medialis, vastus lateralis (VL), vastus intermedius and rectus femoris, all of which have slightly differing lines of pull at their insertions into the patella tendon (Flandry and Hommel, 2011, Oatis, 2009). In order to maintain the optimum position of the patella during functional tasks all of the quadriceps muscles must act in coordination. The patella
centralises the divergent pull from the four quadriceps muscles and transmits these forces to the patella tendon to maintain normal knee function (Schindler and Scott, 2011). In order to achieve this goal, contraction of the vastus medialis oblique (VMO) acts to stabilise the patella in the trochlear notch during contraction of the quadriceps femoris (Oatis, 2009).

In patients suffering from patellofemoral pain syndrome it has been reported that the onset of the VL muscle preceded the onset of the VMO muscle during ascending and descending stair climbing tasks (Cowan et al., 2002, Cowan et al., 2001) as well as during a functional postural challenge (Cowan et al., 2003). Physiotherapy interventions aim to restore the functional coordination of the vasti muscles. These interventions include isometric contractions of the VMO over six weeks and have been shown to result in the EMG onset of VMO preceding the VL in a descending stepping task and the EMG onsets of the VMO and VL occurring simultaneously during an ascending stair climbing task (Cowan et al., 2002) as well as during a functional postural challenge (Cowan et al., 2003). These interventions also resulted in a reduction in reported symptoms and disability (Cowan et al., 2002, Cowan et al., 2003, Crossley et al., 2002).

**Shoulder**
The shoulder is an extremely mobile joint that allows the arm to move through a wide range of motion (Carmichael and Hart, 1985, Hart and Carmichael, 1985, Saha, 1971, Schenkman and Cartaya, 1987). In order to complete everyday arm movements the dynamic stability of the shoulder is controlled by an intricate combination of muscular force couples that produce coordinated movements of the scapula, humerus and clavicle (Hart and Carmichael, 1985, Schenkman and Cartaya, 1987). The primary muscles acting across the shoulder include the four rotator cuff muscles, supraspinatus, infraspinatus,
subscapularis and teres minor, the deltoid (Carmichael and Hart, 1985, Hart and Carmichael, 1985, Saha, 1971, Schenkman and Cartaya, 1987) and the muscles that act on the scapula including the trapezius and serratus anterior (Carmichael and Hart, 1985, Hart and Carmichael, 1985, Schenkman and Cartaya, 1987). Together these muscles act in coordination to provide appropriate scapulohumeral rhythm and shoulder stability throughout the normal range of motion required for function (Struyf et al., 2011b, Struyf et al., 2011a).

The presence of shoulder pain has been reported to be related to changes in the activation patterns of the scapular rotator muscles (Wadsworth and Bullock-Saxton, 1997). More specifically, patients with anterior shoulder instability demonstrate reduced EMG activity in the serratus anterior and supraspinatus muscles during abduction movements of the shoulder (McMahon et al., 1996). In patients with shoulder impingement symptoms a delay in EMG activity has been demonstrated in the middle and lower trapezius during sudden downward falling movements of the shoulder along with reduced coordination between the different parts of the trapezius (Cools et al., 2003) as well as delayed onset and early termination of the EMG activity of the serratus anterior and lower trapezius during abduction movements (Worsley et al., 2013). Furthermore, during abduction movements, an increased EMG activity has been reported in the supraspinatus and latissimus dorsi muscles (Diederichsen et al., 2009). Interestingly, along with these EMG changes occurring on the symptomatic side, concomitant EMG changes have also been reported on the asymptomatic side (Diederichsen et al., 2009). During external rotation movements of the shoulder a reduction in EMG activity was reported in the serratus anterior and infraspinatus muscles on the symptomatic side as well as concomitant increased EMG activities in the supraspinatus, anterior and middle deltoid and upper trapezius muscles on
the asymptomatic side that were different from the changes on the symptomatic side (Diederichsen et al., 2009). Increased variability in the activation patterns of the upper and lower trapezius and serratus anterior muscles of swimmers with impingement symptoms as well as delayed onset of EMG activity in the serratus anterior muscle of the asymptomatic side have also been reported during abduction of the shoulder in the plane of the scapula (Wadsworth and Bullock-Saxton, 1997).

Prescription of specific shoulder exercises results in the restoration of normal function and reduced levels of pain (Ginn and Cohen, 2004, Ginn and Cohen, 2005, Ginn et al., 1997, Worsley et al., 2013). A shoulder rehabilitation programme aimed at optimising the position of the scapula in relation to the thorax and thereby improve the motor control of the muscles that stabilise the scapula during arm movements resulted in the restoration of the muscle recruitment patterns in the serratus anterior and lower trapezius muscles along with a concomitant restoration of the normal kinematics of the scapula (Worsley et al., 2013). Clinically, similar improvements in shoulder function and reduced levels of pain have been reported following conservative rehabilitation programmes aimed at restoring the normal neuromuscular control at the shoulder (Ginn and Cohen, 2004, Ginn and Cohen, 2005, Ginn et al., 1997).

**Cervical Spine**

Longstanding neck pain and/or cervicogenic headache have been reported to result in compromised function of the muscles controlling the head and neck (Falla et al., 2006). Patients diagnosed with mechanical neck pain show reduced active neck extension range of motion as well as decreased isometric neck muscle strength in all directions (Chiu and Sing, 2002). Similarly, chronic neck pain sufferers demonstrate reduced neck muscle strength in cervical extension (Cagnie et al., 2007, Ylinen et al., 2004) as well as flexion and rotation
along with greater fatigue in the sternocleidomastoid and scalene muscles (Falla et al., 2003). Decreased active cervical extension range of motion along with reductions in muscle strength of the cervical flexors and extensors and decreased endurance of the anterior cervical musculature have also been reported in patients with chronic headache (Placzek et al., 1999). Furthermore, in patients with a combination of chronic unilateral neck pain and headache, reduced neck flexion peak and average forces have also been reported (Barton and Hayes, 1996). These results tend to suggest the presence of changes in the activation patterns of the cervical musculature in people with chronic neck pain disorders (Jull et al., 2009). Indeed, when performing a low load cranio-cervical flexion task, participants in this patient group demonstrated an increased amplitude of EMG activity in the superficial sternocleidomastoid (Jull et al., 2004) and anterior scalene (Falla et al., 2004a) muscles associated with reduced EMG activation of the deep cervical flexors and reduced cranio-cervical flexion range of motion (Falla et al., 2004a). Moreover, possible changes in the automatic feedforward control of the cervical spine may also occur as demonstrated by a delay in the activation of the superficial and deep cervical flexor muscles during a rapid arm movement task (Falla et al., 2004b).

The prescription of specific therapeutic exercise regimens targeting deficiencies in the cervical musculature in patients with neck pain disorders have been demonstrated to reduce pain (Ylinen et al., 2007, Ylinen et al., 2003, Jull et al., 2002, O’Leary et al., 2007), increase cervical range of motion (Ylinen et al., 2007, Ylinen et al., 2003), improve disability and health related quality of life measures (Salo et al., 2012, Ylinen et al., 2007, Ylinen et al., 2003) and return the altered muscle activation patterns to states that are similar to those seen in asymptomatic individuals (Falla et al., 2006, Jull et al., 2009). In particular, undertaking a low load training programme of the cranio-cervical flexor
muscles has been demonstrated to reduce the EMG activity of the superficial anterior neck flexors, sternocleidomastoid and anterior scalene (Falla et al., 2006) with a concomitant increase in the EMG activity of the deep cervical flexors (Jull et al., 2009). Using pressure biofeedback in patients with chronic neck pain to target the deep upper cervical flexors, longus capitis and longus colli, and simultaneously minimise the action of the sternocleidomastoid and anterior scalene, resulted in an increase in EMG amplitude in the deep cervical flexors and a decreased EMG amplitude in the sternocleidomastoid and anterior scalene (Jull et al., 2009). Furthermore, although no significant differences were found in the relative onset of EMG activities when completing a functional rapid arm movement task, more participants that completed the low load cranio-cervical flexion training showed earlier EMG onsets of the deep cervical flexors, which resembled those of asymptomatic volunteers (Falla et al., 2004a), compared to the group that completed an unskilled, non-specific progressive resistance exercise programme aimed at increasing the general strength of the cervical flexor muscles (Jull et al., 2009). These results may suggest that, in patients with chronic neck pain disorders, specific low load training aimed at preferentially optimising the activity of the deep cervical flexor muscles could improve the motor control patterns of the muscles of the neck and thereby be a possible mechanism that underlies the resolution of symptoms.

**Lumbar Spine**
People with acute or subacute onset of unilateral low back pain (LBP) show signs of muscle atrophy, as evidenced by a reduced cross-sectional area in the ipsilateral multifidus at the level corresponding to their symptoms on manual palpation (Hides et al., 1994). Unfortunately there appears to be no spontaneous recovery of the reduced cross sectional area following spontaneous recovery of the LBP symptoms (Hides et al., 1996). However,
subjects that undertook a four-week specific exercise programme, consisting of facilitated active, isometric multifidus contraction in co-contraction with the deep abdominal muscles, responded with a more rapid and more complete recovery in the cross-sectional area of multifidus at follow-up (Hides et al., 1996) as well as reduced rates of recurrence of symptoms following a first episode of LBP (Hides et al., 2001).

Compared to asymptomatic matched controls, patients with chronic LBP who have minimal or no symptoms at the time of testing exhibit a delayed onset of EMG activity in the transversus abdominis muscle during a rapid arm movement task (Hodges and Richardson, 1996). The direction specific nature of the delay in the transversus abdominis EMG activity also indicated that the reported changes were a fundamental deficiency of the motor control of this muscle thereby rendering the muscle ineffective in maintaining the stability of the spine in preparation for and during external perturbations (Hodges and Richardson, 1996). This may provide a mechanism for the increased risk of recurrence and/or exacerbation of symptoms in people with chronic LBP.

Undertaking specific exercise programmes that target the stabilising role of the abdominals with co-contraction of the lumbar multifidus muscles have resulted in reduced pain and functional disability levels (O'Sullivan et al., 1997) as well as an increased ratio of internal oblique EMG activation relative to rectus abdominis (O'Sullivan et al., 1998). A growing body of recent evidence also suggests that the deficits in the preparatory or feedforward recruitment of transversus abdominis can be reduced through specific training of this muscle in patients with chronic LBP (Tsao and Hodges, 2007, Tsao and Hodges, 2008). Compared to a general sit-up training task, performing three sets of ten repetitions of gentle contractions of the transversus abdominis muscles in isolation from other abdominal
muscles while maintaining normal respiration results in earlier EMG onset of transversus abdominalis following one training session that resembles the pattern of activation observed in healthy, asymptomatic individuals (Tsao and Hodges, 2007, Tsao and Hodges, 2008). Furthermore, the earlier EMG onset of transversus abdominalis during a rapid arm movement task is further enhanced and more closely resembles those of healthy individuals when the training regimen is extended over four weeks (Tsao and Hodges, 2008). Performing non-specific co-contraction exercises of the abdominal muscles, on the other hand, does not produce the same earlier EMG activation of the transversus abdominalis muscle during a rapid arm movement task either immediately (Hall et al., 2009, Tsao and Hodges, 2007) or after four-weeks training (Tsao and Hodges, 2008). From these findings it was suggested that the motor control changes reported during preparatory or feedforward adjustments following motor relearning interventions in patients with LBP could be the result of neural plasticity of the nervous system at many levels including changes in the excitability of motoneurones, the sensorimotor cortex and/or the cerebellum (Tsao and Hodges, 2007). Preliminary evidence may corroborate this hypothesis: transcranial magnetic stimulation has revealed reduced motor thresholds for the stimulation of transversus abdominalis and a reorganisation of the cortical representation of trunk muscles in people with recurrent LBP and that this reorganisation was associated with postural control deficits of delayed onset of EMG activity in the transversus abdominalis muscle during rapid arm movement tasks (Tsao et al., 2008). This reorganisation of the motor cortex found in LBP patients has further been demonstrated to be reversible with the application of specific exercises consisting of voluntary activation of transversus abdominalis independently from other trunk muscles for two weeks (Tsao et al., 2010). At the completion of this skilled training programme the motor cortical representation of
transversus abdominis moved more anteriorly and medially and hence closely resembled those found in healthy, asymptomatic individuals (Tsao et al., 2008) with concomitant earlier activation of the transversus abdominis during a rapid arm movement task and reduced pain (Tsao et al., 2010). It was postulated that these changes in the reorganisation of the motor cortex following specific exercise training may be mediated by changes in the connectivity of the neural networks associated with activation of transversus abdominis which may include unmasking of latent horizontal connections and/or modifications in the strength of synaptic contacts (Tsao et al., 2010).

**Pain and Motor Control**
Collectively, the results from the knee, shoulder and cervical and lumbar spines, suggest that the presence of pain may be associated with abnormalities in muscle activation patterns and/or the presence of poor motor control. This reduced motor control may thereby contribute or predispose individuals to the persistence or exacerbation of existing symptoms or the production of new symptoms experienced by patients with musculoskeletal disorders at these joints. However, the relationship between motor control and the presence of symptoms remains unclear (Hodges and Tucker, 2011, Hodges and Smeets, 2015) with various theories having been proposed that attempt to explain the movement changes that are associated with pain (Murray and Peck, 2007, Roland, 1986, Leeuw et al., 2007, Hodges and Tucker, 2011, Hodges and Smeets, 2015, Lund et al., 1991). The vicious cycle theory, also known as the pain-spasm-pain cycle, was drawn on clinical evidence that patients with LBP also presented with muscle spasm and that their symptoms may have been exacerbated by a cycle of pain, spasm and further pain (Roland, 1986). This theory hypothesised that, in some cases, a painful event resulted in an increase
in muscle activity which, if sustained, could lead to ischaemia, the accumulation of algesic agents and thereby further exacerbating pain (Hodges and Tucker, 2011, Roland, 1986).

On the other hand, based on experimental findings, the pain-adaptation model, proposed that there was a reduction in activity in the agonist muscle(s) that were painful or that produced painful movement (Hodges and Tucker, 2011, Lund et al., 1991). Moreover pain also caused small increases in the opposing antagonist muscles (Lund et al., 1991). These changes then resulted in a reduction in the force produced by the agonist muscle(s) and the range and velocity of movement of the affected body part (Lund et al., 1991).

Another example is the fear-avoidance theory of musculoskeletal pain which proposes a cognitive behavioural model wherein the way in which a painful event is interpreted may result in one of two different possible pathways (Leeuw et al., 2007). If the painful event is interpreted as non-threatening then the individual is likely to maintain their engagement in activities of daily living which assists to promote functional recovery (Leeuw et al., 2007). On the other hand, if the painful event is (mis)interpreted to have catastrophic consequences, this may lead to a fear of pain and/or pain anxiety resulting in the avoidance of activities which have a perceived threat of pain (Leeuw et al., 2007). The avoidance behaviour could then result in disuse and disability which was interpreted as a possible mechanism for the reduced lumbar range of motion and altered EMG patterns observed in chronic LBP patients (Geisser et al., 2004).

More specifically, in the orofacial region the integrated pain adaptation model proposes that there is an unique interaction between an individual’s multidimensional experience of pain and the organisation of their anatomically and functionally complex sensorimotor system (Peck et al., 2008, Murray and Peck, 2007). This complex interaction determines
the effect of pain on the motor activity of the muscles of mastication resulting in a new
recruitment strategy that is biomechanically optimised to activate those motor units that
have the best mechanical advantage and least metabolic demand to produce the masticatory
movement task (Peck et al., 2008, Murray and Peck, 2007). This produces an unique motor
response aimed at minimising pain and maintaining homeostasis (Peck et al., 2008, Murray
and Peck, 2007). In some situations however this new motor response could be associated
with the exacerbation of symptoms or the development of new pain (Peck et al., 2008,
Murray and Peck, 2007).

Even though these theories are based on and are consistent with a range of clinical and
experimental observations they nevertheless have their limitations in explaining the motor
adaptations to pain (Peck et al., 2008, Murray and Peck, 2007, Hodges and Tucker, 2011,
Hodges and Smeets, 2015). Unfortunately these theories, in particular the vicious cycle
theory, the pain-adaptation model and the fear-avoidance theory, are unable to account for
all the variability observed both experimentally and in clinical practice (Peck et al., 2008,
Murray and Peck, 2007, Hodges and Tucker, 2011, Hodges and Smeets, 2015) and have
therefore been described as being relatively simplistic (Hodges and Tucker, 2011, Hodges
and Smeets, 2015). In light of this, a new theory describing the motor adaptation to pain
proposes more flexible solutions than previously described to achieve the basic premise
that any adaptations have a short-term aim to reduce the pain and protect the painful part
(Hodges and Smeets, 2015, Hodges and Tucker, 2011). This contemporary model of the
motor adaptation to either actual, or the threat of, injury or pain, proposes that the motor
system adaptation may either precede or follow the injury or the onset of pain (Hodges and
Smeets, 2015). These adaptations also involve a spectrum of changes in motor behaviour
from subtle redistributions in muscle activity to complete avoidance of movement and/or
function (Hodges and Smeets, 2015). The motor adaptations are also individual and possibly task specific and therefore may be influenced by a variety of issues including psychosocial features (Hodges and Smeets, 2015). If the motor adaptations are maintained, excessive or inappropriate, they have the potential to have long-term consequences dependent on the underlying mechanism for the persistence of symptoms (Hodges and Smeets, 2015). Multiple mechanisms occurring at various levels of the nervous system are responsible for the motor adaptations to pain, all of which are potentially influenced by biological, psychological and social aspects of the pain experience (Hodges and Smeets, 2015).

The variety of theories proposed to describe the mechanisms underlying the motor adaptations to pain has led some authors to conclude that the relationship between pain and motor control is unlikely to be a simple one (Hodges and Smeets, 2015). Nevertheless, current evidence tends to suggest that motor control deficiencies may be reversible with the application of appropriate specific exercise training (Cowan et al., 2003, Worsley et al., 2013, Falla et al., 2004a, Tsao et al., 2010). Furthermore, the effectiveness of the prescribed training modality is likely to be dependent on the individual and unlikely to deliver optimal results if implemented in a generic fashion without considering the specific needs of that individual (Hodges and Smeets, 2015).

The human masticatory system is also a complex musculoskeletal system under the control of its own specific somatosensory and motor centres, similar to the patellofemoral joint, shoulder, cervical spine and lumbar spine. Moreover, the human masticatory system is also subject to musculoskeletal disorders with common signs and symptoms as those found to afflict the other joints. With these commonalities in mind it is further possible to suggest
that the TMJ and its controlling musculature could be affected by similar motor control disturbances as those demonstrated in other joints. It is therefore plausible that similar physiotherapy modalities, such as the prescription of specific resistance exercise regimens, could also be applied to the masticatory system as part of the management strategies utilised in the treatment of chronic orofacial pain conditions of musculoskeletal origin such as TMD.

**Temporomandibular Disorders**

Temporomandibular disorders are musculoskeletal conditions that involve the muscles of mastication, the temporomandibular joints and their associated structures (Manfredini and Nardini, 2010, Leeuw et al., 2008, Lund, 2001). Classically, the clinical signs of TMD have been described as a triad of: pain in the muscles of mastication and/or the TMJ, TMJ sounds, and restriction, deviation or deflection of mouth opening movements (Laskin, 1969). The American Dental Association expanded this triad slightly and suggested the term TMD to refer to a cluster of disorders characterised by: pain in the preauricular area, the TMJ or the muscles of mastication; TMJ sounds during mandibular function; and limitations or deviations in mandibular range of motion (Solberg, 1983, Bell, 1983). Historically, many variants of this classical triad of clinical signs have been used to diagnose and categorise TMD in both the clinical and research settings which has resulted in some confusion in diagnoses (Okeson, 2013) and has led to difficulties in comparing results between studies in terms of outcome measures for various physiotherapy treatment modalities (McNeely et al., 2006, Medlicott and Harris, 2006). In an attempt to standardise the diagnostic process and to better categorise potential study participants into more homogenous groups the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) was developed (Dworkin and Le Resche, 1992). The RDC/TMD was an
initial attempt at developing an evidence based universal classification system utilising a biopsychosocial model of pain intended primarily for the research environment (Dworkin and Le Resche, 1992, Sessle, 2014). The RDC/TMD has since been refined following extensive reliability and validity testing and which have resulted in modifications to the initial diagnostic criteria and in 2014 the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) were published (Schiffman et al., 2014). The musculoskeletal signs and symptoms of TMD associated with the diagnostic algorithms of Axis I of the DC/TMD include: Familiar pain in the masticatory system, including the TMJ and muscles of mastication (i.e. temporalis and masseter); Headache in the temporal region; Modification of familiar pain and headache with jaw movement, function and parafunction; Joint noises (clicking, crepitus); Locking; and Limitations of jaw opening (Schiffman et al., 2014).

Temporomandibular disorders are the most prevalent orofacial pain condition of non-dental origin (Manfredini and Nardini, 2010) and after chronic LBP are reported to be the second most common musculoskeletal condition that results in pain and disability (National Institute of Dental and Craniofacial Research, 2014). In many cases TMD can impact significantly on a patient’s everyday functions and may result in detrimental effects on their work, family and social activities and interactions (Sessle, 2000, Stohler, 1999). It has been estimated that 65% to 85% of people in the United States will experience at least one symptom of TMD during their lives with approximately 12% developing prolonged pain or disability leading to chronic symptoms (Blasberg and Greenberg, 2008, Dworkin et al., 1990). Interestingly, despite the high prevalence, only 5% to 7% of people with at least one symptom of TMD have symptoms that are severe enough for them to seek appropriate treatment (Blasberg and Greenberg, 2008, Dworkin et al., 1990, Greene and Marbach, 1982, Schiffman et al., 1990) with up to 15% of Dutch adults perceiving a need for
treatment (De Kanter et al., 1992). Even though only a small proportion of people with symptoms of TMD present for treatment, over the past decade, the annual management costs (excluding medical imaging) associated with TMD have doubled to $US 4 billion in the United States alone (National Institute of Dental and Craniofacial Research, 2014). The peak onset for TMD is between the ages of 20 to 40 years of age with a higher proportion of females being affected compared to males throughout the age groups investigated (Blasberg and Greenberg, 2008, Manfredini et al., 2010). Although the natural history of TMD remains largely unclear, the lower prevalence in the older age groups suggests that the condition may be self-limiting (Blasberg and Greenberg, 2008, Manfredini et al., 2010).

**Physiotherapy Management of Temporomandibular Disorders**

Various treatment modalities have been proposed by clinicians and researchers from a variety of health professions in an attempt to alleviate the symptoms of musculoskeletal pain and dysfunction. In particular, physiotherapy, including the prescription of specific exercises, may be effective in the treatment of many musculoskeletal conditions including, as described above: patellofemoral pain syndrome (Cowan et al., 2002, Cowan et al., 2001, Cowan et al., 2003); shoulder pain (Ginn and Cohen, 2004, Ginn and Cohen, 2005, Ginn et al., 1997) and impingement (Worsley et al., 2013); chronic cervical spine pain (Jull et al., 2002, O'Leary et al., 2007, Ylinen et al., 2007, Ylinen et al., 2003); and chronic LBP (O'Sullivan et al., 1998, O'Sullivan et al., 1997, Tsao et al., 2010). As described above, a common clinical finding associated with these musculoskeletal conditions is an altered pattern of muscle recruitment between the numerous muscles that control the movements at these joints (Cowan et al., 2001, Hodges and Richardson, 1996, Jull et al., 2009, Wadsworth and Bullock-Saxton, 1997). Specific therapeutic exercise has resulted in the
restoration of muscle activation patterns resembling those found in healthy asymptomatic individuals along with reduced pain and a return to normal function (Cowan et al., 2002, Cowan et al., 2003, Falla et al., 2004a, Jull et al., 2009, Tsao and Hodges, 2007, Worsley et al., 2013). This restoration of the normal kinematics and biomechanics of these joints has been postulated to be the result of changes in the motor control of the muscles acting across these joints (Tsao et al., 2010, Tsao and Hodges, 2007, Worsley et al., 2013).

As in the cases of the patellofemoral joint, shoulder, cervical spine and lumbar spine, the TMJ is also a musculoskeletal system under the control of the somatosensory and motor systems. Furthermore, resistance exercise programmes have been prescribed to reduce or eliminate TMJ clicking (Au and Klineberg, 1993) and bruxism (Quinn, 1995) and for the reduction of pain in patients diagnosed with internal derangement of the TMJ (Nicolakis et al., 2001) and myofascial pain dysfunction (Nicolakis et al., 2002). Although the results of these studies appear promising, the mechanism(s) underlying the relief of symptoms are yet to be elucidated. More recent systematic reviews have also reported that therapeutic exercise may be of benefit in the management of TMD (List and Axelsson, 2010, McNeely et al., 2006, Medlicott and Harris, 2006). However, these systematic reviews concluded that the results should be viewed with caution due to methodological limitations in the studies reviewed (McNeely et al., 2006) and due to the lack of consensus on defining TMD, inclusion and exclusion criteria, and the use of reliable and valid outcome measures (List and Axelsson, 2010, Medlicott and Harris, 2006). Moreover, many of the reviewed studies utilised the prescription of specific exercises in combination with other physiotherapy modalities which makes it difficult to assess the efficacy of the exercise component alone in the management of TMD.
Nevertheless, it remains plausible that specific resistance exercise may be capable of producing neuroplastic changes that can influence the motor control of the muscles of mastication and thereby provide a possible mechanism for the relief of symptoms of TMD and restoration of normal TMJ function. Indeed, it has recently been reported that the application of an isotonic resistance exercise task resulted in a reduction in the duration of EMG activity in the ipsilateral anterior temporalis with a concomitant increase in the time to peak EMG activity in the contralateral masseter and ipsilateral digastric during a standardised jaw movement task (Wirianski, 2009, Wirianski et al., 2014). These findings suggested a change in the pattern of recruitment of the muscles in order to maintain the vertical position of the mandible during the completion of the standardised lateral jaw movement task (Wirianski, 2009, Wirianski et al., 2014) and may be the result of neuroplastic changes similar to those reported in patients with LBP at the completion of a specific exercise programme (Tsao et al., 2008) and following the training of a novel tongue protrusion task (Boudreau et al., 2007).

**Functional Anatomy of the Masticatory System**

In order for clinicians to determine the appropriate indications for specific exercise programmes in the management of TMD it is important to have an understanding of the relevant functional anatomy of the masticatory system, as well as normal TMJ biomechanics and kinematics. Herein, an overview of the functional anatomy of the masticatory system relevant to this thesis will be discussed. For a more in depth review of the anatomy of the TMJ and its supporting musculature the reader is directed to the relevant references.
In brief, the muscles of mastication have classically been described simply as jaw openers and closers (Langenbach and van Eijden, 2001, Weijs, 1994) however this does not fully explain the diversity of jaw movement patterns reported during mastication (Proschel, 1987, Langenbach and van Eijden, 2001). Variations in the anatomy of the masticatory system between individuals as well as the internal architecture of the muscles of mastication have been described (Hannam and McMillan, 1994, Schumacher, 1961). The complex multipennate architecture and the divergent fibre alignments described in the muscles of mastication allow these muscles the flexibility of multiple vectors of tendon pull (Hannam and McMillan, 1994, Schumacher, 1961) making the masticatory system mechanically and kinematically redundant (Van Eijden et al., 1990, Langenbach and van Eijden, 2001). This would provide the potential for a large number of possible patterns of muscle activation that can be utilised to perform jaw movements (Lobbezoo et al., 2004, Van Eijden et al., 1990) to achieve a particular task and thereby may be a contributing mechanism that helps explain the large variability in masticatory motion paths (Proschel, 1987, Langenbach and van Eijden, 2001).

Although the masticatory muscles are described individually below, it is important to remember that mandibular movements occur simultaneously within all three anatomical planes (Miller, 1991) and are therefore rarely the result of one muscle acting individually on the mandible. Rather the muscles of mastication have been described to work synergistically in functional groups or triplets (Weijs, 1994) and may work together through motoneurone task groups (Loeb, 1985) in order to produce jaw movements required for mastication. This along with the anatomical constraints of the TMJ (Baragar and Osborn, 1984, Liu et al., 2004, Liu and Herring, 2000) may be a factor that limits the number of motor recruitment strategies that are utilised to produce the commonly
described masticatory movement patterns of which the teardrop shape is the most predominant (Proschel, 1987).

**Temporomandibular Joint**
The mandible articulates with the skull by way of two separate condylar synovial joints that act in unison to allow the production of jaw movements that are controlled by the muscles of mastication (Bermejo-Fenoll, 2010, Hawthorn and Flatau, 1990). The left and right TMJ is 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 (O'Rahilly, 1986) on each respective ipsilateral side. An articular disc divides the joint into two compartments which function as a lower hinge joint and a larger, upper plane joint (Cunningham and Romanes, 1986, O'Rahilly, 1986). This structural design enables the TMJ to be capable of hinge-type movements (ginglymoid) as well as gliding movements (arthrodial) and it has therefore been classified as a ginglymodiarthroidial joint (Okeson, 2003, Blasberg and Greenberg, 2003). Each TMJ is surrounded by an articular capsule which has a lateral triangular thickening forming the temporomandibular ligament (Cunningham and Romanes, 1986). The sphenomandibular and stylomandibular ligaments also attach the mandible to the skull. Even though it has been suggested that the articular capsule and its lateral ligament are the only true primary restraints of the TMJ, together the ligaments and the articular capsule provide little if any dynamic stability or strength to the TMJ with its integrity maintained primarily by the muscles of mastication (Cunningham and Romanes, 1986). On the other hand, mathematical modelling of open, close and lateral jaw movements has demonstrated that the position of the condyle could be accurately predicted by considering the articular eminence, the temporomandibular ligament and to a lesser extent, the sphenomandibular
ligament, as constraints to mandibular movement (Baragar and Osborn, 1984). Moreover, *in vivo* porcine animal studies have demonstrated elongation of the soft tissues of the lateral joint capsule during mastication (Liu and Herring, 2000, Liu et al., 2004). These findings suggest that, in combination with the muscles of mastication, the joint capsule and the ligaments may in fact contribute to the dynamic stability of the TMJ and act as constraints to jaw movements during function.

**Muscles of Mastication**

The dynamic stability and integrity of the TMJs is maintained by the four paired muscles of mastication: masseter; temporalis; medial pterygoid; and lateral pterygoid (Cunningham and Romanes, 1986, Okeson, 2003) which are innervated by branches of the motor root of the mandibular division of the trigeminal nerve (Blasberg and Greenberg, 2003, Cunningham and Romanes, 1986, O'Rahilly, 1986). The paired digastric muscles also contribute to the production of jaw movements even though they are not considered to be part of the muscles of mastication (Okeson, 2003) and will also be discussed briefly.

**Masseter**

The masseter is a thick, quadrate muscle that originates from the inferior border of the medial aspect of the zygomatic arch (Figure 1.1, Panel A; Cunningham and Romanes, 1986, O'Rahilly, 1986). It extends downwards and posteriorly to insert onto the lateral aspect of the ramus of the mandible from the region of the second molar tooth, anteriorly, to the angle, posteriorly (Okeson, 2003). The masseter has a complex multipennate architecture (Brunel et al., 2003, Gaudy et al., 2000, Hannam and McMillan, 1994, Schumacher, 1961) and has been described as having two (Cunningham and Romanes, 1986, Miller, 1991) or more commonly three heads of origin (Brunel et al., 2003, Gaudy et al., 2000, Hannam and McMillan, 1994, O'Rahilly, 1986) dividing it into deep,
intermediate and superficial portions. Each of the three heads of masseter are separated by
thick, multileaved internal aponeuroses which are roughly aligned in the parasagittal plane
(Brunel et al., 2003, Hannam and McMillan, 1994, Schumacher, 1961). Some of the
aponeuroses traverse downwards through masseter 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 (Hannam and McMillan,
1994, Schumacher, 1961). These septa provide the anchorage for the multipennate
arrangement of masseter’s muscle fibres which mostly insert into adjacent interleaved
aponeuroses with some inserting into the bone of the zygomatic arch or the mandibular
ramus (Brunel et al., 2003, Hannam and McMillan, 1994).

Masseter’s complex architecture of multiple heads with interleaved aponeuroses and
multipennate fibre arrangement is also accompanied by regional differences in muscle
fibre, sarcomere and tendon lengths (Hannam and McMillan, 1994, Van Eijden and
Raadsheer, 1992). Moreover, motor unit recruitment was found to be dependent on bite
force magnitude, direction and duration and consequently attributed to the structural and
physiological differences between the superficial and deep muscle heads (Ogawa et al.,
2006). Furthermore, regional task dependent differences in motor unit firing have also been
demonstrated and attributed to task dependent differences in the excitation of the
trigeminal motoneurone pool related to the performed task (McMillan and Hannam, 1992).
These anatomical and physiological features provide masseter with a variety of options that
would allow it to adapt to the needs of varying functional demands and contribute to
producing appropriate and effective jaw movements (Table 1.1).
Figure 1.1: The muscles of mastication showing their direction of pull (wide red arrows) and their subsequent action on the mandible to produce jaw movements (narrow red arrows). Panel A: Masseter. Panel B: Temporalis. Panel C: Lateral and medial pterygoids. Panel D: The suprahyoid and infrahyoid muscles. (Modified from Oatis, 2009).

**Temporals**

Temporalis is a large, fan-shaped muscle that lies in the temporal fossa (Figure 1.1, Panel B; Cunningham and Romanes, 1986, Miller, 1991, Okeson, 2003, O'Rahilly, 1986, Blasberg and Greenberg, 2003, Hannam and McMillan, 1994). The majority of its fibres
originate from its bony attachment on the floor of the temporal fossa (O’Rahilly, 1986) with a soft tissue attachment being from the deep surface of the temporal aponeurosis or fascia (Hannam and McMillan, 1994, O’Rahilly, 1986). Temporalis passes deep to the zygomatic arch to insert into the coronoid process and the anterior border of the ramus of the mandible (Cunningham and Romanes, 1986, Okeson, 2003, O’Rahilly, 1986, Hannam and McMillan, 1994). Temporalis has been described as having a central conspicuous tendon formed by the convergence of the muscle’s fan-shaped collection of long fibres (Cunningham and Romanes, 1986, Okeson, 2003, Hannam and McMillan, 1994). This central tendon extends superiorly into the muscle as an internal central aponeurosis that separates the muscle into its superficial and deep parts and provides temporalis with a resultant bipennate arrangement of its fibres, especially in the anterior region, when viewed in the frontal plane (Cunningham and Romanes, 1986, Hannam and McMillan, 1994).

Temporalis can be further divided into anterior, middle and posterior portions according to the direction of its fibres and resultant function (Okeson, 2003). The fibres in the anterior portion are aligned vertically at low pennation angles (Okeson, 2003, Hannam and McMillan, 1994) and therefore contribute primarily to raising the mandible vertically (Table 1.1; Cunningham and Romanes, 1986, Miller, 1991, Okeson, 2003, O’Rahilly, 1986). In the middle portion the fibres run obliquely and posteriorly across the lateral aspect of the skull (Okeson, 2003) fanning out at different lengths and angles of pennation (Hannam and McMillan, 1994) and therefore contributing to elevation and retrusion of the mandible (Table 1.1; Okeson, 2003). The posterior portion is thin (Hannam and McMillan, 1994) with its fibres aligned almost horizontally (Okeson, 2003, Hannam and McMillan, 1994) thus contributing to retrusion of the mandible (Table 1.1; Cunningham and Romanes, 1986, Miller, 1991, O’Rahilly, 1986). When temporalis contracts as a whole it acts to
maintain the resting posture of the mandible and to elevate the mandible into molar occlusion (Table 1.1). 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 1.1; Cunningham and Romanes, 1986, O'Rahilly, 1986).

**Medial Pterygoid**

Medial pterygoid is a rectangular muscle that lies on the medial aspect of the ramus of the mandible and possesses two heads of origin that embrace the inferior head of the lateral pterygoid and unite passing downward, backward and laterally to insert between the mandibular foramen and the angle of the mandible (Figure 1.1, Panel C; Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, O'Rahilly, 1986). The larger, deep head arises from the medial surface of the lateral pterygoid plate and the pyramidal process of the palatine bone (Cunningham and Romanes, 1986, Hannam and McMillan, 1994). The smaller, superficial and more inferior head arises from the pyramidal process of the palatine bone and the maxillary tuberosity (Cunningham and Romanes, 1986, Hannam and McMillan, 1994). The medial pterygoid’s fibres run nearly parallel to those of the superficial fibres of masseter (Cunningham and Romanes, 1986) together forming a muscular sling supporting the vertical position of the mandible at its angle (Okeson, 2003).

Morphologically, the medial pterygoid has similar features to those of masseter with six to eight interleaved aponeuroses positioned closely together creating a multipennate arrangement of short muscle fibres (Hannam and McMillan, 1994). This divergent angulation of the fibres suggests that medial pterygoid has the ability to produce different actions on the mandible with muscle tension being potentially generated along at least two principal axes: one anteriorly and directed upward, medially and forward; and the other posteriorly and directed upward, medially and further forward (Hannam and McMillan,
Thus, medial pterygoid, acting in synergy with masseter, elevates the mandible (Table 1.1; Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, Okeson, 2003, O'Rahilly, 1986). Also, acting together with lateral pterygoid it is capable of protruding the mandible (Table 1.1; Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Okeson, 2003, O'Rahilly, 1986) and unilateral contraction produces contralateral lateral movement of the mandible (Table 1.1; Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, Okeson, 2003, O'Rahilly, 1986).

**Lateral Pterygoid**

Lateral pterygoid occupies the infratemporal fossa and possesses two distinct heads of origin (Table 1.1, Panel C; Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, O'Rahilly, 1986) which have been described to function differently (Okeson, 2003). The inferior head has been described as being three times larger than the superior head (Miller, 1991) and having twice the cross-sectional area (Hannam and McMillan, 1994). It arises from the lateral surface of the lateral pterygoid plate (Cunningham and Romanes, 1986, Miller, 1991, Okeson, 2003, O'Rahilly, 1986, Hannam and McMillan, 1994) 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 (Miller, 1991, Okeson, 2003, O'Rahilly, 1986). The thin, flat band of fibres of the superior head arise from the infratemporal ridge and infratemporal surface and crest of the greater wing of sphenoid (Cunningham and Romanes, 1986, Miller, 1991, Okeson, 2003, O'Rahilly, 1986, Hannam and McMillan, 1994) and travel posteriorly, laterally and caudally to converge on their insertions into the anterior surface of the articular capsule, the anterior margin of the

When viewed together, the two heads of lateral pterygoid have a curved, fan-shaped attachment that sweeps through an arc of near horizontal fibres in the superior head to near vertical fibres in the inferior head (Figure 1.1, Panel C; Hannam and McMillan, 1994). This divergent fibre alignment allows the lateral pterygoid a varied line of pull dependent on function leading some authors to describe the two heads as being capable of independent actions (Miller, 1991, O'Rahilly, 1986). The superior head has been described as having more biomechanical advantage to close the mandible, especially when acting in conjunction with the elevators, with the inferior head being more biomechanically efficient at lowering the condyle to allow its translation downward and anteriorly across the articular eminence (Table 1.1; Miller, 1991, Okeson, 2003). This, in conjunction with the superior head’s attachment to the articular disc and anterior portion of the articular capsule, result in the lateral pterygoid being the chief protractor of the articular disc and hence the mandible by translating 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) and thereby contributing to protrusion and/or depression of the mandible (Table 1.1).

At their insertion the two heads of lateral pterygoid converge and their individual fibres become difficult to separate (Hannam and McMillan, 1994). This arrangement explains more recent findings that the two heads of the lateral pterygoid could be considered as a system of fibres that act in unison 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). A wide arc of origin that converges 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 to produce the desired movement.

