THE EFFECT OF SHOULDER PAIN ON THE NEUROMUSCULAR ACTIVITY OF THE SCAPULAR STABILIZING MUSCLES

A dissertation in partial fulfillment of the requirements for the Degree of Masters of Philosophy in Sports Physiotherapy (Mphil Sports Physiotherapy) from the University of Cape Town (December 2005)

By

Delphine Oliver
Diploma in Physiotherapy, Zurich, Switzerland
BRNREG002

Departement of Physiotherapy and UCT/MRC Research Unit for Exercise Science and Sports Medicine,
Departement of Human Biology
Faculty of Health Sciences,
University of Cape Town,
South Africa

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DEDICATION

To my dearest husband Simon, who taught me how to love and play

ACKNOWLEGEMENT

This study would not have been possible without the dedicated support of many people

- My supervisors Prof A St Clair Gibson and Janine Gray
- My co-supervisors Dr Lester John and Sharhidd Taliep
- Dr Basi Vrettos, Dr Steve Roche and all my physio colleagues for referring subjects with shoulder pain
- Prof Mike Lambert, for help with the statistical analysis and continuous support
- All the subjects for taking part in my study
- All the staff and student representatives at the Unit of Exercise Science and Sports Medicine for allowing me to use a desk
- Ikraam for fixing the EMG machine many times
- Paul Rossouw for his help and support with technical problems
- Dr Wayne Viljoen for his help with the EMG recording and EMG analysis, his friendship, support and laughter throughout the process
- Dale Rae for sharing her maticulate research expertise so generously, for proof reading the research article of my thesis, for her many words of encouragement that are true pearls of wisdom
- Ross Tucker for his help in analyzing the data, his friendship and contagious enthusiasm for research
- My office mates Dale Rae, Zandile Mciza, Mariam Hassen and Yolande Harley for their friendship, advice and many discussions
- The friendships of all the students at ESSM, in particular Lize Havemann, Tina Plattner, Wayne Viljoen, Dale Rae, Zandile Mciza, Mariam Hassen, Yolande Harley, Hitesh Harribai, Yumna Albertus, Leani Beretta de Azevedo, Dr Tamara Hews and Bill Butler, George Mokone, Jonathan Dugas and many more
- Janet Keet for proof reading the literature review of my thesis and for finishing her own MPhil. research thesis, thus showing me that it can be done

- The brain research group for the many challenging discussions
- All the staff at the physio practice Calligeris and Diale for their support
- All the teachers in my life that taught me how to ask questions and trying to answer them
- My friends and family for keeping me going
- Simon Oliver, my dearest husband, for believing in me and his unconditional love and support
- My God and Creator, for my life, my community, my relationships and the amazing world we live in

DECLARATION

I, Regula Delphine Oliver-Brunner, hereby declare that the work on which this dissertation is based is my original work (except where acknowledgements indicate otherwise), and that neither the whole work, nor part of it, is being, or is to be submitted for another degree in this or any other university.

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Date:_____

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ABBREVIATIONS

ABD (Abduction)

ANOVA (Analysis of variance)

CNS (Central nervous system)

D (Deltoid)

EEG (Electromyography)

EMG (Electromyography)

SD (Standard deviation)

LT (Lower trapezuis)

SA (Serratus anterior)

UT (Upper trapezius)

CHAPTER 1

INTRODUCTION AND SCOPE OF THESIS

Shoulder pain has been described to be the third most common musculoskeletal pain after lower back pain and knee pain (Rekola *et al.*, 1993; Urwin *et al.*, 1998; Solomon *et al.*, 2001). Acute as well as chronic shoulder injuries are contributing factors to the development of shoulder pain. However, rotator cuff lesions, such as impingement syndrome, partial and full thickness tears have mostly been described to be responsible for chronic shoulder pain in particular (Solomon *et al.*, 2001). Biomechanical factors such as dysfunctions of the scapula, the humerus and the scapular stabilizing muscles are understood to play an integral part in the development and chronicity of chronic shoulder disorders (Hawkins and Kennedy, 1980; Ludewig and Cook, 2000; Ludewig and Cook, 2002; Michener *et al.*, 2003). However, in conjunction with biomechanical factors contributing to the pathological process, the literature also describes genetic and vascular factors which can contribute to the susceptibility of the tendon structures to the development of chronic injuries(Rathbun and Macnab, 1970; Harvie *et al.*, 2004). In addition, changes in muscle activity have also been described to contribute to the development and chronicity of chronic shoulder pain (Kibler and McMullen, 2003). Therefore, the underlying mechanism responsible for the development and chronicity of chronic shoulder disorders and chronic shoulder pain is multi-faceted.

The aim of this thesis was to review the current literature on factors contributing to chronic shoulder disorders and to report on a research study that was designed to investigate the neuromuscular activity of the scapular stabilizing muscles during an abduction movement of subjects with and without chronic shoulder impingement syndrome. EMG (electromyography) as well as EEG (electroencephalography) data was recorded during the testing procedure. For the purpose of this thesis only the EMG data was used for analysis.

The thesis is presented in the format of two papers that are to be submitted for publication in relevant clinical journals. The first paper (chapter 2) is the review of chronic shoulder pain. The second paper (chapter 3) is the original research paper entitled: "Neuromuscular activity of the scapular stabilizing muscles during a shoulder abduction movement in subjects with and without chronic shoulder impingement syndrome".

CHAPTER 2

REVIEW PAPER

A REVIEW OF CHRONIC SHOULDER PAIN

2.1 INTRODUCTION

The shoulder joint is unique due to its wide range of motion that allows skillful, yet powerful movements such as throwing in baseball. Optimal function of the shoulder requires a delicate balance between mobility and functional stability (Wilk *et al.*, 2002). Functional stability as well as movement of the shoulder joint are provided by the rotator cuff and the scapular stabilizing muscles, which are exquisitely coordinated by the central nervous system (Sarrafian, 1983; Norkin and Levangie, 1992). Yet the shoulder does not function in isolation. Rather it is part of a kinetic chain in which individual body segments are coordinated in their movements by muscle activity and body positions. This allows summation and transfer of forces through the individual body segments to the terminal link (Kibler, 1998). The benefits of the kinetic chain are both to optimize function and prevent overuse of certain structures. However, the shoulder joint is very susceptible to injuries due to the limited bony structures, which provide little articular stability yet allowing a wide range of movement (Hess, 2000). The musculotendinous complex of the rotator cuff, which provides most stability of the shoulder joint, has been described to be particularly at risk for acute as well as chronic overuse injuries (Vanvan der Windt *et al.*, 1995). Biomechanical factors have been described to predominantly contribute to the development of shoulder injuries (Wilk *et al.*, 2002; Burkhart *et al.*, 2003). Acute injuries, causing tissue damage, result in a local inflammatory response, thus irritating the peripheral nociceptors and so triggering a pain experience in the brain. Acute pain usually has an identifiable relationship to injury and tissue damage (Melzack, 1999). Chronic shoulder pain in contrast often persists beyond the duration of tissue healing. The specific tissue structure causing chronic shoulder pain is often not

identifiable. This is accentuated by the results of Yamaguchi et al (2001), demonstrating that rotator cuff tears are present in asymptomatic subjects. In addition, the mechanism underlying the transition of acute to chronic pain is unclear (Andrews, 2005). The relationship between tissue injury of the shoulder and chronic shoulder pain is therefore uncertain. However, genetic as well as psychological factors are suggested to play a role in developing acute and sustaining chronic shoulder injuries and shoulder pain (Takeuchi *et al.*, 2004; Harvie *et al.*, 2004).

This review explores the different models described in literature that attempt to explain the mechanism underlying chronic shoulder injury and pain.

2.2 EPIDEMIOLOGY

Musculoskeletal symptoms accounted for 54% of visits to a primary care facility in Norway (Hagen *et al.*, 2000). Shoulder disorders have shown to be the third most common musculoskeletal complaint after lower back and knee pain (Rekola *et al.*, 1997; Urwin *et al.*, 1998; Solomon *et al.*, 2001). Shoulder disorders are common in the work environment (Wofford *et al.*, 2005) as well as amongst the sporting community, in particular overhead athletes (McLeod and Andrews, 1986). The nature of shoulder disorders have been described to be of traumatic or degenerative origin (Vecchio *et al.*, 1995; van der Windt *et al.*, 1995; Wofford *et al.*, 2005). Young, male adults tend to present more often with traumatic, injury related shoulder disorders, whereas females of 35-74 years of age tend to complain more often of shoulder and neck pain of degenerative nature (Rekola *et al.*, 1993). However, in both genders the incidence of

shoulder pain has shown to increase with age (Urwin *et al.*, 1998). Several studies showed rotator cuff lesions (including impingement, partial and full thickness tears) to be the most commonly diagnosed pathology in shoulder disorders, followed by osteoarthritis, capsulitis, tendonitis, acromioclavicular joint pain and referred pain from the cervical spine (McLeod and Andrews, 1986; Vecchio *et al.*, 1995; van der Windt *et al.*, 1995; Croft *et al.*, 1996; Solomon *et al.*, 2001; Wofford *et al.*, 2005). Recurrent episodes of shoulder pain are common and can account for up to 50% of the incidence of shoulder complaints over a one year period (van der Windt *et al.*, 1995). Croft et al (1996) demonstrated in a prospective cohort study that only 21% of patients presenting with an acute shoulder disorder obtained full recovery after a 6 months period and 49% of the patients obtained full recovery after 12 months. Anatomical and biomechanical factors have predominantly been described to contribute to the development and recurrence of shoulder disorders (Wilk *et al.*, 2002; Burkhart *et al.*, 2003).

2.3 ANATOMY AND BIOMECHANICS

2.3.1 Introduction

Shoulder motion is unique due to its wide range of movement, which is achieved by several functional units. The glenohumeral joint, suprahumeral joint, sub-coracoacromial joint, scapulocostal joint, acromioclavicular joint, sternoclavicular joint, sternocostal joints, costovertebral joint and the facet joints of the thoracic and cervical spine all contribute to optimal function of the shoulder complex. The joints primarily contributing to the range of movement of the shoulder are the glenohumeral joint, which is the joint between the humeral head and the scapula, the acromioclavicular joint and the

sternoclavicular joint, which provides the only connection of the shoulder girdle to the thorax (Saha, 1983; Sarrafian, 1983). For optimal shoulder function there needs to be a fine balance between mobility and stability. The limited bony congruency of the humerus and the glenoid fossa renders the glenohumeral joint inherently unstable (Hess, 2000) . Stability of the shoulder complex is therefore critical and is provided by passive, active and neural stabilizing mechanisms (Wilk *et al.*, 1997; Hess, 2000).

2.3.2 Passive stabilizing mechanism

2.3.2.1 Glenohumeral joint

Bony geometry

The articular surface of the glenoid fossa is four times smaller than the humeral head, which allows excessive joint mobility and osseous stability is consequently very limited (Saha, 1983).

Glenoid labrum

The glenoid labrum is a rim that slightly deepens the genoid fossa, providing a larger and deeper contact area for the humeral head. It serves a cushioning function and provides an attachment for the glenohumeral ligaments. Nonetheless, its contribution to joint stability has been debated in literature (Sarrafian, 1983; Howell *et al.*, 1988).

Capsule and ligaments

The capsule attaches around the circumference of the glenoid labrum and the humeral head. The ligaments infiltrating into the capsule are the superior, middle and inferior glenohumeral ligaments as well as the coracohumeral ligament. The inferior glenohumeral ligament is the longest and strongest of the glenohumeral ligaments (Sarrafian, 1983). Its anterior band has been described to be the most important restraint for preventing anterior glenohumeral instability (Takubo *et al.*, 2005). Nerve endings, characteristic for joint position sense (proprioception), are concentrated in the inferior glenohumeral ligament in particularly. This neural component of the capsulo-ligamentous structures adds to joint stability by activating the rotator cuff via the reflex arch (Calvert, 1996). In the mid position of the range of movement of the glenohumeral joint, there is considerable laxity in the capsulo-ligamentous structures, allowing a wide range of motion. Aspects of the capsulo-ligamentous structures are selectively tightened in specific arm positions by winding up its fibers, stabilizing the glenohumeral joint at the end of the range of movement (Wilk *et al.*, 1997).

Intra-articular pressure

A negative pressure generated within the shoulder joint of approximately minus 4 mm/Hg, has been described to contribute to joint stability (Carr, 1996).

Joint cohesion mechanism

The synovial fluid contributes to joint stability while allowing movement to take place. Its high tensile strength resists pulling the joint surfaces apart, while its low shear strength maintains low resistance to the joint surfaces sliding on each other (Carr, 1996).

2.3.2.2 Acromioclavicular joint

The acromioclavicular joint attaches the scapula to the clavicle, providing a distal link between the upper limb and the chest wall. The primary function of the acromioclavicular joint is to maintain the relationship between the clavicle and the scapula in the early stages of elevation and to allow the scapula additional range of rotation in the latter stages of elevation (Norkin and Levangie, 1992).

2.3.2.3 Sternoclavicular joint

The sternoclavicular joint serves as a proximal and only link between the upper limb and the chest wall. Any movement of the upper limb would therefore be transmitted to the sternoclavicular joint (Norkin and Levangie, 1992). With other words, movement impairment of the sternoclavicular joint would compromise optimal shoulder function (Poppen and Walker, 1976).

In summary, the passive stabilizing structures only provide limited stability to the shoulder joint, while allowing a wide range of movement. The musculature surrounding the glenohumeral joint such as the rotator cuff and the scapular stabilizing muscles

(active stabilizing system) and the way they are coordinated (neural stabilizing system) are therefore critical for optimal shoulder function.

2.3.3 Active stabilizing mechanism

2.3.3.1 Rotator cuff

The rotator cuff is a musculotendinous complex formed by the supraspinatus, infraspinatus, teres minor and subscapularis muscle. These four muscles envelop the glenohumeral joint, with the supraspinatus lying superiorly, the infraspinatus and teres minor posteriorly and the subscapularis inferiorly to the joint. The rotator cuff muscles blend into the capsuloligamentous complex, thus stabilizing the glenohumeral joint (Bechtol, 1980). While the labrum and the capsuloligamentous structures provide glenohumeral joint stability at the end of the joint range, in the middle zone, the resting tone of the rotator cuff transmits tension to the capsuloligamentous structures, compressing the humeral head in the glenoid fossa (Sarrafian, 1983; Guanche *et al.*, 1995; Inman *et al.*, 1996; Schiffern *et al.*, 2002). The primary function of the rotator cuff together with the deltoid and biceps tendon is concerned with joint stability. However, activity of the rotator cuff also produces movement of the glenohumeral joint (Sarrafian, 1983; Norkin and Levangie, 1992; Rodosky *et al.*, 1994). For optimal shoulder function, the central nervous system needs to coordinate the rotator cuff muscles in such a way as to produce maximal range of movement, without compromising the stabilizing function of the muscles (neural stabilizing system).

As the glenoid fossa of the scapula provides the proximal joint surface of the glenohumeral joint, stability of the scapula is crucial for optimal shoulder function. The scapula gains stability from the scapular stabilizing muscles (Paine and Voight, 1993).

2.3.3.2 Scapular controlling muscles

The lack of ligamentous restraints between the scapula and the ribcage increases the demands on the scapular muscles to stabilize and control movements of the scapula. The muscles controlling the scapula are primarily the serratus anterior, upper, middle and lower trapezius, levator scapulae, rhomboid minor and major and pectoralis minor (Mottram, 1997; Kibler and McMullen, 2003). The serratus anterior takes its origin from the first eight ribs and runs along the ribcage to insert on the medial border of the scapula. Its primary role has been described to compress the scapula against the ribcage as well as providing upward rotation and posterior tipping of the scapula during glenohumeral elevation. With this complex function in mind, the serratus anterior has been classified as the primary stabilizing muscle in the scapulothoracic region (Basmajian J.V., 1963; Scovazzo *et al.*, 1991; Paine and Voight, 1993; Ruwe *et al.*, 1994; Magarey and Jones, 2003). The trapezius muscle finds its broad medial attachment along the nuchal line, occipital protuberance, ligamentum nuchea, spinous processes and supraspinous ligaments of C7-T12. The nuchal and occipital fibres of the upper trapezius run downwards and transversely, inserting on the lateral third of the clavicle. The fibers of the middle trapezius run horizontally to the acromion and the crest of the scapula (Johnson G *et al.*, 1994). These researchers propose that instead of elevating the scapula, as is commonly understood, the transverse fibers of the upper and middle trapezius draw

the scapula and clavicle backwards by rotating the clavicle at the sternoclavicular joint, thus elevating the acromion in the later phases of humeral elevation. These fibers do not change length much during rotation of the scapula and therefore it is suggested that their main function is to stabilize rather than move the scapula. The fibers of the lower trapezius ascend diagonally and insert at the medial end of the spine of the scapula. These fibers upwardly rotate the scapula during humeral elevation and resist the lateral displacement of the scapula caused by the pull of the serratus anterior. The lower trapezius has shown to be resistant to fatigue with regards to the time of onset of muscle activity as well as the amount of muscle activity, thus indicating the importance of its stabilizing function (McQuade *et al.*, 1998; Cools *et al.*, 2002). The levator scapulae originates from the spinous processes of C2-C5 and attaches to the superior angle of the scapula (Paine and Voight, 1993). Its function has been described to be rotation and stabilization of the scapula during humeral elevation (BASMAJIAN, 1963; Inman *et al.*, 1996). The rhomboid minor takes origin at the spinous process of C7 and T1 and inserts into the medial border of the scapula. The rhomboid major originates from the spinous processes of T2-T5 and inserts into the medial border of the scapula, below the rhomboid minor (Paine and Voight, 1993). The function of the rhomboids have been described to be concentric in nature during retraction of the scapula during the cocking and acceleration phase of the throwing motion, and eccentric in nature during the deceleration phase of the throwing motion (Ferlic and DiGiovine, 1988). The concentric and eccentric phase of muscle contraction needs to be well coordinated by the central nervous system in order to provide scapular movement and stability during functional activities such as throwing. The pectoralis minor originates from the second to sixth rib and finds its

insertion on the coracoid process of the scapula. Its function is multi-faceted producing depression, downward rotation and anterior tilt of the scapula (Paine and Voight, 1993).

Adapting Panjabi's (1992) model of stability of the lumbar spine, it is suggested that the function of the scapular stabilizing muscles are coordinated by the central nervous system in such a way as to provide optimal movement and stability to the shoulder complex (neural stabilizing system).

2.3.4 Neural control mechanism

2.3.4.2 Proprioception

The central nervous system demonstrates exquisite control of the afferent input obtained from mechanoreceptors in the joint capsule as well as rotator cuff muscles and tendons, and the efferent output in order to ensure dynamic stability of the glenohumeral joint throughout the range of movement (Carpenter *et al.*, 1998). Coordinated movement requires an intact joint position sense (proprioception) and appropriate muscle recruitment patterns (Guanche *et al.*, 1995; Myers and Lephart, 2000). In subjects with chronic shoulder impingement syndrome, decreased proprioception and a change in muscle activation patterns have been shown, thus indicating a change in the function of the central nervous system in the presence of pain and tissue damage (Wadsworth and Bullock-Saxton, 1997), (Machner *et al.*, 2003).

