Adductor co-contraction during abduction: a friend or foe

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Altered Co-contraction Patterns of Humeral Head Depressors in Patients with Subacromial Pain Syndrome: A Cross-sectional Electromyography Analysis.

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ABSTRACT

Background

In approximately 29% to 34% of all patients with subacromial pain syndrome (SAPS) there is no anatomic explanation for symptoms, and behavioural aspects and/or central pain mechanisms may play a more important role than previously assumed. A possible behavioural explanation for pain in patients with SAPS is insufficient active depression of the humerus during abduction by the adductor muscles. Although the adductor muscles, specifically the teres major, have the most important contribution to depression of the humerus during abduction, these muscles have not been well studied in patients with SAPS.

Questions/purposes

Do patients with SAPS have altered contraction patterns of the arm adductors during abduction compared with asymptomatic people?

Methods

SAPS was defined as nonspecific shoulder pain lasting for longer than 3 months that could not be explained by specific conditions such as calcific tendinitis, full-thickness rotator cuff tears, or symptomatic acromioclavicular arthritis, as assessed with clinical examination, radiographs, and magnetic resonance arthrography. Of 85 patients with SAPS who met the prespecified inclusion criteria, 40 were eligible and agreed to participate in this study. Thirty asymptomatic spouses of patients with musculoskeletal complaints, aged 35 to 60 years, were included; the SAPS and control groups were not different with respect to age, sex, and hand dominance. With electromyography, we assessed the contraction patterns of selected muscles that directly act on the position of the humerus relative to the scapula (the latissimus dorsi, teres major, pectoralis major, and deltoid muscles). Co-contraction was quantified through the activation ratio ([AR]; range -1 to 1). The AR indicates the task-related degree of antagonist activation relative to the same muscle's degree of agonist activation, equalling 1 in case of sole agonist muscle activation and equalling -1 in case of sole antagonistic activation (co-contraction). We compared the AR between patients with SAPS and asymptomatic controls using linear mixed-model analyses. An effect size of $0.10<AR<0.20$ was subjectively considered to be a modest effect size.

Results

Patients with SAPS had a 0.11 higher AR of the teres major (95% CI, 0.01 to 0.21; $p = 0.038$), a 0.11 lower AR of the pectoralis major (95% CI, -0.18 to -0.04; $p = 0.003$), and a 0.12 lower AR of the deltoid muscle (95% CI, -0.17 to -0.06; $p < 0.001$) than control participants did. These differences were considered to be modest. With the numbers available, we

found no difference in the AR of the latissimus dorsi between patients with SAPS and controls (difference = 0.05; 95% CI, -0.01 to 0.12; p = 0.120).

Conclusions

Patients with SAPS showed an altered adductor co-contraction pattern with reduced teres major activation during abduction. The consequent reduction of caudally directed forces on the humerus may lead to repetitive overloading of the subacromial tissues and perpetuate symptoms in patients with SAPS. Physical therapy programs are frequently effective in patients with SAPS, but targeted approaches are lacking. Clinicians and scientists may use the findings of this study to assess if actively training adductor co-contraction in patients with SAPS to unload the subacromial tissues is clinically effective. The efficacy of training protocols may be enhanced by using electromyography monitoring.

INTRODUCTION

Chronic shoulder pain is the second most common musculoskeletal disorder in the general population, with prevalence rates ranging between 15% and 22%[1-3]. A specific anatomic basis for perceived symptoms, such as full-thickness rotator cuff tears or calcific tendinitis, is observed in many patients[4]. However, in approximately 29% to 34% of all patients with chronic shoulder pain, referred to here as subacromial pain syndrome (SAPS), the subacromial (suprahumeral) tissues are inflamed, but there is no structural anatomic cause that could explain persisting symptoms[5,6]. The fact that altering bony shapes with surgical interventions yields unsatisfactory results comparable to those of physical therapy also suggests that behavioural and/or central pain mechanisms may play a more important role than previously assumed[7,8].