**Digastric**

Digastric consists of two muscle bellies that are united by an intervening tendon attached to the hyoid bone (Figure 1.1, Panel D; Cunningham and Romanes, 1986, Hannam and McMillan, 1994, Miller, 1991, Okeson, 2003, O’Rahilly, 1986). The larger posterior belly originates from the mastoid notch of the temporal bone traveling downward, anteriorly and medially while the shorter anterior belly originates from the digastric fossa on the lower border of the mandible close to the symphysis and is directed backward and downward (Cunningham and Romanes, 1986, Miller, 1991, Okeson, 2003, O’Rahilly, 1986, Hannam and McMillan, 1994). Both the anterior and posterior bellies insert into the hyoid via the intervening tendon attached to the body and the greater horn of the hyoid bone (Hannam and McMillan, 1994, O’Rahilly, 1986).

The orientation of the fibres of the anterior belly of digastric allow it 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 (Table 1.1; Miller, 1991). Therefore, with the hyoid bone held stable by the combined action of the infrahyoid muscles and the posterior digastric, contraction of the anterior belly of digastric would produce a vector that would pull the chin backward and downward and thereby assist the lateral pterygoid in rotating the mandibular condyle in the mandibular fossa during early
mouth opening (Figure 1.1 Panel D; Cunningham and Romanes, 1986, Miller, 1991, Okeson, 2003, O'Rahilly, 1986).

**Electromyography**

Electromyography has been used extensively to investigate the static and dynamic function of the masticatory system since the late 1940’s (Carlsoo, 1952, Moyers, 1949, Suvinen and Kemppainen, 2007). It provides a measure of the electrical activity in muscles (Basmajian and Luca, 1985, Miller, 1991) as well as being an accurate dynamic measure of muscle recruitment that complements anatomical and biomechanical findings (Miller, 1991) and has therefore been utilised in the study of both asymptomatic and dysfunctional muscles (Suvinen and Kemppainen, 2007). Electromyography can thus provide valuable insight into the recruitment patterns of the muscles of mastication and help contribute to the understanding of the motor control of the masticatory system.

The most frequently investigated muscles of mastication have been the masseter and temporalis due to their ease of accessibility for non-invasive surface electrode recordings (Suvinen and Kemppainen, 2007, Svensson and Graven-Nielsen, 2001). Furthermore, by utilising the EMG waveform of the anterior temporalis and masseter muscles bilaterally during a simulated bruxing task, it is possible to predict typical jaw movements such as left and right lateral movement and protraction 96% of the time (Long, 2004). Moreover, recruitment pattern changes have been demonstrated in the anterior temporalis and masseter following an isotonic resistance exercise task (Wirianski, 2009, Wirianski et al., 2014). Recently, with the modifications of the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD), the temporalis and masseter muscles are now the only muscles of mastication included in the diagnostic algorithms of Axis 1 of the
Diagnostic Criteria for Temporomandibular Disorders (DC/TMD; Schiffman et al., 2014).

Therefore, these muscles appear to be sensitive to changes in jaw movements and would be worthy of recording when assessing if exercise training regimens change muscle activation strategies when producing typical jaw movements during mastication.

The cell bodies of the motoneurones supplying the muscles of mastication reside in the trigeminal motor nucleus (Miller, 1991) with their axons terminating on individual muscle fibres associated with each motor unit (Basmajian and Luca, 1985). Under the influence of its central motor control the excitation of a muscle fibre by its motoneurone elicits a transient change in membrane sodium channels resulting in depolarisation of the muscle fibre’s membrane of around 50 to 80 mV (Miller, 1991) which is immediately restored (repolarisation) in around 1 to 2 ms by the backward exchange of ions via the active ion pump producing an action potential (Konrad, 2006). This action potential is then propagated as a wave of electrical activity throughout the innervated muscle fibre (Miller, 1991) causing it to contract and the resultant potential difference can be recorded extracellularly in μV (Miller, 1991). Two electrodes are commonly used to record the EMG signal from individual muscles (Miller, 1991). 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 (Miller, 1991). Differential amplification is then applied wherein the potential difference between each electrode and a distant ground electrode is measured (Miller, 1991). One potential difference is then subtracted from the other and amplified which results in amplification of the signals generated close to the electrodes and reduces spurious signals from distant sources, such as physiological cross-talk and electronic noise (Miller, 1991, Fridlund and Cacioppo, 1986).
The raw EMG signal is a stochastic train of motor unit action potentials and is generally considered unsuitable for quantification without further processing (Fridlund and Cacioppo, 1986) and therefore rarely used in the research or clinical setting (Miller, 1991). Instead, it is usually processed in order to effectively quantify the EMG activity of the active muscles by first rectifying the raw EMG signal and then applying an assimilated integration technique in order to produce a smooth curve (Miller, 1991). The resultant smooth curve provides a visual display of the onset and offset of muscle activity as well as when the muscle is most active and an indication of the level of activity (Miller, 1991). Digital Butterworth filtering also produces a smoothed curve that is reflective of the rectified EMG signal with the added benefit of being able to be applied recursively in order to minimise phase shift phenomena that are common in other forms of EMG filtering, such as moving average and root mean square (RMS, Konrad, 2006). The Butterworth filter provides smoothing by sacrificing 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). Because it suits applications that require preservation of amplitude linearity in the passband region, the Butterworth filter is an ideal candidate for conditioning the EMG signal (The Mathworks Inc, 2000).

Several important limitations need to be considered when using electromyography in a research or clinical setting, with the main factors effecting the quality of EMG recordings including the signal-to-noise ratio, cross-talk and distortion (Fridlund and Cacioppo, 1986, Long, 2004). Any part of the raw EMG signal that is not produced by the action potential train generated in the muscle of interest is generally considered to be signal noise and therefore unwanted. Noise sources are varied and include: mains electricity lines;
computers and their monitors, especially cathode ray tubes; the EMG testing system (Fridlund and Cacioppo, 1986, Konrad, 2006); the electrical activity of the participant’s heart (Drake and Callaghan, 2006); cross-talk from adjacent muscles; and movement artefact, either from the electrodes moving on the skin, movement of the cables connecting the electrodes to the amplifier or the participant’s movements during data collection. In order to collect EMG data that accurately reflect the activity of the muscles being tested 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 may occur when the electrical activity from adjacent muscles is inadvertently sampled due to the inaccuracy of the electrode placement. Accurate and reproducible placement of the electrodes can minimise the occurrence of cross-talk and it has been 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) in standardised positions based on anatomical landmarks (Castroflorio et al., 2008).

**Jaw Movement**

The movements of the jaw are produced by coordinated actions of the muscles of mastication on the mandible which are initially controlled by task specific excitation from the motor cortex (Lund, 1991, Nakamura and Katakura, 1995, Yamada et al., 2005). The overall function of each TMJ is then modulated by the interaction of the recruitment of the muscles of mastication with soft and hard tissue constraints, such as articular morphology and dental contact (Lobbezoo et al., 2004, Sarinnaphakorn et al., 1997) as well as the
intrinsic elasticity or tightness of the soft tissues surrounding the TMJ. The role of the soft and hard tissue constraints can either be active, passive or a combination of both. Active constraints such as the contraction of the muscles of mastication can produce jaw movement or act on other soft tissues into which they insert, such as the joint capsule (Christo et al., 2005, Widmalm et al., 1987), resulting in increased tightness in this soft tissue structure, or the articular disc (Murray et al., 2004, Widmalm et al., 1987), where it acts to assist in the movement of the disc during jaw opening (Huang et al., 2005, Miller, 1991, Murray et al., 1999). Passive constraints such as articular morphology, dental contact (Lobbezoo et al., 2004, Sarinnaphakorn et al., 1997) or tight soft tissues may work to guide the direction of the jaw movement in a particular functional or even parafunctional direction. Thus the final trajectory of mandibular movement is affected by the centrally controlled actions of the muscles of mastication (Lund, 1991, Nakamura and Katakura, 1995, Yamada et al., 2005) interacting with the passive constraints including the shape of the articular surfaces and the articular disc (Lobbezoo et al., 2004), the ligamentous and capsular constraints and/or the occlusion (Lobbezoo et al., 2004, Sarinnaphakorn et al., 1997). The primary factors contributing to the movements of the jaw are summarised in Table 1.1.
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<th>Primary Factors Contributing to Mandibular Movement</th>
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Table 1.1: The movements of the mandible and the primary factors contributing to each of the movements (Modified from Bermejo-Fenoll, 2010, Cunningham and Romanes, 1986, Miller, 1991, O’Rahilly, 1986).
During mastication the mandible moves simultaneously within all three anatomical planes (Miller, 1991): antero-posterior movements occur in the transverse plane; superior-inferior movements occur in the sagittal plane; and medio-lateral movements occur in the coronal plane. Masticatory movement cycle paths have been commonly divided into three basic phases which relate to the movement of the mandibular incisors in the coronal plane: 1) Opening, during which the mandible moves predominantly downwards to open the mouth; 2) Closing, during which the mandible moves predominantly upwards and laterally to close the mouth and capture the food bolus between the posterior teeth; and 3) the Power Stroke, during which the mandible continues to move upward but medially towards occlusion (Figure 1.2, Panel A; (Langenbach and van Eijden, 2001, Miller, 1991, Morimoto et al., 1985, Takada et al., 1994). Eight different patterns of mandibular movement have been described in asymptomatic individuals, with the predominant pattern having a teardrop shape (Proschel, 1987). Individuals generally chew with a particular pattern which can adapt from cycle to cycle based on the task requirements primarily through changes in the closing and power strokes (Miller, 1991). Others have divided each chewing cycle into different numbers of phases depending on movement characteristics, such as: two phases, open and close (Snipes et al., 1998); three phases, open, close and occlusal (Karlsson and Carlsson, 1990, Karlsson et al., 1991, Mongini et al., 1986, Mongini et al., 1989), or opening, fast closing and slow closing (Peyron et al., 1997, Schwartz et al., 1989, Yamada and Yamamura, 1996); four phases, slow open, fast open, fast close and power stroke (Langenbach and van Eijden, 2001, Youssef et al., 1997b, Youssef et al., 1997a); or, five phases, three opening phases and two closing phases (Lund and Enomoto, 1988). For simplicity, to briefly describe the EMG activation patterns during mastication, only three phases of jaw movement will be discussed: open; close; and power stroke.
Figure 1.2: Patterns of mandibular movement during mastication as shown in the coronal plane. Panel A: The phases of the masticatory movement cycle path: Opening (1) during which the mandible predominantly moves downward; Closing (2 and 3) during which the mandible predominantly moves laterally and upwards; and the Power Stroke (4) during which the mandible predominantly moves upward and medially (Modified from Oatis, 2009). Panel B: Eight different patterns of mandibular movement (labelled A, B, C, D, ED, E1, E2 and I) described in asymptomatic individuals, with the predominant pattern having a teardrop shape (Proschel, 1987)

The coordinated actions of the muscles of mastication, under their central control, produce the mandibular movements required for mastication (Langenbach and van Eijden, 2001). Electromyographic studies have revealed that the suprahyoid muscles, including digastric, geniohyoid and mylohyoid, are most active during the opening phase (Miller, 1991). Their EMG activities commence before the opening phase begins, with digastric starting before the closing muscles complete their maximum discharge, and increase gradually until the mandible reaches its maximum velocity at approximately midrange of opening (Miller, 1991).
During the closing phase of mastication EMG activity is greater on the ipsilateral, or working side than on the contralateral, or balancing side (Miller, 1991). At the lowest point of opening the EMG activity of the ipsilateral temporalis initiates lateral movement of the mandible in the coronal plane as well as posterior movement in the sagittal plane. The lateral jaw movement also involves the contralateral lateral and medial pterygoids as well as the contralateral masseter (Miller, 1991). When unopposed, the lateral and medial pterygoids protrude the mandible and rotate it to the opposite side, while the temporalis, with its posteriorly directed vector of pull, retrudes the mandible on the ipsilateral side (Miller, 1991). When acting together under the control of coactivation patterns the closing muscles raise the mandible during the closing phase (Miller, 1991). As the mandible increases its closing velocity there is a gradual increase in the EMG activity of the closing muscles until the beginning of the power stroke when mandibular movement becomes more medial and the velocity decreases despite the increased EMG activity in the jaw closing muscles (Miller, 1991). The EMG activity of the jaw closing muscles ceases for approximately 100 ms when the mandible reaches its most vertical position which usually is not full occlusion (Miller, 1991). This cessation in EMG activity in the jaw closing muscles is thought to serve as a mechanism to allow the relatively smaller jaw opening muscles to generate enough force to counteract the larger closing muscles in order to commence the opening phase (Miller, 1991).

At the beginning of the power stroke the mandible is moved medially by the ipsilateral lateral and medial pterygoid which contract in combination with the ipsilateral masseter and contralateral temporalis (Miller, 1991). Electromyographic activity commences in the contralateral temporalis and ipsilateral masseter in order to initiate the medial movement followed by the contralateral temporalis and ipsilateral masseter some 50 to 100 ms later.
(Miller, 1991). Although EMG activity commences in the contralateral masseter first it develops a poorly defined maximum while the ipsilateral masseter gradually increases its EMG activity to primarily discharge in the power stroke (Miller, 1991). This pattern of EMG activity has been suggested to demonstrate that masseter is capable of applying forces differently depending on the functional task it is performing (Hannam and McMillan, 1994, Miller, 1991). On the ipsilateral side masseter is capable of developing large forces when crushing the food bolus, while, simultaneously, the contralateral masseter contributes to moving the mandible laterally along with the stabilising role of the temporalis (Hannam and McMillan, 1994, Miller, 1991). In general, peak EMG activity in the ipsilateral masseter and anterior and posterior temporalis muscles occurs during the middle of the power stroke when maximum food breakdown is required (Byrd and Garthwaite, 1981, Miller, 1991). However, the contralateral masseter demonstrates three regions of peak EMG activity during the closing phase with the highest peak occurring during the power stroke, a mid-range peak occurring approximately mid-way during the closing phase and a third lower peak occurring approximately 3 mm after the commencement of the closing phase (Byrd and Garthwaite, 1981).

**Measurement of Jaw Movement**

Many different methods of tracking mandibular movements have been developed over the years in an attempt to document human mastication. Early graphic methods included tracing devices, such as the Gothic arch, kinematic face-bows and the pantograph (Bates et al., 1975). These devices used clutches attached to the teeth which traced the movement path in two dimensions but were unable to simultaneously record any timing information (Buschang et al., 2000). Photography, in the form of multiple film exposures (Mohl et al., 1990), and cinematographic techniques are reported to have first been used in 1889 and
1914, respectively (Bates et al., 1975), and these later eliminated the need for the previously used cumbersome clutches (Buschang et al., 2000). As each frame of film represented an instantaneous time-point during mandibular movement, these techniques allowed for the calculation of jaw movement timing, but were still limited to recording single plane movement data (Buschang et al., 2000). With advances in technology, mechanical devices were developed to measure masticatory movement patterns in six degrees of freedom and record jaw movement trajectory data for later playback and computer analyses (Gibbs et al., 1971).

More recently, electromagnetic (Jankelson et al., 1975, Kirihara et al., 2003) and optoelectronic (Gallo, 2005, Kang et al., 1993, Mesqui and Palla, 1985) devices have been developed in an attempt to meet the requirements necessary to accurately measure mandibular movements during jaw function, which include: ease of use; being non-invasive; not interfering with the function of the jaw and soft tissues; not restricting head movement; and being capable of recording the movement of the whole mandible (Airoldi et al., 1994). Although jaw tracking devices have been reported to have little evidence for their efficacy in the diagnosis of TMD (Mohl et al., 1990) they are nevertheless considered an important tool in documenting jaw movements especially in the research of masticatory function.

Kinesiographic instruments are commonly used electromagnetic jaw tracking systems, of which the K7 Cranio-Mandibular Evaluation System (Myotronics-Noromed Inc., Tukwila, WA, USA) is one example. These systems use a lightweight headgear placed on the participant’s head from which two parallel arrays of four sensors each are suspended on either side of the face. The horizontal axis of these sensor arrays are then aligned parallel
to the Frankfort horizontal plane (FHP). Throughout the data collection procedure the sensor arrays are maintained in a constant position on the skull by a frame made of aluminium tubing connected to eyeglass frames that are fixed securely to the participant’s head. The bilateral sensor arrays detect alterations of a magnetic field generated by the movement of a small magnet with high magnetic field strength that is fixed to the participant’s labial vestibule just below the mandibular incisors and centred over the mid-incisor point (MIPT). As the participant moves their mandible, the movement of the magnet is tracked by the sensor arrays. The generated jaw movement data thus represents the spatial position of the mandibular MIPT relative to the FHP. The linear accuracy of this system during mastication has been reported as being good with measurement errors of 0.4 mm and 0.3 mm when measuring vertical and lateral displacements, respectively, without major distortion, especially within a vertical range of motion of 40 mm (Balkhi et al., 1991). Furthermore, good reproducibility has also been demonstrated under rigorously controlled recording conditions (Baba et al., 2000, De Souza et al., 2009) especially with accurate placement of the magnet and referencing to fixed craniofacial landmarks (Hannam et al., 1980).

Another electromagnetic jaw tracking system used in the research setting is the 3SPACE® FASTRAK® (Pollhemus, Colchester, Vermont USA). This system, linked to a personal computer, is capable of recording the position of the mandible with six degrees of freedom which is achieved through the use of a stationary transmitter unit and a moveable receiver unit. The stationary transmitter unit which consists of an assembly of three concentric antennae generates near field, low frequency magnetic field vectors. During movements the field vectors are detected by the moveable remote receiver unit which consists of three concentric sensing antennae. The detected signals are then input to a mathematical
algorithm and the position and orientation of the receiver unit relative to the stationary transmitter unit is calculated. This system has a reported static accuracy of 0.8 mm RMS for the X, Y or Z position of the receiver unit, and 0.15° RMS for the orientation of the receiver unit (Polhemus, 2012). At a range of 300 mm, the position resolution and the orientation resolution of the system have been reported to be 0.0058 mm and 0.0026° respectively (Polhemus, 2012). Similar findings to these results from manufacturer tests have been reported in in vitro studies. A linear accuracy magnitude of less than 0.5 mm has been reported when the remote receiver unit is placed between 50 to 650 mm away from the transmitter unit along with an angular accuracy of less than 0.5° when the remote receiver unit is placed between 100 to 350 mm away from the transmitter unit (Ribeiro et al., 2011). This system has also been shown to be a reliable measure of range of motion in the cervical spine and shoulder (Amiri et al., 2003, Jordan et al., 2000). More specifically, when used to measure TMJ motion the measurement error has been reported as being less than 100 μm for displacement and less than 0.02° for angulation when the transmitter and receiver were placed 300 mm apart (Kiriha et al., 2003).

Modern jaw tracking systems are capable of collecting data related to the position of the mandible relative to fixed craniofacial landmarks in at least three orthogonal planes and up to six degrees of freedom. Regardless of which system is used to track mandibular motion paths, a large amount of data is collected, even from a single chewing cycle, which then need to be processed and analysed. In an attempt to describe masticatory cycle paths, many different measurements have been developed that relate to the timing and excursion of individual chewing cycles (Buschang et al., 2000). For example, many studies have measured average chewing cycle duration (Byrd, 1981, Chew et al., 1988) with some studies further dividing each cycle into different numbers of phases depending on
movement characteristics, such as: two phases, open and close (Snipes et al., 1998); three phases, open, occlusal and close (Karlsson and Carlsson, 1990, Karlsson et al., 1991, Mongini et al., 1986, Mongini et al., 1989), opening, closing and power stroke (Miller, 1991, Morimoto et al., 1985, Takada et al., 1994) or opening, fast closing and slow closing (Peyron et al., 1997, Schwartz et al., 1989, Yamada and Yamamura, 1996); four phases, slow open, fast open, fast close and power stroke (Langenbach and van Eijden, 2001, Youssef et al., 1997b, Youssef et al., 1997a); or, five phases, three opening phases and two closing phases (Lund and Enomoto, 1988). Other quantitative features describing the masticatory chewing path that have been studied include, velocity and acceleration (Karlsson and Carlsson, 1990, Karlsson et al., 1991) as well as the approach angles of the mandible into centric occlusion (Karlsson et al., 1991). The majority of these jaw movement features are two-dimensional descriptors of the masticatory movement path. Mandibular masticatory movement paths are three-dimensional continuous curves and it has therefore been argued that much information about the form of the chewing movement path is discarded when analysing these variables (Buschang et al., 2000, Hattori et al., 2010).

In an attempt to capture as much information describing the masticatory movement path as possible, the opening and closing phases of individual chewing cycles have been divided into equally spaced time intervals with each time-point designated by three-dimensional cycle coordinates for each masticatory cycle in the chewing sequence (Buschang et al., 2000, Hattori et al., 2010, Igari et al., 2012, Kobayashi et al., 2009). Novel algorithms have been developed to describe the movement paths and using multilevel statistical analyses of these mathematical models has provided a more complete and objective hierarchical
description of the kinematic patterns of jaw movements and their variations during chewing (Buschang et al., 2000).

Another method of analysing the variations in the cycle paths during human mastication combines the use of elliptic Fourier descriptors (Ferrario et al., 1990, Kuhl and Giardina, 1982) and principal component analysis (Hattori et al., 2010). This method has been used to quantify movement trajectories during the chewing of different types of food where it was found that the linear combination of only three independent movement variations accounted for an average of 93% of the total variations in the masticatory cycle paths in eight healthy male participants when chewing gum and gummy candy (Hattori et al., 2010). These three extracted movement variations were further found to be similar among the eight participants suggesting that masticatory motion was controlled by identical strategies amongst the participants (Hattori et al., 2010). This technique has also been used in conjunction with cluster analysis to classify chewing cycles (Igari et al., 2012). Ten participants chewed cooked rice until swallowing and the chewing sequences were divided equally into three stages based on the number of chewing cycles (Igari et al., 2012). The resultant appearance ratios of the chewing cycles were found to be different between the stages indicating that the groups of chewing cycles that differed in the shape of their movement trajectories may also differ in function (Igari et al., 2012). These methods have demonstrated that the use of elliptic Fourier descriptors is capable of capturing the kinematics of the masticatory path in detail (Ferrario et al., 1990, Hattori et al., 2010). Furthermore, analysis of the elliptic Fourier descriptors with principal component analysis has the advantage of describing the variations of the masticatory cycle paths during a chewing sequence and may prove useful in the study of the association between changes in masticatory kinematics during different orofacial functional tasks, including masticatory
performance (Hattori et al., 2010, Wilding and Lewin, 1994) or following specific interventions, such as a resistance exercise task.

**Mastication and Orofacial Motor Control**

Mastication, or chewing, is an intermittent rhythmical movement of the jaws that prepares a food bolus for swallowing (Lund, 1991, Lund and Kolta, 2006, Nakamura and Katakura, 1995, Yamada et al., 2005). It is a complex neuromuscular task that involves the coordinated actions of the tongue, facial and masticatory muscles that results in the food being placed between the teeth, cut and ground up into smaller pieces and mixed with saliva to produce a food bolus ready for swallowing (Lund, 1991, Ferrario and Sforza, 1996, Sessle et al., 2013). The intrinsic rhythmical pattern of mastication is controlled by its central pattern generator located in the brainstem (Lund, 1991, Nakamura and Katakura, 1995, Yamada et al., 2005, Avivi-Arber et al., 2011, Sessle et al., 2013) and is modulated by feedback from sensory inputs from the tongue and oral mucosa as well as periodontal structures (Manns et al., 1991, Lund et al., 1998, Hiiemae and Palmer, 2001, Avivi-Arber et al., 2011, Sessle et al., 2013). This sensory feedback provides information about the size, shape, texture and hardness of the food bolus as well as its position within the mouth (Foster et al., 2006, Langenbach and van Eijden, 2001, Peyron et al., 2004, Peyron et al., 2002, Woda et al., 2006a, Woda et al., 2006b). By utilising the information provided by this sensory feedback adaptations can be made to the chewing movement pattern to accommodate the continuously changing intrinsic characteristics of the food bolus and its position within the mouth such that mastication can be completed successfully and the bolus prepared for deglutition (Foster et al., 2006, Langenbach and van Eijden, 2001, Peyron et al., 2004, Peyron et al., 2002, Woda et al., 2006a, Woda et al., 2006b).
In mammals there is great diversity in jaw motor behaviour (Langenbach and van Eijden, 2001, Weijs, 1994) which may be a reflection of the diversity of diet (Weijs, 1994), occlusal forces (Langenbach and van Eijden, 2001, Rossignol et al., 1988), muscle morphology and/or the anatomy of the TMJ and occlusal contact patterns (Langenbach and van Eijden, 2001) that is seen between species. Despite these differences a basic uniformity in the patterns of jaw motor behaviour has been suggested (Hiemae, 1978, Langenbach and van Eijden, 2001) which has been refined into a classification of five masticatory muscle activity patterns based on the function of the muscles of mastication during chewing (Weijs, 1994). This classification, based on EMG recordings, defines three functional muscle groups related to the direction of tendon pull that are active during different times of the closing phase of mastication thereby describing the cooperative action of the jaw closers (Weijs, 1994). The first functional group is active during the start of the fast closing phase and includes the symmetrical vertical closers of the deep head of masseter and the anterior temporalis (Langenbach and van Eijden, 2001, Weijs, 1994). The second functional group moves the mandible to the working side (ipsilateral) during the middle to late fast closing phase and includes a triplet of muscles, namely, ipsilateral temporalis and contralateral masseter and medial pterygoid (Triplet I, Langenbach and van Eijden, 2001, Weijs, 1994). The third functional group moves the mandible to the balancing side (contralateral) during the power stroke and includes another triplet of muscles, namely, contralateral temporalis and ipsilateral masseter and medial pterygoid (Triplet II, Langenbach and van Eijden, 2001, Weijs, 1994). This classification of the jaw closers enabled the diverse masticatory patterns among different species to be classified into five patterns according to the timing of the cooperative activity of the muscles within these three functional groups (Figure 1.3, Langenbach and van Eijden, 2001, Weijs, 1994).
Figure 1.3: A classification of five masticatory muscle activity patterns in mammals based on the function of the muscles of mastication during the fast closing (FC), power stroke (PS) and initial opening (O₁₂) phases of chewing. Three functional muscle groups related to the direction of tendon pull are shown from top to bottom: the symmetrical vertical closers of the deep head of masseter and the anterior temporalis (ZYMA; open bar); Triplet I, the working side (ws) temporalis (TEM; shaded bar) and balancing side (bs) masseter (MAS; diagonal hashed bar) and medial pterygoid (MPT; dotted bar); and Triplet II, balancing side temporalis and working side masseter and medial pterygoid. In rodents and transverse grinders the working side lateral pterygoid activity is also indicated (asterisk filled bar). Bars with dashed lines indicate very low level or inconsistent muscle activity (From Weijs, 1994).
Another theory that could be applied to help describe the organisation of the masticatory sensorimotor system is the motoneurone task group hypothesis (Loeb, 1985). Task groups consist of muscle fibres, extrafusal and intrafusal motoneurones along with their associated proprioceptive afferents and act in unison in the performance of specific tasks (Loeb, 1985). These groups of motor units and their feedback mechanism(s) not only reside in a single area of individual muscle but may in fact appear in many regions across multiple muscles (Loeb, 1985, Murray and Peck, 2007). In this configuration, the motor system is capable of utilising many different types of control principles and tailor the recruitment of the muscles within the task group with their respective proprioceptive feedback in order to produce a specific movement task (Loeb, 1985).

This notion of motoneurone task groups (Loeb, 1985) may be consistent with the actions of the functional groups of the jaw closer muscles that have also been described (Langenbach and van Eijden, 2001, Weijs, 1994) and may contribute to understanding the mechanisms of masticatory function. The complex and diverse architecture of the muscles of mastication allows for multiple options for tendon pull (Hannam and McMillan, 1994, Schumacher, 1961). This results in an inherent degree of mechanical redundancy in the jaw sensorimotor system that provides the potential for a large number of possible patterns of muscle activation that can be utilised to perform jaw movements (Lobbezoo et al., 2004, Van Eijden et al., 1990, van Eijden and Turkawski, 2001). In light of this functional heterogeneity and complexity within the masticatory system (Hannam and McMillan, 1994, van Eijden and Turkawski, 2001), in producing jaw movement tasks it is plausible that the motor command may preferentially activate those motor units that have the optimal biomechanical capacity to produce that specific task rather than activating specific muscles (Murray and Peck, 2007, Sessle et al., 2013). Furthermore, the distribution of this motor
unit activity may be reflected in the preferential activation of those motor units with the best mechanical advantage and lowest metabolic cost to produce the desired specific jaw movement task (Murray and Peck, 2007, De Troyer et al., 2005, Gandevia et al., 2006, Sessle et al., 2013). This may provide an explanation for the classification of mandibular movements into eight different patterns in asymptomatic individuals, with the predominant pattern having a teardrop shape (Proschel, 1987). Therefore, despite the potential availability of a large number of possible patterns of muscle activation (Lobbezoo et al., 2004, Van Eijden et al., 1990, van Eijden and Turkawski, 2001) and even though mandibular movement patterns continuously adapt to the changes in the food bolus during mastication (Foster et al., 2006, Langenbach and van Eijden, 2001, Peyron et al., 2004, Peyron et al., 2002, Woda et al., 2006a, Woda et al., 2006b), it is plausible that the masticatory system may commonly only be required to use up to eight masticatory movement patterns (Proschel, 1987) to complete a given chewing task. This confinement of motor patterns may be a reflection of the recruitment of motoneurone task groups (Loeb, 1985, Murray and Peck, 2007) via the described muscle functional groups and triplets (Langenbach and van Eijden, 2001, Weijs, 1994) and thereby contributes to the motor control of mastication.

**Orofacial Motor Control**

Transcranial magnetic stimulation has been used in the orofacial region to assess corticomotor control of the tongue muscles (Svensson et al., 2003, Svensson et al., 2006, Boudreau et al., 2007) as well as the muscles of mastication (Nordstrom, 2007) in healthy, dentate (Pearce et al., 2003, Goiato et al., 2010) and edentulous individuals (Goiato et al., 2010) as well as in patients with facial palsy (Türk et al., 1994), sleep bruxism (Gastaldo et al., 2006) and TMD (Cruccu et al., 1997, Goiato et al., 2010). Transcranial magnetic
stimulation has also been used to demonstrate exercise induced changes in the orofacial region. Neuroplastic changes have been reported in the tongue primary motor cortex following periods of 15 minutes (Boudreau et al., 2007), one hour (Svensson et al., 2006) and one week (Svensson et al., 2003) training of a novel tongue protrusion task. These studies have reported significant enhancement of the motor evoked potentials recorded from the tongue musculature and decreases in the tongue primary motor cortex thresholds with concomitant improvements in performance of the novel tongue protrusion task (Boudreau et al., 2007, Svensson et al., 2006, Svensson et al., 2003). These changes were also accompanied by significant increases in the size of the cortical motor maps of the tongue primary motor cortex (Svensson et al., 2003, Svensson et al., 2006) as well as a lateral and anterior shift in the centre of gravity coordinates of the cortical map at the seven days follow-up after one hour of tongue protrusion training (Svensson et al., 2006). More specifically, isotonic resistance exercise has also been shown to produce significant differences in the temporal characteristics of EMG activation of the muscles of mastication compared to a Control group during a standardised jaw movement task (Wirianski, 2009, Wirianski et al., 2014).

These findings in the orofacial region have similarities with recent LBP studies that have also utilised transcranial magnetic stimulation (Tsao et al., 2010, Tsao et al., 2008). These LBP studies reported reorganisation of the cortical representation of trunk muscles in people with recurrent LBP which was associated with postural control deficits of delayed onset of EMG activity (Tsao et al., 2008). Furthermore these neuroplastic changes were demonstrated to be reversible following the application of a specific training programme (Tsao et al., 2010).
Statement of the Problem

Movements of the jaw during chewing are produced by the coordinated action of the muscles of mastication under centrally mediated motor control (Langenbach and van Eijden, 2001, Lund and Enomoto, 1988). Deviation from the normal movement patterns of the jaw have been associated with TMD (Bell, 1983, Laskin, 1969, Schiffman et al., 2014, Solberg, 1983). Therapeutic exercises are often prescribed in the management of TMD and recent systematic reviews have reported that they may be of benefit in its management (List and Axelsson, 2010, McNeely et al., 2006, Medlicott and Harris, 2006) however there is a paucity of literature regarding their mechanism of action. On the other hand, the prescription of specific exercise regimens aimed at restoring normal neuromuscular control has been shown to be effective in the treatment of many other musculoskeletal conditions affecting the knee (Cowan et al., 2002, Cowan et al., 2001, Cowan et al., 2003), shoulder (Ginn and Cohen, 2004, Ginn and Cohen, 2005, Ginn et al., 1997, Worsley et al., 2013), cervical spine (Jull et al., 2002, O'Leary et al., 2007, Ylinen et al., 2007, Ylinen et al., 2003) and lumbar spine (O'Sullivan et al., 1998, O'Sullivan et al., 1997, Tsao et al., 2010).

Moreover, EMG and cortical changes have been demonstrated in patients presenting with patellofemoral pain (Cowan et al., 2002, Cowan et al., 2001, Cowan et al., 2003), shoulder pain and impingement (Wadsworth and Bullock-Saxton, 1997, McMahon et al., 1996, Cools et al., 2003, Worsley et al., 2013), cervical spine pain (Jull et al., 2004, Jull et al., 2009, Falla et al., 2004a, Falla et al., 2004b) and LBP (Hodges and Richardson, 1996, Tsao and Hodges, 2007, Tsao and Hodges, 2008). These cortical changes have also been shown to be reversible and associated with improvements in motor control and reduction of symptoms in patients with chronic LBP following the completion of a specific exercise programme (Tsao et al., 2010).
Recently, in asymptomatic individuals, EMG changes have been reported in the muscles of mastication during standardised jaw movements following the application of a specific resistance exercise task (Wirianski, 2009, Wirianski et al., 2014). Neuroplastic changes have also been reported in the tongue primary motor cortex following training of a novel tongue protrusion task (Boudreau et al., 2007, Svensson et al., 2006, Svensson et al., 2003). These findings were demonstrated during novel jaw and tongue movement tasks and thus may not be indicative of the changes that may occur during the normal function of the masticatory system. Nevertheless, these findings suggest the possibility that similar motor control changes could occur in the somatosensory cortex associated with the orofacial region, and in particular the muscles of mastication, as those that have been demonstrated in patients with patellofemoral pain, shoulder pain and impingement, cervical spine pain and LBP, as outlined above.

Aims and Hypotheses

This thesis attempts to investigate the effects of isometric resistance exercise training on the movement patterns of the jaw and the EMG activation patterns of the muscles of mastication during the functional task of chewing. In so doing, this thesis will document mandibular movement patterns along with the EMG activity patterns of the muscles of mastication in normal, asymptomatic volunteers. The effect of isometric resistance exercise on the masticatory movement and EMG patterns will be investigated in both the immediate timeframe and after two weeks of a home-based isometric resistance exercise programme. Hence, the aims of this thesis are:
1. To determine if the application of a specific resistance exercise task in asymptomatic individuals will result in the jaw muscles being used differently in order to produce different jaw movement patterns during chewing; and,

2. If changes in jaw movement patterns occur following the application of a specific resistance exercise task then the second aim is to define the EMG activity patterns in the muscles of mastication that may accompany any changes in jaw movement patterns.

Two studies, utilising different data collection and analysis techniques, are described in an attempt to address these aims. Each study has a specific hypothesis to be tested as stated below:

Study 1. That the application of an isometric resistance exercise task against lateral jaw movement will result in changes in the masticatory movement path during the functional task of chewing cooked rice (Chapter 2). The alternate null hypothesis would therefore be that jaw movement patterns would remain unchanged following the application of the isometric resistance exercise task.

Study 2. That the application of an isometric resistance jaw exercise task against lateral jaw movement will result in a change in EMG activity in the jaw muscles and more reproducible activity patterns. Furthermore, in some of the tested EMG variables, there will be significant changes in the Control group between the testing sessions while in the Exercise group the values of these variables will be maintained at baseline levels (Chapter 3).
Clinical Relevance

Some patients that present with TMD also demonstrate jaw movements that deviate from normal masticatory patterns (Bell, 1983, Laskin, 1969, Schiffman et al., 2014, Solberg, 1983). These altered jaw movement patterns may be associated with reversible neuroplastic changes that may affect the motor control of the muscles of mastication and interfere with normal masticatory function. Understanding the mechanisms that underlie the motor control deficits associated with altered jaw movement patterns may provide effective strategies for the management of TMD.
Chapter 2

This chapter presents the first of two studies described in this thesis. It reports the immediate effects of an isometric resistance exercise task against lateral jaw movement on the movement trajectories of the jaw during free chewing of cooked rice.

This study was conducted under the supervision of Professor Yoshinori Hattori at the Division of Aging and Geriatric Dentistry, Department of Oral Function and Morphology, Graduate School of Dentistry, Tohoku University, Sendai, Japan. It was supervised in collaboration with Professor Christopher C. Peck and Professor Gregory M. Murray from the Jaw Function and Orofacial Pain Research Unit, Westmead Centre for Oral Health, Faculty of Dentistry, The University of Sydney.

The completion of this study was made possible by the generous assistance of Dr Yuko Komine, Dr Yasue Tanaka, Dr Yohei Igari and Dr Mai Sato from the Division of Aging and Geriatric Dentistry, Department of Oral Function and Morphology, Graduate School of Dentistry, Tohoku University, Sendai, Japan. The author was supported by the 2010 Prime Minister’s Australia Asia Endeavour Award (Postgraduate Outgoing) from the Australian Government Department of Education and Training.
Isometric resistance jaw exercise alters jaw movement patterns during chewing.

Abstract

Therapeutic exercise is a common treatment modality used in the management of temporomandibular disorders, although the mechanism of action is not known. To test the hypothesis that exercise modifies jaw movement, the effects of an isometric resistance exercise task applied to the jaw muscles on masticatory movement of the human jaw were investigated using principal component analysis. Fourteen asymptomatic volunteer participants were randomly allocated to either a Control group (n = 7) or an Exercise group (n = 7). Jaw movement data were collected from each participant during five trials of chewing cooked rice until swallowing before and after an “exercise condition”. During the “exercise condition” participants in the Exercise group completed an isometric resistance exercise task at 30% of maximum voluntary contraction against right lateral jaw movements for 15 minutes while participants in the Control group sat quietly for 15 minutes and did not complete the exercise task. Five principal components of a total of 123 principal components explained 92.1% of the total variance in the chewing cycles of all participants. Furthermore, in asymptomatic individuals, isometric resistance exercise applied against a lateral jaw movement resulted in masticatory movement paths that were significantly (p < 0.001) more horizontally orientated in the coronal plane and more protruded in the sagittal plane. Applying the findings from this study to an appropriate patient population warrants further investigation in a clinical setting utilising appropriately matched controls. This may help to elucidate the mechanisms whereby therapeutic exercise affects the muscles of mastication and thereby contributes to the effectiveness of that therapeutic exercise in the management of temporomandibular disorders.
Introduction

Therapeutic exercise is a common treatment modality used in the management of temporomandibular disorders (TMD, Glass et al., 1993, Michelotti et al., 2005). Several systematic reviews have reported that therapeutic exercise may be effective in the treatment of TMD (List and Axelsson, 2010, McNeely et al., 2006, Medlicott and Harris, 2006). However, it is difficult to determine the contribution of the prescribed exercises to symptom resolution, partly because the mechanisms whereby therapeutic exercise affects the muscles of mastication remain poorly understood. This paucity of information related to the mechanisms of action of therapeutic exercise on jaw muscles may also contribute to the difficulty in assessing the effectiveness of therapeutic exercise in the management of TMD. An understanding of the mechanism(s) of action of treatments helps answer why, how, and when such treatments should be applied. Furthermore, standardised treatment regimens enable a more consistent approach to clinical research which in turn facilitates the development of more effective management strategies to TMD sufferers.