Force couples

The muscles around the shoulder work together in an intricate way, coordinated by the central nervous system, in order to provide dynamic stability while facilitating functional movement. The coordination of activation patterns of opposing muscles around a joint is termed a force couple, which is a word coined by the WHO (World Health Organization). During elevation of the shoulder, dynamic stability of the shoulder is maintained by three force couples (Bechtol, 1980; Norkin and Levangie, 1992; Inman *et al.*, 1996). The force couple in the frontal plane is formed by the deltoid and supraspinatus, superiorly, and the lower elements of the rotator cuff, inferiorly. During elevation of the shoulder, the lower rotator cuff muscles counteract the superior glide of the humeral head, produced by the pull of the deltoid, preventing impaction of the humeral head against the coracoid process (Sarrafian, 1983). In the transverse plane, the force couple produced by the subscapularis, anteriorly, and the infraspinatus/teres minor, posteriorly, plays a key role in maintaining joint stability around a normal axis of rotation throughout the range of movement (Bechtol, 1980; Saha, 1983; Norkin and Levangie, 1992). In the scapulothoracic area the force couple formed by the serratus anterior and the upper and lower trapezius plays a pivotal role in stabilizing the scapula during elevation of the shoulder, with the scapula providing a stable base for the humeral head (Inman *et al.*, 1996; Mottram, 1997).

In summary, coordination of the amount of activity in the muscles working as a force couple determines the mechanical displacement of the adjacent joint partners during a movement. However, the timing of the onset of muscle activity has also been shown to

impact on the function of the force couples and movements of the joint surfaces (David *et al.*, 2000).

2.3.4.4 Pre-activation

The timing of onset of muscle contraction in relation to the start of a movement has been studied in several joints such as the glenohumeral joint, scapulothoracic joint, cervical spine, lumbar spine and the knee joint. These studies have shown a distinct activation pattern of the muscles surrounding a particular joint, with some muscles contracting prior to the onset of movement and other muscles contracting after the onset of movement (Wadsworth and Bullock-Saxton, 1997; Hodges and Richardson, 1999b; David *et al.*, 2000; Cowan *et al.*, 2001; Falla *et al.*, 2004). Particular importance has been attributed to the muscles contracting prior to the onset of movement, as it is hypothesized that the pre-activation of these muscles provide joint stability prior to the onset of joint movement (Wadsworth and Bullock-Saxton, 1997; Hodges and Richardson, 1999b; David *et al.*, 2000; Cowan *et al.*, 2001; Falla *et al.*, 2004).

David et al (2000) investigated the timing of onset of muscle activity of the supraspinatus, infraspinatus, subscapularis, biceps, the anterior, middle and posterior deltoid, as well as the pectoralis major, in subjects without a shoulder disorder, during internal and external rotation of the glenohumeral joint. Their results demonstrated that the entire rotator cuff was activated prior to the onset of more superficial muscles, as well as prior to the onset of movement. Although the infraspinatus and subscapularis are considered to be the primary movers for internal and external rotation, their activity prior

to the onset of movement together with the entire rotator cuff supports the principal stabilizing function of these muscles. In the scapulothoracic area, Wadsworth and Bullocks (1997) have shown consistent pre-activation of the upper trapezius during bilateral abduction (ABD) movements of the shoulder in the scapular plane in subjects with and without shoulder disorder, while the serratus anterior and lower trapezius were activated after the onset of shoulder movement. It is hypothesized that the activation of the upper trapezius before the beginning of glenohumeral motion would provide a stable origin for the deltoid and supraspinatus muscles, hence optimizing their length-tension relationship (Wadsworth and Bullock-Saxton, 1997).

The concept of pre-activation has also been observed on a more global scale with regards to the entire kinetic chain with the stabilizing muscles in the pelvic region preceding movement of the shoulder (Hodges and Richardson, 1999a). It is understood that the proximal stability of the pelvic region would provide a stable base for the scapular stabilizing muscles, hence improving scapular stability during shoulder movement.

In summary, stability as well as the optimal movement of the shoulder complex is primarily provided by the rotator cuff and scapular stabilizing muscles, which are coordinated by the central nervous system. However, optimal shoulder movement is not just reliant on well coordinated musculature, but also on well functioning joints of the shoulder complex that contribute to shoulder movement such as the glenohumeral, scapulothoracic, acromioclavicular and the sternoclavicular joints, as well as the thoracic and cervical spine(Saha, 1983; Sarrafian, 1983; Kibler, 1998). Nevertheless, any

movement between two joint partners is in itself a composite of a movement that changes the angle between the two leverages and a rolling and gliding motion of the two joint surfaces on each other (arthrokinematics). Therefore, optimal shoulder function depends on sound coordination of the musculature as well as the integrity of each joint contributing to movement of the shoulder complex.

2.3.5 Arthrokinematics

2.2.5.1 Introduction

Arthrokinematics refers to the study of the movement of adjacent joint surfaces on each other during a movement of the joint that changes the angle between the two leverages. The joint capsule and the muscles that stabilize the joint control these arthrokinematic movements (Norkin and Levangie, 1992).

2.2.5.2 Roll and glide

A rolling and gliding motion takes place between two joint surfaces in order to increase the range of movement of the joint. At the glenohumeral joint, the head of the humerus is four times the size of the glenoid fossa, which renders the two joint surfaces incongruent. For the glenohumeral joint to produce a wide range of movement, a simultaneous rolling and gliding takes place of the humeral head on the glenoid fossa (Saha, 1983; Howell *et al.*, 1988; Harryman *et al.*, 1990; Norkin and Levangie, 1992; Ludewig and Cook, 2002). A posterior translation of the humeral head relative to the center of the glenoid fossa has been demonstrated between 30°-90° of ABD, followed by an anterior translation between 90°-120° of ABD. There is controversy in the literature about the superior and inferior

glides of the humeral head during ABD of the shoulder. General consensus is that the head of the humerus is only centered between 30°-150° of shoulder ABD (Howell *et al.*, 1988; Harryman *et al.*, 1990; Graichen *et al.*, 2000; Ludewig and Cook, 2002; Graichen *et al.*, 2005). Therefore, as the axis of rotation of the glenohumeral joint changes throughout the range of movement in a superior/inferior and anterior/posterior direction, the action of the rotator cuff muscles surrounding the joint has to adapt continuously, maintaining a centered humeral head and a stable glenohumeral joint (Graichen *et al.*, 2000; Graichen *et al.*, 2005).

In summary, the rolling and gliding motion of the humeral head on the glenoid surface increases the range of movement locally at the glenohumeral joint as well as maintaining a centered humeral head providing glenohumeral stability. From a more global perspective, combined movement at the scapulothoracic and glenohumeral joint allows an even wider range of movement of the shoulder.

2.3.5.3 Humero-scapulo-thoracic gliding mechanism

Shoulder motion represents an associated pattern of glenohumeral and scapulothoracic motion (Lukasiewicz *et al.*, 1999). The ratio of humeral and scapular contribution to shoulder movement has been described to be approximately 2:1, although individual differences do occur (Poppen and Walker, 1976; Norkin and Levangie, 1992). During glenohumeral elevation the scapula glides on the thorax in order to adjust the axis of rotation at the scapulothoracic joint from being at the root of the scapular spine at the beginning of elevation and subsequently moving towards the acromioclavicular joint at

the end of elevation (Bagg and Forrest, 1988). The contribution of the scapulothoracic articulation to shoulder motion plays an important role with regards to optimal shoulder function. Firstly, movement of the scapula orientates the glenoid fossa so that it can provide a stable base for articulation with the humeral head (Norkin and Levangie, 1992; Paine and Voight, 1993; Kibler and McMullen, 2003). Secondly, the combination of glenohumeral and scapulothoracic movement allows a much wider range of movement of the shoulder (Saha, 1983; Norkin and Levangie, 1992; Hess, 2000). Thirdly, the upward motion of the acromion (lateral rotation of the scapula) during elevation and ABD prevents impaction of the humeral head against the acromion (Saha, 1973). And lastly, as the scapula provides a base for the attachment of the rotator cuff and scapulothoracic stabilizing muscles, scapulothoracic movement associated with glenohumeral movement optimizes the length–tension relationship of the muscles that have attachments on the humerus and the scapula (Norkin and Levangie, 1992; Kibler and McMullen, 2003).

Arthrokinematic studies of the glenohumeral and scapulothoracic joint show that during the initial 60° of elevation and 30° of ABD, shoulder movement consists primarily of glenohumeral movement, which is associated with contraction of the supraspinatus and deltoid muscles (Saha, 1983; Burkhart *et al.*, 2003). During the initial part of elevation, the limited involvement of the scapulothoracic joint is associated with activity of the upper fibers of trapezius initiating upward rotation of the acromion and activity of the serratus anterior stabilizing the scapula against the thorax. Further elevation requires the scapula to engage with movements that involve upward rotation (around an anterior-posterior axis), posterior tipping (around a medial-lateral axis) and external rotation

(around a superior-inferior axis) to allow full glenohumeral elevation (Saha, 1983; Ludewig *et al.*, 1996; Mottram, 1997; Lukasiewicz *et al.*, 1999; McClure *et al.*, 2001). Muscle activity, during the latter part of elevation, entails a decrease in the activity of the upper trapezius, while the lower fibres of the trapezius and the serratus anterior muscles increase activity (BASMAJIAN, 1963).

In summary, optimal shoulder function requires stability, as well as mobility, of the entire shoulder complex, which is provided by anatomical and biomechanical aspects, as well as well coordinated muscle activation patterns of the rotator cuff and the scapula stabilizing muscles. However, the limited bony stabilizing structures of the glenohumeral joint, although allowing a wide range of movement, cause the shoulder complex to be very susceptible to acute and overuse injuries.

2.4 PATHOPHYSIOLOGY

2.4.1 Introduction

The pathophysiology underlying chronic shoulder disorders has been described to predominantly be caused by rotator cuff tendon pathology (Vecchio *et al.*, 1995; Wofford *et al.*, 2005). These pathologies have shown to be on a continuum from mild edema, to tendinopathy, to micro tear, to full thickness tear of the rotator cuff tendons (Lyons and Orwin, 1998). Rotator cuff pathologies are common in the general population and are exacerbated by work or sport related overhead activities, which implies that biomechanical aspects are contributing to the development of the pathology. Studies have shown that rotator cuff pathologies often occur bilaterally, although only one side may be

symptomatic (Sher *et al.*, 1995; Yamaguchi *et al.*, 2001). This poses an interesting question as to the origin of chronic shoulder pain as tissue damage is not entirely responsible for the perceived pain. Various models have been described to explain the pathophysiological mechanisms underlying chronic shoulder disorders, as well as chronic shoulder pain (Rathbun and Macnab, 1970; Neer, 1983; Harvie *et al.*, 2004).

2.4.2 Biomechanical model

2.4.2.1 Introduction

The biomechanical model is an attempt to explain the mechanisms underlying the development of the most common shoulder disorder which has been described as rotator cuff pathology. Neer (1983) first described the pathomechanics of rotator cuff pathology to be of a mechanical nature, causing compression of the subacromial structures between the anterior undersurface of the acromion, the acromioclavicular ligament, as well as the acromioclavicular joint and the greater tuberosity of the humeral head (Neer, 1983). Subacromial structures include the rotator cuff tendons, the tendon of the long head of biceps, the subacromial bursa and the capsule of the shoulder joint. Neer (1983) termed the disorder of mechanical compression of the subacromial structures, subacromial impingement syndrome. All the subacromial structures can be affected by mechanical compression although pathologies of the rotator cuff tendons are most common. Neer's classification of subacromial impingement syndrome is based on the degree of tissue injury. Stage 1 entails edema and hemorrhage in the tendon and is found in young patients under the age of 25 years who participate in overhead activities in sport and work. This stage is fully reversible and treated conservatively. Progression to stage 2 is

characterized by further degeneration of the tendon with development of fibrosis and tendinosis and is found in 25 - 40 year old patients. These patients report recurrent painful episodes with activity. A conservative rehabilitative approach to treatment is mostly applied. Further progression of the pathology to stage 3 is defined by the development of a bony spur and partial or full thickness tendon rupture affecting those over 40 years of age. Reported symptoms include recurrent episodes of pain and weakness. A surgical approach to treatment is predominantly used with repair of the tendon rupture and/or acromioplasty (Hawkins and Kennedy, 1980; Neer, 1983).

Neer's model of subacromial impingement syndrome does offer an explanation as to the pathomechanics of rotator cuff pathology. However the model does not include an explanation of the underlying mechanisms causing the narrowing of the subacromial space. Literature describes changes of the acromion and acromioclavicular joint, scapular dysfunction, glenohumeral dysfunction and postural dysfunctions to be contributing factors to the narrowing of the subacromial space resulting in compression of the subacromial structures (Neer, 1983; Greenfield *et al.*, 1995; Kibler, 1998; Magarey and Jones, 2003).

2.4.2.2 Acromial and acromiolavicular joint morphology

Hereditary prominence of the acromion at the anterior edge on its undersurface and a prominent greater humeral tuberosity that limit the subacromial space, have been described as contributing factors in the development of shoulder impingement (Hawkins and Kennedy, 1980; Neer, 1983; Flatow *et al.*, 1994). Acquired osteoarthritic changes of

the acromioclavicular joint with osteophytic lipping have also been described to compromise the subacromial space resulting in mechanical tendon injuries (Jerosch *et al.*, 1990). However, bony exostosis such as osteophytic lipping have been described to be a result of long term tissue overuse and degeneration and may not be the primary cause of tendon pathology (Budoff *et al.*, 1998). Some authors have therefore debated the relevance of acromioplasty, a common surgical procedure to increase the subacromial space, as it does not address the underlying problem of tissue overuse (Budoff *et al.*, 1998).

2.4.2.3 Scapula

Scapular movement plays an integral part during humeral elevation and is reliant on optimal function of the scapular stabilizing muscles. Subjects with chronic shoulder disorders have shown abnormal movement patterns of the scapula, as well as dysfunction of the scapular stabilizing muscles. Kibler (1998) defined the abnormal patterns of the scapula, termed scapular dyskinesis, as observable alterations in the position of the scapula and the patterns of scapular motion in relation to the thorax. Dyskinesis of the scapula has been described to include decreased scapular posterior tipping (Lukasiewicz *et al.*, 1999; Ludewig and Cook, 2000; Hebert *et al.*, 2002), decreased scapular upward rotation (Ludewig and Cook, 2000) and increased scapular internal rotation (Warner *et al.*, 1992; Ludewig and Cook, 2000). The underlying cause for scapular dyskinesis is thought to be dysfunction of the scapular stabilizing muscles. A number of studies have reported dysfunction of the upper trapezius, lower trapezius and serratus anterior muscle in subjects with chronic shoulder pain compared to pain free subjects (Scovazzo *et al.*,

1991; Ruwe *et al.*, 1994; Kibler, 1998; Ludewig and Cook, 2000). In a study on swimmers with and without subacromial impingement syndrome the firing pattern of the upper trapezius, lower trapezius and serratus anterior muscle has shown to be consistent with the activation of the upper trapezius prior to the onset of movement, while the lower trapezius and serratus anterior were activated after the onset of movement (Wadsworth and Bullock-Saxton, 1997). Nevertheless, the subjects with subacromial impingement syndrome showed increased variability in the onset of muscle contraction of the upper trapezius, lower trapezius as well as serratus anterior and a significant delay in serratus anterior activation on the non-injured side. Muscle dysfunction and scapular dyskinesis have shown to be a non-specific phenomenon of painful conditions of the shoulder complex.

The mechanism underlying muscular dysfunction is not well understood, although it is hypothesized by many authors that changes in the central nervous system as a result of pain may inhibit muscular activity (Warner *et al.*, 1992; Kibler and McMullen, 2003). Inhibition is a process in which the output of the motor system is reduced by an active process. For instance there is evidence of hyperexcitability of the motor neuron at the level of the spinal cord associated with acute pain (Dubner and Ruda, 1992). However, it is not possible from the available literature to determine whether pain inhibition underlies the changes in muscular activity in subjects with chronic shoulder pain.

In summary, dysfunction of the muscles controlling the scapula and the resulting biomechanical abnormality of the scapula, are thought to compromise the subacromial

joint space during function, hence impinging the rotator cuff tendon under the acromion (Neer, 1983; Warner *et al.*, 1992; Hebert *et al.*, 2002). However, a reduction of the subacromial space also occurs functionally in healthy subjects during elevation of the humeral head between 90°-110° or when the body is held in a kyphotic posture of the thoracic spine with protracted scapulae (Lewis *et al.*, 2005b). It is when the subacromial space is reduced repetitively or over extended periods of time that the subacromial structures may develop pathologies due to the mechanical compression. Alternatively, if pathologies of the subacromial structures are present, reduction of the subacromial space and mechanical compression may maintain and exacerbate the pathologies. It is unclear which problem develops first, the pathology of the subacromial structures, the muscular dysfunction or the biomechanical changes. Most likely, it is a combination of factors contributing to the development of pathologies of the subacromial structures.

2.4.2.4 Glenohumeral joint

In the normal shoulder, the rotator cuff muscles that work as force couples centering the humeral head in the glenoid fossa throughout glenohumeral elevation, grant optimal shoulder function (Howell *et al.*, 1988; Magarey and Jones, 2003). In subjects with subacromial impingement syndrome, dysfunction of the rotator cuff muscles have been demonstrated, as well as a loss of a centered humeral head in the glenoid fossa due to altered translations of the humeral head (Poppen and Walker, 1976; Reddy *et al.*, 2000; Magarey and Jones, 2003). A significant decrease in the activity of the infraspinatus, subscapularis and middle deltoid muscles have been identified in subjects with subacromial impingement syndrome during ABD in the scapular plane between 30°-60°

(Reddy *et al.*, 2000). The dysfunction of the force couple between the infraspinatus and the subscapularis results in abnormal translations of the humeral head in an anterior and superior direction (Poppen and Walker, 1976; Sharkey and Marder, 1995; Payne *et al.*, 1997; Reddy *et al.*, 2000). It is hypothesized that these biomechanical changes contribute to the reduction of the subacromial space resulting in an increased mechanical compression of the subacromial structures (Flatow *et al.*, 1994; Ludewig and Cook, 2002). Another common feature in subjects with chronic shoulder impingement syndrome is a tight posterior capsule of the glenohumeral joint, identified by a reduction of glenohumeral internal rotation, which is a common feature in subjects with subacromial impingement syndrome. Posterior capsular tightness is possibly a result of disuse due to pain in subjects with subacromial impingement syndrome, as well as biomechanical changes of the scapula and the humeral head. It has been found that posterior capsular tightness increases anterior and superior translation of the humeral head, which leads to further reduction of the subacromial space, thus increasing mechanical compression of the subacromial structures (Tyler *et al.*, 2000). The compression of the subacromial structures, due to a reduced subacromial space, becomes particularly problematic between 90°-110° of humeral elevation. This range of movement corresponds, firstly, with the highest value of subacromial pressure (Nordt *et al.*, 1999) and, secondly, with the greatest contact of the rotator cuff and biceps tendon with the subacromial arc (Flatow *et al.*, 1994). Hence, a painful arc from 90°-110°, is a common finding in people with chronic impingement syndrome (Neer, 1983).

In summary, a reduction of the subacromial space, and subsequent mechanical compression of the subacromial structures, may be caused by either dysfunction of the force couples around the glenohumeral joint or by a tight posterior capsule.