In patients with SAPS, pain is frequently exacerbated during abduction, suggesting that motion-related (kinematic) factors contribute to the perpetuation of symptoms[4,5]. Open MRI and radiographic studies have attributed this particular pain pattern, the painful arc, to insufficient humeral-head depression during abduction[9,10]. We believe that patients with SAPS could benefit from mechanical unloading of the subacromial tissues during abduction by the active contribution of shoulder muscles that act as humeral-head depressors[11-13]. The craniocaudal position of the humerus relative to the scapula is directly determined by a balance of cranial forces generated by the shoulder abductors and caudal forces generated by co-contraction of the rotator cuff and arm adductors[14,15]. In both research and clinical practice, there has been a focus on the rotator cuff, while the arm adductors, specifically the teres major, contribute the most to depression of the humerus during abduction[11,14,15].

Because of this, studying co-contraction of the arm adductors in patients with SAPS seems to be worthwhile; if adductor co-contraction is altered in patients with SAPS, this could indicate a treatable imbalance between the abductors and adductors. Accordingly, we asked: Do patients with SAPS have altered contraction patterns of the arm adductors during abduction compared with asymptomatic people?

PATIENTS AND METHODS

In this study, we defined SAPS as shoulder pain lasting for longer than 3 months with no specific anatomic abnormalities that could explain the pain and could benefit from specific treatment (such as acromioclavicular osteoarthritis, calcific

tendinitis, or full-thickness rotator cuff tears)[4]. Between April 2010 and September 2016, consecutive patients with a clinical diagnosis of SAPS who were referred to the outpatient clinics of the Leiden University Medical Centre, Haaglanden Medical Centre, or Alrijne Hospital were evaluated for inclusion in this cross-sectional cohort study (Trial registry number NTR2283)[16]. Patients were selected through clinical examination, radiographs, and MR arthrography.

The inclusion criteria were unilateral shoulder pain for at least 3 months, positive results of a Hawkins-Kennedy test (passive anteflexion of the shoulder to 90° with subsequent internal rotation of the shoulder to provoke subacromial pain) and Neer lidocaine impingement test (examining for immediate relief of pain after subacromial infiltration with lidocaine), and at least one of the following symptoms: pain during daily life activities with arm abduction, extension, and/or internal rotation; pain at night or incapability of lying on the shoulder; painful arc; diffuse pain during palpation of the greater tuberosity; scapular dyskinesis; and a positive full-can test, empty-can test, or Yocum test result[16]. Exclusion criteria were insufficient Dutch-language skills, age younger than 35 years or older than 60 years, inflammatory arthritis of the shoulder, clinical signs of glenohumeral or acromioclavicular osteoarthritis, previous fracture, dislocation or surgery of the shoulder, cervical radiculopathy, glenohumeral instability, decreased passive function (frozen shoulder), malignancy, full-thickness rotator cuff tears, calcific tendinitis, labrum or ligament pathology, pulley lesion, biceps tendinopathy, os acromiale, cartilage lesion, or a bony cyst[16]. All MR arthrography studies were evaluated by an experienced independent radiologist[5]. Of 85 patients who were referred with the clinical diagnosis of SAPS, 45 were excluded, leaving 40 patients for evaluation in this study (**Figure 1**).

To select control participants, we recruited the spouses of patients with musculoskeletal complaints at the outpatient clinic of Leiden University Medical Centre between January 2016 and November 2016. Inclusion criteria were age between 35 and 60 years, no current or past shoulder concerns, no visit to a physician for shoulder related concerns, and no past shoulder discomfort for more than 1 week. Exclusion criteria were impaired passive and active shoulder function during clinical examination, insufficient Dutch-language skills, prior shoulder surgery, injections, shoulder fracture or dislocation, radiculopathy, frozen shoulder, osteoarthritis or rheumatoid arthritis, and neurologic or muscle disease. No additional imaging was performed in the control group, because we only used imaging in the SAPS group to exclude specific anatomic conditions that would have explained a patient's symptoms.