Recently, in asymptomatic individuals, electromyographic (EMG) changes have been reported in the muscles of mastication during standardised jaw movements following the application of a specific resistance exercise task (Wirianski, 2009, Wirianski et al., 2014). These findings suggested a change in the pattern of recruitment of the muscles in order to maintain the vertical position of the mandible during the completion of the standardised lateral jaw movement task (Wirianski, 2009, Wirianski et al., 2014). The prescription of therapeutic exercise has also been shown to be associated with restoration of normal EMG activity patterns and kinematics during functional tasks across a range of joints in the body (Cowan et al., 2003, Ginn and Cohen, 2005, O'Leary et al., 2007, Tsao et al., 2010). It is plausible therefore that the application of a specific resistance exercise task to the muscles
of mastication may also affect kinematic changes that may result in changes in mandibular movement paths during the functional task of mastication.

Investigating the effects of therapeutic exercise on masticatory motion paths may help to elucidate the mechanisms of action of this modality, viz., predictable alteration of jaw movement and consequently jaw muscle and articular loading. One method of analysing the movement path of the temporomandibular joint (TMJ), and in particular, the variations in the cycle paths during human mastication, combines the use of elliptic Fourier descriptors (EFD, Kuhl and Giardina, 1982) and principal component analysis (PCA, Hattori et al., 2010). This method has been used to quantify movement trajectories during the chewing of different types of food (Hattori et al., 2010). In their study it was found that the linear combination of only three independent movement variations accounted for an average of 93% of the total variations in the masticatory cycle paths in eight healthy male participants when chewing gum and gummy candy (Hattori et al., 2010). Since the controlled degrees of freedom of the masticatory movements were much less than the total number of muscles involved in the production and control of the movement, their results also suggested the existence of muscle synergies in the production and control of masticatory movements (Hattori et al., 2010). Furthermore, the three extracted variations were found to be similar among the eight participants suggesting to the authors that masticatory motion was controlled by identical strategies amongst the participants (Hattori et al., 2010).

This technique, in conjunction with cluster analysis, has also been used to classify chewing cycles (Igari et al., 2012). In their study, ten participants chewed cooked rice until swallowing and the chewing sequences were divided equally into three stages based on the
number of chewing cycles (Igari et al., 2012). It was found that the appearance ratios of the chewing cycles were different between the stages indicating that the groups of chewing cycles that differed in the shape of their movement trajectories may also differ in function (Igari et al., 2012).

The current study investigated the effects of isometric resistance exercises applied to the human jaw on masticatory movement patterns by utilising the previously described method of combining EFD and PCA (Hattori et al., 2010). We hypothesised that the application of an isometric resistance exercise task against lateral jaw movement would result in changes in the masticatory movement path during the functional task of chewing cooked rice. An improved understanding of the mechanisms of therapeutic exercise may simplify and improve the appropriate selection of effective treatment modalities through a systematic, evidence-based approach.
Methods

Participants
Fourteen asymptomatic adult volunteers (7 females, 7 males) participated in this study. Participants with a past or current history of pain or dysfunction (e.g. TMD) in and around the orofacial region were excluded. Participants provided informed consent to participate in the study. All experimental procedures were approved by the Tohoku University Human Research Ethics Committee (No: 22-30; Appendix 1).

Each participant attended one data collection session conducted during the Research Summer School of the Japanese Stomatognathic Society, held at the Fujiyoshida Campus, Showa University, Yamanashi, Japan. Participants were randomly allocated into one of two groups: a Control group (n = 7); or an Exercise group (n = 7). During the data collection session, each participant quietly sat upright in a comfortable chair. Jaw movement data were collected on two separate occasions during the data collection session. First, baseline jaw movement data were collected during five trials of chewing a 10 gm sample of cooked rice (Sato no Gohan, Sato Shokuhin Inc., Niigata, Japan). Jaw movement data collection was repeated a second time approximately 15 minutes after the first data collection occasion. In between the two occasions of data collection participants in the Control group sat quietly, while participants in the Exercise group performed a simple isometric resistance exercise task against right sided jaw movement.

Jaw movement
Movement of the jaw was recorded simultaneously in three orthogonal planes (aligned parallel to the clinically-determined Frankfort horizontal plane (FHP) and mid-sagittal plane) during the chewing of a 10 gm sample of cooked rice. Each participant was asked to chew the sample of rice on their right side until swallowing (one chewing trial) and asked
to repeat this chewing trial a further four times during each data collection occasion. This resulted in a total of five chewing trials during the baseline data collection occasion and a further five chewing trials after the 15 minutes interval in between.

Jaw movement data were collected using an electromagnetic jaw tracking system (K7 Cranio-Mandibular Evaluation System, Myotronics-Noromed Inc., Tukwila, WA, USA). A lightweight headgear was placed on the participant’s head. This headgear suspended two parallel arrays of four sensors each, one on the left and the other on the right side of the face (Figure 2.1, Panel A). The horizontal axis of these sensor arrays were aligned parallel to the FHP. The sensor arrays were maintained in a constant position on the skull throughout the data collection by a frame made of aluminium tubing connected to eyeglass frames that were fixed securely to the participant’s head with a Velcro strap (Figure 2.1, Panel A). The sensor arrays detect alterations of a magnetic field. A magnet (12.7 mm long x 6.4 mm wide x 3.2 mm thick) with high magnetic field strength was fixed with double sided tape to the participant’s labial vestibule just below the mandibular incisors and centred over the mid-incisor point (MIPT). The movement of the magnet was then tracked by the sensor arrays as the participant moved their mandible during chewing of the bolus of cooked rice. The generated jaw movement data represented the spatial position of the mandibular MIPT relative to the head’s reference planes (outlined above). The linear analysis of chewing movements with this system has been demonstrated to be accurate without major distortion, especially within a vertical range of motion of 40 mm (Balkhi et al., 1991). Furthermore, good reproducibility has also been demonstrated under rigorously controlled recording conditions (Baba et al., 2000, De Souza et al., 2009).
Figure 2.1: Jaw tracking and force measurement equipment used in this study. Panel A: Anterior view of a participant showing the position of the K7 Cranio-Mandibular Evaluation System and the acrylic faceplate containing the miniature load cell. Participants held the acrylic faceplate with their right hand against the skin such that the miniature load cell was positioned over the right mandible adjacent to the canine root tip. The white arrow shows the direction of the force generated by the muscles of mastication during the maximum voluntary contraction (MVC) and resistance exercise tasks. Participants held the mandible stationary in the neutral position by their right hand. Panel B: Acrylic faceplate showing the position of the miniature load cell. The anterior surface of the acrylic faceplate was placed against the skin over the right mandible adjacent to the canine root tip with the lower border of the mandible resting on the mandibular positioning ledge.

The K7 was connected to a data logger (NR-500, Keyence, Osaka, Japan) through an analogue to digital converter (sampling rate 62.5 Hz, NR-HA08 Keyence, Osaka, Japan). The data logger saved the raw jaw movement data as an individual text file for each chewing sequence. Each individual text file contained the three dimensional coordinates representing the spatial position of the trajectory of the mandibular MIPT relative to the skull during the chewing sequence. This text file was automatically loaded into a custom-
made Excel Visual Basic for Applications (VBA) macro (2010, version 14, Microsoft Corp., Redmond, WA, USA) which processed each chewing sequence into separate chewing cycles and saved the data for each chewing cycle into individual text files for further offline processing and analysis.

**Maximum voluntary contraction (MVC)**

The measurement of the maximum voluntary contraction (MVC) of a muscle or muscle group is widely used to assess the maximum isometric force that can be generated by both single and multi-joint muscles (Wilson and Murphy, 1996). In the orofacial region MVC has generally been used to measure the maximum bite force of the muscles of mastication and/or their associated EMG activity during maximum teeth clenching in asymptomatic individuals (Hellmann et al., 2011, Uchida et al., 2008, Tartaglia et al., 2011, Forrester et al., 2010) as well as in patients with TMD (Tartaglia et al., 2011, Tartaglia et al., 2008, Ferrario et al., 2007). No studies have been found that investigated the reliability of the measurement of MVC in the muscles of mastication specifically, however it has been reported to be a reliable measure of isometric strength in the neck muscles (Asghar et al., 2003, Marjan et al., 2008, Almosnino et al., 2010, Salmon et al., 2015), neck and shoulder muscles (Tornøe et al., 2013), shoulder muscles (Fischer et al., 2011), quadriceps femoris (Morton et al., 2005, Blacker et al., 2013) and for measuring grip strength of the hand (Demura et al., 2001). By virtue of its widespread use and that it has been reported to be a reliable measure of the assessment of isometric muscle performance, MVC was chosen to measure the maximum force produced by the muscles of mastication as described below.

The maximum force exerted during an isometric contraction of the muscles of mastication in the direction of right lateral jaw movement with the mandible in the neutral position was determined in order to standardise the applied force during the resistance exercise training.
section (Exercise group). Force data were collected with a miniature load cell (L6 miniature load cell and an L6 - 20K compensation sensor pipe extension cord, SSK, Tokyo, Japan) connected to a signal conditioning unit (6M91, Sanei-Sokki, Tokyo, Japan). The load cell was placed in an acrylic faceplate (Figure 2.1, Panel B) and held against the skin over the right mandible adjacent to the canine root tip by the participant with their right hand (Figure 2.1, Panel A). The output of the signal conditioning unit was connected to an analogue voltmeter which measured the amplified output voltage of the load cell. When instructed, participants in the Exercise group applied an isometric resistance force against a right lateral jaw movement, with the mandible held stationary in the rest position of the mandible, as hard as they could without pain for approximately five seconds (Figure 2.1, Panel A). The displayed voltage provided the participant with visual feedback of the magnitude of the force they exerted during each isometric MVC trial. Participants were also given verbal encouragement during each MVC trial from the experimenter (AW) to push as hard as they could against the load cell without causing pain or discomfort. This was completed three times and the highest voltage reading on the analogue voltmeter was used as a measure of the participant’s MVC against right sided isometric resistance. Each participant’s exercise training isometric force was then calculated at 30% MVC.

**Isometric resistance exercise training task**

The isometric resistance exercise task consisted of a total of five sets of 10 repetitions of isometric resistance exercise against right lateral jaw movement at 30% MVC. A mark was placed on the analogue voltmeter at the voltage that represented the calculated 30% MVC level which provided the participant with feedback of the force they exerted during the exercise task. During each repetition, participants were instructed to apply and maintain the training force for approximately 10 s without moving the jaw from its neutral position. The
training force was applied to the right mandible in the same way as described for the MVC trials above. Isometric resistance against right lateral movement was selected in order to train the contralateral (left) lateral pterygoid which is a prime agonist for opening and lateral jaw movement phases of mastication. Participants in the Exercise group performed 10 repetitions of this isometric task with 5 s to 10 s rest between each repetition. At the completion of the 10 repetitions (one set), participants were instructed to rest quietly for 20 s to 30 s. The set of 10 isometric muscle contractions was then repeated a further four times with a 20 s to 30 s rest between each set. The complete exercise task took approximately 15 minutes to complete. During the isometric resistance exercise task participants were monitored for signs of pain and fatigue as well as regularly asked if they had symptoms of pain and/or fatigue in and around their jaw, the jaw muscles or the orofacial region. If a participant reported any symptoms of pain and/or fatigue in these areas they were allowed to rest until these symptoms subsided before continuing with any remaining exercises or the next movement recording occasion. On completion of the exercise task, jaw movement data were collected during five trials of chewing a 10 gm sample of cooked rice. Participants in the Control group did not undertake the exercise task. Instead they sat quietly for 15 minutes, at the end of which, jaw movement data were also collected during five trials of chewing a 10 gm sample of cooked rice.

**Data Analysis**

Each chewing cycle path was described using the three-dimensional coordinates along the trajectory of the mandibular MIPT at time-points which divided the opening and closing phases of the cycle into equal intervals: 20 data points were along the opening phase of the chewing cycle; 20 data points were along the closing phase; and one data point at the transition point between opening and closing. This procedure made it possible to describe
the trajectory of any single chewing cycle with a total of 123 variables regardless of the duration of the chewing cycle. The variation in the mandibular motion paths during mastication of the bolus of cooked rice was evaluated using PCA on a variance-covariance matrix (Hattori et al., 2010). The principal components (PCs) that accounted for at least 90% of the variation in the mandibular movement paths were selected for further analysis.

Multivariate analysis of variance (MANOVA) was used to determine the within-subjects and between-groups differences of the selected PCs before and after the application of the isometric resistance exercise task with statistical significance set at \( p < 0.05 \). PASW Statistics (Version 17, SPSS Inc., Chicago, IL, USA) was used to perform the PCA and MANOVA.

The mean values of the PCs were zero. For the purpose of visualising the contribution of a particular PC to differences in mandibular motion, the mandibular motion paths were reconstructed using the inverse eigenvector matrix and by altering the value of the selected single PC to plus or minus twice the standard deviation of the PC with the values of other PCs maintained at zero. According to the attribute of the eigenvector matrix, the inverse eigenvector matrix is equal to the transverse matrix of the original eigenvector matrix.
Results

Chewing Trials

Fourteen participants completed the study. A total of 3825 chewing cycles were analysed in the PCA (Table 2.1). Data were recorded for an average of 25.8 (SD 9.95, range: 9 – 46) chewing cycles in the Control group and 28.8 (SD 10.23, range: 14 – 53) chewing cycles in the Exercise group (Table 2.1).

<table>
<thead>
<tr>
<th>Statistic</th>
<th>Control Group</th>
<th>Exercise Group</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>895</td>
<td>914</td>
<td>3825</td>
</tr>
<tr>
<td>Mean</td>
<td>25.6</td>
<td>26.1</td>
<td>27.3</td>
</tr>
<tr>
<td>SD</td>
<td>9.62</td>
<td>10.37</td>
<td>10.17</td>
</tr>
<tr>
<td>Min</td>
<td>9</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>Max</td>
<td>47</td>
<td>46</td>
<td>53</td>
</tr>
</tbody>
</table>

Table 2.1: Summary of the number of chewing cycles (n) in each experimental group that were analysed using principal component analysis (PCA). Also shown are the mean number of chewing cycles and standard deviation (SD), as well as the minimum (Min) and maximum (Max) number of chewing cycles in each group.
There were no significant differences in the mean number of chewing cycles per trial between the Control group and the Exercise group (Table 2.1 and Figure 2.2; $F_{(1, 12)} = 100.823$, $p < 0.001$).

![Graph showing mean number of chewing cycles per trial for Control and Exercise groups and all participants combined](image)

Figure 2.2: Mean number of chewing cycles per trial in both the Control group (Left blue columns) and the Exercise group (Centre green columns) as well as for all participants combined (Right red column). Error bars indicate the standard deviation (SD). (Pre-Ex: pre-exercise condition; Post-Ex: post-exercise condition).
Principal Component Analysis (PCA)

Principal component analysis of the EFD revealed that five PCs of a total of 123 PCs explained 92.1% of the total variance in the chewing cycles of all participants (Table 2.2).

### Total Variance Explained

<table>
<thead>
<tr>
<th>Principal Component (PC)</th>
<th>Initial Eigenvalues*</th>
<th>Extraction Sums of Squared Loadings</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>% of Variance</td>
</tr>
<tr>
<td>Raw</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PC1</td>
<td>413.02</td>
<td>51.7</td>
</tr>
<tr>
<td>PC2</td>
<td>158.82</td>
<td>19.9</td>
</tr>
<tr>
<td>PC3</td>
<td>87.24</td>
<td>10.9</td>
</tr>
<tr>
<td>PC4</td>
<td>51.80</td>
<td>6.5</td>
</tr>
<tr>
<td>PC5</td>
<td>24.75</td>
<td>3.1</td>
</tr>
</tbody>
</table>

Extraction Method: Principal Component Analysis.

a. When analysing a covariance matrix, the initial eigenvalues are the same across the raw and rescaled solution.

Table 2.2: Extract from the PASW Principal Component Analysis showing the Total Variance explained by the first five principal components (PCs) of the chewing cycles of all 14 participants (taken from a total of 123 components). Based on the initial eigenvalues and the extraction sums of squared loadings, the total variance in the chewing cycles that was explained by the first five PCs was 51.7% for PC1, 19.9% for PC2, 10.9% for PC3, 6.5% for PC4 and 3.1% for PC5. The Cumulative % column shows that the first five PCs accounted for 92.1% of the total variance in the chewing cycles.
The scree plot of this PCA confirms the point of inflexion of the curve to be at the fifth PC (Figure 2.3).

![Scree Plot]

**Figure 2.3:** Scree plot of the principal component analysis (PCA) confirming that the fifth PC (PC5) is at the elbow or inflexion point (arrow).

**Reconstruction of Jaw Displacements**

The three-dimensional coordinates along the trajectory of the mandibular MIPT of the first five PCs were plotted for all 14 participants (Figure 2.4). The mean trajectories were plotted in the coronal plane (Top row, Figure 2.4, solid black lines and solid black squares) and the sagittal plane (Bottom row, Figure 2.4, solid black lines and solid black squares) along with the respective trajectories wherein the corresponding PC value was altered to plus twice the standard deviation (+ 2SD; Figure 2.4, dashed red lines and solid red circles) or minus twice the standard deviation (- 2SD; Figure 2.4, short dashed blue lines and solid blue triangles). These reconstructed trajectory curves provide a qualitative and quantitative representation of the contribution of each PC to the movement path of the jaw during the chewing cycles and are briefly described below.
Figure 2.4: Graphical representations of the displacement of the jaw for each of the first five principal components (PCs) that explained at least 90% of the variance of the chewing cycles in all 14 participants in the coronal plane (Top row) and the sagittal plane (Bottom row). For each PC, solid black lines and solid black squares represent the mean movement displacement, dashed red lines and solid red circles represent the mean displacement + 2SD and short dashed blue lines and solid blue triangles represent the mean displacement - 2SD.
Principal component 1 (PC1) accounted for 51.7% of the variance of the chewing cycles (Table 2.2), and contributed to the magnitude of the vertical displacement of the MIPT in both the coronal and sagittal planes (Figure 2.4). In the coronal plane, PC1 also contributed to a change in the width of the trajectory. The shape of the trajectory appears relatively similar between the mean trajectory and the +/- 2SD trajectories. The change in length of the displacement appears to elongate and widen the elliptical shape of the trajectory when moving from the mean + 2SD trajectory (dashed red lines and solid red circles) to the mean trajectory (solid black lines and solid black squares) and thence to the mean - 2SD trajectory (short dashed blue lines and solid blue triangles), thus showing an increase in length and width with a reduction in the PC score.

Principal component 2 (PC2) accounted for a further 19.9% of the variance of the chewing cycles (Table 2.2), and contributed to the angle of the trajectory in both the coronal and sagittal planes (Figure 2.4). The change in angulation of the displacement became more vertical in the coronal plane and less protruded in the sagittal plane with a reduction in the PC score.

Principal component 3 (PC3) accounted for a further 10.9% of the variance of the chewing cycles (Table 2.2), and contributed to angular and length changes of the chewing trajectories in both the coronal and sagittal planes as well as shape changes in the coronal plane (Figure 2.4). The change in angulation of the displacement became more vertical in the coronal plane and more protruded in the sagittal plane with a reduction in the PC score. As the movement trajectories became more vertical they also became longer in both the sagittal and coronal planes. Furthermore, the shape of the trajectories became more
elongated and narrower in the coronal plane as the trajectory became more vertically orientated.

Principal component 4 (PC4) and PC5 accounted for a further 6.5% and 3.1% of the variance of the chewing cycles, respectively (Table 2.2). These 2 PCs contributed primarily to shape changes in the movement trajectories in the coronal plane (Figure 2.4). There were no angular changes in the sagittal plane, with both PCs showing similar angles of movement displacement. For PC4 the shape of the trajectory became narrowed and slightly shorter with a reduction in the PC score. Conversely, for PC5 the shape of the trajectory became rounder and slightly less elongated with a reduction in the PC score.

In order to visualise the effects of the isometric resistance exercise task the three-dimensional coordinates along the trajectory of the mandibular MIPT were also plotted for the three PCs that showed significant changes in the seven Control group participants (Figure 2.5) as well as in the seven Exercise group participants (Figure 2.6). The significant changes in the trajectory of the mandibular MIPT following the application of the isometric resistance exercise task are described below along with the multivariate and univariate analysis of variance (ANOVA) results.
Figure 2.5: Graphical representations of the displacement of the jaw of the 3 principal components (PCs) that showed significant changes of the 5 PCs that explained at least 90% of the variance of the chewing cycles in the 7 Control group participants before and after the “Exercise” condition. Jaw displacements are depicted in the coronal plane (Top row) and the sagittal plane (Bottom row). For each PC, solid black lines and solid black squares represent the mean movement displacement during the pre-exercise condition, dashed red lines and solid red circles represent the mean displacement + 2SD during the pre-exercise condition, short dashed blue lines and solid blue triangles represent the mean displacement - 2SD during the pre-exercise condition, dotted pink lines and open pink squares represent the mean movement displacement of each PC in the post-exercise condition, dash-dot green lines and open green circles represent the mean displacement + 2SD for each PC in the post-exercise condition and short dash-dot purple lines and open purple triangles represent the mean displacement - 2SD for each PC in the post-exercise condition.
Figure 2.6: Graphical representations of the displacement of the jaw of the 3 principal components (PCs) that showed significant changes of the 5 PCs that explained at least 90% of the variance of the chewing cycles in the 7 Exercise group participants before and after the “Exercise” condition. Jaw displacements are depicted in the coronal plane (Top row) and the sagittal plane (Bottom row). For each PC, solid black lines and solid black squares represent the mean movement displacement during the pre-exercise condition, dashed red lines and solid red circles represent the mean displacement + 2SD during the pre-exercise condition, dotted pink lines and open pink squares represent the mean movement displacement of each PC in the post-exercise condition and dash-dot green lines and open green circles represent the mean displacement + 2SD for each PC in the post-exercise condition.
**Multivariate ANOVA of the Principal Components**

The MANOVA conducted on the PC scores found the following significant differences:

1. There was a significant difference in the PC scores between the Pre-exercise and Post-exercise conditions ($F(5, 3817) = 4.896, p < 0.001$) for all the test statistics;

2. There was a significant difference between the PC scores in the Control group compared to the PC scores in the Exercise group ($F(5, 3817) = 176.148, p < 0.001$), and;

3. There was a significant difference in the interaction between the Pre-exercise and Post-exercise conditions and whether participants were in the Control group or the Exercise group ($F(5, 3817) = 10.885, p < 0.001$).

In order to determine the nature of these significant effects the MANOVA provided univariate analyses. The results of the univariate analyses should be viewed in conjunction with the respective profile plots (Table 2.3).

**Univariate Analyses**

**Significant Changes in PC1**

There was a significant difference between the Pre-exercise condition and the Post-exercise condition in PC1 ($F(1, 3821) = 7.676, p = 0.006$) as well as a significant interaction between the Pre-exercise and Post-exercise conditions and whether participants were in the Control group or the Exercise group ($F(1, 3821) = 4.084, p = 0.043$). The Control group had a reduction in PC1 from 0.067 (SE 0.033) to -0.088 (SE 0.033) after the “Exercise” condition. A *post-hoc* power analysis revealed a calculated statistical power of 0.93 for the reduction of PC1 in the Control group (Decision Support Systems LP, 2012, accessed on 1 May 2013). In the Control group, a reduction in PC1 resulted in an increase in the width of the mean trajectory of the mandibular motion path in the coronal plane and an increase in
the vertical displacement in the coronal and sagittal planes (Figure 2.5 and Table 2.3).

There was no significant change in the PC score of the Exercise group.

**Significant Changes in PC2**

There was a significant difference in PC scores between the Control and Exercise groups in PC2 \( (F_{(1, 3821)} = 131.917, p < 0.001) \) as well as a significant interaction between the Pre-exercise and Post-exercise conditions and whether participants were in the Control group or the Exercise group \( (F_{(1, 3821)} = 25.322, p < 0.001) \). Analysis of covariance (ANCOVA), adjusting for the differences between the Exercise Group and the Control Group as a covariate, showed a significant difference in the PC scores between the groups \( (F_{(1, 3822)} = 130.857, p < 0.001) \) as well as a significant contrast between the Control and Exercise groups between the Pre-exercise and Post-exercise conditions \( (F_{(1, 3822)} = 130.857, p < 0.001) \). The Control group had a reduction in PC2 from -0.120 (SE 0.033) to -0.262 (SE 0.032) and the Exercise group had an increase from 0.085 (SE 0.031) to 0.262 (SE 0.031) after the “Exercise” condition. *Post-hoc* power analyses revealed a calculated statistical power of 0.84 for the reduction of PC2 in the Control group and a statistical power of 0.99 for the increase in PC2 in the Exercise group (Decision Support Systems LP, 2012, accessed on 1 May 2013). In the Control group, a reduction in PC2 resulted in the mean trajectory of the mandibular motion path to become more vertically angulated in the coronal plane and less protruded in the sagittal plane (Figure 2.5 and Table 2.3). In the Exercise group, an increase in PC2 resulted in the mean trajectory of the mandibular motion path to become more horizontally angulated in the coronal plane and more protruded in the sagittal plane (Figure 2.6 and Table 2.3).
**Significant Changes in PC3**

There was a significant difference between the Pre-exercise condition and the Post-exercise condition in PC3 ($F_{(1, 3821)} = 6.446, p = 0.011$). There was a significant difference in PC scores between the Control and Exercise groups in PC3 ($F_{(1, 3821)} = 687.853, p < 0.001$). There was a significant interaction between the Pre-exercise and Post-exercise conditions and whether participants were in the Control group or the Exercise group in PC3 ($F_{(1, 3821)} = 8.969, p = 0.003$). ANCOVA, adjusting for the differences between the Exercise Group and the Control Group as a covariate, showed a significant difference in the PC scores between the Pre-exercise and Post-exercise conditions ($F_{(1, 3822)} = 5.655, p = 0.017$) as well as between the groups ($F_{(1, 3822)} = 686.115, p < 0.001$). Univariate contrast testing revealed a significant contrast between the Control and Exercise groups between the Pre-exercise and Post-exercise conditions ($F_{(1, 3822)} = 130.857, p < 0.001$). The Control group had an increase in PC3 from 0.329 (SE 0.031) to 0.493 (SE 0.030) after the “Exercise” condition. A post-hoc power analysis revealed a calculated statistical power of 0.995 for the increase of PC3 in the Control group (Decision Support Systems LP, 2012, accessed on 1 May 2013). In the Control group, an increase in PC3 resulted in the mean trajectory of the mandibular motion path to become more horizontal, shorter and rounder in the coronal plane and less protruded and shorter in the sagittal plane (Figure 2.5 and Table 2.3). There was no significant change in the PC score of the Exercise group.

**Significant Changes in PC4**

There was a significant interaction between the Pre-exercise and Post-exercise conditions and whether participants were in the Control group or the Exercise group in PC4 ($F_{(1, 3821)} = 6.457, p = 0.011$). The Exercise group had an increase in PC4 from -0.049 (SE 0.031) to 0.071 (SE 0.032) after the “Exercise” condition. A post-hoc power analysis revealed a
calculated statistical power of 0.77 for the increase of PC4 in the Exercise group (Decision Support Systems LP, 2012, accessed on 1 May 2013). In the Exercise group, an increase in PC4 resulted in the mean trajectory of the mandibular motion path to become longer and rounder in the coronal plane (Figure 2.6 and Table 2.3). There was no significant change in the PC score of the Control group.

**Significant Changes in PC5**

There was a significant difference between the Pre-exercise condition and the Post-exercise condition in PC5 ($F_{(1, 3821)} = 9.022, p = 0.003$) as well as a significant interaction between the Pre-exercise and Post-exercise conditions and whether participants were in the Control group or the Exercise group ($F_{(1, 3821)} = 5.989, p = 0.014$). The Exercise group had an increase in PC5 from -0.061 (SE 0.031) to 0.116 (SE 0.032) after the “Exercise” condition. A *post-hoc* power analysis revealed a calculated statistical power of 0.99 for the increase of PC5 in the Exercise group (Decision Support Systems LP, 2012, accessed on 1 May 2013). In the Exercise group, an increase in PC5 resulted in the mean trajectory of the mandibular motion path to become narrower and shorter in the coronal plane (Figure 2.6 and Table 2.3). There was no significant change in the PC score of the Control group.
### Displacements of the MIPT with Reduction in PC score

<table>
<thead>
<tr>
<th>Sagittal plane</th>
<th>Coronal plane</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase magnitude of the vertical displacement.</td>
<td>Increase width of the trajectory.</td>
</tr>
<tr>
<td>Increase magnitude of the vertical displacement.</td>
<td>Angulation becomes less protruded.</td>
</tr>
</tbody>
</table>

### Table 2.3: Summary of the significant changes in principal component (PC) scores of the first five PCs and their respective effects on the mandibular masticatory movement paths. Shown are the profile plots of the mean PC scores, with error bars depicting standard error (SE), during the pre-exercise condition (Pre) and the post-exercise condition (Post) in the Control group (blue lines) and the Exercise group (green lines). A summary of the Significant Effects following the univariate analyses is listed for each PC and each condition that showed a significant difference (see text for details). The last four columns offer a brief interpretation of the significant differences in each PC in both the Control group and the Exercise group and how these differences affected the mandibular movement paths (Table continued over page).
Table 2.3 Continued: Summary of the significant changes in PC scores of the first five PCs and their respective effects on the mandibular masticatory movement paths. Shown are the profile plots of the mean PC scores, with error bars depicting standard error (SE), during the pre-exercise condition (Pre) and the post-exercise condition (Post) in the Control group (blue lines) and the Exercise group (green lines). A summary of the Significant Effects following the univariate analyses is listed for each PC and each condition that showed a significant difference (see text for details). The last four columns offer a brief interpretation of the significant differences in each PC in both the Control group and the Exercise group and how these differences affected the mandibular movement paths.

<table>
<thead>
<tr>
<th>Profile Plots of the Mean PC Scores (SE)</th>
<th>Univariate Analysis Significant Effects</th>
<th>Group Changes</th>
<th>Displacements of the MIPT with Reduction in PC score</th>
</tr>
</thead>
<tbody>
<tr>
<td>PC4</td>
<td>PrePost x Exercise</td>
<td>No change.</td>
<td>Increase in PC4, i.e. rounder and longer in the Coronal plane.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Shape of the trajectory becomes narrowed and slightly shorter.</td>
</tr>
<tr>
<td></td>
<td>PrePost</td>
<td>No change.</td>
<td>Increase in PC4, i.e. rounder and longer in the Coronal plane.</td>
</tr>
<tr>
<td></td>
<td>PrePost x Exercise</td>
<td></td>
<td>Shape of the trajectory becomes narrowed and slightly shorter.</td>
</tr>
</tbody>
</table>

Table 2.3 Continued:
Discussion

This study demonstrated the use of PCA on a variance-covariance matrix in the quantitative analysis of masticatory motion paths of the human jaw before and after the application of isometric resistance exercises to the muscles of mastication. The results are consistent with the findings of previous studies that have utilised the same or similar analysis techniques (Hattori et al., 2010, Igari et al., 2012). The current study found that a total of five PCs, or independent movement variations, out of a total 123 PCs described 92.1% of the total variance in the masticatory motion paths when chewing cooked rice.

Many variables have been used to describe the kinematics of human jaw movement, such as, maximum excursion in the vertical, lateral and anteroposterior directions, angles of approach of the mandible into centric occlusion, cycle and phase durations, velocities and accelerations (Bates et al., 1975, Buschang et al., 2000). Unfortunately, a large amount of information that describes the overall kinematics of the masticatory cycle is discarded when utilising these variables (Buschang et al., 2000). Furthermore, previous quantitative studies have used arbitrary points of the masticatory cycle for the analyses of the variations of the motion paths of the jaw (Hattori et al., 2010). The use of PCA on a variance-covariance matrix, on the other hand, was capable of describing the kinematics of the masticatory motion paths in detail without the loss of valuable information that may otherwise contribute to the overall description of the masticatory motion path. Moreover, the results of the current study suggest that only five key independent movement variations are required to describe the majority of the variations in the mandibular motion paths that contribute to the masticatory sequence.
These results compare favourably with a previous study, in which participants chewed gum and gummy candy, where it was found that the linear combination of three independent movement variations accounted for an average of 93% (range, 88% to 96%) of the total variations in the masticatory cycle paths (Hattori et al., 2010). It is well recognised that the chewing of different types of foods results in different functional requirements from the masticatory system (Foster et al., 2006, Peyron et al., 2004, Peyron et al., 2002, Woda et al., 2006a, Woda et al., 2006b). It is therefore plausible that the chewing of cooked rice, as used in the current study, requires different functional requirements than the chewing of gum and/or gummy candy (Hattori et al., 2010) and hence, results in different movement patterns in the jaw during mastication. These different movement patterns therefore may have contributed to the different numbers of independent movement variations that contributed to the total variations in the masticatory cycle paths; namely three independent movement variations (or PCs) in the previous study (Hattori et al., 2010) compared to five independent movement variations (or PCs) in the current study.

Gum and gummy candy have a more even consistency and, in the case of gum in particular, have the potential to maintain their consistency throughout the chewing sequence. Cooked rice, on the other hand, is less likely to maintain its original shape and form during the chewing sequence. As cooked rice is transported and comminuted in the mouth it would gradually reduce in size and change consistency, and therefore change its shape and form during mastication as it is prepared appropriately for deglutition. These differences in the food bolus presented to participants, and its processing during mastication may account for the differences seen in the number of PCs that described the majority of the variance in the masticatory motion paths in the current study compared to previous works (Hattori et al., 2010, Igari et al., 2012). Further studies are warranted to verify these differences.
In general, resistance exercises have been shown to increase range of motion in the knee (Crossley et al., 2005, Doucette and Child, 1996) and in the shoulder (Ginn and Cohen, 2004, Ginn and Cohen, 2005, Ginn et al., 1997). More specifically, therapeutic exercise may also be effective in the reduction of symptoms in TMD patients (List and Axelsson, 2010, McNeely et al., 2006, Medlicott and Harris, 2006). Recently, EMG changes have been reported in the muscles of mastication of asymptomatic individuals during standardised jaw movements following the application of a specific resistance exercise task (Wirianski, 2009, Wirianski et al., 2014). As far as the author is aware, the current study is the first to demonstrate significant jaw movement path changes in asymptomatic individuals during mastication following the application of a resistance exercise task. The results suggest that isometric resistance to right sided mandibular movement may produce changes in the masticatory movement path. Of the five PCs that contributed to the majority of the variations in the masticatory cycle paths in our sample of participants, three PCs showed significant changes following the resistance exercise task, namely PC2, PC4 and PC5 (Figure 2.6 and Table 2.3). Right sided resistance exercise resulted in a more horizontal and protruded masticatory movement path in PC2, a rounder and longer masticatory movement path in PC4 and a narrower and shorter masticatory movement path in PC5. Further, PC2 contributed to almost 20% of the variations in the masticatory cycle paths, whereas PC4 and PC5 contributed to less than 10% of the variations in the masticatory cycle paths combined (Table 2.2). This suggests that PC2 may have a larger influence on the masticatory movement path of the jaw following the application of isometric resistance exercise. In our sample of participants, therefore, this may imply that the application of resistance exercise to a lateral jaw movement could more likely result in a masticatory movement path which is more horizontally orientated in the coronal plane.
and more protruded in the sagittal plane. Future studies incorporating a wider sample of the population may help to confirm the repeatability of these findings, which in turn may assist clinicians in making more generalisable decisions when formulating management plans.

Furthermore, this result could suggest a possible mechanism for these observed changes. Mastication is a functional movement that results from the combination of jaw movements in the three anatomical planes: open and closing movements and protrusion and retraction in the sagittal plane; open and closing and lateral movements in the coronal plane; and lateral movements and protrusion and retraction in the transverse plane. The production of these active jaw movements is therefore the result of the coordinated actions of the muscles of mastication: opening produced primarily by the action of the lateral pterygoid and digastric; closing is produced primarily by the action of masseter, medial pterygoid and temporalis (Hannam and McMillan, 1994, Miller, 1991); lateral movements of the mandible produced primarily by the action of the contralateral medial and lateral pterygoid, ipsilateral temporalis and masseter (Hannam and McMillan, 1994, Miller, 1991, Murray et al., 1999, Uchida et al., 2001, Uchida et al., 2002); protrusive movements of the mandible are primarily produced by the action of the medial and lateral pterygoid, and masseter (Hannam and McMillan, 1994, Miller, 1991, Murray et al., 1999); and retraction being primarily produced by the posterior temporalis and anterior digastric (Hannam and McMillan, 1994, Miller, 1991). Specific resistance jaw exercise has been shown to produce changes in the temporal characteristics of the EMG activity patterns in the muscles of mastication during a standardised unilateral jaw movement task (Wirianski, 2009, Wirianski et al., 2014). Furthermore, resistance exercise is known to increase muscle strength and improve task performance possibly as a result of neuroplastic changes in the central and peripheral nervous systems and structures that control lateral jaw movements.
(Boudreau et al., 2007, Sale and MacDougall, 1981). Therefore increasing the strength of these muscles and improving the performance of the specific task (i.e. laterotrusion and/or protrusion) may lead to these muscles contributing more to the production of the more complex functional task of mastication (a sequence of jaw movements that combine both laterotrusion and protrusion along with opening and closing) and thereby producing a more horizontal and protruded masticatory path. This might suggest that patients with TMD who present with a reduced and/or a more retruded lateral movement pattern may benefit from an ipsilateral resistance exercise task. This hypothesis warrants further investigation in a controlled clinical setting.

In addition, the resistance exercise task may have affected transient changes in the physical properties of the soft tissues surrounding the TMJ especially at the wider jaw movements during opening and closing (Peck et al., 2000, Peck et al., 2002). Viscosity of the soft tissues provides the major resistance to jaw movement (Peck et al., 2002) and articular stability and jaw posture may also be affected by thixotropic properties of the TMJ which may result in increased stiffness with inactivity and reduced stiffness with increased activity (Peck et al., 2002, Lobbezoo et al., 2004). It is plausible therefore that the increased level of activity brought about by completion of the resistance exercise task may have reduced the stiffness in the surrounding soft tissues. A transient reduction in TMJ stiffness may have contributed to the changes in the masticatory movement path resulting in the more horizontally orientated chewing pattern in the coronal plane and more protruded chewing pattern in the sagittal plane as demonstrated in this study.

Significant changes in the jaw movement trajectories also occurred in the Control group in three of the five PCs that contributed to the majority of the variations in the masticatory
cycle paths, namely PC1, PC2 and PC3 (Figure 2.5 and Table 2.3). Furthermore, there were no significant changes in PC1 and PC3 in the Exercise group, which tended to maintain these two PCs at pre-exercise levels. These findings of changes in the Control group may suggest a level of variability in the available motor control patterns in the human masticatory system during mastication of cooked rice. The maintenance of the pre-exercise values in the Exercise group suggests that the isometric resistance exercise may result in stabilisation of the motor strategy (Sale and MacDougall, 1981) which may reflect the neuroplastic changes that can occur at multiple sites throughout the central nervous system (Boudreau et al., 2007, Sale and MacDougall, 1981, Wolpaw and Tennissen, 2001) and this may be significant to understanding improvement in symptoms (Au and Klineberg, 1993, Feine and Lund, 1997, List and Axelsson, 2010, McNeely et al., 2006, Medlicott and Harris, 2006, Nicolakis et al., 2001, Nicolakis et al., 2002, Rocabado and Iglarsh, 1991).