2.4.2.5 Posture

Postural dysfunctions of the upper body, as described by Kendall et al (1983), have been identified in people with chronic shoulder impingement syndrome (Kendall and Kendall McCreary, 1983; Greenfield *et al.*, 1995) and are clinically thought to contribute to the development of the pathology. These postural dysfunctions include kyphosis of the thoracic spine, protraction of the cervical spine (lower cervical flexion with upper cervical extension) as well as scapular protraction (increased scapular elevation, anterior tilt and internal rotation) (Greenfield *et al.*, 1995; Kebaetse *et al.*, 1999). It is postulated that dysfunction of the scapula may reduce the subacromial space with subsequent mechanical compression of the subacromial structures (Warner *et al.*, 1992; Kebaetse *et al.*, 1999). The postural alignment of the scapula has shown to be affected by a kyphotic posture of the thoracic spine resulting in increased scapular elevation, decreased posterior tilt and increased internal rotation, which may reduce the subacromial space (Kebaetse *et al.*, 1999; Finley and Lee, 2003). Although, in the clinical setting, postural dysfunctions of the upper body are commonly thought to contribute to the development of shoulder impingement syndrome, there is insufficient evidence supporting these theories (Lewis *et al.*, 2005a). In addition, postural dysfunctions of the upper body are not exclusive to subjects with shoulder impingement syndrome but are a common feature in non-symptomatic people as well. However, postural changes of the thoracic spine from a

kyphotic to a more upright posture resulted in an increase of the range of shoulder elevation in subjects with shoulder impingement syndrome (Lewis *et al.*, 2005b). Although the change of posture had no effect on the intensity of shoulder pain, the point in the range of shoulder elevation at which the subjects experienced pain was significantly higher (Lewis *et al.*, 2005b). Therefore, postural dysfunctions of the upper body may not be a cause of shoulder impingement syndrome but they have shown to affect shoulder function once tendon pathology exists.

In summary, the biomechanical model suggests that muscular dysfunction of the rotator cuff and scapular stabilizing muscles, biomechanical changes of the glenohumeral joint and scapulothoracic joint, as well as postural dysfunctions of the upper body, may be linked together in a pathological cycle, reducing the subacromial space and thus resulting in mechanical compression of the subacromial structures. However, it is unclear where the pathological cycle starts, namely with muscular dysfunction, biomechanical changes, postural dysfunctions or tissue pathology. Secondly, the biomechanical model does not elucidate the pathomechanics at a structural level of the pathological tendon and, thirdly, it does not explain the origin of pain. Therefore, the vascular, genetic and neural model makes an attempt to explain further aspects of chronic shoulder disorders such as the pathomechanics of tendon pathology at a structural level, genetic predisposition to shoulder disorders and the mechanism of pain.

2.4.3 The vascular model

In the blood supply of a healthy supraspinatus and biceps tendon an avascular zone has been identified at the area of the attachment when the glenohumeral joint is in a neutral position (Moseley and Glodie, 1963; Rathbun and Macnab, 1970). Several studies have shown that the avascular zone in these tendons corresponds to the most common site of tendinosis and tendon rupture (Moseley and Glodie, 1963; Rathbun and Macnab, 1970; Stein *et al.*, 2000). It is hypothesized by these authors that this avascular zone in the tendon hinders the healing process of tissue breakdown induced by overuse of the tendon. Although a restricted blood supply of the tendon at its attachment may predispose the tendon to pathologies induced by overuse, with the progression of the degenerative process in the tendon, secondary vascular changes in form of hypervascularisation have also been observed (Rathbun and Macnab, 1970; Alfredson and Ohberg, 2005). Nevertheless, it is unknown whether the hypervascularisation of the tendon, which has been linked to the degenerative process, enhances the pathological process or supports the healing process of the tendon.

2.4.4 The genetic model

In a study on the etiology of rotator cuff tears, it has been shown that siblings had more than twice the relative risk of developing full thickness rotator cuff tears compared to a control group consisting of their spouses, and five times the risk of developing associated symptoms (Harvie *et al.*, 2004). These results indicate a genetic susceptibility to rotator cuff tears at the level of the ultra structure of the tendon. These findings are supported by a study on achilles tendons, that has recently shown an association between the guanine-

thymine dinucleotide repeat polymorphism within the tenacin-C gene and achilles tendon injuries (Mokone *et al.*, 2005). However, studies have shown that not all rotator cuff tears are symptomatic (Yamaguchi *et al.*, 2001). The study of Harvie et al (2004) supports the findings of asymptomatic tears and demonstrated that genetic factors contribute to the development of symptoms associated with rotator cuff tears. It may be hypothesized that genetic factors at any point along the sensori-neural pathway may contribute to the development of symptoms associated with rotator cuff tears (Harvie *et al.*, 2004).

2.3.4 The neural model

Patients seeking medical attention due to a shoulder disorder most frequently complain of pain or pain in combination with weakness or stiffness. Less common symptoms associated with shoulder disorders are weakness or stiffness without pain (van der Windt *et al.*, 1995; Rekola *et al.*, 1997; Wofford *et al.*, 2005). It is commonly postulated that pain associated with shoulder disorders is due to an inflammatory process in the tissue, initiated in the acute phase of injury (Akgun *et al.*, 2004). However, clinical observation shows that pain frequently persists into the period of chronicity, when tissue healing should have passed the inflammatory stage. In addition, the absence of inflammatory cells has been described in injured tendons (Khan *et al.*, 1999). Further, studies have shown that not all rotator cuff tears are symptomatic (Yamaguchi *et al.*, 2001). Therefore, as rotator cuff tendon pathology is the most frequently described diagnosis in shoulder disorders, the question as to where the pain comes from, is of great interest (Sher *et al.*, 1995; Khan *et al.*, 2000). Consequently, it may be postulated that either the biochemical substance causing pain in tendon injuries has not yet been identified (Khan *et al.*, 2000)

and/or that pain is not exclusively dependant on tissue injury (Melzack, 1999). Moseley (2003) recently explained pain to be: "an output of the brain that is produced whenever the brain concludes that body tissue is in danger and action is required". When pain is felt, an individual–specific pain neuromatrix is activated in the brain which is a combination of cortical mechanisms that, when stimulated, produce pain (Melzack, 1999; Moseley, 2003). The pain neuromatrix may be activated and modulated by noxious stimuli, as in the case of tissue injury, but also via non-noxious stimuli such as memory of pain, belief system about pain, meaning of pain, culture and genetically determined aspects. When pain persists into the period of chronicity, changes have been observed at the level of the spinal cord and the brain, which result in an increased sensitivity to noxious and non-noxious stimuli, hence activating the pain neuromatrix at a lower threshold level (Dubner and Ruda, 1992; Flor *et al.*, 1997). Therefore, it may be postulated that initially shoulder pain might be partially related to tissue injury but more complex dynamics of the central nervous system may be involved, particularly in developing and sustaining chronic shoulder pain.

2.5 DISCUSSION

The literature describes rotator cuff lesions to be the most commonly diagnosed pathology in shoulder disorders (McLeod and Andrews, 1986; Vecchio *et al.*, 1995; Croft *et al.*, 1996; Solomon *et al.*, 2001; Wofford *et al.*, 2005). Most frequently, the biomechanical model is used to explain the pathogenesis underlying the development of rotator cuff lesions (Neer, 1983). A narrowing of the subacromial space is understood to increase mechanical load on the rotator cuff tendons that then undergo a progressive

degenerative process (Hawkins and Kennedy, 1980). The subacromial space may be reduced due to the morphology of the acromion itself or indirectly due to biomechanical changes of the scapula and/or the humeral head. The biomechanical changes of the scapula and the humeral head have been associated with postural imbalances of the thoracic and cervical spine, as well as muscular dysfunction of the scapular stabilizing muscles and the rotator cuff (Jerosch *et al.*, 1990; Kebaetse *et al.*, 1999; Ludewig and Cook, 2000). The mechanism underlying muscular dysfunction is not well understood. In the acute phase after a soft tissue injury, changes in muscular control may be initiated by reflex inhibition on a spinal cord level due to the presence of pain, effusion and the inflammatory response in the injured tissue (Shakespeare *et al.*, 1985). But the changes in muscular function are often sustained beyond the acute phase and may contribute to the chronicity of musculoskeletal problems. It has been suggested that the central nervous system may accentuate these changes in efferent output, such as altered muscle activation pattern, muscle strength and motor planning (Scovazzo *et al.*, 1991; Ludewig and Cook, 2000; Sterling *et al.*, 2001; Cools *et al.*, 2004; Moseley *et al.*, 2004b). However, the central nervous system may also be affected by changes in the afferent sensory input or in the way the brain interprets the afferent sensory input. A change in afferent sensory feedback to the brain has been observed with a decrease in proprioception in subjects with shoulder impingement syndrome (Machner *et al.*, 2003). In the brain itself, cortical re-organization of the representation of the body in the somatosensory motor cortex, may indicate a change in interpreting sensory feedback in the brain in individuals with phantom limb pain, after upper limb amputation (Flor *et al.*, 1997). Therefore, changes at any point in the central nervous system loop of afferent input, interpretation of sensory

input in the brain and efferent output, may set off a pathological cycle, which can lead to chronicity in shoulder disorders. It is hypothesized that pain, effusion and an inflammatory response, due to an acute or chronic overuse injury of the rotator cuff, may set off the pathological cycle, by changing the motor output (Shakespeare *et al.*, 1985). A change in muscular function has shown to result in scapular and humeral dysfunction causing a narrowing of the subacromial space, thus increasing the mechanical load and pathological process in the rotator cuff tendons. The rotator cuff tendons have been described to be intrinsically susceptible to developing pathologies due to an avascular zone close to the attachment of the tendon, which may compromise any healing process (Rathbun and Macnab, 1970). Local degeneration of the tendon may cause effusion or an inflammatory response as a result of a tendon tear and consequently alter the motor output. Additionally, genetic factors have also been shown to predispose subjects to tendon pathologies (Harvie *et al.*, 2004).

Several interventions have been proposed for chronic shoulder pain. Eccentric training programs have been described to be beneficial in treating pathologies of the achilles and the patellar tendon as well as the rotator cuff tendons (Silbernagel *et al.*, 2001; Jonsson *et al.*, 2005; Young *et al.*, 2005). It may be hypothesized that the eccentric training program normalizes the tendon structure and therefore interrupts the pathological cycle. However, pain has been described to be the most common symptom in subjects with a shoulder disorder and corticosteroid injections into the subacromial space are used in an attempt to reduce the pain. Although not supported by available literature, it is commonly understood that muscular dysfunction of the rotator cuff or scapular stabilizing muscles

are a result of changes in the central nervous system due to pain. Therefore it is hypothesized that a progressive rehabilitation program may normalize the efferent output (Akgun *et al.*, 2004). A reduction of the muscular dysfunctions may lead to an optimization of the afferent input allowing the brain to normalize the cortical representation of the shoulder in the somatosensory motor cortex. However, the pain neuromatrix in the brain can be activated by noxious (tissue damage) as well as non-noxious (belief system, memory, culture, genetics) stimuli (Melzack, 1999). Anticipation of lower back pain has shown to alter the muscle activation pattern of the trunk muscle, which would be associated with a non-noxious stimulus (Moseley *et al.*, 2004b).

Treatment of chronic shoulder impingement syndrome mainly focuses on reducing pain (via noxious stimuli) by addressing biomechanical aspects with rehabilitation programs correcting muscle imbalances and posture (Kibler *et al.*, 2001). However, Moseley et al (2004) have recently shown that when patients suffering from chronic lower back pain, were informed about noxious and non-noxious aspects of the neurobiology of chronic pain instead of the biomechanical factors that may be responsible for their pain, the outcome was superior. It may be suggested that this treatment approach could be applied to patients with chronic shoulder pain. However, it is recommended that a multi-faceted approach in the treatment of patients with chronic shoulder impingement syndrome be used. This approach needs to address the following parameters; firstly, biomechanical factors by normalizing muscle dysfunction, biomechanical changes and postural correction, secondly, the pathological tendon structure via an eccentric training program,

and thirdly, the belief systems of pain and fear of pain via education, in an attempt to normalize afferent input, interpretation in the brain and efferent output.

2.6 CONCLUSION

The pathomechanics underlying chronic shoulder disorders have been described, exploring different models such as the biomechanical, vascular, genetic and neural model. It is most likely that a combination of biomechanical, vascular, genetic and neural mechanisms predisposes subjects to development of shoulder disorders and sustains the pathological changes into the phase of chronicity. However, muscular dysfunctions of the scapular stabilizing muscles are commonly described in subjects with chronic shoulder disorders and are understood to result in scapular dyskinesis, thus increasing the compression of the subacroial structures against the acromion (Scovazzo *et al.*, 1991; Pink *et al.*, 1993; Ruwe *et al.*, 1994; Ludewig and Cook, 2000). Therefore, the muscular dysfunctions of the scapular stabilizing muscles need to be accurately defined for individuals with chronic shoulder disorders, so that specific rehabilitation programs can be devised. These can be used within a multi-faceted approach towards treatment, addressing biomechanical factors, the pathological tendon structure and the pain. Research that will enlighten such a specific rehabilitation program could investigate the effect of the intensity and chronicity of chronic shoulder pain on the activation of the scapular stabilizing muscles that have been described to be an integral part of the pathological cycle of shoulder disorder. A better understanding of the mechanism underlying muscle dysfunction may serve to effectively address muscle dysfunction and scapular dysfunction.

CHAPTER 3

RESEARCH PAPER

NEUROMUSCULAR ACTIVITY OF THE SCAPULAR STABILIZING MUSCLES DURING A SHOULDER ABDUCTION MOVEMENT IN SUBJECTS WITH AND WITHOUT SHOULDER IMPINGEMENT SYNDROME

3.1 ABSTRACT

3.1.1 Title

Neuromuscular activity of the scapular stabilizing muscles in subjects with and without chronic shoulder impingement syndrome.

3.1.2 Study design

Case control study

3.1.3 Objectives

To examine differences in neuromuscular activity of the scapular stabilizing muscles in subjects with and without chronic shoulder impingement syndrome during an abduction movement of the shoulder.

3.1.4 Background

Muscular dysfunctions of the scapular stabilizing muscles have been identified in subjects with chronic shoulder impingement syndrome. It is hypothesized that muscular dysfunctions of the scapular stabilizing muscles are part of a pathological cycle resulting in scapular dyskinesis, thus narrowing the subacromial space, leading to increased mechanical load on the subacromial structures and progression of the pathological process of the subacromial structures. Although it is unclear which element of the pathological cycle initiates the process, muscular dysfunction appears to be an integral part in maintaining the pathological cycle.

3.1.5 Methods and Measures

Seven subjects with chronic shoulder impingement syndrome (pain group) and fourteen subjects without chronic shoulder impingement syndrome (control group) performed three repetitions of an abduction movement of the right, injured shoulder in the frontal plane. EMG (electromyography) activity of the deltoid, which indicated the onset of shoulder movement, and the upper trapezius, lower trapezius and serratus anterior muscles, was recorded during the shoulder movements. The normalized iEMG activity was measured during time epoch A (500ms) at the beginning and time epoch B (500ms) at the end of the abduction movement and compared between groups using an independent t-test. The percentage difference between the values of time epoch A and B was calculated and compared between groups using an independent t-test. The normalized iEMG activity of the three middle seconds of the five second abduction movement was calculated for the three repetitions of abduction and compared between groups, using a one-way analysis of variance (ANOVA) with repeated measures, identifying a repetition by group interaction. Correlation matrices were calculated between the duration of pain and the total pain score during the testing procedure expressed on the numeric pain scale (NPS) and all outcome measures.

3.1.6 Results

During time epoch A, no differences were found between groups with regards to the normalized iEMG activity of the deltoid, upper trapezius, lower trapezius and serratus anterior muscles. During time epoch B significant differences were found between groups with a reduction of the normalized iEMG activity of the upper trapezius ($p = 0.036$) and

serratus anterior ($p = 0.016$) muscles in the pain group. The percentage difference between the iEMG values of time epoch A and B showed no significant differences between groups. The repetition by group interaction effect was not significant with regards to the normalized iEMG activity during the three middle seconds of the five second abduction movement. During time epoch B, iEMG activity of the upper trapezius muscle ($p = 0.040$) and the serratus anterior muscle ($p = 0.020$) was significantly correlated to the duration of pain, showing that the longer pain had been present, the lower the iEMG activity. The percentage difference between the iEMG values of time epochs A and B for the serratus anterior muscle ($p = 0.017$) was significantly correlated to the pain duration, showing that the longer pain had been present, the bigger the percentage difference between the iEMG activity during time epochs A and B. No significant correlation was found between the total pain score on the numeric pain scale (NPS) measured during the testing procedure and all the outcome measures and muscles tested.

3.1.7 Conclusion

Muscle dysfunctions of the upper trapezius and serratus anterior muscles have been identified in subjects with chronic shoulder impingement syndrome during the later part of the abduction movement. The range of glenohumeral movement between 60° - 120° of abduction corresponds with the range of the painful arc, when the subacromial space is biomechanically reduced due to the greater humeral tuberosity approaching the acromion. The muscle dysfunctions of the upper trapezius and the serratus anterior have been shown to increase the longer the pain had been present.

3.1.8 Keywords

Shoulder; chronic shoulder impingement syndrome; scapular stabilizing muscles; Electromyography (EMG)

3.2 INTRODUCTION

Shoulder pain has been described to be the third most common musculoskeletal pain after lower back pain and knee pain (Rekola *et al.*, 1993; Urwin *et al.*, 1998; Solomon *et al.*, 2001). Rotator cuff lesions, such as impingement syndrome, partial and full thickness tears have mostly been described to be responsible for shoulder pain (McLeod and Andrews, 1986; Solomon *et al.*, 2001; Wofford *et al.*, 2005). The mechanism underlying chronic shoulder impingement syndrome has been explained to be mechanical overload of the subacromial structures due to a narrowing of the subacromial space (Hawkins and Kennedy, 1980; Neer, 1983). Scapular dyskinesis in subjects with chronic shoulder impingement syndrome has been associated with a reduction of the subacromial space and subsequent mechanical overload of the subacromial structures (Hawkins and Kennedy, 1980; Ludewig and Cook, 2000; Ludewig and Cook, 2002; Michener *et al.*, 2003). Scapular dyskinesis has been related to muscle dysfunctions of the scapular stabilizing muscles. In a comparison of swimmers with and without painful shoulders an increase in the activity of the subscapularis and infraspinatus muscle and a decrease in the activity of the teres minor, supraspinatus, and the upper trapezius muscle has been demonstrated in the group of swimmers with painful shoulders during breastroke (Ruwe *et al.*, 1994). These results have been confirmed by a study comparing swimmers with and without painful shoulders during butterfly stroke (Pink *et al.*, 1993). In addition, the latter study demonstrated a decrease in the activity of the serratus anterior muscle and an increase in the activity of the posterior deltoid muscle in swimmers with shoulder pain, whereas no difference in muscle activity was found in the rhomboid, pectoralis major, latissimus dorsi and anterior and middle deltoid muscle. In contrast, comparing

swimmers with and without painful shoulders during freestyle stroke (Scovazzo *et al.*, 1991), the group with shoulder pain demonstrated decreased activity in the anterior and middle deltoid as well as subscapularis muscle, whereas a decrease in serratus anterior muscle activity corresponds with the findings of the former study. The study on freestyle stroke also found a decrease in lower trapezius activity in the group with shoulder pain. Ludewig and Cook (2000) demonstrated conflicting results comparing subjects with and without painful shoulders during elevation of the shoulder in the scapular plane with an increase in upper and lower trapezius muscle activity in the group with shoulder pain, whereas a decrease in serratus anterior activity is consistent with previous studies.

The aim of this study was to compare neuromuscular activity of the deltoid muscle, which was chosen to indicate the onset of shoulder movement, and the scapular stabilizing muscles such as the upper trapezius, lower trapezius and serratus anterior muscles during an ABD (abduction) movement of the shoulder in the frontal plane in subjects with pain due to chronic shoulder impingement syndrome and healthy control subjects.

