Biomechanical Analysis of the Shoulder Joint Motion in Patients with Rotator Cuff Tendinopathy

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Preface

The work presented in this dissertation was performed at the Lisbon Biomechanics Laboratory of Instituto Superior Técnico (Lisbon, Portugal), during the period February 2018 - May 2019, under the supervision of Prof. Miguel Tavares da Silva and Dr. Guilherme Ferreira Dos Santos.
Declaration

I declare that this document is an original work of my own authorship and that it fulfills all the requirements of the Code of Conduct and Good Practices of the Universidade de Lisboa.
Acknowledgments

First of all, I would like to thank my supervisors, Professor Miguel Tavares da Silva, who always gave me direction, believing in my work and never ceased to support me through all my decisions and moments of insecurity; to Doctor Guilherme Ferreira Dos Santos, who initiated this project and allowed me to be apart of it. Thank you for all your support and encouragement, I hope your unwearied energy continues to influence everyone around you.

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Resumo

As patologias do ombro são muito comuns e prevalentes na população em geral, apresentando taxas de prevalência entre 1% a 66.7%. Tradicionalmente, os métodos de diagnóstico clínico são baseados na dor, redução da amplitude de movimento e redução da força muscular, sintomas comuns à ampla variedade de patologias do ombro. O objetivo deste estudo é avaliar a viabilidade da utilização de variáveis cinemáticas mensuráveis e dados de eletromiografia de superfície como critério para um diagnóstico mais objetivo e preciso da tendinopatia da coifa dos rotadores. Para a aquisição cinemática tridimensional do ombro, 22 marcadores retro-reflexivos foram monitorizados usando um sistema de medição opto-eletrônico, composto por 14 câmaras digitais de infravermelho. A eletromiografia foi obtida simultaneamente com os dados cinemáticos. Vinte e quatro sujeitos participaram neste estudo: 9 doentes diagnosticados com tendinopatia unilateral do supraespinhoso e um grupo controlo de 15 indivíduos saudáveis. Os movimentos considerados foram elevação do braço nos planos frontal e sagital. Os resultados mostraram diferenças mais notáveis durante os primeiros 60° de elevação umerotorácica no plano frontal (abdução) onde os ombros sintomáticos apresentaram uma diminuição da rotação superior, inclinação posterior e rotação externa da escápula, quando comparados com o grupo de controlo e o ombro contralateral. Diferenças foram também encontradas nos padrões do sinal normalizado de sEMG, onde os deltóides apresentaram uma evolução mais rápida no ombro sintomático do grupo patológico quando comparado com o ombro asintomático e o grupo controlo.

Palavras-chave: Cinemática do Ombro, Ritmo Escapuloumeral, sEMG do Ombro, Tendinopatia da Coifa dos Rotadores
Abstract

Shoulder disorders are very common and prevalent in the general population, having lifetime prevalence rates ranging from 1% to 66.7%. Traditionally, clinical diagnosis methods are based on pain, reduced range of motion and reduced muscle force, which are common symptoms to the wide variety of shoulder disorders. The aim of this study is to evaluate the feasibility of using measurable kinematic variables and surface electromyography (sEMG) data as criteria for a more objective and accurate diagnosis of rotator cuff tendinopathy (RCT). To measure the 3D kinematics of the shoulder, 22 retro-reflective markers were tracked using an opto-electronic measurement system composed of 14 digital infrared cameras. sEMG was recorded synchronously with the kinematic data. Twenty-four subjects participated in this study: 9 subjects diagnosed with unilateral supraspinatus tendinopathy (UST) and a control group (CG) of 15 healthy subjects. The movements considered were arm elevation in both frontal and sagittal planes. Results showed more notable differences during the first 60° of humerothoracic (HT) elevation in the frontal plane (abduction), where the symptomatic shoulders decreased upward rotation, posterior tilt and external rotation of the scapula when compared to CG and its contralateral shoulder. Differences were also found in the targeted muscles normalized sEMG patterns, as the deltoid muscle showed a faster evolution of the signal on the pathological group (PG)’s symptomatic shoulder.

Keywords: Shoulder Kinematics, Scapulohumeral Rhythm, Rotator Cuff Tendinopathy, Shoulder sEMG
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Glossary

AD  anterior deltoid.

BMI  body mass index.

CG  control group.

EF  elevation factor.

EMG  electromyography.

GH  glenohumeral.

IF  increment factor.

IS  infraspinatus.

IT  inferior trapezius.

m-DYN  mean dynamic amplitude.

MD  medial deltoid.

MEI  movement elevation interaction.

MF  movement factor.

MII  movement increment interaction.

PD  posterior deltoid.

PG  pathological group.

PM  pectoralis major.

RCT  rotator cuff tendinopathy.

SA  serratus anterior.

sEMG  surface electromyography.
SHR  scapulohumeral rhythm.
SIS  shoulder impingement syndrome.
SS  subscapularis.
SS  supraspinatus.
ST  superior trapezius.
STIR  scapulothoracic internal rotation.
STT  scapulothoracic tilt.
STUR  scapulothoracic upward rotation.
TM  teres minor.
UST  unilateral supraspinatus tendinophaty.
Chapter 1

Introduction

1.1 Motivation

Shoulder disorders are very common and prevalent in the general population, having lifetime prevalence rates ranging from 1% to 66.7%, depending on the specific shoulder disorder (Luime et al., 2004; VanDerHeijden, 1999; Walker-bone et al., 2004). They are one of the main causes for seeking medical care (VanDerHeijden, 1999; J. et al., 2003), and in many cases translate in movement restrictions and thereby limitations in daily and work activities (VanDerHeijden, 1999; Luime et al., 2004). Traditional diagnostic methods lack accuracy as they are based on criteria such as pain, reduced range of motion and muscle strength, which are of general application and not specific to the wide variety of possible shoulder disorders. In other words, there is an overlap of symptoms between the different disorders, which may mislead the implementation and management of treatments protocols (Walker-bone et al., 2004; Lewis, 2008; VanDerHeijden, 1999).

Due to the lack of accuracy of the traditional clinical methods there has been a growing interest on studying the shoulder kinematics using computational techniques. The first computational methods used to study kinematics were applied to gait, and the methods now utilised to study the shoulder joint have evolved from these first studies. However, due to the different nature of upper body movements, data analysis is more complex. Arm motion is not cyclic and can achieve a given position with different combinations of joint rotations. Despite these challenges, shoulder biomechanics has progressed rapidly in the last 20 years. However, there is still little research on pathological movements and how this research can be used as a complementary diagnostic tool or as a way to improve rehabilitation programmes.

It is very difficult to find measurable variables on shoulder kinematics due to the complex anatomy of this joint. The scapulohumeral rhythm (SHR) is one of the most widely used parameters to study and evaluate shoulder movement. It describes the motion of the scapula relative to the humerus, during arm elevation and has proven to have clinical relevance when evaluating shoulder kinematics, since for a healthy condition it is expected to translate a specific pattern of joint rotations. However, there is not full consensus on this value or other indicators of shoulder kinematics. Hence, it is essential to continue studying shoulder biomechanics, whilst having a well designed experimental protocol with movements
as standardized as possible and an accurate motion tracking technique to guarantee reliable kinematic measurements.

1.2 Scope and Objectives

The main objective of this thesis was to assess the biomechanical differences in shoulder joint motion between subjects with unilateral supraspinatus tendinopathy (UST) and asymptomatic individuals, during shoulder motion performed in the sagittal and frontal planes of humerothoracic elevation. To do so, an optoelectronic tracking system was used to acquire shoulder kinematics, while surface electromyography (sEMG) was applied to record the activation patterns of a set of shoulder muscles. In order to fully understand the kinematic alterations on the symptomatic subjects, their asymptomatic shoulder was also analysed and compared with both its contralateral shoulder and the Control group. This study also intended to analyse the reliability of using both an optoelectronic tracking system and sEMG to differentiate between pathologic shoulder kinematics and the physiological pattern of muscle activation.

1.3 Literature Review

1.3.1 Clinical Methods

Diagnosing shoulder disorders has always proven to be a challenging task, mainly due to the underlying nature of the traditional diagnostic methods used in clinical practice (Lewis, 2008; Hegedus et al., 2008; Calis et al., 2000).

Historically, the diagnostic methods were based on the fact that different shoulder structures can be isolated and tested in order to find the cause of a patient’s symptoms. The tests applied would either compress, stretch or require the tissue of interest to contract, having pain as a positive result for some kind of shoulder disorder. These kinds of tests can be identified as symptom alteration tests. Imaging techniques along with other tests (blood test, diagnostic injection) can be used to gather more information to achieve an accurate diagnosis (Lewis, 2008).

In 1972, Neer introduced the concept of shoulder impingement syndrome (SIS) and a test to diagnose this pathology. For the Neer test, while the examiner prevents scapular rotation with one hand, the arm of the patient is forced to elevation at an angle between flexion and abduction, by the other hand (Neer, 1972; Calis et al., 2000; Fodor et al., 2009).

Silliman & Hawkins (1994) invented the Hawkins test, also used to diagnose SIS based on contemporary physical examinations signs. This test is performed by placing the patient’s arm at 90° of strict forward elevation with the elbow at 90° flexion, and then forcing the arm to rotate medially by lowering the forearm (Silliman & Hawkins, 1994; Calis et al., 2000; Fodor et al., 2009).

However, this kind of approach to diagnose a specific shoulder disorder is flawed. The shoulder anatomy is complex, so the premise of being able to isolate different structures is wrong. When trying to compress or stretch a given shoulder structure, most likely another structure will also suffer the same
consequences and therefore the results will lose specificity. Additionally, imaging techniques cannot fulfill the lack of specificity of physical examination tests, since there is not a direct correlation between radiological imaging and symptoms (Lewis, 2008).

Shoulder pathologies have also been associated with alterations of normal scapular kinematics (Sil-liman & Hawkins, 1994; Lange et al., 2017; Kibler et al., 2013; Tripp & Ph, 2009) - scapular dyskinesis - which can translate to alterations in the normal SHR. Clinical methods have also been developed with the objective of measuring and quantifying SHR, both in normal and in injured shoulders (Tripp & Ph, 2009).

A few of the first tests that tried to measure scapular dyskinesia were the lateral scapular slide test (Kibler, 1998), the posterior displacement test (Turczany et al., 1997), and the scapular upward rotation measurement (Johnson et al., 2001). These tests used static measures that assessed scapular position in 1 plane or, at most, 2 planes (Tripp & Ph, 2009). These kinds of techniques make use of measuring instruments, such as inclinometers and specially designed instruments to measure displacement (Johnson et al., 2001; Kibler, 1998), or some type of criterion-based visual observation (Turczany et al., 1997). Despite a more objective approach of these observational/measuring tests the lack of sensitivity and specificity remains.

In order to tackle the lack of diagnose accuracy and reliability of clinical diagnosing methods, Kibler et al. (2013) suggested a combination of several clinical tests to identify scapular dyskinesis: 1) visual observation, 2) effect of manual correction on shoulder symptoms (symptom alteration tests), and 3) examination of surrounding anatomic structures. Despite scapular dyskinesis not being a specific symptom of rotator cuff tendinophaty (RCT) it is proven to be related to shoulder pathology and as such it is essential to study pathological shoulder kinematics (Lange et al., 2017). Kibler et al. (2013) recommendation provides a more reliable method to identify it and consequently provide a more accurate diagnoses.

A tendinopathy is defined as an overuse condition that manifests itself as pain in and around tendons. Factor & Dale (2014) stated that this painful condition is associated with tendon disorganization and thickening that reduces its physical properties, which causes the tendon to fatigue, further exacerbating the painful condition with ultimate failure. Several authors have suggested that during arm elevation the scapula needs to upwardly rotate, posteriorly tilt and externally rotate to avoid pathology (Ludewig et al., 2009a; Kibler et al., 2013; Lawrence et al., 2014). Alterations to this kinematic pattern may deregulate healthy tendon function and lead to a tendinopathy. As such it is very important to study possible harmful shoulder dyskinesia, in order to understand its causes and/or consequences.

1.3.2 Computational Methods - Kinematic analysis

Biomechanics of movement has a very long history, starting from the animal movement studies of Aristotle, followed by the anatomic drawings of Leonardo da Vinci and the work of many other scientists who tried to understand how human kinematics and kinetics work.

The early studies on biomechanics focused on human gait. Most methods used to study upper body
kinematics were initially conceived to study gait. However, due the recent improvements in 3D motion analysis techniques and the lack of previous research on upper body kinematics, there has been an increased interest on this field of study.

The main reason behind the lack of research on upper body kinematics is the complex nature of upper body motion when compared to gait. In contrast to gait, arm movements are not cyclic. Additionally, due to the high variety of arm movements it is difficult to construct a specific pattern for a given movement, since the same movement can be achieved by different joint adaptations.

Although initial studies on human motion kinematics focused on the movement of the lower limbs, there were also contemporary studies on the movement of the upper limbs where only range of motion was studied.

Hancock (1968) published a literature review on upper-extremity motion effectiveness where he stated that arm range of motion had already been studied using goniometers and photography by 1921. However, it was only almost 40 years later that someone tried to measure the coordinate motion of daily activities. These studies were meant to construct orthoses or prostheses and to provide data for biomechanical models.

Taylor & Blaschke (1951), introduced a new kinematic analysis of the shoulder motions, arm and hand using cine camera, two mirrors and passive markers. In this early approach to photography as a tracking technique, the two mirrors were used to allow two different views besides the one obtained from the cine camera. The markers were meant to follow bone movement around the centre of joint rotation. The motivation behind this work was to study the functional requirements of arm and hand prostheses, and to do so, joint rotation was studied by looking at the markers path across time. Later, more studies made use of this technology but with more cameras (Langrana, 1981), improving the line of sight of the markers.

Engin et al. (1984a), introduced a sonic digitizing technique where a set of microphones/sensor assembly, an electronic control unit and a generator/multiplexer are used to determine direction and location of body segments. This technique is based on the principle of sound triangulation. The several generators placed on the skin surface emit sonic impulses that are received by a microphone assembly. In order to get a more accurate result, the three microphones closest to a given impulse triangulate the signal and give the location of each emitter. To get the direction of a body segment, 4 sensors are placed per segment to guarantee that at least 3 emitters are detected at all times, giving the direction of the respective body segment (Engin et al., 1984a,b). In a second part article, Engin et al. (1984b) applied this sonic digitizing technique and a Force Applicator device in a drawer test, in order to study shoulder stiffness. The sonic emitters would give the location and direction of the force, since the subjects arm would be tested in different orientations.

Video-based methods or sonic digitizing techniques make use of rotation matrices to provide location and direction of body segments. However, a given arm position can be achieved with different combinations of body segment rotations. In order to tackle this problem, Johnson & Anderson (1990), studied the advantages of using an electromagnetic movement sensor to measure spatial motion.

Electromagnetic tracking systems consist of an electromagnetic source and a sensor. The source
generates an electromagnetic field (detected by the sensor) and the electronics package calculates the relative position and orientation from the detected magnetic field with the full six degrees of freedom required in 3-D (Johnson & Anderson, 1990).

To validate the technique suitability to study shoulder movement, a set of standardized movements were tested: flexion, extension and abduction without humeral rotation; abduction, with an attempt to maintain 90º of external rotation throughout; internal rotation as measured by asking the subject to place the arm as far behind the back as possible. In order to study these movements the angles as function of time were calculated using two different methods: a traditional clinical method where the movement of the shoulder is described in two planes (sagittal and frontal) together with the rotation about the long axis of the humerus; and a method suggested by Kapandji (2000), using spherical polar coordinates (Johnson & Anderson, 1990).

The results of this study proved that electromagnetic tracking systems, along with spherical polar coordinates, could be applied to a clinical setting, with the main advantage of providing the possibility of analysing true 3-D movement as well as the out of plane movements (Johnson & Anderson, 1990).

Electromagnetic sensors can also be applied through bone-fixed pins, attaching the sensors to surgically placed pins (Lawrence et al., 2014; Ludewig et al., 2009a). Although bone-fixed tracking provides a direct tracking of bone movement, Cook et al. (2002) proved that for certain movements it is possible to use surface sensors with minimal errors at slow velocities. Additionally, surface sensors do not require such an invasive approach, making them the most common way to apply an electromagnetic system.

On the studies previously mentioned, upper limbs biomechanics was based on the study of ranges of motion and/or joint angles as a function of time. However, when it comes to the shoulder joint the analysis becomes very complex, since its motion depends on scapular and humeral rotation. To provide a more accurate insight on shoulder motion, Högfors et al. (1991) studied the relationship between scapular and humeral rotations, in order to understand if there was a pattern in their relative motions. This relative motion is called rhythm of the human shoulder.

To do so, Högfors et al. (1991) also introduced a new tracking technique based on roentgenographic techniques, that make use of x-rays to study joint kinematics. The authors adapted the technique so that the amount of radiation applied to the subjects was as low as possible. Spherical tantalum balls were inserted percutaneously on the shoulder. To identify the positions of the tantalum balls in the three bone-fixed coordinate systems a normal dose stereo exposure of the shoulder was made (Högfors et al., 1991). Afterwards, polynomial functions approximating the clavicular and scapular angles were calculated with the humeral angles considered as independent variables. The rhythm was described by this polynomial fit.

R. John Runciman (1993) also applied a different tracking method so that a 3-D analysis of the shoulder movement was possible. The author used an optoelectronic system, Vicon, which involved a set of infrared cameras and reflective markers. The cameras emit pulsed infrared light and monitor the reflection of this light from anything in its field of view. The markers are placed in specific body landmarks and will reflect the infrared light providing its location in space. With the combination of the individual measurements of each camera the three-dimentional coordinates of each marker can be calculated.
Shoulder kinematics is determined by creating lines between markers in order to represent the different body segments. Since the markers position are known, and the coordinates of the markers can be calculated as a function of time, it is possible to track body segment movement and consequently calculate joint angles through time.

The author only tested one movement, abduction in the frontal plane, calculating the moments at the glenohumeral joint and the scapula and clavicle kinematics.

Anglin & Wyss (2000) introduced a different kind of optoelectronic system, Optotrak, where active markers are used, i.e. the markers placed on the skin surface emit infrared light that are captured by cameras (Costigan et al., 1992). Using this method, functional movements were studied instead of just the standardized ones, such as flexion and abduction.

One of the main disadvantages of electromagnetic and optoelectronic systems is the fact that they need communication between emitter and receiver devices. Additionally, with electromagnetic systems, results are more prone to error when there is a ferromagnetic material close to the system.

1.3.3 Electromyography

Early studies on shoulder motion recognized the essential role of the muscles surrounding the shoulder joint to its stability and consequent healthy motion.

