

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

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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 Patient group's (PG) symptomatic shoulder.

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

1. Introduction

Shoulder disorders are one of the main causes for seeking medical care, translating in many cases in movement restrictions that lead, ultimately, to limitations in daily and work activities. Rotator cuff tendinopathy (RCT) is considered the most common shoulder disorder as it may affect up to 30% of the population [1]. 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 [17]. There is high uncertainty as to what are the causes of RCT, as well as to what causes different patients to experience such different levels of pain and functional limitation.

Subacromial impingement has long been proposed as a mechanism of shoulder pain and one of the possible causes of RCT. It is described as a repeated compression on the RC tendons and associated tissues within the subacromial space [25]. However, the causes and/or effects of this mechanism are still poorly understood. Most studies of shoulder biomechanics have focused on healthy

shoulder kinematics, and the ones that did study pathological kinematics still present some contradictory results, specially when considering the Scapulohumeral Rhythm (SHR). The SHR is an important variable to consider, since it describes the relative motion of the scapula to the humerus during arm elevation, allowing for a relative analysis between the two main joint rotations in humerothoracic elevation, the scapulothoracic upward rotation (STUR) and the glenohumeral (GH) elevation.

Furthermore, traditional clinical tests, based on observation and clinical examination, often present a weak correlation with the specific diagnosis, while imaging methods cannot provide a fully reliable diagnosis since there is no direct association between symptoms and structural failure [17]. In order to tackle the referred limitations at the hospitalar setting, this work 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.

2. Shoulder Anatomy and Biomechanics

2.1. Shoulder Anatomy

The shoulder joint is composed of four joints and three 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. Together they form three anatomical and one physiological joint, namely the sternoclavicular (SC), the acromioclavicular (AC), the glenohumeral joint (GH) and the scapulothoracic joint, which is the physiological joint.

There are nine muscles that cross the shoulder joint, all of them originating on the scapula except for the pectoralis major and latissimus dorsi. Four of the nine muscles make up the Rotator Cuff (RC), namely the Supraspinatus, Infraspinatus, Teres minor, and Subscapularis. This complex of muscles is an essential component of the shoulder joint, and the most common source of injury of the musculoskeletal system [6].

This complex shoulder structure allows for 3 degrees of freedom. However its stability becomes more intricate too. Contrary to the lower body joints, shoulder stability is mostly achieved by its muscles [20]. 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 [4]. Thus, it is essential to have a deep comprehension of both shoulder anatomy and biomechanics in order to understand pathology mechanisms and how to develop effective treatment programmes.

2.2. Healthy Biomechanics

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.

In healthy individuals it is known that during elevation of the arm, the scapula should upwardly rotate and posteriorly tilt on the thorax [19, 9]. 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, with values raging from 1.7 to 3.4 in the scapular plane.

Regarding the muscle activity, abduction is believed to be mostly performed by the supraspinatus and anterior and middle deltoid muscle [13, 31, 2]. The subscapularis was also found to play an impor-

tant role during abduction, stabilising the shoulder anteriorly by compressing the humeral head in the glenoid fossa [33] and also showing potential abduction capability. Additionally, the infraspinatus shares the load with the supraspinatus, and the trapezius also showed a stabilising role, promoting scapula movement throughout the whole abduction exercise [33].

Flexion is mainly performed by the supraspinatus and anterior deltoid [13, 31]. 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. Pathological Biomechanics

Scapular and glenohumeral kinematics are altered in unstable or pathological shoulders [26, 22]. This 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 [22] and/or increase the subacromial space [21]. 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 [23].

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 [23, 26, 27, 14]. Regarding glenohumeral kinematics, an increase on glenohumeral elevation as been observed [14].

Despite the significant number of studies on symptomatic subjects, there are very few that use the SHR to characterize kinematic differences between populations and their results are contradictory [8, 11, 22]. Nonetheless, given the kinematic alterations found in RCT populations an increase on SHR for the symptomatic population should be expected, result also found by [22].

One of the factors that may lead to RCT is a lack of coordination and neuromuscular balance between the RC muscles [5]. 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.

Additionally, there may also be alterations on the activation pattern of the shoulder muscles. Despite not existing a consensus on the variation in muscle onset patten between asymptomatic and symptomatic populations, deficits in the activity onset of scapular muscles when compared to the asymptomatic subjects were found, along with slower Lower Trapezius (LT) and Serratus Anterior (SA) activations [16].