3.3 METHODOLOGY

3.3.1 Experimental design

The study design is that of a case control study, investigating the effect of chronic shoulder pain on neuromuscular activation patterns of the scapular stabilizing muscles during ABD of the right shoulder in the frontal plane. All subjects were required to visit the laboratory on one occasion, where they completed an informed consent form

(Appendix 1), the "Edinburgh Handedness Questionnaire" (Appendix 2) and a physical questionnaire examining the history of their shoulder pain and the functional ability of their shoulder during activities of daily living and sporting activities (Appendix 3). All subjects underwent a physical examination of the shoulder and neck by a physiotherapist, assessing the presence of pain (Appendix 4). The Ethics and Research Committee of the University of Cape Town approved the study.

3.3.2 Population

Twenty-one right-handed male subjects between the ages of 27 – 50 years were recruited for the study. Only male subjects were recruited because data was collected to investigate electromyography (EMG) as well as electroencephalography (EEG) recordings and gender differences have been described in EEG patterns (Wada *et al.*, 1994). For the purpose of this study only the EMG data was analyzed. Fourteen healthy control subjects and 7 pain subjects with shoulder impingement syndrome were recruited. The subjects were matched for the anthropometrical data (age, hight, mass, skinfolds) as well as for activity level.

3.3.2.1 Pain group

The 7 subjects in the pain group were recruited from local sport clubs and by referral from local physicians and physiotherapists.

Inclusion criteria for the pain group

Symptoms of shoulder impingement syndrome were determined by history and confirmed by a physical examination to detect signs of chronic shoulder impingement syndrome (Appendices 3 and 4). The subjects must have experienced pain for at least 3 weeks. The level of pain had to be of such a grade that it affected the subject's activities of daily living and sporting activity or prevented the subject from participating in sport altogether (Grade 3 or 4 injury) (Neer, 1983). Subjects were included in the pain group if they presented with at least three out of the following four criteria during the physical examination: 1) positive Neer's sign, reproduction of pain when the examiner passively flexes the humerus to end range with overpressure (Neer, 1983); 2) positive Hawkins sign, reproduction of pain when the shoulder is passively placed in 90° of forward flexion and internally rotated to end range (Hawkins and Kennedy, 1980); 3) positive Jobe's sign, reproduction of pain and lack of force production with an isometric contraction in the direction of elevation in the scapular plane in internal rotation of the shoulder (empty can) (Jobe and Jobe, 1983); 4) a painful arc of movement during ABD (60°-120°) (Kibler *et al.*, 2001) (Appendix 4).

Exclusion criteria for the pain group

Subjects were excluded from the study if they had: 1) a current or past traumatic injury of the shoulder, 2) a history of shoulder surgery, 3) bilateral shoulder pain, 4) current pain in any other part of the body. Any dysfunction of the neck was excluded by an examination

of the neck as outlined by (Brukner and Kahn, 2001). Active range of motion of the neck was tested with the subject in sitting. The subject then moved his head actively into flexion, extension, lateral flexion to the right and left and rotation to the right and left. The occurrence of pain was noted. These movements were repeated with overpressure at the end of active range of motion. The cervical spine was regarded as clear when there was full range of motion with overpressure without pain during the testing procedure. The subjects may have undergone previous physiotherapy treatment for shoulder pain that failed in the past. The subjects should not have received a corticosteroid injection or an injection with a local anesthetic into the shoulder joint at least 5 weeks prior to testing. The subjects should not have taken pain-relieving medication within 48 hours of testing.

3.3.2.2 Control group

The control group consisted of 14 healthy subjects without any pathology of either the shoulders or neck regions. They were recruited from the local university and sport clubs. The control subjects filled in the same questionnaires and underwent the same physical examination as the pain subjects.

Inclusion criteria for the control group

Inclusion criteria for the control group were: 1) Males between 27 and 50 years of age, 2) right dominant, 3) no current or previous acute or chronic shoulder injury and 4) no more than 2 hours overhead activity per week.

Exclusion criteria for the control group

Exclusion criteria for the control group included: 1) a history of shoulder pain, 2) a history of traumatic shoulder injury and 3) a history of shoulder surgery. Any dysfunction

of the shoulder and neck were excluded by the physical examination of the shoulder and neck as described for the pain group.

3.3.3 Procedure

3.3.3.1 Subject characteristics

Anthropometrical data was recorded for all subjects. Age (yrs), height (m), mass (kg) and skinfolds (mm) (biceps, triceps, subscapular, abdominal, suprailiac, thigh, calf) were recorded.

3.3.3.2 Electromyography instrumentation

Electromyography (EMG) data was collected with surface electrodes from the muscle bellies of the middle deltoid , upper trapezius , lower trapezius and serratus anterior during ABD of the shoulder (McQuade *et al.*, 1998; Ludewig and Cook, 2000; Cools *et al.*, 2002). Two electrodes were placed on the belly of each muscle with an inter-electrode distance of 10 mm and carefully taped. Before application of the surface electrodes, the subject's skin was shaven, abraded, and wiped with alcohol. For the upper trapezius, EMG electrodes were placed midway between the spinous process of the seventh cervical vertebrae and the posterior tip of the acromion process along the line of the trapezius (McQuade *et al.*, 1998). For the lower trapezius, EMG electrodes were placed obliquely upward and laterally along a line between the intersection of the spine of the scapula with the vertebral border of the scapula and the seventh thoracic spinous process (McQuade *et al.*, 1998). The electrode for the serratus anterior was placed at the lateral side of the thorax 3 cm caudal to the inferior spine of the scapula obliquely

upwards and posteriorly, following a rib (McQuade *et al.*, 1998). For the middle deltoid, electrodes were placed midway between the deltoid tuberosity and the acromion process (McQuade *et al.*, 1998). A reference electrode was placed over the clavicle (McQuade *et al.*, 1998). The EMG activity of the muscles was recorded using the telemetric EMG system (Telemyo 900, Noraxon, USA, Inc., Arizona, USA). The EMG signal was transmitted from the electrodes to a transmitter box via separate channels. A telemetric signal was relayed to an antenna connected to an online computer (MyoResearch 2.11). The MyoResearch (2.11) software was used to clean and analyze the recorded data.

3.3.3.3 Testing procedure

Both groups underwent the same testing procedure. The subjects were seated with their back against a wall on which a contraption was mounted that allowed individual adjustment to ensure 50° and 110° of right sided shoulder ABD. A 1.1 kg weight was applied to the subject's right wrist in order to elicit more muscle activity, which allowed a clearer EMG reading (Figure 1).

Shoulder ABD to 110°

EMG data was recorded while the subjects performed a standardized ABD movement of the shoulder with external rotation (thumb up) to 110°. An audible beep signaled the start of the recording time as well as the start of the ABD movement of 5-second duration to 110°. At 110° of ABD the subjects paused for 1 second before lowering the arm during 5 seconds returning to the starting position. The subjects were instructed to maintain an upright posture during the testing procedure. To control the speed of the movement, the investigator gave the following command: "go-one-two-three-four-five-hold-go-one-two-

three-four-five-stop". The subjects carried out 2 repetitions of shoulder ABD for familiarization, followed by 3 repetitions during recording. The ABD movement from neutral to 110° ABD was used for data analysis in order to evaluate changes in the EMG activity between pain and control subjects when moving the shoulder into the painful arc above 60° of ABD. The painful arc coincides with the range of movement during glenohumeral elevation when the subacromial space is compromised due to the greater humeral tuberosity approaching the acromion (60° - 120°) (Nordt *et al.*, 1999). All pain subjects experienced pain while moving above 60° of ABD, whereas all the control subjects were pain free (Table 1).

Isometric contraction at 50°

EMG data was recorded during a 5-second period while the subjects performed 3 repetitions of isometric contractions while holding the arm at 50° of ABD against the force of gravity. A one-minute break was allowed between repetitions. The EMG data recorded during the 5-second isometric contractions was used for normalization purpose of the EMG data, which was collected during the ABD movement. The EMG normalization process negates the potentially confounding effects of differences in lean muscle mass and percentage body fat between subjects and the individual variation introduced as a result of electrode placement (Lehman and McGill, 1999). For the normalization procedure 50° of ABD was chosen as it is outside the painful arc. None of the pain subjects experienced pain during the isometric contraction, which allowed normalization of the EMG activity of control and pain subjects without the interference of pain.

3.3.3.4 Pain evaluation

The pain subjects were asked to report their shoulder pain on a numeric pain scale (NPS) before and after every repetition during the testing procedure (Appendix 4), (Table 1). The PNS has proven to be a reliable method of assessing pain (Olagun *et al.*, 2003).

3.3.4 Evaluation of data

3.3.4.1 Subject characteristics

The sum of seven skinfolds (mm) was calculated by summing the values of the biceps, triceps, subscapular, abdominal, suprailiac, thigh and calf skinfolds. Percentage body fat (% BF) was determined according to the methods of Durnin and Womersley. Body mass index (BMI) was calculated by dividing the mass (kg) of the subject by the square of his height (m) (Durnin and Womersley, 1974).

3.3.4.2 iEMG activity during time epochs A and B

The raw EMG data was filtered using a 50Hz notch filter followed by a 15-500 Hz band pass filter and sampled at 2000Hz. This allowed noise and movement interference to be cut out below 15 Hz and above 500 Hz. The data was smoothed and iEMG (μv) was calculated by the root mean square (RMS) with MyoResearch (2.11) for two time epochs of 500ms for each muscle for the three repetitions of ABD. The start of epoch A was selected for each muscle coinciding with the visually determined onset of deltoid contraction (Hodges and Bui, 1996). Epoch B entailed the last 500ms of the ABD movement of 5- second duration (Figure 2). The data of time epochs A and B was normalized to the equivalent time epochs of

the submaximal isometric contraction at 50° ABD. The average of the normalized value of the 3 repetitions of ABD was calculated for each muscle for time epochs A and B.

3.3.4.3 Percentage difference of the normalized iEMG activity of time epochs A and B

The percentage difference of the iEMG activity of the time epoch A and B was calculated for each muscle group (calculated in excel: (epoch B – epoch A) / epoch A x 100). The percentage difference indicated the change in activity from the start of the movement as a proportion of this initial value.

3.3.4.4 iEMG activity of the 3 middle seconds of the 5-second abduction movement

The middle 3 seconds of the EMG recording of the deltoid, upper trapezius, lower trapezius and serratus anterior of 3 repetitions of ABD movement of 5-second duration were selected for processing. The data collected during the ABD movement and was normalized to the 3 middle seconds of a 5- second isometric contraction at 50° of ABD using excel (iEMG (μv) during 3 seconds ABD / iEMG (μv) during 3 seconds isometric contraction at 50° ABD x 100 = percentage of submaximal iEMG activity). The average iEMG value of the 3 repetitions of ABD was calculated for each muscle.

3.3.5 Statistical analysis

3.3.5.1 Subject characteristics

The independent t-test (Statistica 7 software package StatSoft Inc, Tulsa, OK, USA) was used to identify differences between the control and pain group with regards to age,

height, weight, sum of skinfolds, percentage body fat (% BF) and body mass index (BMI).

3.3.5.2 iEMG activity during time epochs A and B

The independent t-test (Statistica 7 software package StatSoft Inc, Tulsa, OK, USA) was used to evaluate differences between the control and pain groups with regards to the normalized iEMG activity of time epochs A and B respectively. When the p-variance was below 0.05, the non-parametric statistical analysis (Mann-Whitney) was performed. Statistical significance was accepted when $p < 0.05$.

3.3.5.3 Percentage difference of the normalized iEMG activity of time epochs A and B

The average value of the three repetitions of ABD was used for the statistical analysis of the percentage difference between the normalized iEMG value of time epochs A and B. The independent t-test (Statistica 7 software package StatSoft Inc, Tulsa, OK, USA) was used to determine any significant differences between pain and control subjects for descriptive characteristics. When the p-variance was below 0.05, the non-parametric statistical analysis (Mann-Whitney) was performed. Statistical significance was accepted when $p < 0.05$.

3.3.5.4 iEMG activity of the 3 middle seconds of the 5-second abduction movement

A Levine's test followed by an analysis of variance (ANOVA) with repeated measures were used (Statistica 7 software package StatSoft Inc, Tulsa, OK, USA) to determine a group effect and a repetition effect of the 3 repetitions of ABD movement as well as the

interaction effect between the groups and the repetitions when assessing the normalized iEMG activity of the 3-second period during ABD. A significant interaction effect between the groups and the 3 repetitions of ABD movement was accepted when $p < 0.05$.

3.3.5.5 Correlations

Correlation matrices were calculated (StatSoft Inc, Tulsa, OK, USA) for the pain group between the duration of pain in weeks as well as the total pain score on the numeric pain scale (NPS) during the 3 repetitions of ABD, and the normalized iEMG activity during time epochs A and B, the percentage difference between the normalized iEMG activity of time epochs A and B and the normalized iEMG activity of the 3 middle seconds of the 5-second ABD movement. R-values and p values were calculated for each muscle and outcome measure. Statistical significance was accepted when $p < 0.05$.

3.4 RESULTS

3.4.1 Subject Characteristics

No significant differences were shown between groups for the following factors: age, height, weight, percentage body fat (%BF) and body mass index (BMI). The sum of skinfolds showed a significant difference between the control and pain group ($p = 0.032$) (Table 2).

3.4.2 Analysis of iEMG activity

During time epoch B at the end of the ABD movement a significant difference was found for the upper trapezius with a decrease of iEMG activity of 36 % ($p = 0.036$) in the

pain group compared to the control group. A similar finding for the serratus anterior muscle occurred with a decrease of iEMG activity of 40 % ($p = 0.016$) in the pain group compared to the control group. No significant differences were found between the control and pain group during time epoch B for the lower trapezius and deltoid muscles (Figures 3 and 4).

No significant differences were found for the deltoid, upper trapezius, lower trapezius and serratus anterior muscles between the control and pain groups with regards to the normalized iEMG activity during time epoch A and the percentage difference of the normalized iEMG activity of time epochs A and B. With regards to the iEMG value during the three middle seconds of the ABD movement, the group by repetition interaction effect was not significant and no group effect and repetition effect was demonstrated for all the tested muscles.

3.4.3 Correlations

A significant relationship was found between the duration of pain and the normalized iEMG activity during time epoch A with regards to the serratus anterior muscle activity ($p = 0.007$) and time epoch B with regards to upper trapezius muscle activity ($p = 0.040$) and serratus anterior muscle activity ($p = 0.020$) (Figures 6 and 7). A significant correlation was also demonstrated between the duration of pain and the percentage difference of the normalized iEMG activity of time epochs A and B with regards to the serratus anterior muscle activity ($p = 0.017$) (Figure 8). No relationship was found

between the duration of pain and the normalized iEMG activity during the 3 middle seconds of the 5-second ABD movement (Figure 9).

The total pain score on the numeric pain scale (NPS) during the 3 repetitions of ABD and the normalized iEMG activity during time epoch A, time epoch B, the percentage difference between the normalized iEMG activity of time epochs A and B as well as the normalized iEMG activity during the 3 middle seconds of the 5-second ABD movement, demonstrated no significant relationships (Figures 10 - 13).

3.5 DISCUSSION

3.5.1 Subject characteristics

The sum of skinfolds showed a significant difference between the control and pain groups ($p = 0.032$). However, the percentage body fat (% BF) and body mass index (BMI) showed no differences between the groups. As the measurement of the sum of skinfolds affects the quantity of recorded EMG activity, the difference between the groups may be a confounding factor (Lehman and McGill, 1999). One subject in the control group had a very low sum of skinfolds (38.2mm), which would explain the large standard deviation for the control group. However, the role of normalization is to control for differences in skin fold thickness.

3.5.2 iEMG activity during time epochs A and B

Time epoch A

This study showed no significant difference between the pain and control groups when comparing the normalized iEMG activity of the deltoid, upper trapezius, lower trapezius and serratus anterior muscles during time epoch A with a 1.1 kg added wrist weight. These results may correspond with the findings of Ludewig and Cook (2000) that investigated the iEMG activity of the upper trapezius, lower trapezius and serratus anterior during an ABD movement in the scapular plane with no load and added loads of 2.3 kg and 4.6 kg between 31° - 60°. Time epoch A (500ms after the onset of deltoid activity) may partially overlap with the initial part of the range of movement between 31°-60°. It may be hypothesized that the absence of a significant difference between the pain and control groups with regards to the iEMG activity in this study may be due to the fact that none of the subjects experienced pain during time epoch A. Most likely the subjects did not experience pain because time epoch A corresponds with a range of movement below 60° of ABD, when there is only minimal stress on the subacromial structures (Nordt *et al.*, 1999). Therefore, the effect of pain on muscle activity would be minimal (Shakespeare *et al.*, 1985).

Time epoch B

The time epoch B is defined as the last 500ms of a 5-second ABD movement of the shoulder in the frontal plane. At the end of the ABD movement the glenohumeral joint reaches 110° of ABD. Therefore, time epoch B corresponds with the range of movement of 60° - 120° during which the subacromial space is compromised due to the greater

tuberosity of the humerus approaching the acromion (Nordt *et al.*, 1999). Hence the subacromial structures are likely to be compressed and subjects with pathology of the subacromial structures would experience pain at 60° - 120° of ABD (painful arc). During time epoch B a significant difference between the pain and control group with regards to the normalized iEMG activity has been demonstrated for the upper trapezius ($p = 0.036$). The pain group showed a 36 percent decrease in upper trapezius activity compared to the control group. The function of the upper and middle trapezius has been described to draw the scapula and clavicle backwards by rotating the clavicle at the sternoclavicular joint, thus elevating the acromion in the later phases of humeral elevation, preventing compression of the subacromial structures. The fibers of the upper and middle trapezius do not change length much during rotation of the scapula and therefore it is suggested that their main function is to stabilize rather than move the scapula (Johnson G *et al.*, 1994). A decrease in upper trapezius activity may therefore result in a decrease in scapular stability and a decrease in elevation of the acromion, resulting in a decrease in subacromial space during humeral elevation and compression of the subacromial structures. Therefore, the pathology of the subacromial structures would be exacerbated. However, the results of this study are in contradiction with the findings of Ludewig and Cook, which demonstrate an increase in upper trapezius activity in the pain group by 11 percent between 91° - 120° of ABD in the scapular plane with a load of 4.6 kg (Ludewig and Cook, 2000). The inconsistencies between the study of Ludewig and Cook and this study are likely to be explained by the difference in weight that is lifted and the difference in normalization method.

In this study, a significant difference was also demonstrated in the normalized iEMG activity of the serratus anterior muscle ($p = 0.016$) with 40 percent less muscle activity in the pain group compared to the control group. The reduction in serratus anterior muscle activity in subjects with shoulder impingement syndrome is in accordance with literature (Scovazzo *et al.*, 1991; Ruwe *et al.*, 1994; Ludewig and Cook, 2000). The primary role of the serratus anterior has been described to compress the scapula against the ribcage as well as providing upward rotation and posterior tipping of the scapula during glenohumeral elevation in order to avoid compression of the subacromial structures (Scovazzo *et al.*, 1991; Paine and Voight, 1993; Ruwe *et al.*, 1994; Magarey and Jones, 2003). Dysfunction of the serratus anterior has been described to result in a decrease in upward rotation and posterior tipping of the scapula, which has been associated with a reduction of the subacromial space. Subsequent mechanical compression of the subacromial structures may reinforce the pathological process (Scovazzo *et al.*, 1991; Ruwe *et al.*, 1994; Ludewig and Cook, 2000; Kibler and McMullen, 2003).

3.5.3 Percentage difference of the normalized iEMG activity of time epochs A and B

The percentage difference of the normalized iEMG activity of time epoch A at the beginning and time epoch B at the end of the ABD movement indicates a dynamic change of neuromuscular activity during the ABD movement. No statistical difference has been demonstrated between the control and pain groups, most likely due to a small sample size.

