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Original Articles

Pain does not explain reduced teres major co-contraction during abduction in patients with Subacromial Pain Syndrome

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ABSTRACT

Background: Patients with Subacromial Pain Syndrome show reduced co-contraction of the teres major during abduction. Consequent insufficient humeral depressor function may contribute to painful irritation of subacromial tissues and offers a potential target for therapy. A crucial gap in knowledge is whether the degree of teres major co-contraction in these patients is influenced by pain itself. To gain insight into this matter, we assessed whether relief of subacromial pain with local analgesics leads to increased adductor co-contraction in 34 patients with subacromial pain.
Methods: In a single-arm interventional study with 34 patients, electromyographic activity of the latissimus dorsi, pectoralis major, teres major and deltoid was assessed during isometric force tasks in 24 directions before and after subacromial Lidocaine injection. Co-contraction was quantified using the activation ratio; range [−1 (sole antagonistic activation, i.e. co-contraction) to 1 (sole agonistic activation)].
Findings: There were no changes in activation ratio of the teres major after the intervention (Z-score: −0.6, $p =$ 0.569). The activation ratio of the latissimus dorsi increased to 0.38 (quartiles: 0.13–0.76), indicating decreased co-contraction (Z-score: −2.0, $p = 0.045$).
Interpretation: Subacromial analgesics led to a decrease in co-contraction of the latissimus dorsi, whereas no change in the degree of teres major co-contraction was observed. This study shows that decreased teres major co-contraction in patients with subacromial pain, likely is not the consequence of pain itself, opening a window for physical therapy with training of teres major co-contraction to reduce subacromial irritation and pain.
Level of evidence: Level II treatment study.

1. Introduction

Compared to age-matched controls, patients with Subacromial Pain Syndrome (SAPS) show reduced co-contraction of the teres major during abduction (Overbeek et al., 2019a). While the rotator cuff muscles are regarded as the major humeral head depressors during abduction, teres major co-contraction may also play a role in humeral head depression during this movement (Hik and Ackland, 2019). Hence, observed reduction of teres major contraction during abduction in patients with SAPS may explain painful irritation of subacromial tissues and represent a target for therapy (Graichen et al., 2005; Halder et al., 2001; Overbeek et al., 2018). However, it has not yet been made clear whether decreased teres major co-contraction in patients with SAPS is owing to pain or underlying pathology, which is crucial information for the direction of treatment (Overbeek et al., 2019a; Overbeek et al., 2019b).

From studies involving asymptomatic individuals it is known that older individuals demonstrate increased contraction of the teres major and latissimus dorsi during abduction, compared to younger individuals (Overbeek et al., 2019b). This trend is associated with ageing and explained as a compensation mechanism for age-related degeneration of shoulder tissues (in particular the rotator cuff). The increased co-contraction enhances glenohumeral stability and protection of subacromial tissues by producing a caudally directed force counter-balancing the cranially directed force of the deltoid during abduction (Hik and Ackland, 2019; Overbeek et al., 2019b). These findings suggest that changes in teres major co-contraction relate to biomechanical

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demands and not particularly to pain.

Therefore, the goal of this study was to assess whether reducing the degree of pain in patients with SAPS, results in increased teres major activation during abduction, i.e. co-contraction. In a single-arm interventional study, contraction of the deltoid muscle and simultaneous co-contraction of the latissimus dorsi, pectoralis major and teres major is measured before and after injection of subacromial anesthetics.

2. Methods

Thirty-four patients were recruited between April 2010 and December 2012 at the Leiden University Medical Center, Haaglanden Medical Center and Alrijne Hospital, under a previously registered and published study protocol (Trial register no. NTR2283) (de Witte et al., 2011). Consecutive patients with SAPS were screened using physical examination, shoulder radiographs and magnetic resonance arthrography by dedicated shoulder surgeons. In this study, we defined SAPS as shoulder pain lasting for longer than 3 months with no specific anatomic abnormalities that could explain complaints and require specific treatment (e.g. acromioclavicular osteoarthritis, calcific tendinitis, full thickness rotator cuff tears). The inclusion criteria were patients who were aged 35–60 years, who had unilateral shoulder complaints for >3 months and who received a clinical diagnosis of SAPS based on a positive Hawkins test and Neer impingement test with lidocaine (de Witte et al., 2011). The exclusion criteria were insufficient language skills, inflammatory glenohumeral (GH) arthritis, clinical signs of GH or acromioclavicular osteoarthritis, previous shoulder surgery, fracture or dislocation, cervical radiculopathy, GH instability, decreased passive GH mobility (e.g. frozen shoulder), and presence of electronic implants (e.g. pacemaker). Additionally, patients were excluded in case other specific conditions were diagnosed on radiographs or magnetic resonance arthrography such as calcific tendinitis, full-thickness rotator cuff tear, and labral or ligament pathology (de Witte et al., 2011). The review board of the institutional medical ethical committee approved this study (P09.227) and all patients gave written informed consent.