3. Experimental and Computational Methods

Twenty-four subjects (15 healthy and 9 patients) were recruited from the university setting and from Hospital Curry Cabral, in Lisboa, respectively, to participate in this study. The control group (CG) did not present any history of shoulder pathologies or pain. Because RCT is a broad definition of a group of different tendinopathies, it is important to study the effect of each tendinopathy on the shoulder motion and muscle function separately. Here, participants of the pathological group (PG) had been previously diagnosed with unilateral supraspinatus tendinopathy (UST), a particular case of RCT, by means of an ultrasound scan.

The 3D kinematics of the shoulders was acquired at the Lisbon Biomechanics Laboratory by means of an opto-electronic measurement system composed of 14 Qualisys digital infrared cameras. Following the recommendations of the International Society of Biomechanics, 22 retro-reflective markers were used to track the thorax, clavicle, scapula, humerus and forearm [34] of each CG subjects dominant arm, and of both arms of the PG. Complementarily, two marker clusters were placed per arm: one on the upper arm and another one on the acromion, according to the acromial tracker method [28].

Because the acromion cluster used by Shaheen et al. [2010] presented very high noise interference, a custom made acromiom cluster was design based on the original one, with variation on the length and inclination of the bars holding the markers.

Using a Delsys Myomonitor III system, sEMG was recorded synchronously with the movement for 8 muscles on the CG. The targeted muscles were the anterior (AD), medial (MD) and posterior (PD) deltoid, the superior (SupT) and inferior (IT) trapezius, the serratus anterior (SA), the pectoralis major (PM) and the infraspinatus (IS). For the PG only the AD, MD, PD, SA and the IS muscles were measured. After giving informed consent, all subjects were instructed to perform abduction in the frontal plane and flexion in the sagittal plane, until their maximum elevation, without moving the trunk. The subjects on the CG repeated each movement 10 times and on the PG 7, with some exceptions due to pain or fatigue.

Software developed in house was used to gen-

erate the anatomical co-ordinate 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.

In this work, the shoulder joints analysed were the scapulothoracic (ST) and glenohumeral (GH) joint. For the ST all three rotations were studied, namely, upward/downward rotation, protraction/retraction, and anterior/posterior tilt. For the GH joint only its elevation angle was analysed. Shoulder kinematics were evaluated comparing these joint angles and the scapulohumeral rhythm (SHR).

In order to calculate the SHR, the movement was divided in four motion increments, and a linear regression was applied to the values of glenohumeral versus scapulothoracic angle on each increment. Then slope of the linear regression corresponded to the SHR. Additionally, another linear regression was 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 the following intervals of HT elevation:

- First Increment (I1) - [Min - 30°];
- Second Increment (I2) - [30° - 60°];
- Third Increment (I3) - [60° - 90°];
- Fourth Increment (I4) - [60° - 90°];

3.1. Statistical Analysis

Descriptive statistics were calculated across groups for each joint angle, SHR and sEMG results (onset and mean normalized sEMG data). The experimental study design used a two-way repeated-measures analysis of variance (ANOVA) model for all results mentioned above, except for muscle onset. For this value, one-way repeated-measures analysis of variance (ANOVA) was used.

Regarding the two-way repeated-measures applied to the joint rotation analysis, comparisons among groups/subgroups used a group factor (pathological and control group; Pain or no pain subgroups) and an elevation angle factor (30°, 60°, 90°, and 120° of HT elevation). Comparisons between contralateral shoulders used a shoulder factor (symptomatic and asymptomatic) and an elevation factor. For the SHR and mean normalized sEMG data analysis, because these two results refer to an elevation interval and not a specific elevation angle, the elevation factor is replaced by an increment factor (I1, I2, I3 and I4). The second factor on both group and contralateral shoulder analysis remains the same as for the joint rotation analysis.

For the one-way repeated-measures analysis of the onset, no elevation/increment factor were considered as this value does not depend on elevation. Hence, a group factor was used when comparing groups, and a shoulder factor when comparing contralateral shoulders.

In the presence of significant interactions, a pairwise comparison was applied at each level of the interacting factor. 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 or increment.

Regarding the sEMG data, a set of MATLAB built-in routines were used to process the sEMG data. For each subject trial, a bandpass 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. Each trial was normalized using its mean normalized sEMG data. Thus, each representation of the sEMG mean normalized sEMG data is as a percentage of the m-DYN (% m-DYN). 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 analysis were done:

- Onset analysis;
- Mean normalized sEMG data analysis;

The onset time was defined as when 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 mean normalized sEMG data was defined as the averaged value of the processed sEMG data across the same 30 intervals defined above (Increments 1 to 4).