3.5.4 iEMG activity of the 3 middle seconds of the 5-second abduction movement

The hypothesis for the investigation of the repetition X group interaction was that the pain free group may show more variability in EMG activity between repetitions and thus overuse of the muscles may be prevented. This hypothesis has been refuted as no significant difference was found between groups, repetitions and repetition X group interaction. The three middle seconds are problematic for interpretation because of the dramatic change in EMG activity over this period and the meaning of a single value may be questionable.

3.5.5 Correlations

3.5.4.1 Duration of pain

Time epoch A

During the testing procedure the pain subjects did not experience pain at 50° of ABD. Time epoch A at the beginning of the ABD movement is situated below 50° of ABD and therefore pain during the testing procedure can be excluded as a contributing factor for changes in the EMG activity. This hypothesis has been confirmed with the finding that demonstrated no significant differences between control and pain group with regards to the normalized iEMG activity during time epoch A. However, although the pain subjects did not experience pain during time epoch A, the normalized iEMG activity with regards to serratus anterior muscle activity has shown to be affected by the duration of shoulder pain as reported by the subjects. With an increase of the duration of pain, a reduction of the normalized iEMG activity can be demonstrated for the serratus anterior muscle.

However, the duration of pain the subjects reported is clustered between 3 and 24 weeks, whereas only one subject reported the duration of pain to be 96 weeks. Although statistically significant, due to the uneven spread of the data with regards to duration of pain, a reduction of serratus anterior iEMG activity with increased chronicity of shoulder pain during a pain free movement must be interpreted with caution. A more even spread of data would strengthen the finding of this study. However, it may be hypothesized that chronicity of shoulder pain may be responsible for a reduction of serratus anterior muscle iEMG activity, resulting in scapular dyskinesis during a pain free range of shoulder ABD. Scapular dyskinesis due to muscle dysfunction of the serratus anterior muscle may therefore already be set up before the critical range of movement of 60° - 120 ° of ABD is reached, when the subacromial space is biomechanically reduced due to the greater humeral tuberosity approaching the acromion (Nordt *et al.*, 1999). The possible scapular dyskinesis at the beginning of the ABD movement may therefore contribute to further reduction of the subacromial space between 60° - 120 ° ABD, thus increasing the mechanical compression of the subacromial structures resulting in pain (painful arc).

Time epoch B

Time epoch B is situated at the end of the ABD movement, covering the range of glenohumeral movement when the subacromial space is biomechanically reduced due to the greater humeral tuberosity approaching the acromion (60° - 120°) (Nordt *et al.*, 1999). Due to possible mechanical compression of the subacromial structures during this critical range of movement, subjects with chronic shoulder impingement syndrome frequently report a painful arc between 60° - 120° of shoulder elevation (Neer, 1983). The pain subjects in this study reported pain during this latter part of the ABD movement. In

addition, during time epoch B, a reduction of serratus anterior muscle and upper trapezius muscle iEMG activity has been demonstrated in the pain group compared to the control group. It may therefore be concluded that pain during the latter part of the ABD movement is most likely responsible for the reduction of serratus anterior and upper trapezius muscle activity in form of pain inhibition (Shakespeare *et al.*, 1985). Additionally, there is a significant relationship between the duration of pain reported by the subjects and the reduction of serratus anterior and upper trapezius muscle iEMG activity. Although the data representing the duration of pain is not evenly spread between 3 and 96 weeks, it may be concluded that during the range of glenohumeral movement, when the subacromial space is biomechanically reduced (60° - 120°), pain during the movement as well as the chronicity of shoulder pain, may contribute to a reduction of serratus anterior and upper trapezius muscle iEMG activity. A reduction of serratus anterior and upper trapezius muscle activity has been associated with scapular dysfunction, thus further progressing the pathological cycle (Ludewig and Cook, 2000).

Percentage difference of the normalized iEMG activity of time epochs A and B

The percentage difference of the normalized iEMG activity between time epochs A and B of the serratus anterior muscle demonstrated a significant relationship with the duration of shoulder pain. Due to the uneven spread of the data with regards to duration of pain, the interpretation of the findings must be done with caution. However, it may be hypothesized that chronicity of shoulder pain results in an increase of the percentage difference of the normalized iEMG activity between time epochs A and B with regards to serratus anterior muscle activity. During time epoch B, a decrease in normalized iEMG activity has been demonstrated for the serratus anterior muscle. Therefore, an increase of

the percentage difference from the time epoch B perspective may be due to an increase in iEMG activity during time epoch B, which would reflect a normalization of the muscle dysfunction of the serratus anterior muscle with increased chronicity of shoulder pain. However, the results of this study indicate a tendency of a reduction of normalized iEMG activity during time epoch B with increased chronicity. Nevertheless, an increase of the percentage difference of the normalized iEMG activity between time epochs A and B of the serratus anterior muscle may also be explained by a reduction of iEMG muscle activity during time epoch A with increased chronicity of shoulder pain. Supported by the results of this study demonstrating a reduction of iEMG activity of the serratus anterior muscle with increased chronicity of shoulder pain during time epoch A, it may be hypothesized that chronicity of shoulder pain may lead to an increase in serratus anterior muscle dysfunction in form of a reduction of iEMG activity during the pain free range of movement. Hence it may be concluded that with increased chronicity of shoulder pain, muscle dysfunction of the serratus anterior muscle during a pain free range of movement may lead to scapular dyskinesis even before reaching the painful arc, thus increasing the progression of the pathology of the subacromial structures.

3.5.4.2 Total pain score on the numeric pain scale

The total pain score on the numeric pain scale (NPS) during the 3 repetitions of ABD and the normalized iEMG activity during time epoch A, time epoch B, the percentage difference of the normalized iEMG activity of time epochs A and B as well as the normalized iEMG activity during the middle 3 seconds of the 5-second ABD movement,

demonstrated no significant relationships (Figures 10 - 13). Therefore it may be concluded that the amount of muscle dysfunction due to pain inhibition may not be related to the amount of pain, as reported on the numeric pain scale (NPS), experienced during the movement. However, a tendency has been demonstrated that the amount of muscle dysfunction may be related to the chronicity of shoulder pain.

3.5.6 Normalization process

The EMG normalization process, as opposed to working with raw EMG data, negates the potentially confounding effects of differences in lean muscle mass and percentage body fat between subjects and the individual variation introduced as a result of electrode placement (Lehman and McGill, 1999). Normalization using a maximal voluntary contraction in pain subjects has received criticism in the literature due to the changes of neuromuscular output associated with pain (Marras and Davis, 2001). It is therefore suggested that in the presence of pain, a muscle contraction would be submaximal in nature rather than maximal. It is hypothesized that in subjects with chronic shoulder impingement syndrome the central nervous system suppresses the EMG activity of the scapular stabilizing muscles even in a pain free range of motion. Any contraction of the affected muscles would therefore always be submaximal in nature. In view of that, the EMG data of this study was normalized to a standardized pain free submaximal isometric contraction at 50° of shoulder ABD with an added wrist weight of 1.1kg. Fifty degrees of ABD were chosen for normalization purpose as it is below the range of glenohumeral movement when the subacromial space is biomechanically reduced due to the greater humeral tuberosity approaching the acromion (Nordt *et al.*, 1999). Therefore the risk of a painful impingement of the subacromial structures is reduced. However, it is not possible

to exclude that the subjects with shoulder pain had abnormal activity of the tested muscles even in the pain free range of 50° ABD. As it is not possible to determine whether activity of the tested muscles was increased or decreased in this position the data can not be interpreted with full confidence.

3.5.7 Limitations of the study

Only right handed, right injured male subjects were recruited for the study in order to avoid confounding factors that would affect the EEG (electroencephalogram) data that was collected together with the EMG data. The purpose of the EMG aspect of this study was to investigate changes in muscle activity due to pain inhibition. Therefore, the strict homogeneity of the studied population reduces the risk of confounding factors such as dominance and side of injury as well as gender related factors concerning the visco-elastic properties of tendon structures, which may be a possible risk factor for tendon injuries (Kubo *et al.*, 2003). However, the strict homogeneity of the studied population restricts extrapolation of the EMG results on female subjects and subjects with different combinations of dominance and side of injury. An additional limitation of the study is the small sample size, particularly in the pain group (control n = 14, pain n = 7), which poses a major restrictive factor for the investigation of neuromuscular changes due to a shoulder disorder. A larger sample size of the control and pain group may highlight differences in muscle activity between control and pain subjects during a pain free range of movement and not just during the painful range of movement. The significant relationship between the duration of pain and the reduction of serratus anterior muscle iEMG activity, which has been demonstrated in this study, could be strengthened with a

bigger sample size of the pain group and a more evenly spread duration of pain reported by the pain subjects. A strong correlation between chronicity of shoulder pain and serratus anterior muscle activity would have great clinical implications in terms of the necessity of treating acute shoulder pain effectively in order to minimize muscular dysfunction.

3.5.8 Future research

Muscular dysfunction of the scapular stabilizing muscles as well as scapular dyskinesis have been described in subjects with chronic shoulder disorders. It is generally understood that muscle dysfunctions of the scapular stabilizing muscles are due to pain-inhibition, resulting in scapular dyskinesis (Warner *et al.*, 1992), (Ludewig and Cook, 2000). However, scapular dyskinesis has also shown to be present in pain free subjects (Warner *et al.*, 1992). A prospective study investigating the incidence of pain free subjects with scapular dyskinesis developing shoulder disorders would be of clinical significance in terms of evaluating to what degree scapular dyskinesis is a risk factor for developing a painful shoulder disorder.

The mechanism of muscle inhibition due to pain is not well understood. The results of this study showed a correlation between chronicity of shoulder pain and dysfunction of the serratus anterior muscle activity during a painful as well as non-painful range of shoulder ABD movement. However, it is unclear whether it is the continuous presence of pain with chronicity that maintains the muscle dysfunction or whether once the muscle dysfunction is triggered the central nervous system adapts continuing to activate the

muscles in a dysfunctional way even though the pain has resolved. A study investigating the muscle activity of the scapular stabilizing muscles in pain free subjects, after induction of pain with a saline injection into the subacromial space and again after the pain has resolved would help to understand the mechanism underlying muscle inhibition due to pain. Pain intensity as expressed on the visual analogue scale (VAS) has not shown a significant correlation with the amount of EMG activity for the deltoid, upper trapezius, lower trapezius and serratus anterior muscle during an ABD movement of the shoulder in this study. However, it would be of interest to observe the relationship between the intensity of pain on the visual analogue scale and the muscle dysfunction in a study as described above with induced shoulder pain and over time when shoulder pain is resolving.

Studies describing rehabilitation strategies for the scapular stabilizing muscles have been described in literature (Kibler *et al.*, 2001), (Wilk *et al.*, 2002). In accordance with literature, the results of this study emphasis the importance of the rehabilitation of the serratus anterior muscle (Paine and Voight, 1993). However the function of the upper trapezius that stabilizes the scapula during glenohumeral elevation may be underestimated in clinical practice (Johnson G *et al.*, 1994). The results of this study showed a decrease of upper trapezius EMG activity in pain subjects. Nevertheless, in clinical practice, the upper trapezius muscle is frequently regarded as being overactive due to postural dysfunctions such as prolonged elevation of the shoulder girdle (Mork and Westgaard, 2005). However, the amount of upper trapezius EMG activity recorded either during a functional movement or at rest, does not elucidate information about

upper trapezius muscle strength. Therefore, the investigation of strength of the upper trapezius as well as the effect of rehabilitation strategies for the upper trapezius may help to improve the clinical understanding of dysfunctions of the upper trapezius due to impingement syndrome.

3.6 CONCLUSION

The results of this study demonstrate a reduction of iEMG activity of the upper trapezius and serratus anterior muscles in the pain group compared to the control group, during time epoch B, which corresponds with the later, painful phase of the ABD movement of the shoulder between 60° - 120°. The range of movement between 60° - 120° of shoulder elevation corresponds with the range, during which the greater humeral tuberosity approaches the acromion, thus narrowing the subacromial space (Nordt *et al.*, 1999). Subjects with pathology of the subacromial structures commonly experience pain between 60° - 120° of ABD (painful arc) (Neer, 1983). Therefore, muscle dysfunction of the upper trapezius and serratus anterior muscles as demonstrated in this study, are specific to the pain range. Further, during time epoch B, a significant correlation was found between the duration of pain and muscle dysfunction of the upper trapezius as well as serratus anterior muscle in the form of a reduction of iEMG activity in the pain group compared to the control group. Therefore, the results of this study demonstrate that the longer pain has been present, the more pronounced the muscle dysfunction of the upper trapezius and serratus anterior muscles becomes during the painful range of shoulder movement. However, the results of this study also demonstrate a significant correlation between the duration of pain and muscle dysfunction in the form of a decrease in iEMG

activity of the serratus anterior muscle in the pain group compared to the control group, during time epoch A, which corresponds with the initial, pain free part of the ABD movement. Consequently, increased duration of shoulder pain also results in an increase of muscle dysfunction of the serratus anterior muscle during the pain free range of shoulder movement.

It therefore is suggested, that the muscular dysfunction of the upper trapezius and serratus anterior muscles needs to be accurately defined for individuals with chronic shoulder pain in the painful as well as pain free range of movement, so that specific rehabilitation programs can be devised.

CHAPTER 4

SUMMARY AND CONCLUSION

Muscle dysfunction of the scapular stabilizing muscles due to pain inhibition have been described in literature (Scovazzo *et al.*, 1991; Pink *et al.*, 1993; Ruwe *et al.*, 1994; Ludewig and Cook, 2000). The results of this study are in support of literature, demonstrating a change in neuromuscular function in subjects with chronic shoulder impingement syndrome with regards to the upper trapezius and the serratus anterior muscle activity during an ABD movement of the shoulder in the frontal plane when the range of movement reaches the painful arc (60° - 120°) (Nordt *et al.*, 1999). The Upper trapezius and serratus anterior muscles both demonstrated a decrease of normalized iEMG activity in the pain subjects compared to the control subjects. A decrease in serratus anterior muscle activity has been described to result in scapular dysfunction and it is suggested that this may lead to a decrease of the subacromial space and increase the mechanical load on the subacromial structures (Kibler, 1998). Rehabilitation programs have been suggested to improve the function of the serratus anterior muscle, thus minimizing the scapular dysfunction and compression of the subacromial structures. The results of the upper trapezius muscle activity demonstrating a decrease in EMG activity in pain subjects is in conflict with a similar study by Ludewig and Cook (2000). A difference in the testing protocol may be responsible for the conflicting results. It is suggested that muscle dysfunction of the upper trapezius due to shoulder pain may be activity related. However, the function of the upper trapezius has been described to be

concerned with stabilizing the scapula during glenohumeral elevation, acting as a force couple together with the lower trapezius and serratus anterior muscles (Johnson *et al.*, 1994; Kibler, 1998). Therefore, a rehabilitation program normalizing muscular activity of the upper trapezius as part of the force couple acting on the scapula is suggested, in order to improve scapular stability. The mechanism underlying muscle dysfunction due to pain inhibition is not well understood. However, the results of this study demonstrate a correlation between chronicity of shoulder pain and muscle dysfunction of the upper trapezius and serratus anterior muscles, showing that the longer the pain had been present, the lower the iEMG activity of the upper trapezius and serratus anterior muscles.

Based on the results of this study, it is therefore suggested that a rehabilitation program for the upper trapezius and serratus anterior muscles is an important component of a multi-faceted treatment approach of individuals with chronic shoulder pain in order to normalize the scapular dysfunction, thus minimizing the compression of the subacromial structures against the acromion. Further more, the literature suggests that the treatment should address postural dysfunctions (Lewis *et al.*, 2005a) and the pathological tendon structure via an eccentric training program (Jonsson *et al.*, 2005). A reduction in pain has been shown in patients with chronic lower back pain when they were given education about the neurobiology of chronic pain and when the belief system and fear of pain were addressed in an attempt to normalize the interpretation of the pain in the brain, thus affecting the efferent output (Moseley *et al.*, 2004a). It is suggested that a similar treatment strategy may be of value in patients with chronic shoulder pain.

CHAPTER 5

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CHAPTER 6

TABLES

Table 1. Pain duration and pain experienced during the testing procedure for the pain subjects

Pain subjects	**Duration of pain (weeks)**	**Sum of NPS**
Subject 1	8	5
Subject 2	36	13
Subject 3	3	18
Subject 4	3	21
Subject 5	24	9
Subject 6	8	12
Subject 7	96	18
Mean and standard deviation	**25 ± 33**	**12 ± 7**

Note: The control subjects did not experience pain during the testing procedure.
Abbreviations: NPS = Numeric Pain Scale (amount of pain on a scale between 0 {no pain} and 10 {maximum pain}).
The sum of the NPS refers to the added up amount of pain on the NPS that the subjects reported during the three repetitions of shoulder ABD movement.

Table 2. Subject characteristics for the Control and Pain groups

Variables	Control (n = 14)	Pain (n = 7)	p - value
Age (years)	36.5 ± 6.8	36.4 ± 7.6	0.983
Height (m)	1.82 ± 8.55	1.80 ± 9.62	0.676
Weight (kg)	80.7 ± 11.9	83.3 ± 6.4	0.601
Sum of Skinfolds (mm)	84.9 ± 35.7	120.9 ± 28.0	0.032*
% BF	19.8 ± 4.6	22.2 ± 4.6	0.268
BMI ($kg \cdot m^{-2}$)	24.4 ± 2.9	25.9 ± 3.4	0.300

Data are expressed as the mean ± standard deviation
* The sum of skinfolds showed a significant difference between groups
Abbreviations: % BF = Percentage body fat, BMI = Body mass index

Table 3. Percentage difference in iEMG (μv) activity between time epochs A and B for the Deltoid, Upper trapezius, Lower trapezius and Serratus anterior muscles

Muscles	Control	Pain	p - value
Deltoid	610 ± 334	806 ± 706	0.881
Upper trapezius	531 ± 252	253 ± 362	0.068
Lower trapezius	186 ± 160	219 ± 145	0.656
Serratus anterior	720 ± 778	352 ± 228	0.081

Data are expressed as the mean ± standard deviation
Abbreviation: iEMG = integrated EMG

CHAPTER 7

FIGURES

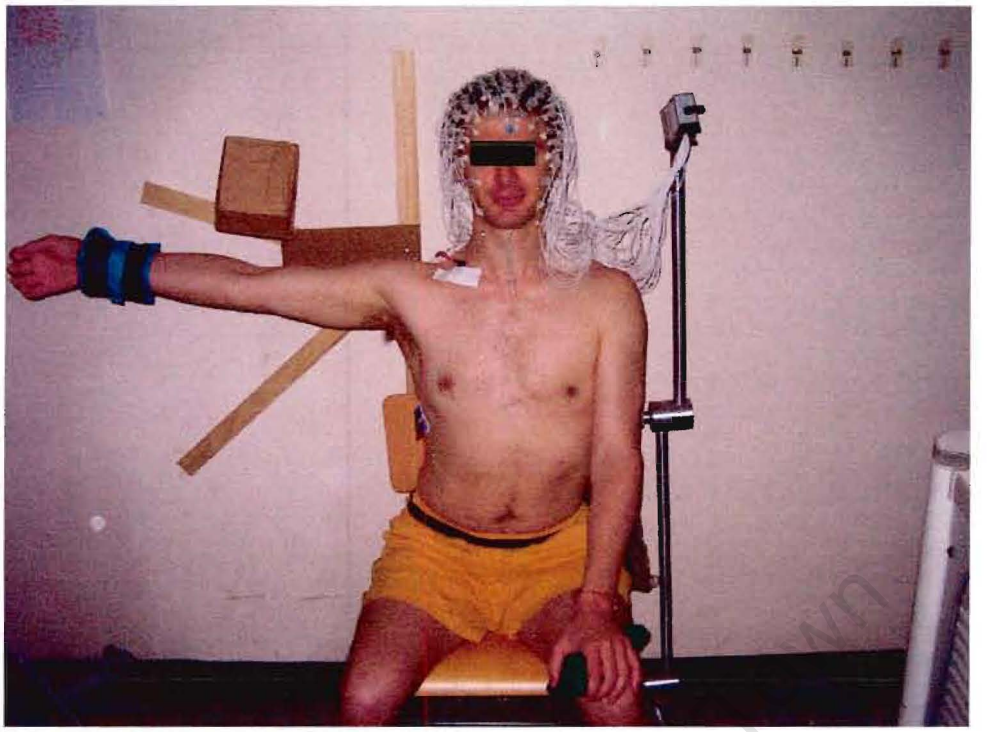

Figure 1. Set-up for testing procedure. The reference electrode as well as the electrode for the serratus anterior muscle is illustrated in the picture. The electrodes for the upper trapezius, lower trapezius and serratus anterior are situated on the dorsal side of the subject. The net on the skull served to collect the EEG (electroencephalography) data. For the purpose of this study, only the EMG (electromyography) data was analysed.