Interestingly, similar findings of significant changes in the Control group along with maintenance of the pre-exercise values in the Exercise group have been reported in the temporal characteristics of the EMG activity patterns in the muscles of mastication during a standardised unilateral jaw movement (Wirianski, 2009, Wirianski et al., 2014). Those results suggested a level of variability between recording sessions in the recruitment patterns of the muscles of mastication when producing the same right lateral jaw movement and that isotonic resistance exercise may reduce this variability (Wirianski, 2009, Wirianski et al., 2014). Future studies combining an 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.
The sample size of each group (n = 7) in this study was small. *Post-hoc* power analyses (Decision Support Systems LP, 2012, accessed on 1 May 2013) of the six experimental conditions in which the PCs showed significant changes after the application of resistance exercises to the muscles of mastication revealed calculated statistical power ranging from 0.77 (PC4) to 0.995 (PC3). These power analyses are supportive that the majority of the significant between group effects occurred as a result of the application of isometric resistance exercise to the muscles of mastication.

Only the immediate effects of specific resistance exercises have been assessed in this study. Although the current results appear promising they would only reflect changes in the jaw movement paths that occurred within 15 to 20 minutes. This time period would reflect a normal treatment time in a clinical setting and interestingly, in the orofacial region, neuroplastic changes have been reported in conjunction with improvements in performance of a novel tongue protrusion task after 15 minutes of training (Boudreau et al., 2007). However, no conclusions can be made as to the longevity of these changes over longer periods of time. Therefore, further studies investigating the longer term effects and the possible maintenance of these immediate changes over longer periods of time are warranted.

The current study only investigated the effects of resistance jaw exercises on the masticatory movement path in asymptomatic participants. Whether these movement path changes seen in asymptomatic individuals have a causal relationship in the reduction or resolution of symptoms in TMD sufferers warrants further investigation. Although promising, the current results need to be tested in a randomised control trial in order to
assess the clinical effects of resistance exercises applied to the jaw in TMD patients with appropriately matched controls.
Conclusion

The effects of an isometric resistance exercise task applied to the muscles of mastication on the masticatory motion paths of the human jaw were investigated using principal component analysis (PCA) on a variance-covariance matrix. In asymptomatic individuals, isometric resistance exercise applied against a lateral jaw movement resulted in masticatory movement paths that were more horizontally orientated in the coronal plane and more protruded in the sagittal plane. Applying the findings from this study to an appropriate patient population warrants further investigation in a clinical setting utilising appropriately matched controls. This may help to elucidate the mechanisms whereby therapeutic exercise affects the muscles of mastication and thereby contributes to the effectiveness of that therapeutic exercise in the management of TMD.
This chapter presents the second of two studies described in this thesis. It reports the effects of an isometric resistance exercise task against lateral jaw movement on the electromyographic (EMG) activation patterns of the muscles of mastication during free chewing of gum. Both the immediate and longer-term effects, after two weeks of a home-based exercise programme, will be discussed.

This study was conducted under the supervision of Assistant Professor Ichiro Minami at the Removable Partial Prosthodontics Unit in the Department of Masticatory Function Rehabilitation, Division of Oral Health Sciences, Graduate School, Tokyo Medical and Dental University, Japan. It was supervised in collaboration with Professor Christopher C. Peck and Professor Gregory M. Murray from the Jaw Function and Orofacial Pain Research Unit, Westmead Centre for Oral Health, Faculty of Dentistry, The University of Sydney.

The completion of this study was made possible by the generous assistance of Dr Ryosuke Harakawa, Dr Antoine Couturier and Dr John Gal. The author was supported by the 2010 Prime Minister’s Australia Asia Endeavour Award (Postgraduate Outgoing) from the Australian Government Department of Education and Training and the Fellowship of the International Scientific Exchange Fund from the Japan Dental Association.
Isometric resistance jaw exercise alters electromyographic activity in the jaw muscles during mastication.

Abstract
Temporomandibular disorders (TMD) are often associated with limitations and/or deviations in jaw movement which can impact on an individual’s ability to chew. These jaw movement deviations may be the result of alterations in the activation patterns of the muscles of mastication due to changes in motor control. Alterations in muscle activation patterns have been reported in other musculoskeletal systems. In particular, the prescription of specific exercise regimens for patients with recurrent low back pain has been associated with the restoration of these altered electromyographic (EMG) patterns along with cortical reorganisation of the motor representation of transversus abdominis to closely resemble those found in healthy, asymptomatic individuals. This study tested the hypothesis that isometric resistance exercise can change the patterns of EMG activation of the muscles of mastication during free chewing. Surface EMG activity was recorded from the anterior temporalis and masseter muscles bilaterally during chewing a single pellet of gum in twenty asymptomatic adult volunteers. Participants were randomly allocated to either a Control group (n = 10) or an Exercise group (n = 10). Jaw movement and EMG activity were simultaneously collected: i) at baseline, before the exercise task; ii) immediately after the exercise task (isometric resistance at between 20% to 30% maximum voluntary contraction against right lateral jaw movements); iii) after two weeks of a home-based exercise programme; and iv) at four-weeks follow-up. Performance of the exercise task resulted in a significantly (p < 0.05) earlier onset of the EMG activity in the ipsilateral anterior temporalis and the ipsilateral masseter during the masticatory cycle. These results
may reflect changes in the central motor control of mastication brought about by the application of the exercise task.
Introduction

Pain associated with jaw function and parafunction along with limitations of jaw opening are common symptoms reported by patients presenting with temporomandibular disorders (TMD, Schiffman et al., 2014), all of which could result in difficulties with mastication. Specific therapeutic jaw exercises are commonly used in the management of TMD (Glass et al., 1993, Michelotti et al., 2005) with several systematic reviews reporting some evidence for their efficacy, especially when used in conjunction with other treatment modalities (List and Axelsson, 2010, McNeely et al., 2006, Medlicott and Harris, 2006). However, the mechanism(s) of action of therapeutic jaw exercises remains poorly understood which may underlie the lack of stronger evidence for their management effectiveness (List and Axelsson, 2010, McNeely et al., 2006, Medlicott and Harris, 2006).

Specific therapeutic exercise has been demonstrated to be effective in reducing pain and disability and improving function in the knee (Cowan et al., 2002, Cowan et al., 2003, Crossley et al., 2002), shoulder (Ginn and Cohen, 2004, Ginn and Cohen, 2005, Ginn et al., 1997), cervical spine (Falla et al., 2006, Jull et al., 2009) and lumbar spine (Hides et al., 1996). In these musculoskeletal pain conditions, the pain and disability was associated with alterations in the activation patterns of the muscles that control the respective joints (Cowan et al., 2002, Cools et al., 2003, Falla et al., 2004b, Tsao and Hodges, 2008). The prescription of specific exercise regimens that addressed the altered muscle activation patterns resulted in electromyographic (EMG) activity patterns that closely resembled those of healthy individuals (Cowan et al., 2002, Tsao and Hodges, 2008, Jull et al., 2009, Worsley et al., 2013, Falla et al., 2004a). These findings suggest that the prescribed motor relearning interventions may be associated with neural plasticity of the nervous system at many levels including changes in the excitability of motoneurones, the sensorimotor cortex...
and/or the cerebellum (Tsao et al., 2008, Tsao and Hodges, 2007) and thus resulting in improved task performance (Boudreau et al., 2007, Iida et al., 2013, Sale and MacDougall, 1981, Wolpaw and Tennissen, 2001).

Recent evidence from our group has shown that resistance exercise has significant effects on the EMG activity patterns of the muscles of mastication during a standardised jaw movement task (Wirianski, 2009, Wirianski et al., 2014). In this study, for a reproducible lateral jaw movement, subjects who undertook isotonic resistance jaw exercises at 60% maximum voluntary contraction (MVC), demonstrated a reduction in the duration of EMG activity in the ipsilateral anterior temporalis with a concomitant increased EMG activity duration to peak activity in the contralateral masseter and ipsilateral digastric compared to participants in the Control group that undertook no jaw exercises (Wirianski, 2009, Wirianski et al., 2014). These findings suggested that, within an individual, completion of a standardised task can be achieved by a change in the muscle recruitment and activity patterns (Wirianski, 2009, Wirianski et al., 2014). This fits with the concept that the masticatory system is mechanically redundant, with a wide range of muscle activation patterns available to produce a desired jaw movement (Lobbezoo et al., 2004, Van Eijden et al., 1990, Langenbach and van Eijden, 2001).

Interestingly, the majority of changes occurred in the Control group, suggesting variability in the utilisation of the available motor control patterns in the human masticatory system (Lobbezoo et al., 2004, Van Eijden et al., 1990, Proschel, 1987, Langenbach and van Eijden, 2001) when completing standardised jaw movement tasks (Wirianski, 2009, Wirianski et al., 2014). In contrast, the Exercise group tended to maintain the pre-exercise values in the tested variables over the eight-weeks testing period (Wirianski, 2009,
Wirianski et al., 2014). This suggested that the resistance exercise task may have resulted in a stabilisation of the motor strategy possibly reflecting neuroplastic changes in the central nervous system resulting in improved task performance (Boudreau et al., 2007, Iida et al., 2013, Sale and MacDougall, 1981, Wolpaw and Tennissen, 2001).

The application of isometric resistance jaw exercises also appears to result in changes in jaw movement patterns during chewing (Chapter 2). Participants that completed 15 minutes of an isometric resistance exercise task at 30% MVC against ipsilateral mandibular movement were found to have masticatory movement paths that were significantly more horizontally orientated in the coronal plane and more protruded in the sagittal plane during chewing of cooked rice compared to a Control group that completed no jaw exercises (Chapter 2). Although EMG activity was not recorded in this study, changes in movements require changes in EMG activity, and therefore these results may be consistent with the changes in muscle recruitment patterns described earlier (Wirianski, 2009, Wirianski et al., 2014). Furthermore, significant changes in jaw movement trajectories during mastication also occurred in the Control group (Chapter 2), and this observation of significant motor changes in the Control group is also consistent with the findings of the previous study (Wirianski, 2009, Wirianski et al., 2014).

Therefore, resistance exercise appears to result in changes in EMG activity patterns of the muscles of mastication during a standardised movement task (Wirianski, 2009, Wirianski et al., 2014) as well as changes in the trajectory of the mandible during free chewing of cooked rice (Chapter 2). With the inherent mechanical redundancy of the masticatory system (Van Eijden et al., 1990, Langenbach and van Eijden, 2001), these changes may reflect changes in the coactivation patterns of the recruited muscles (Van Eijden et al.,
under the control of the masticatory central pattern generator and higher motor cortical regions. Therefore, the aim of the present study was to further investigate the effects of specific resistance jaw exercises on the EMG activity patterns of the muscles of mastication during natural chewing of a standardised soft and consistent bolus, a pellet of gum, with the view of further clarifying the effects of resistance exercise on jaw muscle EMG activity patterns. Based on the results of the previous two studies (Wirianski, 2009, Wirianski et al., 2014), it is hypothesised that the application of an isometric resistance jaw exercise task against lateral jaw movement would result in a change in EMG activity in the jaw muscles and more reproducible activity patterns during chewing. Moreover, during lateral jaw movements, the ipsilateral anterior temporalis and the contralateral masseter muscles have greater EMG activity (Van Eijden et al., 1990). Also, the ipsilateral temporal muscles and the contralateral masseter are activated first while the ipsilateral masseter and temporal muscles demonstrate more EMG activity during unilateral chewing of gum (Moller, 1966). Therefore it would be plausible to expect greater EMG activity changes in these muscles during chewing following the application of a lateral resistance exercise task. Furthermore, in some of the tested EMG variables, we hypothesise that there will be significant changes in the Control group between the testing sessions while in the Exercise group the values of these variables will be maintained at baseline levels.
Methods

Participants
Twenty asymptomatic adult volunteers (8 females, 12 males; aged 21.8 to 45.8 years) participated in this study. Participants with a past or current history of pain or dysfunction (e.g. TMD) in and around the orofacial region were excluded. Participants provided informed consent to participate in the study. All experimental procedures were approved by the Tokyo Medical and Dental University Human Research Ethics Committee (No: 874; Appendix 1).

Figure 3.1 shows a flowchart depicting the experimental design. Each participant attended three data collection sessions, each spaced two weeks apart, conducted at The Department of Removable Partial Dentures, Faculty of Dentistry, Tokyo Medical and Dental University. During each of the three data collection sessions participants sat comfortably in an upright chair with their head unsupported in a neutral and comfortable position. Participants were randomly allocated into one of two groups: a Control group; or, an Exercise group. Jaw movement and EMG data were collected simultaneously on four separate occasions over the course of the study. Data collection session 1 (S1) was divided into three sections. During the first section of S1 (S1-Baseline), baseline jaw movement and EMG data were collected during five trials of chewing a standard pellet of sugar-free chewing gum (Xylitol: Lotte Co., Ltd, Tokyo, Japan). During the second section of S1, participants in the Control group were asked to sit quietly in an upright chair for approximately 15 minutes, while participants in the Exercise group were asked to perform an isometric resistance exercise task to the jaw muscles as described below (see page 108). Finally, during the third section of S1 (S1-Post), jaw movement and EMG data collection was repeated a second time approximately 15 minutes after S1-Baseline (Figure 3.1). Data
collection session 2 (S2) took place two weeks after S1 and data collection session 3 (S3) took place four weeks after S1. During S2 and S3 the same jaw movement and EMG data collection, as performed during S1-Baseline and S1-Post, was repeated a third and fourth time respectively (Figure 3.1).
Participant recruitment 
(n = 20)

Randomisation into Control group or Exercise group.

Record MVC x3 each for: 
- Right and left lateral movements.

Record jaw movement and EMG during chewing of a standard pellet of gum (x5 Trials)

Control Group
No resistance jaw exercise training 
(n = 10)

Exercise Group
Perform Isometric lateral resistance jaw exercise training on preferred chewing side. 
5 sets of 10 repetitions at 20-30% of the measured MVC 
(n = 10)

Record jaw movement and EMG during chewing of a standard pellet of gum (x5 Trials)

Control Group
Continue normal activities 
No exercise for 2 weeks 
(n = 10)

Record MVC x3 each 
Record jaw movement and EMG 
(n = 10)

Exercise Group
Perform resistance jaw exercise 
training for 2 weeks 
(n = 10)

Record MVC x3 each 
Record jaw movement and EMG 
(n = 10)

Control Group
Continue normal activities 
No exercise for 2 weeks

Record MVC x3 each 
Record jaw movement and EMG 
(n = 10)

Exercise Group
Continue normal activities 
No exercise for 2 weeks

Record MVC x3 each 
Record jaw movement and EMG 
(n = 10)

Figure 3.1: Flowchart depicting the experimental design with sample sizes (n) in each group. 
(MVC: maximum voluntary contraction; EMG: electromyographic activity).
**Maximum Voluntary Contraction (MVC)**

Maximum voluntary contraction (MVC) is widely used to assess the maximum isometric force that can be generated by both single and multi-joint muscles (Wilson and Murphy, 1996). Furthermore, it has been reported to be a reliable measure of isometric strength in the neck muscles (Asghar et al., 2003, Marjan et al., 2008, Almosnino et al., 2010, Salmon et al., 2015), neck and shoulder muscles (Tornøe et al., 2013), shoulder muscles (Fischer et al., 2011), quadriceps femoris (Morton et al., 2005, Blacker et al., 2013) and for measuring grip strength of the hand (Demura et al., 2001). Therefore, MVC was chosen to measure the maximum force produced by the muscles of mastication as described below.

The maximum force produced during an isometric contraction of the muscles of mastication during right and left lateral jaw movements with the mandible in the neutral position was determined for all participants. For participants in the Exercise group, this MVC force produced in the direction of their preferred (ipsilateral) chewing side was used to standardise the applied force during the resistance exercise training section. The force was measured with a miniature load cell (LMB-A-200N: Kyowa Electronic Instruments Co., Ltd., Tokyo, Japan) connected to a sensor interface (PCD-300A: Kyowa Electronic Instruments Co., Ltd., Tokyo, Japan). The load cell was placed in an acrylic faceplate (Figure 3.2, Panel A) and held against the skin over the right mandible adjacent to the canine root tip by the right hand of the participant (Figure 3.2, Panel B). The output of the sensor interface was connected to a personal computer which calculated, displayed and stored the magnitude of the force exerted during each isometric MVC trial on a screen placed in front of the participant (Figure 3.3). When instructed, participants applied an isometric resistance force against a right lateral jaw movement as hard as they could without pain for approximately five seconds with the mandible held stationary in the
neutral position (Figure 3.2, Panel B and Figure 3.3). To encourage the participant’s best effort during the MVC trials, a visual display of the magnitude of the force they exerted during each isometric MVC trial was provided on a screen placed in front of them. Participants were also given verbal encouragement during each MVC trial from the experimenter (AW) to push as hard as they could against the load cell without causing pain or discomfort. This was completed three times and the highest displayed force reading was used as a measure of the participant’s MVC against right sided isometric resistance. This set of three MVC trials were then repeated on the left side. For participants in the Exercise group the exercise training isometric force was then calculated. Due to the difficulty of maintaining a training force exactly at a predetermined level, participants in the Exercise group were asked to maintain the training force during the exercises between a range of 20% to 30% of their MVC for their preferred chewing side (Figure 3.3).
Figure 3.2: Acrylic faceplate and its positioning during the maximum voluntary contraction (MVC) and resistance exercise trials. Panel A: The components of the acrylic faceplate showing the miniature load cell *in situ*. Panel B: The participant held the anterior surface of the acrylic faceplate against the skin over the mandible adjacent to the canine root tip with the lower border of the mandible resting on the mandibular positioning ledge and the participant’s chin lightly touching the anterior-posterior positioning strip.
Figure 3.3: Force biofeedback during the maximum voluntary contraction (MVC) and resistance exercise tasks. The acrylic faceplate containing the miniature load cell was held by participants with their hand against the skin such that the miniature load cell was positioned over the mandible adjacent to the canine root tip, as in Figure 3.2, Panel B. The white arrow shows the direction of the resistance force applied against the lateral movement of the mandible during the MVC and resistance exercise tasks. Participants held the mandible stationary in the neutral position by their hand. A monitor placed in front of the participants during the MVC and resistance exercise trials provided feedback of their performance while generating the applied force onto the load cell. Here the two white horizontal lines placed on the monitor depict the 20% MVC (lower line) and the 30% MVC (upper line) values used during the resistance exercise trials.

**Jaw movement**

Movement of the jaw was recorded simultaneously in three orthogonal planes (antero-posterior, X; horizontal, Y; and vertical, Z) during the chewing of a standard pellet of chewing gum. Each participant was seated comfortably in an upright position with a natural head posture and their occlusal plane was used as the reference plane for the
recording of jaw movement data. Each participant was asked to chew the standard pellet of gum on their preferred chewing side for approximately 20 to 30 chewing cycles (one chewing trial) and asked to repeat this chewing trial a further four times during each data collection occasion. This resulted in a total of five chewing trials during S1-Baseline, five chewing trials during S1-Post after the 15 minutes interval in between, and a further five chewing trials at S2 (two weeks following S1-Baseline) and S3 (four weeks following S1-Baseline).

Jaw movement data were collected using an electromagnetic jaw tracking system at a sampling rate of 120 samples per second (3SPACE® FASTRAK®, Polhemus, Colchester, Vermont USA) linked to a personal computer. This system records the position of the mandible with six degrees of freedom by generating near field, low frequency magnetic field vectors from a stationary transmitter unit which consists of an assembly of three concentric antennae. A moveable remote receiver unit consisting of three concentric sensing antennae detect the field vectors during movements of the receiver unit. The detected signals are then input to a mathematical algorithm that calculates the position and orientation of the receiver unit relative to the stationary transmitter unit. The manufacturer has reported that this system has a static accuracy of 0.8 mm root mean square (RMS) for the X, Y or Z position of the receiver unit, and 0.15° RMS for the orientation of the receiver unit (Polhemus, 2012). Furthermore, at a range of 300 mm, the position resolution and the orientation resolution of the 3SPACE® FASTRAK® system has been reported to be 0.0058 mm and 0.0026° respectively (Polhemus, 2012). In in vitro studies, this system has been shown to have a linear measurement error of less than 0.5 mm when the remote receiver unit is placed between 50 to 650 mm away from the transmitter unit (Ribeiro et al., 2011) and to be a reliable measure of range of motion in the cervical spine and shoulder.
(Amiri et al., 2003, Jordan et al., 2000). Furthermore, when used to measure temporomandibular joint motion the measurement error has been reported as being less than 100 μm for displacement and less than 0.02° for angulation when the transmitter and receiver were placed 300 mm apart (Kirihara et al., 2003).

On a separate day, prior to the commencement of any data collection, customised acrylic clutches were fabricated for each participant. Maxillary and mandibular impressions (Aroma Fine Mixer Type, GC Corporation, Tokyo Japan) were taken from each participant and dental stone casts poured (New Fujirock, GC Corporation, Tokyo Japan). The casts were hand articulated in the intercuspal position (IP; maximum intercuspation of opposing teeth) and mandibular and maxillary acrylic (Unifast III, GC Corporation, Tokyo Japan) clutches were custom fabricated for each participant using their respective casts. The reference plane was the occlusal plane as determined from the casts when placed in the IP. Figure 3.4 illustrates the experimental setup for each participant. At the beginning of each data collection session the clutches were fixed to the participant’s mandibular and maxillary anterior teeth from canine to canine with cyanoacrylate adhesive. The mandibular and maxillary clutches were aligned parallel with the occlusal plane and centred about the mandibular and maxillary mid-incisor points (MIPTs) respectively. The mandibular clutch had a recessed area fabricated into the bottom side of its distal portion into which the base of the receiver unit was fixed with double sided adhesive tape prior to the commencement of data collection. This ensured the reproducible placement of the receiver unit with respect to the mandibular MIPT and the transmitter unit between data collection sessions. The maxillary acrylic clutch was fixed to a light weight, reinforced, acrylic jig (Figure 3.4; PLA Plate 1.7 mm thick, Tamiya Inc., Shizuoka, Japan) at its distal end. This acrylic jig was angled upwards and posteriorly to rest gently on the participant’s
Figure 3.4: Oblique view of a participant showing the position of the EMG electrodes and the 3SPACE® FASTRAK® system. The EMG electrodes were placed on the skin overlying the anterior temporalis and masseter muscles bilaterally. The transmitter unit was positioned on the participant’s head in its jig and attached to the maxillary teeth via an acrylic clutch. In order to maintain the position of the transmitter unit during data collection the acrylic jig was secured to the participants head by an elastic strap. The receiver unit was attached to the mandibular teeth via an acrylic clutch. Both clutches and the transmitter unit were aligned parallel to the occlusal plane.

forehead and was aligned with their facial midline. The transmitter unit was then attached to the top of this acrylic jig via a light weight removable acrylic baseplate attached to the jig that rested lightly and comfortably on the participant’s head and was aligned parallel to the maxillary acrylic clutch and hence the occlusal plane. The position of this removable baseplate was fixed in a custom fitting position for each participant (Figure 3.4). The acrylic jig was secured to the participants head by an elastic strap in order to maintain the position of the transmitter unit during data collection. Thus the custom fitted acrylic
clutches and jig allowed for the reproducible placement of the receiver and transmitter units of the jaw tracking system with respect to the MIPTs between data collection sessions.

**Electromyographic (EMG) Data**
Bipolar silver-silver chloride (Ag-AgCl) surface electrodes (Duo-Trode, Myotronics, Washington, USA) were placed bilaterally over the middle of the anterior temporalis and masseter muscles and aligned in the direction of the muscle fibres (Figure 3.4). A common ground electrode was placed on the dorsum of the participant’s right wrist.

The EMG signals were amplified (1,000-10,000 times) and filtered (low pass = 400 Hz; high pass = 10 Hz) by an isolated bioelectric amplifier (P-EMG Plus: Osaka Electronic Equipment Co., Ltd. Kanabe, Japan), digitised by a data acquisition card (ADA16-32/2(CB)F; Contec, Osaka, Japan) and digitally sampled at 12,000 samples per second with custom modified data acquisition software (LabDAQ5-CT, Matsuyama Advance Co., Ltd., Ehime, Japan).

**Synchronisation of Jaw Movement and EMG Data**
The 3SPACE® FASTRAK® system and the data acquisition card used to collect EMG data were connected to a personal computer and digitally sampled at 1200 samples per second with custom developed software. This software was customised in order to synchronise the jaw movement and EMG activity data with respect to the commencement of each chewing trial. The synchronised jaw movement and EMG data were then saved as individual text files for each chewing trial for subsequent off-line processing.

**Isometric resistance exercise training task**
The isometric resistance exercise task consisted of a total of five sets of 10 repetitions of isometric resistance exercise against right or left lateral jaw movement at between 20% to
30% MVC. Each participant applied the training force to their mandible on their preferred chewing side in the same way as described for the MVC trials above. Two horizontal lines were placed on the display screen at levels that represented the calculated 20% MVC and 30% MVC which provided the participant with feedback of the force they exerted during the exercise task (Figure 3.3). During each repetition, participants were instructed to apply and maintain the training force for approximately 10 s without moving the jaw from its neutral position. Isometric resistance against lateral movement was selected for two reasons: 1) pure lateral mandibular movements are relatively novel jaw movements that are rarely performed in isolation during normal daily functions of the jaw, and; 2) to preferentially train the contralateral (left) lateral pterygoid which is a prime agonist for opening and lateral jaw movement phases of mastication. It is proposed that these two considerations will reduce the possibility of erroneous training effects taking place as a result of normal jaw function. Thus, any changes would be more likely to be the result of the resistance exercise task. Participants performed 10 repetitions of this isometric task with approximately 5 s rest between each repetition. At the completion of the 10 repetitions (one set), participants were instructed to rest quietly for 20 s to 30 s. The set of 10 isometric muscle contractions was then repeated a further four times with a 20 s to 30 s rest between each set. The complete exercise task took approximately 15 minutes to complete. Participants in the Control group did not undertake the exercise task: instead they sat quietly for 15 minutes. On completion of the exercise task by the Exercise group and the period of quiet sitting by the Control group, jaw movement and EMG data were collected during five trials of chewing a standard pellet of chewing gum (S1-Post).

On completion of S1, participants in the Exercise group were asked to continue to perform the isometric resistance exercise task at home for two weeks. This home-based resistance
exercise programme consisted of completing five sets of 10 repetitions of the isometric resistance exercise task three times per day (morning, afternoon and evening) for two weeks, with each five sets of 10 repetitions taking approximately 15 minutes to complete. To assist compliance, participants in the Exercise group were asked to complete a daily training diary (Appendix 2) and the experimenter (AW) contacted each participant on a weekly basis. Participants in the Control group were instructed to continue with their normal activities of daily living. This would then test the effects of a two-weeks home-based resistance exercise programme on the calculated EMG parameters (in the Exercise group). At the end of two weeks (S2), all participants returned for data collection of the same jaw movement and EMG data as performed during S1-Baseline and S1-Post (Figure 3.1). To test whether the longer term effects were maintained over the second two-weeks period of the study, at the completion of S2, all participants were instructed to continue with their normal activities of daily living and did not perform any jaw exercises for a further two weeks at which time the third data collection session was performed (S3; Figure 3.1).

**Data Processing**

**Selection of Chewing Trials and Chewing Cycles**

The previously saved individual text files containing the synchronised jaw movement and EMG data for each chewing trial were processed offline at the completion of the study. Of the five chewing trials completed during each of the four data collection sections, only data from the second and third chewing trials were processed for further analysis. Data from the first chewing trial were omitted from the analysis to mitigate any potential training effects that may have occurred as a result of the participant performing a novel task under experimental conditions and to allow the participant to acclimatise to the experimental
protocol. The fourth and fifth chewing trials were omitted to mitigate any possible fatigue effects that may have resulted towards the end of each data collection section. Thus, only data from the second and third chewing trials were processed as it was felt that, during these two chewing trials, participants would have had the greatest likelihood of performing the most stable chewing cycles without the influence of extraneous effects such as learning to perform the requested chewing task under experimental conditions or becoming fatigued as a result of nearing completion of the experimental protocol. From each of the second and third chewing trials, five chewing cycles were then selected for further processing. Chewing cycles six to 10 were selected as it was felt that, during these five chewing cycles, participants would have had the greatest likelihood of performing the most stable chewing cycles without the influence of extraneous effects as mentioned above. Data from the first to the fifth chewing cycles were omitted from the analysis to allow participants to settle into a natural chewing rhythm. Thus, data from a total of 10 chewing cycles per data collection section were used for the calculation of the tested variables for each participant.

Processing Movement Data

Raw movement data collected by the 3SPACE® FASTRAK® system were saved in six degrees of freedom. Therefore, each sampled data point contained the values of the X- (antero-posterior), Y- (lateral) and Z- (superior-inferior) coordinates of the position of the receiver unit attached to the participant’s mandibular teeth within a three-dimensional Cartesian system, as well as the angular values (azimuth, elevation and roll respectively) for the orientation of the receiver unit with respect to the transmitter unit placed on the participant’s head. The mean position of the receiver was 60.6 mm (Range: 55.5 to 62.2) anterior to the mandibular MIPT in the occlusal plane (Figure 3.4). The position of the mandibular MIPT was first calculated from the receiver position data for each subject. This
produced the three-dimensional data representing the trajectory of the mandibular MIPT in the antero-posterior (x-), lateral (y-) and open-close (z-) directions during each chewing trial. The open-close (z-direction) data were then used to determine the movement reference points for the analysed chewing cycles.

**Processing EMG Data**

Prior to determining the EMG reference points the raw EMG data signals (Figure 3.5) were digitally rectified and filtered (OriginPro 9.1.0, OriginLab Corporation, Northampton, MA, USA). First, the raw EMG data were bandpass filtered (Order: 2; Sample Frequency: 1200 samples per second; Lowpass Cutoff: 400 Hz; Highpass Cutoff: 25 Hz; recursive). The bandpass filtered data were then Butterworth filtered (Figure 3.6; Order: 3; Sample Frequency: 1200 samples per second; Lowpass Cutoff: 25 Hz; recursive). The Butterworth filtered data were then used to determine the EMG reference points for the analysed chewing cycles (Figure 3.7).
Figure 3.5: Synchronised raw jaw opening displacement (cm; top panel) and electromyographic (EMG; bottom four panels) data from a typical chewing sequence from one participant. (mV: millivolts).
Figure 3.6: Synchronised jaw opening displacement (cm; top panel) and processed electromyographic (EMG; bottom four panels) data from a typical chewing sequence from one participant. The sixth to tenth chewing trials were used for data analysis as shown approximately between the vertical blue lines. (mV: millivolts).
Figure 3.7: The synchronised movement (Top panel) and Butterworth filtered electromyographic (EMG) data (Bottom four panels) from the tenth chewing cycle of a typical chewing trial from one participant. (RAT: right anterior temporalis; LAT: left anterior temporalis; RMASS: right masseter; LMASS: left masseter)


**Reference Points**

To determine the movement and EMG reference points (Figure 3.7) the processed movement and EMG data were plotted onto the same graph (EMG Toolbar v3.5 for OriginPro 9.1.0, Antoine Couturier, INSEP, France). This allowed for the automated detection of the reference points in the synchronised data sets. A sample of this synchronised data for a single chewing cycle is shown in Figure 3.8. The following software settings were used for both the movement and EMG data files to automatically determine the onsets and offsets: Envelope type: RMS windowing; Window detection size: 120 data points; Duration threshold: 60 data points; Rest detection: 1200 data points. For each movement and EMG datafile an envelope was computed and drawn on the respective plot (denoted as a red line superimposed on the processed data traces in Figure 3.8). A threshold for detection of the onsets and offsets was set at five standard deviations (5 SD) above the calculated rest level for each movement and EMG datafile (horizontal red line superimposed on the processed data traces in Figure 3.8). This threshold level was selected as it enabled the automatic detection of the onset (green upward arrows in Figure 3.8) and offset (red downward arrows in Figure 3.8) reference points most consistently over the majority of the datafiles during preliminary testing of the detection method. On the relatively small number of datafiles where the automatic threshold calculation failed to calculate the onset and offset reference points then the horizontal threshold line was adjusted manually until the onset and offset reference points were calculated and revealed across all the chewing cycles in that chewing sequence. The resultant synchronised jaw movement and processed EMG datafiles at the completion of the automated detection of the reference points in a typical chewing sequence are shown in Figure 3.8.
Figure 3.8: Example of representative output graphs following the automated detection of peaks (short vertical red lines with time-point values), onsets (green upward arrows) and offsets (red downward arrows) of the synchronised jaw opening displacement (Mz; cm; top panel) and processed rectified electromyographic (EMG) data from the left anterior temporalis (LAT; middle panel) and left masseter (LMass; bottom panel) from a typical chewing sequence from one participant.
The jaw movement displacement and EMG peaks (denoted by the short vertical red lines with time-point values) as well as the onset and offset of the movement trajectory and EMG activity for each chewing cycle were then determined from the computed envelope and defined as follows:

**Jaw Movement and EMG Onset**
The time-point where the envelope of the movement and processed EMG datafile became greater than the threshold level of five standard deviations above the calculated rest level. This time-point was denoted by a green upward arrow (Figure 3.8).

**Jaw Movement Peak**
The time-point at which the jaw movement trajectory reached its maximum opening displacement from the calculated rest level for each chewing cycle. This time-point was denoted by a short vertical red line superimposed on the peak of the movement datafile and a number representing the time in seconds following the onset of data collection (Figure 3.8).

**Jaw Movement Displacement**
The maximum opening displacement of the mandible was taken as the value of the Z-coordinate at the Jaw Movement Peak of the chewing cycle.

**EMG Peak**
The time-point at which the Butterworth filtered EMG activity reached its maximum value from the calculated rest level for each chewing cycle. This time-point was denoted by a short vertical red line superimposed on the peak of the processed EMG datafile and a number representing the time in seconds following the onset of data collection (Figure 3.8).
**Jaw Movement and EMG Offset**

The time-point where the envelope of the movement and processed EMG datafile became less than the threshold level of five standard deviations above the calculated rest level. This time-point was denoted by a red downward arrow (Figure 3.8).
Calculated Variables

The following variables were calculated from the reference points as defined above:

**Jaw Movement Duration**

\[
\text{Jaw Movement Duration} \ (\text{sec}) = \text{Jaw Movement Offset} \ (\text{sec}) - \text{Jaw Movement Onset} \ (\text{sec})
\]

...Equation 1

**Jaw Movement Open Time**

\[
\text{Jaw Movement Open Time} \ (\text{sec}) = \text{Jaw Movement Peak} \ (\text{sec}) - \text{Jaw Movement Onset} \ (\text{sec})
\]

...Equation 2

**Jaw Movement Close Time**

\[
\text{Jaw Movement Close Time} \ (\text{sec}) = \text{Jaw Movement Offset} \ (\text{sec}) - \text{Jaw Movement Peak} \ (\text{sec})
\]

...Equation 3

**Chew Cycle Duration**

\[
\text{Chew Cycle Duration} \ (\text{sec}) = \text{Jaw Movement Onset} \ (\text{sec}) - \text{Jaw Movement Onset} \ (\text{Cycle n})
\]

Where \( n \) is the chew cycle number.

**Jaw Movement Open Time as a Percentage of Chew Cycle Duration**

\[
\text{Jaw Movement Open Time as a Percentage of Chew Cycle Duration} \ (%) = \frac{\text{Jaw Movement Open Time}}{\text{Chew Cycle Duration}} \times 100\%
\]

...Equation 5

**Jaw Movement Close Time as a Percentage of Chew Cycle Duration**

\[
\text{Jaw Movement Close Time as a Percentage of Chew Cycle duration} \ (%) = \frac{\text{Jaw Movement Close Time}}{\text{Chew Cycle Duration}} \times 100\%
\]

...Equation 6

**Jaw Movement Duration as a Percentage of Chew Cycle Duration**

\[
\text{Jaw Movement Duration as a Percentage of Chew Cycle duration} \ (%) = \frac{\text{Jaw Movement Duration}}{\text{Chew Cycle Duration}} \times 100\%
\]

...Equation 7
**Jaw Movement Opening Velocity**

\[
\text{Jaw Movement Opening Velocity (mm.s}^{-1}\text{)} = \frac{\text{Jaw Movement Displacement (mm.s}^{-1}\text{)}}{\text{Jaw Movement Open Time (s)}}
\]

...Equation 8

**Jaw Movement Closing Velocity**

\[
\text{Jaw Movement Closing Velocity (mm.s}^{-1}\text{)} = \frac{\text{Jaw Movement Displacement (mm.s}^{-1}\text{)}}{\text{Jaw Movement Close Time (s)}}
\]

...Equation 9

**Chewing Velocity**

\[
\text{Chewing Velocity (mm.s}^{-1}\text{)} = \frac{2 \times \text{Jaw Movement Displacement (mm.s}^{-1}\text{)}}{\text{Jaw Movement Duration (s)}}
\]

...Equation 10

**Chewing Frequency**

\[
\text{Chewing Frequency (cycles.s}^{-1}\text{)} = \frac{1}{\text{Chew Cycle Duration (s)}}
\]

...Equation 11

**Time to Peak EMG activity**

\[
\text{Time to Peak EMG Activity (sec)} = \text{EMG Peak (sec)} - \text{EMG Onset (sec)}
\]

...Equation 12

**EMG Duration**

\[
\text{EMG Duration (sec)} = \text{EMG Offset (sec)} - \text{EMG Onset (sec)}
\]

...Equation 13

**EMG Onset relative to Jaw Movement Onset**

\[
\text{EMG Onset relative to Jaw Movement Onset (sec)} = \text{EMG Onset (sec)} - \text{Jaw Movement Onset (sec)}
\]

...Equation 14

**Time to Peak EMG activity relative to Jaw Movement Onset**

\[
\text{Time to Peak EMG Activity relative to Jaw Movement Onset (sec)} = \text{EMG Peak (sec)} - \text{Jaw Movement Onset (sec)}
\]

...Equation 15
EMG offset relative to Jaw Movement Onset

\[ \text{EMG Offset relative to Jaw Movement Onset (sec)} = \text{EMG Offset (sec)} - \text{Jaw Movement Onset (sec)} \]

...Equation 16

EMG offset relative to Jaw Movement Offset

\[ \text{EMG Offset relative to Jaw Movement Offset (sec)} = \text{EMG Offset (sec)} - \text{Jaw Movement Offset (sec)} \]

...Equation 17

Relative EMG Onset as a Percentage of Jaw Movement Duration

\[ \text{Relative EMG Onset as a percentage of Jaw Movement Duration (sec)} = \frac{\text{EMG Onset relative to Jaw Movement Onset (sec)}}{\text{Jaw Movement Duration (sec)}} \times 100\% \]

...Equation 18

Relative EMG Offset as a Percentage of Jaw Movement Duration

\[ \text{Relative EMG Offset as a percentage of Jaw Movement Duration (sec)} = \frac{\text{EMG Offset relative to Jaw Movement Offset (sec)}}{\text{Jaw Movement Duration (sec)}} \times 100\% \]

...Equation 19

Relative EMG Onset as a Percentage of Chew Cycle Duration

\[ \text{Relative EMG Onset as a percentage of Chew Cycle Duration (sec)} = \frac{\text{EMG Onset relative to Chew Cycle Onset (sec)}}{\text{Chew Cycle Duration (sec)}} \times 100\% \]

...Equation 20

Relative EMG Offset as a Percentage of Chew Cycle Duration

\[ \text{Relative EMG Offset as a percentage of Chew Cycle Duration (sec)} = \frac{\text{EMG Offset relative to Chew Cycle Onset (sec)}}{\text{Chew Cycle Duration (sec)}} \times 100\% \]

...Equation 21
Statistical Analyses
All statistical analyses were conducted using PASW Statistics (version 17.0: IBM Corporation, Armonk, NY, USA) and have been previously described (Wirianski, 2009, Wirianski et al., 2014). In brief, the variables were calculated from each of the 10 chewing cycles as selected above and the mean value of each variable was calculated for each participant. The calculated mean values of each variable were first tested with the Kolmogorov-Smirnov Test of Normality. If the data were not significantly different (p > 0.05) from the normal distribution then an analysis of variance (ANOVA) was used with three planned contrasts that reflected the tested conditions in terms of: 1) the significant changes following the initial application of isotonic resistance exercise during S1 (i.e. immediate effects); 2) the significant changes in the variables between S1-Baseline and S2 (i.e. training effects); and 3) the significant changes between S2 and S3 (i.e. whether any training effects were maintained over the longer-term). The Bonferroni correction determined the critical p-value to be 0.0167 (i.e. 0.05/3) for statistical significance of the contrasts tested. For the normally distributed data the mean and standard deviation (SD) are reported. If the data were significantly different (p < 0.05) from the normal distribution, non-parametric tests were used (Mann-Whitney test, two-tailed, and Friedman test; significance accepted at p < 0.05) and the median and standard error (SE) are reported. In the situations where the Friedman test revealed significant within subjects differences across all the testing sessions a post hoc Wilcoxon test was performed using the same three planned contrasts as those used for the ANOVA. In these cases a similar Bonferroni correction (critical p-value of 0.0167) was applied to determine the level of significance of each of the three contrasts.
Results

Participants
Twenty participants (12 male and 8 female) completed the study. The mean age of the subjects was 29.6 (SD 5.8; range 21.8 to 45.8) years. Ten participants were recruited into both the Control group and the Exercise group. The demographic breakdown of participants in each group is shown in Table 3.1. There was no statistically significant difference in the age of the participants between groups (Independent Samples T-Test, t_{18} = 1.227, p = 0.236, 2-tailed).

