# Adductor co-contraction during abduction: a friend or foe

Overbeek, C.L.

# 3

# Increased co-contraction of arm adductors is associated with a favorable course in subacromial pain syndrome.

Celeste L. Overbeek, MD[1, 2]
Arjen Kolk, MD[1, 2]
Jochem Nagels, MD[1,2]
Pieter Bas de Witte[1]
Peer van der Zwaal MD, PhD[3]
Cornelis P.J. Visser MD, PhD[4]
Marta Fiocco, MSc, PhD[5]
Rob G.H.H. Nelissen, MD, PhD[1, 2]
Jurriaan H. de Groot, Msc., PhD[2]

*[1] Department of Orthopaedics, Leiden University Medical Centre, Postzone J-11-R, PO box 9600, 2300RC Leiden, the Netherlands*
*[2] Laboratory for Kinematics and Neuromechanics, department of Orthopaedics and Rehabilitation, Leiden University Medical Centre, Postzone J-11-R, PO box 9600, 2300RC Leiden, the Netherlands.*
*[3] Department of Orthopaedics, Haaglanden Medical Centre, The Netherlands*
*[4] Department of Orthopaedics, Alrijne Hospital, The Netherlands.*
*[5] Department of Medical Statistics and Bioinformatics, Leiden University Medical Centre, The Netherlands.*

*J Shoulder Elbow Surg; 2018; DOI: 10.1016/j.jse.2018.06.015.*

# ABSTRACT

**Background**

Enhancement of arm adductor activity during abduction (i.e. adductor co-contraction), may be effective in the treatment of Subacromial Pain Syndrome (SAPS). We assessed whether an increase of adductor co-contraction is associated with a favourable course of SAPS.

**Methods**

At baseline and after nearly 4 years of follow-up, electromyography of the latissimus dorsi (LD), teres major (TM), pectoralis major and deltoid muscle was obtained during isometric abduction and adduction tasks in 26 patients with SAPS. Changes in co-contraction were assessed with change in the activation ratio ($\Delta AR$). The AR ranges between -1 and 1, where lower values indicate more co-contraction. Clinical course was determined from an anchor question (*reduced, persistent* or *increased* complaints), the Visual Analogue Scale for pain (*VAS*), and the Western Ontario Rotator Cuff score (*WORC*).

**Results**

In patients indicating persistent complaints (31%), the *VAS* and *WORC* remained stable. In patients who indicated reduced complaints (69%), the *VAS* reduced (z score -3.4, p=0.001) and *WORC* increased (z score 3.6, p<0.001). Unchanged ARs associated with *complaints persistence*, whereas decreased AR of the LD ($\Delta AR_{LD}$: -0.21, 95%CI: -0.36 to -0.06) and TM ($\Delta AR_{TM}$: -0.17, 95% CI: -0.34 to -0.00) coincided with *reduced complaints*. There was a significant between-group difference in $\Delta AR_{LD}$ (-0.35, 95% CI: -0.60 to -0.10) and $\Delta AR_{TM}$ (-0.36, 95% CI: -0.66 to -0.05).

**Conclusions**

Increased co-contraction of the LD and TM is associated with a favourable course of SAPS. This may be explained by widening of the subacromial space accomplished by adductor co-contraction.

**Level of evidence**

Level I; Prospective Design; Prognostic Study



**Key Words**

Shoulder impingement syndrome; electromyography; biomechanical phenomena; co-contraction; teres major; latissimus dorsi

# INTRODUCTION

During abduction of the arm, muscles that generate the moment for shoulder movement simultaneously generate a resultant force through the glenoid that stabilises the glenohumeral joint[1]. Studies have suggested that this active stabilisation is compromised in the Subacromial Pain Syndrome (SAPS), leading to painful upward migration of the humerus[2-6]. Model simulation and radiographic analyses show that humerus cranialisation may be counteracted with activation of arm adductors during abduction (i.e. adductor co-contraction)[7-9]. Therefore, increasing co-contraction of arm adductors like the latissimus dorsi (LD), teres major (TM) and pectoralis major (PM), may be beneficial for patients with SAPS.