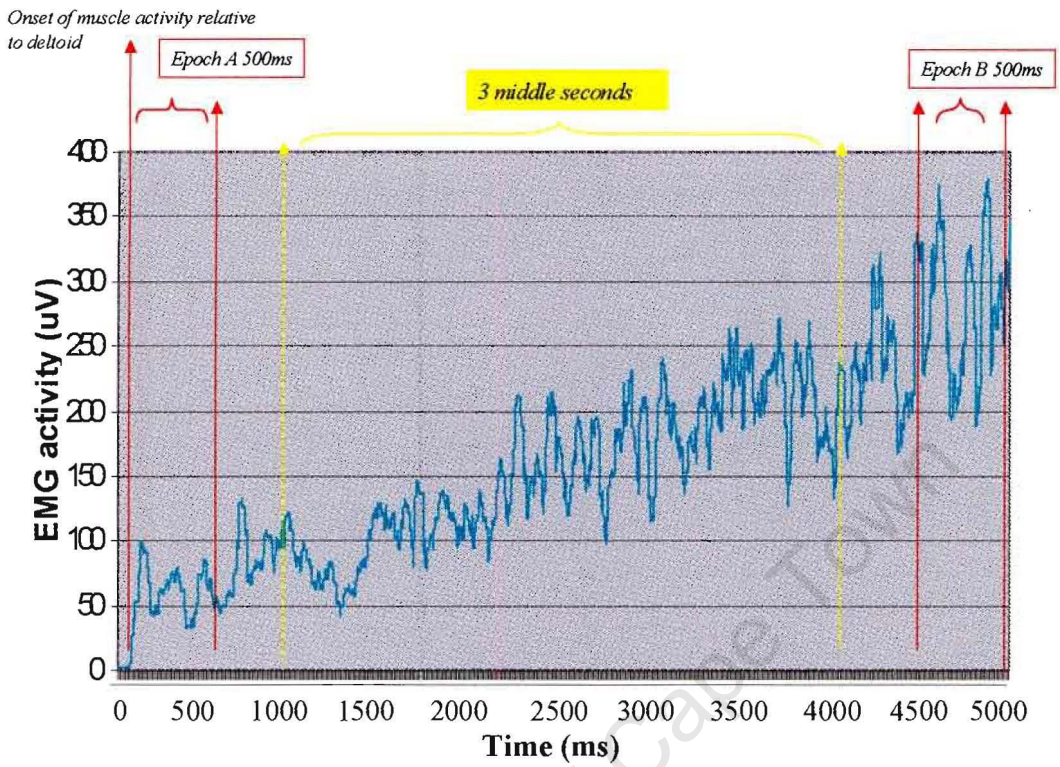

Figure 2: Representation of EMG data of the serratus anterior muscle (example of one subject during) collected during a 5-second abduction movement. In particular, the onset of muscle activity, time epoch A (500ms), middle three seconds, time epoch B (500ms) are shown.

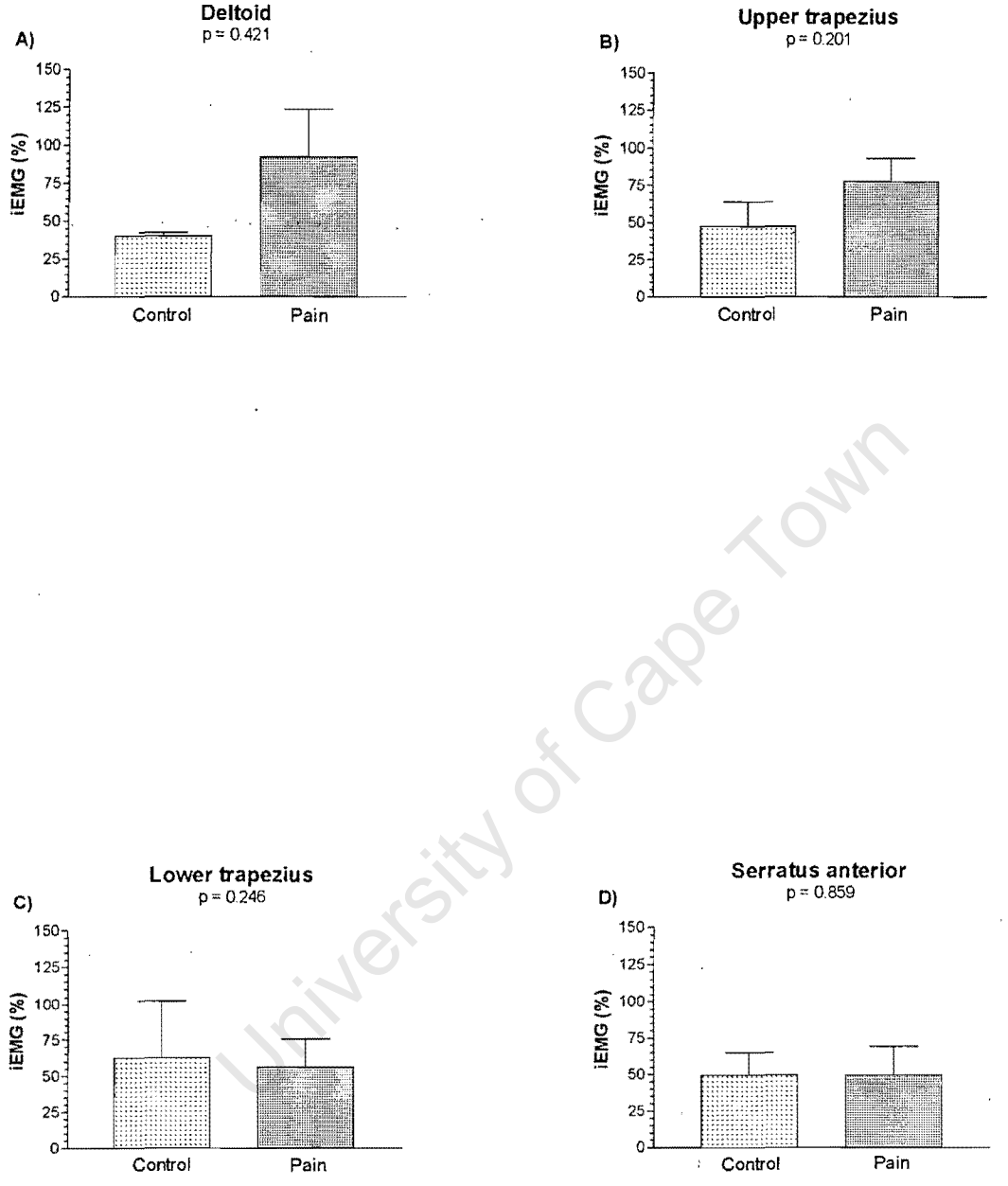

Figure 3. Differences in iEMG activity during **time epoch A** of the abduction movement between the Control and Pain groups for the Deltoid A), Upper trapezius B), Lower trapezius C) and Serratus anterior D) muscles. Data are presented as the mean ± standard deviation. iEMG is expressed as a percentage of the submaximal iEMG value to which it was normalized.

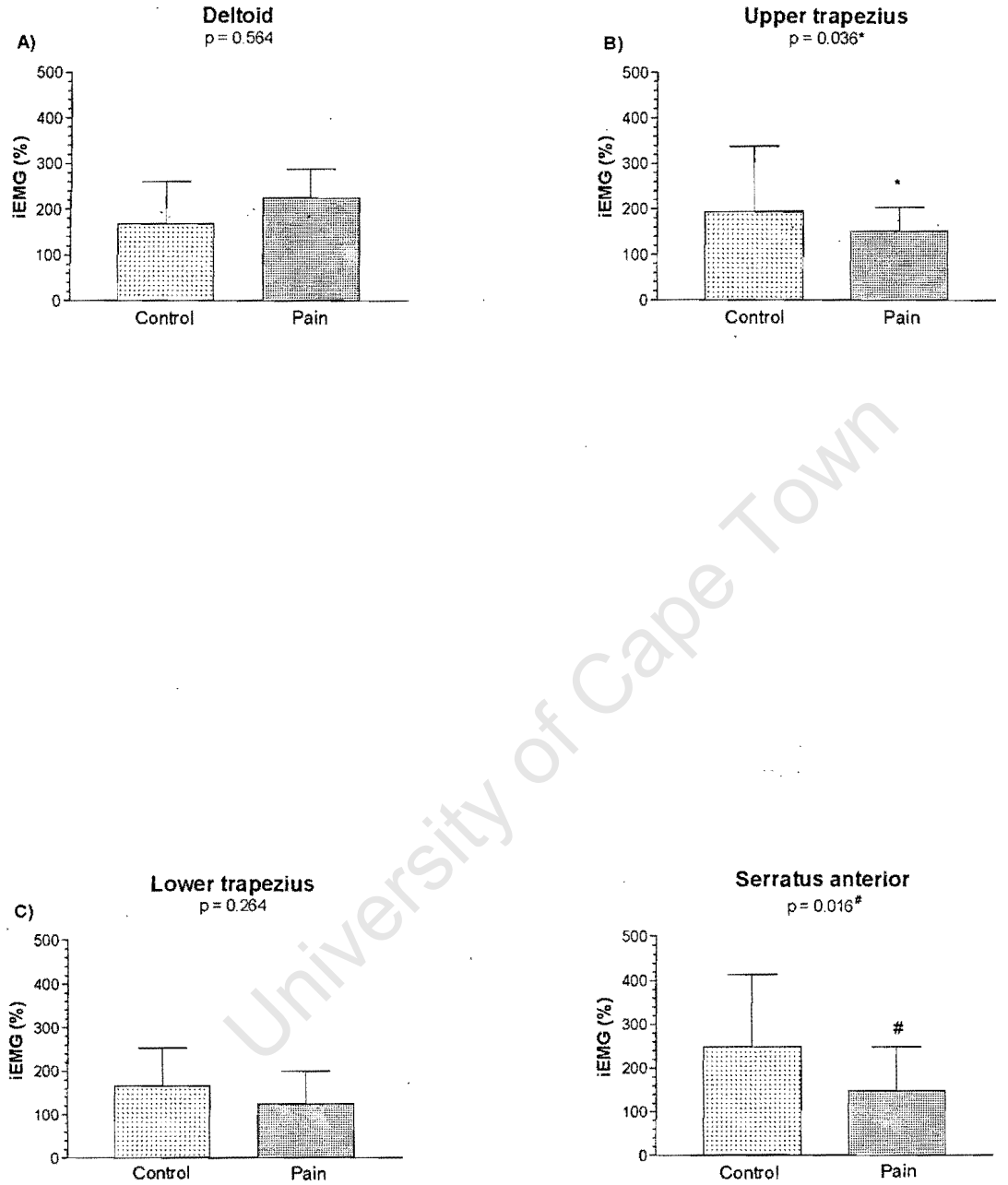

Figure 4. Differences in iEMG activity during **time epoch B** of the abduction movement between the control and pain groups for the Deltoid A), Upper trapezius B), Lower trapezius C) and Serratus anterior D) muscles. Data are presented as the mean ± standard deviation. iEMG is expressed as a percentage of the submaximal iEMG value to which it was normalized. There was a significant difference in iEMG activity of the Upper trapezius muscle * ($p = 0.036$) and the Serratus anterior muscle $^{\#}$ ($p = 0.016$).

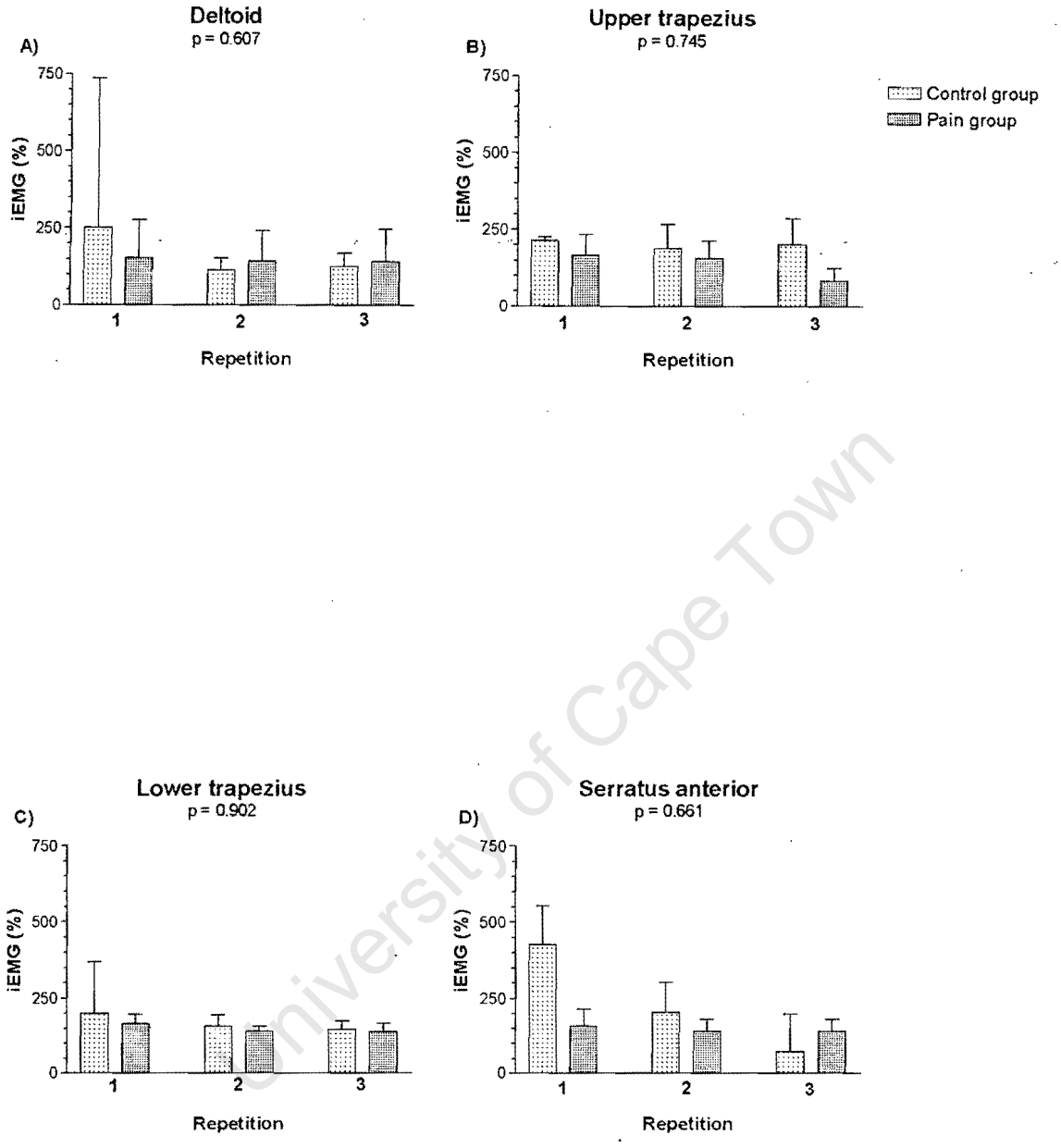

Figure 5. iEMG activity of the Deltoid A), Upper trapezius B), Lower trapezius C) and Serratus anterior D) muscles during the **3 middle seconds** of the 5-second abduction movement for the control and pain groups. Data are presented as the mean ± standard deviation. iEMG is expressed as a percentage of the submaximal iEMG value to which it was normalized. The 3 repetitions of the abduction movement are represented on the graphs for the control and pain groups. The p-value reflects the repetition / group interaction.

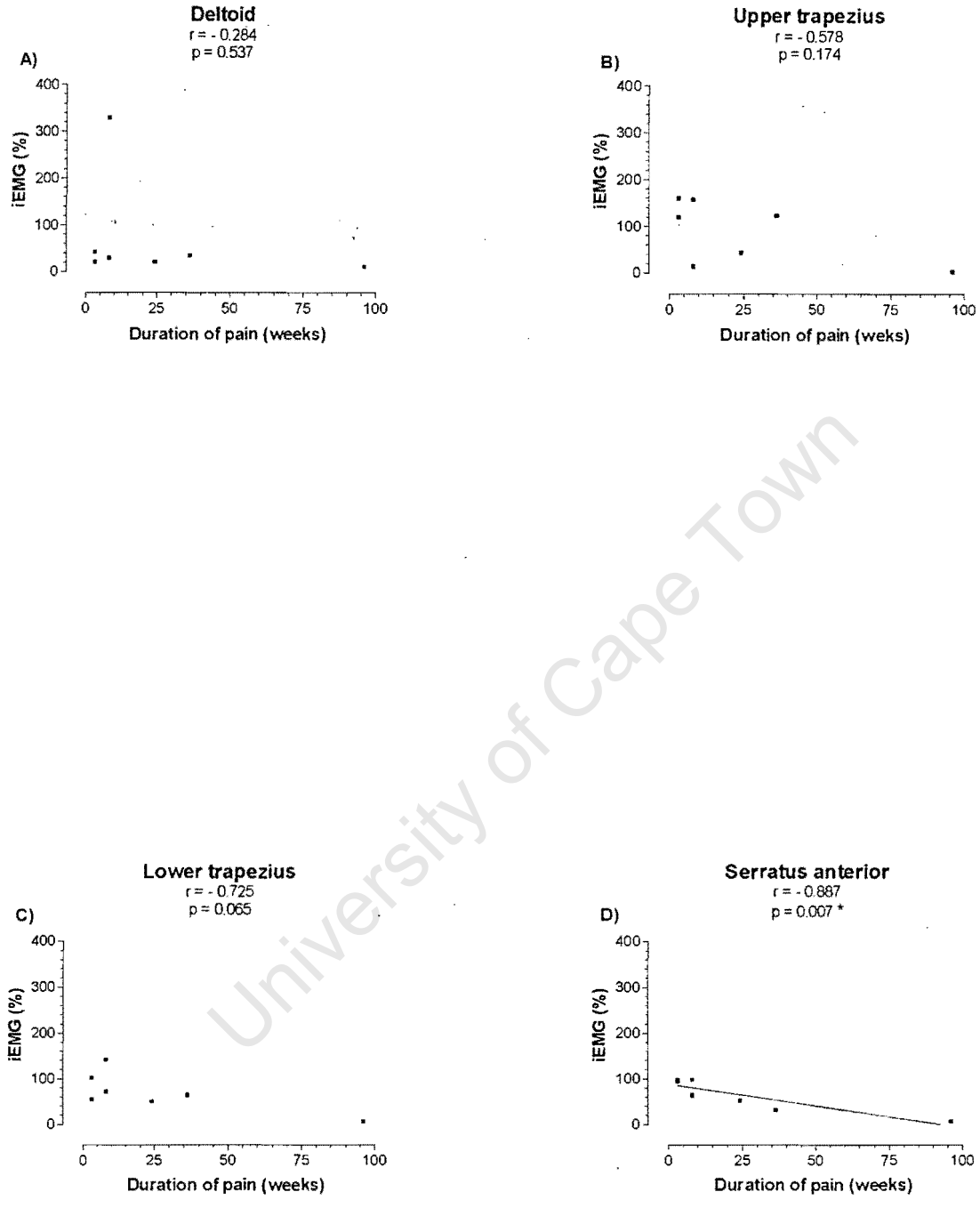

Figure 6. The correlation between the iEMG activity of the Deltoid A), Upper trapezius B), Lower trapezius C), Serratus anterior D) muscles during **time epoch A** and the duration of pain (weeks). IEMG is expressed as a percentage of the submaximal iEMG value to which it was normalized. The duration of pain reflects the time since the onset of the shoulder pain as reported by the pain subjects. The iEMG of the Serratus anterior muscle was significantly correlated to the duration of pain ($p = 0.007$) *, showing that the longer pain had been present, the lower the iEMG activity.