4. Results

The results will be organized in two sections, first the description of the Pathological group, comparing symptomatic with asymptomatic shoulder, as well as with the Control group; and then, 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.

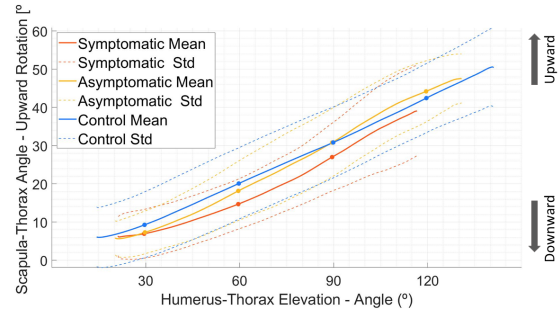
4.1. Kinematics

4.1.1 Control and Pathological Group

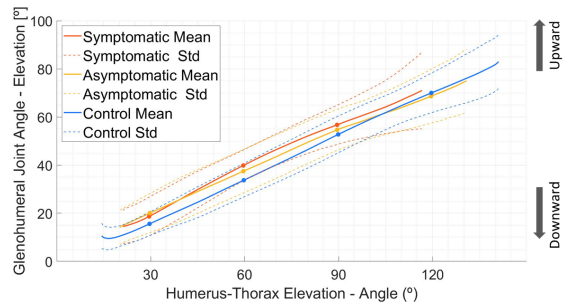
This analysis included 15 Control individuals (8 men, 7 women), with a mean age of 24 ± 5.5 years (range, 18-39 years). Their mean height was 1.72 ± 0.10 m and weight was 67.97 ± 13.16 kg. Their self-reported dominant arms were right in 13, left in 1 and ambidextrous in 1.

Regarding the Pathological group (PG), 10 patients (2 men, 8 women) were recruited with mean age of 46.67 ± 13.70 years (range, 23-68 years). Their mean height was 1.64 ± 0.09 m and weight was 66.88 ± 10.24 kg. Their self-reported dominant arms are right in 7 and left in 3.

Figures 1 and 2 depict the STUR and GH elevation of both PG's shoulders and CG, during abduction and flexion, respectively.



(a) Scapulothoracic Upward Rotations - Abduction

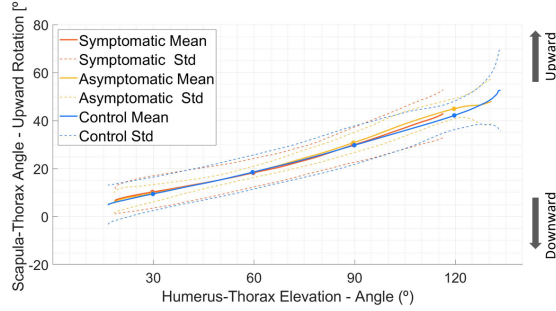


(b) Glenohumeral Elevation - Abduction

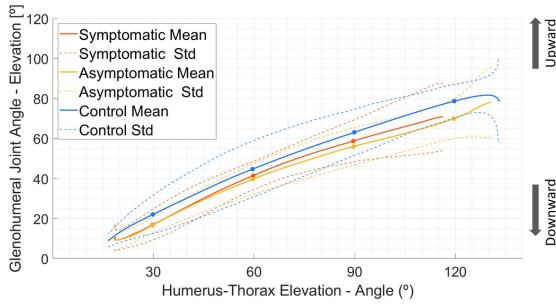
Figure 1: PG - Scapulothoracic and glenohumeral joint motion during abduction

When comparing the PG's contralateral shoulders, no significant interactions between elevation and shoulder was found during either movement. Meaning that the pattern of joint rotation between contralateral shoulders remained similar across the whole PG's range of motion. Significant shoulder effect was found for STIR and STUR during both movements, where the symptomatic shoulder had a more internally and upwardly rotated scapula.

The comparison between symptomatic shoulder and CG, showed a significant group effect on the



(a) Scapulothoracic Upward Rotations - Flexion



(b) Glenohumeral Elevation - Flexion

Figure 2: PG - Scapulothoracic and glenohumeral joint motion during flexion

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° and 60° elevation, at the 90° and 120° , the symptomatic shoulder was more upwardly rotated. Additionally, the group effect was also significant on GH elevation during flexion ($p < 0.01$, $F = 7.866$), where the symptomatic shoulder had a lower GH elevation throughout the complete movement.