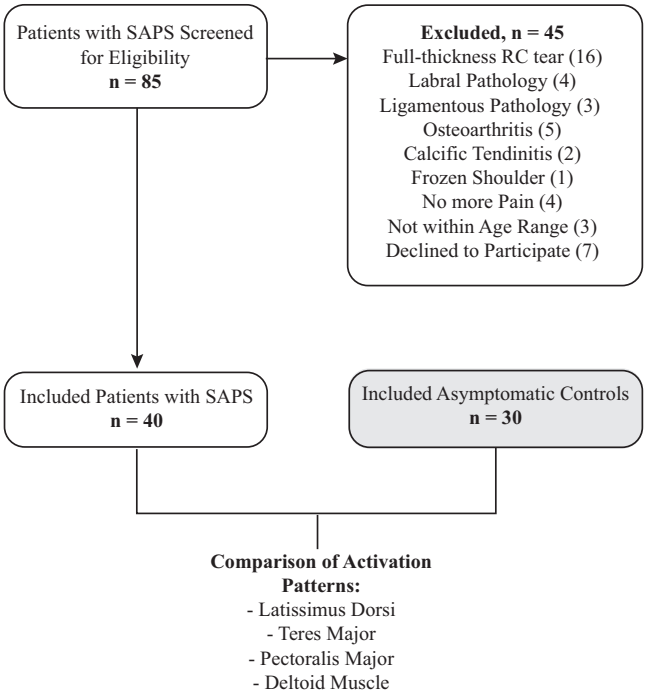

Figure 1 | A flow diagram of the participant inclusion process is shown.

Forty patients with SAPS and 30 asymptomatic controls were compared. The SAPS and control groups were not different with respect to age, sex, and hand dominance (**Table 1**).

The research was conducted according to the principles of the Declaration of Helsinki (64th WMA General Assembly, Fortaleza, Brazil, October 2013) and in accordance with the Medical Research Involving Human Subjects Act. The review board of our institutional ethical medical commission approved this study (P09.227 & P15.046) and all participants provided written informed consent.

Table 1 | Baseline characteristics of patients with SAPS and controls

	SAPS	**Controls**	
Demographics	*n=40*	*n=30*	*p-value*
Age, yrs (mean, SD)	50 (6)	51 (5.7)	0.740
Female (n, %)	23 (58)	17 (57)	0.944
Right side dominance (n, %)	35 (88)	25 (83)	0.622
Dominant side measured/affected (n, %)	25 (63)	17 (57)	0.622
Complaints duration in months (median, percentiles)	18 (12-29)	N/A	N/A

We used a standardised testing protocol[17]. In this study, we were interested in evaluating the activation patterns of muscles that directly act on the craniocaudal position of the humerus with respect to the scapula during abduction. In biomechanical evaluations and a recent systematic review on the topic, it has been shown that the deltoid muscle contributes the most to upward migration of the humerus during abduction[14,15]. The arm adductors, specifically the latissimus dorsi, teres major, and, to a lesser extent, the pectoralis major, are the strongest humeral-head depressors during abduction[14,15]. Other muscles that may contribute to humeral depression are the teres minor and the lower parts of the infraspinatus and subscapular muscles[14]. Because evaluating these muscles with EMG requires indwelling (fine wire) electrodes, we limited our evaluation to the deltoid, latissimus dorsi, teres major, and pectoralis major muscles.

With the target arm in external rotation at the side and attached to a one-dimensional force transducer at the wrist, we recorded activation of the latissimus dorsi, teres major, pectoralis major (clavicular part), and deltoid muscles (medial part) with surface EMG during rest and isometric abduction and adduction tasks (DelSys system Bagnoli-16, Boston, MA, USA; inter-electrode distance, 10 mm; bandwidth 20-450 Hz)[17].

The EMG and force signals were analog-digitally converted and recorded simultaneously at a sample rate of 2500 Hz. Offline, the EMG signals were subtracted from the mean EMG signals for offset removal, rectified, and combined with the moving average over intervals of 0.1 seconds, using custom-made software in Matlab (Math-Works Inc., R2018b, Natick, MA, USA).