2.1. Intervention

Using a 50 mm 21 gauge needle, 5 ml 1% Lidocaine was injected in the subacromial space. The needle was inserted 1 to 2 cm inferior and medial to the posterolateral corner of the acromion, directing to the anterolateral corner of the acromion (soft spot). Patients were then given a 30 min adjustment period and were asked to move their arm in order to disperse the drug within the subacromial bursa. Following subacromial analgesics, all patients verbally reported reduced pain.

2.2. Electromyography (EMG) measurement set-up

Before and 30 min after subacromial analgesics, muscle activation patterns of the latissimus dorsi, teres major, pectoralis major (pars clavicularis) and deltoid (pars medialis) were assessed with EMG during isometric force tasks (de Groot et al., 2004). Bipolar surface electrodes (inter-electrode distance 10 mm) were adhered to abraded and ethanol cleaned skin overlying the middle of the muscle belly of the latissimus dorsi, pectoralis major (clavicular part), teres major and the middle part of the deltoid muscle (Table 1). The EMG was band pass filtered (20–500 Hz) before recording. Force and EMG signals were Analogue-Digitally converted and recorded simultaneously at a sample rate of 2000 Hz.

Table 1
Shoulder muscles and localization of the electrodes.

Muscle	Location electrode
M. latissimus dorsi (LD)	6 cm below angulus inferior scapulae
M. pectoralis major, pars clavicularis (PM)	1/2 clavicula, 1 cm caudally
M. teres major (TM)	4 cm cranial to angulus inferior and 2 cm lateral to LD
M. deltoideus, pars medialis (DM)	Middle muscle belly, 2-4 cm below acromion, lateral

Subjects were in seated position with the arm in a splint such that the upper arm was in 60° of anteflexion, in 30° adduction, and 45° internal rotation (Fig. 1) (de Witte et al., 2014). The elbow was 90° flexed. The force transducer was mounted on a sled and all gravitational forces and GH moments were neutralized by contra-weights, to ensure that participants only exerted forces perpendicular to the humeral longitudinal axis and prevent subjects from generating supplementary moments. The exerted force was visualized through a cursor on a video screen to help subjects to control both force direction and magnitude.

Before and after the injection of subacromial anesthetics, the following procedure was carried out. First, the Maximum Voluntary Force (MVF) was determined by asking subjects to perform 24 tasks in a range from 0 to 360° with 15° increments, in a random sequence, at the highest level of force wherein patients could comfortably fulfill the tasks for 2 s. Then, subjects were asked to perform 2 s force tasks visualized by a cursor on the video screen in the same 24 randomly sequenced directions at 75% of the lowest MVF. The raw EMG signal during two seconds of rest and raw EMG signals during the 75% MVF force tasks were rectified and averaged. The offset was removed by subtracting the rectified EMG signal during the rest task from the rectified EMG signals during the force tasks. These EMG-values were used for the calculation of the degree of co-contraction (below).

2.3. Calculation of co-contraction

The degree of adductor co-contraction was expressed using the activation ratio (AR), which represents the degree of antagonistic activation respective to the same muscles agonistic activation. The AR ranges from −1 to 1, equaling 1 in case of pure agonistic muscle activation and − 1 in case of pure antagonistic activation (de Groot et al., 2004; Meskers et al., 2004; Steenbrink et al., 2010). Calculation of the AR is based on the muscle's principal action as previously determined in young healthy participants (de Groot, 1998; de Groot et al., 2004). These