<table>
<thead>
<tr>
<th>Group</th>
<th>Males</th>
<th>Females</th>
<th>Total</th>
<th>Mean Age (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>6</td>
<td>4</td>
<td>10</td>
<td>28.1 (7.0)</td>
</tr>
<tr>
<td>Exercise</td>
<td>6</td>
<td>4</td>
<td>10</td>
<td>31.2 (2.9)</td>
</tr>
<tr>
<td>Total</td>
<td>12</td>
<td>8</td>
<td>20</td>
<td>29.6 (5.8)</td>
</tr>
</tbody>
</table>

Table 3.1: Demographic breakdown of the 20 participants recruited into the study.

Preferred Chewing Side
Of the 20 recruited participants, nine preferred to chew on their left side and 11 preferred to chew on their right side (Table 3.2). In the Control group, three participants preferred to chew on their left side and seven preferred to chew on their right. In the Exercise group, six participants preferred to chew on their left side while four preferred to chew on their right.
<table>
<thead>
<tr>
<th>Preferred Chewing Side</th>
<th>Control Group</th>
<th>Exercise Group</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left</td>
<td>3</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>Right</td>
<td>7</td>
<td>4</td>
<td>11</td>
</tr>
<tr>
<td>Total</td>
<td>10</td>
<td>10</td>
<td>20</td>
</tr>
</tbody>
</table>

Table 3.2: Preferred chewing side of the 20 participants recruited into the study.

**Initial Exercise Time and Time Between Data Collection Sessions**

On average, the Exercise group spent 15.7 (SD 1.0) minutes completing the isometric resistance exercise task while the Control group sat quietly for 15.4 (SD 0.8) minutes (Table 3.3). There was no significant difference between the two groups (Independent Samples T-test, $t_{(18)} = -0.589$, $p = 0.563$, 2-tailed).

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean Exercise Time (minutes) (SD)</th>
<th>Mean Time between S1 and S2 (days) (SD)</th>
<th>Mean Time between S2 and S3 (days) (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>15.4 (0.8)</td>
<td>14.1 (0.7)</td>
<td>14.6 (2.1)</td>
</tr>
<tr>
<td>Exercise</td>
<td>15.7 (1.0)</td>
<td>14.3 (0.9)</td>
<td>13.8 (0.6)</td>
</tr>
<tr>
<td>All Participants</td>
<td>15.6 (0.9)</td>
<td>14.2 (0.8)</td>
<td>14.2 (1.6)</td>
</tr>
</tbody>
</table>

Table 3.3: Mean (SD) times between data collection sections for both the Control group and the Exercise group. (S1: Data Collection Session 1; S2: Data Collection Session 2; and S3: Data Collection Session 3).

For all participants, the mean number of days between Data Collection Session 1 (S1) and Data Collection Session 2 (S2) was 14.2 (SD 0.8) days and the mean number of days
between S2 and Data Collection Session 3 (S3) was also 14.2 (SD 1.6) days (Table 3.3). ANOVA revealed no significant differences in the mean time between data collection sessions either within-subjects ($F_{(1,18)} = 1.304$, $p = 0.268$) or between subjects ($F_{(1,18)} = 0.720$, $p = 0.407$).

**Participants’ Adherence to the Home Exercise Programme**

All 10 participants in the Exercise group completed their Training Diaries for the two weeks of the home-based resistance exercise programme. On average, participants in the Exercise group completed 2.4 (SD 0.7; Range 0 to 3) resistance exercise sessions each day, out of a maximum of three sessions per day (80%). This was comprised of an average of 8.2 (SD 4.6; Range 0 to 15) sets of 10 repetitions per day, out of a maximum of 15 sets per day (55%), and an average of 2.7 (SD 2.1; Range 0 to 10) exercise sets per session, out of a maximum requested number of five sets per session (54%). The maximum value of 10 exercise sets per session resulted from one participant (Participant 11) completing 10 sets during the morning session, nil sets during the afternoon session and five sets during the evening session on Day 1 of their participation. Figure 3.9, Panel A illustrates the average number of exercise sets performed by all participants in the Exercise group during each of the three sessions they were asked to perform on each of the 14 days of the home-based resistance exercise programme. The mean number of sets performed during the morning, afternoon and evening sessions was 2.86 (SD 2.06; 57%), 2.01 (SD 2.13; 40%) and 3.28 (SD 2.01; 66%), respectively (Figure 3.9, Panel B). There were no significant differences between the mean number of sets performed in the morning versus the afternoon sessions ($F_{(1,2)} = 6.166$, $p = 0.267$) nor between the morning versus the evening sessions ($F_{(1,2)} = 6.166$, $p = 0.474$). There was however a significant difference between the mean number of sets performed in the afternoon versus the evening sessions ($F_{(1,2)} = 6.166$, $p = 0.021$).
Figure 3.9: Mean number of exercise sets performed by all participants in the Exercise group (n = 10). Error bars show SD of the mean. Panel A: Mean number of exercise sets performed on each of the 14 days of the home-based resistance exercise programme during the morning session (black line), afternoon session (red line) and evening session (blue line). Panel B: Mean number of sets performed during each of the three training sessions for all 14 training days. The asterisk (*) shows a significant difference between the mean number of sets performed in the afternoon versus the evening session ($F_{(1,2)} = 6.166$, $p = 0.021$).

**Tested Variables**

**Jaw Movement Variables**

There were no significant differences in the following jaw movement variables either within subjects or between groups: Jaw Movement Open Time as a Percentage of Chew Cycle Duration ($F_{(3,54)} = 0.638$, $p = 0.594$ for within subjects effects and $F_{(1,18)} = 0.389$, $p =$...
Jaw Movement Close Time as a Percentage of Chew Cycle Duration ($F_{(3,54)} = 2.433$, $p = 0.075$ for within subjects effects and $F_{(1,18)} = 0.107$, $p = 0.747$ for between groups effects). The jaw movement variables that showed significant differences during the study are shown in Figure 3.10, Figure 3.11, Figure 3.12 and Figure 3.13 and are described below.

**Jaw Movement Duration (sec)**

Figure 3.10, Panel A shows the mean (SD) jaw movement durations for the Control group and Exercise group across the data collection sessions. ANOVA revealed a significant within subject effect (Test Time) in the jaw movement duration ($F_{(3,54)} = 15.810$, $p < 0.001$). There was no significant interaction between the data collection session and the group ($F_{(3,54)} = 0.035$, $p = 0.991$). Within subjects contrasts (Test Time) testing showed significant differences in the jaw movement duration between S1-Baseline and S1-Post ($F_{(1,18)} = 37.243$, $p < 0.001$) and S1-Baseline and S2 ($F_{(1,18)} = 19.632$, $p < 0.001$). In the Control group there was a significant reduction in the mean (SD) jaw movement duration from 0.50 s (0.04) at S1-Baseline to 0.43 s (0.03) at S1-Post. There was a similar significant reduction in the mean (SD) jaw movement duration from S1-Baseline to 0.43 s (0.04) at S2. In the Exercise group there was a significant reduction in the mean (SD) jaw movement duration from 0.55 s (0.14) at S1-Baseline to 0.49 s (0.11) at S1-Post. There was a similar significant reduction in the mean (SD) jaw movement duration from S1-Baseline to 0.48 s (0.10) at S2. There were no significant within subjects interactions between the data collection sessions and the groups for any of the three tested contrasts ($p > 0.0167$). There were no significant differences in the between groups effects ($F_{(1,18)} = 2.127$, $p = 0.162$).
Figure 3.10: Jaw movement duration related variables across all data collection sessions in the Control group (blue solid squares and solid line) and the Exercise group (red solid circles and dashed line). Panel A: Mean jaw movement duration. Panel B: Mean chew cycle duration. Panel C: Mean jaw movement open time. Panel D: Mean jaw movement close time. Error bars show SD of the mean. Asterisks (*) depict the significant within subjects contrasts in Panel A, Panel B and Panel C, and the significant within subjects effects between S1-Baseline and S1-Post in Panel D (S1-Baseline: baseline measures in data collection session 1; S1-Post: measures taken after the initial exercise task approximately 15 minutes after S1-Baseline; S2: data collection session 2 undertaken two weeks after data collection session 1; and S3: data collection session 3 undertaken four weeks after data collection session 1).
Chew Cycle Duration (sec)

Figure 3.10, Panel B shows the mean (SD) chew cycle durations for the Control group and Exercise group across the data collection sessions. ANOVA revealed a significant within subject effect (Test Time) in the chew cycle duration ($F_{(3,54)} = 24.393, p < 0.001$). There was no significant interaction between the data collection session and the group ($F_{(3,54)} = 0.795, p = 0.502$). Within subjects contrasts (Test Time) testing showed significant differences in the chew cycle duration between S1-Baseline and S1-Post ($F_{(1,18)} = 27.796, p < 0.001$) and S1-Baseline and S2 ($F_{(1,18)} = 33.368, p < 0.001$). In the Control group there was a significant reduction in the mean (SD) chew cycle duration from 0.70 s (0.11) at S1-Baseline to 0.61 s (0.07) at S1-Post. There was a similar significant reduction in the mean (SD) chew cycle duration from S1-Baseline to 0.60 s (0.05) at S2. In the Exercise group there was a significant reduction in the mean (SD) chew cycle duration from 0.78 s (0.21) at S1-Baseline to 0.70 s (0.18) at S1-Post. There was a significant reduction in the mean (SD) chew cycle duration from S1-Baseline to 0.65 s (0.14) at S2. There were no significant within subjects interactions between the data collection sessions and the groups for any of the three tested contrasts ($p > 0.0167$). There were no significant differences in the between groups effects ($F_{(1,18)} = 1.519, p = 0.234$).

Jaw Movement Open Time (sec)

Figure 3.10, Panel C shows the mean (SD) jaw movement open times for the Control group and Exercise group across the data collection sessions. ANOVA revealed a significant within subject effect (Test Time) in the chew cycle duration ($F_{(3,54)} = 13.655, p < 0.001$). There was no significant interaction between the data collection session and the group ($F_{(3,54)} = 0.022, p = 0.996$). Within subjects contrasts (Test Time) testing showed significant differences in the jaw movement open time between S1-Baseline and S1-Post.
(F(1,18) = 35.333, p < 0.001) and S1-Baseline and S2 (F(1,18) = 22.550, p < 0.001). In the Control group there was a significant reduction in the mean (SD) jaw movement open time from 0.27 s (0.02) at S1-Baseline to 0.23 s (0.02) at S1-Post. There was a similar significant reduction in the mean (SD) jaw movement open time from S1-Baseline to 0.23 s (0.03) at S2. In the Exercise group there was a significant reduction in the mean (SD) jaw movement open time from S1-Baseline to 0.26 s (0.07) at S1-Post. There was a similar significant reduction in the mean (SD) jaw movement open time from S1-Baseline to 0.26 s (0.05) at S2. There were no significant within subjects interactions between the data collection sessions and the groups for any of the three tested contrasts (p > 0.0167). There were no significant differences in the between groups effects (F(1,18) = 2.410, p = 0.138).

**Jaw Movement Close Time (sec)**

Figure 3.10, Panel D shows the mean (SD) jaw movement close times for the Control group and Exercise group across the data collection sessions. ANOVA revealed a significant within subject effect (Test Time) in the jaw movement close time (F(3,54) = 8.039, p < 0.001). There was no significant interaction between the data collection session and the group (F(3,54) = 0.058, p = 0.981). Within subjects contrasts (Test Time) testing showed significant differences in the jaw movement close time between S1-Baseline and S1-Post (F(1,18) = 16.625, p = 0.001). In the Control group there was a significant reduction in the mean (SD) jaw movement close time from 0.22 s (0.03) at S1-Baseline to 0.20 s (0.02) at S1-Post. In the Exercise group there was a significant reduction in the mean (SD) jaw movement close time from 0.24 s (0.06) at S1-Baseline to 0.22 s (0.05) at S1-Post. There were no significant differences in the within subjects interactions between the data collection sessions and the exercise groups for any of the three tested contrasts (p >
There were no significant differences in the between groups effects ($F_{(1,18)} = 1.271$, $p = 0.274$).

**Jaw Movement Duration as a Percentage of Chew Cycle Duration**

Figure 3.11 shows the median (SE) jaw movement durations as a percentage of the chew cycle duration for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects across all the testing sessions ($\chi^2_{(3)} = 8.34$, $p = 0.039$). In the Control group there was a reduction in the median (SE) jaw movement duration as a percentage of the chew cycle duration across all data collection sessions from 75.60% (2.36) at S1-Baseline to 74.29% (1.90) at S1-Post, 73.49% (2.02) at S2 and 72.83% (1.84) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical $p$-value of 0.0167) revealed no significant difference in the Control group between S1-Baseline and S1-Post ($T = 25.00$, $r = -0.057$, $z = -0.255$, $p = 0.799$, 2-tailed), S1-Baseline and S2 ($T = 26.00$, $r = -0.034$, $z = -0.153$, $p = 0.878$, 2-tailed) or S2 and S3 ($T = 25.00$, $r = -0.057$, $z = -0.255$, $p = 0.799$, 2-tailed). In the Exercise group there was a reduction in the median (SE) jaw movement duration as a percentage of the chew cycle duration from 69.81% (1.93) at S1-Baseline to 69.23% (2.03) at S1-Post. After two weeks of the home-based exercise programme the median (SE) jaw movement duration as a percentage of the chew cycle duration increased to 74.67% (1.27) at S2 and after ceasing the home-based exercise programme it increased to 74.81% (1.63) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical $p$-value of 0.0167) revealed no significant difference in the Exercise group between S1-Baseline and S1-Post ($T = 25.00$, $r = -0.103$, $z = -0.459$, $p = 0.646$, 2-tailed), S1-Baseline and S2 ($T = 10.00$, $r = -0.399$, $z = -1.784$, $p = 0.074$, 2-tailed) or S2 and S3 ($T = 22.00$, $r = -0.125$, $z = -0.561$, $p = 0.575$, 2-tailed).
There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

Figure 3.11: Median jaw movement duration as a percentage of chew cycle duration across all data collection sessions in the Control group (blue solid squares and solid line) and the Exercise group (red solid circles and dashed line). Error bars show SE of the median. The asterisk (*) depicts the significant within subjects effects across all the testing sessions (S1-Baseline: baseline measures in data collection session 1; S1-Post: measures taken after the initial exercise task approximately 15 minutes after S1-Baseline; S2: data collection session 2 undertaken two weeks after data collection session 1; and S3: data collection session 3 undertaken four weeks after data collection session 1).
**Jaw Movement Displacement (mm)**

Figure 3.12, Panel A shows the mean (SD) jaw movement displacements for the Control group and Exercise group across the data collection sessions. ANOVA revealed no significant within subject effect (Test Time) in the jaw movement displacement ($F_{(3,54)} = 0.804, p = 0.497$). There was no significant interaction between the data collection session and the group ($F_{(3,54)} = 0.383, p = 0.766$). Within subjects contrasts (Test Time) testing showed significant differences in the chew duration between S1-Baseline and S1-Post ($F_{(1,18)} = 9.322, p = 0.007$). In the Control group there was a significant increase in the mean (SD) jaw movement displacement from 14.83 mm (6.80) at S1-Baseline to 16.32 mm (6.84) at S1-Post. In the Exercise group there was a significant increase in the mean (SD) jaw movement displacement from 15.75 mm (4.88) at S1-Baseline to 17.33 mm (4.78) at S1-Post. There were no other significant differences in the within subjects contrasts ($p > 0.0167$), the within subjects interactions between the data collection sessions and the groups for any of the three tested contrasts ($p > 0.0167$) or in the between groups effects ($F_{(1,18)} = 0.83, p = 0.777$).
Figure 3.12: Jaw movement displacement and velocity related variables across all data collection sessions in the Control group (blue solid squares and solid line) and the Exercise group (red solid circles and dashed line). Error bars show SD of the mean and SE of the median respectively. Panel A: Mean jaw movement displacement. Panel B: Mean jaw movement opening velocity. Panel C: Median jaw movement closing velocity. Panel D: Mean chewing velocity. Asterisks (*) depict the significant within subjects contrasts in Panel A, Panel B and Panel D, and the significant within subjects effects between S1-Baseline and S1-Post in Panel C (S1-Baseline: baseline measures in data collection session 1; S1-Post: measures taken after the initial exercise task approximately 15 minutes after S1-Baseline; S2: data collection session 2 undertaken two weeks after data collection session 1; and S3: data collection session 3 undertaken four weeks after data collection session 1).
**Jaw Movement Opening Velocity (mm.sec⁻¹)**

Figure 3.12, Panel B shows the mean (SD) jaw movement opening velocities for the Control group and Exercise group across the data collection sessions. ANOVA revealed a significant within subject effect (Test Time) in the jaw movement opening velocity ($F_{(3,54)} = 4.644, p = 0.006$). There was no significant interaction between the data collection session and the group ($F_{(3,54)} = 0.459, p = 0.712$). Within subjects contrasts (Test Time) testing showed significant differences in the jaw movement opening velocities between S1-Baseline and S1-Post ($F_{(1,18)} = 42.685, p < 0.001$) and S1-Baseline and S2 ($F_{(1,18)} = 7.289, p = 0.015$). In the Control group there was a significant increase in the mean (SD) jaw movement opening velocity from 30.39 mm.sec⁻¹ (14.08) at S1-Baseline to 37.58 mm.sec⁻¹ (14.87) at S1-Post. There was also a significant increase from S1-Baseline to 35.47 mm.sec⁻¹ (8.54) at S2. In the Exercise group there was a similar significant increase in the mean (SD) jaw movement opening velocity from 29.76 mm.sec⁻¹ (8.24) at S1-Baseline to 36.37 mm.sec⁻¹ (7.88) at S1-Post. There was also a significant increase from S1-Baseline to 35.10 mm.sec⁻¹ (9.66) at S2. There were no significant within subjects interactions between the data collection sessions and the groups for any of the three tested contrasts ($p > 0.0167$). There were no significant differences in the between groups effects ($F_{(1,18)} = 0.205, p = 0.656$).

**Jaw Movement Closing Velocity (mm.sec⁻¹)**

Figure 3.12, Panel C shows the median (SE) jaw movement closing velocities for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in the jaw movement closing velocity across all the testing sessions ($\chi^2_{(3)} = 16.380, p = 0.001$). In the Control group there was an increase in the median (SE) jaw movement closing velocity from 57.63 mm.sec⁻¹ (9.37) at S1-
Baseline to 76.99 mm.sec\(^{-1}\) (9.28) at S1-Post, then reducing to 69.26 mm.sec\(^{-1}\) (6.49) at S2 and increasing to 74.69 mm.sec\(^{-1}\) (8.23) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Control group between S1-Baseline and S1-Post (T = 5.00, r = -0.513, z = -2.293, p = 0.022, 2-tailed), S1-Baseline and S2 (T = 11.00, r = -0.376, z = -1.682, p = 0.093, 2-tailed) or S2 and S3 (T = 11.00, r = -0.376, z = -1.682, p = 0.093, 2-tailed). In the Exercise group there was an increase in the median (SE) jaw movement closing velocity from 69.94 mm.sec\(^{-1}\) (5.36) at S1-Baseline to 82.65 mm.sec\(^{-1}\) (4.94) at S1-Post. After two weeks of the home-based exercise programme the median (SE) jaw movement closing velocity reduced to 78.09 mm.sec\(^{-1}\) (6.68) at S2 and after ceasing the home-based exercise programme it reduced to 77.50 mm.sec\(^{-1}\) (6.80) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S1-Post (T = 2.00, r = -0.581, z = -2.599, p = 0.009, 2-tailed). There was no significant difference in the Exercise group between S1-Baseline and S2 (T = 9.00, r = -0.422, z = -1.886, p = 0.059, 2-tailed) or S2 and S3 (T = 23.00, r = -0.103, z = -0.459, p = 0.646, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

**Chewing Velocity (mm.sec\(^{-1}\))**

Figure 3.12, Panel D shows the mean (SD) chewing velocities for the Control group and Exercise group across the data collection sessions. ANOVA revealed a significant within subject effect (Test Time) in the chewing velocity (F\(_{3,54}\) = 4.644, p = 0.006). There was no significant interaction between the data collection session and the group (F\(_{3,54}\) = 0.459, p = 0.712). Within subjects contrasts (Test Time) testing showed significant differences in the chewing velocity between S1-Baseline and S1-Post (F\(_{1,18}\) = 42.685, p < 0.001) and S1-
Baseline and S2 ($F_{(1,18)} = 7.289$, $p = 0.015$). In the Control group there was a significant increase in the mean (SD) chewing velocity from 60.77 mm.sec$^{-1}$ (28.15) at S1-Baseline to 74.71 mm.sec$^{-1}$ (9.40) at S1-Post. There was also a significant increase from S1-Baseline to 70.95 mm.sec$^{-1}$ (17.07) at S2. In the Exercise group there was a similar significant increase in the mean (SD) chewing velocity from 59.52 mm.sec$^{-1}$ (16.48) at S1-Baseline to 72.74 mm.sec$^{-1}$ (15.75) at S1-Post. There was also a significant increase from S1-Baseline to 70.20 mm.sec$^{-1}$ (19.31) at S2. There were no significant within subjects interactions between the data collection sessions and the groups for any of the three tested contrasts ($p > 0.0167$). There were no significant differences in the between groups effects ($F_{(1,18)} = 0.205$, $p = 0.656$).

**Chewing Frequency (cycles.sec$^{-1}$)**

Figure 3.13 shows the mean (SD) chewing frequency for the Control group and Exercise group across the data collection sessions. ANOVA revealed a significant within subject effect (Test Time) in the chewing frequency ($F_{(3,54)} = 24.081$, $p < 0.001$). There was no significant interaction between the data collection session and the group ($F_{(3,54)} = 0.462$, $p = 0.710$). Within subjects contrasts (Test Time) testing showed significant differences in the chewing frequency between S1-Baseline and S1-Post ($F_{(1,18)} = 26.800$, $p < 0.001$) and S1-Baseline and S2 ($F_{(1,18)} = 40.312$, $p < 0.001$). In the Control group there was a significant increase in the mean (SD) chewing frequency from 1.47 cycles.sec$^{-1}$ (0.18) at S1-Baseline to 1.68 cycles.sec$^{-1}$ (0.19) at S1-Post. There was a similar significant increase in the mean (SD) chewing frequency from S1-Baseline to 1.70 cycles.sec$^{-1}$ (0.13) at S2. In the Exercise group there was a significant increase in the mean (SD) chewing frequency from 1.37 cycles.sec$^{-1}$ (0.36) at S1-Baseline to 1.53 cycles.sec$^{-1}$ (0.37) at S1-Post. There was a similar significant increase in the mean (SD) chewing frequency from S1-Baseline to
1.61 cycles.sec\(^{-1}\) (0.35) at S2. There were no significant within subjects interactions between the data collection sessions and the groups for any of the three tested contrasts (p > 0.0167). There were no significant differences in the between groups effects (F\(_{(1,18)}\) = 0.710, p = 0.410).

![Figure 3.13](image)

Figure 3.13: Mean chewing frequency across all data collection sessions in the Control group (blue solid squares and solid line) and the Exercise group (red solid circles and dashed line). Error bars show SD of the mean. Asterisks (*) depict the significant within subjects contrasts between the baseline measures in data collection session 1 (S1-Baseline) and measures taken after the initial exercise task approximately 15 minutes after S1-Baseline (S1-Post) and data collection session 2 undertaken two weeks after data collection session 1 (S2) respectively (S3: data collection session 3 undertaken four weeks after data collection session 1).
EMG Variables

Time to Peak EMG activity (sec)
There were no significant differences in the time to peak EMG activity for any of the tested muscles (p > 0.05).

EMG Duration (sec)
Figure 3.14 shows the EMG durations of the four tested muscles over the four data collection sessions. The significant findings are described below for each muscle individually.

Ipsilateral Anterior Temporalis
Figure 3.14, Panel A shows the median (SE) EMG duration of the ipsilateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in EMG duration in the ipsilateral anterior temporalis across all the testing sessions ($\chi^2(3) = 16.980, p = 0.001$). In the Control group there was an increase in the median (SE) EMG duration from 0.30 s (0.02) at S1-Baseline to 0.31 s (0.01) at S1-Post, followed by decreases to 0.29 s (0.02) at S2 and 0.27 s (0.02) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Control group between S1-Baseline and S1-Post ($T = 20.00, r = -0.171, z = -0.764, p = 0.445$, 2-tailed), S1-Baseline and S2 ($T = 17.00, r = -0.239, z = -1.070, p = 0.285$, 2-tailed) or S2 and S3 ($T = 9.00, r = -0.422, z = -1.886, p = 0.059$, 2-tailed). In the Exercise group there was a reduction in the median (SE) EMG duration from 0.33 s (0.02) at S1-Baseline to 0.32 s (0.02) at S1-Post. After two weeks of the home-based exercise programme the median (SE) EMG duration increased to 0.33 s (0.02) at S2 and after ceasing the home-based exercise programme it decreased to 0.31 s (0.01) at S3. Post hoc Wilcoxon tests with a Bonferroni correction
Figure 3.14: EMG duration of the tested muscles across all data collection sessions in the Control group (blue solid squares and solid line) and the Exercise group (red solid circles and dashed line). Error bars show SE of the median or SD of the mean respectively. Panel A: Median EMG duration for the ipsilateral anterior temporalis. Panel B: Mean EMG duration for the contralateral anterior temporalis. Panel C: Mean EMG duration for the ipsilateral masseter. Panel D: Mean EMG duration for the contralateral masseter. Asterisks (*) depict the significant within subjects effects across all the testing sessions in Panel A and Panel B and the significant within subjects contrasts between S1 Baseline and S2 in Panel C (S1-Baseline: baseline measures in data collection session 1; S1-Post: measures taken after the initial exercise task approximately 15 minutes after S1-Baseline; S2: data collection session 2 undertaken two weeks after data collection session 1; and S3: data collection session 3 undertaken four weeks after data collection session 1).
(critical p-value of 0.0167) revealed no significant difference in the Exercise group between S1-Baseline and S1-Post (T = 17.00, r = -0.239, z = -1.070, p = 0.285, 2-tailed), S1-Baseline and S2 (T = 7.00, r = -0.467, z = -2.090, p = 0.037, 2-tailed) or S2 and S3 (T = 20.00, r = -0.171, z = -0.764, p = 0.445, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

**Contralateral Anterior Temporalis**

Figure 3.14, Panel B shows the mean (SD) EMG duration of the contralateral anterior temporalis for the Control group and Exercise group across the data collection sessions. ANOVA revealed a significant within subject effect (Test Time) in the EMG duration ($F_{(3,54)} = 4.299, p = 0.009$). In the Control group there was a decrease in the mean (SD) EMG duration across all data collection sessions from 0.32 s (0.07) at S1-Baseline to 0.31 s (0.06) at S1-Post, 0.30 s (0.04) at S2 and 0.29 s (0.05) at S3. In the Exercise group the mean (SD) EMG duration was 0.32 s (0.05) at S1-Baseline and 0.32 s (0.06) at S1-Post. After two weeks of the home-based exercise programme the mean (SD) EMG duration decreased to 0.31 s (0.06) at S2 and after ceasing the home-based exercise programme it decreased to 0.29 s (0.05) at S3. There was no significant interaction between the data collection session and the group ($F_{(3,54)} = 0.101, p = 0.959$). There were no significant differences in the within subjects contrasts (p > 0.0167), the within subjects interactions between the data collection sessions and the groups for any of the three tested contrasts (p > 0.0167) or in the between groups effects ($F_{(1,18)} = 0.050, p = 0.825$).

**Ipsilateral Masseter**

Figure 3.14, Panel C shows the mean (SD) EMG duration of the ipsilateral masseter for the Control group and Exercise group across the data collection sessions. ANOVA revealed a significant within subject effect (Test Time) in the EMG duration ($F_{(3,54)} = 5.409, p = $
0.003). There was no significant interaction between the data collection session and the group (F(3,54) = 2.057, p = 0.117). Within subjects contrasts (Test Time) testing showed significant differences in the EMG duration between S1-Baseline and S2 (F(1,18) = 7.081, p = 0.016). In the Control group the mean (SD) EMG duration was 0.34 s (0.06) at S1-Baseline and 0.32 s (0.05) at S2. In the Exercise group the mean (SD) EMG duration was 0.38 s (0.06) at S1-Baseline and 0.33 s (0.06) at S2. There were no significant within subjects interactions between the data collection sessions and the groups for any of the three tested contrasts (p > 0.0167). There were no significant differences in the between groups effects (F(1,18) = 2.183, p = 0.157).

**Contralateral Masseter**
Figure 3.14, Panel D shows the mean (SD) EMG duration of the contralateral masseter for the Control group and Exercise group across the data collection sessions. ANOVA revealed no significant within subject effect (Test Time) in the EMG duration of the contralateral masseter (F(3,54) = 0.379, p = 0.768). There was no significant interaction between the data collection session and the group (F(3,54) = 0.821, p = 0.488). There were no significant differences in the within subjects contrasts (p > 0.0167), the within subjects interactions between the data collection sessions and the groups for any of the 3 tested contrasts (p > 0.05) or in the between groups effects (F(1,18) = 0.834, p = 0.373).

**EMG Onset Relative to Jaw Movement Onset (sec)**
Figure 3.15 shows the EMG Onset Relative to Jaw Movement Onset of the four tested muscles over the four data collection sessions. The significant findings are described below for each muscle individually.
Figure 3.15: EMG Onset Relative to Jaw Movement Onset of the tested muscles across all data collection sessions in the Control group (blue solid squares and solid line) and the Exercise group (red solid circles and dashed line). Error bars show SE of the median or SD of the mean respectively. Panel A: Median EMG Onset Relative to Jaw Movement Onset for the ipsilateral anterior temporalis. Panel B: Median EMG Onset Relative to Jaw Movement Onset for the contralateral anterior temporalis. Panel C: Median EMG Onset Relative to Jaw Movement Onset for the ipsilateral masseter. Panel D: Mean EMG Onset Relative to Jaw Movement Onset for the contralateral masseter. Asterisks (*) depict the significant within subjects effects across all the testing sessions in Panel A, Panel B and Panel C and the significant within subjects contrasts between the baseline measures in data collection session 1 (S1-Baseline) and measures taken after the initial exercise task approximately 15 minutes after S1-Baseline (S1-Post) and data collection session 2 undertaken two weeks after data collection session 1 (S2) respectively (S3: data collection session 3 undertaken four weeks after data collection session 1).
**Ipsilateral Anterior Temporalis**

Figure 3.15, Panel A shows the median (SE) EMG onset relative to jaw movement onset of the ipsilateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in EMG onset relative to jaw movement onset in the ipsilateral anterior temporalis across all the testing sessions ($\chi^2(3) = 23.760$, $p < 0.001$). In the Control group there was a decrease in the median (SE) EMG onset relative to jaw movement onset from 0.33 s (0.02) at S1-Baseline to 0.25 s (0.04) at S1-Post and to 0.24 s (0.03) at S2 followed by an increase to 0.27 s (0.02) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical $p$-value of 0.0167) revealed a significant difference in the Control group between S1-Baseline and S2 ($T = 2.00$, $r = -0.581$, $z = -2.599$, $p = 0.009$, 2-tailed). There was no significant difference in the Control group between S1-Baseline and S1-Post ($T = 10.00$, $r = -0.399$, $z = -1.784$, $p = 0.074$, 2-tailed) or S2 and S3 ($T = 11.00$, $r = -0.376$, $z = -1.682$, $p = 0.093$, 2-tailed). In the Exercise group there was a decrease in the median (SE) EMG onset relative to jaw movement onset from 0.38 s (0.04) at S1-Baseline to 0.30 s (0.03) at S1-Post. After two weeks of the home-based exercise programme the median (SE) EMG onset relative to jaw movement onset decreased to 0.29 s (0.03) at S2 and after ceasing the home-based exercise programme it decreased to 0.29 s (0.04) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical $p$-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S1-Post ($T = 0.00$, $r = -0.627$, $z = -2.803$, $p = 0.005$, 2-tailed) and S1-Baseline and S2 ($T = 1.00$, $r = -0.604$, $z = -2.701$, $p = 0.007$, 2-tailed). There was no significant difference in the Exercise group between S2 and S3 ($T = 21.00$, $r = -0.148$, $z = -0.663$, $p = 0.508$, 2-tailed). There were no significant between groups effects at any of the testing sessions ($p > 0.05$, 2-tailed).
Contralateral Anterior Temporalis

Figure 3.15, Panel B shows the median (SE) EMG onset relative to jaw movement onset of the contralateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in EMG onset relative to jaw movement onset in the contralateral anterior temporalis across all the testing sessions ($\chi^2(3) = 23.460, p < 0.001$). In the Control group there was a decrease in the median (SE) EMG onset relative to jaw movement onset from 0.33 s (0.01) at S1-Baseline to 0.24 s (0.01) at S1-Post followed by an increase to 0.26 s (0.03) at S2 and to 0.27 s (0.01) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Control group between S1-Baseline and S1-Post ($T = 0.00, r = -0.627, z = -2.803, p = 0.005, 2$-tailed) and S1-Baseline and S2 ($T = 1.00, r = -0.604, z = -2.701, p = 0.007, 2$-tailed). There was no significant difference in the Control group between S2 and S3 ($T = 22.00, r = -0.125, z = -0.561, p = 0.575, 2$-tailed). In the Exercise group there was a significant decrease in the median (SE) EMG onset relative to jaw movement onset from 0.37 s (0.05) at S1-Baseline to 0.29 s (0.04) at S1-Post. After two weeks of the home-based exercise programme the median (SE) EMG onset relative to jaw movement onset increased to 0.30 s (0.06) at S2 and after ceasing the home-based exercise programme it decreased to 0.29 s (0.04) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S1-Post ($T = 2.00, r = -0.581, z = -2.599, p = 0.009, 2$-tailed). There was no significant difference in the Exercise group between S1-Baseline and S2 ($T = 14.00, r = -0.308, z = -1.376, p = 0.007, 2$-tailed) or S2 and S3 ($T = 17.00, r = -0.239, z = -0.239, p = 0.285, 2$-tailed). There was a significant between groups effect at S2 (Mann-Whitney U = 24.000, $p = 0.049, 2$-tailed).
There were no other significant between groups effects at any of the other testing sessions (p > 0.05, 2-tailed).

**Ipsilateral Masseter**

Figure 3.15, Panel C shows the median (SE) EMG onset relative to jaw movement onset of the ipsilateral masseter for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in EMG onset relative to jaw movement onset in the ipsilateral masseter across all the testing sessions ($\chi^2(3) = 21.180, p < 0.001$). In the Control group there was a decrease in the median (SE) EMG onset relative to jaw movement onset from 0.28 s (0.02) at S1-Baseline to 0.23 s (0.02) at S1-Post and to 0.22 s (0.03) at S2 followed by an increase to 0.24 s (0.02) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Control group between S1-Baseline and S1-Post (T = 10.00, r = -0.399, z = -1.784, p = 0.074, 2-tailed), S1-Baseline and S2 (T = 8.00, r = -0.445, z = -1.988, p = 0.047, 2-tailed) or S2 and S3 (T = 39.00, r = -0.262, z = -1.172, p = 0.241, 2-tailed). In the Exercise group there was a decrease in the median (SE) EMG onset relative to jaw movement onset from 0.33 s (0.04) at S1-Baseline to 0.27 s (0.03) at S1-Post. After two weeks of the home-based exercise programme the median (SE) EMG onset relative to jaw movement onset was 0.27 s (0.07) at S2 and after ceasing the home-based exercise programme it decreased to 0.23 s (0.03) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S1-Post (T = 1.00, r = -0.604, z = -2.701, p = 0.007, 2-tailed). There was no significant difference in the Exercise group between S1-Baseline and S2 (T = 10.00, r = -0.399, z = -1.784, p = 0.074, 2-tailed) or S2 and S3 (T = 12.00, r =
-0.353, z = -1.580, p = 0.114, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

**Contralateral Masseter**
Figure 3.15, Panel D shows the mean (SD) EMG onset relative to jaw movement onset of the contralateral masseter for the Control group and Exercise group across the data collection sessions. ANOVA revealed a significant within subject effect (Test Time) in the EMG onset relative to jaw movement onset of the contralateral masseter (F(3,54) = 12.446, p < 0.001). Within subjects contrasts (Test Time) testing showed significant differences in the EMG onset relative to jaw movement between S1-Baseline and S1-Post (F(1,18) = 58.455, p < 0.001) and S1-Baseline and S2 (F(1,18) = 19.891, p < 0.001). In the Control group there was a significant decrease in the mean (SD) EMG onset relative to jaw movement onset from 0.30 s (0.03) at S1-Baseline to 0.23 s (0.03) at S1-Post and to 0.19 s (0.10) at S2. In the Exercise group there was a significant decrease in the mean (SD) EMG onset relative to jaw movement onset from 0.35 s (0.12) at S1-Baseline to 0.29 s (0.11) at S1-Post. After two weeks of the home-based exercise programme the mean (SD) EMG onset relative to jaw movement onset was 0.28 s (0.09) at S2. There were no significant within subjects interactions between the data collection sessions and the groups for any of the three tested contrasts (p > 0.0167). There were no significant differences in the between groups effects (F(1,18) = 2.983, p = 0.101).