Participants first performed maximal abduction and adduction tasks to determine the maximum voluntary force. The maximum voluntary force was set as the lowest value of either the maximum voluntary force during isometric adduction or abduction. Subsequently, a target force of 60% with a tolerance of ± 3.75% of the maximum voluntary force was presented to the participants on a computer screen[17]. Finally, participants performed a 15-second isometric force task in abduction and adduction at equal target force levels for the purpose of computing a standardised measure of the degree of antagonistic versus agonistic activation. Measurements were performed twice, and both assessments were used in this study to reduce variability[16]. We quantified muscle activation with the AR using the following equation:

$$AR_{muscle} = \frac{mEMG^{IP} - mEMG^{OP}}{mEMG^{IP} + mEMG^{OP}} \quad [17]$$

"Muscle" represents either the latissimus dorsi, teres major, pectoralis major, or deltoid muscle, and ^{IP} and ^{OP} indicate "in-phase" agonist activation and "out-phase" antagonist muscle activation, respectively, relative to the force task in abduction or adduction.

The AR ranges between -1 and 1 and indicates the task related degree of antagonist activation relative to the same muscle's degree of agonist activation. The AR equals 1 in case of sole agonist muscle activation and decreases with increasing co-contraction. An AR of -1 indicates sole antagonistic activation and no agonistic activation. Based on this AR, we assessed the difference in muscle activation patterns between patients with SAPS and asymptomatic controls. We also assessed the influence of the target force level on the activation ratio in the statistical analysis.

To prevent overestimation of the degree of co-contraction as assessed with the AR, the mean EMG amplitude during the agonistic task (the activity of the deltoid muscle during abduction and the activity of adductors during adduction) was verified to be twice the mean EMG amplitude of the 10% lowest EMG signals during the relative rest, abduction, and adduction tasks (a signal-to-noise ratio of 2.0). If this condition was not met or if EMG data were corrupt (because of loose electrodes or other technical problems), the ARs were excluded. In the SAPS group, one of 40 ARs of the latissimus dorsi (2.5%), one AR of the teres major (2.5%), one AR of the pectoralis major (2.5%), and one AR of the deltoid muscle (2.5%) had to be excluded because the twofold signal-to-noise ratio was not reached. For the same reason, four of 30 ARs of the latissimus dorsi (13%) and two ARs of the teres major (6.7%) had to be excluded in the control group. Furthermore, because of a disconnected EMG amplifier, three of 30 ARs of the deltoid muscle (10%), four ARs of the latissimus dorsi (13%), four ARs of the pectoralis major (13%) and three ARs of the teres major (10%) could not be used in the control group. Additionally, in the control group, one out of 30 ARs of the latissimus dorsi (3%) and one AR of the pectoralis major (3%) could not be used because of a broken electrode.

Categorical data are described with numbers and percentages and continuous parameters are described with means and either 95% CIs, SDs, or medians with the 25th and 75th percentiles, depending on data distributions. Demographic data were compared using independent-samples t-tests or Mann-Whitney's U test depending on the distribution of data. Linear mixed-model analyses assessed the differences in activation ratios between patients with SAPS and asymptomatic controls. There were separate analyses for each assessed muscle. The dependent variable was AR_{muscle} and the measurement moment was the repeated factor. Independent variables were the patient groups (SAPS or asymptomatic controls) and the target force level (60% maximum voluntary force). An effect size of $0.10<AR<0.20$ was subjectively considered to be a modest effect size.

The patient and control populations were recruited in two different studies, and no a priori sample size analysis was performed for the AR. The statistical analysis was performed using SPSS® version 23 (IBM® Corp., Armonk, NY, USA). The results of the linear mixed-model analyses are presented as estimated regression coefficients, 95% CIs, and p values. A two-sided p value of 0.05 or less was considered statistically significant.