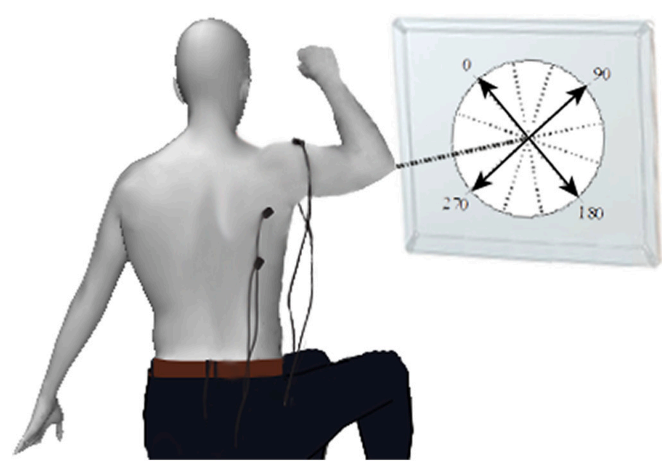

Fig. 1. Measurement set-up.
Subjects were in seated position with the arm in a splint attached to a 3D force transducer such that the upper arm was in 60° of anteflexion, in 30° adduction, and 45° internal rotation, and the elbow 90° flexed (splint depicted in manuscript by De Witte and co-authors (de Witte et al., 2014)). The exerted force was visualized through a cursor on a video screen to help subjects to control both force direction and magnitude. Subjects performed 24 submaximal (75% MVF) force tasks in 24 equidistant directions, 15° apart, ranging from pressing arm straight up (0°) to pushing the arm sideward (90°, 270°) or downward (180°). During these tasks, electromyography was obtained from the latissimus dorsi, pectoralis major, teres major and deltoid using bipolar surface electrodes.

values were used to indicate in which direction of movement the muscle is supposed to be maximally active (de Groot, 1998; de Groot et al., 2004). For instance, the deltoid muscle is expected to have maximum activation during arm abduction, i.e. the principal action (de Groot, 1998). Based on these principal actions, muscle activation can be expressed as the agonistic 'in-phase' activation (EMG^{IP}), and in the opposite direction as antagonistic 'out-of-phase' activation (EMG^{OP}) as depicted in Fig. 2 (de Groot, 1998; de Groot et al., 2004). For the calculation of the mean EMG^{IP}, EMG magnitudes were averaged over seven force tasks, including the force task corresponding to the muscle's principal action and 3 adjacent force tasks on each side. Conversely, for the calculation of EMG^{OP}, EMG was averaged over 7 targets in targets in the exact opposite direction of the EMG^{IP} directions. Subsequently, based on these values the activation ratio (AR) was calculated using Eq. (1). In order to prevent overestimation of the degree of co-contraction as assessed with the AR, the maximum EMG-amplitude was verified to be twice the minimum EMG-amplitude (a signal-to-noise ratio of SNR ≥2.0). In case this condition was not met or in case EMG-data was corrupt (e.g. loose electrode), the ARs were excluded.

$$AR_{muscle} = \frac{EMG^{IP} - EMG^{OP}}{EMG^{IP} - EMG^{OP}} \quad -1 \le \mathrm{AR}_{\mathrm{muscle}} \le 1 \tag{1}$$

2.4. Statistical analysis

Categorical data were described with numbers and percentages. Continuous parameters were described with means and either 95%-confidence intervals (95% CI) or standard deviations (SD), or medians with the 25th and 75th percentiles, depending on data distributions. The changes in activation ratios and unstandardized EMG amplitudes (EMG^{IP} and EMG^{OP}) before and after intervention were assessed by means of paired *t*-tests or Mann Whitney *U* test. The statistical analysis was performed using the Statistical Package of Social Sciences (SPSS®) version 23 (IBM® Corp, Armonk, NY, USA). A two-sided *p*-value of 0.05 or less was considered statistically significant.

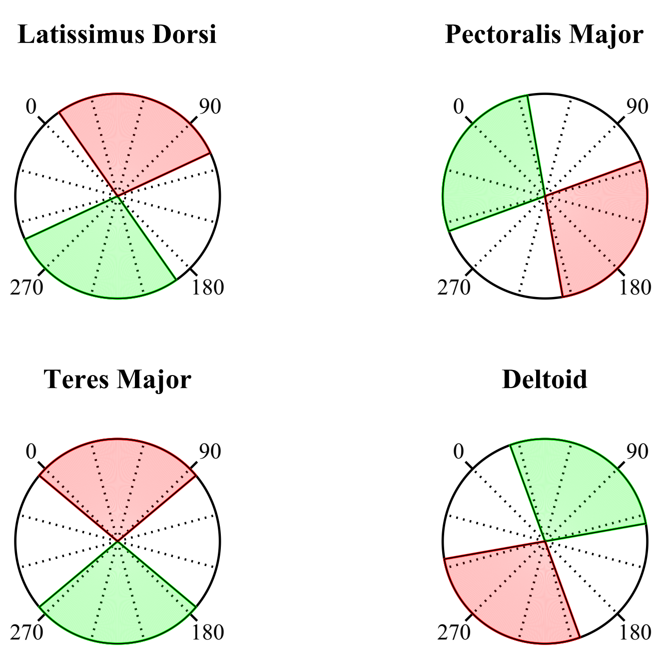

Fig. 2. In-phase and out-phase activation areas for calculation of the activation ratio.
The principal action indicates in which direction of movement the muscle is supposed to be maximally active (de Groot, 1998; de Groot et al., 2004). Based on the muscle's principle action, activation was expressed as agonistic 'in-phase' activation (*green*), and in the opposite direction, antagonistic 'out-of-phase' activation (red). Based on averaged EMG-values within these ranges, the activation ratio was calculated (Eq. (1)).