Figure 3 shows a profile analysis of the mean SHR values for all symptomatic, asymptomatic shoulder and CG, during both movements.

The symptomatic shoulder showed a significant variation in pattern to the CG ($p < 0.05$, $F = 2.154$), along with significant group ($p < 0.1$, $F = 3.064$) and increment effect ($p < 0.01$, $F = 5.681$). 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 ($p < 0.1$, $F = 2.022$).

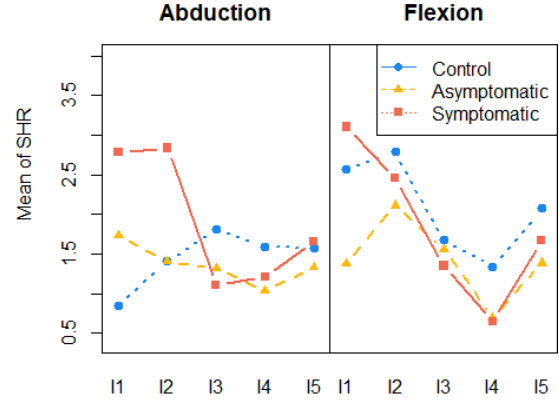


Figure 3: SHR across motion increments

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.

4.1.2 Pain/No Pain Subgroup

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 an 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 groups:

1. Pain group: group of subjects that reported moderate or high pain at a given HT elevation angle, and that elevation point on the symptomatic shoulder was less than 85% of the asymptomatic maximum arm elevation angle.
2. No Pain group: group 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.

In this analysis it is interesting to compare not only symptomatic vs asymptomatic shoulder within each group, but also how shoulder kinematics vary between groups and what differences can be found

when comparing again with the CG. 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 groups 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 more internally rotated scapula. Nonetheless, the elevation effect was significant on both subgroups for all joint rotations and the increment effect significant only during abduction.

Comparing the symptomatic shoulder results on both subgroups, several significant differences were found. 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 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 GH elevation ($p < 0.001$, $F = 55.12$). The Pain group had a less internally rotated scapula and a lower GH elevation at all angles of HT elevation. The elevation effect was significant for all joint rotations during both movements.

Regarding the SHR results, Figure 4 presents the SHR values during both movements for all increments and for the total movement.

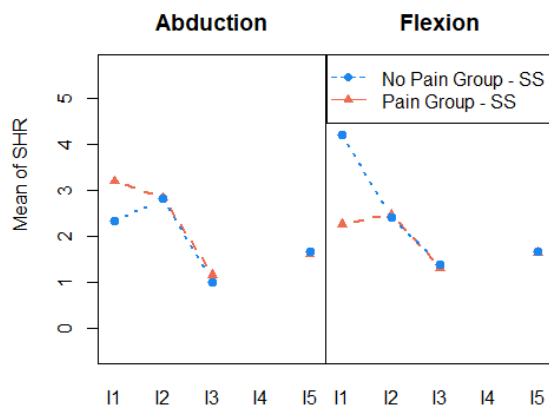


Figure 4: SHR Increments - No Pain group vs Pain group - Symptomatic shoulders

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.

4.2. sEMG

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 and compared for the CG (considering all 8 muscles) and PG and then for the PG's subgroups.

4.2.1 Control and Pathological Group

During abduction, the onset pattern between contralateral shoulders remained similar, only having a significant shoulder effect on the IS ($p < 0.1$, $F = 3.802$). All muscles showed a slight delay on the symptomatic shoulder onset, except for the SA, Figure 5.

During flexion, both contralateral shoulders 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 seems to have a slight delay on all muscle onset, except for the SA, that activated much earlier on the motion, having similar onset values to the IS and PD.