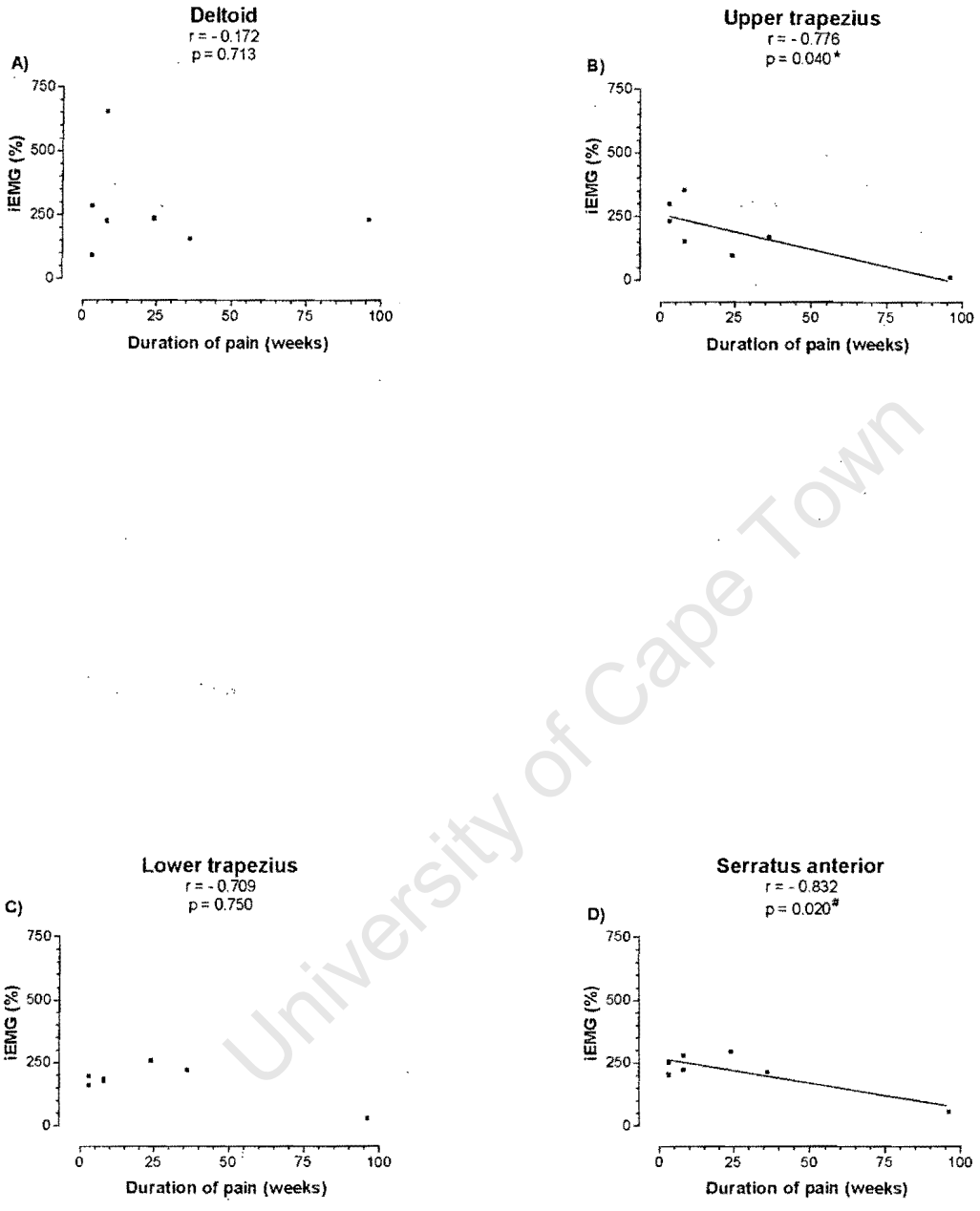

Figure 7. The correlation between the iEMG activity of the Deltoid A), Upper trapezius B), Lower trapezius C), Serratus anterior D) muscles during **time epoch B** and the duration of pain (weeks). IEMG is expressed as a percentage of the submaximal iEMG value to which it was normalized. The duration of pain reflects the time since the onset of the shoulder pain as reported by the pain subjects. The iEMG of the Upper trapezius muscle ($p = 0.040$)* and the Serratus anterior muscle ($p = 0.020$)$^{\#}$ were significantly correlated to the duration of pain, showing that the longer pain had been present, the lower the iEMG activity.

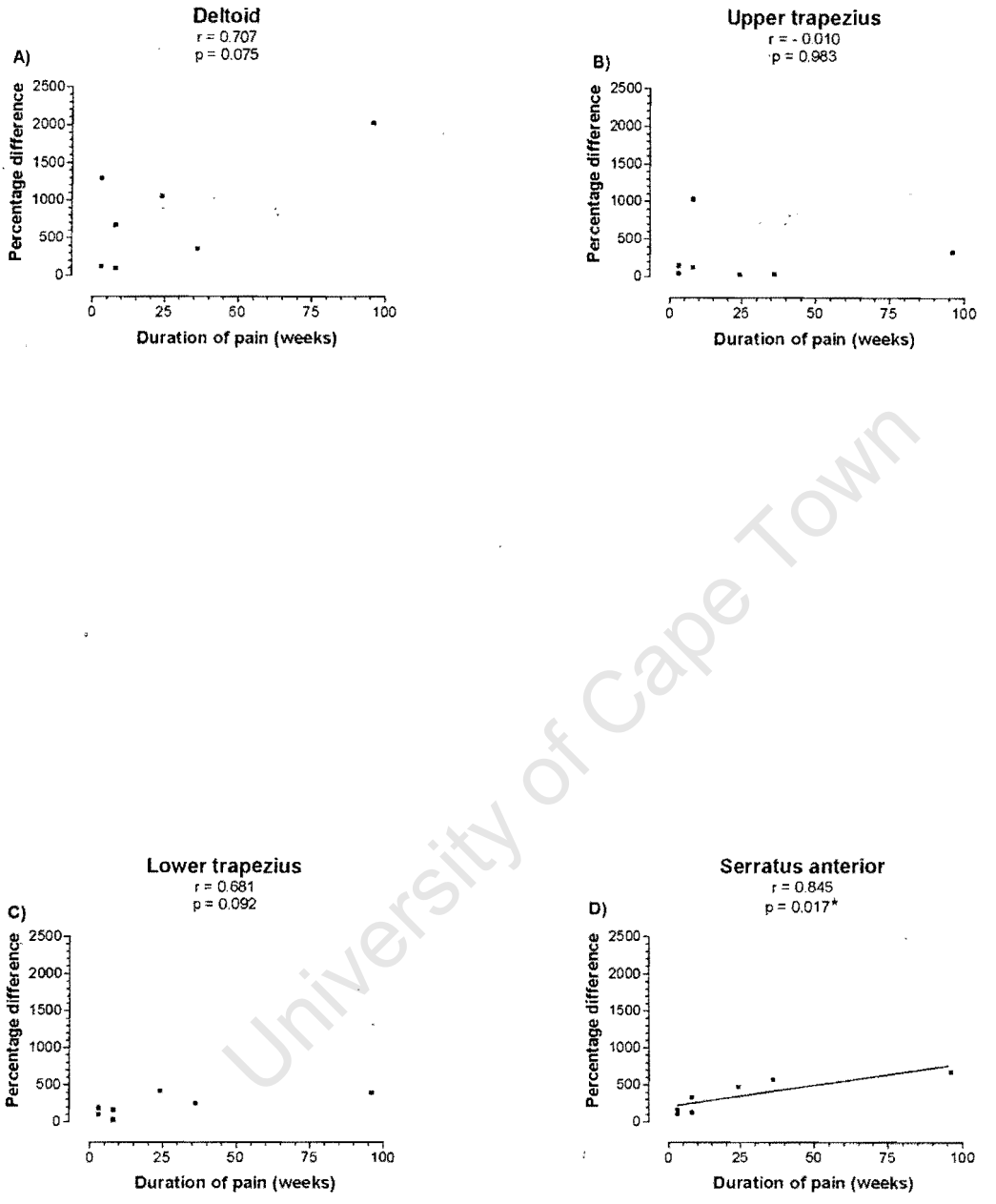

Figure 8. The correlation between the **percentage difference** in iEMG activity between time epoch A and B for the Deltoid A), Upper trapezius B), Lower trapezius C), Serratus anterior D) muscles and the duration of pain (weeks). iEMG is expressed as a percentage of the submaximal iEMG value to which it was normalized. The duration of pain reflects the time since the onset of the shoulder pain as reported by the pain subjects. The percentage difference in iEMG activity between time epochs A and B for the Serratus anterior muscle ($p = 0.017$)* was significantly correlated to the pain duration.

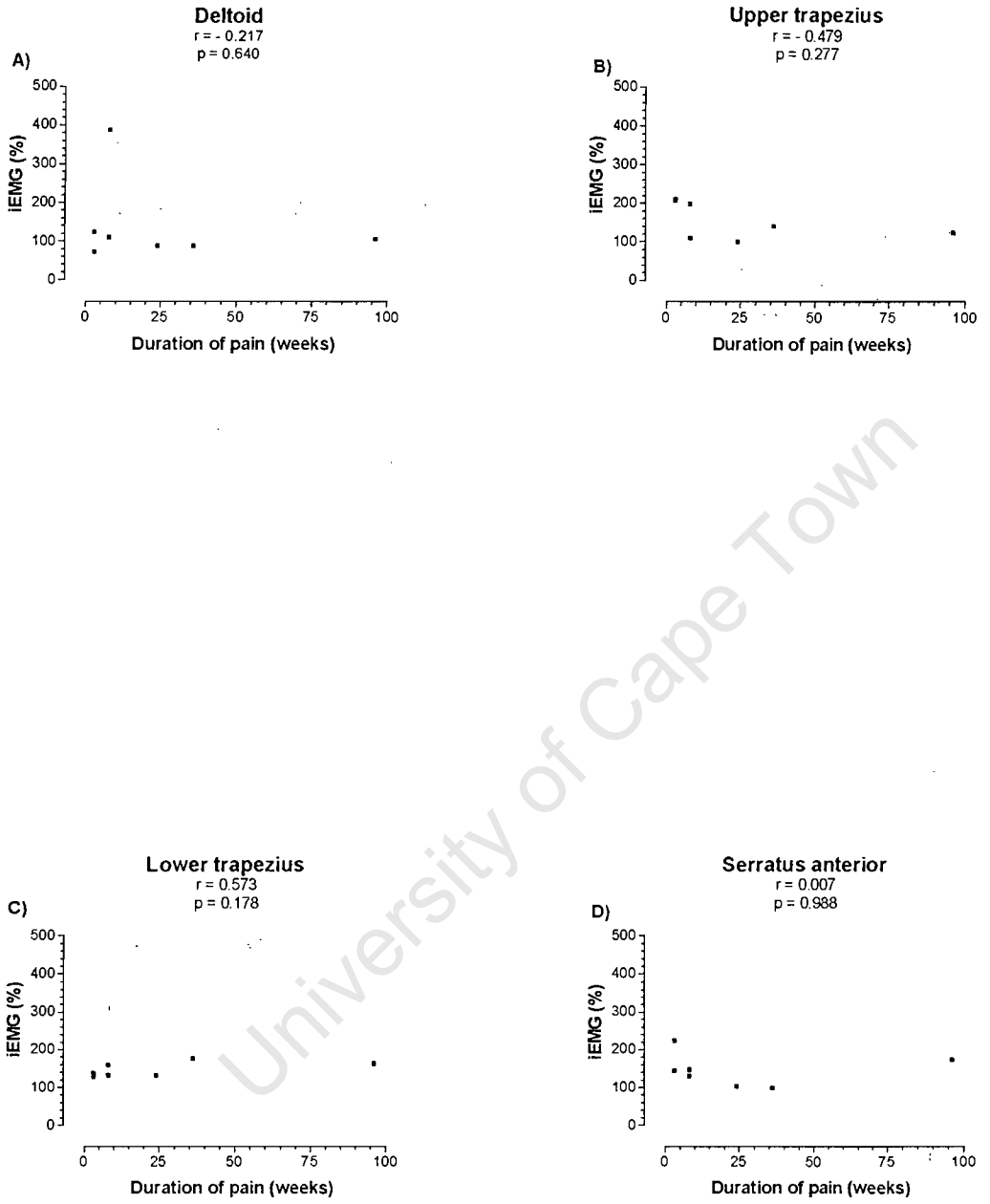

Figure 9. The correlation between the iEMG activity of the Deltoid A), Upper trapezius B), Lower trapezius C), Serratus anterior D) muscles during the **3 middle seconds** of the 5-second abduction movement and the duration of pain (weeks). iEMG is expressed as a percentage of the submaximal iEMG value to which it was normalized. The duration of pain reflects the time since the onset of the shoulder pain as reported by the pain subjects.

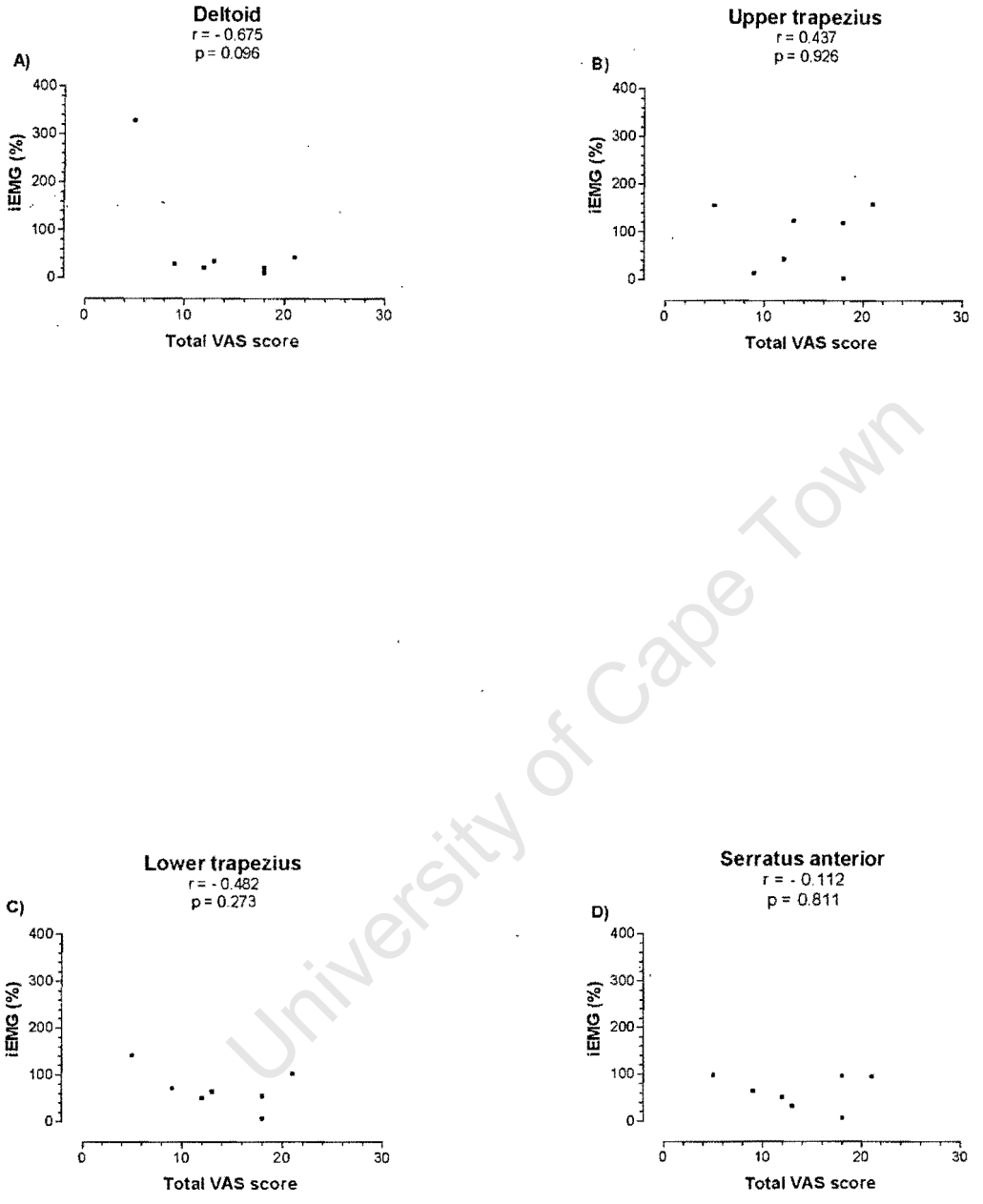

Figure 10. The correlation between the iEMG activity of the Deltoid A), Upper trapezius B), Lower trapezius C), Serratus anterior D) muscles during **time epoch A** and the total score on the visual analogue scale (VAS). iEMG is expressed as a percentage of the submaximal iEMG value to which it was normalized. The total VAS score was determined by the sum of the scores on the visual analogue scale (VAS) during the 3 repetitions of abduction movement.