3. Results

Thirty-four patients with SAPS, with a mean age of 50 years (SD 6.5) were included in this study (Table 2). In four patients post-intervention assessments could not be performed due to a vasovagal syncope ($n = 1$), allergy to lidocaine (n = 1) or refusal to undergo the intervention ($n = 2$). There was no loss of EMG signals. The EMG-measurements were performed at a mean force level of 35 N (SD 8.8) and 35 N (SD 8.0), before and after the intervention respectively.

Before the intervention, the activation ratios of the pectoralis major and the deltoid were around 0.8, indicating predominant agonistic activity (Table 3). In contrast, the median pre-intervention activation ratios of the teres major and latissimus dorsi were 0.48 (quartiles: 0.36–0.63) and 0.26 (quartiles: 0.09–0.66), indicating presence of antagonistic activity.

After intervention, the teres major showed no statistically significant change in activation ratio (*Z*-score: −0.6, $p = 0.569$). The median activation ratio of the latissimus dorsi increased to 0.38 (quartiles: 0.13–0.76), which represented a significant change (Z-score: −2.0, $p = 0.045$). The increase in activation ratio of the latissimus dorsi was explained by a relative decrease of activation during the antagonistic tasks (EMG^{OP}) and an increase in in activation during agonistic tasks (EMG^{IP}) as described in Table 3.

4. Discussion

In this single-arm interventional study, we found that co-contraction of the teres major did not change after the administration of subacromial analgesics, while a statistically significant increase in latissimus dorsi activation ratio after subacromial infiltration was observed, implying a decrease in co-contraction. Thus pain does seem to affect latissimus dorsi co-contraction but has no direct influence on the degree of teres major co-contraction, suggesting different pathophysiological pathways.

The degree of co-contraction of the latissimus dorsi decreased after local administration of Lidocaine, while the degree of agonistic activation increased, resulting in a significant increase in activation ratio (suggesting relatively reduced antagonistic activation). It has been shown in previous studies that in the presence of pain, one may react with decreasing agonistic muscle activity and increasing antagonistic muscle activity, to protect damaged tissue (Lund et al., 1991). Our finding regarding the latissimus dorsi, may be in line with this protective mechanism to pain; due to the reduction of pain after Lidocaine infiltration, co-contraction of the latissimus dorsi may no longer be necessary (Lund et al., 1991).

Table 2
Patient characteristics.

Patient characteristics		SAPS ($n = 36$)
Age	Years (SD)	50 (6.5)
Female	n (%)	22 (61)
Length, cm	Mean (SD)	173 (12)
Weight, kg	Mean (SD)	78 (16)
BMI	Mean (SD)	26 (4.4)
Duration of complaints, months	Median (percentiles)	17 (12–24)
Right side dominance	n (%)	31 (86)
Right side affected	n (%)	21 (58)
Dominant side affected	n (%)	22 (61)
VAS in rest, mm	Median (percentiles)	11 (2.0–25)
VAS during movement, mm	Median (percentiles)	39 (18–59)
Constant Score	Mean (SD)	71 (13)
WORC score	Mean (SD)	58 (18)

SAPS, Subacromial Pain Syndrome; SD, Standard Deviation; n, number; VAS, Visual Analogue Scale; WORC, Western Ontario Rotator Cuff score (Holtby and Razmjou, 2005, Kirkley et al., 2003).

Table 3
Activation Ratio before and after Lidocaine injection.