The glenohumeral joint has the greatest range of motion of all human body joints (Reinold et al., 2004). Consequently, the stability provided by the bony structure is limited, specially during motion. Along with the joint capsule, ligaments and glenoid labrum, the surrounding muscles are the main responsible for shoulder stability (Reinold et al., 2004; Struyf et al., 2014). As such, the study of the muscle activation patterns is very important when studying possibles causes of shoulder pathology.

The first studies using electromyography (EMG) used intramuscular electrode needles (Jobe et al., 1984; McMahon et al., 1996). McMahon et al. (1996) used this technique to compare a healthy population with a SIS one. To do so, the muscles studied were the subscapularis (upper and lower portions), supraspinatus, infraspinatus, rhomboid, serratus anterior, and trapezius (upper and lower portions). Abduction, scapular plane abduction, and forward flexion were performed over the range of motion and later divided into 30 degrees intervals. Finally, the activation of each muscle was normalized and compared between populations.

Intramuscular electrodes provide an accurate signal of a given muscle with little interference from the surrounding muscles. However they are not always the most practical solution in a clinical environment and they can migrate within the muscle during motion (Rajaratnam, 2014; Reinold et al., 2004).

Bandholm et al. (2006) used both intramuscular and surface electrodes to examine the effects of SIS on muscular function. Since each kind of electrode has different characteristics, a selection of electrodes was made depending on the muscle, in order to acquire the best results. Specifically, the surface electrodes were placed on the skin overlying the anterior and middle deltoid, upper and lower trapezius, latissimus dorsi, and serratus anterior muscles. Here, subjects performed isometric abductions, adductions, and internal and external rotations of the upper arm at maximal effort.

6
Despite intramuscular electrodes remaining the golden standard for EMG analysis, surface electrodes can also provide reliable data when used in the right conditions (Rajaratnam, 2014), avoiding the pain and discomfort caused by an invasive technique.

1.4 Contributions

The main contributes of this thesis are:

- The application of an optoelectronic tracking system along with sEMG, to identify and analyse pathological shoulder kinematics;
- The development of an acromion cluster that avoids interference between markers;
- The study of the main kinematic alterations on two different planes of motion, between subjects diagnosed with UST and a control group;
- The use of SHR to better characterise the differences between a pathological and healthy shoulder kinematics;

This work was presented on the 8th Congresso Nacional de Biomecânica, the American Congress of Rehabilitation Medicine 2018 and the 39th Congresso Nacional de Ortopedia e Traumatologia – SPOT 2019.

1.5 Thesis Organization

This thesis aims to evaluate the feasibility of using measurable kinematic variables and sEMG data as criteria for a more accurate and specific diagnosis of the RCT. As such, an experimental protocol was develop, in order to acquire kinematic and surface electromyography data of a Control Group and a Patient Group, diagnosed with Unilateral Supraspinatus Tendinopathy. Before describing this process, followed by its results and corresponding discussion, an literature review was done to understand how motion tracking and sEMG technologies have evolved and how important their impact is on contemporary clinical setting. Additionally, both the anatomy and biomechanics of the shoulder join are described, to provide the needed concepts to understand the shoulder kinematics and its muscle activation patter during arm elevation.

Chapter 1 corresponds to the current introduction where the motivation, scopes and objectives and contributions of this work are presented. Furthermore, a literature review of both clinical and computational methods to study shoulder kinematics, along with the techniques to acquire electromyography data is done. First, the traditional clinical methods used to diagnosed and follow shoulder pathologies are described and their limitations identified. Second, the evolution and the different technologies to track shoulder motion are presented, in order to show the continuous development of these technologies and their potentiality to be applied on a clinical environment. Finally, the traditional techniques on EMG are described.
Chapter 2 presents the anatomy of the shoulder, including the bones, joints and muscles that com-
pose this complex joint. Then, rotator cuff tendinopathy, a broader pathology diagnose of the UST, is
deﬁned and its causes and consequences presented. Finally, the consensus on healthy and pathological
shoulder biomechanics found on the literature, are described.

In Chapter 3, the Control and Pathological Group are described, followed by the experimental pro-
tocol, computational methods and data analysis. This chapter also includes the design process of an
acromial cluster, that is posteriorly mentioned when the instrumentalization of the subjects is described.

Chapter 4 contains the results of all processed data, dividing them in three main subsections. First,
the results of the cluster design are presented, justifying the choice of one of the models for the posterior
acquisition sessions. Second, the kinematic results are displayed, analysing the data on three different
stages. Firstly, the results of the CG, than the PG and finally the results of two subgroups subsequent of
the PG. On the third subsection, the EMG results are described following the same three stages of the
kinematic results.

Chapter 5 follows a similar organization of the three stages of the results description. In other words,
there are three subsections, covering first, the analysis and discussion of the CG results, comparing the
results of abduction and ﬂexion, both kinematic and EMG; second, the analysis of the PG relative to
the CG and also comparing contralateral shoulders on the PG; and third a discussion of the subgroup
results, relative to each other and to the CG again.

Chapter 6 has the main conclusions of this work including possible limitations, followed by the clinical
applications of the procedures here studied. Lastly, future developments that should be considered in
following research on shoulder kinematics alterations in populations with UST.
Chapter 2

Shoulder Anatomy and Biomechanics

2.1 Shoulder Anatomy

The shoulder joint is composed of four joints and three bones. Its complex structure allows for 6 degrees of freedom. However its stability becomes more intricate too. Contrary to the lower body joints, shoulder stability is mostly achieved by its muscles (Lugo et al., 2008). The coordinate action of all these shoulder components enables a wide range of movement, whilst also ensuring its stability. Nonetheless, this stability can be easily disturbed if one of the components fails. Given the complexity of the shoulder structure there are a variety of injuries or pathologies that can provoke shoulder instability and consequently pain or movement loss (Cutti & Veeger, 2009). Thus, it is essential to have a deep comprehension of the shoulder anatomy and physiology in order to understand pathology mechanisms and how to develop effective treatment plans.

2.1.1 Bones

The bone framework of the shoulder consists of the clavicle and scapula, which form the shoulder girdle, and the proximal end of the humerus.

The clavicle, presented on Figure 2.1, is an S-shape long bone, palpable through its whole length. It is the only bony attachment between the trunk and the upper body. Medially, the clavicule has a rounded end, which is called the sternal end, that articulates with the manubrium of the sternum to form the sternoclavicular joint. Laterally, it has a broad, flat, which is called the lateral end, the acromial end, that articulates with the acromion of the scapula to form the acromioclavicular joint. The clavicle has several points of ligament attachment, namely the conoid tubercle on the inferior surface of the lateral end of the bone, which attaches the clavicle and scapula through the conoid ligament and the trapezoid line which is a point of attachment for the costoclavicular ligament, that attaches the clavicle and first rib (Drake et al., 2009; Tortora & Derrickson, 2017).

The scapula, presented on Figure 2.2, is a large, triangular, flat bone positioned superiorly and posteriorly on the thorax at the level of the first seven ribs (Tortora & Derrickson, 2017; Rouvière & Delmas, 2005). At the posterior surface of the scapula a prominent ridge called the spine runs diagonally. The
The lateral end of the spine projects as a flattened, expanded process called the acromion. Superior and inferior to the spine on the posterior surface of the scapula are two fossae: the supraspinous fossa is a surface of attachment for the supraspinatus muscle of the shoulder, and the infraspinous fossa serves as a surface of attachment for the infraspinatus muscle of the shoulder. On the anterior surface of the scapula is a broad concavity called the subscapular fossa, a surface of attachment for the subscapularis muscle. Inferior to the acromion there is the glenoid cavity, that accepts the humerus forming the gleno-humeral joint. At the anterior side of the scapula there is the coracoid process, defined by a thick curved process that projects forward and slightly laterally. This bone has three borders, the medial (vertebral) border, closer to the vertebral column; lateral (axillary) border, closer to the arm; and the superior border. Lateral and medial borders join at the inferior angle (Tortora & Derrickson, 2017; Pronk, 1991).

The humerus is the largest bone of the upper limb. It articulates proximally with the scapula and distally with two bones, the ulna and the radius, to form the elbow joint (Tortora & Derrickson, 2017). The proximal end of the humerus has a rounded head that connects to the glenoid cavity on the scapula, forming the gleno-humeral joint. The distal humerus presents two articular surfaces for articulation with the radius and ulna. Projecting on either side of the distal end of the humerus there are the medial epicondyle and lateral epicondyle (Tortora & Derrickson, 2017; Pronk, 1991). A representation of the articulation between the humerus and the scapula can be observed on Figure 2.3.
2.1.2 Joints

A joint is a point of contact between two bones, between bone and cartilage, or between bone and teeth. Its shape and structure dictate the joint’s strength and mobility. Joints can be characterized using two criteria: (1) do they have a synovial cavity, i.e., a space between the articulating bones; and (2) what is the type of connective tissue that binds the bones together (Tortora & Derrickson, 2017).

Joints with no synovial cavity tend to have little or no movement, because they are held together by dense connective tissue or cartilage. On the other hand joints that do have synovial cavity have more mobility. However they are also less stable (Tortora & Derrickson, 2017).

The shoulder girdle is composed of four joints, three anatomical and one physiological joint.

The sternoclavicular (SC) joint supports the connection of the arms and shoulders to the main skeleton on the front of the chest. It is a small synoval joint that results from the articulation between the sternal end of the clavicle and the manubrium and the first costal cartilages. It is divided in two compartments by an articular disc that is firmly attached to several ligaments. This structure allows for great joint strength. The SC joint can also be significantly mobile, reaching 25°-30° of anteriorly or posteriorly motion during elevation in the frontal plane (Moore et al., 2014).

The acromioclavicular (AC) joint is a synoval joint that results from the articulation of the clavicle with the acromion. Its stability is mainly achieved by the coracoclavicular ligament, that unites the coracoid process of the scapula to the clavicle, anchoring the clavicle to the coracoid process. Along with this ligament the AC joint is also stabilized superiorly by fibres of the trapezius muscle and the superior acromioclavicular ligament, and inferiorly by the inferior acromioclavicular ligament (Tortora & Derrickson, 2017). The AC joint allows for a complementary motion of the scapulothoracic joint by allowing the scapula to continue to rotate beyond its individual range (Pronk, 1991).

The scapulothoracic joint (ST) is physiological (false) joint, formed where the shoulder blade glides against the thorax (the rib cage). It is a very important joint given that its contribution to the shoulder movement allows it to rotate beyond 120° of humerothoracic elevation. Additionally, the ST joint also has a role on enhancing glenohumeral stability. From all muscles responsible for stabilizing and providing
motion to the scapula, the most important are the serratus anterior, which maintains the medial angle against the chest wall, and the trapezius, which helps to rotate and elevate the scapula synchronously with glenohumeral motion (Terry & Chopp, 2000; Lovern, 2010).

The motions at the scapulothoracic joint are described by three rotations, upward and downward rotation, protraction and retraction, and anterior and posterior tilt, rotations represented on Figure 2.4, and two translations, elevation and depression, and abduction and adduction (Terry & Chopp, 2000).

![Figure 2.4: Representation of the scapulothoracic joint rotations. Adapted from (Ludewig et al., 2009a)](image)

The glenohumeral joint (GH) is a “ball and socket” synovial joint, where the humeral head is the ball and the glenoid cavity the socket. This joint allows a wide range of motion. However, it is quite unstable. This instability is provoked by the small surface of the glenoid cavity when compared to the humeral head. To increase GH stability there is a fibrocartilaginous structure named glenoid labrum, that deepens the glenoid cavity. Due to the small contact area between the two articulating surfaces, the GH has a large range of motion capable of flexion/extension, abduction/adduction, internal/external rotation and circumduction. This joint accounts for 120° of full arm elevation (Lovern, 2010; Veeger & van der Helm, 2007).

It is also important to mention the subacromial space, as it plays an essential role on a healthy shoulder rotation. It is the space delimited above by the coracoacromial arc and below by the humeral head, by the tendons of the rotator cuff and of the long head of the biceps. Thus it facilitates movement of the supraspinatus tendon under the coracoacromial arch and of the deltoid over the joint capsule of the glenohumeral joint and the greater tubercle of the humerus (Moore et al., 2014).

### 2.1.3 Muscles

There are nine muscles that cross the shoulder joint, all of them originating on the scapula except for the pectoralis major and latissimus dorsi. These two muscles are called axial muscles, since they originate on the axial skeleton. All nine muscles work alongside to provide joint stability and a wide range of motion, Tables 2.1 and 2.2 describes the origin, insertion and action of each muscle.

Four of the nine muscles make up the Rotator Cuff (RC), namely the Supraspinatus, Infraspinatus, Teres minor, and Subscapularis. They form the RC, a continuous cuff around the humeral head, by intersecting and blending with the adjacent tendons and the subjacent capsule (Lewis, 2009). This
complex of muscles is an essential component of the shoulder joint, and the most common source of injury of the musculoskeletal system (Dela Rosa et al., 2001). The RC has three main functions: it rotates the humerus with respect to the scapula; it acts as a dynamic stabilizer of the glenohumeral joint, by counteracting non–rotator cuff muscles, e.g. deltoid or latissimus dorsi, in order to keep the humeral head in place; and it maintains the integrity of the joint space, preventing leaks of the synovial fluid, and consequently keeping the joint cartilage healthy and preventing cuff tear arthropathy (Dela Rosa et al., 2001; Lewis, 2009).

2.2 Rotator Cuff Tendinopathy

RCT is generally characterized by pain and weakness occurring at the rotator cuff muscles, during common shoulder movements such as elevation and external rotation, as result of excessive external loads (Lewis et al., 2015). It is considered the most common shoulder disorder, as it may affect up to 30% of the population (Abat et al., 2017). However there is a lack of consensus on its aetiology and the criteria to achieve an accurate diagnosis (Michener et al., 2003; De Witte et al., 2011).

There is high uncertainty on the causes of RCT, as well as on the pain experienced by the diagnosed subjects. Besides the lack of sensitivity of the traditional clinical tests, imaging methods do not provide a fully reliable diagnosis, since there is not a direct association between symptoms and structural failure. Additionally, the inflammatory role of the disorder is not completely understood, since the concentration of inflammatory components is not consistent throughout different studies (Lewis et al., 2015).

Classically, RCT mechanisms have been described as extrinsic, intrinsic or a combination of both. Extrinsic factors, first described by Neer (1983), represent a compression on the RC tendons and associated tissues within the subacromial space, under the anterior aspect of the acromion or surrounding structures. Neer (1983) first labelled this mechanism as impingement syndrome, and divided it in three stages, Edema and Hemorrhage (Stage I), Fibrosis and Tendinitis (Stage II) and Bone Spurs and Tendon Rupture (Stage III). Stage I is normally associated with excessive practice of overhead sports or work activities, typically in people with less than 25 years old. Stage II is a consequence of repetitive episodes of inflammation, causing fibrosis and thickening of the bursa. Here, the population affected is mainly between 25 and 40 years old. Finally, the last and more severe stage of impingement syndrome is characterized by an incomplete or complete tear of the rotator cuff, biceps lesion, and bone alterations of the anterior acromion and greater tuberosity.

Compression of the subacromial space occurs due to anatomical factors, biomechanical factors, or a combination of both. Anatomically, there are several factors that contribute to a predisposition for subacromial compression and a lower success rate in treatments. Variations in shape or orientation of the slope/angle of the acromion, subacromial joint spurs and AC joint spurs can lead to tendinopathy (Factor & Dale, 2014).

Idrissi et al. (2017) studied the effect of acromial morphology on rotator cuff tears and concluded that, acromion shape, lateral extension and angle, play an important role on the development of RC pathology. Additionally, changes in acromion morphology have been associated with increasing age.
(Julie et al., 2012), which can justify why there is a higher incidence of RC tears with age (Maffulli et al., 2011).

Table 2.1: Shoulder muscles description. The graphical representation of bones and muscles was obtained from the Anatronica, 3D Interactive Anatomy (2010-2011)

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Info</th>
</tr>
</thead>
</table>
| **Deltoid** | **Origin**: The anterior fibres arise from the anterior border and upper surface of the lateral third of the clavicle, the lateral fibres from the acromion of the scapula, and the posterior fibres from the spine of the scapula.  
**Insertion**: Deltoid tuberosity of the humerus  
**Action**: The anterior fibres flex and medially rotate, the lateral fibres abduct, and the posterior fibres extend and laterally rotate the arm at the shoulder joint. |
| **Trapezius** | **Origin**: Superior nuchal line of occipital line, ligamentum nuchae, and spines of the C7 to T12 vertebrae.  
**Insertion**: Posterior border of the lateral third of the clavicle, acromion process, and spine of the scapula  
**Action**: The superior fibres elevate the scapula, the middle fibres retract the scapula and the inferior fibres depress the scapula; the superior and inferior fibres together rotate the scapula upwardly; as a whole, also contributes to the stabilisation of the scapula. |
| **Serratus** | **Origin**: Usually from the lateral surface of the first to eighth ribs  
**Insertion**: Costal surface of the medial border of the scapula  
**Action**: Protracts the scapula and rotates it upwardly; also stabilises the scapula for arm movements |
| **Pectoralis major** | **Origin**: The superior fibres arise from the anterior surface of the medial half of the clavicle, the middle fibres from the anterior surface of the sternum and the inferior fibres from the superior six costal cartilages.  
**Insertion**: Lateral lip of the bicipital groove of the humerus  
**Action**: The superior fibres, often called clavicular portion, flex the arm while the middle and inferior fibres, often called sternocostal portion, extend the flexed arm. As a whole, it adducts and medially rotates the arm at the shoulder joint. |
<table>
<thead>
<tr>
<th>Muscles</th>
<th>Info</th>
</tr>
</thead>
</table>
| Infraspinatus | **Origin:** Infraspinous fossa of the scapula.  
|             | **Insertion:** Greater tubercle of the humerus  
|             | **Action:** Laterally rotates the arm and stabilises the shoulder joint. |
| Latissimus Dorsi | **Origin:** Spines of T7–L5, lumbar vertebrae, crests of sacrum and ilium, ribs 9–12 via thoracolumbar fascia.  
|             | **Insertion:** Intertubercular sulcus of humerus.  
|             | **Action:** Extends, adducts, and medially rotates arm at shoulder joint; draws arm inferiorly and posteriorly. RMA: Elevates vertebral column and torso. |
| Subscapularis | **Origin:** Subscapular fossa of scapula.  
|             | **Insertion:** Lesser tubercle of humerus.  
|             | **Action:** Medially rotates arm at shoulder joint. |
| Coracobrachialis | **Origin:** Coracoid process of scapula.  
|             | **Insertion:** Middle of medial surface of shaft of humerus.  
|             | **Action:** Flexes and adducts arm at shoulder joint |
| Supraspinatus | **Origin:** Supraspinous fossa of scapula.  
|             | **Insertion:** Greater tubercle of humerus.  
|             | **Action:** Assists deltoid muscle in abducting arm at shoulder joint. |
Degenerative changes in AC joint can also be a factor on RCT. Over time osteophytes can form on the inferior aspect of the distal clavicle, decreasing the supraspinatus outlet and consequently exposing underlying soft tissue to increased mechanical wear and degeneration. These anatomical alterations can lead to RCT.