**Time to Peak EMG activity relative to Jaw Movement Onset (sec)**
Figure 3.16 shows the Time to Peak EMG Activity Relative to Jaw Movement Onset of the four tested muscles over the four data collection sessions. The significant findings are described below for each muscle individually.
Figure 3.16: Time to Peak EMG Activity Relative to Jaw Movement Onset of the tested muscles across all data collection sessions in the Control group (blue solid squares and solid line) and the Exercise group (red solid circles and dashed line). Error bars show SE of the median. Panel A: Median EMG Onset Relative to Jaw Movement Onset for the ipsilateral anterior temporalis. Panel B: Median EMG Onset Relative to Jaw Movement Onset for the contralateral anterior temporalis. Panel C: Median EMG Onset Relative to Jaw Movement Onset for the ipsilateral masseter. Panel D: Median EMG Onset Relative to Jaw Movement Onset for the contralateral masseter. Asterisks (*) depict the significant within subjects effects across all the testing sessions (S1-Baseline: baseline measures in data collection session 1; S1-Post: measures taken after the initial exercise task approximately 15 minutes after S1-Baseline; S2: data collection session 2 undertaken two weeks after data collection session 1; and S3: data collection session 3 undertaken four weeks after data collection session 1).
**Ipsilateral Anterior Temporalis**

Figure 3.16, Panel A shows the median (SE) time to peak EMG activity relative to jaw movement onset of the ipsilateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in time to peak EMG activity relative to jaw movement onset in the ipsilateral anterior temporalis across all the testing sessions ($\chi^2(3) = 25.620, p < 0.001$). In the Control group there was a decrease in the median (SE) time to peak EMG activity relative to jaw movement onset from 0.47 s (0.02) at S1-Baseline to 0.43 s (0.02) at S1-Post and 0.41 s (0.03) at S2 and increased to 0.42 s (0.02) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Control group between S1-Baseline and S1-Post ($T = 0.00, r = -0.627, z = -2.803, p = 0.005, 2$-tailed) and between S1-Baseline and S2 ($T = 1.00, r = -0.604, z = -2.701, p = 0.007, 2$-tailed). There was no significant difference in the Control group between S2 and S3 ($T = 22.00, r = -0.125, z = -0.561, p = 0.575, 2$-tailed).

In the Exercise group there was a decrease in the median (SE) time to peak EMG activity relative to jaw movement onset from 0.54 s (0.05) at S1-Baseline to 0.47 s (0.04) at S1-Post. After two weeks of the home-based exercise programme the median (SE) time to peak EMG activity relative to jaw movement onset increased to 0.49 s (0.03) at S2 and after ceasing the home-based exercise programme it decreased to 0.46 s (0.04) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S1-Post ($T = 2.00, r = -0.581, z = -2.599, p = 0.009, 2$-tailed) and between S1-Baseline and S2 ($T = 2.00, r = -0.581, z = -2.599, p = 0.009, 2$-tailed). There was no significant difference in the Exercise group between S2 and S3 ($T = 21.00, r = -0.148, z = -0.663, p = 0.508, 2$-tailed). There were no significant between groups effects at any of the testing sessions ($p > 0.05, 2$-tailed).
**Contralateral Anterior Temporalis**

Figure 3.16, Panel B shows the median (SE) time to peak EMG activity relative to jaw movement onset of the contralateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in time to peak EMG activity relative to jaw movement onset in the contralateral anterior temporalis across all the testing sessions ($\chi^2(3) = 27.060, p < 0.001$).

In the Control group there was a decrease in the median (SE) time to peak EMG activity relative to jaw movement onset from 0.48 s (0.02) at S1-Baseline to 0.44 s (0.02) at S1-Post and 0.42 s (0.03) at S2 and 0.42 s (0.02) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Control group between S1-Baseline and S1-Post ($T = 0.00, r = -0.627, z = -2.803, p = 0.005$, 2-tailed) and between S1-Baseline and S2 ($T = 0.00, r = -0.627, z = -2.803, p = 0.005$, 2-tailed). There was no significant difference in the Control group between S2 and S3 ($T = 23.00, r = -0.103, z = -0.459, p = 0.646$, 2-tailed). In the Exercise group there was a decrease in the median (SE) EMG onset relative to jaw movement onset from 0.57 s (0.05) at S1-Baseline to 0.50s (0.04) at S1-Post. After two weeks of the home-based exercise programme the median (SE) time to peak EMG activity relative to jaw movement onset was 0.50 s (0.07) at S2 and after ceasing the home-based exercise programme it decreased to 0.46 s (0.04) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant differences in the Exercise group between S1-Baseline and S1-Post ($T = 5.00, r = -0.513, z = -2.293, p = 0.022$, 2-tailed), S1-Baseline and S2 ($T = 12.00, r = -0.353, z = -1.580, p = 0.114$, 2-tailed) or S2 and S3 ($T = 9.00, r = -0.422, z = -1.886, p = 0.059$, 2-tailed). There were no significant between groups effects at any of the three testing sessions ($p > 0.05$, 2-tailed).
Ipsilateral Masseter

Figure 3.16, Panel C shows the median (SE) time to peak EMG activity relative to jaw movement onset of the ipsilateral masseter for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in time to peak EMG activity relative to jaw movement onset in the ipsilateral masseter across all the testing sessions ($\chi^2(3) = 25.740$, $p < 0.001$). In the Control group there was a decrease in the median (SE) time to peak EMG activity relative to jaw movement onset from 0.47 s (0.02) at S1-Baseline to 0.44 s (0.01) at S1-Post and 0.42 s (0.03) at S2 and 0.42 s (0.02) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical $p$-value of 0.0167) revealed a significant difference in the Control group between S1-Baseline and S1-Post ($T = 0.00, r = -0.627, z = -2.803, p = 0.005, 2$-tailed) and between S1-Baseline and S2 ($T = 0.00, r = -0.627, z = -2.803, p = 0.005, 2$-tailed). There was no significant difference in the Control group between S2 and S3 ($T = 22.00, r = -0.125, z = -0.561, p = 0.575, 2$-tailed). In the Exercise group there was a decrease in the median (SE) time to peak EMG activity relative to jaw movement onset from 0.55 s (0.05) at S1-Baseline to 0.50s (0.04) at S1-Post. After two weeks of the home-based exercise programme the median (SE) time to peak EMG activity relative to jaw movement onset was 0.49 s (0.07) at S2 and after ceasing the home-based exercise programme it decreased to 0.47 s (0.04) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical $p$-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S1-Post ($T = 4.00, r = -0.536, z = -2.395, p = 0.0166, 2$-tailed). There was no significant difference in the Exercise group between S1-Baseline and S2 ($T = 11.00, r = -0.376, z = -1.682, p = 0.093, 2$-tailed) or S2 and S3 ($T = 13.00, r = -0.330, z = -1.478, p = 0.139, 2$-tailed). There were no significant between groups effects at any of the testing sessions ($p > 0.05, 2$-tailed).
Contralateral Masseter

Figure 3.16, Panel D shows the median (SE) time to peak EMG activity relative to jaw movement onset of the contralateral masseter for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in time to peak EMG activity relative to jaw movement onset in the contralateral masseter across all the testing sessions ($\chi^2(3) = 28.38, p < 0.01$). In the Control group there was a decrease in the median (SE) time to peak EMG activity relative to jaw movement onset from 0.48 s (0.02) at S1-Baseline to 0.41 s (0.02) at S1-Post and 0.38 s (0.03) at S2 and increased to 0.42 s (0.02) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Control group between S1-Baseline and S1-Post ($T = 0.00, r = -0.627, z = -2.803, p = 0.005, 2$-tailed) and between S1-Baseline and S2 ($T = 0.00, r = -0.627, z = -2.803, p = 0.005, 2$-tailed). There was no significant difference in the Control group between S2 and S3 ($T = 23.00, r = -0.103, z = -0.459, p = 0.646, 2$-tailed). In the Exercise group there was a decrease in the median (SE) time to peak EMG activity relative to jaw movement onset from 0.51 s (0.05) at S1-Baseline to 0.48 s (0.04) at S1-Post. After two weeks of the home-based exercise programme the median (SE) time to peak EMG activity relative to jaw movement onset was 0.48 s (0.03) at S2 and after ceasing the home-based exercise programme it decreased to 0.44 s (0.04) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S2 ($T = 4.00, r = -0.536, z = -2.395, p = 0.0166, 2$-tailed). There was no significant difference in the Exercise group between S1-Baseline and S1-Post ($T = 5.00, r = -0.513, z = -2.395, p = 0.022, 2$-tailed) or between S2 and S3 ($T = 18.00, r = -0.216, z = -0.968, p = 0.333, 2$-tailed). There were no significant between groups effects at any of the testing sessions ($p > 0.05, 2$-tailed).
**EMG offset relative to Jaw Movement Onset (sec)**

Figure 3.17 shows the EMG Offset Relative to Jaw Movement Onset of the four tested muscles over the four data collection sessions. The significant findings are described below for each muscle individually.

**Ipsilateral Anterior Temporalis**

Figure 3.17, Panel A shows the median (SE) EMG offset relative to jaw movement onset of the ipsilateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in EMG offset relative to jaw movement onset in the ipsilateral anterior temporalis across all the testing sessions ($\chi^2(3) = 27.180, p < 0.001$). In the Control group there was a decrease in the median (SE) EMG offset relative to jaw movement onset from 0.61 s (0.03) at S1-Baseline to 0.54 s (0.05) at S1-Post, 0.53 s (0.03) at S2 and to 0.51 s (0.02) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Control group between S1-Baseline and S2 ($T = 0.00, r = -0.627, z = -2.803, p = 0.005, 2$-tailed). There was no significant difference in the Control group between S1-Baseline and S1-Post ($T = 10.00, r = -0.399, z = -1.784, p = 0.074, 2$-tailed) or S2 and S3 ($T = 25.00, r = -0.057, z = -0.255, p = 0.799, 2$-tailed). In the Exercise group there was a decrease in the median (SE) EMG offset relative to jaw movement onset from 0.68 s (0.06) at S1-Baseline to 0.61 s (0.05) at S1-Post. After two weeks of the home-based exercise programme the median (SE) EMG offset relative to jaw movement onset increased to 0.63 s (0.04) at S2 and after ceasing the home-based exercise programme it decreased to 0.61 s (0.04) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S1-Post ($T = 4.00, r = -0.536, z = -2.395, p = 0.0166, 2$-tailed) and
Figure 3.17: Median EMG Offset Relative to Jaw Movement Onset of the tested muscles across all data collection sessions in the Control group (blue solid squares and solid line) and the Exercise group (red solid circles and dashed line). Error bars show SE of the median. Panel A: Median EMG Onset Relative to Jaw Movement Onset for the ipsilateral anterior temporalis. Panel B: Median EMG Onset Relative to Jaw Movement Onset for the contralateral anterior temporalis. Panel C: Median EMG Onset Relative to Jaw Movement Onset for the ipsilateral masseter. Panel D: Median EMG Onset Relative to Jaw Movement Onset for the contralateral masseter. Asterisks (*) depict the significant within subjects effects across all the testing sessions (S1-Baseline: baseline measures in data collection session 1; S1-Post: measures taken after the initial exercise task approximately 15 minutes after S1-Baseline; S2: data collection session 2 undertaken two weeks after data collection session 1; and S3: data collection session 3 undertaken four weeks after data collection session 1).
between S1-Baseline and S2 (T = 1.00, r = -0.604, z = -2.701, p = 0.007, 2-tailed). There was no significant difference in the Exercise group between S2 and S3 (T = 19.00, r = -0.194, z = -0.866, p = 0.386, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

**Contralateral Anterior Temporalis**

Figure 3.17, Panel B shows the median (SE) EMG offset relative to jaw movement onset of the contralateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in EMG offset relative to jaw movement onset in the contralateral anterior temporalis across all the testing sessions ($\chi^2_3 = 32.280$, $p < 0.001$). In the Control group there was a decrease in the median (SE) EMG offset relative to jaw movement onset from 0.63 s (0.02) at S1-Baseline to 0.57 s (0.02) at S1-Post, 0.55 s (0.03) at S2 and to 0.53 s (0.02) at S3.

*Post hoc* Wilcoxon tests with a Bonferroni correction (critical $p$-value of 0.0167) revealed a significant difference in the Control group between S1-Baseline and S1-Post ($T = 0.00$, $r = -0.627$, $z = -2.803$, $p = 0.005$, 2-tailed) and between S1-Baseline and S2 ($T = 0.00$, $r = -0.627$, $z = -2.803$, $p = 0.005$, 2-tailed). There was no significant difference in the Control group between S2 and S3 ($T = 25.00$, $r = -0.057$, $z = -0.255$, $p = 0.799$, 2-tailed). In the Exercise group there was a decrease in the median (SE) EMG offset relative to jaw movement onset from 0.72 s (0.05) at S1-Baseline to 0.63s (0.05) at S1-Post. After two weeks of the home-based exercise programme the median (SE) EMG offset relative to jaw movement onset increased to 0.64 s (0.07) at S2 and after ceasing the home-based exercise programme it decreased to 0.62 s (0.04) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical $p$-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S1-Post ($T = 4.00$, $r = -0.536$, $z = -2.395$, $p = 0.0166$, 2-
tailed). There was no significant difference in the Exercise group between S1-Baseline and S2 (T = 10.00, r = -0.399, z = -1.784, p = 0.074, 2-tailed) or between S2 and S3 (T = 8.00, r = -0.445, z = -1.988, p = 0.047, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

Ipsilateral Masseter
Figure 3.17, Panel C shows the median (SE) EMG offset relative to jaw movement onset of the ipsilateral masseter for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in EMG offset relative to jaw movement onset in the ipsilateral masseter across all the testing sessions ($\chi^2(3) = 24.300, p < 0.001$). In the Control group there was a decrease in the median (SE) EMG offset relative to jaw movement onset from 0.59 s (0.03) at S1-Baseline to 0.55 s (0.02) at S1-Post and 0.54 s (0.03) at S2 and to 0.53 s (0.02) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Control group between S1-Baseline and S1-Post (T = 8.00, r = -0.445, z = -1.988, p = 0.047, 2-tailed), S1-Baseline and S2 (T = 7.00, r = -0.467, z = -2.090, p = 0.037, 2-tailed) or between S2 and S3 (T = 25.00, r = -0.057, z = -0.255, p = 0.799, 2-tailed). In the Exercise group there was a decrease in the median (SE) EMG offset relative to jaw movement onset from 0.71 s (0.06) at S1-Baseline to 0.63 s (0.05) at S1-Post. After two weeks of the home-based exercise programme the median (SE) EMG offset relative to jaw movement onset increased to 0.64 s (0.07) at S2 and after ceasing the home-based exercise programme it decreased to 0.62 s (0.05) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Exercise group between S1-Baseline and S1-Post (T = 5.00, r = -0.513, z = -2.293, p = 0.022, 2-tailed), S1-Baseline and S2 (T = 11.00, r = -0.376, z = -1.682, p = 0.093, 2-tailed)
or between S2 and S3 (T = 11.00, r = -0.376, z = -1.682, p = 0.093, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

**Contralateral Masseter**

Figure 3.17, Panel D shows the median (SE) EMG offset relative to jaw movement onset of the contralateral masseter for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in EMG offset relative to jaw movement onset in the contralateral masseter across all the testing sessions ($\chi^2(3) = 24.900, p < 0.001$). In the Control group there was a decrease in the median (SE) EMG offset relative to jaw movement onset from 0.59 s (0.03) at S1-Baseline to 0.55 s (0.02) at S1-Post, 0.50 s (0.03) at S2 and to 0.50 s (0.02) at S3. **Post hoc** Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Control group between S1-Baseline and S1-Post ($T = 0.00, r = -0.627, z = -2.803, p = 0.005, 2$-tailed) and between S1-Baseline and S2 ($T = 1.00, r = -0.604, z = -2.701, p = 0.007, 2$-tailed). There was no significant difference in the Control group between S2 and S3 ($T = 22.00, r = -0.125, z = -0.561, p = 0.575, 2$-tailed). In the Exercise group there was a decrease in the median (SE) EMG offset relative to jaw movement onset from 0.66 s (0.06) at S1-Baseline to 0.60 s (0.05) at S1-Post. After two weeks of the home-based exercise programme the median (SE) EMG offset relative to jaw movement onset increased to 0.61 s (0.04) at S2 and after ceasing the home-based exercise programme it decreased to 0.60 s (0.04) at S3. **Post hoc** Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S2 ($T = 3.00, r = -0.558, z = -2.497, p = 0.013, 2$-tailed). There was no significant difference in the Exercise group between S1-Baseline and S1-Post ($T = 5.00, r = -0.513, z = -2.293, p = 0.022, 2$-tailed) or S2 and S3 ($T = 26.00, r =
-0.034, \( z = -0.153, p = 0.878 \), 2-tailed). There were no significant between groups effects at any of the testing sessions (\( p > 0.05, \) 2-tailed).

**EMG offset relative to Jaw Movement Offset (sec)**

Figure 3.18 shows the EMG Offset Relative to Jaw Movement Offset of the four tested muscles over the four data collection sessions. The significant findings are described below for each muscle individually.

**Ipsilateral Anterior Temporalis**

Figure 3.18, Panel A shows the median (SE) EMG offset relative to jaw movement offset of the ipsilateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in EMG offset relative to jaw movement offset in the ipsilateral anterior temporalis across all the testing sessions \( (\chi^2(3) = 21.660, p < 0.001) \). In the Control group there was a decrease in the median (SE) EMG offset relative to jaw movement offset from 0.12 s (0.02) at S1-Baseline to 0.10 s (0.05) at S1-Post and it was 0.10 s (0.04) at S2 and 0.10 s (0.01) at S3.

*Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Control group between S1-Baseline and S1-Post (\( T = 17.00, r = -0.239, z = -1.070, p = 0.285, \) 2-tailed), S1-Baseline and S2 (\( T = 6.00, r = -0.490, z = -2.191, p = 0.028, \) 2-tailed) or S2 and S3 (\( T = 23.00, r = -0.103, z = -0.459, p = 0.646, \) 2-tailed). In the Exercise group there was a decrease in the median (SE) EMG offset relative to jaw movement offset from 0.16 s (0.01) at S1-Baseline to 0.13 s (0.02) at S1-Post. After two weeks of the home-based exercise programme the median (SE) EMG offset relative to jaw movement offset decreased to 0.12 s (0.01) at S2 and after ceasing the home-based exercise programme it was 0.12 s (0.01) at S3. *Post hoc* Wilcoxon tests with a Bonferroni
correction (critical p-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S2 (T = 0.00, r = -0.627, z = -2.805, p = 0.005, 2-tailed).

Figure 3.18: Median EMG Offset Relative to Jaw Movement Offset of the ipsilateral anterior temporalis (Panel A), contralateral anterior temporalis (Panel B), ipsilateral masseter (Panel C) and contralateral masseter (Panel D) across all data collection sessions in the Control group (blue solid squares and solid line) and the Exercise group (red solid circles and dashed line). Error bars show SE of the median. Asterisks (*) depict the significant within subjects effects across all the testing sessions (S1-Baseline: baseline measures in data collection session 1; S1-Post: measures taken after the initial exercise task approximately 15 minutes after S1-Baseline; S2: data collection session 2 undertaken two weeks after data collection session 1; and S3: data collection session 3 undertaken four weeks after data collection session 1).
There was no significant difference in the Exercise group between S1-Baseline and S1-Post (T = 13.00, r = -0.330, z = -1.478, p = 0.139, 2-tailed) or S2 and S3 (T = 27.00, r = -0.011, z = -0.051, p = 0.959, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

**Contralateral Anterior Temporalis**

Figure 3.18, Panel B shows the median (SE) EMG offset relative to jaw movement offset of the contralateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in EMG offset relative to jaw movement offset in the contralateral anterior temporalis across all the testing sessions ($\chi^2(3) = 18.540, p < 0.001$). In the Control group there was a decrease in the median (SE) EMG offset relative to jaw movement offset from 0.13 s (0.02) at S1-Baseline to 0.12 s (0.01) at S1-Post and it was 0.12 s (0.04) at S2 and 0.12 s (0.01) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Control group between S1-Baseline and S1-Post (T = 10.00, r = -0.399, z = -1.784, p = 0.074, 2-tailed), S1-Baseline and S2 (T = 5.00, r = -0.513, z = -2.293, p = 0.022, 2-tailed) or S2 and S3 (T = 25.00, r = -0.057, z = -0.255, p = 0.799, 2-tailed). In the Exercise group there was a significant decrease in the median (SE) EMG offset relative to jaw movement offset from 0.18 s (0.01) at S1-Baseline to 0.14 s (0.02) at S1-Post. After two weeks of the home-based exercise programme the median (SE) EMG offset relative to jaw movement offset was 0.14 s (0.05) at S2 and after ceasing the home-based exercise programme it was 0.14 s (0.01) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Exercise group between S1-Baseline and S1-Post (T = 15.00, r = -0.285, z = -1.274, p = 0.203, 2-tailed), S1-Baseline and S2 (T = 10.00, r = -0.399, z = -1.784, p = 0.074, 2-tailed)
or S2 and S3 (T = 16.00, r = -0.262, z = -1.172, p = 0.241, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

**Ipsilateral Masseter**

Figure 3.18, Panel C shows the median (SE) EMG offset relative to jaw movement offset of the ipsilateral masseter for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in EMG offset relative to jaw movement offset in the ipsilateral masseter across all the testing sessions ($\chi^2(3) = 15.120, p = 0.002$). In the Control group there was a decrease in the median (SE) EMG offset relative to jaw movement offset from 0.12 s (0.02) at S1-Baseline to 0.11 s (0.01) at S1-Post and it was 0.11 s (0.04) at S2 and 0.12 s (0.01) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Control group between S1-Baseline and S1-Post (T = 26.00, r = -0.034, z = -0.153, p = 0.878, 2-tailed), S1-Baseline and S2 (T = 19.00, r = -0.194, z = -0.866, p = 0.386, 2-tailed) or S2 and S3 (T = 26.00, r = -0.034, z = -0.878, p = 0.878, 2-tailed). In the Exercise group there was a decrease in the median (SE) EMG offset relative to jaw movement offset from 0.18 s (0.01) at S1-Baseline to 0.15 s (0.02) at S1-Post. After two weeks of the home-based exercise programme the median (SE) EMG offset relative to jaw movement offset was 0.13 s (0.05) at S2 and after ceasing the home-based exercise programme it was 0.14 s (0.02) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Exercise group between S1-Baseline and S1-Post (T = 13.00, r = -0.330, z = -1.478, p = 0.139, 2-tailed), S1-Baseline and S2 (T = 10.00, r = -0.399, z = -1.784, p = 0.074, 2-tailed) or S2 and S3 (T = 27.00, r = -0.011, z = -0.051, p = 0.959, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).
**Contralateral Masseter**

Figure 3.18, Panel D shows the median (SE) EMG offset relative to jaw movement offset of the contralateral masseter for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in EMG offset relative to jaw movement offset in the contralateral masseter across all the testing sessions ($\chi^2(3) = 12.480, p = 0.006$). In the Control group there was a decrease in the median (SE) EMG offset relative to jaw movement offset from 0.10 s (0.02) at S1-Baseline to 0.09 s (0.02) at S1-Post and it was 0.10 s (0.03) at S2 and 0.10 s (0.02) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical $p$-value of 0.0167) revealed no significant difference in the Control group between S1-Baseline and S1-Post ($T = 24.00, r = -0.080, z = -0.357, p = 0.721, 2$-tailed), S1-Baseline and S2 ($T = 12.00, r = -0.353, z = -1.580, p = 0.114, 2$-tailed) or S2 and S3 ($T = 18.00, r = -0.216, z = -0.968, p = 0.333, 2$-tailed). In the Exercise group there was a decrease in the median (SE) EMG offset relative to jaw movement offset from 0.13 s (0.01) at S1-Baseline to 0.12 s (0.01) at S1-Post. After two weeks of the home-based exercise programme the median (SE) EMG offset relative to jaw movement offset was 0.11 s (0.01) at S2 and after ceasing the home-based exercise programme it was 0.12 s (0.01) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical $p$-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S2 ($T = 4.00, r = -0.536, z = -2.395, p = 0.0166, 2$-tailed). There was no significant difference in the Exercise group between S1-Baseline and S1-Post ($T = 16.00, r = -0.262, z = -1.172, p = 0.241, 2$-tailed) or S2 and S3 ($T = 13.00, r = -0.330, z = -1.478, p = 0.139, 2$-tailed). There were no significant between groups effects at any of the testing sessions ($p > 0.05, 2$-tailed).
Relative EMG Onset as a Percentage of Chew Duration (%)

Figure 3.19 shows the Relative EMG Onset as a Percentage of Chew Duration of the four tested muscles over the four data collection sessions. The significant findings are described below for each muscle individually.

Ipsilateral Anterior Temporalis

Figure 3.19, Panel A shows the median (SE) relative EMG onset as a percentage of chew duration of the ipsilateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in the relative EMG onset as a percentage of chew duration in the ipsilateral anterior temporalis across all the testing sessions ($\chi^2(3) = 15.540, p = 0.001$). In the Control group there was a decrease in the median (SE) relative EMG onset as a percentage of chew duration from 60.39% (2.12) at S1-Baseline to 55.30% (8.40) at S1-Post and it was 56.67% (6.38) at S2 and 61.67% (2.93) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical $p$-value of 0.0167) revealed a significant difference in the Control group between S2 and S3 ($T = 0.00, r = -0.627, z = -2.803, p = 0.005, 2$-tailed). There was no significant difference in the Control group between S1-Baseline and S1-Post ($T = 15.00, r = -0.285, z = -1.274, p = 0.203, 2$-tailed) or S1-Baseline and S2 ($T = 6.00, r = -0.490, z = -2.191, p = 0.028, 2$-tailed). In the Exercise group there was a decrease in the median (SE) relative EMG onset as a percentage of chew duration from 65.19% (2.60) at S1-Baseline to 62.11% (3.44) at S1-Post. After two weeks of the home-based exercise programme the median (SE) relative EMG onset as a percentage of chew duration was 58.83% (2.37) at S2 and after ceasing the home-based exercise programme it was 61.76% (3.99) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical $p$-value of 0.0167) revealed no
Figure 3.19: Relative EMG Onset as a Percentage of Chew Duration of the tested muscles across all data collection sessions in the Control group (blue solid squares and solid line) and the Exercise group (red solid circles and dashed line). Error bars show SE of the median and SD of the mean respectively. Panel A: Median Relative EMG Onset as a Percentage of Chew Duration for the ipsilateral anterior temporalis. Panel B: Median Relative EMG Onset as a Percentage of Chew Duration for the contralateral anterior temporalis. Panel C: Median Relative EMG Onset as a Percentage of Chew Duration for the ipsilateral masseter. Panel D: Mean Relative EMG Onset as a Percentage of Chew Duration for the contralateral masseter. Asterisks (*) depict the significant within subjects effects across all the testing sessions in Panel A, Panel B and Panel C and the significant within subjects contrasts between the baseline measures in data collection session 1 (S1-Baseline) and measures taken after the initial exercise task approximately 15 minutes after S1-Baseline (S1-Post) and data collection session 2 undertaken two weeks after data collection session 1 (S2) in Panel D (S3: data collection session 3 undertaken four weeks after data collection session 1).
significant difference in the Exercise group between S1-Baseline and S1-Post (T = 9.00, r = -0.422, z = -1.886, p = 0.059, 2-tailed), between S1-Baseline and S2 (T = 5.00, r = -0.513, z = -2.293, p = 0.022, 2-tailed) or between S2 and S3 (T = 27.00, r = -0.011, z = -0.051, p = 0.959, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

**Contralateral Anterior Temporalis**

Figure 3.19, Panel B shows the median (SE) relative EMG onset as a percentage of chew duration of the contralateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in the relative EMG onset as a percentage of chew duration in the contralateral anterior temporalis across all the testing sessions ($\chi^2(3) = 13.380$, $p = 0.004$). In the Control group there was a decrease in the median (SE) relative EMG onset as a percentage of chew duration from 67.95% (2.84) at S1-Baseline to 57.16% (2.58) at S1-Post and it was 57.31% (6.72) at S2 and 61.39% (2.42) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Control group between S1-Baseline and S1-Post (T = 0.00, r = -0.627, z = -2.803, p = 0.005, 2-tailed). There was no significant difference in the Control group between S1-Baseline and S2 (T = 6.00, r = -0.490, z = -2.191, p = 0.028, 2-tailed) or between S2 and S3 (T = 23.00, r = -0.103, z = -0.459, p = 0.646, 2-tailed). In the Exercise group there was a decrease in the median (SE) relative EMG onset as a percentage of chew duration from 72.08% (3.67) at S1-Baseline to 63.45% (3.50) at S1-Post. After two weeks of the home-based exercise programme the median (SE) relative EMG onset as a percentage of chew duration was 63.48% (9.27) at S2 and after ceasing the home-based exercise programme it was 62.31% (3.80) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a
significant difference in the Exercise group between S1-Baseline and S1-Post (T = 3.00, r = -0.558, z = -2.497, p = 0.013, 2-tailed). There was no significant difference in the Exercise group between S1-Baseline and S2 (T = 19.00, r = -0.194, z = -0.866, p = 0.386, 2-tailed) or S2 and S3 (T = 20.00, r = -0.171, z = -0.764, p = 0.445, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

**Ipsilateral Masseter**

Figure 3.19, Panel C shows the median (SE) relative EMG onset as a percentage of chew duration of the ipsilateral masseter for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in the relative EMG onset as a percentage of chew duration in the ipsilateral masseter across all the testing sessions ($\chi^2(3) = 11.700, p = 0.008$). In the Control group there was a decrease in the median (SE) relative EMG onset as a percentage of chew duration from 56.11% (4.04) at S1-Baseline to 51.52% (3.07) at S1-Post and it was 53.03% (6.88) at S2 and 60.22% (3.37) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Control group between S1-Baseline and S1-Post (T = 10.00, r = -0.399, z = -1.784, p = 0.074, 2-tailed), S1-Baseline and S2 (T = 11.00, r = -0.376, z = -1.682, p = 0.093, 2-tailed) or S2 and S3 (T = 11.00, r = -0.376, z = -1.682, p = 0.093, 2-tailed). In the Exercise group there was a decrease in the median (SE) relative EMG onset as a percentage of chew duration from 61.25% (3.11) at S1-Baseline to 56.41% (3.30) at S1-Post. After two weeks of the home-based exercise programme the median (SE) relative EMG onset as a percentage of chew duration was 57.15% (10.47) at S2 and after ceasing the home-based exercise programme it was 51.37% (3.67) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and
S1-Post (T = 4.00, r = -0.536, z = -2.395, p = 0.0166, 2-tailed). There was no significant difference in the Exercise group between S1-Baseline and S2 (T = 16.00, r = -0.262, z = -1.172, p = 0.241, 2-tailed) or S2 and S3 (T = 13.00, r = -0.330, z = -1.478, p = 0.139, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

**Contralateral Masseter**

Figure 3.19, Panel D shows the mean (SD) relative EMG onset as a percentage of chew duration of the contralateral masseter for the Control group and Exercise group across the data collection sessions. ANOVA revealed a significant within subject effect (Test Time) in the relative EMG onset as a percentage of chew duration in the contralateral masseter ($F_{(3,54)} = 4.625$, $p = 0.006$). Within subjects contrasts (Test Time) testing showed significant differences in the relative EMG onset as a percentage of chew duration between S1-Baseline and S1-Post ($F_{(1,18)} = 23.057$, $p < 0.001$) and between S1-Baseline and S2 ($F_{(1,18)} = 9.082$, $p = 0.007$). In the Control group there was a significant decrease in the mean (SD) relative EMG onset as a percentage of chew duration from 60.66% (5.03) at S1-Baseline to 52.31% (5.28) at S1-Post and to 44.89% (21.35) at S2. In the Exercise group there was a significant decrease in the mean (SD) relative EMG onset as a percentage of chew duration from 61.57% (10.03) at S1-Baseline to 56.99% (11.09) at S1-Post. After two weeks of the home-based exercise programme the mean (SD) relative EMG onset as a percentage of chew duration was 56.86% (9.72) at S2. There were no significant within subjects interactions between the data collection sessions and the groups for any of the three tested contrasts ($p > 0.0167$). There were no significant differences in the between groups effects ($F_{(1,18)} = 1.638$, $p = 0.217$).
Relative Offset as a Percentage of Chew Duration (%)
There were no significant differences in the relative offset as a percentage of chew duration for any of the tested muscles either within subjects or between groups (p > 0.05).

Relative Onset as a Percentage of Chew Cycle Duration (%)
Figure 3.20 shows the relative EMG onsets as a percentage of chew cycle duration of the four tested muscles over the four data collection sessions. The significant findings are described below for each muscle individually.

Ipsilateral Anterior Temporalis
Figure 3.20, Panel A shows the median (SE) relative EMG onset as a percentage of chew cycle duration of the ipsilateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in relative EMG onset as a percentage of chew cycle duration in the ipsilateral anterior temporalis across all the testing sessions ($\chi^2(3) = 10.860, p = 0.013$). In the Control group there was a decrease in the median (SE) relative EMG onset as a percentage of chew cycle duration from 45.79% (1.83) at S1-Baseline to 41.35% (4.89) at S1-Post and remained relatively stable at 41.36% (4.95) at S2 followed by an increase to 41.75% (2.56) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Control group between S1-Baseline and S1-Post (T = 13.00, r =0.3304908470, z = -1.478, p = 0.139, 2-tailed), S1-Baseline and S2 (T = 8.00, r = -0.445, z = -1.988, p = 0.047, 2-tailed) or S2 and S3 (T = 9.00, r = -0.422, z = -1.886, p = 0.059, 2-tailed). In the Exercise group there was a decrease in the median (SE) relative EMG onset as a percentage of chew cycle duration from 46.83% (2.14) at S1-Baseline to 43.59% (2.08) at S1-Post. After two weeks of the home-based exercise programme the median (SE) relative EMG onset as a percentage of chew cycle duration
Figure 3.20: Relative EMG Onset as a Percentage of Chew Cycle Duration of the tested muscles across all data collection sessions in the Control group (blue solid squares and solid line) and the Exercise group (red solid circles and dashed line). Error bars show SE of the median and SD of the mean respectively. Panel A: Median Relative EMG Onset as a Percentage of Chew Cycle Duration for the ipsilateral anterior temporalis. Panel B: Median Relative EMG Onset as a Percentage of Chew Cycle Duration for the contralateral anterior temporalis. Panel C: Median Relative EMG Onset as a Percentage of Chew Cycle Duration for the ipsilateral masseter. Panel D: Mean Relative EMG Onset as a Percentage of Chew Cycle Duration for the contralateral masseter. Asterisks (*) depict the significant within subjects effects across all the testing sessions in Panel A, Panel B and Panel C and the significant within subjects contrasts between the baseline measures in data collection session 1 (S1-Baseline) and data collection session 2 undertaken two weeks after data collection session 1 (S2) in Panel D (S1-Post: measures taken after the initial exercise task approximately 15 minutes after S1-Baseline; S3: data collection session 3 undertaken four weeks after data collection session 1).
decreased to 43.22% (1.78) at S2 and after ceasing the home-based exercise programme it increased to 45.59% (2.24) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S1-Post (T = 4.00, r = -0.536, z = -2.395, p = 0.0166, 2-tailed). There was no significant difference in the Exercise group between S1-Baseline and S2 (T = 16.00, r = -0.262, z = -1.172, p = 0.241, 2-tailed) or S2 and S3 (T = 26.00, r = -0.034, z = -0.153, p = 0.878, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

**Contralateral Anterior Temporalis**

Figure 3.20, Panel B shows the median (SE) relative EMG onset as a percentage of chew cycle duration of the contralateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in relative EMG onset as a percentage of chew cycle duration in the contralateral anterior temporalis across all the testing sessions ($\chi^2(3) = 14.640$, $p = 0.002$). In the Control group there was a decrease in the median (SE) relative EMG onset as a percentage of chew cycle duration from 48.92% (2.36) at S1-Baseline to 41.77% (1.67) at S1-Post followed by an increase to 42.85% (5.10) at S2 and to 44.99% (2.08) at S3. *Post hoc* Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Control group between S1-Baseline and S1-Post (T = 1.00, r = -0.604, z = -2.701, p = 0.007, 2-tailed). There was no significant difference in the Control group between S1-Baseline and S2 (T = 8.00, r = -0.445, z = -1.988, p = 0.047, 2-tailed) or S2 and S3 (T = 21.00, r = -0.148, z = -0.663, p = 0.508, 2-tailed). In the Exercise group there was a decrease in the median (SE) relative EMG onset as a percentage of chew cycle duration from 50.43% (2.52) at S1-Baseline to 44.35% (2.52) at S1-Post. After two weeks
of the home-based exercise programme the median (SE) relative EMG onset as a percentage of chew cycle duration increased to 46.93% (5.51) at S2 and after ceasing the home-based exercise programme it decreased to 46.77% (1.97) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed a significant difference in the Exercise group between S1-Baseline and S1-Post (T = 0.00, r = -0.627, z = -2.803, p = 0.005, 2-tailed). There was no significant difference in the Exercise group between S1-Baseline and S2 (T = 22.00, r = -0.125, z = -0.561, p = 0.575, 2-tailed) or S2 and S3 (T = 20.00, r = -0.171, z = -0.764, p = 0.445, 2-tailed). There were no significant between groups effects at any of the testing sessions (p > 0.05, 2-tailed).

**Ipsilateral Masseter**

Figure 3.20, Panel C shows the median (SE) relative EMG onset as a percentage of chew cycle duration of the ipsilateral masseter for the Control group and Exercise group across the data collection sessions. The Friedman Test revealed significant within subjects effects in relative EMG onset as a percentage of chew cycle duration in the ipsilateral masseter across all the testing sessions (χ²(3) = 11.220, p = 0.011). In the Control group there was a decrease in the median (SE) relative EMG onset as a percentage of chew cycle duration from 41.92% (3.56) at S1-Baseline to 38.16% (1.69) at S1-Post followed by an increase to 39.22% (5.06) at S2 and 40.36% (1.81) at S3. Post hoc Wilcoxon tests with a Bonferroni correction (critical p-value of 0.0167) revealed no significant difference in the Control group between S1-Baseline and S1-Post (T = 10.00, r = -0.399, z = -1.784, p = 0.074, 2-tailed), S1-Baseline and S2 (T = 9.00, r = -0.422, z = -1.886, p = 0.059, 2-tailed) or S2 and S3 (T = 13.00, r = -0.330, z = -1.478, p = 0.139, 2-tailed). In the Exercise group there was a decrease in the median (SE) relative EMG onset as a percentage of chew cycle duration from 43.02% (2.11) at S1-Baseline to 40.95% (1.95) at S1-Post. After two weeks of the
home-based exercise programme the median (SE) relative EMG onset as a percentage of 
chew cycle duration was 43.67% (6.30) at S2 and after ceasing the home-based exercise 
programme it decreased to 37.72% (1.90) at S3. Post hoc Wilcoxon tests with a Bonferroni 
correction (critical p-value of 0.0167) revealed a significant difference in the Exercise 
group between S1-Baseline and S1-Post (T = 4.00, r = -0.536, z = -2.395, p = 0.0166, 2-
tailed). There was no significant difference in the Exercise group between S1-Baseline and 
S2 (T = 26.00, r = -0.034, z = -0.153, p = 0.878, 2-tailed) or S2 and S3 (T = 14.00, r = 
-0.308, z = -1.376, p = 0.169, 2-tailed). There were no significant between groups effects at 
any of the testing sessions (p > 0.05, 2-tailed).

**Contralateral Masseter**

Figure 3.20, Panel D shows the mean (SD) relative EMG onset as a percentage of chew 
cycle duration of the contralateral masseter for the Control group and Exercise group 
across the data collection sessions. ANOVA revealed significant within subject effects 
(Test Time) in the relative EMG onset as a percentage of chew cycle duration of the 
contralateral masseter (F(3,54) = 3.290, p = 0.027). Within subjects contrasts (Test Time) 
testing showed a significant differences in the relative EMG onset as a percentage of chew 
cycle duration between S1-Baseline and S1-Post (F(1,18) = 25.171, p < 0.001). In the 
Control group there was a significant decrease in the mean (SD) relative EMG onset as a 
percentage of chew cycle duration from 43.45% (5.38) at S1-Baseline to 37.43% (2.82) at 
S1-Post and it was 32.41% (15.79) at S2 and 38.77% (5.05) at S3. In the Exercise group 
there was a significant decrease in the mean (SD) relative EMG onset as a percentage of 
chew cycle duration from 43.41% (6.96) at S1-Baseline to 39.95% (7.30) at S1-Post. After 
two weeks of the home-based exercise programme the mean (SD) relative EMG onset as a 
percentage of chew cycle duration was 41.91% (6.11) at S2. There were no significant
within subjects interactions between the data collection sessions and the groups for any of the three tested contrasts (p > 0.0167). There were no significant differences in the between groups effects (F(1,18) = 2.034, p = 0.171).