Muscle activity	SAPS (n = 34)		Paired difference	
	Before intervention (median, quartiles)	After intervention (median, quartiles)	Z-score	p-Value
Activation ratio				
Latissimus dorsi	0.26 (0.09–0.66)	0.38 (0.13–0.76)	−2.0	**0.045**
Pectoralis major	0.83 (0.72–0.89)	0.78 (0.64–0.88)	−1.2	0.229
Teres major	0.48 (0.36–0.63)	0.51 (0.32–0.68)	−0.6	0.569
Deltoid	0.81 (0.74–0.94)	0.87 (0.79–0.94)	−1.1	0.254
Unstandardized agonistic EMG (μV)				
Latissimus dorsi	2.3 (1.5–4.3)	2.8 (1.3–4.2)	−0.32	0.750
Pectoralis major	14 (9.3–26)	17 (7.5–25)	−1.9	0.057
Teres major	7.7 (4.6–11)	7.9 (5.2–13)	−0.32	0.750
Deltoid	15 (8.0–27)	18 (11–30)	−0.03	0.975
Standardized antagonistic EMG (μV)				
Latissimus dorsi	1.0 (0.4–2.2)	0.8 (0.4–1.5)	−1.9	0.057
Pectoralis major	1.3 (0.7–3.0)	1.5 (0.8–3.8	−0.09	0.926
Teres major	2.6 (1.3–3.7)	2.8 (1.0–4.4)	−0.48	0.633
Deltoid	1.1 (0.5–2.9)	1.1 (0.7–2.6)	−0.42	0.673

Muscle activation patterns assessed using electromyography and expressed as the Activation Ratio (AR) are compared before and after lidocaine infiltration with paired t-tests or Wilcoxon Signed Rank test depending on the distribution of data. SAPS, Subacromial Pain Syndrome. Statistically significant differences are presented in bold.

A different pattern was observed regarding the teres major where there was no difference in co-contraction before and after administration of subacromial analgesics. There are currently no studies to compare our results with, however, our finding may be explained in light of previous findings using the activation ratio. First, in a study showing that patients with SAPS exhibit decreased teres major co-contraction during abduction, the theory was raised that painful irritation of subacromial tissues in SAPS may (in part) be explained by insufficient humeral head depression during abduction by the teres major (Hik and Ackland, 2019; Overbeek et al., 2019a). In a second study, increased teres major co-contraction in patients with SAPS towards the degree observed in asymptomatic controls, was associated with reduction of pain, again suggesting that teres major co-contraction is a physiologic finding that may protect from pain (Beaudreuil et al., 2011; Overbeek et al., 2018; Overbeek et al., 2019a). Thus, assuming that teres major co-contraction is physiologic and not a consequence of pain, it seems plausible that the degree of teres major co-contraction in this study did not change after subacromial Lidocaine infiltration.

Lastly, regarding the pectoralis major, no changes in activation ratio were observed after the intervention. Although biomechanical evaluations have subscribed a role to the pectoralis major muscle as a potential humeral head depressor, no clinical evidence is present yet in patients with SAPS (Hik and Ackland, 2019; Steenbrink et al., 2009). In our study, this may partly be explained by the positioning of the arm during measurements in anteflexion, adduction and internal rotation.

This study has several limitations. First, although all patients verbally reported reduced subacromial pain after intervention, it is well likely that the responsiveness to subacromial injections varied per patient (e.g. due to uncontrolled administration) reducing the power to find differences in activation patterns after intervention. The fact that the level of latissimus dorsi co-contraction did change after the intervention suggests that the decrease in pain was sufficient to elicit changes in muscle activation patterns. Second, in this study we assumed that if reduced adductor co-contraction in patients with SAPS is a reaction to pain, it would increase right after administration of local analgesics. The fact that the activation pattern of the latissimus dorsi indeed changed after the intervention (in the expected direction) suggests that this is rightful assumption, however it should be noted that a more gradual reaction is possible. Thirdly, we did not perform an a-priori sample size calculation as this study was part of a larger project, and therefore effect sizes may have be underestimated (de Witte et al., 2011). Fourthly, we used surface electrodes for measurement of EMG-activity, and cannot exclude crosstalk from nearby muscles. Lastly, we evaluated a selection of muscles that affect the craniocaudal position of the humerus relative to the scapula (Halder et al., 2001; Holtermann et al., 2010; Steenbrink et al., 2009). The conclusions of this study may be further put in perspective by adding an analysis of other glenohumeral stabilizers, for example, the teres minor and the infraspinatus and subscapularis.

To conclude, we found that during an abduction movement, pain affects latissimus dorsi co-contraction but has no direct influence on the degree of teres major co-contraction in a group of 34 patients with SAPS. It has been previously shown that patients with SAPS exhibit decreased teres major co-contraction and that increasing co-contraction towards the degrees of observed in asymptomatic age-matched controls, is associated with reduction of pain (Beaudreuil et al., 2011; Overbeek et al., 2018; Overbeek et al., 2019a). The present study confirms that the deficit in teres major co-contraction observed in patients with SAPS is not the consequence of pain, and may represent a target for therapy to overcome perpetuating irritation of subacromial tissues during abduction by increasing humeral head depression.

Declaration of Competing Interest

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