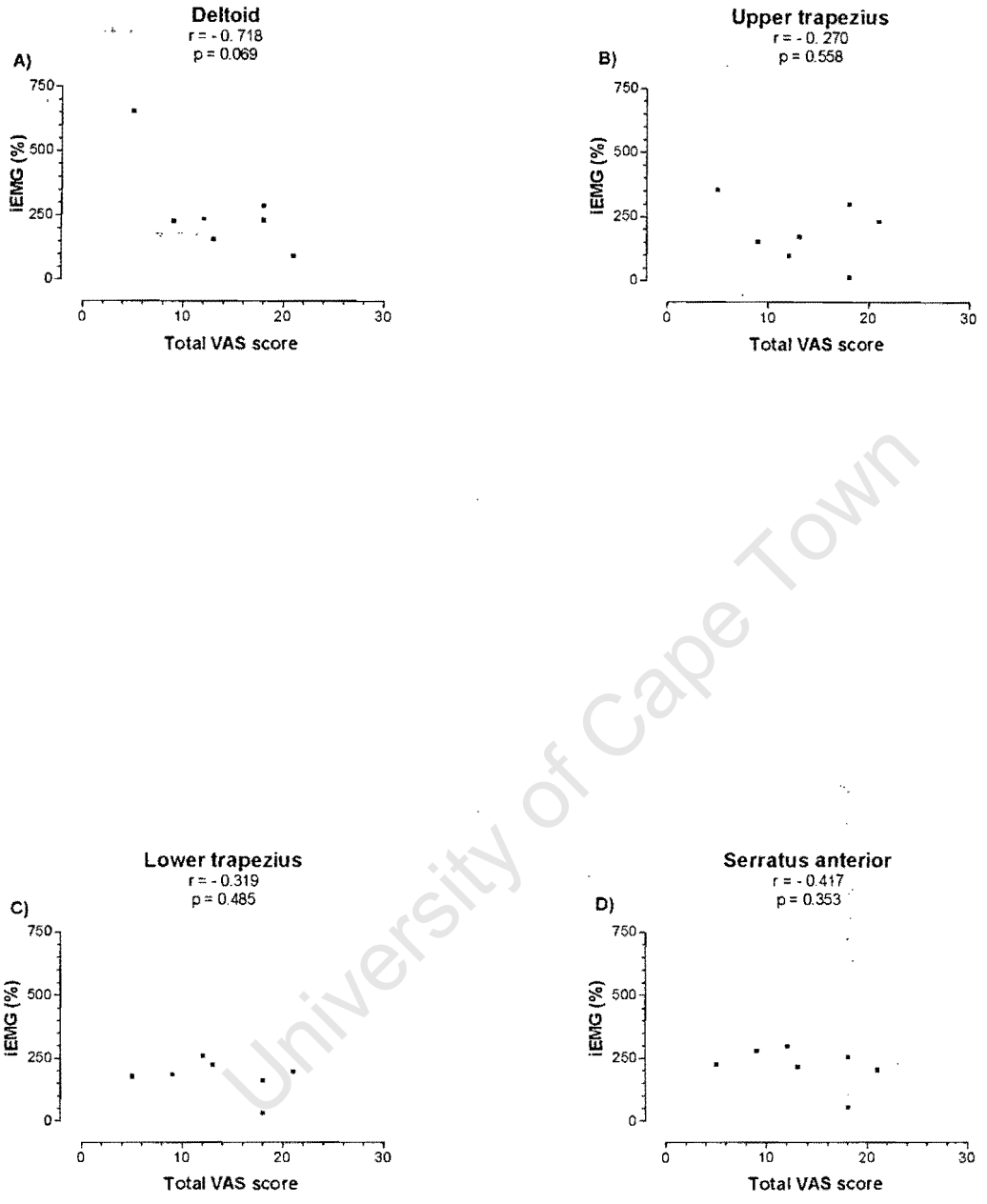

Figure 11. The correlation between the iEMG activity of the Deltoid A), Upper trapezius B), Lower trapezius C), Serratus anterior D) muscles during **time epoch B** and the total score on the visual analogue scale (VAS). iEMG is expressed as a percentage of the submaximal iEMG value to which it was normalized. The total VAS score was determined by the sum of the scores on the visual analogue scale (VAS) during the 3 repetitions of abduction movement.

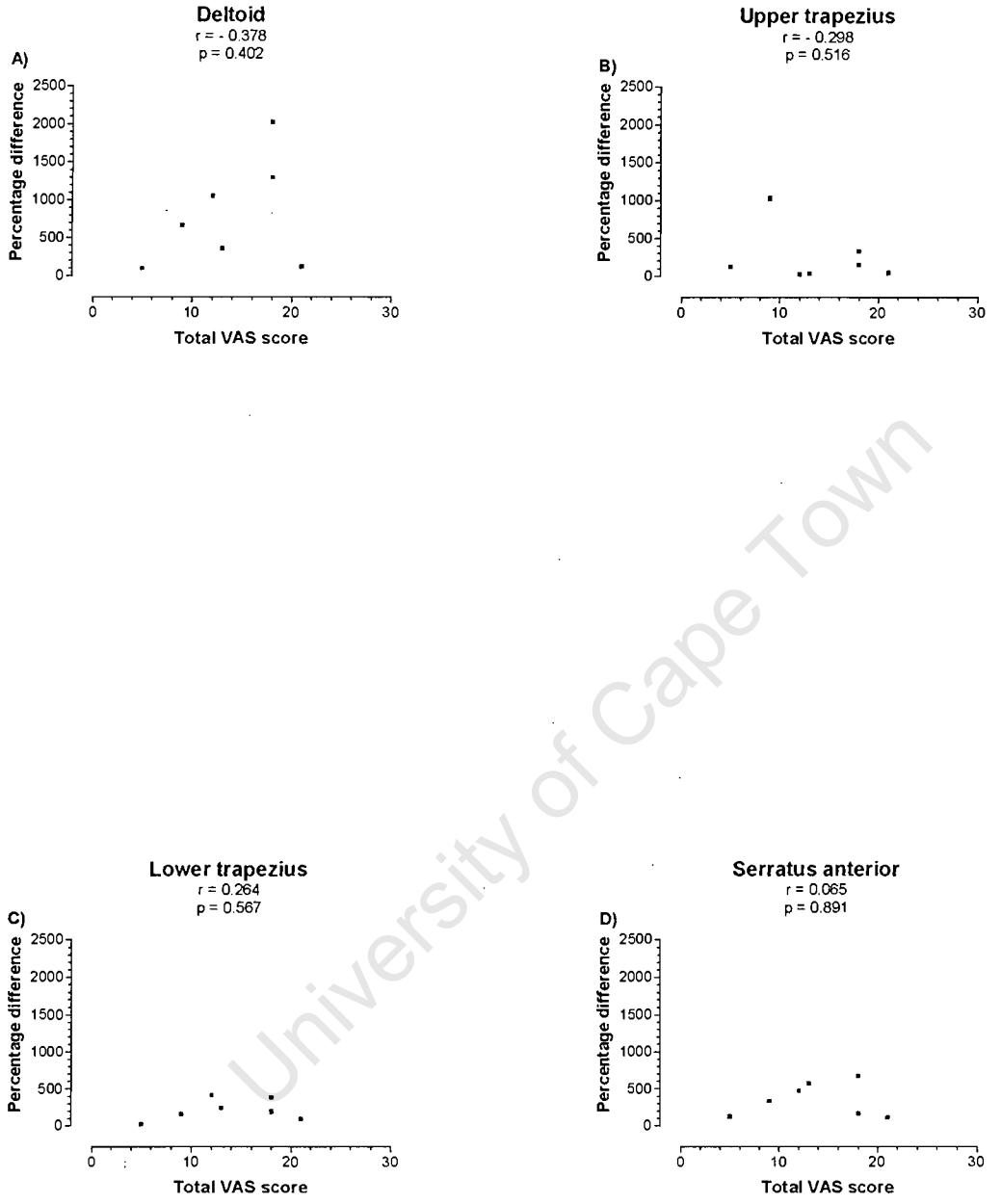

Figure 12. The correlation between the **percentage difference** in iEMG activity between time epoch A and B for the A) deltoid, B) upper trapezius, C) lower trapezius, D) serratus anterior muscles and the total score on the visual analogue scale (VAS). IEMG is expressed as a percentage of the submaximal iEMG value to which it was normalized. The total VAS score was determined by the sum of the scores on the visual analogue scale (VAS) during the 3 repetitions of abduction movement.

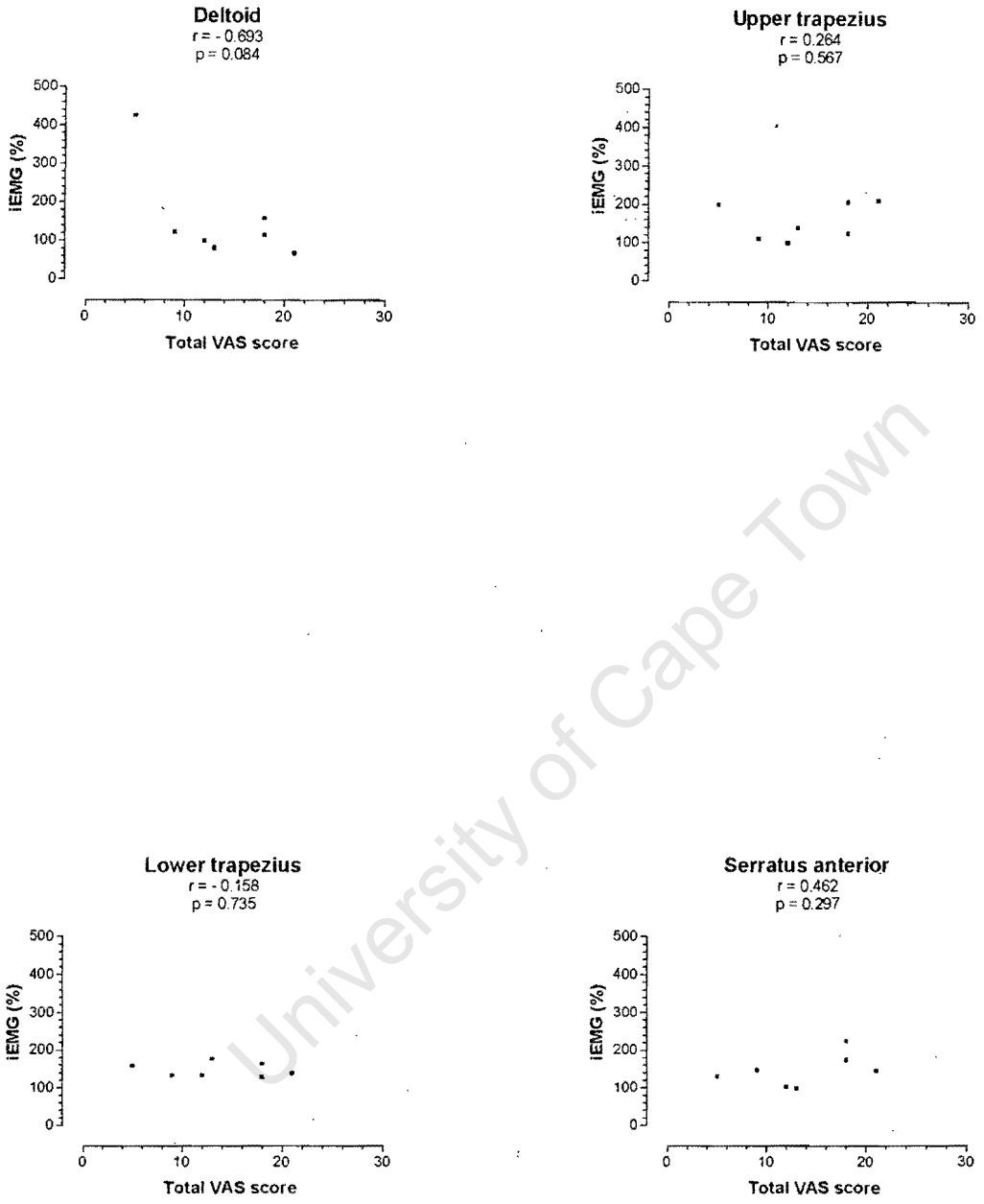

Figure 13. The correlation between the iEMG activity of the Deltoid A), Upper trapezius B), Lower trapezius C), Serratus anterior D) muscles during the **3 middle seconds** of the 5-second abduction movement and the total score on the visual analogue scale (VAS). iEMG is expressed as a percentage of the submaximal iEMG value to which it was normalized. The total VAS score was determined by the sum of the scores on the visual analogue scale (VAS) during the 3 repetitions of abduction movement.

CHAPTER 8

APPENDICES

Appendix 1

INFORMED CONSENT FORM

Study: The effect of chronic shoulder pain on neuromuscular activity and EEG patterns

INTRODUCTION

This study attempts to determine the effect of chronic shoulder pain on neuromuscular activity of the scapular stabilizing muscles and establish an understanding of the mechanism by which the possible supraspinal factors may contribute to the chronic shoulder pain.

PROCEDURE

The following procedures are involved during this trial:

1) The completion of a physical questionnaire on the history of shoulder pain and any other injury or discomfort experienced at present or in the past.

2) The completion of a questionnaire to determine your handedness.

3) Body measurements such as height, weight and skin folds.

4) A physical examination of the shoulder and neck.

5) The measurement of EMG (electromyogram) activity of the shoulder blade muscles. This involves the marking of the skin of the shoulder blades with a washable body marker and 8 hypo-allergic sticky disks will be placed on these markings on the skin. To improve the stickiness, the skin will be wiped with an alcoholic wipe and if necessary body hair will be shaved with a disposable razor.

6) The measurement of EEG (electroencephalogram) activity. Before the EEG net is fitted on the head, the hair will be washed with Johnson's Baby Shampoo. This procedure will ensure optimal conduction of the EEG electrodes.

7) The performances of several arm movements and the activation.

8) After the testing procedure the referring orthopedic surgeon will administer a corticosteroid injection into the painful shoulder. This is the standard treatment for chronic shoulder impingement syndrome.

9) The data collected may be used for scientific purposes and publications in a scientific manner, and all the individual data will be treated confidentially.

RISKS

1) There is a risk that the shoulder pain may increase during this trial. But this risk will be no more than when reaching for an object during activities of daily living. Ice will be available immediately after testing should the pain be increased. The corticosteroid injection, which is administered by an orthopedic surgeon after the testing procedure, will provide appropriate treatment of any exacerbation of shoulder pain as a result of the testing.

2) The risk of any injection is an infection of the injection site. The procedure will be administered under sterile conditions and therefore this risk is minimal. The skin on the side of the shoulder will be cleaned with a hypo allergic disinfectant. An orthopedic shoulder surgeon will then inject corticosteroid into the shoulder with a

sterile needle. The skin of the shoulder will be disinfected once more and a protective plaster will be applied on the injection site.

3) Corticosteroids are potent anti-inflammatory agents. Rapid relief of symptoms after corticosteroid injection may result in overuse of damaged tissue, promoting further tissue degeneration.

4) The EMG and EEG are only used for receiving electrical impulses from the brain and as such, place no inherent risks on myself.

BENEFITS

The subjects will receive information on the pathophysiology of their condition and treatment advice will be given. An estimate of the percentage body fat will be given to the subjects, should they desire it. This research will add to the body of physiotherapy knowledge by increasing the understanding of the effect of chronic shoulder pain on muscle activity.

I, _____ am aware that I will be free to withdraw from the study at any time and that I will not be subjected to any pressure whatsoever to remain in the trial. I understand the implications of my consent and that questions have been answered to my satisfaction.

"The University of Cape Town and its team of researchers, who are working under the mandate of the university, will be responsible for treating any adverse or untoward events arising from participation in this research study."

	Name	**Signature**	**Date**
Subject			
Researcher			
Witness			

Appendix 2

THE ASSESSMENT AND ANALYSIS OF HANDEDNESS (The Edinburgh inventory)

Name:_____

Have you ever had any tendency to left-handedness?

YES	NO

Please indicate your preferences in the use of hands in the following activities by putting + in the appropriate column. Where the preference is so strong that you would never try to use the other hand unless absolutely forced to, put ++. If in any case you are really indifferent put + in both columns. Some of the activities require both hands. In these cases the part of the task, or object, for which hand-preference is wanted is indicated in brackets. Please try to answer all the questions, and only leave a blank if you have no experience at all of the object or task.

		R	L
1	Writing		
2	Drawing		
3	Throwing		
4	Scissors		
5	Comb		
6	Toothbrush		
7	Knife (without fork)		
8	Spoon		
9	Hammer		
10	Screwdriver		
11	Tennis Racket		
12	Knife (with fork)		
13	Cricket Bat (lower hand)		
14	Golf Club (lower hand)		
15	Broom (upper hand)		
16	Rake (upper hand)		
17	Striking match (match)		
18	Opening box (lid)		
19	Dealing cards (card being dealt with)		
20	Threading needle (needle, thread according to which is moved)		
21	Which foot do you prefer to kick with?		
22	Which eye do you use when using only one?		

Appendix 3

PHYSICAL QUESTIONNAIRE

Name:

1. Are you taking part in any sport?

Y	N

If YES, what kind of sport?

Tennis	
Cricket	
Volleyball	
Swimming	
Other, specify.....	

_____Times per week

_____ Hours per week

2. Are you experiencing any neck pain presently?

Y	N

3. Have you experienced any neck pain in the past?

Y	N

If YES, _____Weeks ago _____ Months ago _____Years ago

4. Are you experiencing pain or discomfort in any part of your body presently?

Y	N

If YES, where?.....

_____ Weeks ago _____Months ago _____Years ago

5. Have you had shoulder pain in the past?

Y	N

If YES, under what circumstances?.....

_____ Weeks ago _____ months ago _____years ago

6. Are you experiencing any shoulder pain presently?

Y	N

8. How did your shoulder pain start?.....

9. In which shoulder are you experieing pain?

Left	Right	Both

10. When did your shoulder pain start?

_____Weeks ago _____ months ago __ years ago

11. Please tick the most appropriate answer about your shoulder pain:

Pain is always present and unbearable	
Pain is always present but bearable	
Pain is not always present but sometimes unbearable for short periods	
No pain or little at rest, present during light activities	
Pain is present during heavy or particular activities	

12. Please tick the most appropriate answers about what aggravates your pain:

More than one answer can be ticked

Unable to use limb	
Pain while driving	
Pain while brushing hair	
Pain while dressing / undressing	
Able to do light activities	
Able to do most housework	
Able to work above shoulder level	

13. Please tick the most appropriate answer about your sporting activities:

Pain after sport but not during or affecting sport	
Pain during sport but not affecting sport	
Pain during sport and affecting sport	
Pain prevents all participation in sport	

14. Please tick the appropriate answers about treatment modalities:

    More than one answer can be ticked

Physiotherapy		Date of last treatment:
Cortisone injection		Date of last injection:
Pain relieving medication		Date of last tablet taken: Time of last tablet taken:
Chiropractor		Date of last consultation
Other treatment modalities	Specify:	Date of last treatment:

Appendix 4

PHYSICAL EXAMINATION PROTOCOL

NAME:

ANTHROPOMETICAL DATA

WEIGHT	
HEIGHT	
AGE	
SKINFOLDS	
BICEPS	
TRICEPS	
SUBSCAPULAR	
ABDOMEN	
SUPRAILIAC	
TIGH	
CALF	

IMPINGEMENT TESTS

HAWKINS'S SIGN	RIGHT	LEFT
POSITIVE		
NEGATIVE		
NEER'S SIGN	RIGHT	LEFT
POSITIVE		
NEGATIVE		
JOBE'S SIGN	RIGHT	LEFT
POSITIVE		
NEGATIVE		
PAINFUL ARCH	RIGHT	LEFT
POSITIVE		
NEGATIVE		

EXAMINATION OF THE NECK

ACTIVE NECK MOVEMENTS	FLEXION	EXTENSION	LATERAL FLEXION RIGHT	LATERAL FLEXION LEFT	ROTATION RIGHT	ROTATION LEFT
NO PAIN						
PAIN						
OVERPRESSURE	FLEXION	EXTENSION	LATERAL FLEXION RIGHT	LATERAL FLEXION LEFT	ROTATION RIGHT	ROTATION LEFT
NO PAIN						
PAIN						

VISUAL ANALOGUE SCALE (VAS):

0 **10**

|----------|

NO PAIN MAXIMUM PAIN

BEFORE PROCEDURE	
FAMILIRISATION ABD	
ABD 1	
ABD 2	
ABD 3	
ISOMETRIC CONTRACTION 1	
ISOMETRIC CONTRACTION 2	
ISOMETRIC CONTRACTION 3	