RESULTS

Patients with SAPS showed less co-contraction of the teres major and more co-contraction of the pectoralis major than controls did. Patients with SAPS had a 0.11 higher AR of the teres major (95% CI, 0.01 to 0.21; p = 0.038), a 0.11 lower AR of the pectoralis major (95% CI, -0.18 to -0.04; p = 0.003), and a 0.12 lower AR of the deltoid muscle (95% CI, -0.17 to -0.06; p < 0.001) than controls did. In terms of effect size, these differences were considered to be modest. With the number of patients and controls available, there was no difference in the degree of latissimus dorsi co-contraction between the groups (difference = 0.05, 95% CI, -0.01 to 0.12; p = 0.120) (**Table 2**). The average activation ratios of the latissimus dorsi, teres major, pectoralis major and deltoid muscle were 0.78 (SD 0.14), 0.53 (SD 0.23), 0.68 (SD 0.23) and 0.73 (SD 0.18) in patients with SAPS, and 0.73 (SD 0.19), 0.41 (0.27), 0.82 (0.08) and 0.85 (0.10) in controls (**Figure 2**).

Table 1 | Difference in activation ratios between patients with SAPS and controls

	Activation Ratio		
Independent variables	*Estimate*	*95% CI*	*p-value*
Latissimus dorsi			
Intercept	0.67	(0.57 – 0.77)	–
SAPS patients vs. controls	0.05	(-0.01 – 0.12)	0.120
Force task	0.08	(-0.02 – 0.17)	0.108
Teres major			
Intercept	0.43	(0.28 – 0.59)	–
SAPS patients vs. controls	0.11	(0.01 – 0.21)	**0.038**
Force task	0.0	(-0.13 – 0.13)	0.994
Pectoralis major			
Intercept	0.73	(0.62 – 0.83)	–
SAPS patients vs. controls	-0.11	(-0.18 – -0.04)	**0.003**
Force task	0.09	(0.01 – 0.18)	**0.036**
Deltoid muscle			
Intercept	0.90	(0.81 – 0.98)	–
SAPS patients vs. controls	-0.12	(-0.17 – -0.06)	**<0.001**
Force task	-0.05	(-0.13 – 0.03)	0.200

DISCUSSION

In approximately 29% to 34% of all patients with SAPS there is no anatomic explanation for symptoms, and behavioural aspects and/or central pain mechanisms may play a more important role than previously assumed[5,6]. A possible behavioural explanation for pain in patients with SAPS is insufficient active depression of the humerus during abduction achieved by adductor co-contraction[11-13]. The adductor muscles, specifically the teres major, have the most important contribution to humeral depression; however, this subject hast not been studied well in patients with SAPS[12,15]. In the current study, we sought to determine whether patients with SAPS have altered co-contraction patterns of the arm adductors compared with asymptomatic controls, which could point towards a treatable imbalance between the abductor and adductor muscles. We found that patients with SAPS predominantly contracted with the pectoralis major, while controls did so with the teres major. To unload subacromial tissues, it may be more effective to co-contract with the teres major[11,15].