Few studies have investigated arm adductor co-contraction in SAPS, and there is currently no evidence for alterations in activation patterns[10-12]. Moreover, longitudinal electromyography (EMG) assessments to support the theory that increasing adductor co-contraction is beneficial in SAPS, are yet lacking. In this study, we tested the hypothesis that increased arm adductor co-contraction would be associated with a favourable course of SAPS. In a prospective cohort with EMG assessment, changes in muscle activation of the LD, TM, PM and deltoid muscle (DM) were related to changes in complaints after nearly 4 years of follow-up.



# MATERIALS AND METHODS

Between April 2010 and December 2012, 32 patients were recruited at the Leiden University Medical Center, Haaglanden Medical Center and Alrijne Hospital, under a previously registered and published study protocol (Netherlands Trial Register No. NTR2283)[13]. Patients with SAPS were selected using strict criteria on clinical examination and magnetic resonance arthrography[13]. Inclusion criteria were a positive Neer impingement test, a positive Hawkins test, and 1 or more additional criteria, including painful arc, shoulder complaints for longer than 3 months or diffuse pain during palpation of the greater tuberosity[13]. Exclusion criteria included, but were not limited to the presence of previous fracture or dislocation of the shoulder, frozen shoulder, comorbidities of the affected shoulder (e.g. tumor, instability), full-thickness rotator cuff tears or calcific tendinitis[13]. All patients gave written informed consent. After a period of usual care (e.g. physical therapy, subacromial injections), the 34 included patients were contacted for a follow-up visit between June 2014 and September 2015.

**Measurement set- up**

For EMG-measurements, participants were standing with the affected arm in external rotation at the side, facing a screen where the recorded force exertion was visualised (**Figure 1**). This testing position with the arm at the side was chosen so that all patients with SAPS could be evaluated, including those who could not abduct (fully) because of pain. We were also interested in typifying muscle activation strategies that patients use to generate an abduction moment, rather than in assessing the influence of pain on muscle activation patterns. In this position of relative rest and during abduction and adduction tasks against a 1-dimensional force transducer at the wrist, EMG of 3 shoulder adductors (LD, TM, PM, clavicular part) and the main shoulder abductor (DM, medial part) were recorded with bipolar surface EMG (DelSys system Bagnoli-16, Boston, MA, USA, interelectrode distance 10 mm, bandwidth 20 to 450 Hz) as previously described in detail[13]. EMG and force signals were analogue-digitally (AD) converted and recorded simultaneously at a sample rate of 2500 Hz. For offset removal, the mean was subtracted and the EMG-signals were rectified and enveloped (moving average) using custom made MATLAB software (MathWorks Inc., Natick, MA, USA). Corrupt EMG data or EMG signals that did not reach a 2-fold signal-to-noise ratio were excluded.



During the measurements, the maximal voluntary force (MVF) was first determined as the lowest absolute value of the *MVF* during isometric abduction and adduction. Second, participants performed an abduction and adduction force task at 60% ± 3.75% MVF. Muscle co-contraction was quantified using the activation ratio (AR), which is a reliable method to interpret EMG activity in a standardised manner and based on the muscles' principal action[14,15]. According to the principle action, muscle activation is expressed as agonistic "in-phase" activation ($EMG^{IP}$) and antagonistic "out-of-phase" activation ($EMG^{OP}$)[15]. For example, activation of the DM, during the isometric abduction *force task* is called $EMG^{IP}$ and activation during the adduction *force task* is called $EMG^{OP}$. These values were used to calculate ARs for the LD, TM, PM or DM ($AR_{muscle}$) using Eq. 1:

$$AR_{muscle} = \frac{EMG^{IP} - EMG^{OP}}{EMG^{IP} + EMG^{OP}} \qquad -1 \leq AR_{muscle} \leq 1 \qquad \textbf{Eq.1}$$