The biomechanical factors that may lead to RCT, are abnormal posture, muscle deficit, or aberrant scapular and humeral kinematics. Scapular kinematic abnormalities have been identified in patients with RCT. In this cases altered motion may contribute to a reduction of subacromial space leading to RC tendon compression. However, there are contradictory results regarding the scapular kinematics relative to RCT. Codman (1934a) suggested that there are no significant differences between RCT patients and healthy controls. Nonetheless, a small subgroup of patients, with a difference of >2.5 standard deviations from the mean in normal controls showed significant differences in joint rotations, suggesting that more obvious scapular dyskinesis may contribute to an extrinsic mechanism of RC tendinopathy.

The affected RC muscles in RCT are the supraspinatus, infraspinatus and teres minor in the posterior part of the shoulder and the subscapularis in the anterior part. These muscles are responsible for a variety of upper extremity movements, and for the stability of the shoulder joint at the same time. Hence, aberrant activity or muscle deficit may be linked to abnormal scapular kinematics, leading to RCT. Additionally, soft tissue tightness and an abnormal posture may change the scapula positioning, also affecting its kinematics (Myers et al., 2009; Lewis et al., 2015; Lewis, 2009; Abat et al., 2017).

Intrinsic mechanisms relate to factors that directly influence tendon health and quality, including ageing (Yamamoto et al., 2010), genetics, vascular changes, and altered loading. Excessive loading is one of the main reasons for developing RCT (Lewis et al., 2015).

Ageing has been proven to have negative effects on the tendons biomechanical properties (Maffulli et al., 2011). There is a decreased toe-region on the stress strain curve, decreased elasticity, and decreased overall tensile strength of tendons with age. Additionally, calcification, fibrovascular proliferation, degeneration are more present in elderly patients too (Nho et al., 2008).

The role of vascularity as an intrinsic mechanism is not completely understood. Codman (1934b) first described an area within the supraspinatus with decreased vascularity, as a critical zone, since it was considered the most common site for RC tendon injury. However, more recent studies have challenged this theory by suggesting that there is no such critical area (Levy et al., 2008), or that the hypovascularity is only limited to the articular side and not the bursal side of the tendon (Rudzki et al., 2008).
2.3 Shoulder Biomechanics

2.3.1 Healthy

Quantifying shoulder kinematics is an intricate field of study, mainly due to the complex shoulder anatomy and the large range of motions this joint can perform. Therefore, it is essential to accurately measure the kinematics of all the shoulder joints and understand how they interact with each other.

Before analysing what is known about healthy shoulder kinematics it is important to define the planes of motion studied in the literature, and consequently the ones analysed in this work. When studying pure shoulder movements, these may be described by the anatomical planes in which the arm elevates in, namely the frontal and sagittal plane. The frontal, or coronal, plane divides the body into anterior and posterior halves. When the arm elevates in this plane, the movement is called abduction. The sagittal, or medial, plane crosses the body from front to back and divides it into right and left sections. Here, when the arm elevates, the movement is referred to as flexion. Additionally, when studying shoulder kinematics, most studies consider the scapular plane, which corresponds to a plane of the scapula in its resting position, more specifically at 30° from the frontal plane.

![Figure 2.5: Representation of the three Anatomical Planes](http://picdeer.com/media/1880774086800480859_7579525751)

Most studies of shoulder biomechanics have focused on healthy shoulder kinematics, measuring clavicular, scapular, and humeral motion during arm elevation, as well as the scapulohumeral rhythm (SHR). This last variable was introduced by Codman (1934a), and is used to describe the relative motion of the scapula to the humerus, during arm elevation. Moreover, it represents the ratio of the glenohumeral movement to the scapulothoracic movement during arm elevation. Tables 2.3 displays the SHR values found in the literature for the arm movement as a whole.

Inman (1944) described the combined motion of the different bony structures of the shoulder, proving that although the four joints within the shoulder can move independently, in a normal healthy movement they rotate simultaneously.

Scapular position and control during motion are an essential component of a healthy movement. In healthy individuals it is known that during elevation of the arm, the scapula should upwardly rotate and

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1http://picdeer.com/media/1880774086800480859_7579525751 consulted at March 27th
posteriorly tilt on the thorax (Ludewig et al., 2009b; Habechian et al., 2013). However, it is important to note that although the scapula kinematics described previously are consistent across planes of elevation, it is not the same for its pattern (Struyf et al., 2011). For instance, during shoulder elevation, the scapula is more upwardly rotated at 60° in the frontal plane, when compared to the other two planes. Scapular internal rotation is less consistent during humeral elevation, across subjects, planes of elevation, and point in the range of motion of elevation (Ludewig et al., 2009b; Habechian et al., 2013). Scapulothoracic tilt (STT) has not shown significant differences within the three planes of humeral elevation (Struyf et al., 2011).

Table 2.3: Healthy Subjects - SHR for the whole movement

<table>
<thead>
<tr>
<th>Article</th>
<th>Plane of Motion</th>
<th>SHR</th>
<th>Elevation interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yoshizaki et al. (2009)</td>
<td>Scapular and sagittal</td>
<td>3.0-4.0</td>
<td>[Min - Max]</td>
</tr>
<tr>
<td>Ludewig &amp; Cook (2000)</td>
<td>Scapular plane</td>
<td>2.3-3</td>
<td>[30° - 120°]</td>
</tr>
<tr>
<td>McClure et al. (2001)</td>
<td>Scapular plane</td>
<td>1.7</td>
<td>[Min - Max]</td>
</tr>
<tr>
<td>Matsuki et al. (2011)</td>
<td>Scapular plane</td>
<td>2.6-2.7</td>
<td>[Min - Max]</td>
</tr>
<tr>
<td>Braman (2010)</td>
<td>Without a controlled plane</td>
<td>2.3</td>
<td>Without a controlled elevation angle</td>
</tr>
</tbody>
</table>

Inman (1944) also introduced the value of the SHR to be a 2:1 ratio, meaning that for each 2 degrees of the glenohumeral joint rotation the scapulothoracic joint would rotate 1 degree. Since then, more studies have measured the SHR, showing a lack of consensus on this value as it can be observed on Table 2.3. On one hand, different planes of motion have been tested and it was proven that they can influence the SHR (Struyf et al., 2011). Nonetheless, within the same plane of motion, for instance in the scapular plane, SHR can range from 1.7 to 3.4 (Yoshizaki et al., 2009; Ludewig & Cook, 2000; Struyf et al., 2011; Matsuki et al., 2011; Braman, 2010). On the other hand, the incremental motion of the scapulothoracic and glenohumeral joint can vary within the same motion (Inman, 1944; Braman, 2010; Mell et al., 2005; Hosseinimehr et al., 2015; Turgut et al., 2016b; Scibek et al., 2008). To take this in consideration the SHR can be calculated in elevation intervals. Here, following the work of Braman (2010) each movement was divided in intervals of 30° increment: [Min,30°]; [30°,60°]; [60°,90°]; [90°,120°] and [Min,120°]. Table 2.4 displays the SHR values calculated for the arm movement divided in different elevation intervals.

In addition to kinematic analysis, electromyography (EMG) early studies (Inman, 1944) have also been used to study healthy and/or pathological shoulder motion. More recently, different authors have study pure motions as abduction, flexion and internal/external rotation (Heuberer & Kranzl, 2015; Wickham et al., 2010; Reinold et al., 2004).

Despite the several studies that have focused on pure movements as abduction and flexion, there
is still not full consensus on the role of each muscle during shoulder movement. Heuberer & Kranzl (2015) found that abduction in the healthy shoulder was mainly performed by the supraspinatus and anterior and middle deltoid muscle, a finding that was similar to other studies (Townsend et al., 1991; Alpert et al., 2000). The subscapularis was also found to play an important role during abduction, stabilising the shoulder anteriorly by compressing the humeral head in the glenoid fossa (Wickham et al., 2010) and also showing potential abduction capability. Additionally, the infraspinatus shared the load with the supraspinatus, and the trapezius also showed a stabilising role, promoting scapula movement throughout the whole abduction exercise (Wickham et al., 2010).

Table 2.4: Healthy Subjects - SHR for divided movement

<table>
<thead>
<tr>
<th>Article</th>
<th>Plane of Motion</th>
<th>Elevation interval</th>
<th>SHR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hosseinimehr et al. (2015)</td>
<td>Frontal plane</td>
<td>45°</td>
<td>8.2/8.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>90°</td>
<td>6.2/6.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>135°</td>
<td>4.2/4.4</td>
</tr>
<tr>
<td>Mell et al. (2005)</td>
<td>Scapular plane</td>
<td>Phase I</td>
<td>2.13 = (1/0.47)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Phase II</td>
<td>2.5 = (1/0.36)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Phase III</td>
<td>2.78 = (1/0.36)</td>
</tr>
<tr>
<td>Mell et al. (2005)</td>
<td>Sagittal plane</td>
<td>Phase I</td>
<td>3.45 = (1/0.29)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Phase II</td>
<td>2.56 = (1/0.39)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Phase III</td>
<td>2.44 = (1/0.41)</td>
</tr>
<tr>
<td>Braman (2010)</td>
<td>None</td>
<td>Min-Max</td>
<td>2.37 ± 0.44</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30°-Max</td>
<td>2.31 ± 0.50</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Min-30°</td>
<td>3.71 ± 2.15</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30°-60°</td>
<td>2.36 ± 0.75</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60°-90°</td>
<td>2.71 ± 0.85</td>
</tr>
<tr>
<td></td>
<td></td>
<td>90°-120°</td>
<td>2.50 ± 0.90</td>
</tr>
</tbody>
</table>

Phase I, II and III - Mell et al. (2005) divided the movement in three equal phases based on minimum and maximum normalized humeral elevation.

Wickham et al. (2010) reached similar conclusions. However in his work a temporal analysis to muscle activation was also done. The author found that supraspinatus, middle trapezius and middle deltoid were on average activated before the movement began and that the lower and upper subscapularis and infraspinatus had a later activation. It was suggested that the early activation of the supraspinatus confirmed its role as a abduction initiator, and that the middle trapezius would have a stabilizer role in order to provide an initial stable base to allow the scapulo-humeral muscles to generate force. The late activation of the lower and upper subscapularis and infraspinatus suggests that their stabilizing role is only needed starting from 10° of humerothoracic elevation angle. Additionally, serratus anterior and upper trapezius were also found to have a dual role on arm elevation, as upward rotators and stabilisers.

Heuberer & Kranzl (2015) also studied flexion and found that it is performed mainly by the supraspinatus and anterior deltoid (AD) (Townsend et al., 1991). All three trapezius subparts promoted scapulothoracic movement with increasing forward flexion, whereas serratus anterior and the infraspinatus seemed
to have a stabilization role during the movement.

### 2.3.2 Symptomatic

Scapular and glenohumeral kinematics are altered in unstable or pathological shoulders (Ogston & Ludewig, 2007; Mell et al., 2005). However, the alterations described in the literature are not unanimous. It has been described that this kinematic alterations can be compensatory or contributory to an impingement mechanism. In the first case, there can be a adaptation to a painful shoulder by increasing the scapulothoracic joint rotation in order to reduce the requirement for elevation at the glenohumeral joint (Mell et al., 2005) and/or increase the subacromial space (Mcclure et al., 2006). Regarding a possible contributory effect to the pathology, since the subacromial space is relatively small any changes in shoulder kinematics could result in compression of the subacromial tissues during glenohumeral elevation (Michener et al., 2003).

Nonetheless, differences in shoulder kinematics have been found between healthy and symptomatic shoulders. Most studies tend to focus on scapular kinematics. Although there is some variations in their results, the majority found that symptomatic shoulders tend to have decreased scapular upward rotation, posterior tilt and external rotation (Ludewig & Cook, 2000; Michener et al., 2003; Turgut et al., 2016a; Lawrence et al., 2014; Lefèvre-Colau et al., 2018). Regarding glenohumeral kinematics, an increase on glenohumeral elevation as been observed (Lawrence et al., 2014).

Despite the significant number of studies on symptomatic subjects, there are very few that use the SHR to characterize kinematic differences between populations (Graichen et al., 2001; Hallström & Kärrholm, 2009; Mell et al., 2005). Considering the kinematic alterations previously described, an increase in SHR for the symptomatic population may be expected. This result has been proven by Mell et al. (2005). However, Graichen et al. (2001); Hallström & Kärrholm (2009) found no significant differences between symptomatic and asymptomatic subjects. Despite the lack of studies including the SHR as a result, an increase on SHR for the symptomatic population should be expected, since these kinematic results have been corroborated by a higher number of studies.

Studying scapular and glenohumeral kinematics is essential to understand the overall shoulder movement. However, there is a dependency between both joints that can change as an adaptation to a given pathology (Ogston & Ludewig, 2007; Mcclure et al., 2006). On the other hand, alterations in their relative movement can be the cause of impingement or other pathologies (Michener et al., 2003; Hbert et al., 2002). Hence, calculating the SHR is also essential for an accurate and complete study of shoulder kinematics.

One of the factors that may lead to RCT is a lack of coordination and neuromuscular balance between the RC muscles, which includes the supraspinatus (SS), infraspinatus (IS), subscapularis (SS), and teres minor (TM) (de Oliveira et al., 2017). Proper RC musculature activation is crucial for maintaining a stable shoulder motion. Reddy et al. (2000) suggested that on a population with chronic subacromial impingement, there is a lack of balance between muscle forces, leading to inadequate humeral head depression during elevation, especially during the initiation of movement. If the humeral head is not adequately
depressed, the head could rise during elevation, exacerbating the symptoms of impingement. In this study the overall decreased activity of the deltoid and rotator cuff muscles during abduction was found mainly in the early arcs of motion.

Myers et al. (2009) measured rotator cuff (supraspinatus, infraspinatus and subscapularis) coactivation and middle deltoid muscle activation during elevation of the arm in subjects with shoulder impingement and control subjects. Similar results to the ones described above were presented, where subjects with impingement showed significantly decreased rotator cuff coactivation and increased middle deltoid activation on the initial stages of the motion.

When studying muscle activation onsets and patterns between healthy subjects and subjects with RCT, most studies only consider scapulothoracic muscles, namely upper trapezius (UP), medial trapezius (MU), lower trapezius (LT) and serratus anterior (SA). However, there is no consensus regarding their results. Leong et al. (2017) studied the effects of RCT on muscle activity onset and kinematics of scapula and found that the symptomatic subjects had all muscles (UP, MU, LT and SA) activated at unison to stabilize the scapula. The authors also found that these same subjects showed deficits in the activity onset of scapular muscles when compared to the asymptomatic subjects. The LT and SA were activated slower. In opposition, Moraes et al. (2008) found no significant differences on muscle activation between the two populations, demonstrating that the muscles were recruited in the same sequence, starting by the UT, followed by SA, MT, and LT.
Chapter 3

Experimental and Computational Methods

3.1 Participants

Despite the increasing interest on shoulder kinematics, there is still no full consensus on what should be expected to be found between control and pathological movements. In order to tackle this inconsistency on previous studies, a control group (CG) was created, with the following inclusion criteria:

1. Did not present shoulder pain or pathology history;
2. Did not practice frequently overhead movements, as sports or work related activities;
3. Did not present overweight BMI indexes;

The criteria described are mainly focused on guaranteeing an homogeneous group, with no possible kinematic alteration factors. Alterations in scapular and glenohumeral kinematics are not always related with shoulder pathology (Kibler, 1998; Kebaetse et al., 1999; Gupta et al., 2013), hence it is important to identify and use these benign factors of kinematic alterations as an exclusion criteria.

Repetitive overhead movements and excessive body weight can translate into shoulder kinematic alterations. On one hand, overhead athletes have shown asymmetrical shoulder kinematics, due to muscle adaptations that consequently alter scapular kinematics (Hosseinimehr et al., 2015; Oyama et al., 2008). On the other hand, increased arm weight, due to body overweight, can also lead to kinematic alterations. There is an increased scapula upward rotation, as a way to use momentum to overcome the greater inertia of an arm with increased mass (Gupta et al., 2013).

The PG, was composed of male or female patients, observed at the External Consultation of Rheumatological Rehabilitation of the Physical Medicine and Rehabilitation Specialty of the Centro Hospitalar Universitário de Lisboa Central and fulfilled the following criteria:

1. Had more than 18 and less than 80 years old;
2. Had clinical and imaging diagnosis (by articular ultrasound) of tendinopathy/tendinitis of the supraspinatus, without clinical or imagiologic evidence of total or partial rupture;

3. Did not present history of previous surgery to the shoulder;

4. Did not present personal ancestors of pathology of the central or peripheral nervous system with reaching the scapular girdle.

All subjects provided their informed consent to participate in this study, approved by the scientific committee of the Service of Physical Medicine and Rehabilitation of the Centro Hospitalar Universitário de Lisboa Central, E.P.E..

### 3.2 Instrumentation

#### 3.2.1 Kinematics

To evaluate 3D-kinematics motion capture was accomplished using the Qualysis opto-electronic measurement system comprising 14 digital infrared cameras (Pro Reflex MCU 1000), set at a sampling frequency of 100 Hz, 22 retro-reflective markers (with a diameter of 19 mm) and two marker clusters per arm.

The retro-reflective markers were attached to the bony landmarks of the thorax, clavicle, scapula, humerus, and forearm, of each subject’s dominant arm, for the control group, and of both arms, for the PG, as recommended by the ISB (Wu et al., 2005).

Table 3.1 describes and Figure 3.1 shows all bony landmarks to be identified.