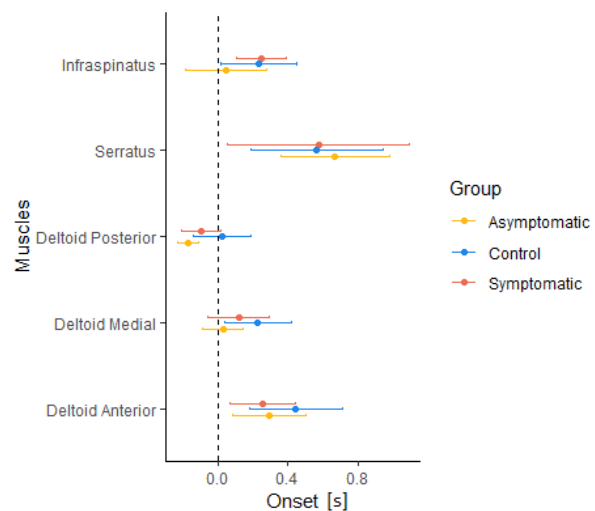
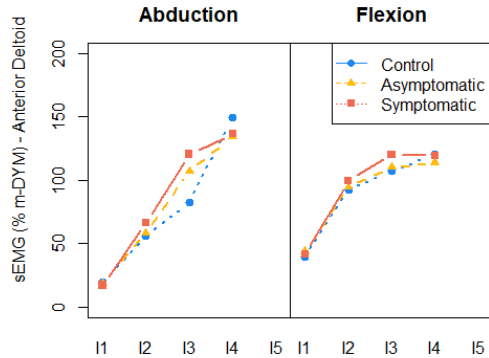


Figure 5: Onset - Abduction

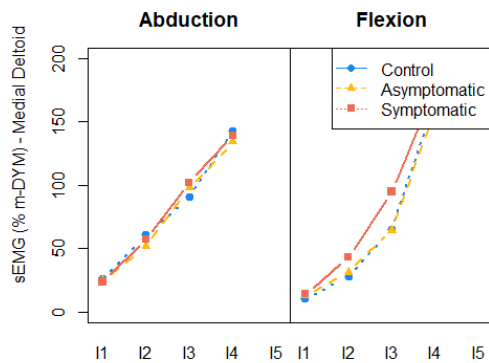
Regarding the comparison between PG and CG, significant differences were found on the PD ($p < 0.05$, $F = 4.996$) during abduction. However, despite the lack of statistical significance, there was a notable later activation of all three deltoids on the CG.

Looking now to the mean normalized sEMG data results, Figure 6 and 7 the mean normalized EMG activity of all muscles is shown, during both movements.

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.



(a) Anterior Deltoid

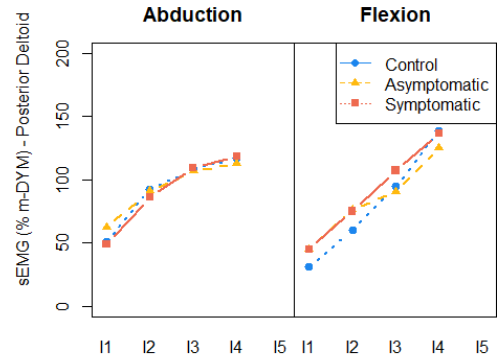


(b) Medial Deltoid

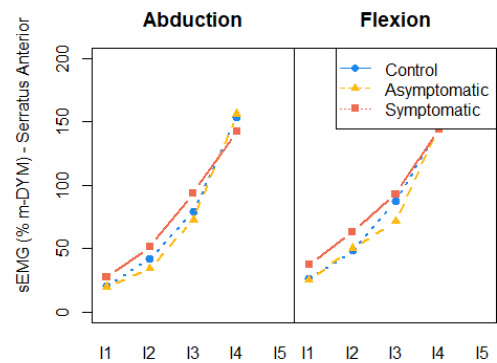
Figure 6: Mean normalized sEMG data - Abduction

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.

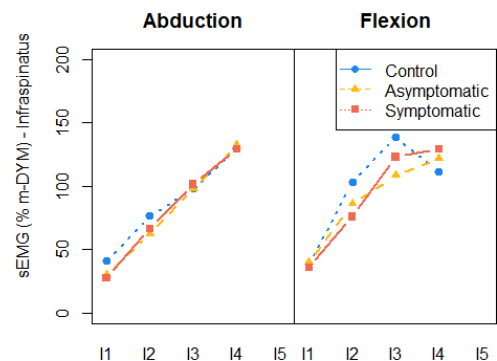
When comparing the PG with the CG, a significant shoulder, increment interaction was found on the AD ($p < 0.001$, $F = 2.849$) during abduction, and on the ME ($p < 0.1$, $F = 13.059$) and IS ($p < 0.01$, $F = 5.469$) during flexion. Additionally, the shoulder effect was also found significant on the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



(a) Posterior Deltoid



(b) Serratus Anterior



(c) Infraspinatus

Figure 7: Mean normalized sEMG data - Abduction

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 sRMG 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.