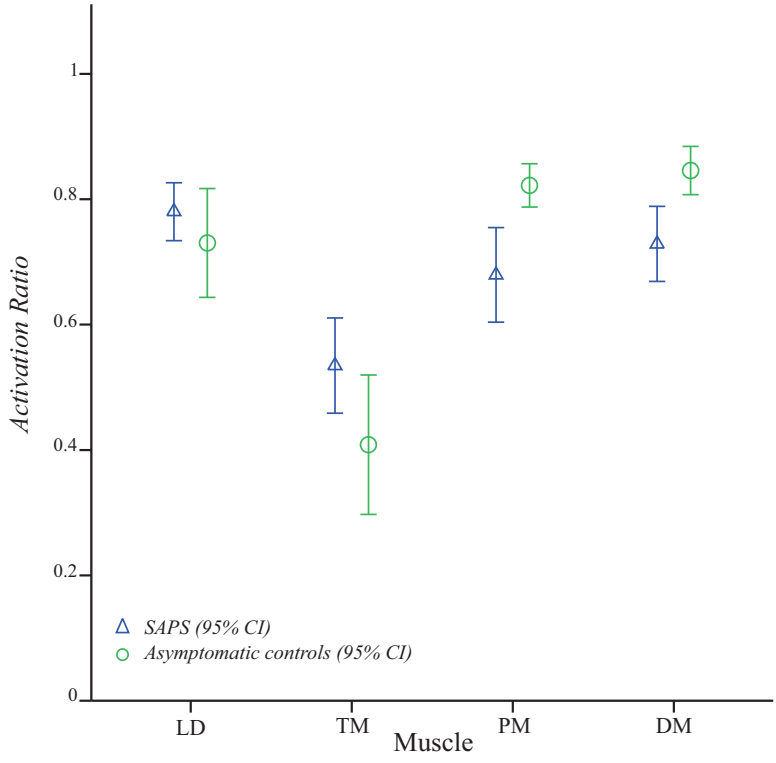

Figure 2 | The activation ratios of four shoulder muscles in patients with SAPS and controls are shown. LD = latissimus dorsi; TM = teres major; PM = pectoralis major; DM = deltoid muscle

Our study results have some limitations. First, the common chronic symptoms in the SAPS group were likely caused by a variety of anatomic factors. SAPS is a syndrome, not a specific anatomic diagnosis, and our conclusions need to be interpreted in light of this. A specific anatomic cause should be sought in patients presenting with shoulder pain, and treatment, other than nonsurgical therapies, should only be initiated when a specific, treatable anatomic basis is found for the symptoms[8]. Second, no a priori analysis was performed because the patient and control populations were recruited in two studies. Negative results may therefore have originated from underpowering. In light of the large amount of missing data for the latissimus dorsi in the control group (nine of 30 ARs; 30%), it plausible that there is a difference between patients with SAPS and controls in co-contraction of the latissimus dorsi, we may have been able to detect this difference due to underpowering. We do not consider it likely that missing data introduced a bias, because the predominant cause was failure of the EMG equipment (20 of 26 total missing values, 77%). Third, we did not control for potential confounding variables such as sports participation and BMI. Although we selected our control group from the patients' spouses, we cannot exclude the influence of these factors. Fourth, we only evaluated a selection of muscles that affect the craniocaudal position of the humerus the most[11,14,15]. Our conclusion may be supported by adding an analysis of other adductors, for example, the teres minor and lower parts of the infraspinatus and subscapularis.

Few studies have assessed the activation patterns of arm adductors during abduction tasks in patients with SAPS[18-20], and these studies contradicted one another, perhaps because of small sample sizes[18] or different testing positions[19]. In addition to altered adductor activation patterns, we observed a lower activation ratio of the deltoid muscle in patients with SAPS, originating from reduced activation during abduction. As suggested in previous studies[21,22], it seems that patients with SAPS attempt to avoid pain by reducing abductor activation at the cost of function (that is, reduced target force level). Co-contraction with the teres major, as we observed in the control group, may protect the patient from pain while preserving function.

Our EMG assessment of muscles that determine the craniocaudal position of the humerus during abduction provides new insight regarding the function of the teres major. Patients with SAPS predominantly co-contracted with the pectoralis major, whereas controls did so with the teres major (a glenohumeral muscle). Both muscles contribute to glenohumeral stabilisation. However, to reduce loading on subacromial tissues, it is more effective to use teres major co-contraction because this muscle is more capable of pulling the humerus downward (away from the acromion) than other muscles are[11,15].

Recently, it has been shown that surgical interventions commonly yield unsatisfactory results in treating patients with SAPS, and physical therapy is preferable[7,8,23]. We believe that such nonsurgical approaches can be improved with targeted approaches[23]. Supporting our findings, other clinical studies have suggested that increasing co-contraction of the arm adductors is a viable treatment option for patients with SAPS[12,13,24]. To improve targeted treatment approaches that enhance teres major (and latissimus dorsi) co-contraction in patients with SAPS, we suggest performing trials in which EMG is used[25-27].

In this cross-sectional EMG evaluation, we found decreased co-contraction of the teres major and increased co-contraction of the pectoralis major in patients with SAPS. We based our study on the rationale that insufficient humeral depression during abduction leads to perpetuation of SAPS, by overloading of the subacromial tissues[5,9,10]. For depressing the humerus, increasing teres major co-contraction as observed in the control group could be more effective[11,12,15]. Future studies using EMG monitoring should assess if actively training teres major (and latissimus dorsi) co-contraction could be a target for physical therapy protocols for patients with SAPS.

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