Figure 3.1: Posterior, in (a), lateral (b) and anterior, in (c), views of the upper limb bony landmarks.
Table 3.1: Anatomical landmarks proposed by the ISB

<table>
<thead>
<tr>
<th>Bone</th>
<th>Bony landmark</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thorax</td>
<td>C7</td>
<td>Spineous process of the seventh cervical vertebra</td>
</tr>
<tr>
<td></td>
<td>T8</td>
<td>Spineous process of the eighth thoracic vertebra</td>
</tr>
<tr>
<td></td>
<td>IJ</td>
<td>Deepest point of Incisura Jugularis</td>
</tr>
<tr>
<td></td>
<td>PX</td>
<td>Processus Xiphoideus, most caudal point on the sternum</td>
</tr>
<tr>
<td>Clavicle</td>
<td>SC</td>
<td>Most ventral point on the SC joint</td>
</tr>
<tr>
<td></td>
<td>AC</td>
<td>Most dorsal point on the AC joint</td>
</tr>
<tr>
<td>Scapula</td>
<td>TS</td>
<td>Trigonium Spinae, the midpoint of the triangular surface on the medial border of the scapula in line with the scapular spine</td>
</tr>
<tr>
<td></td>
<td>AI</td>
<td>Angulus Inferior, most caudal point of the scapula</td>
</tr>
<tr>
<td></td>
<td>AA</td>
<td>Angulus Acromialis, most laterodorsal point of the scapula</td>
</tr>
<tr>
<td></td>
<td>PC</td>
<td>Most ventral point of processus coracoideus</td>
</tr>
<tr>
<td>Humerus</td>
<td>GH</td>
<td>GH rotation centre (estimated)</td>
</tr>
<tr>
<td></td>
<td>EL</td>
<td>Most caudal point on the lateral epicondyle</td>
</tr>
<tr>
<td></td>
<td>EM</td>
<td>Most caudal point on the medial epicondyle</td>
</tr>
<tr>
<td>Forearm</td>
<td>RS</td>
<td>Most caudal–lateral point on the radial styloid</td>
</tr>
<tr>
<td></td>
<td>US</td>
<td>Most caudal–medial point on the ulnar styloid</td>
</tr>
<tr>
<td>NOVO</td>
<td></td>
<td>Replacement of IJ</td>
</tr>
</tbody>
</table>

To describe the 3D motion of a bone, ideally 3 markers are needed. However, not all bones have 3 bony landmarks, so markers from different bones have to be used to define them, consequently creating a position-dependency. Nonetheless, despite 3 markers per bone are enough to provide a representation of any bone motion, in some cases, the error associated to motion acquisition may be too high to provide reliable data. That is the case of the scapula, since it moves with respect to the skin causing a displacement between the markers and the corresponding anatomical landmarks as large as 2 cm (de Groot et al., 1998). In order to improve the tracking accuracy of the scapula, a dynamical tracking methodology was used, following the work of Shaheen et al. (2010), which shows acceptable accuracy up to 120° of arm elevation. To do so, a passive four-marker cluster made of copolymer polypropylene, covered with four retro-reflective markers, was placed laterally on the upper arm and held in position using elastic bands, as depicted in Figure 3.1 (arm cluster).

A second passive marker, the acromial cluster, was placed following the acromial tracker method (Shaheen et al., 2010), at the meeting point between the acromion and the scapular spine, as represented in Figure 3.1. Because the cluster used by Shaheen et al. (2010) presented very high noise interference, a custom made acromioclavicular was design based on the original one, with variations on the length and inclination of the bars holding the markers. Further explanations on the construction and testing of the acromial cluster will be present in Section 3.3.

The use of clusters rather than single markers and their placement at sites known to experience minimal overlying skin movement, minimises skin movement artefacts associated with standard motion analysis techniques (Cappozzo et al., 1995). Nonetheless, it is important to note that the acromial cluster...
is more susceptible to errors, since its accuracy is highly dependent on the right placement. In order to minimize the errors of the acromial tracker method, the cluster was placed accordingly to the study of Shaheen et al. (2010) and calibrated for the range of movement being measured.

### 3.2.2 sEMG

sEMG was recorded synchronously with 3D motion acquisition using Delsys’ Myomonitor III system for 8 muscles in the Control group: AD, medial deltoid (MD) and posterior deltoid (PD), superior trapezius (ST) and inferior trapezius (IT), SA, pectoralis major (PM) and IS; and 5 muscles in the pathological group: AD, MD and PD, SA and IS. Tables 3.2 and 3.3 summarizes the muscles analysed on each group.

The MOCAP system was used to triggered and synchronize kinematics and sEMG data. For the Pathological group less muscles were considered in order to decrease the instrumentation time, since on this group both arms would be tested. The muscles chosen were the ones where it was expected to find more notable differences given the pathology considered in this study.

Before placement of the surface electrodes the skin was abraded and defatted to decrease skin impedance. The electrodes were placed with an inter electrode distance of 20 mm, orientation parallel to the muscle fibers and with a (double sided) tape fixation method, as recommended by SENIAM 1. The reference electrode was placed on the elbow.

Table 3.2: Muscles Analysed on each Group. X: Muscle analysed; O: Muscle not analysed

<table>
<thead>
<tr>
<th></th>
<th>Anterior Deltoid</th>
<th>Medial Deltoid</th>
<th>Posterior Deltoid</th>
<th>Serratus Anterior</th>
<th>Infraspinatus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control Group</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Pathological Group</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>

Table 3.3: Muscles Analysed on each Group (Continued). X: Muscle analysed; O: Muscle not analysed

<table>
<thead>
<tr>
<th></th>
<th>Infraspinatus</th>
<th>Superior Trapezius</th>
<th>Inferior Trapezius</th>
<th>Pectoralis Major</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control Group</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Pathological Group</td>
<td>X</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
</tbody>
</table>

### 3.3 Acromial Cluster Design

The acromial cluster, shown in Figure 3.2, was designed using SolidWorks, and is the result of a series of tests with 16 different cluster designs. Each one had a base to be in contact with skin and three bars, each with a circular base at the top, where the markers would be attached.

1SENIAM: http://www.seniam.org/
The main objective of each design change was to decrease the interferences between markers, without causing instability on the cluster. The end result was a cluster with different lengths and inclinations between bars with strategic circular base angles, so that each marker would be shielded from the others’ reflections.

The tests were done in three series. The first series included 5 versions of the clusters, the second 9 and the last one 2. Each following series improved on the previous designs until the final one was chosen. The alterations between designs were in the length and inclinations of the bars, and the angle of the circular bases at the top of the bars. A MJP3600 printer of 3DS, with multijet technology, was used to print all cluster designs with visijet crystel.

The description of all cluster designs is in Table 3.4. If the circular base angle as a “No” it means that the bars did not have a circular base.

Table 3.4: Cluster designs characteristics

<table>
<thead>
<tr>
<th>Cluster</th>
<th>Length [cm]</th>
<th>Inclination [°]</th>
<th>Circular Base Angle [°]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bars</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>1</td>
<td>2.5</td>
<td>2.5</td>
<td>2.5</td>
</tr>
<tr>
<td>2</td>
<td>2.5</td>
<td>2.5</td>
<td>2.5</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>6</td>
<td>4.33</td>
<td>3.06</td>
<td>2.5</td>
</tr>
<tr>
<td>7</td>
<td>4.33</td>
<td>3.06</td>
<td>2.5</td>
</tr>
<tr>
<td>8</td>
<td>4.33</td>
<td>3.06</td>
<td>2.5</td>
</tr>
<tr>
<td>9</td>
<td>4.33</td>
<td>3.06</td>
<td>2.5</td>
</tr>
<tr>
<td>10</td>
<td>4.33</td>
<td>3.06</td>
<td>2.5</td>
</tr>
<tr>
<td>11</td>
<td>4.33</td>
<td>3.06</td>
<td>2.5</td>
</tr>
<tr>
<td>12</td>
<td>4.33</td>
<td>3.06</td>
<td>2.5</td>
</tr>
<tr>
<td>13</td>
<td>4.33</td>
<td>3.06</td>
<td>2.5</td>
</tr>
<tr>
<td>14</td>
<td>3</td>
<td>3</td>
<td>2.12</td>
</tr>
<tr>
<td>15</td>
<td>4.33</td>
<td>3.06</td>
<td>3</td>
</tr>
<tr>
<td>16</td>
<td>4.33</td>
<td>3.06</td>
<td>3</td>
</tr>
</tbody>
</table>

All cluster designs had the same base area as the original cluster, 16 mm x 16 mm. In the first series different lengths and inclinations were tested, maintaining the three bar characteristics per cluster the same or with slight differences. In this series none of the clusters had circular bases.

Cluster designs in the second series were based on cluster 5, with different lengths and the addition of circular bases. The alteration in length was done in order to achieve higher stability, since the different bar inclinations provoked a moment of force that could influence the cluster results. The new lengths were calculated by minimizing the cluster moment. Regarding the circular bases angle, different angles were tested, always relative to the vertical axis, to avoid noise by marker reflection.

Finally, in the third series, the last two designs were based on cluster 10 with an alteration in the third
bar circular base angle. Here angles of 30 and 40 were tested, this time relative to the horizontal axis, to protect this marker from the reflections of the AC marker, since the optimal position of the cluster would leave these markers close to each other.

![Figure 3.2: Acromiom Cluster](image)

After printing each cluster, a series of kinematic acquisition trials was done with one subject. A marker was attached in each circular base, and the cluster placed at the position described by Shaheen et al. (2010). Next, the closest markers to the cluster were also positioned on their respective places, namely markers AA and AC, in order to test for possible interferences between them. Afterwards, 2 repetitions of a sequence of movements were performed: abduction, flexion and circumduction.

When all clusters were tested, the resulting files were analysed in order to identify noise causes and possible improvements to the cluster designs. The procedure of redesigning, printing, and testing the clusters was repeated until a good solution, with low noise, was found.

### 3.4 Experimental Protocol

The experimental protocol can be divided in 6 phases:

1. Camera calibration;
2. Placement of markers and clusters;
3. Placement of surface EMG electrodes;
4. Initial calibration;
5. Movements;
6. Final calibrations.
Firstly, the camera system must be calibrated. To do so, an L-shaped calibration frame is placed in the centre of the laboratory, surrounding one of the weigh plates. The long arm of the frame defines the x-axis of the Global Co-ordinate System (GCS), the short arm of the frame defines the y-axis of the GCS, and the z-axis is vertically upward, perpendicular to the x and y axes. A wand, with two markers, is moved around in the space were the movement will be acquired during 30 seconds. This calibration process is done minutes before the instrumentation of each subject, in order to guarantee an accurate acquisition of kinematic data.

The placement of markers and clusters is done through palpation of the bony landmarks previously described. Afterwards the positioning of the sEMG electrodes becomes easier, since SENIAM uses them as reference to describe the optimal placement for the electrodes. When all markers were set, tests were done to ensure the quality of the data acquired, i.e., to evaluate the noise caused by marker interference and the optimal placement of the sEMG electrodes.

In phase 4, two static positions are acquired, followed by a small amplitude movement acquisition. The initial static positions were the same, straight posture, elbows close to the thorax, with approximately 90° angle and the hand pronated. In the first one, all markers were placed on the subject except for the SC. Next, the same position is done, however the IJ marker is removed and the SC is fixed. Afterwards, the markers were kept the same, and the subject was instructed to make movements with small arm elevation angles.

Phase 5 consists of a series of different movements, enumerated in Table 3.5. Before each movement, instructions were given to the subjects on how to perform it, and then some practice time was allowed before the actual acquisition of the movements.

Table 3.5: Movement Description

<table>
<thead>
<tr>
<th>Movement</th>
<th>Description</th>
<th>Plane of Motion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abduction and Adduction</td>
<td>Arm elevation and lowering</td>
<td>Coronal</td>
</tr>
<tr>
<td>Flexion and Hiperextention</td>
<td>Arm elevation and lowering</td>
<td>Sagital</td>
</tr>
<tr>
<td>Horizontal Abduction and Adduction</td>
<td>Initial arm elevation until the arm is at approximately at 90° from the thorax. Abduction and adduction of the arm.</td>
<td>Transverse</td>
</tr>
<tr>
<td>Internal and External Rotation</td>
<td>Initial position with the elbows close to the thorax, with approximately 90° angle. Abduction and adduction of the forearm, maintaining the forearm in the same position</td>
<td>Transverse</td>
</tr>
<tr>
<td>Brush Hair</td>
<td>Movement of brushing air, starting at the resting position, reaching the fore head, then back of the head and returning to the initial position</td>
<td>Complex</td>
</tr>
<tr>
<td>Catch Lateral</td>
<td>Movement of catching something laterally and diagonally at a high of the subject's head.</td>
<td>Complex</td>
</tr>
<tr>
<td>Catch Medial</td>
<td>Movement of catching something medially and diagonally at a high of the subject's head.</td>
<td>Complex</td>
</tr>
<tr>
<td>Touch Back</td>
<td>Movement of touching their own back</td>
<td>Complex</td>
</tr>
<tr>
<td>Circumduction</td>
<td>Complete range of motion of the shoulder</td>
<td>Complex</td>
</tr>
</tbody>
</table>
For the CG and PG each movement was performed 10 and 7 times, respectively, with some exceptions in the pathological group in case of pain or fatigue.

The last phase consists of two new static positions, holding the arm in the sagittal and afterwards the coronal plane at approximately 120° arm elevation. In this step the subject holds each position while the TS and AI markers are repositioned, and then static data is be acquired for a few seconds.

3.5 Computational Methods

Software developed in-house (Quental, 2013) was used to generate the anatomical coordinate systems for each bone segment and to calculate the joint and body segment rotations according to the I.S.B. recommended standards from the files created by the tracking system. The glenohumeral joint centre is estimated using the algorithm of Gamage & Lasenby (2002).

Before analysing the files of the movements, a double calibration of the acromion cluster was made, this time for a second position acquired in the final calibration phase of the experimental protocol.

Finally, the files corresponding to the movements to be studied are analysed providing the joint and body segment rotations. Secondly, the movement was divided into four motion increments, described below, and a linear regression was applied to the values of glenohumeral versus scapulothoracic angles. Then the slope of the linear regression corresponded to the SHR. Additionally, a linear regression was also applied to the complete motion data in order to calculate an overall motion SHR. The four increments into which each movement was divided were defined as:

- First Increment (I1) - Minimum to the 30° HT elevation angle;
- Second Increment (I2) - 30° to 60° HT elevation angle;
- Third Increment (I3) - 60° to 90° HT elevation angle;
- Fourth Increment (I4) - 90° to 120° HT elevation angle;

3.6 Data Analysis

3.6.1 Kinematic Data

The kinematic data of each joint angle was averaged firstly across trials, within each subject, and secondly across subjects, within each group. Descriptive statistics were calculated across groups for each joint angle. The experimental study design used a two-way repeated measures analysis of variance (ANOVA) model.

From this model two values are presented, the F value and P value. The P value tests the null hypothesis, which states that the data from all groups are drawn from populations with identical means. When the P value is small, it is unlikely that the differences observed are due to random sampling. It is then possible to reject the null hypothesis, i.e., that all populations have identical means. The F value
is a ratio of 2 different measure of variance for the data. If the null hypothesis is true, the F value is likely
to be close to 1.0. If the null hypothesis is not true, the F value is likely to be greater than 1.0.

The repeated measure model is used when a variable of interest is recorded several times on each
unit in the data set (here the unit being each subject). In this work, each joint rotation is analysed in
four different HT elevation points. However, considering that all rotation measurements were performed
on the same subject, it is likely they will not be fully independent, but rather present a correlation be-
tween them. The advantage of this statistical method of analysis is that it takes this dependency into
consideration, and aims at characterizing the change in the repeated values of the response variable
(Everitt & Hothorn, 2011). Here, a two-way repeated measurements model was applied, since two dif-
ferent factors were studied on each analysis. One corresponding to the elevation factor (EF), and the
other to what characterises the difference between the samples being tested, i.e., a movement factor
(MF), when comparing abduction with flexion, a shoulder factor (SF), when comparing symptomatic with
asymptomatic shoulders, a group factor (GF), when comparing the CG with the PG.

This model can provide three different outcomes: (1) One of the factors has statistical significance (its
p-values are at least lower than 0.1); (2) Both factors have statistical significance; (3) and the interaction
between factors has statistical significance.

Regarding the first outcome, one factor having statistical significance means that the differences
based on that factor are significant. For example, if the group factor is significant, it means that the
results between data samples are significantly different. Since one of the data samples represents the
CG, a significant group effects implies a significant effect of UST on the PG shoulder kinematics. The
same reasoning is applied to any other factor.

In case the interaction between factors is significant, the variation of results based on one factor also
depends on the other. For example, if the interaction between shoulder and elevation factors is signif-
icant, it means that there is a significant variation between asymptomatic and symptomatic shoulders.
However, this variation depends on the elevation point being considered. Therefore, the pattern of joint
rotation angle across the four HT elevation points is significantly different between shoulders.

Table 3.6 summarizes the factors that will be consider on each analysis, along with their respective
interaction.

Tukey-Kramer post hoc testing was used where appropriate, to adjust for multiple pairwise compar-
isons of the two factors under analysis. Firstly, each factor and the interaction between factors were
tested to determine any potential influence. If there was a significant interaction, Tukey-Kramer post hoc
test was then applied, to test for possible significant differences on a specific elevation point.

The data analysis will be organized into three sections, (1) the description of the Control group
results, with comparison of the two movements under consideration, abduction and flexion; (2) the
description of the Pathological group, comparing symptomatic with asymptomatic shoulder, as well as
both shoulders with the Control group; and (3) a brief description of the results of the two subgroups
Pain and No Pain, with main focus on the comparison between them and the Control group.

On all three sections, the kinematic variables are analysed as a function of HT elevation. However,
depending on the variable being testes, joint rotation angles or SHR, a different factor concerning ele-
elevation is used. The joint rotation angles are analysed at four different HT elevation angles, at 30°, 60°, 90° and 120°. For higher elevation angles the tracking system loses sensitivity (Shaheen et al., 2010). For these variables, an elevation factor will be considered as specific data points of the movement are used. Regarding the SHR, since it is calculated by applying a linear regression on a given movement interval, an increment factor will be used to represent the four elevation increments. The second factor on the statistical model will depend on what comparison it is being analysed.

The elevation and increment factors refer to the effect of the HT elevation on all dependent variables, the first focusing on the values on specific elevation points and the latter on 30° incremental elevations. The movement factor represents the effect of the different movements, and therefore, it is used when comparing the results of kinematic data between abduction and flexion. The shoulder factor is applied, in order to analyse the differences between symptomatic and asymptomatic shoulder on the Pathological group. Finally, the group factor is used to study the effect of the groups or subgroups on the results, i.e., when comparing shoulders between Pathological and Control group or between subgroups.