**Relative Peak as a Percentage of Chew Cycle Duration (%)**
There were no significant differences in the relative peak as a percentage of chew cycle duration for any of the tested muscles either within subjects or between groups (p > 0.05).

**Relative Offset as a Percentage of Chew Cycle Duration (%)**
Figure 3.21 shows the relative EMG offsets as a percentage of chew cycle duration of the four tested muscles over the four data collection sessions. The significant findings are described below for each muscle individually.

**Ipsilateral Anterior Temporalis**
Figure 3.21, Panel A shows the median (SE) relative EMG offset as a percentage of chew cycle duration of the ipsilateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Mann-Whitney Test revealed no significant differences between the Control group and the Exercise group at S1-Baseline (Mann-Whitney U = 47.00, p = 0.821, 2-tailed), S1-Post (Mann-Whitney U = 36.00, p = 0.290, 2-tailed), S2 (Mann-Whitney U = 43.00, p = 0.597, 2-tailed) or S3 (Mann-Whitney U = 28.000, p = 0.096, 2-tailed). The Friedman Test revealed no significant within subjects effects in relative EMG offset as a percentage of chew cycle duration in the ipsilateral anterior temporalis across all the testing sessions ($\chi^2_{(3)} = 0.180$, p = 0.981).
Figure 3.21: Median Relative EMG Offset as a Percentage of Chew Cycle Duration of the ipsilateral anterior temporalis (Panel A), contralateral anterior temporalis (Panel B), ipsilateral masseter (Panel C) and contralateral masseter (Panel D) across all data collection sessions in the Control group (blue solid squares and solid line) and the Exercise group (red solid circles and dashed line). Error bars show the SE of the median. Asterisks (*) depict the significant between groups differences at the respective testing sessions in Panel B, Panel C and Panel D. There were no significant differences in the relative EMG offset as a percentage of chew cycle duration for the ipsilateral anterior temporalis (Panel A). (S1-Baseline: baseline measures in data collection session 1; S1-Post: measures taken after the initial exercise task approximately 15 minutes after S1-Baseline; S2: data collection session 2 undertaken two weeks after data collection session 1; and S3: data collection session 3 undertaken four weeks after data collection session 1).
Contralateral Anterior Temporalis

Figure 3.21, Panel B shows the median (SE) relative EMG offset as a percentage of chew cycle duration of the contralateral anterior temporalis for the Control group and Exercise group across the data collection sessions. The Mann-Whitney Test revealed no significant differences between the Control group and the Exercise group at S1-Baseline (Mann-Whitney U = 48.00, p = 0.880, 2-tailed), S1-Post (Mann-Whitney U = 41.00, p = 0.496, 2-tailed) or S3 (Mann-Whitney U = 35.00, p = 0.257, 2-tailed). However, there was a significant difference between the Control group and the Exercise group at S2 (Mann-Whitney U = 23.00, p = 0.041, 2-tailed). At S2 the median (SE) relative EMG offset as a percentage of chew cycle duration of the contralateral anterior temporalis for the Control group was 92.17% (5.08) and for the Exercise group was 93.87% (4.52). The Friedman Test revealed no significant within subjects effects in relative EMG offset as a percentage of chew cycle duration in the contralateral anterior temporalis across all the testing sessions ($\chi^2(3) = 1.860, p = 0.602$).

Ipsilateral Masseter

Figure 3.21, Panel C shows the median (SE) relative EMG offset as a percentage of chew cycle duration of the ipsilateral masseter for the Control group and Exercise group across the data collection sessions. The Mann-Whitney Test revealed no significant differences between the Control group and the Exercise group at S1-Baseline (Mann-Whitney U = 43.00, p = 0.597, 2-tailed) or S1-Post (Mann-Whitney U = 47.00, p = 0.821, 2-tailed). However, there was a significant difference between the Control group and the Exercise group at S2 (Mann-Whitney U = 20.00, p = 0.023, 2-tailed) and S3 (Mann-Whitney U = 23.00, p = 0.041, 2-tailed). At S2 the median (SE) relative EMG offset as a percentage of chew cycle duration of the ipsilateral masseter for the Control group was 91.55% (4.91)
and for the Exercise group was 94.82% (4.49). At S3 the median (SE) relative EMG offset as a percentage of chew cycle duration of the ipsilateral masseter for the Control group was 91.98% (1.12) and for the Exercise group was 95.31% (1.37). The Friedman Test revealed no significant within subjects effects in relative EMG offset as a percentage of chew cycle duration in the ipsilateral masseter across all the testing sessions ($\chi^2_{(3)} = 2.940, p = 0.401$).

**Contralateral Masseter**

Figure 3.21, Panel D shows the median (SE) relative EMG offset as a percentage of chew cycle duration of the contralateral masseter for the Control group and Exercise group across the data collection sessions. The Mann-Whitney Test revealed no significant differences between the Control group and the Exercise group at S1-Baseline (Mann-Whitney U = 49.00, p = 0.940, 2-tailed), S1-Post (Mann-Whitney U = 47.00, p = 0.821, 2-tailed) or S2 (Mann-Whitney U = 37.00, p = 0.326, 2-tailed). However there was a significant difference between the Control group and the Exercise group at S3 (Mann-Whitney U = 21.00, p = 0.028, 2-tailed). At S3 the median (SE) relative EMG offset as a percentage of chew cycle duration of the contralateral masseter for the Control group was 89.91% (1.61) and for the Exercise group was 93.26% (1.36). The Friedman Test revealed no significant within subjects effects in relative EMG offset as a percentage of chew cycle duration in the contralateral masseter across all the testing sessions ($\chi^2_{(3)} = 5.040, p = 0.169$).

**Jaw Kinematic and EMG Features**

The EMG features of a typical single chewing cycle are shown as a percentage of the chewing cycle in Figure 3.22, with jaw movement onset occurring at 0% and the end of the chewing cycle occurring at 100% of a typical chewing cycle (Figure 3.22, Panel A). Maximum jaw opening and jaw movement offset occurred at 35.4% and 71.3% of the
chewing cycle respectively. The baseline EMG activity of all 20 participants was pooled and the mean onset, peak and offset are shown for each of the four tested muscles (Figure 3.22, Panel B). In all four muscles the EMG activity occurred during the closing and occlusal phases of the movement cycle (Figure 3.22, Panel B). In the ipsilateral anterior temporalis the mean onset of EMG activity occurred at 44.9% (SD 6.1%), reaching its peak at 70.7% (SD 4.6%) and its offset occurred at 91.1% (SD 4.4%). In the contralateral anterior temporalis the mean onset of EMG activity occurred at 47.8% (SD 7.6%), reaching its peak at 72.7% (SD 4.3%) and its offset occurred at 91.9% (SD 5.2%). In the ipsilateral masseter the mean onset of EMG activity occurred at 41.5% (SD 9.1%), reaching its peak at 72.1% (SD 4.9%) and its offset occurred at 90.9% (SD 9.0%). In the contralateral masseter the mean onset of EMG activity occurred at 43.4% (SD 6.1%), reaching its peak at 69.7% (SD 5.2%) and its offset occurred at 87.7% (SD 5.5%).
Figure 3.22: The kinematic and electromyographic (EMG) features of a typical single chewing cycle expressed as a percentage of the chewing cycle duration. Panel A: The trajectory of the opening and closing of the mandible showing the Onset, Peak and Offset of a typical jaw opening movement as well as the End of that Chewing Cycle which corresponds to the Onset of the following chewing cycle. Panel B: Corresponding EMG activities of the 4 tested muscles showing the mean Onset, Peak and Offset for each muscle as a percentage of the chewing cycle. Error bars show SD of the mean Onset, Peak and Offset respectively (n = 20).
Discussion

This study has shown that the application of a specific resistance exercise task to the muscles of mastication can produce changes to the temporal characteristics of the activation of these muscles relative to the movement of the jaws during chewing. It has previously been demonstrated that a unilateral resistance exercise task applied to the mandible can produce changes in the pattern(s) of EMG activation in some of the muscles of mastication during a novel standardised unilateral jaw movement task (Wirianski, 2009, Wirianski et al., 2014) as well as changes to the jaw movement trajectories during chewing of cooked rice: a food bolus that changes shape and consistency throughout the chewing sequence (Chapter 2). The current study has provided further evidence that a specific resistance exercise task applied to the jaw may be capable of altering the motor control patterns of the muscles of mastication during chewing a standard pellet of gum: a food bolus that maintains its consistency throughout the chewing sequence. In each of these three studies a similar resistance exercise task was performed and movement and EMG data were collected during three different jaw movement tasks from three different groups of participants. Each study has reported changes to either muscle activation patterns (Wirianski, 2009, Wirianski et al., 2014 and the current study) or jaw movement patterns (Chapter 2) and together, these results may suggest that the application of a unilateral resistance exercise task may result in a learning/training effect which may be in part the result of changes in the motor control of the muscles of mastication.

Adherence with the Home Exercise Programme

All participants in the Exercise group completed some of the prescribed home exercise programme. On average this equated to participants completing approximately 80% of the three exercise sessions and 55% of the prescribed number of exercise sets per day. These
figures indicate a lower level of adherence to the prescribed exercise regimen than that in
an earlier low back pain (LBP) study which asked participants to complete three sets of 10
repetitions twice per day and reported an average adherence rate of 81% in the skilled
training group (Tsao et al., 2010). The current study, however, requested participants to
complete five sets of 10 repetitions thrice per day. Also the participants in the current study
consisted of students and staff from a busy Dental teaching hospital within the
metropolitan area of a large capital city. All participants had busy clinical and academic
workloads which may have contributed to the lower level of adherence compared to the
LBP study, especially in the morning and afternoon sessions (Figure 3.9). It is plausible
that it was difficult for participants in the current study to complete the prescribed
exercises during these two sessions as they may have had to prepare for and complete their
work duties. Furthermore, the participants in the current study were healthy adults with no
signs or symptoms of TMD. Patients with LBP or other painful conditions may hold
perceptions that the prescribed exercise intervention may be beneficial for symptom
reduction and therefore may be more likely to adhere to their exercise programme. Healthy
individuals, on the other hand, may have no preconceived perceptions of the benefit of the
exercise programme and this may contribute to the lower adherence rate reported in this
study.

No other studies have been found that report adherence to an exercise programme in
patients with TMD. Nevertheless, the adherence rate of the current study compares
favourably to reports of other exercise programmes prescribed for musculoskeletal
conditions. Community-based general physical activity regimens have been reported to
have an adherence rate ranging between 60% to 102% in sedentary adults aged 65 years or
more, over a 12-month intervention period (King et al., 2000). In obese adults with
osteoarthritis of the knee, adherence to an 18-month aerobic and resistance-training exercise programme was reported to be 66% during the initial four months and 54% overall (van Gool et al., 2005). Although an average adherence rate of 81% has been reported in patients with recurrent LBP that undertook a skilled training exercise programme (Tsao et al., 2010) and approximately 60% adherence was reported in patients aged 50 years and over with chronic LBP that underwent a 12-month exercise programme (Hicks et al., 2012) between 50% to 70% of patients with chronic LBP are not adherent to their prescribed home-based exercise programme (Beinart et al., 2013). With such varied rates of adherence it is clearly important that future studies assess and report the adherence rates of participants undergoing the tested intervention(s). Reporting adherence would improve the determination of the clinical effectiveness of the tested exercise regimen and assist clinicians in their clinical reasoning when prescribing appropriate interventions for their patients.

**Jaw Movement Changes**

Significant changes occurred in some of the movement parameters over the course of this study. Although these differences in the movement parameters were found between the different testing sessions there were no significant differences between the two groups of participants. Furthermore, these changes were similar and followed similar patterns of change across the testing sessions in both the Control group and the Exercise group (Figure 3.10, Figure 3.12 and Figure 3.13). This suggests that the jaw movement characteristics were similar in both groups at each of the testing sessions. Therefore, any significant between groups differences in the EMG parameters that occurred in the Exercise group would be more likely to have resulted from the application of the exercise modality rather than from changes in the jaw movement parameters.
The lack of between groups differences in the jaw movement parameters may be in contrast to the results of the previous study that reported immediate changes in jaw movement trajectories during chewing cooked rice following the application of a similar resistance exercise task (Chapter 2). Post hoc testing in the current study did however reveal an immediate significant increase in the closing velocity of the jaw in the Exercise group which did not occur in the Control group (Figure 3.12, Panel C). Velocity of jaw movement along with many other variables, such as maximum jaw displacement, cycle and phase durations and acceleration, have been used previously to describe the kinematics of human jaw movement (Bates et al., 1975, Buschang et al., 2000). However, it has been suggested that much of the information that describes the masticatory cycle is lost when using these variables (Buschang et al., 2000). Many of these variables describe the masticatory movement path in two dimensions and arbitrary points in the masticatory cycle are generally chosen to demarcate phase changes in order to calculate the measures used to analyse chewing motion paths (Buschang et al., 2000, Hattori et al., 2010). However, the masticatory movement path of the mandible is a three-dimensional continuous curve with no evident demarcations (Hattori et al., 2010) and thus, information about the form of the mandibular movement path is discarded when analysing variations in these variables during chewing (Buschang et al., 2000, Hattori et al., 2010). It is plausible therefore that these may not be the best variables to describe human jaw movement. In contrast, the use of elliptic Fourier descriptors (Ferrario et al., 1990, Kuhl and Giardina, 1982) is capable of capturing the kinematics of the masticatory path in detail (Ferrario et al., 1990, Hattori et al., 2010). Furthermore, the analysis of the elliptic Fourier descriptors using principal component analysis on a covariance-variance matrix has been successfully implemented to accurately describe masticatory motion paths without the loss of valuable information.
(Hattori et al., 2010, Igari et al., 2011) and may be a more appropriate method for describing masticatory kinematics in future studies.

Interestingly, the immediate significant increase in the closing velocity of the jaw in the Exercise group may reflect changes in the jaw movement trajectory (Chapter 2). Following the application of a resistance exercise task it was demonstrated that the masticatory movement trajectory became more horizontally orientated in the coronal plane and more protruded in the sagittal plane (Chapter 2) which may have resulted in a slightly longer opening movement path. This would therefore necessitate a faster closing velocity in order to complete the chewing cycle with the same duration. Although the current study demonstrated an immediate significant increase in the Jaw Movement Displacement in both the Exercise and Control groups, it did not find a significant difference between the groups following the resistance exercise task. This may reflect the method of defining maximum opening in the current study as the greatest downward mandibular displacement. Defining the maximum mandibular opening in this manner has been described as being arbitrary and has the potential of losing valuable information that describes the complete masticatory cycle (Buschang et al., 2000). The use of elliptic Fourier descriptors in combination with principal component analysis on a covariance-variance matrix on the other hand may be a more appropriate method for describing masticatory motion paths without losing valuable information.

Although there was a lack of statistically significant differences between groups in the jaw movement parameters there does appear to be differences in the Median Jaw Movement Duration expressed as a Percentage of the Chewing Cycle Duration between the Exercise and Control groups, especially at S1-Baseline and S1-Post. Two plausible explanations
may help to describe these phenomena. Firstly, this may reflect differences in the durations of the occlusal phases of the masticatory cycle. The duration of intercuspation is related to the way posterior temporalis and deep masseter muscle activity is dispersed (Moller, 1966, Miller, 1991, Hannam and McMillan, 1994) which may result in differences in the way an individual uniquely manages the late and mediotrusive phases of the power stroke. Therefore it is possible that the manner in which even a few participants in either experimental group used their dentition could have affected any comparison of muscle-timing data. And secondly, the data were not normally distributed and therefore non-parametric statistical analyses were used and the standard error was reported as the measure of the variability of the Median Jaw Movement Duration expressed as a Percentage of the Chewing Cycle Duration instead of the standard deviation. The standard error is smaller than the standard deviation. This may have resulted in the data points illustrated in Figure 3.11 appearing to be different even though there were no statistically significant differences between the two experimental groups.

**Electromyographic Changes**

Two variables showed differences between the groups: the EMG Onset Relative to Jaw Movement Onset (Figure 3.15); and the Relative EMG Offset as a Percentage of Chew Cycle Duration (Figure 3.21). After two weeks of the home-based exercise programme the onset of the EMG activity in the contralateral anterior temporalis occurred 0.04 s later in the chewing cycle compared to the Control group (Figure 3.15, Panel B). The delay in the onset of the contralateral anterior temporalis may indicate that other muscles may need to become more active around the same time in the chewing cycle. Electromyographic activity in the contralateral anterior temporalis commences shortly after the commencement of the closing phase of the chewing cycle (Miller, 1991) and increasing
EMG activity in the inferior head of the lateral pterygoid and the medial pterygoid muscles has been reported just after the changeover between opening and closing phases (Miller, 1991). Along with activation of the temporalis and masseter muscles there is coactivation of the inferior head of the lateral pterygoid, medial pterygoid and digastric during closing and medial jaw movements (Hannam and McMillan, 1994, Miller, 1991) which occur in combination during the closing phase of the masticatory cycle. It is plausible therefore that there may have been a concomitant increase in the EMG activities or earlier onsets of these muscles, which were not measured in this study, in order to compensate for the delayed onset of the contralateral anterior temporalis in order to initiate the closing phase of the chewing cycle. Indeed, specific exercise training has resulted in earlier activation of the targeted muscles in the knee (Cowan et al., 2002, Cowan et al., 2003), shoulder (Worsley et al., 2013), cervical spine (Falla et al., 2004a) and the lumbar spine (Tsao and Hodges, 2007, Tsao and Hodges, 2008). Moreover, variables related to the onset of jaw muscle activation showed significant reductions in the ipsilateral masseter and the ipsilateral anterior temporalis in the current study. Therefore, it is possible that the resistance exercise programme may have resulted in earlier onsets of the activation of the inferior head of the lateral pterygoid, medial pterygoid and/or digastric which then allowed the contralateral anterior temporalis to demonstrate a delay in the onset of its activity during the closing phase of the chewing cycle. Future studies incorporating the use of intramuscular electrodes to investigate the EMG activity of the pterygoid muscles, in particular, would help elucidate these mechanisms.

Furthermore, at the same data collection time-point, the offset of the EMG activity of the contralateral anterior temporalis and ipsilateral masseter occurred 1.7% and 3.3% later in the chewing cycle, respectively, when measured as a percentage of the chewing cycle.
(Figure 3.21, Panel B and Panel C). These findings, along with the delayed onset of the contralateral anterior temporalis, complement a previous report of a reduced EMG duration in the ipsilateral anterior temporalis with a concomitant delay in the time to peak EMG activity of the contralateral masseter and ipsilateral digastric following the application of a resistance exercise task (Wirianski, 2009, Wirianski et al., 2014). These findings are also consistent with the view that the masticatory system possesses an inherent degree of mechanical redundancy (Langenbach and van Eijden, 2001, Lobbezoo et al., 2004, Van Eijden et al., 1990, van Eijden and Turkawski, 2001). Through their complex and diverse architecture the muscles of mastication possess multiple options for tendon pull (Hannam and McMillan, 1994, Schumacher, 1961). This provides the potential for a large number of possible patterns of muscle activation that can be utilised to perform jaw movements (Lobbezoo et al., 2004, Van Eijden et al., 1990, van Eijden and Turkawski, 2001) which can be called upon by the masticatory central pattern generator, the cortical masticatory area and/or the sensorimotor cortex to control the movement of the jaw in order to appropriately adapt the chewing movement trajectories to changes in the oral environment specific to the performed task (Avivi-Arber et al., 2011, Lund, 1991, Lund and Kolta, 2006, Nakamura and Katakura, 1995, Yamada et al., 2005).

After completing the initial resistance exercise task the onset of EMG activity in the contralateral anterior temporalis occurred 0.08 s earlier in the chewing cycle in the Exercise group (Figure 3.15, Panel B). Interestingly, over the same initial period in the Control group, the onset of EMG activity in the contralateral anterior temporalis also occurred 0.09 s earlier in the chewing cycle even though participants in the Control group did not complete the resistance exercise task. Furthermore, compared to the baseline measure, the onset of EMG activity in the contralateral anterior temporalis in the Control
group also occurred 0.07 s earlier in the chewing cycle after two weeks (S2). Although there was a similar reduction in the Exercise group after completing the exercise task, this group did not show any significant differences between the baseline measure and after completing two weeks of the home-based exercise programme suggesting that there was no change in the Exercise group over this period. This finding complements the results of our previous study which also demonstrated that many of the significant changes occurred in the Control group, while the Exercise group tended to maintain the majority of the tested variables at pre-exercise baseline values (Wirianski, 2009, Wirianski et al., 2014). It was postulated that in asymptomatic individuals there was a level of variability between recording sessions in the recruitment patterns of some of the muscles of mastication for the production of the same right lateral jaw movement and that isotonic resistance exercise may reduce this variability (Wirianski, 2009, Wirianski et al., 2014). The results of the current study and our previous work may indicate that completing a home-based resistance exercise task may result in a stabilisation of the motor strategy when completing a standardised jaw movement task (Wirianski, 2009, Wirianski et al., 2014) as well as during mastication.

Four variables showed differences in two muscles immediately after completing the resistance exercise task and one variable showed differences in two muscles after two weeks of the home-based resistance exercise programme. Three of these variables were related to the onset of the muscles’ activation: EMG Onset Relative to Jaw Movement Onset (Figure 3.15); Relative EMG Onset as a Percentage of Chew Duration (Figure 3.19); and Relative EMG Onset as a Percentage of Chew Cycle Duration (Figure 3.20). All three of these variables showed a reduction in the ipsilateral masseter and two of the variables, namely, the EMG Onset Relative to Jaw Movement Onset and the Relative EMG Onset as
a Percentage of Chew Cycle Duration also showed a reduction in the ipsilateral anterior temporalis. These findings indicate that the onset of EMG activity in these two ipsilateral muscles occurred earlier in the chewing cycle following the application of the resistance exercise task. During unilateral chewing of gum the ipsilateral masseter and temporal muscles demonstrate greater EMG activity with the ipsilateral temporal muscles being activated first during the closing phase of this task (Moller, 1966). The earlier onset of EMG activity in these muscles reported in this study may demonstrate that these muscles might be more susceptible or responsive to the effects of the prescribed resistance exercise task, perhaps in part due to the recruitment of motoneurone task groups (Loeb, 1985, Murray and Peck, 2007) and/or the preferential activation of motor units with the best mechanical advantage and lowest metabolic cost to produce the desired specific jaw movement (Murray and Peck, 2007, De Troyer et al., 2005, Gandevia et al., 2006, Sessle et al., 2013).

The application of specific exercise training has also resulted in earlier activation of the targeted muscles in the knee (Cowan et al., 2002, Cowan et al., 2003), shoulder (Worsley et al., 2013), cervical spine (Falla et al., 2004a) and the lumbar spine (Tsao and Hodges, 2007, Tsao and Hodges, 2008). In these musculoskeletal systems, the therapeutic exercises were prescribed for the management of known biomechanical deficiencies of delayed muscle activation at these joints resulting from pain or injury. It is yet to be determined if patients with TMD also present with similar biomechanical deficiencies that display delayed muscle activation patterns. If future studies reveal the presence of delayed muscle activity patterns in patients with TMD symptoms then the findings of the current study could indicate that a specific exercise task may reverse the muscle activation delays and perhaps provide an avenue for the management of TMD.
**Patterns of Jaw Movement and EMG Changes**

Interestingly, some of the EMG parameters also demonstrated similar patterns of change across several muscles between the testing sessions. Furthermore, significant changes also occurred in the movement parameters, and as with the EMG changes, some followed similar patterns of changes between the testing sessions. These changes also tended to be similar in both the Control group and the Exercise group. For example, jaw movement duration, chew cycle duration, jaw movement open time and jaw movement close time all showed significant decreases between the testing sessions over the course of the study (Figure 3.10). Jaw movement displacement, opening velocity, closing velocity and chewing velocity all showed a significant initial increase between S1-Baseline and S1-Post and the opening velocity and chewing velocity showed significant increases after two weeks of the study (Figure 3.12).

Similar findings have been demonstrated in an experimental pain model where no significant between group differences were found in either jaw movement amplitude or velocity or jaw muscle EMG activity during a standardised or free chewing task following the resolution of a short period of moderate to severe masseter muscle pain that subsided within 10 minutes (Inamoto, Whittle and Murray, unpublished observations). There were however significant increases in velocity and amplitude of chewing between data collection blocks during the free chewing task but not the chewing task that was standardised for timing only (Inamoto, Whittle and Murray, unpublished observations). This absence of an increase in the jaw movement and EMG activity during the standardised chewing task suggested that factors such as a motivation to complete the experimental protocol may be more influential on the increased velocity and amplitude of jaw movement seen in the free chewing task rather than the presence of a practice or
training effect (Inamoto, Whittle and Murray, unpublished observations). This effect may also have occurred in the current study and the inclusion of a standardised chewing task in future studies may help determine the mechanisms of the similar pattern changes reported in both the Control group and the Exercise group. Furthermore, studies combining principal component analysis of the movement trajectory with the collection of EMG data will help determine whether the EMG changes reported in the current study and previously (Wirianski, 2009, Wirianski et al., 2014) are associated with movement trajectory changes (Chapter 2).

**Jaw Motor Control**

It is plausible that the resistance exercise task may have contributed to central neuroplastic changes either within the central pattern generator or higher cortical centres such as the primary motor cortex controlling the muscles of mastication and/or its respective cortical somatosensory area and/or the cortical masticatory area. The results of the current study complement the findings of previous work which investigated the effects of similar specific resistance exercise regimens during a standardised lateral jaw movement task (Wirianski, 2009, Wirianski et al., 2014) and free chewing of cooked rice (Chapter 2). The application of an isotonic resistance jaw exercise during a lateral jaw movement resulted in a reduction in the duration of EMG activity in the ipsilateral anterior temporalis with a concomitant increase in the time to peak EMG activity in the contralateral masseter and ipsilateral anterior digastric (Wirianski, 2009, Wirianski et al., 2014). It was postulated that the increased time to peak EMG activity in the contralateral masseter and ipsilateral anterior digastric allowed these masticatory muscles to produce their peak EMG activity later in the movement cycle to compensate for the reduced duration of EMG activity in the ipsilateral anterior temporalis in order to complete the same lateral jaw movement task.
and/or maintain the vertical position of the mandible while completing the standardised movement task (Wirianski, 2009, Wirianski et al., 2014). Moreover, undertaking a laterally directed isometric resistance jaw exercise resulted in changes in the trajectory of the mandible during the free chewing of cooked rice whereby the movement trajectory became significantly more horizontally orientated in the coronal plane and more protruded in the sagittal plane (Chapter 2). Unfortunately, EMG data were not recorded and therefore no correlation could be made between the changes in the masticatory movement path and the activation patterns of the muscles of mastication following the application of the resistance exercise task (Chapter 2). However, given that resistance exercise can change the temporal characteristics of the EMG activation patterns during a standardised jaw movement task (Wirianski, 2009, Wirianski et al., 2014) as well as changing the masticatory movement path during the functional task of chewing (Chapter 2), it is plausible that the resistance exercise task performed in the current study, that resulted in the changes in the temporal characteristics of the activation of the muscles of mastication, may have also contributed to changes in the trajectory of the masticatory movement path which was not recorded. Future studies combining the measurement of the jaw movement path with EMG data will help determine the association of these two findings.

Exercise induced neuroplastic changes have been described in the orofacial region with both animal and human studies reporting neuroplastic changes in the face primary motor cortex (Avivi-Arber et al., 2011, Boudreau et al., 2007, Sessle et al., 2007) and the face somatosensory cortex (Avivi-Arber et al., 2011) following successful training of novel motor tasks. More specifically in humans, changes have been demonstrated in the tongue primary motor cortex following 15 minutes (Boudreau et al., 2007), one hour (Svensson et al., 2006) and one week (Svensson et al., 2003) training of a novel tongue protrusion task.
These studies, utilising transcranial magnetic stimulation, reported significant enhancement of the motor evoked potentials recorded from the tongue musculature and decreases in the tongue primary motor cortex thresholds with concomitant improvements in performance of the novel tongue protrusion task (Boudreau et al., 2007, Svensson et al., 2006, Svensson et al., 2003). These changes were also accompanied with significant increases in the size of the cortical motor maps of the tongue primary motor cortex (Svensson et al., 2003, Svensson et al., 2006) as well as a lateral and anterior shift in the centre of gravity coordinates of the cortical map at the seven-days follow-up after one hour of tongue protrusion training (Svensson et al., 2006). Complementary findings have also been reported in LBP patients where reorganisation of the motor cortex representation of transversus abdominis was demonstrated following two weeks of specific voluntary activation training of the transversus abdominis muscle (Tsao et al., 2008).

Based on these previous reports, the results of the current study are consistent with the general view that specific exercise training may result in changes in the motor control patterns of the muscles that produce joint movements. The temporal changes in the EMG activation of the muscles of mastication demonstrated in the current study along with the changes in jaw movement trajectories demonstrated in our previous study (Chapter 2) may reflect changes in the central motor control of mastication brought about by the application of the exercise task. Furthermore, these motor control changes may be the result of reversible neural plasticity at many levels of the nervous system including changes in the excitability of motoneurones, the reorganisation of the sensorimotor cortex and/or the cerebellum (Boudreau et al., 2007, Svensson et al., 2006, Svensson et al., 2003, Tsao et al., 2010, Tsao and Hodges, 2007).
The rhythmic pattern of mastication is produced and controlled by the masticatory central pattern generator located in the pons and medulla (Lund and Kolta, 2006, Lund and Enomoto, 1988). In turn the masticatory central pattern generator output drive is modulated by inputs from the primary motor and somatosensory cortices of the face, the cortical masticatory area and by feedback from mechanoreceptors including those in the periodontal ligament, intraoral touch receptors and muscle spindles in the jaw closing muscles (Langenbach and van Eijden, 2001, Lund and Kolta, 2006, Rossignol et al., 1988). With such a vast array of neuromuscular structures contributing to the control of mastication it is plausible that any one, or a combination, of these areas may be affected by the application of jaw resistance exercises. Different tasks could effect changes in the primary motor and somatosensory cortices and the cortical masticatory area which in turn could affect the masticatory central pattern generator. These changes in cortical motor drive/output could in turn lead to changes in alpha motoneurones possibly facilitated by changes in the recruitment of different motoneurone task groups (Loeb, 1985, Murray and Peck, 2007) which in turn may lead to changes in jaw EMG activity and therefore motor patterns. This is clearly an area worthy of further investigation.

**Strengths of the study**

The current study complements previous work from different laboratories utilising different recording equipment and analysis techniques (Wirianski, 2009, Wirianski et al., 2014). These previous studies demonstrated temporal changes in EMG activation of the muscles of mastication during a standardised right lateral jaw movement task following four weeks of an isotonic jaw resistance exercise task (Wirianski, 2009, Wirianski et al., 2014) and changes in the pattern of jaw movement trajectories during chewing cooked rice immediately following the application of an isometric jaw resistance exercise task (Chapter
2). Now, the current study has demonstrated temporal changes in EMG activation of the muscles of mastication during the chewing of a standardised soft and consistent bolus of gum. These results suggest that the application of resistance jaw exercises can change the temporal characteristics of the activation of the muscles of mastication during a standardised jaw movement task (Wirianski, 2009, Wirianski et al., 2014) as well as during a functional rhythmical task such as chewing gum, as shown in the current study. Furthermore, the application of resistance jaw exercises can also change jaw movement patterns during a similar functional chewing task (Chapter 2). It is therefore plausible to suggest that the changes in jaw movement trajectories may have been accompanied by, or were a result of, changes in the temporal characteristics of the EMG activation of the muscles of mastication. This hypothesis is a subject for further investigation and studies combining the recording of EMG activity and jaw movement trajectories with the use of elliptic Fourier descriptors in conjunction with principal component analysis on a covariance-variance matrix will help elucidate this possible mechanism of jaw muscle control.

**Limitations**

Even though the results of this study appear promising and they complement the findings of previous studies they should be interpreted with a level of caution. Logistical difficulties with participant recruitment within a fixed time period limited the sample size to n = 10 for each group. Moreover, multiple parameters were recorded and calculated over the course of this study. Both these factors may have contributed to the analyses being underpowered. However, as noted in our initial hypotheses, only one primary outcome variable was chosen for the statistical analysis of the study. The effect of two weeks of resistance jaw exercise was assessed by testing for the difference in this variable between S1-baseline and
S2 (two weeks after the baseline). The level of significance was set at 5% and based on the results of our previous study (Wirianski, 2009, Wirianski et al., 2014) an estimate of sample size was calculated (Dell et al., 2002). Based on a probability of a Type I error set at $\alpha = 0.05$ and a power ($1-\beta$) of 0.8, these calculations revealed a sample size estimate for the Time to Peak Muscle EMG Activity in the masseter muscle of $n = 12.63$. Thus a sample size of $n = 13$ for each of the two groups was selected for this current study. All other measured parameters were considered secondary outcomes. These were analysed in an exploratory analysis with the significance level set at 5% for each secondary outcome measure. In the same way, any changes between S2 and S3 for all the measured parameters were considered as secondary outcomes. Therefore there will be a possibility of false positive results amongst the secondary analyses.

Furthermore, increasing the number of statistical tests increases the chance of a Type I error. To limit the overall Type I error to below 0.05, the Bonferroni correction was used, which is a very conservative test (Field, 2005). This has the possibility of increasing the probability of Type II error (i.e. not detecting an effect when one actually existed) and also resulting in a loss of statistical power. This was an exploratory study to attempt to determine meaningful variables to examine in future works. Apart from our previous study (Wirianski, 2009, Wirianski et al., 2014) there have been little if any other studies that have investigated the temporal characteristics of the EMG activity in the muscles of mastication following the application of a resistance exercise task. Therefore, little is known about which variables are most appropriate to measure. This current work, along with our previous study (Wirianski, 2009, Wirianski et al., 2014), will allow future more focused studies to be conducted on a narrower subset of variables with more rigorous statistical analyses.
Future Studies
Despite the limitations of this study, the results suggest that resistance exercise may be capable of changing the EMG activity patterns of the muscles of mastication during chewing. Whether these EMG activity changes are the result of changes in the motor control of the masticatory system is yet to be determined. Nevertheless, the findings of the current study are consistent with our previous work (Wirianski, 2009, Wirianski et al., 2014) as well as reports from other musculoskeletal systems, including the knee (Cowan et al., 2002, Cowan et al., 2003), shoulder (Worsley et al., 2013), cervical spine (Falla et al., 2004a) and the lumbar spine (Tsao and Hodges, 2007, Tsao and Hodges, 2008). Future studies incorporating transcranial magnetic stimulation may help to determine the effects of resistance exercise on the central motor control of mastication. Combining this with the use of elliptic Fourier descriptors to effectively describe jaw movement trajectories in conjunction with principal component analysis on a covariance-variance matrix and incorporating EMG data, including the investigation of single motor unit activity may also help elucidate the mechanisms underlying these changes, especially in determining if the EMG changes are concomitant with previously reported jaw movement trajectory changes (Chapter 2). Ultimately, applying and investigating these and future findings to a patient population in a randomised control trial, with the inclusion of an appropriate control and/or sham intervention, will aid the development of effective, evidence-based treatment modalities for the management of jaw movement disorders in patient’s diagnosed with temporomandibular disorders.
Conclusion

The effects of a specific isometric resistance exercise task applied to the muscles of mastication on the temporal characteristics of the EMG activation patterns of these muscles were investigated during chewing. In asymptomatic individuals, isometric resistance exercise applied against a lateral jaw movement resulted in an earlier onset of the EMG activity in the ipsilateral anterior temporalis and the ipsilateral masseter after two weeks. Musculoskeletal pain conditions are associated with biomechanical deficiencies of delayed muscle activation at other joints including the knee, shoulder, cervical spine and lumbar spine. At these joints, the application of specific exercise training has resulted in earlier activation of the targeted muscles and in the lumbar spine, these earlier muscle activation patterns were associated with reversible cortical neuroplastic changes as well as a reduction in pain. At present it is unclear if patients with TMD also display similar biomechanical deficiencies of delayed activation of the muscles of mastication. Future studies investigating the presence of delayed muscle activation patterns in patients with deviations in mandibular movement patterns will help determine the efficacy of specific resistance exercise in the management of TMD.
Chapter 4
Discussion

Overview

This thesis investigated the effects of isometric exercise training on the muscles of mastication during the functional task of chewing. The specific aims of this thesis were: firstly, to determine if the application of a specific resistance exercise task in asymptomatic individuals will result in the jaw muscles being used differently in order to produce different jaw movement patterns during chewing; and secondly, if changes in jaw movement patterns occur following the application of a specific resistance exercise task then the second aim is to define the EMG activity patterns in the muscles of mastication that may accompany any changes in jaw movement patterns. In relation to the postulated hypotheses, this thesis demonstrated that performing an isometric resistance exercise task can change the patterns of jaw movement (Chapter 2), as well as the patterns of EMG activation of the muscles of mastication (Chapter 3) during free chewing. In particular, jaw movement trajectories became more horizontally orientated in the coronal plane and more protruded in the sagittal plane (Chapter 2) and an earlier onset of the EMG activities in the ipsilateral masseter and ipsilateral anterior temporalis were demonstrated (Chapter 3) following the application of the resistance exercise task. The findings of this thesis appear consistent with those from other musculoskeletal systems where the application of specific exercise training has also been demonstrated to result in earlier EMG activation of the targeted muscles in the knee (Cowan et al., 2002, Cowan et al., 2003), shoulder (Worsley et al., 2013), cervical spine (Falla et al., 2004a) and the lumbar spine (Tsao and Hodges, 2007, Tsao and Hodges, 2008). These results are further complemented in the lumbar spine, in particular, where reorganisation of the cortical representation of trunk muscles
associated with postural control deficits of delayed onset of EMG activity in the transversus abdominis muscle during rapid arm movement tasks were demonstrated in people with recurrent low back pain (LBP, Tsao et al., 2008). This reorganisation of the motor cortex found in LBP patients was subsequently demonstrated to be reversible following the completion of two weeks of specific exercises consisting of voluntary activation of transversus abdominis independently from other trunk muscles, which resulted in the motor cortical maps resembling those found in healthy, asymptomatic individuals with concomitant earlier activation of the transversus abdominis during a rapid arm movement task and reduced pain (Tsao et al., 2010).

Exercise induced neuroplastic changes have also been reported in the orofacial region where changes have been demonstrated in the tongue primary motor cortex following the training of a novel tongue protrusion task (Boudreau et al., 2007, Svensson et al., 2006, Svensson et al., 2003). More recently, isotonic resistance exercise has also been shown to produce significant differences in the temporal characteristics of the EMG activation of the muscles of mastication during a standardised jaw movement task, with a reduction in the duration of EMG activity in the ipsilateral anterior temporalis and a concomitant increase in the duration to peak EMG activity in the contralateral masseter and ipsilateral digastric being demonstrated (Wirianski, 2009, Wirianski et al., 2014). Therefore, when viewed in the context of previous findings in other musculoskeletal systems, and more specifically when compared to the orofacial region, the combined results of this thesis are consistent with the general view that specific exercise training may result in changes in the motor control patterns of the muscles that produce joint movements.
These findings may also have a clinical application. Deviations in jaw movements from normal masticatory patterns are seen in some patients that present with temporomandibular disorders (TMD, Schiffman et al., 2014, Laskin, 1969, Solberg, 1983, Bell, 1983). These altered jaw movement patterns may result in reversible neuroplastic changes, similar to those demonstrated in LBP patients (Tsao et al., 2008) and in the tongue primary motor cortex (Boudreau et al., 2007, Svensson et al., 2006, Svensson et al., 2003), that may affect the motor control of the muscles of mastication and interfere with normal masticatory function. The application of appropriately directed specific resistance exercise to the jaw may contribute to reversing these neuroplastic changes such that they resemble those found in healthy, asymptomatic individuals, as previously demonstrated in LBP patients (Tsao et al., 2010). Understanding the mechanisms that underlie the motor control deficits associated with altered jaw movement patterns may provide effective strategies for the management of TMD.