4.2.2 Pain/No Pain Subgroup

During abduction, no significant differences were found between subgroups, as they presented similar onsets for both shoulders. However, the Pain group's symptomatic shoulder had the serratus anterior activated much earlier than the asymptomatic shoulder, with onset values close the infraspinatus and anterior deltoid.

For flexion, more variations on the onset patterns can be observe, mainly for the infraspinatus and posterior deltoid. However only for the latter statistical significance existed ($p < 0.05$, $F = 8.962$). The infraspinatus on the symptomatic shoulders had a much later activation on the No Pain subgroup, along with the posterior deltoid.

Regarding the mean normalized sEMG data results, 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 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.

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.

5. Discussion

5.1. Pathological vs Control

When comparing the PG's symptomatic shoulder with the CG, more noticeable differences were found than with the PG's contralateral shoulders, especially during abduction. The kinematic alterations observed during this movement corresponded to

what is described on the literature, while during flexion the results are not as straight forward.

During abduction, even though only the STUR present statistical significance between groups, 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 [25]. A decrease on this two joint rotations, may lead to the anterior aspect of the acromion fail to move away from the humeral head during arm elevation and consequently contributing to a reduction of subacromial space and external RC compression [18]. Given the lower STUR, the GH is expected to elevate more as a compensatory response [14].

During flexion, different results were found. The plane of motion influences the pattern of shoulder kinematics [30, 15], 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 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 substantive risk for 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 its relative rotations that ultimately lead to a full range of motion. SHR provides that relation between the two joints responsible for most of the motion during arm elevation, STUR and GH.

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 is especially noticeable during abduction.

[14] 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 an 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.

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. However, the results presented for each individual increment show how the SHR can vary at different intervals of the movement. As such 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.

Alterations in shoulder kinematics have also been associated with muscle dysfunction, which can be a result of muscle strength deficits or muscle activation alterations [3]. 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 [29]. 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.

The muscle normalized sEMG pattern varied mainly on the deltoids. 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 data pattern between symptomatic and asymptomatic shoulders [32, 24, 16]. However, the values described in the literature are closer to the motion initiation or even before it, both on control and pathological populations [32, 33, 16].

The difference in SA onsets results may be related with the use of different methodologies to the ones used in the literature. [33] used wire intramuscular electrodes, possibly having more reliable data. Additionally, both [32] and [16] 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 [10]. 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 [7, 18]. 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.

Additionally, it should be pointed out that the significant differences found 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.

5.2. Pain vs No Pain

Differently to the results described on the previous subsection, the Pain and No pain subgroups, almost did not show significant differences between contralateral shoulders. The sample size on these subgroups is smaller, so it is harder to find statistically significance on its results, nonetheless, other studies have found similar shoulder kinematics between symptomatic and asymptomatic shoulders on a Pathological population [12, 8]. However, when comparing the subgroups with each other, several joint rotations showed differences with statistical significance.

During abduction, what it is interesting to notice is that 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. During flexion, the GH joint seems to have an compensatory response to the STUR, as on the phases with lower STUR the GH shows a higher elevation.

Regarding the SHR results during both movements, higher values are observed on all motion increments for the Pain subgroup, meaning that for the whole range of motion the GH joint showed a higher contribution than the STUR to the HT elevation, on this subgroup. Hence, there may be a lower capability of rotating the scapula properly, decreasing not only its upward rotation but also its posterior tilt, possibly leading to a reduction of sub-acromial space and external RC compression [18], that consequently may intensify their symptoms.

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 [24, 33], 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.

6. Conclusions

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 UCT. However, it should also be noted that the asymptomatic shoulders of the PG also showed dif-

ferences 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.

The sEMG results showed the most significant differences in the mean normalized sEMG data of the deltoid muscles, with increased mean normalized sEMG data mostly from the 30 degrees of humerothoracic elevation onwards, suggesting an adaptation of the motor strategies to elevate the shoulder. Additionally, the serratus anterior and infraspinatus muscle showed little variations, suggesting 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 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 HT elevation.

There are some limitations on this study that should be considered. Firstly, the small sample size on each group, and its different demographic and body composition. Secondly, when considering the study of shoulder kinematics, the present protocol was designed to study shoulder joint movement to a maximum of 120 of HT elevation. Lastly, the sEMG normalization process tends to remove the true biological variation within a group [29], becoming harder to identify differences between the two groups.

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