Table 3.6: Two-way repeated measures (ANOVA) model

<table>
<thead>
<tr>
<th>Factors</th>
<th>Abduction vs Flexion</th>
<th>Symptomatic vs Asymptomatic</th>
<th>Group vs Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Movement Factor (MF); Increment (IF) or Elevation Factor (EF)</td>
<td>Movement Factor (MF); Increment (IF) or Elevation Factor (EF)</td>
<td>Movement Factor (MF); Increment (IF) or Elevation Factor (EF)</td>
<td></td>
</tr>
<tr>
<td>Interaction</td>
<td>Movement + Elevation (MEI) or Movement + Increment (MII)</td>
<td>Shoulder + Elevation (SEI) or Shoulder + Increment (SII)</td>
<td>Group + Elevation (GEI) or Group + Increment (GII)</td>
</tr>
</tbody>
</table>

### 3.6.2 sEMG

A set of MATLAB built-in routines were used to process the sEMG data. For each subject trial, a band-pass filter from 20 Hz to 400 Hz was applied, in order to remove high frequency and motion-related noise. Next, the mean value of each muscle along the trial was calculated and subtracted from the original data, so the signal oscillates around 0. The signal was rectified using a root mean square (RMS), and subsequently normalized using a mean dynamic amplitude (m-DYN) method (Myers et al., 2009; Bolgla & Uhl, 2007). Each trial was normalized using its mean value. Thus, each representation of the sEMG mean normalized sEMG data is as a percentage of the m-DYN (% m-DYN). The normalized sEMG data was averaged firstly across trials, within each subject, and secondly across subjects, within each group. This normalization technique was selected mainly due to the patient's inability to perform maximum voluntary contractions.

With the rectified and normalized data two different analyses were done:

- Onset analysis;
- Mean sEMG normalized data analysis;
The onset time was defined as the moment the muscles reached 5% of their maximum amplitude from the moment the motion was detected. This moment was obtained using the tracking system software to detect the first instant at which the subject arm started moving. The onset value was calculated for each trial and then averaged for each subject. Afterwards, it was averaged across subject within each group. For this analysis, a one-way repeated measures analysis of variance (ANOVA) model was also applied with different factors depending on the comparison being made. These comparisons were the same as the ones described for the kinematic data analysis, with the difference that they do not include the elevation and increment effects, since the onset is unique per trial.

The mean sEMG normalized data was defined as the averaged value of the processed sEMG data across the same 30° intervals described above (increments 1 to 4). Here, a two-way repeated-measures analysis of variance (ANOVA) model was applied with the same factors described for the kinematics data (Table 3.6).
Chapter 4

Results

4.1 Acromion Cluster

The original cluster used by Shaheen et al. (2010) presented noisy results, mainly due to the proximity of the markers. Their reflection interfered with each other, causing oscillations in their position or even merges between them. Figure 4.1 represents the trajectory coordinates of one of the cluster markers in the original cluster. There were high oscillations and sudden changes of position. Because they do not represent the real motion of the markers, they would introduce errors when calculating scapula kinematics. Since scapula kinematics is such an essential component to study shoulder kinematics, it is very important to have an acromion cluster that can provide data with as little noise as possible.

![Figure 4.1: Marker 2 of the Acromion Cluster Trajectory without interpolations for the original cluster. X-axis: Frame number; Y-axis: Coordinates (mm)](image)

Regarding the first series of cluster designs, the overall results showed less noise between the cluster markers, however some noise remained in the AA and AC markers. On the first series of printed clusters, the cluster that presented best results was Cluster 5. Nonetheless it still showed notable noise, not only in the cluster markers but in the surrounding ones also.

The second series had 8 variations of the cluster 5. To this cluster, circular bases were added to the
bar ends, as a protection of marker reflection to the other markers. In each design, one of the circular bases had a given angle relative to the bar, in order to find the best configuration to avoid unwanted reflections.

The results between clusters were quite similar, however one of the clusters showed less noise on all markers. Because some of the clusters had only slight alterations in the circular base angle, but showed notable differences in the resulting amount of noise, it was also hypothesised that the positioning of the cluster was important to avoid marker reflection, i.e., the relative position of the bars to the surrounding markers.

Taking this hypothesis and the last results into consideration, a final set of 2 clusters were designed. The cluster that present the best results, meaning, the least amount of noise or interference, was Cluster 15, described in Table 4.1.

<table>
<thead>
<tr>
<th>Bar Number</th>
<th>Length (cm)</th>
<th>Inclination</th>
<th>Circular Base Angle</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4.33</td>
<td>60°</td>
<td>0°</td>
</tr>
<tr>
<td>2</td>
<td>3.06</td>
<td>45°</td>
<td>30° (Horizontal)</td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>30°</td>
<td>40° (Vertical)</td>
</tr>
</tbody>
</table>

In Figure 4.2 the trajectory of the chosen cluster for the same movement is depicted. There were no longer steep oscillations or sudden changes of position, this cluster provided smoother data. Moreover, it can be observed that the addition of angled circular bases to the bars, along with their configuration of lengths and inclinations, avoided unwanted reflection between the three markers of the cluster and the surrounding ones. Hence, providing a more accurate motion data of the cluster and consequently more realistic scapula kinematic results.
4.2 Kinematics

4.2.1 Control Group

This analysis included 15 Control individuals (8 men, 7 women), with a mean age of 24 ± 5.5 years (range, 18-39 years). Their mean height was 1.72 ± 0.10 m and weight was 67.97 ± 13.16 kg, and their self-reported dominant arms were right in 13, left in 1 and ambidextrous in 1. None of the volunteers had shoulder pain or a medical history of shoulder disorders. Each participant was informed of the details of the study and provided signed consent before participation.

Table 4.2 describes the CG studied.

Table 4.2: Subjects characteristics on the CG

<table>
<thead>
<tr>
<th>ID</th>
<th>Dominant Arm</th>
<th>Age</th>
<th>Sex</th>
<th>Weight (kg)</th>
<th>Height (m)</th>
<th>Sport</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO1</td>
<td>Right</td>
<td>22</td>
<td>F</td>
<td>62</td>
<td>1.71</td>
<td>No</td>
</tr>
<tr>
<td>CO2</td>
<td>Right</td>
<td>23</td>
<td>F</td>
<td>57</td>
<td>1.64</td>
<td>Casual</td>
</tr>
<tr>
<td>CO3</td>
<td>Right</td>
<td>23</td>
<td>M</td>
<td>67</td>
<td>1.75</td>
<td>No</td>
</tr>
<tr>
<td>CO4</td>
<td>Right</td>
<td>27</td>
<td>M</td>
<td>86</td>
<td>1.84</td>
<td>Usually</td>
</tr>
<tr>
<td>CO5</td>
<td>Right</td>
<td>21</td>
<td>M</td>
<td>77</td>
<td>1.86</td>
<td>Usually</td>
</tr>
<tr>
<td>CO6</td>
<td>Right</td>
<td>21</td>
<td>M</td>
<td>84</td>
<td>1.86</td>
<td>Usually</td>
</tr>
<tr>
<td>CO7</td>
<td>Right</td>
<td>23</td>
<td>F</td>
<td>56.5</td>
<td>1.63</td>
<td>Usually</td>
</tr>
<tr>
<td>CO8</td>
<td>Ambidextrous</td>
<td>19</td>
<td>F</td>
<td>60</td>
<td>1.6</td>
<td>Usually</td>
</tr>
<tr>
<td>CO9</td>
<td>Right</td>
<td>39</td>
<td>M</td>
<td>85</td>
<td>1.8</td>
<td>Occasional</td>
</tr>
<tr>
<td>CO10</td>
<td>Right</td>
<td>22</td>
<td>F</td>
<td>58</td>
<td>1.64</td>
<td>Occasional</td>
</tr>
<tr>
<td>CO11</td>
<td>Right</td>
<td>18</td>
<td>M</td>
<td>80</td>
<td>1.75</td>
<td>No</td>
</tr>
<tr>
<td>CO12</td>
<td>Right</td>
<td>21</td>
<td>F</td>
<td>47</td>
<td>1.54</td>
<td>Casual</td>
</tr>
<tr>
<td>CO13</td>
<td>Left</td>
<td>35</td>
<td>M</td>
<td>73</td>
<td>1.8</td>
<td>Usually</td>
</tr>
<tr>
<td>CO14</td>
<td>Right</td>
<td>22</td>
<td>M</td>
<td>80</td>
<td>1.74</td>
<td>Usually</td>
</tr>
<tr>
<td>CO15</td>
<td>Right</td>
<td>23</td>
<td>F</td>
<td>50</td>
<td>1.57</td>
<td>Casual</td>
</tr>
</tbody>
</table>

For the first three Control subjects, an analysis of both arms was done in order to verify if the kinematic results between them were similar, as the literature suggested (Matsuki et al., 2011; Hosseinimehr et al., 2015; Yoshizaki et al., 2009).

Little variations were found on shoulder kinematics and between dominant and non-dominant shoulders. Nonetheless, it should be taken into account that this results were only used as reference to the following acquisition, since the sample of subjects is very small to allow for a reliable conclusion. Be-
cause these results suggest the findings presented by Matsuki et al. (2011); Hosseinimehr et al. (2015); Yoshizaki et al. (2009), the remaining 12 Control subjects were only tested for their dominant arm. For the following analysis the data respecting the non dominant arms were excluded from the CG data.

Figure 4.3 depicts the scapulothoracic and glenohumeral motion during HT elevation on the frontal plane, or Abduction, and HT elevation on the sagittal plane, or Flexion, respectively.

During abduction, the scapulothoracic joint was characterized by a decrease in the internal rotation of 26°; an increase of upward rotation, reaching an average maximum rotation of 52.56°; and an increase in posterior tilting of 25° (Figures 4.3(a), 4.3(b) and 4.4(a)).

![Figure 4.3: Scapulothoracic and glenohumeral joint motion during abduction and flexion](image)

Regarding flexion, both scapulothoracic internal rotation (STIR) and STT showed more of a quadratic behaviour. STIR reached its minimum value of 23° at approximately 80° of HT elevation, and STT reached its maximum value of 24° close to the 100° of HT elevation. The STUR increased continuously, reaching an average maximum rotation of 56.83°.

Looking now to the glenohumeral motion, Figure 4.4(b) shows a continuous elevation with an average maximum elevation of 82.75° and 86.14°, during abduction and flexion, respectively.
The movement effect was significant on the STIR ($p<0.001$, $F=15.244$) and glenohumeral (GH) ($p<0.001$, $F=55.481$). The interaction between increment and movement was significant on STIR ($p<0.05$, $F=2.929$) and STT ($p<0.1$, $F=2.318$). Only the scapulothoracic upward rotation (STUR) did not present significant differences between movements. The elevation effect was significant on all joint rotations.

The scapula was more internally and posteriorly tilted during flexion until approximately 100° and 80° of HT elevation, respectively. The GH elevation was also higher during flexion for the complete movement.

Table 4.3 presents the SHR for the total movement as well as for 30° increments between the starting position and 120°. This data is also represented on Figure 4.5, through a profile analysis of the mean SHR values between movements.

Significant movement ($p<0.001$, $F=16.807$) and increments ($p<0.1$, $F=2.249$) effect were found, as well as significant interaction ($p<0.001$, $F=6.929$). A post hoc comparison showed statistical significance on the first two increments (I1: $p<0.01$, I2: $p<0.01$), with higher SHR during flexion, having differences of...
Table 4.3: SHR values at intervals throughout both abduction and flexion

<table>
<thead>
<tr>
<th>Elevation Intervals</th>
<th>Min-30°</th>
<th>30°-60°</th>
<th>60°-90°</th>
<th>90°-120°</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abduction Mean SHR</td>
<td>0.50</td>
<td>1.41</td>
<td>1.81</td>
<td>1.59</td>
<td>1.57</td>
</tr>
<tr>
<td>Flexion Mean SHR</td>
<td>2.02</td>
<td>2.78</td>
<td>1.68</td>
<td>1.34</td>
<td>2.08</td>
</tr>
</tbody>
</table>

1.52 and 1.37, respectively. From 30° of HT elevation, the SHR values were similar between movements, with a slight higher value on the third increment (I3) during abduction. For flexion, a larger difference across increments was found, with the highest value of 2.78 occurring early in on the motion, at the second increment (I2).

Figure 4.5: SHR in 30° Increments

4.2.2 Pathological Group

This study included 9 patients (2 men, 7 women), recruited from the the Hospital Curry Cabral, in Lisbon. All patients with proven UCT using imagiology tests, but with no rotator cuff tears were refereed to the Laboratório de Biomecânica de Lisboa, where the acquisition test took place.

The PG has mean age of 46.67 ± 13.70 years (range, 23-68 years). The mean height was 1.64 ± 0.09 m and the mean weight was 66.88 ± 10.24 kg. Their self-reported dominant arms were right in 7 and left in 3. Each participant was informed of the details of the study and provided signed consent before participation. Table 4.4 describes the PG studied.

Figures 4.7 to 4.9 depict the scapulothoracic and glenohumeral joint motions of both shoulders, during abduction. Figure 4.10 to 4.13 shows the same joint motions during flexion.

During both abduction and flexion, no significant interactions between elevation and shoulder effects were found, meaning that the pattern of joint rotation between contralateral shoulders remained similar across the whole PG’s range of motion. However, as can be observed in Figures 4.7 to 4.13, despite the similarity in pattern of motion, the rotation angles were different between shoulders. Significant shoulder effect was found for STIR and STUR during both movements, where the symptomatic shoulder had a less internally and upwardly rotated scapula.
Table 4.4: Pathological Group Description

<table>
<thead>
<tr>
<th>ID</th>
<th>Dominant Arm</th>
<th>Symptomatic Shoulder</th>
<th>Age</th>
<th>Sex</th>
<th>Weight [kg]</th>
<th>Height [m]</th>
<th>Profession</th>
</tr>
</thead>
<tbody>
<tr>
<td>PO1</td>
<td>Left</td>
<td>Left</td>
<td>43</td>
<td>M</td>
<td>72</td>
<td>1.72</td>
<td>Student and Hairdresser</td>
</tr>
<tr>
<td>PO3</td>
<td>Right</td>
<td>Right</td>
<td>23</td>
<td>F</td>
<td>58</td>
<td>1.62</td>
<td>Student and Hairdresser</td>
</tr>
<tr>
<td>PO4</td>
<td>Right</td>
<td>Right</td>
<td>46</td>
<td>F</td>
<td>63</td>
<td>1.62</td>
<td>Hairdresser</td>
</tr>
<tr>
<td>PO5</td>
<td>Right</td>
<td>Right</td>
<td>55</td>
<td>F</td>
<td>76</td>
<td>1.58</td>
<td>Treasurer</td>
</tr>
<tr>
<td>PO6</td>
<td>Right</td>
<td>Right</td>
<td>45</td>
<td>F</td>
<td>62</td>
<td>1.69</td>
<td>Nurse</td>
</tr>
<tr>
<td>PO7</td>
<td>Right</td>
<td>Left</td>
<td>68</td>
<td>M</td>
<td>80</td>
<td>1.83</td>
<td>Economist</td>
</tr>
<tr>
<td>PO8</td>
<td>Left</td>
<td>Left</td>
<td>44</td>
<td>F</td>
<td>76</td>
<td>1.51</td>
<td>Unemployed (Former Hairdresser)</td>
</tr>
<tr>
<td>PO9</td>
<td>Right</td>
<td>Right</td>
<td>65</td>
<td>F</td>
<td>-</td>
<td>1.61</td>
<td>Housekeeper</td>
</tr>
<tr>
<td>PO10</td>
<td>Left</td>
<td>Left</td>
<td>31</td>
<td>F</td>
<td>48</td>
<td>1.61</td>
<td>IT Support</td>
</tr>
</tbody>
</table>

Both symptomatic and asymptomatic shoulders, present similar motion patterns relative to the CG, however, just as described above, the rotation angles values vary significantly in some cases.

The asymptomatic shoulder on the PG showed only a significant group effect on the STIR, during both abduction ($p<0.05$, $F=5.416$), and flexion ($p<0.1$, $F=2.806$), having a more internally rotated scapula. On the other hand, the comparison between symptomatic shoulder and CG showed a significant group effect on the STUR, during abduction ($p<0.05$, $F=5.656$) and flexion ($p<0.05$, $F=4.413$). During abduction, the symptomatic shoulder was less upwardly rotated. During flexion, both shoulders had similar values for upward rotation at the $30^\circ$ and $60^\circ$ elevation, at the $90^\circ$ and $120^\circ$, the symptomatic shoulder was more upwardly rotated. Additionally, the group effect was also significant on glenohumeral
elevation during flexion (p<0.01, F=7.866), where the symptomatic shoulder had a lower glenohumeral elevation throughout the complete movement.

Figure 4.7: Scapulothoracic Internal Rotations - Abduction - Symptomatic vs asymptomatic vs CG

Figure 4.8: Scapulothoracic Posterior Tilt - Abduction - Symptomatic vs asymptomatic vs CG

Figure 4.9: Glenohumeral Elevation - Abduction - Symptomatic vs asymptomatic vs CG
Figure 4.10: Scapulothoracic Internal Rotations - Flexion - Symptomatic vs asymptomatic vs CG

Figure 4.11: Scapulothoracic Upward Rotations - Flexion - Symptomatic vs asymptomatic vs CG

Figure 4.12: Scapulothoracic Posterior Tilt - Flexion - Symptomatic vs asymptomatic vs CG
Table 4.5 presents the SHR mean values for the incremental motion, as well as the total motion. Additionally, Figure 4.14 shows a profile analysis of the mean SHR values for all symptomatic, asymptomatic and CG, during both movements.

Table 4.5: Mean SHR for the Pathological Group

<table>
<thead>
<tr>
<th>Elevation Intervals</th>
<th>Min-30°</th>
<th>30°-60°</th>
<th>60°-90°</th>
<th>90°-120°</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Abduction</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASS</td>
<td>1.73</td>
<td>1.40</td>
<td>1.32</td>
<td>1.03</td>
<td>1.33</td>
</tr>
<tr>
<td>SS</td>
<td>2.78</td>
<td>2.84</td>
<td>1.10</td>
<td>1.21</td>
<td>1.65</td>
</tr>
<tr>
<td><strong>Mean SHR</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASS</td>
<td>1.37</td>
<td>2.11</td>
<td>1.55</td>
<td>0.69</td>
<td>1.38</td>
</tr>
<tr>
<td>SS</td>
<td>3.11</td>
<td>2.46</td>
<td>1.35</td>
<td>0.64</td>
<td>1.67</td>
</tr>
</tbody>
</table>

Starting with the abduction movement, when comparing the asymptomatic shoulder with the CG, a significant interaction between group and increment was found \((p<0.01, F=3.803)\), meaning that the pattern between groups was significantly different. As it can be observed on Figure 4.14, the asymptomatic shoulder had its highest SHR on the first motion increment and decreased it continually on the following increments. On the other hand, the CG had its minimum SHR on the first increment and increased it on the following to increments.