**Masticatory Movement Changes**

Many different variables have been used to describe jaw movement kinematics, including measurements of maximum vertical, lateral and anteroposterior excursions, angles of approach of the mandible into centric occlusion, cycle and phase durations, velocities and accelerations (Bates et al., 1975, Buschang et al., 2000). The majority of these jaw movement features are two-dimensional descriptors of the masticatory movement path and studies using these variables generally select arbitrary points in the masticatory cycle to easily demarcate phase changes and thereby calculate the desired measures used to analyse chewing motion paths (Buschang et al., 2000, Hattori et al., 2010). However, the masticatory movement path of the mandible is a three-dimensional continuous curve with no evident demarcations (Hattori et al., 2010). Therefore, it has been argued that much
information about the form of the mandibular movement path is discarded when analysing
variations in these variables during chewing (Buschang et al., 2000, Hattori et al., 2010).
On the other hand, the use of elliptic Fourier descriptors (Ferrario et al., 1990, Kuhl and
Giardina, 1982) has been demonstrated to be capable of capturing the kinematics of the
masticatory path in detail (Ferrario et al., 1990, Hattori et al., 2010). Moreover, the
analysis of the elliptic Fourier descriptors using principal component analysis allows for
the description of variations of the masticatory cycle paths during a chewing sequence
(Hattori et al., 2010, Igari et al., 2011). These features of this combination of analysis
techniques would be useful in the study of the association between changes in masticatory
kinematics during different orofacial functional tasks following the application of a
specific resistance exercise task.

Isometric resistance exercise resulted in changes in the mandibular movement path during
free chewing (Chapter 2). The use of principal component analysis on a variance-
covariance matrix in the quantitative analysis of masticatory motion paths of the human
jaw before and after the application of isometric resistance exercises to the muscles of
mastication found that five principal components (PCs), or independent movement
variations, out of a total 123 PCs were capable of describing around 92% of the total
variance in the masticatory motion paths when chewing cooked rice (Chapter 2). Of those
five PCs that contributed to the majority of the variations in the masticatory cycle paths,
three PCs showed significant changes following the resistance exercise task, namely PC2,
PC4 and PC5 (Figure 2.6 and Table 2.3). The application of a right sided resistance
exercise task resulted in a more horizontal and protruded masticatory movement path in
PC2, a rounder and longer masticatory movement path in PC4 and a narrower and shorter
masticatory movement path in PC5 (Figure 2.6 and Table 2.3). Furthermore, PC2
contributed to more than twice the variations in the masticatory cycle paths compared to PC4 and PC5 combined (Table 2.2), suggesting that PC2 may have a larger influence on the masticatory movement path of the jaw following the application of isometric resistance exercise. Clinically therefore, this may imply that the application of resistance exercise to a lateral jaw movement could more likely result in a masticatory movement path which is more horizontally orientated in the coronal plane and more protruded in the sagittal plane.

These findings are consistent with previous studies that have utilised either the same (Hattori et al., 2010) or similar analysis techniques (Igari et al., 2012, Kobayashi et al., 2009) to describe and analyse masticatory movement patterns. This thesis has demonstrated similar mandibular masticatory movement patterns to those described previously (Hattori et al., 2010, Igari et al., 2012, Kobayashi et al., 2009, Proschel, 1987) with the movement patterns found in the five PCs that described more than 90% of the total variance in the masticatory motion paths when chewing cooked rice resembling the two most common patterns found when chewing gum (Kobayashi et al., 2009) as well as resembling the majority of the eight different patterns of mandibular movement that have been described in asymptomatic individuals chewing standardised pieces of winegum and soft bread, with the predominant pattern having a teardrop shape (Proschel, 1987). Further, it has been previously shown that the linear combination of three independent movement variations can account for an average of 93% of the total variations in the masticatory cycle paths when chewing gum or gummy candy (Hattori et al., 2010). This compares favourably with the five independent movement variations that described around 92% of the total variance in the masticatory motion paths when chewing cooked rice (Chapter 2). Chewing different types of foods results in different functional requirements from the masticatory system (Foster et al., 2006, Peyron et al., 2004, Peyron et al., 2002, Woda et al.,
2006a, Woda et al., 2006b). Even though chewing cooked rice demonstrated similar movement patterns as those described when chewing gum (Kobayashi et al., 2009), cooked rice is a food bolus that changes shape and consistency throughout the chewing sequence while a standard pellet of gum is a food bolus that maintains its consistency throughout the chewing sequence. Therefore, these two different foodstuffs may impose different functional demands on the masticatory system during their respective chewing sequences. This may explain the different numbers of independent movement variations that contribute to the total variations in the masticatory cycle paths when chewing cooked rice (five PCs; Chapter 2) and gum or gummy candy (three PCs, Hattori et al., 2010).

Interestingly, significant differences were also demonstrated in the movement characteristics of the masticatory cycle following the application of a resistance exercise task during the chewing of gum (Chapter 3). These differences in the movement parameters were found between different testing sessions and not between the two groups of participants. Furthermore, these changes followed similar patterns of change across the testing sessions in both the Control group and the Exercise group (Figure 3.10, Figure 3.12 and Figure 3.13) thus suggesting that the jaw movement characteristics were similar in both groups at each of the testing sessions. This appears to be in contrast to the significant changes in masticatory movement paths when chewing cooked rice (Chapter 2). However, post hoc testing did reveal an immediate significant increase in the closing velocity of the jaw following the application of the resistance exercise task (Chapter 3). This lack of significant differences in the jaw movement characteristics may be consistent with the view that information about the form of the mandibular movement path may be lost when analysing variations in these variables during chewing (Buschang et al., 2000, Hattori et al., 2010). The use of elliptic Fourier descriptors in combination with principal component
analysis may therefore be a more appropriate method for describing masticatory
kinematics in future studies due to its capability of capturing the kinematics of the
masticatory path in detail (Ferrario et al., 1990, Hattori et al., 2010).

Significant changes in some of the movement parameters demonstrated in the current
thesis occurred in both the Control group and the Exercise group with similar patterns of
change reported in both experimental groups over the course of the second study in
particular (Chapter 3). Similar findings have been recently demonstrated in an
experimental pain model (Inamoto, Whittle and Murray, unpublished observations).
Following the resolution of a short period of moderate to severe masseter muscle pain that
subsided within 10 minutes, no significant between group differences were found in either
jaw movement amplitude or velocity or jaw muscle EMG activity during a free chewing
task that was standardised for timing (Inamoto, Whittle and Murray, unpublished
observations). However, significant increases in velocity and amplitude of chewing
between data collection blocks were demonstrated during a free chewing task but not
during the standardised chewing task. The absence of an increase in the jaw movement and
EMG activity during the standardised chewing task suggested that factors such as a
motivation to complete the experimental protocol may be more influential on the increased
velocity and amplitude of jaw movement seen in the free chewing task rather than the
presence of a practice or training effect (Inamoto, Whittle and Murray, unpublished
observations). A similar effect may also have occurred in the studies reported in this thesis
and may explain the similarities in the changes demonstrated in both the Control group and
the Exercise group. The inclusion of a standardised chewing task in future studies may
help determine the mechanisms of these similar pattern changes.
Electromyographic Changes During Mastication

Electromyographic changes were demonstrated in some of the muscles of mastication following the application of an isometric resistance exercise task. Upon completion of two weeks of the home-based isometric resistance exercise programme the contralateral anterior temporalis demonstrated a delayed onset in its EMG activity compared to the Control group, as well as a delay in its offset as a percentage of the chewing cycle. This may indicate that the contralateral anterior temporalis was active later in the closing phase of the chewing cycle after the completion of the home-based resistance exercise task. The contralateral anterior temporalis commences its EMG activity shortly after the commencement of the closing phase of the masticatory cycle (Miller, 1991). There is also coactivation of the inferior head of the lateral pterygoid, medial pterygoid and digastric during the closing phase of the masticatory cycle (Hannam and McMillan, 1994, Miller, 1991). The EMG activity of these muscles was not measured in the reported study (Chapter 3) however it is plausible to suggest that the activation patterns of these muscles may have also changed in order to compensate for the delayed onset of the contralateral anterior temporalis during the early stage of the closing phase of the masticatory cycle. These changes in the muscle activation patterns may be the result of neuroplastic changes in the primary motor cortex related to the muscles of mastication similar to those changes reported in tongue primary motor cortex following training of a novel tongue protrusion task (Boudreau et al., 2007, Svensson et al., 2006, Svensson et al., 2003) and/or the transversus abdominis primary motor cortex following the completion of a specific exercise programme involving its voluntary activation independently from other trunk muscles for two weeks (Tsao et al., 2010).
Along with the above mentioned changes in the contralateral anterior temporalis, the ipsilateral masseter also demonstrated a delayed offset as a percentage of the chewing cycle at the completion of the home-based resistance exercise task indicating that it may have remained active for a longer proportion of the chewing cycle (Figure 3.21, Panel C). Together, these findings provide further evidence for the inherent mechanical redundancies within the masticatory system (Langenbach and van Eijden, 2001, Lobbezoo et al., 2004, Van Eijden et al., 1990). The complex and diverse architecture of the muscles of mastication allows for multiple options for tendon pull (Hannam and McMillan, 1994, Schumacher, 1961) which in turn provide for a substantial array of potential patterns of muscle activation to produce jaw movements (Lobbezoo et al., 2004, Van Eijden et al., 1990). The masticatory central pattern generator, cortical masticatory area and/or the sensorimotor cortex are then capable of utilising these potential muscle activation patterns to appropriately control the movement of the jaw perhaps via changes in the recruitment of motoneurone task groups (Loeb, 1985, Murray and Peck, 2007) and adapt the masticatory movement paths to changes in the oral environment specific to the performed task (Avivi-Arber et al., 2011, Lund, 1991, Lund and Kolta, 2006, Nakamura and Katakura, 1995, Yamada et al., 2005).

Interestingly, following the initial period in which the Exercise group completed the resistance exercise task, the contralateral anterior temporalis demonstrated an earlier onset of EMG activity relative to jaw movement onset in both the Control group and Exercise group (Figure 3.15, Panel B). This earlier onset of EMG activity in the Control group appeared to remain after two weeks (S2) while the EMG onset of the Exercise group was more delayed than that of the Control group but did not demonstrate a significant difference from its baseline measure. Similar patterns of change in EMG activities, where
significant changes in both groups occurred after the initial resistance exercise task and only the Control group demonstrating changes after two weeks were also seen in the Time to Peak EMG Activity Relative to Jaw Movement Onset in the ipsilateral masseter (Figure 3.16, Panel C) and the EMG Offset Relative to Jaw Movement Onset in the contralateral anterior temporalis (Figure 3.17, Panel B). These findings suggest there was no change in the Exercise group after completion of the home-based resistance exercise programme in these variables. Moreover, these patterns of change have also been reported previously where, following four weeks of a home-based isotonic resistance exercise programme, participants in the Exercise group tended to maintain the majority of the tested variables at pre-exercise baseline values while participants in the Control group demonstrated significant changes over the same time period (Wirianski, 2009, Wirianski et al., 2014). Such results may indicate that undertaking a home-based resistance exercise task may result in a stabilisation of the motor strategy during jaw movements. Specific muscle training results in neural changes that allow an individual to better coordinate the activation of the trained muscle groups (Sale and MacDougall, 1981). Furthermore, the acquisition and maintenance of motor performance involves activity dependent plasticity at multiple sites throughout the nervous system (Sale and MacDougall, 1981, Wolpaw and Tennissen, 2001). In particular, neuroplasticity has been demonstrated following the training and subsequent improved performance of a novel tongue protrusion task (Boudreau et al., 2007, Svensson et al., 2006, Svensson et al., 2003) and following the completion of a specific exercise programme targeting the activation of transversus abdominis in patients with LBP (Tsao et al., 2010). Although improvements in the performance of the masticatory task were not measured in the studies reported in this thesis, it is plausible that a stabilisation of the motor strategy during jaw movements may indicate
a reduced variability in the choice of potential muscle activation patterns utilised by the masticatory system and may be indicative of a muscle training effect. This in turn may allow an individual to better coordinate the activation of the muscles of mastication during the functional task of chewing.

Application of a specific resistance exercise task against lateral jaw movement may change the onset of EMG activity of the jaw muscles during mastication. In particular, during the chewing cycle the ipsilateral masseter and anterior temporalis demonstrated earlier onsets of EMG activity. This was evidenced in the ipsilateral masseter by a reduction in the EMG Onset Relative to Jaw Movement Onset (Figure 3.15, Panel C), the Relative EMG Onset as a Percentage of Chew Duration (Figure 3.19, Panel C), and, the Relative EMG Onset as a Percentage of Chew Cycle Duration (Figure 3.20, Panel C), and in the ipsilateral anterior temporalis by a reduction in the EMG Onset Relative to Jaw Movement Onset (Figure 3.15, Panel A) and the Relative EMG Onset as a Percentage of Chew Cycle Duration (Figure 3.19, Panel A). Therapeutic exercises have been prescribed for the management of known biomechanical deficiencies of delayed muscle activation resulting from pain and/or injury at the patellofemoral joint (Cowan et al., 2002, Cowan et al., 2003), shoulder (Worsley et al., 2013), cervical spine (Falla et al., 2004a) and the lumbar spine (Tsao and Hodges, 2007, Tsao and Hodges, 2008) and have resulted in earlier activation of the targeted muscles during movements of the respective joints. At present it is unknown whether patients suffering from TMD also present with altered EMG patterns, especially delayed EMG onsets during the chewing cycle, similar to those reported in other joints. If future studies reveal delayed EMG onsets in the muscles of mastication in patients diagnosed with TMD then these findings may indicate that the prescription of an appropriate specific resistance
exercise to the muscles that display delayed EMG onsets may result in earlier EMG onsets in those muscles and may provide a mechanism for the resolution of TMD symptoms.

**Motor Control of Mastication**

The rhythmical pattern of mastication is produced and controlled by the masticatory central pattern generator located in the pons and medulla (Lund and Kolta, 2006, Lund and Enomoto, 1988) and is modulated by inputs from the primary motor cortex and primary somatosensory cortex of the face, the cortical masticatory area and by feedback from mechanoreceptors including those in the periodontal ligament, intraoral touch receptors and muscle spindles in the jaw closing muscles (Lund and Kolta, 2006, Rossignol et al., 1988). Specific muscle training induces neural changes that allow for better coordinated activation of the trained muscle groups (Sale and MacDougall, 1981) and the acquisition and maintenance of motor performance involves activity dependent plasticity at multiple sites throughout the nervous system (Sale and MacDougall, 1981, Wolpaw and Tennissen, 2001). Therefore, the application of specific resistance jaw exercises could produce changes in any one, or a combination, of the neuromuscular areas involved in the production of mastication and thereby influence its motor control. Different tasks could produce changes in the primary motor cortex, the primary somatosensory cortex and/or the cortical masticatory area which in turn could affect the masticatory central pattern generator. These cortical changes may in turn facilitate changes in the recruitment patterns of motoneurone task groups (Loeb, 1985, Murray and Peck, 2007) perhaps via functional muscle groups or triplets (Langenbach and van Eijden, 2001, Weijs, 1994) and thereby change the motor output to the muscles of mastication facilitating changes in jaw EMG activity and therefore motor patterns.
The application of specific resistance exercise to the muscles of mastication has been shown to produce jaw movement changes (Chapter 2) as well as EMG changes in these muscles (Chapter 3) during a chewing task. Together these results may provide further evidence that a specific resistance exercise task applied to the jaw may be capable of altering the motor control patterns of the muscles of mastication. A unilateral resistance exercise task applied to the mandible has previously been demonstrated to produce changes in the patterns of EMG activation in some of the muscles of mastication during a novel standardised unilateral jaw movement task (Wirianski, 2009, Wirianski et al., 2014). Similarly, changes in EMG activation patterns have been demonstrated in other musculoskeletal systems such as the knee (Cowan et al., 2002, Cowan et al., 2003), shoulder (Worsley et al., 2013), cervical spine (Falla et al., 2004a) and lumbar spine (Tsao and Hodges, 2007, Tsao and Hodges, 2008). In the lumbar spine, cortical changes have also been demonstrated in patients with chronic LBP (Tsao et al., 2008) which have subsequently been shown to be reversible following the application of a specific training programme for the transversus abdominis muscle (Tsao et al., 2010).

In the orofacial region, animal and human studies have demonstrated cortical neuroplasticity in face primary motor cortex and face primary sensory cortex following successful training of novel motor tasks (Boudreau et al., 2007, Svensson et al., 2006, Svensson et al., 2003, Avivi-Arber et al., 2011, Sessle et al., 2007). In humans, transcranial magnetic stimulation has shown significant enhancement of the motor evoked potentials recorded from the tongue musculature and decreases in the tongue primary motor cortex thresholds with concomitant improvements in performance following 15 minutes (Boudreau et al., 2007), one hour (Svensson et al., 2006) and one week (Svensson et al., 2003) training of a novel tongue protrusion task. Along with these findings, significant
increases in the size of the cortical motor maps of the tongue primary motor cortex have also been reported (Svensson et al., 2003, Svensson et al., 2006) as well as a lateral and anterior shift in the centre of gravity coordinates of the cortical map at the seven-days follow-up after one hour of tongue protrusion training (Svensson et al., 2006).

The current evidence from studies of the patellofemoral joint, shoulder, cervical and lumbar spines as well as the orofacial region suggests that specific exercise training may result in changes in the motor control patterns of the muscles that produce joint movements and that these changes may be driven by and/or produce reversible neuroplastic changes. The application of a specific unilateral resistance exercise to the mandible demonstrated temporal EMG changes in the muscles of mastication during a novel lateral jaw movement task (Wirianski, 2009, Wirianski et al., 2014). The current thesis has further complemented these findings by demonstrating changes in jaw movement trajectories (Chapter 2) along with temporal changes in the EMG activation of the muscles of mastication (Chapter 3) during the functional task of chewing. Unfortunately, neither of the two studies presented in this thesis combined the use of elliptic Fourier descriptors (Kuhl and Giardina, 1982) in conjunction with principal component analysis on a variance-covariance matrix in the quantitative analysis of masticatory motion paths of the jaw (Hattori et al., 2010, Igari et al., 2011) with the simultaneous collection of EMG data from the muscles of mastication. Nevertheless, based on the previous reports, the findings demonstrated in the current thesis may reflect changes in the central motor control of mastication brought about by the application of the exercise task. Moreover, these motor control changes may be the result of reversible neural plasticity at many levels of the nervous system including changes in the excitability of motoneurones, the reorganisation of the sensorimotor cortex and/or the cerebellum (Boudreau et al., 2007, Svensson et al., 2006, Svensson et al., 2003, Tsao et al.,
2010, Tsao and Hodges, 2007). Future studies combining the use of elliptic Fourier descriptors in conjunction with principal component analysis to quantify jaw movement trajectories with simultaneous collection of EMG data from the muscles of mastication will help elucidate the mechanisms of the motor control of the muscles of mastication and the resultant masticatory movement paths.

**Clinical Relevance**

Common signs and symptoms of TMD include limitations and/or deviations of mandibular movements (Schiffman et al., 2014, Laskin, 1969, Solberg, 1983, Bell, 1983) which may impact on an individual’s ability to chew. Masticatory movement patterns are produced and controlled by the masticatory central pattern generator and modulated by inputs and feedback from structures in both the central and peripheral nervous systems (Lund and Kolta, 2006, Avivi-Arber et al., 2011, Rossignol et al., 1988, Lund and Enomoto, 1988). Resistance exercise training increases muscle strength and improves task performance possibly as a result of neuroplastic changes in the central and peripheral nervous systems and structures that control joint movements (Boudreau et al., 2007, Sale and MacDougall, 1981) such as the muscles of mastication. This thesis has demonstrated that an isometric resistance exercise task applied against unilateral mandibular movement may produce changes in the masticatory movement path such that it becomes more horizontally orientated in the coronal plane and more protruded in the sagittal plane during chewing (Chapter 2). Clinically, this result may suggest a possible mechanism and/or indication for the application of a specific resistance exercise in the management of symptomatic jaw movement deviations in patients with TMD. For example, in the case where a patient diagnosed with TMD presents with a symptomatic left lateral deviation of their mandible during chewing, the application of a right sided isometric resistance exercise task, similar
to that demonstrated in this thesis, may strengthen the muscles of mastication that contribute to right lateral jaw movements. The resistance exercise task may also contribute to central and/or peripheral neuroplastic changes that may facilitate changes in the recruitment patterns of motoneurone task groups (Loeb, 1985, Murray and Peck, 2007) resulting in improved task performance (Boudreau et al., 2007, Sale and MacDougall, 1981) of the masticatory muscles in producing a right lateral force on the mandible. This may assist the muscles of mastication to compensate for the left lateral deviation and contribute to a more normal masticatory movement pattern thereby facilitating a reduction in TMD symptoms.

Musculoskeletal pain conditions have been reported to be associated with alterations in the activation patterns of the muscles that control the knee (Cowan et al., 2002, Cowan et al., 2001), shoulder (Cools et al., 2003, Wadsworth and Bullock-Saxton, 1997), cervical spine (Falla et al., 2004b, Falla et al., 2004a, Jull et al., 2004) and lumbar spine (Hodges and Richardson, 1996, O'Sullivan et al., 1998, Tsao and Hodges, 2008, Tsao and Hodges, 2007). Common findings of these pain conditions include biomechanical deficiencies of delayed muscle activation at these joints which are potentially either caused by or result in pain or injury. Specific exercise training has been demonstrated to result in earlier activation of the targeted muscles as well as reducing pain and disability and improving function in the knee (Cowan et al., 2002, Cowan et al., 2003, Crossley et al., 2002), shoulder (Ginn and Cohen, 2005, Ginn and Cohen, 2004, Ginn et al., 1997, Worsley et al., 2013), cervical spine (Falla et al., 2006, Jull et al., 2009, Falla et al., 2004a) and lumbar spine (Hides et al., 1996, Tsao and Hodges, 2008, Tsao and Hodges, 2007). This thesis demonstrated a significant reduction in variables related to the onset of EMG activities in the ipsilateral masseter and ipsilateral anterior temporalis following the application of the
resistance exercise task (Chapter 3). These findings indicate that the application of the resistance exercise task resulted in the onset of EMG activity occurring earlier in the masticatory cycle in these two ipsilateral muscles. Some patients with TMD present with altered mandibular movement patterns (Schiffman et al., 2014, Laskin, 1969, Solberg, 1983, Bell, 1983) which significantly impact on their ability to chew. If these altered jaw movement patterns are associated with altered muscle activation patterns of delayed EMG onsets in the muscles of mastication then the results of the current thesis suggest that a specific resistance exercise task may reverse the muscle activation delays and perhaps provide an avenue for the management of TMD.

Significant differences in the masticatory movement trajectories and EMG activation patterns of the muscles of mastication were demonstrated in some variables in the Control group over the course of the two presented studies whereas the values of these variables remained unchanged from baseline levels in the Exercise group (Chapter 2 and Chapter 3). Similar findings in asymptomatic individuals have been reported following the application of an isotonic resistance exercise task where significant changes also occurred in the Control group, while the Exercise group tended to maintain the majority of the tested variables at pre-exercise baseline values (Wirianski, 2009, Wirianski et al., 2014). These results tend to suggest that, in asymptomatic individuals, there may be a level of variability between recording sessions in the available motor control patterns in the human masticatory system during mastication (Chapter 2) as well as in the temporal characteristics of jaw movements and the recruitment patterns of some of the muscles of mastication (Wirianski, 2009, Wirianski et al., 2014) during chewing. Indeed, the complex and diverse architecture of the muscles of mastication could allow multiple options for tendon pull (Hannam and McMillan, 1994, Schumacher, 1961) thereby providing a large
array of potential patterns of muscle activation to produce jaw movements (Lobbezoo et al., 2004, Van Eijden et al., 1990, van Eijden and Turkawski, 2001) during mastication. The maintenance of the pre-exercise values in the Exercise groups demonstrated in this thesis and in our previous work (Wirianski, 2009, Wirianski et al., 2014) suggests that isotonic and isometric resistance exercises may result in stabilisation of the motor strategy (Sale and MacDougall, 1981). This stabilisation of motor strategies may reflect the neuroplastic changes that can occur with the acquisition and maintenance of specific motor performance at multiple sites throughout the central nervous system (Boudreau et al., 2007, Sale and MacDougall, 1981, Wolpaw and Tennissen, 2001) and therefore may be significant to understanding improvement in TMD symptoms (Au and Klineberg, 1993, Feine and Lund, 1997, List and Axelsson, 2010, McNeely et al., 2006, Medlicott and Harris, 2006, Nicolakis et al., 2001, Nicolakis et al., 2002, Rocabado and Iglarsh, 1991).

**Limitations**

Although the results demonstrated in this thesis appear promising it is important to recognise the small sample size in both of the two studies presented. The sample sizes of each group were n = 7 and n = 10 for the first (Chapter 2) and the second (Chapter 3) study respectively. In the first study, *post hoc* power analyses (Decision Support Systems LP, 2012, accessed on 1 May 2013) of the six experimental conditions in which the principal components (PCs) showed significant changes after the application of the resistance exercise task to the muscles of mastication revealed a calculated statistical power ranging from 0.77 (PC4) to 0.995 (PC3). In the second study, *post hoc* power analyses (Decision Support Systems LP, 2012, accessed on 4 August 2015) revealed a calculated statistical power of 0.77 for the primary outcome variable which was the EMG Time to Peak for the contralateral masseter. These power analyses are supportive that the majority of the
significant between group effects occurred as a result of the application of isometric resistance exercise to the muscles of mastication.

Even though significant changes were demonstrated with the current sample sizes, it is acknowledged that larger sample sizes may have yielded more robust results and therefore better quality information that may have contributed to a better understanding of the underlying mechanisms. In order to maximise the chance of detecting a true effect and the likelihood that a statistically significant result also reflects a true effect it is important to design a study with sufficiently high statistical power (Button et al., 2013). Therefore, future studies, especially those investigating the effects of resistance exercise tasks in a clinical setting of symptomatic participants, need to be designed with sufficiently high statistical power in order for researchers to be confident that statistically significant results reflect a true effect of the intervention being investigated (Button et al., 2013, Campbell et al., 1995). A priori sample size estimates should therefore be calculated in order to maximise the likelihood of detecting clinically relevant effects and help determine the appropriateness and efficacy of the investigated intervention(s).

Two separate studies have been presented in this thesis. The first study demonstrated significant differences in masticatory movement paths during the chewing of cooked rice following the application of an isometric resistance exercise task (Chapter 2) while the second study demonstrated temporal EMG changes in the muscles of mastication during the chewing of gum following the application of a similar isometric resistance exercise task (Chapter 3). Inferences have been made that the EMG changes may be associated with the masticatory jaw movement changes and that they are perhaps the result of neuroplastic changes in the central and/or peripheral nervous systems that control mastication. However,
these conclusions should be interpreted with a level of caution. It is important to recognise that each study investigated the effects of a similar resistance exercise task during two different yet similar chewing tasks. It is well recognised that chewing different types of foods results in different functional requirements from the masticatory system (Foster et al., 2006, Peyron et al., 2004, Peyron et al., 2002, Woda et al., 2006a, Woda et al., 2006b).

Cooked rice is a foodstuff that would gradually reduce in size and change consistency during mastication and therefore would be less likely to maintain its original shape and form during the chewing sequence. Chewing gum, on the other hand, has a more even consistency and therefore would have the potential to maintain its consistency throughout the chewing sequence. These differences between the food boluses may have contributed to the reported changes in the two studies through separate and unrelated mechanisms.

Combining the use of elliptic Fourier descriptors in conjunction with principal component analysis to quantify masticatory movement paths with the simultaneous collection of EMG activity from the muscles of mastication while chewing the same foodstuff would help to determine whether masticatory jaw movement changes are associated with concomitant changes in the EMG activation patterns of the muscles of mastication during chewing.

It is also important to acknowledge the possibility that the results of the reported studies may be chance findings. Both studies reported within-participant changes as well as between-groups changes. The second study was in part an heuristic, exploratory study that attempted to determine meaningful variables to examine in future works. Little is known about which variables are most appropriate to measure when investigating the temporal characteristics of the EMG activity in the muscles of mastication following the application of a resistance exercise task. Therefore many variables were analysed in an attempt to better understand which variables best reflect meaningful changes in muscle activity.
patterns during mastication which increases the chance of a Type I error. However, as described above, the Bonferonni correction was used to limit the overall Type 1 error and the power analyses are supportive that the majority of the significant between group effects occurred as a result of the application of isometric resistance exercise to the muscles of mastication.

**Future Directions**

As mentioned previously, the two studies presented in this thesis investigated the effects of a similar resistance exercise regimen on two slightly different masticatory tasks. Moreover, the two studies utilised different analysis techniques and investigated different aspects of mastication. The first study utilised elliptic Fourier descriptors in conjunction with principal component analysis to investigate the effects of the isometric resistance exercise task on masticatory movement paths during the chewing of cooked rice (Chapter 2) while the second study investigated the temporal EMG changes in the muscles of mastication during the chewing of gum (Chapter 3). Thus any inferences related to the association between the changes to the masticatory jaw movements and changes to the EMG activation patterns of the muscles of mastication should be viewed with caution. In order to investigate any associations that may result from the application of a resistance exercise task it would be prudent to combine the principal component analysis techniques with the analysis of the temporal characteristics of the EMG activity patterns of the muscles of mastication. Furthermore, the current thesis was limited to investigating the bilateral pair of the superficial muscles of mastication: anterior temporalis; and masseter. Incorporating the collection of EMG data from the other muscles of mastication including, inferior and superior head of lateral pterygoid, medial pterygoid and digastric, would yield more detail of the contributions of these muscles to the masticatory movement paths and further our
understanding of the effects of specific resistance exercise on the activation patterns of these muscles during mastication.

Many musculoskeletal pain conditions are associated with alterations in the activation patterns of the muscles that control particular joints including the knee (Cowan et al., 2002, Cowan et al., 2001), shoulder (Cools et al., 2003, Wadsworth and Bullock-Saxton, 1997), cervical spine (Falla et al., 2004b, Falla et al., 2004a, Jull et al., 2004) and lumbar spine (Hodges and Richardson, 1996, O'Sullivan et al., 1998, Tsao and Hodges, 2008, Tsao and Hodges, 2007). A common alteration that has been reported is a delay in the activation of the muscles controlling these joints. Therapeutic exercises prescribed to address these biomechanical deficiencies have resulted in earlier activation of the targeted muscles in the knee (Cowan et al., 2002, Cowan et al., 2003), shoulder (Worsley et al., 2013), cervical spine (Falla et al., 2004a) and the lumbar spine (Tsao and Hodges, 2007, Tsao and Hodges, 2008). Investigating a clinical population of patients with TMD who also have deviations in mandibular movements may help determine if the movement deviations are also associated with alterations in the patterns of EMG activation of the muscles of mastication. If it is revealed that patients with TMD symptoms demonstrate delayed muscle activation patterns then further studies could investigate the efficacy of a resistance exercise task, similar to the one utilised in the current thesis, in reversing the muscle activation delays. This would then assist clinicians in determining the appropriateness of prescribing specific resistance jaw exercises in this patient group to facilitate symptom reduction and a return to normal masticatory function.

Transcranial magnetic stimulation has been used extensively to demonstrate exercise induced neuroplastic changes in the orofacial region following successful training of novel
motor tasks, particularly in face primary motor cortex (Boudreau et al., 2007, Svensson et al., 2006, Svensson et al., 2003, Avivi-Arber et al., 2011, Sessle et al., 2007) and face primary sensory cortex (Avivi-Arber et al., 2011). Moreover, in people with recurrent LBP, transcranial magnetic stimulation has also demonstrated reduced motor thresholds for the stimulation of transversus abdominis and a reorganisation of the cortical representation of trunk muscles associated with postural control deficits of delayed onset of EMG activity in the transversus abdominis muscle during rapid arm movement tasks (Tsao et al., 2008). This reorganisation of the motor cortex found in LBP patients has further been demonstrated to be reversible with the application of specific exercises (Tsao et al., 2010). At the completion of two weeks of this skilled training programme the motor cortical representation of transversus abdominis became more anteriorly and medially placed and closely resembled those found in healthy, asymptomatic individuals (Tsao et al., 2008) with concomitant earlier activation of the transversus abdominis during a rapid arm movement task and reduced pain (Tsao et al., 2010). It is plausible therefore that transcranial magnetic stimulation could be utilised to investigate cortical changes in patients with TMD who also present with symptomatic deviations in mandibular movements. Furthermore, if cortical changes are demonstrated in this patient group then transcranial magnetic stimulation could also be used to investigate the effects of specific resistance exercise training of the muscles of mastication on neuroplasticity of the cortical representation of these muscles. Information gained from the results of these studies could then help to further elucidate the mechanisms of motor control of the masticatory system.
Conclusion

Temporomandibular disorders (TMD) are often associated with deviations in mandibular movements that may result in limitations of normal jaw function such as chewing and swallowing. Mastication is an intermittent, semiautomatic, rhythmical function that is produced and controlled by the masticatory central pattern generator located in the brainstem and modulated by the cortical masticatory area, the primary motor cortex, the primary somatosensory cortex and by feedback from peripheral mechanoreceptors including those in the periodontal ligament, intraoral touch receptors and muscle spindles in the jaw closing muscles. The complex neuromuscular interaction between these central and peripheral structures produces coordinated movements of the tongue, facial and masticatory muscles that result in food being placed between the teeth, cut and ground up into smaller pieces and mixed with saliva to produce a food bolus ready for swallowing.

This thesis investigated the effects of specific isometric resistance jaw exercises on mandibular movement patterns and the EMG activity patterns of the muscles of mastication during chewing. In asymptomatic individuals, resistance exercise applied against lateral jaw movements resulted in masticatory movement paths that were more horizontally orientated in the coronal plane and more protruded in the sagittal plane. Performance of the exercise task also resulted in an earlier onset of the EMG activity in the ipsilateral anterior temporalis and the ipsilateral masseter during the masticatory cycle.

The temporomandibular joint is one of many joints that comprise the human musculoskeletal system. In other joints, painful musculoskeletal conditions are often associated with biomechanical deficiencies of delayed muscle activation and the application of specific exercise training at these joints has resulted in earlier activation of
the targeted muscles. In patients with recurrent low back pain, these earlier muscle activation patterns have also been demonstrated to be associated with reversible neuroplastic changes of the cortical representation of the targeted transversus abdominis muscle that closely resembled those found in healthy, asymptomatic individuals as well as a reduction in pain. Currently it is unclear if patients with TMD also display similar biomechanical deficiencies of delayed activation of the muscles of mastication. However, neuroplastic changes associated with improvements in task performance have been demonstrated in the orofacial region following the training of a novel tongue protrusion task. These cortical changes in the orofacial region along with those reported in recurrent low back pain patients suggest that specific exercise training may result in centrally mediated changes in the motor control patterns of the muscles that produce and control joint movements. The changes in masticatory jaw movement trajectories and the temporal changes in EMG activation patterns of the muscles of mastication during chewing demonstrated in this thesis may also reflect changes in the central motor control of mastication brought about by the application of the exercise task.

Future studies are warranted to investigate the presence of delayed muscle activation patterns in patients with TMD who also present with deviations in masticatory movement patterns. If these biomechanical deficiencies are revealed in this patient group then the results of this thesis suggest that a specific exercise task may be capable of reversing the muscle activation delays as well as modifying the deviations in masticatory mandibular movements. This may contribute to a more normal masticatory movement pattern thereby facilitating a reduction in TMD symptoms and perhaps provide an avenue for the management of TMD. Furthermore, investigating the presence of cortical changes associated with any delayed muscle activation patterns and whether the application of a
specific resistance exercise task is associated with reversible neuroplasticity will help further develop our understanding of the mechanisms of motor control of the masticatory system. This will then assist clinicians in prescribing appropriate and effective exercise regimens as part of their management of patients with TMD.
References


Appendix 1: Ethics Committee Approvals

Tohoku University

Copy of the ethics approval letter dated 8 March 2011 from the Tohoku University Human Research Ethics Committee (No: 22-30). No English translation was made available.
Tokyo Medical and Dental University

Copy of the ethics approval letter dated 16 January 2013 from the Ethics Committee at the Faculty of Dentistry, Tokyo Medical and Dental University (No: 874). An English translation dated 15 May 2013 appears on the following page.
Ethics Committee Approval

The study entitled “The effects of exercise on Jaw Function of the human jaw during chewing” was approved by the ethics committee at Faculty of Dentistry, Tokyo Medical and Dental University.

Researcher: Ichiro Minami, DDS, PhD
Assistant Professor
Removable Partial Denture Prosthodontics, Graduate School of Medical and Dental Sciences, Tokyo Medical and Dental University,

Research number: 874
Date of approval: January/16/2013

Junji Tagami
Dean, Faculty of Dentistry
Date May 15, 2013

The ethics committee at Faculty of Dentistry, Tokyo Medical and Dental University
5-45, Yushima 1-chome, Bunkyo-ku,
Tokyo 113-8549 Japan
Appendix 2: Training Diary

The following pages show copies of the Training Diary developed for the study conducted at Tokyo Medical and Dental University (Chapter 3). The first Training Diary is the English version and the second is the Japanese translation. These Training Diaries were used by the participants that were randomly allocated to the Exercise Group. Participants were asked to complete the Training Diary during the two weeks that they completed the isometric resistance jaw exercise task.
Training Diary

Thank you for participating in this study. Please complete the Training Diary below every time you complete your jaw exercises session. Remember to do the exercise in the same way that we taught you. That is:

Push the right side of your jaw against your fingers or the palm of your right hand, whichever is more comfortable, at the level of force that we taught you (20-30% of your maximum).

Hold the force against the right side of your jaw for 10 seconds, and then relax the force.

Rest for 5 seconds.

Repeat this 10 times and then rest for 20-30 seconds.

You should NOT experience any pain or discomfort when doing these exercises.

If you feel any pain or discomfort stop the exercise and write this down in the comments section. Retry the exercises on the next scheduled occasion. If you continue to have pain or discomfort please contact the researchers.
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トレーニングダイヤリー

このたびは研究にご協力いただき、まことにありがとうございます。下記のトレーニングダイヤリーのご記載をお願いいたします。顎運動のトレーニング毎にチェックしてください。トレーニングは以下になります。

・手指あるいは掌を右のおとがい部に当てて、右方向に指定した力の大きさで下顎を動かし、同時に下顎が動かないように抵抗するようにして下さい。

・10秒間力を入れて5秒間休憩を10回繰り返し、20ないし30秒間の休憩を入れて下さい。

・これを5回繰り返して下さい。

One set of resistance jaw exercises

Exercise at 20-30% MVC

Repetitions of Resistance Jaw Exercises within each Set

1 2 3 4 5 6 7 8 9 10

Rest

Time (sec)

0 15 30 45 60 75 90 105 120 135 150 160

X 5

・この一連のトレーニングを朝、昼、夜と一日3回、2週間行って下さい。

・トレーニングは痛みがなく、かつ不快にならない程度で行って下さい。

・痛みや不快感が出た場合は、トレーニングはそこでまでとしてその旨をコメント欄に記載して下さい。これが続く場合は連絡を下さい。
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