The symptomatic shoulder also showed a significant variation in pattern to the CG \((p<0.001, F=11.940)\), along with significant group \((p<0.01, F=10.366)\) and increment effect \((p<0.1, F=2.129)\). On the first two motion increments, the symptomatic shoulder had significant higher SHR relative to the CG (I1 - \(p<0.001\); I2 - \(p<0.001\)). On the following motion increment, the symptomatic shoulder showed an opposite behaviour to the CG, decreasing steeply on the third increment and then increasing on the last one. This variation in SHR across the complete range of motion also proved to be statistically significant when compared to its contralateral shoulder \((p<0.1, F=2.022)\).

For flexion, a significant shoulder, increment interaction was found between contralateral shoulders \((p<0.05, F=3.129)\). The symptomatic shoulder showed higher SHR on the first two motion increments,
with statistical significance on the first one (p<0.01). From the 90° elevation onwards, the symptomatic shoulder followed similar values to the asymptomatic shoulder. When comparing CG with PG, only significant increment effect was found.

Figure 4.14: SHR Increments - Symptomatic vs asymptomatic vs CG

Regarding the SHR for the complete motion, it is possible to observe similar relative values between groups than the ones found throughout the motion increments, i. e., during abduction the asymptomatic shoulder showed mostly the lowest SHR values, while the symptomatic shoulder had a very high SHR at the fist and second increment that influenced significantly the SHR of the complete motion. As such, the symptomatic shoulder showed the highest SHR for the complete movement and the asymptomatic the lowest. For flexion the same can be said, the CG had the highest values throughout the movement, while the asymptomatic had the lowest. The same result is observed on the SHR for the complete movement.

4.2.3 Pain/No Pain Subgroups

Despite the homogeneity of the PG, variations in symptoms and movement constriction were found. The main differences identified during the acquisition procedure was the extent of motion constrain in the symptomatic shoulders and the pain that each patient described during the procedure.

Three of the nine patients reported pain at a given HT elevation angle, stopping their movement at that point. Furthermore, one patient described its movement constrain as a anatomical/physiological blockage of the motion and not pain inflicted. The remaining five patients did not show a notable difference in range of motion between shoulders and the pain reported was low or non-existing.

Given the variations in pain and range of motion described above the PG could be separated in two subgroup:

1. Pain subgroup: subgroup of subjects that reported moderate or high pain at a given HT elevation angle, and that elevation point on the symptomatic shoulder was less then 85% of the asymptomatic maximum arm elevation angle.
2. No Pain subgroup: subgroup of subjects that reported low pain throughout the range of motion, and that the maximum HT elevation angle on the symptomatic shoulder was at least 85% of the asymptomatic.

Table 4.6 shows the maximum angles of scapula upward rotation and glenohumeral and HT elevation between contralateral shoulders for both subgroups. The differences between subgroups were more noticeable in the maximum HT elevation: for the No Pain subgroup the values between contralateral shoulders were very similar, i.e. there were no movement constrains, while for the Pain subgroup there is a clear difference in the the maximum HT elevation achieved between contralateral shoulders.

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Max Scapula Upward Rotation</th>
<th>Max Glenohumeral Joint Angle</th>
<th>Max HT Elevation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AS</td>
<td>SS</td>
<td>AS</td>
</tr>
<tr>
<td>Pain</td>
<td>50.72</td>
<td>31.12</td>
<td>72.10</td>
</tr>
<tr>
<td>No Pain</td>
<td>46.64</td>
<td>47.56</td>
<td>78.60</td>
</tr>
</tbody>
</table>

In this analysis it is interesting to compare not only symptomatic vs asymptomatic shoulder within each subgroup, but also how shoulder kinematics vary between subgroups and what differences can be found when comparing again with the CG.

**Subgroups Comparison**

On this subsection, the results of both Pathological subgroups will be described and compared, in order to evaluate the possible influence of the variation in symptoms between subjects. Because the No Pain subgroup had motion constraints, the subjects did not reach the 120° HT elevation. Hence, the analysis here described will only consider the first three motion increments.

Both subgroups presented very similar shoulder kinematics between their relative contralateral shoulders. Only the No pain subgroup showed a significant shoulder effect during flexion, on the STIR, where the symptomatic shoulder had a less internally rotated scapula. Nonetheless, the elevation effect was significant on both subgroups for all joint rotations and the increment effect significant only during abduction.

Figures 4.15 to 4.22 depict the glenohumeral and scapulothoracic joint motion of the symptomatic shoulders of both subgroups. The figures regarding the comparison between asymptomatic shoulders may be consulted on Appendix A, Figures A.1 to A.8.

Regarding the asymptomatic shoulder, during abduction, there was a significant subgroup effect on the glenohumeral elevation (p<0.1, F=4.151), where the Pain subgroup showed higher values at the 60° and 90° of HT elevation. A significant subgroup effect and interaction was found for STIR (p<0.001, F=47.26), (p<0.05, F=3.64, respectively). Pairwise comparisons indicated that the Pain subgroup had the scapula more internally rotated at 30° (p<0.1, mean difference, 3.54°), 60° (p<0.01 mean difference, 9.05°) and 90° (p<0.01 mean difference, 14.63) (p<0.001 mean difference, 18.03).
During flexion, similar results regarding STIR were found, with significant subgroup effect \((p<0.001, F=33.544)\) and significant interaction between subgroup and elevation \((p<0.01, F=5.850)\). Pairwise comparisons indicated that the Pain subgroup had the scapula more internally rotated at 60° \((p<0.1, \text{mean difference, } 5.62°)\) and 90° \((p<0.01 \text{ mean difference, } 12.46)\) \((p<0.001 \text{ mean difference, } 18.78)\). Regarding the glenohumeral elevation angles, the No Pain subgroup showed higher values from approximately the 60° of the HT elevation. Additionally, the elevation effect was significant for all joint rotations during both abduction and flexion.

Looking now to the symptomatic shoulders of both groups, during abduction, the group effect was found significant for STIR \((p<0.01, F=9.155)\), STT \((p<0.01 , F=11.085)\) and GH elevation \((p<0.01, F=10.454)\). The Pain group had a less posteriorly tilted and internally rotated scapula and a more elevated GH joint, at all angles of HT elevation.

During flexion, the group effect was found significant for STIR \((p<0.01, F=10.711)\) and glenohumeral elevation \((p<0.001, F=55.12)\). The No Pain group had a more internally rotated scapula and a higher glenohumeral elevation at all angles of HT elevation. Additionally, the elevation effect was significant for all joint rotations during both abduction and flexion.

![Figure 4.15: Pain vs No Pain subgroup - SS - Scapulothoracic Internal Rotation - Abduction](image)

![Figure 4.16: Pain vs No Pain subgroup - SS - Scapulothoracic Upward Rotation - Abduction](image)
Concerning the SHR results for the asymptomatic shoulders, Figure 4.23 shows the SHR values during both movements for all increments and for the total movement.

During flexion, the subgroup effect was found significant for STIR (p<0.01, F=10.711) and gleno-humeral elevation (p<0.001, F=55.12). The Pain subgroup had a more internally rotated scapula and a
lower glenohumeral elevation at all angles of HT elevation. Additionally, the elevation effect was significant for all joint rotations during both abduction and flexion.

Figure 4.20: Pain vs No Pain subgroup - SS - Scapulothoracic Upward Rotation - Flexion

Figure 4.21: Pain vs No Pain subgroup - SS - Scapulothoracic Posterior Tilting - Flexion

Figure 4.22: Pain vs No Pain subgroup - SS - Glenohumeral Elevation - Flexion

Regarding the SHR results for the symptomatic shoulder, Figure 4.24 presents the SHR values during both movements for all increments and for the total movement.
Significant increment effect was found for both movements. The only noticeable difference between subgroups was on the first motion increment, where the Pain subgroup had a higher value during abduction and a lower one during flexion. For the complete motion SHR, both subgroups had similar values for the two movements.

Figure 4.23: SHR Increments - No Pain subgroup vs Pain subgroup - Asymptomatic shoulders

Figure 4.24: SHR Increments - No Pain subgroup vs Pain subgroup - Symptomatic shoulders
4.3 EMG Results

The analysis of the sEMG results will follow two steps, first the presentation of the onset values of each muscle and then the description of their normalized sEMG data patterns. These results will be first presented for the CG (considering all 8 muscles) and then for the PG and respective subgroups.

4.3.1 Control Group

The sEMG results referring to the CG will be presented with both movements side-by-side as a way to describe and compare them afterwards.

Onset Results

Figure 4.25 shows the mean onset values with respective standard deviation, in seconds, for both abduction and flexion movements.

![Figure 4.25: Onset - CG - Abduction vs Flexion](image)

Significant differences were found between movements for the AD, MD and PD (p < 0.01, F=11.1; p < 0.001, F=26.85; p < 0.001, F=22.64; respectively), not only showing a significant variance in value but also in its pattern. During abduction, the sequence of deltoid activation was, first the PD, followed by the medial and finally the AD, all with approximately 0.2 s between them. On the other hand, during flexion, the AD was the first to be activated, followed by the posterior, and then the MD. The activation interval between the first two deltoids was approximately 0.18 s, however the MD activated later in the movement with an interval from the PD of almost 0.5 s.

Additionally, the pectoralis major also had a significant difference between movements (p < 0.05, F=4.514), with an earlier onset during flexion. The remaining muscles showed similar values, with Flexion having earlier onsets on all muscles, except for the superior trapezius.

Moreover, there were different patterns of muscle onset between movements. For abduction, the PD and superior trapezius were the first muscles to activate, with onsets lower than 0.1 s. Next, the MD...
and infraspinatus activated at approximately 0.3 s. The remaining muscles activated approximately after 0.45 s of the movement start, being the inferior trapezius the last to be activated at 0.74 s.

Regarding flexion, the AD was the first to be activated at approximately 0.15 s, followed by the infraspinatus and superior trapezius 0.02 s later. After approximately 0.14 s the PD and pectoralis major activate. Only after 0.4 s of the movement start the SA, inferior trapezius and middle deltoid activate, in this order, with the latest activation at 0.83 s.

Normalized EMG Data Results

Figure 4.26 and 4.27 depicts the normalized EMG data of all muscle per motion increment, during abduction and flexion.

![Normalized EMG Data Results](image)

All three deltoids had a significant movement, increment interaction (p<0.001, F= 28.39 for the AD; p<0.001, F= 19.30 for the MD; p<0.001, F= 22.76 for the PD). A post hoc analysis showed significant
differences on all motion increments (I1: p<0.001, I2: p<0.001, I3: p<0.001, I4: p<0.001). During flexion, the AD showed a faster increase, reaching its mean value on the second increment, while during abduction the sEMG curve was beneath its mean value until the third increment. The MD and PD showed an opposite behaviour, reaching its mean value faster during abduction than during flexion (I2, I3 and I4 - p<0.001 for MD; I1 - p<0.01, I2 and I4 - p<0.001 for PD).

The IS and PM also showed a significant movement, increment interaction (p<0.001, F= 13.283 for the IS; p<0.001, F= 11.437 for the PM). Regarding the IS, a more noticeable change in normalized sEMG data pattern could be observed. During abduction, the normalized sEMG of the IS showed an almost constant increase, while during flexion, there was a faster progression of the signal until the third increment, and from the 90° of HT elevation onward the normalized sEMG decreased to be lower than during abduction. A post hoc analysis showed statistical significance on the second and third increment (I2 - p<0.05 and I3 - p<0.001 ).

The PM, showed similar differences in pattern between movements, to what was described for the
AD. It reached its mean value faster during flexion, at the second increment, stabilizing from the point onwards. During abduction the normalized sEMG had a slower but more constant increase. A post hoc analysis showed statistical significance on the second and fourth increment (I2 - p<0.001 and I4 - p<0.05).

The SA, and both trapezius showed similar mean normalized sEMG results throughout abduction and flexion. All muscles showed significant increment effects.

### 4.3.2 Pathological Group

**Onset Results**

Figures 4.28 and 4.29 depict the onset values of the symptomatic and asymptomatic shoulders on the PG and of the CG, along with a representation of the movement start (dashed line), during abduction and flexion, respectively.

![Figure 4.28: Onset - Abduction - Symptomatic vs Asymptomatic](image)

During abduction, the onset pattern between contralateral shoulders remained similar, only having a significant shoulder effect on the infraspinatus (p<0.1, F=3.802). Despite no other significant differences were found, a slight delay on the symptomatic shoulder onset can be observed on all muscles, except for the SA.

For both shoulders on the PG, the PD activated prior to the beginning of the motion, followed by the MD within less than 0.03 s and 0.12 s of the motion start, for the asymptomatic and symptomatic shoulders respectively. For the asymptomatic shoulder, the infraspinatus activated closely to the MD, at 0.04 s. In contrast, for the symptomatic shoulder, these muscles activated later, at approximately 0.23 s, closer to the activation of the AD. For the remaining two muscles, AD and SA, both shoulders also showed similar onset values.
Figure 4.29: Onset - Flexion - Symptomatic vs Asymptomatic

During flexion, both contralateral shoulders also showed a similar onset pattern, however with a significant shoulder effect on the AD (p<0.1, F=3.82) and SA (p<0.1, F=3.484). Once more, the symptomatic shoulder seemed to have a slight delay on all muscles onset, except for the SA, however, for this motion the SA of the symptomatic shoulder activated much earlier on the motion, having similar onset values to the infraspinatus and PD. Regarding the AD, despite showing a significant shoulder effect, the difference between shoulder values was 0.04s, one of the smallest.

The onset pattern, mostly shared by contralateral shoulders, starts with the activation of the AD, before the start of the motion for the asymptomatic shoulder, and 0.01s afterwards for the symptomatic. Then, the PD and infraspinatus activate, all within 0.16 s after the motion begin. The MD was the last muscle to activate, approximately at 0.65s for both shoulders. As mentioned before, the SA showed a different behaviour on each shoulder. For the asymptomatic shoulder it activated closer to the infraspinatus, at 0.2s, and for the symptomatic shoulder, activated later in the motion, closer to the MD at 0.50s.

Regarding the comparison between PG and CG, significant differences were found on the PD (p<0.05, F=4.996, between SS and CG and p<0.05, F=7.162, between ASS and CG) and SA (p<0.05 F=5.412 between ASS and CG) during abduction. However, despite the lack of statistical significance, consistent pattern variation between groups could be observed during both movements. There was a later activation of all three deltoids on the CG during both movements. For the infraspinatus and SA the onset differences varied depending on the PG shoulder being considered. On one hand, during abduction, the onset values of the CG were similar to the symptomatic shoulder for both muscles. When compared to the asymptomatic shoulder, the CG had an earlier activation on the infraspinatus and a later on the SA. On the other hand, during flexion, the CG had a closer onset value to the asymptomatic shoulder on the SA. For the infraspinatus, the CG had a similar value to the symptomatic shoulder, with a later activation then the asymptomatic.
Normalized EMG Data Results

Figures 4.30 and 4.31 depict the mean normalized EMG data of all muscles, during abduction and flexion.

Between contralateral shoulders, during abduction, both anterior and medial deltoid showed a significant shoulder effect ($p<0.05$, $F=5.239$ for the AD; $p<0.1$, $F=3.203$ for the MD). All five muscles showed a significant increment effect.

During flexion, the MD had a significant interaction between shoulder and increment ($p<0.05$, $F=2.932$). The symptomatic shoulder showed a more linear normalized sEMG behaviour, while the asymptomatic shoulder had more of a quadratic behaviour, having a slower progression across increments. The SA showed a significant shoulder effect ($p<0.1$, $F=2.849$) and all muscles showed a significant increment effect.

When comparing the PG with the CG, a significant shoulder, increment interaction was found on the AD ($p<0.001$, $F=2.849$) during abduction, and on the ME ($p<0.1$, $F=13.059$) and IS ($p<0.01$, $F=5.469$) during flexion. Additionally, the shoulder effect was also found significant on the PD ($p<0.01$, $F=9.293$)
During flexion, the increment effect was significant on all five muscles during both movements.

During abduction, the AD on the symptomatic shoulder had a faster evolution until the third motion increment (I₃: \( p < 0.001 \)). From the 90° elevation onwards the CG had a steep increase, while the symptomatic shoulder stabilized.

For flexion, the MD showed a faster progression in the normalized sEMG on the symptomatic shoulder throughout the movement, with statistical significance on the third increment (\( p < 0.01 \)). Regarding the IS, a significant variation in normalized sEMG data pattern could be observed. The PG's symptomatic shoulder showed a slower increase until the third increment, stabilizing from that point onwards, while the CG showed a decrease on the normalized sEMG signal. For the PD, the symptomatic shoulder reached faster its mean value. However, a faster progression on the normalized sEMG can be observed on the CG.

Regarding the comparison between PG's asymptomatic shoulder and the CG, significant interaction between shoulder and increment effects was found on the AD (\( p < 0.01, F=5.906 \)) during abduction, and on the PD (\( p < 0.05, F=3.909 \)) and IS (\( p < 0.05, F=3.482 \)) during flexion. A significant shoulder effect was also found on the PD (\( p < 0.1, F=2.775 \)) during abduction. The increment effect was significant on all five muscles during both movements.

During abduction, the AD showed a similar variation in normalized sEMG data pattern as the one described above for the symptomatic shoulder vs CG. The asymptomatic shoulder had a faster evolution until the third motion increment (I₃: \( p < 0.05 \)). From the 90° elevation onwards the CG had a steep increase, while the symptomatic shoulder stabilized.

During flexion, the PG's asymptomatic shoulder had a slower evolution from the second increment onwards. Regarding the IS, once more, the PG's asymptomatic shoulder had a similar normalized sEMG data pattern to its contralateral shoulder, having a slower progression on the normalized sEMG signal until the third increment than the CG.

![Figure 4.31: Mean normalized sEMG data - Symptomatic vs Asymptomatic](image)
4.3.3 Pain/No Pain Subgroups

On this subsection the results description will be based on a comparison between the two subgroups, comparing the symptomatic shoulders of both subgroups, as well as the asymptomatic shoulders.

Onset Results

Figures 4.32 and 4.33 show the comparison between symptomatic and asymptomatic shoulders of Pain and No pain subgroups during abduction and flexion, respectively.

Figure 4.32: Onset - (a) Pain and (b) No Pain - Symptomatic vs Asymptomatic - Abduction

Figure 4.33: Onset - (a) Pain and (b) No Pain - Symptomatic vs Asymptomatic - Flexion
During abduction, no significant differences were found between subgroups, as they presented similar onset patterns for both shoulders. However, the SA showed notable differences between subgroups. For the Pain group the symptomatic shoulder had the SA activated much earlier than the asymptomatic shoulder, with onset values close the infraspinatus and AD. For the No Pain subgroup the onset values between shoulders were closer and the symptomatic shoulder had a later SA activation.

For flexion, more variations on the onset patterns can be observed, mainly for the infraspinatus and PD, however only for the latter statistical significance existed (p<0.05, F=8.962). For the infraspinatus, the difference between subgroups was on the symptomatic shoulders, since, on one hand the Pain group had a later activation, relative to the asymptomatic, and the No Pain group had a previous activation, almost at the same time as the motion began. Regarding the PD, the Pain group showed activation on both shoulder prior to the motion start, with earlier activation on the symptomatic shoulder. The No Pain group had a later activation on both shoulders, first the asymptomatic shoulder, with onset of approximately 0.1s and the symptomatic shoulder 0.25s.

**Normalized EMG Data Results**

Figures 4.34, 4.35 depict the mean normalized EMG data of all muscles, during abduction and flexion, comparing symptomatic shoulders between Pain and No Pain subgroups. The figures regarding the comparison between asymptomatic shoulders, A.9 and A.10, may be consulted on Appendix A.

During abduction and regarding the symptomatic shoulder, the interaction between subgroup and increment was significant on the MD (p<0.05, F=4.878) and the group effect significant on the PD (p<0.05, F=7.221) and SA(p<0.01, F=15.056). The increment effect was significant on all muscles.

For the MD, the No pain subgroup showed a similar normalized sEMG pattern to the CG, while the Pain subgroup had a faster evolution of the normalized signal until the third motion increment. The PD on the Pain subgroup shoulder had a faster increase from the first to the second motion increment. However it also stabilized earlier. The SA showed a faster evolution of the normalized sEMG on the Pain subgroup, especially from the second to the third motion increment.

For the asymptomatic shoulder comparison, only the PD showed a group effect (p<0.05, F=5.744), with higher mean normalized sEMG data for the Pain subgroup, especially on the second and third increments. The increment effect was also found significant on all muscles.

Regarding flexion, the comparison between symptomatic shoulders showed significant interaction between subgroup and increment on the MD (p<0.05, F=3.656) and a significant group effect on the PD (p<0.05, F=5.630). The MD showed a faster increase on the normalized sEMG from the second to third motion increment (p<0.01). Regarding the PD, the Pain group showed a faster evolution of the signal from the first to second increment, while the No pain subgroup had a faster increase from the second to third increment.

For the asymptomatic shoulder comparison, there was a significant increment, shoulder interaction on the MD (p<0.05, F=3.656). The MD showed a faster progression on the Pain subgroup on all three motion increments.
Figure 4.34: Mean normalized sEMG data - Pain vs No Pain subgroup - Symptomatic shoulder

(a) Anterior Deltoid

(b) Medial Deltoid

(c) Posterior Deltoid

Figure 4.35: Mean normalized sEMG data - Pain vs No Pain subgroup - Symptomatic shoulder

(a) Serratus Anterior

(b) Infraspinatus
Chapter 5

Discussion

In order to demonstrate the validity of the acquisition process and further analysis of the results, several comparisons were made, not only between groups, but also between movements and between subgroups. Both kinematic and sEMG analyses were sensitive enough to differentiate between asymptomatic and symptomatic shoulders within the PG, showing significant differences mainly on the STIR and STUR for the kinematic results, and on the deltoids for the sEMG activity pattern.

On the subsequent sections the results will be discussed following the organization adopted in the previous chapter, i.e., first looking at the CG and the comparison between movements, then comparing asymptomatic and symptomatic shoulders within the Pathological group and afterwards with the CG and, finally, the discussion of the subgroups results.

5.1 Abduction vs Flexion

The first analysis aimed at studying how the two movements differ in joint motion and, consequently, on SHR. Most of the joint rotations presented significant movement effect or significant interaction between movements and elevation/increment, suggesting relevant differences in joint rotations in order to elevate the arm in different planes.

Table 5.1 summarizes the comparison between movements on the CG. For this particular comparison the two-way repeated measures analysis used a MF and the EF, when considering joint motion (STIR, STUR, STT, GH), and an increment factor (IF) when considering the SHR. The interaction between factors is represented by movement elevation interaction (MEI) for the interaction between movement and elevation factors, and movement increment interaction (MII) for the interaction between movement and increment factors. For each dependent variable, Table 5.1 presents which of the factors and/or interaction was statistical significant, with the confidence interval represented with the * symbol. Because there were significant interaction of factors, Table 5.2 shows the post hoc analysis between movements on each individual elevation point or increment. Here, a qualitative representation of the results is presented. Taking as an example, the first entry of the Table, the STIR on the 30° of elevation was significantly higher during flexion (represented by the upward direction of the arrow), with a confidence interval
of p<0.1. The entries with no confidence interval symbol were not statistical significant.

Table 5.1: Two-way repeated measures analysis.

<table>
<thead>
<tr>
<th>Abduction</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>STIR</td>
<td>Flexion</td>
</tr>
<tr>
<td>STUR</td>
<td>MF***, EF**, MII*</td>
</tr>
<tr>
<td>STT</td>
<td>EF***</td>
</tr>
<tr>
<td>GH</td>
<td>MF***, EF***</td>
</tr>
<tr>
<td>SHR</td>
<td>MF*, IF*, MII*</td>
</tr>
</tbody>
</table>

Legend: STIR: Scapulothoracic Internal Rotation; STUR: Scapulothoracic Upward Rotation; STT: Scapulothoracic Tilt; GH: Glenohumeral Elevation; SHR: Scapulohumeral Rhythm; P-values: . - p<0.1; * - p<0.05; ** p<0.01; *** - p<0.001.

Table 5.2: Post Hoc analysis to the comparison between movements on each individual elevation point or motion increment.

<table>
<thead>
<tr>
<th>Abduction</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>STIR</td>
<td>Flexion</td>
</tr>
<tr>
<td>STUR</td>
<td>30 Flex ↗, Flex ↘, Flex ↗</td>
</tr>
<tr>
<td>STT</td>
<td>60 Flex ↘, Flex ↗, Flex ↗</td>
</tr>
<tr>
<td>GH</td>
<td>90 Flex ↘, Flex ↗, Flex ↗</td>
</tr>
<tr>
<td>SHR</td>
<td>120 Flex ↘, Flex ↗, Flex ↘</td>
</tr>
</tbody>
</table>

Legend: P-values: . - p<0.1; * - p<0.05; ** p<0.01; *** - p<0.001.

Firstly, it is important to note that despite the variations in joint kinematics the range of motion achieved on both movements remained similar. This result agrees with the fact that the shoulder joint as a whole has a wide range of motion, that can result from different combinations of joint rotations. The significant variations in SHR prove the different relative motion of the ST with the GH. However, this difference was mainly present in the first 60° of HT elevation, suggesting that the early phases of the motion are more important on defining the correct placement of the humeral head within the glenoid to achieve the intended movement.

In this work, STIR showed the highest variation across movements. However, it is important to note that the STIR shows the most variability across subjects and investigations (Ludewig et al., 2009b), so these results should be interpreted with caution. Nonetheless, it is expected to find higher STIR during flexion. The scapula, along with the clavicle, are responsible for aligning the glenoid in the appropriate plane to preserve congruency with the humeral head (Ludewig et al., 2009a; Fung et al., 2001). Maintaining a higher internal rotation may be needed to achieve the correct placement of the arm.
in the sagittal plane.

The STUR was the most predominant scapulothoracic motion on both movements, along with the GH elevation. Interestingly, despite the higher GH elevation for the same HT elevation during flexion, the STUR remained similar between movements, meaning that, during abduction, other joints rotations had a higher contribution on this movement to achieve the same HT elevation.

Since most of the significant differences between movements were identified on the first 60° of HT elevation, the early behaviour of the shoulder muscles has increased importance. Interestingly, the main responsible for each movement showed a faster activation and evolution of the normalized sEMG signal. The AD and PM during flexion, and the MD for abduction. The infraspinatus showed similar onset between movements. However the faster progression of the normalized sEMG signal during flexion at the first two motion increments, suggests a higher need for scapula stabilization during this movement.

For the following analysis it is important to consider that there are significant kinematic differences between abduction and flexion, as well as different muscle pattern activations and onsets. As such, UST may also show different repercussion depending on the plane of motion being studied. Most studies focused on the scapular plane. However, due to the wide shoulder range of motion, it is also important to study other planes of motion and their specific kinematic alterations.

5.2 Pathological vs Control

Table 5.3 shows the significant effects and/or interaction of effects between asymptomatic and symptomatic shoulders on the Pathological group and its subgroups.

Within the Pathological group, symptomatic and asymptomatic shoulders showed similar patterns on all joint rotations, which is supported by no significant interactions having been observed. However, the symptomatic shoulder showed a significantly less internally and upwardly rotated scapula during both movements. Despite the shoulder effect not being statistically significant, the scapula on the symptomatic shoulder was also less posteriorly tilted and the glenohumeral joint more elevated. All these results are in agreement with literature (Michener et al., 2003; Ogston & Ludewig, 2007; Turgut et al., 2016a; Lawrence et al., 2014). Regarding the comparison between groups, more noticeable differences were found between symptomatic shoulder and CG and significant variations were also found between asymptomatic shoulder and CG.

These variations on group and contralateral shoulder comparisons raise questions regarding both groups. Can the asymptomatic shoulder of the Pathological group be considered equivalent to the CG? How reliable can the CG be to represent a standard shoulder motion? In order to answer these questions and others that were already described or that may arise it is essential to deeply study and compare all results.

Looking first at the PG’s asymptomatic shoulder and its kinematic differences to the CG, Table 5.4 describes the results of the statistical model analysis. For the group effect, significant differences were found only on STIR. However, it is interesting to notice that all joint rotations during both movements had the same relation between PG’s asymptomatic shoulder and the CG as between symptomatic shoulder...
When interpreting these results, it is important to consider that most of the subjects on the PG performed heavy or repetitive shoulder movements in their work and daily activities (6 out 9), hence, they may have developed compensation strategies on both shoulders in order to continue working regardless of the pain or discomfort felt (Ludewig & Cook, 2000). Despite a patient presenting stronger symptoms on one of its shoulders, the contralateral shoulder may not be completely healthy, so it may not be enough to study its kinematic alterations relative to the supposed asymptomatic shoulder. Therefore, it is also important to consider the possible risk factors present on each patient daily lives.

Eight out of the nine patients had the symptomatic shoulder coinciding with the dominant shoulder, only one patient, that did not present a profession prone to develop shoulder problems, had the non-dominant shoulder as the symptomatic one. Despite the apparent equal exposure on both shoulders to risky shoulder movements during daily activities, the dominant shoulder tended to be more affected, possibly due to its preferential use on a daily bases. Even though similar shoulder kinematics between contralateral shoulders are expected to be found in healthy people, subjects that perform routinely overhead movements or use their arms in elevated positions may have increased risk for developing shoulder problems on their dominant shoulder, as it is more exposed to repeated movements (Yamamoto et al., 2010).

When comparing the PG’s symptomatic shoulder with the CG (Table 5.5), more notable differences were found during abduction. Even though only the STUR presented statistical significance on the group factor, it is also important to consider the variations in all joint rotations. Upward rotation and posterior tilting of the scapula have an extremely important role on arm elevation, as upward rotation elevates the lateral acromion preventing impingement under the lateral acromial edge, and posterior tilting elevates the anterior acromion, which is the predominant site of impingement (Neer, 1983). A decrease on these two joint rotations, may prevent the anterior aspect of the acromion from moving away from the humeral head during arm elevation and, consequently, contributing to a reduction of the subacromial space and external RC compression (Ludewig & Cook, 2000). Additionally, the decrease in STUR may also lead to an increase in GH elevation as a compensatory response (Lawrence et al., 2014).

As studied in Chapter 2, RC tendon compression is one of the main factors that contribute to UCT, hence, the altered shoulder kinematics discussed during abduction may be a possible cause of the UST. On the other hand, it has been observed similar alterations in shoulder kinematics between asymptomatic shoulder and the CG. Yet, in 8 out of 9 patients, the dominant shoulder had more evident kinematic alterations, which may suggest that the altered kinematics may be caused by the development of the pathology and not its precursor. Further research is needed to conclude whether kinematic alterations in subjects with symptoms of UST are precursors to the development of the pathology or a result of the condition.
Table 5.3: Comparison between Asymptomatic and Symptomatic shoulder.

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<tr>
<th></th>
<th>Abduction</th>
<th>Flexion</th>
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<tbody>
<tr>
<td>Symptomatic</td>
<td>STIR</td>
<td>STUR</td>
</tr>
<tr>
<td>Pathological group</td>
<td>GF ., EF**</td>
<td>GF**, EF***</td>
</tr>
<tr>
<td>Pain subgroup</td>
<td>EF***</td>
<td>EF***</td>
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<tr>
<td>No pain subgroup</td>
<td>EF***</td>
<td>EF***</td>
</tr>
</tbody>
</table>

Legend: GF: Group Factor; EF: Elevation Factor; GEI: Group Elevation Factor; IF: Increment Factor; GII: Group Increment Interaction P-values: . - p<0.1; * - p<0.05; ** p<0.01; *** - p<0.001.
Table 5.4: Comparison between Control and Asymptomatic shoulder.

<table>
<thead>
<tr>
<th>Control group</th>
<th>Flexion</th>
</tr>
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<tbody>
<tr>
<td>Abduction</td>
<td></td>
</tr>
<tr>
<td>STIR</td>
<td>STUR</td>
</tr>
<tr>
<td>Pathological Group</td>
<td>GF*, EF***</td>
</tr>
<tr>
<td>Pain Subgroup</td>
<td>EF**, GF**</td>
</tr>
<tr>
<td>No Pain Subgroup</td>
<td>EF***</td>
</tr>
</tbody>
</table>

Legend: GF: Group Factor; EF: Elevation Factor; GEI: Group Elevation Factor; IF: Increment Factor; GII: Group Increment Interaction P-values: . - p < 0.1; * - p < 0.05; ** p < 0.01; *** - p < 0.001.

Table 5.5: Comparison between Control and Symptomatic shoulder.

<table>
<thead>
<tr>
<th>Control group</th>
<th>Flexion</th>
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<tbody>
<tr>
<td>Abduction</td>
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<tr>
<td>STIR</td>
<td>STUR</td>
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<tr>
<td>Pain Subgroup</td>
<td>EF**, GF**</td>
</tr>
<tr>
<td>No Pain Subgroup</td>
<td>EF***</td>
</tr>
</tbody>
</table>

Legend: GF: Group Factor; EF: Elevation Factor; GEI: Group Elevation Factor; IF: Increment Factor; GII: Group Increment Interaction P-values: . - p < 0.1; * - p < 0.05; ** p < 0.01; *** - p < 0.001.
During flexion, fairly different results were found relative to abduction. On one hand, the STUR did not vary as much between either groups or contralateral shoulders. On the other hand, the GH elevation results were not consistent to what was described for abduction. Furthermore, the symptomatic shoulder GH elevation did show the same relative behaviour to the CG as it did to its contralateral shoulder. The plane of motion influences the pattern of shoulder kinematics (Struyf et al., 2011; Lawrence et al., 2018), hence, it is important to adapt the interpretation of the results depending on the plane of motion being considered. By studying the effect of the glenohumeral plane of elevation on subacromial proximity, Lawrence et al. (2018) suggested that movements on the sagittal plane are more protective than movements on the scapular or frontal plane, meaning that it is less likely to have mechanical impingement during flexion than in any other movement on the scapular or frontal plane. This protective behaviour may be associated to the clearance of the supraspinatus tendon from under the coracoacromial arch. Additionally, Lawrence et al. (2018) also concluded that the subacromial compression risk is highest below the 60° of HT elevation, since from that point onwards the supraspinatus tendon has already cleared the coracoacromial arch and is no longer at substantial risk of compression in most subjects. The little variation in STUR may be due to the lower probability of causing impingement during flexion.

Alterations in joint motions should not be considered in isolation. As such, it is important to analyse their relative rotations that ultimately lead to the full range of motion. SHR provides that relation between the two joint responsible for most of the motion during arm elevation, STUR and GH. Just as described for these two joint rotations individually, the differences between groups was higher than between contralateral shoulders on the PG.

Despite the higher differences in joint angles being mostly present on the end range of the motion, the significant kinematic alterations were found on the initial stage of the movement, where the joint rotation rates have a higher discrepancy. Because the SHR by definition is a rotation rate between two joints, it allows for an analysis where these alterations in joint rotations rates are more prominent and, hence, complement the analysis of the difference in rotation angle.

The first two motion increments showed the largest variations in results for both movements. The higher SHR values on the first two motion increments for the symptomatic shoulder suggest a higher contribution of the GH joint during the movement initiation until 60° of HT elevation. On the last two increments, the symptomatic shoulder decreased its SHR, meaning that from the 60° of HT elevation the scapula contributes more for the arm elevation, resembling more the asymptomatic shoulder kinematics. This decrease in SHR from the 60° of HT elevation onwards may represent an approximation of the PG shoulder kinematics to the CG one, since the GH elevation decreases and the STUR increases to values closer to the CG ones. This behaviour was especially noticeable during abduction.

Lawrence et al. (2014) also showed similar results, where the symptomatic group had a less upwardly rotated scapula until the 90° of HT elevation, and then both symptomatic and asymptomatic groups showed similar STUR. The authors suggested that this apparent compensation can be explained by a continuous higher sternoclavicular elevation, relative to the asymptomatic group, contributing to the STUR and eventually leading to a similar STUR of the asymptomatic group. It is also noteworthy that, at the latter stages of motion, the supraspinatus does not have such a prominent role on shoulder elevation,
hence, the differences between shoulders may start to fade as the supraspinatus action decreases.

The SHR for the complete motion can, therefore, give some clues to the relative rotation between GH and STUR throughout the HT elevation. However, the results presented for each individual increment show how the SHR can vary at different intervals of the movement. As such, it is very important to study different stages of the motion, in order to analyse how the shoulder joint behaves throughout the complete range of motion of HT elevation.

It is also important to consider the different characteristics of each group that may influence the kinematic results, namely, the difference in age and in body mass index (BMI) values. The PG is composed of subjects in average 23 years older than the subjects on the CG, and their average BMI places these subjects on an overweight category, while the CG subjects are in a Normal (Healthy weight) category. Both age and weight are important factors to consider when studying shoulder movement, as they have been proven to influence shoulder kinematics. Ageing can have a negative effect on tendons elasticity and overall tensile strength, being a significant factor in the intrinsic pathoetiology of RCT (Seitz et al., 2011). Additionally, high BMI can influence the scapulothoracic motion, increasing upward rotation with humerothoracic elevation (Gupta et al., 2013).

Alterations in shoulder kinematics have also been associated with muscle dysfunction, which can be a result of muscle strength deficits or muscle activation alterations (Castelein et al., 2017). However, it is important to notice that, even within the studies that found altered shoulder muscle activity, the results are not unanimous. The EMG is highly variable and is dependent upon several variables that can not be fully controlled in an experimental setting. Hence, a proper normalization procedure is essential to analyse and compare subject groups (Sousa & Tavares, 2012). Nonetheless, it should be noted that the different normalization methods have relevant implications on the interpretation of the results attained. Caution should be taken when interpreting and comparing EMG results that did not follow similar methodologies.

In this work, a mean dynamic method was used to normalize the sEMG data, given the patients inability to perform a maximum contraction. Although this method does bring some advantages, as a decrease in inter-subject variability, it tends to remove the true biological variation within a group (Sousa & Tavares, 2012). Hence, using this method can make it harder to identify differences between the two groups.

The muscle normalized sEMG pattern varied mainly on the deltoids. However, the most noticeable variations were not found on the motion stage where the supraspinatus has a more active role on elevating the shoulder, the first 30° of HT elevation. At this stage, increased deltoid action may compromise the GH joint stability by superiorly translating the humerus (Heuberer & Kranzl, 2015; Moore et al., 2014). Therefore, the faster deltoid normalized sEMG signal on the symptomatic shoulder, later in the movement, may represent a motor strategy to increase the GH elevation to compensate for the lack of STUR.

Regarding the SA, conflicting results to what is presented in the literature were found. The SA was one of the last muscles to activate for both CG and PG, which is not what should be expected given the dual role of the SA as upward rotator and stabiliser. Several other authors have found significant
variations on the SA onset and normalized sEMG pattern between symptomatic and asymptomatic shoulders (Wadsworth & Bullock-Saxton, 1997; Moraes et al., 2008; Leong et al., 2017). However, the values described in the literature are closer to the motion initiation or even before it, both on control and pathological populations (Wadsworth & Bullock-Saxton, 1997; Wickham et al., 2010; Leong et al., 2017).

The difference in SA onsets results may be related with the use of different methodologies to the ones used in the literature. Wickham et al. (2010) used wire intramuscular electrodes, possibly having more reliable data. Additionally, both Wadsworth & Bullock-Saxton (1997) and Leong et al. (2017) studied competitive freestyle swimmers populations, which in theory would allow for a clearer data acquisition given the higher muscle development and lower body fat percentage. Furthermore, despite the SA being one of the strongest muscles of the pectoral girdle, because the only superficial parts of serratus anterior accessible for surface electrode placements are over the slips attaching to the ribs, it is likely that geometric displacement would have a stronger effect on the surface electrode recordings of this muscle (Hackett et al., 2014). Additionally, the fatty tissue present on the placement region of the SA electrode can be higher than on other more superficial muscles, especially on women, increasing the noise component of the signal. Besides having a higher proportion of female subjects on the PG, the average BMI is already considered to be on the overweight category, so the fatty tissue effect on the sEMG signal is even higher on this group. Hence, it is important to take caution when interpreting the SA normalized sEMG data results.

For the SA normalized sEMG results, most of the literature points to a decrease mean normalized sEMG data on the SA for pathological groups (Diederichsen et al., 2009; Ludewig & Cook, 2000). However, the work of both Bandholm et al. (2006) and Roy et al. (2008) also showed no significant variations in SA normalized sEMG data between an impingement syndrome and CGs. Similarly, the IS showed little variation in mean normalized sEMG data, result that agrees with the work of Roy et al. (2008) and Lopes et al. (2015). These results suggest that subjects with UST may not always have a compromised ability to control muscle force and, as such, can present different motor strategies in order to perform dynamic movements.

It is also noteworthy that the PG’s symptomatic shoulder had a later activation of the IS relative to the medial deltoid during abduction. The IS, as one of the RC muscles, is responsible for counteracting the unwanted superior transitory pull of deltoid on the humerus. However, in a healthy population, it as been shown to activate only from the 10° of HT elevation onwards, as until that point the supraspinatus, being one of the main movement initiators, may be enough to offset the deltoid forces during the early stages of abduction (Wickham et al., 2010). A later activation of the infraspinatus, relative to the deltoids, may suggest a lack of coordination between these muscles. In order to have a better understanding of the effects of uncoordination between RC and deltoid muscles, future studies should also consider the remaining RC muscles.

Additionally, it should be pointed out that the significant differences found between between CG and the PG’s asymptomatic shoulder resemble the differences found between CG and symptomatic shoulder. There may be an adaptation of the asymptomatic shoulder muscle activity to the alter motor strategies of the symptomatic shoulder.
5.3 Pain vs No Pain

Tables 5.6 and 5.7 show the significant effects and/or interaction of effects, between Pain and No pain subgroups, for both asymptomatic and symptomatic shoulder, respectively. Here, the results of both subgroups will be compared with each other, in order to study the possible influence of pain and restricted motion on the shoulder kinematics and muscle normalized sEMG data pattern.

Table 5.6: Comparison Pain vs No pain subgroups - Asymptomatic shoulders.

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<td>STIR</td>
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<td>Pain</td>
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<tr>
<td>Group</td>
<td>GF***,</td>
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<td></td>
<td>EF*</td>
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Legend: GF: Group Factor; EF: Elevation Factor; GEI: Group Elevation Factor; IF: Increment Factor; GII: Group Increment Interaction P-values: . - p < 0.1; * - p < 0.05; ** p < 0.01; *** - p < 0.001.

Table 5.7: Comparison Pain vs No pain subgroups - Symptomatic shoulders.

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<td>STIR</td>
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<tr>
<td>Pain</td>
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<td></td>
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<tr>
<td>Group</td>
<td>GF***</td>
<td>EF***</td>
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Legend: GF: Group Factor; EF: Elevation Factor; GEI: Group Elevation Factor; IF: Increment Factor; GII: Group Increment Interaction P-values: . - p < 0.1; * - p < 0.05; ** p < 0.01; *** - p < 0.001.

The Pain and No pain subgroups, showed no significant differences between contralateral shoulders. The sample size on these subgroups is small, so it is harder to find statistically significant results, nonetheless, other studies have found similar shoulder kinematics between symptomatic and asymptomatic shoulders on a Pathological population (Hbert et al., 2002; Graichen et al., 2001).

The similarity in shoulder kinematics may be explained by several different factors. Firstly, it is important to remember that the results depend greatly on the methodological acquisition process, such as a symmetrical initial posture, and on the subjects performance of the asked movements. On one hand, an asymmetrical resting posture can lead to errors when calculating the joint angle motion, since the reference points vary between shoulders (Hbert et al., 2002). Despite possible differences in shoulder kinematics, an asymmetrical resting posture may result on a apparent similar results. On the other hand, poor posture, throughout the movement, has been associated with shoulder kinematic alterations. Ke-
Baetse et al. (1999) reported decreased scapular upward rotation and posterior tilting in the slouched position. As such, even though the resting posture may be symmetrical, it may change and restrict the degrees of freedom of the scapula during arm motion (Hbert et al., 2002).

However, when comparing the subgroups with each other, several joint rotations showed differences with statistical significance. Interestingly, the No pain group had similar joint kinematics to the CG, hence the subjects on the Pain subgroup were the main responsible for the kinematic variations previously described between PG and CG. Looking solely to the kinematic differences between subgroups, the results suggest that the constricting pain felt by the subjects on the Pain subgroup may be a significant factor to their kinematic alterations.

For the muscle mean normalized sEMG data results, the Pain subgroup showed, in general, a faster evolution of the normalized sEMG signal than the No pain subgroup, especially on the SA. Since this muscles plays such an important role on stabilizing and rotating superiorly the scapula (Moraes et al., 2008; Wickham et al., 2010), a faster evolution of the normalized sEMG signal throughout the motion may represent an adaptation to pain, in order to follow the higher deltoid sEMG data progression, maintaining the SHR. Interestingly, the SHR results show how similar the relative GH and ST rotations are between subgroups, despite the significant differences in their individual angle rotations.

The lower capability of ST rotation may be due to other ST muscles dysfunction. The trapezius muscles are the prime movers of the scapula upward rotation and on patients with UST it is expected to find slower normalized sEMG data evolution on the inferior trapezius and a higher on the superior trapezius, when compared to healthy subjects (Phadke et al., 2009). Despite not being possible to confirm this result on the Pathological group, since this muscles were not considered on this analysis, it is possible that despite the faster increase of the normalized sEMG signal of the SA on the Pain subgroup, their trapezius had a stronger dysfunction resulting on lower STUR.

Pain seems to have an important effect on the shoulder kinematics, as the alteration in shoulder kinematics was more prominent on the Pain subgroup. However, it is not possible to conclude if pain is a causing or contributory factor to the kinematic alterations or if it is a result of a more significant kinematic alteration or muscle dysfunction. As such, it is important to stress, that a more in depth study would be needed to take any conclusions regarding the effect of pain on the shoulder kinematics of a UST population. The results were presented provided some clues to a possible differentiation of patients with different symptoms or in different stages of the pathology. In future studies it would be important to homogenize the PG in order to fully understand the effects of UST on a variety of different symptomatology.
Chapter 6

Conclusion

6.1 Main Conclusions & Clinical Applications

Shoulder disorders rank amongst the most prevalent disorders of the human musculoskeletal system, with RCT being responsible for a majority of the cases, thus representing a worldwide important medical and socio-economic burden. Despite the recent interest in the study of shoulder joint kinematics by the international scientific community, knowledge about the motion of the joint is still limited, especially when we consider kinematics of the pathological shoulder. This lack of insight may be explained by several factors, from the complex nature of the joint itself, to the wide variety of different symptoms and movement limitations that each disorder can provoke.

This work aimed to contribute to the growing pool of knowledge on the kinematics and muscle sEMG normalized data patterns of healthy and pathological shoulders, the latter being represented by a group of shoulders from 9 patients clinically diagnosed with RCT. With this intention, an experimental protocol was designed using an optoelectronic tracking system to study shoulder kinematics and a sEMG system to study the shoulder muscles.

6.1.1 Shoulder Kinematics

The results of the protocol showed differences between the kinematics of the shoulder joint motion in the PG (PG asymptomatic shoulders versus PG symptomatic shoulders) as well as when comparing the CG with the PG (CG shoulders versus PG symptomatic shoulders), the latter being the most noticeable. Regarding the plane of movement, isolated abduction was the most affected motion, with the symptomatic shoulders of the PG having lower scapulothoracic upward rotation, lower external rotation, lower posterior tilting, and higher glenohumeral elevation, when compared with the shoulders of the CG. Additionally, SHR analysis showed that the first 60 degrees of humerothoracic elevation experienced the biggest kinematic alterations, with the symptomatic shoulders of the PG having a higher contribution of the glenohumeral joint to the humerothoracic elevation, when compared to the shoulders of the CG.

The supraspinatus has a more prominent role on the early stages of the motion, as such the increased kinematic alterations during the first 60° of abduction may be associated to the higher risk of
subacromial compression during this stage. From the 60° onwards, the supraspinatus tendon has already cleared the coracoacromial arch and is no longer at substantive risk for compression. Hence the convergence of the symptomatic shoulder kinematics to the CG at the latter stages of the movement.

Differences were also found between the kinematics of the asymptomatic shoulders and symptomatic shoulders of the PG, with the symptomatic shoulder showing lower scapulothoracic internal rotation, lower posterior tilting, and higher glenohumeral elevation in both planes of movement (isolated flexion and isolated abduction). Regarding SHR analysis, differences between the asymptomatic and symptomatic shoulders of the PG were also higher in the first two increments of the isolated abduction motion (0-30 degrees; 31-60 degrees), with the difference being most noticeable in the second movement increment (31-60 degrees).

The kinematic differences reported in the results of this protocol suggest that clinicians who work in the area of musculoskeletal disorders should focus their attention on the first two increments of isolated abduction motion when examining patients who have been referred for further evaluation of RCT. However, it should also be noted that the asymptomatic shoulders of the PG also showed differences when compared to the shoulders of the CG. These were similar to the ones observed between the symptomatic shoulders of the PG and the shoulders of the CG, albeit smaller. As such, clinicians should bear in mind that patients with a diagnosis of RCT may also show differences in the motion of the non-affected shoulder.

6.1.2 Shoulder Muscles sEMG Data

The results of the protocol showed differences between the normalized sEMG data patterns of shoulder muscles in the PG (PG asymptomatic shoulders versus PG symptomatic shoulders) as well as when comparing the CG with the PG (CG shoulders versus PG symptomatic shoulders). The most significant differences were found in the normalized sEMG signal of the deltoid muscles, with faster progressions mostly from the 30 degrees of humerothoracic elevation onwards, suggesting an adaptation of the motor strategies to elevate the shoulder.

The serratus anterior and infraspinatus muscle showed little variations during flexion. The SA had faster evolution of the normalized sEMG signal on the symptomatic shoulder when compared to its contralateral, and the IS slower evolution of the normalized sEMG signal on the symptomatic shoulder when compared to the CG on the second and third motion increments (31-60 degrees; 61-90 degrees). These results suggest that subjects with UST may not always have a compromised ability to control muscle force, and as such can present different motor strategies in order to perform dynamic movements.

The results of the sEMG study seem to point towards an adaptation of the shoulder muscles to the patient pathological condition, rather than a muscle deficit.

6.1.3 Pain Influence on Shoulder Motion

The results of the protocol showed more significant alterations in shoulder kinematics and in the normalized sEMG data patterns of shoulder muscles when a subgroup analysis of the PG symptomatic
shoulders was performed. Although insufficient for further considerations at the present time, these results suggest that pain may have an important role on the normalized sEMG data pattern of shoulder muscles as well as on shoulder joint kinematics beyond the limitation in humerothoracic elevation.

6.1.4 Limitations

Firstly, the small sample size limits the statistical power to detect group differences, especially when considering the influence of pain in shoulder joint kinematics and the normalized sEMG data patterns of shoulder muscles.

Secondly, subjects on the CG and PG have important demographic (average age of subjects) and body composition (average BMI) differences, which have been proven to influence shoulder joint kinematics in previous studies. Additionally, a higher BMI may also compromise the reliability of the sEMG data, which is important considering that on average PG subjects BMI was over 25, which places them in the overweight category.

Thirdly, in regards to the instrumentation process it is important to consider that manual positioning of both reflective markers and sEMG sensors always entails placing errors. Furthermore, when replacing the scapula markers during the second stage of calibration, the marker placement becomes harder due to the rotated position of the scapula, thus more propitious to error. In addition, when considering the study of shoulder kinematics it is important to consider that the present protocol was designed to study shoulder joint movement to a maximum of 120 degrees of humerothoracic elevation, given the loss of data reliability from that point onwards.

Lastly, the sEMG normalization process tends to remove the true biological variation within a group (Sousa & Tavares, 2012), becoming harder to identify differences between the two groups.

6.2 Future Work

Studying pathological shoulder kinematics is important not only to better understand the different pathology mechanisms and how they can be more easily identified, but also to create personalised rehabilitation programmes and improve its success rates. As such, future work should also focused on following patients through a rehabilitation programme, based on their individual kinematic and muscle normalized sEMG data alterations. Nonetheless, before focusing on the effects of a rehabilitation programme on a pathological population, a more in depth study should be taken in a standardize healthy shoulder kinematics.

There are several factors that have been proven to influence shoulder kinematics, namely anatomic factors, age, BMI, the practice of sports, etc. Hence, it is very important to first study the healthy shoulder kinematics that may represent the most standardized movements as possible, then understand what may be benign kinematic alterations and finally use this knowledge to create the control groups that provides the best comparison for the pathological population in study. To do so, different control groups, ideally with more than the 15 subjects here selected, with different characteristics that represent possible
variations of a healthy population, should be analysed.

Regarding the movements studied, it would also be important to consider daily movements, as they tend to be the most faithful representation of the patient limitations and they may provide more information to better align rehabilitation programmes relative to the patient needs. On the experimental stage of this work, several daily movements were acquired. However, due to the intentional focus on simpler motions, they were not analysed.

Movement of the shoulder complex is carried out by several muscles. However, due to the limitation of time for each acquisition session, for the PG only 5 muscles were analysed. Nonetheless, the trapezius muscle has an essential role on moving and stabilizing the scapula. It has also proven to be one of the affected muscles on RCT, as such, future studies should also include them.

The Pain and No pain subgroup analysis led to an important question, is pain a result or consequence of RCT? Pathological groups with a higher number of subjects, the same diagnosis and similar symptomologies, should be further studied in order to study the role of pain on shoulder kinematics.
References


Codman, E.A. 1934b. The shoulder: rupture of the supraspinatus tendon and other lesions in or about the subacromial bursa. 1 edn.


Hancock, Roger P. 1968. Interfacial Couplings for Man-Machine System. *U.S. Army Medical Biomechanical Research Laboratory Walter Reed Army Medical Center.*


Hosseinimehr, Seyed Hossein, Anbarian, Mehrdad, Norasteh, Ali Asghar, Fardmal, Javad, & Khosravi, Mohammad Taghi. 2015. The comparison of scapular upward rotation and scapulohumeral rhythm


Appendix A

Results

A.1 Kinematic Results

Figure A.1: Pain vs No Pain Group - ASS - Scapulothoracic Internal Rotation - Abduction

Figure A.2: Pain vs No Pain Group - ASS - Scapulothoracic Upward Rotation - Abduction
Figure A.3: Pain vs No Pain Group - ASS - Scapulothoracic Posterior Tilting - Abduction

Figure A.4: Pain vs No Pain Group - ASS - Glenohumeral Elevation - Abduction

Figure A.5: Pain vs No Pain Group - ASS - Scapulothoracic Internal Rotation - Flexion
Figure A.6: Pain vs No Pain Group - ASS - Scapulothoracic Upward Rotation - Flexion

Figure A.7: Pain vs No Pain Group - ASS - Scapulothoracic Posterior Tilting - Flexion

Figure A.8: Pain vs No Pain Group - ASS - Glenohumeral Elevation - Flexion
A.2 EMG Results

Figure A.9: Mean normalized sEMG data - Pain vs No Pain subgroup - Asymptomatic shoulder

(a) Anterior Deltoid
(b) Medial Deltoid
(c) Posterior Deltoid

Figure A.10: Mean normalized sEMG data - Pain vs No Pain subgroup - Asymptomatic shoulder

(a) Serratus Anterior
(b) Infraspinatus