**Outcome measures**

***Co-contraction***

Changes in co-contraction were monitored using the AR (-1 to 1), where lower values indicate relatively more antagonistic activity (i.e. co-contraction)[14]. We also recorded the unstandardised group averages of the agonistic $EMG^{IP}$ and antagonistic $EMG^{OP}$ activity. Lastly, we used the magnitude of the *force task* to assess whether this mediated changes in AR.

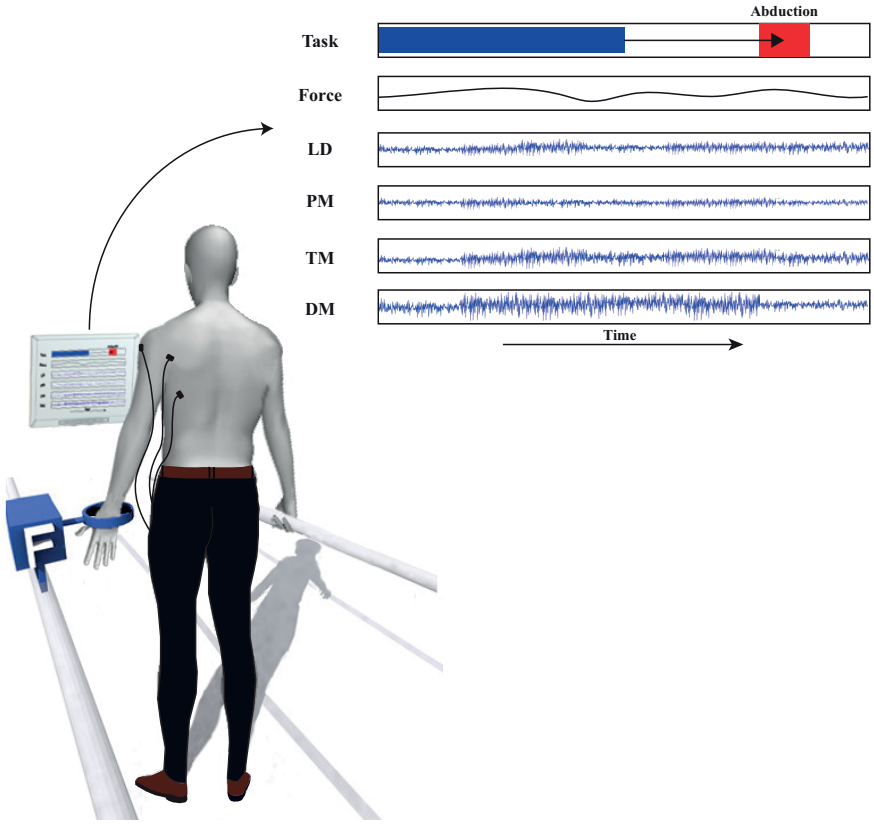


**Figure 1 |** Electromyography measurements during isometric force tasks. *LD*, latissimus dorsi; *PM*, pectoralis major; *TM*, teres major; *DM*, deltoid muscle.

### *Clinical course*

- Anchor question for complaints persistence: The primary end point was an anchor question that assessed whether complaints had changed compared with the first visit, with 3 possible answers: *persistent complaints, reduced complaints* or *more complaints*. For the analyses of the association between ARs and the clinical course, patients were subgrouped according to their answers on the anchor uestion.
- Visual Analogue Scale for pain during motion *(VAS):* pain during arm movement was scored at baseline and follow-up using a 100mm *VAS* scale where 0 indicated no pain and 100 indicated maximal pain. We assessed whether changes in the *VAS* over time corresponded with answers on the anchor question and whether the change in the *VAS* score exceeded the minimal clinically important difference (MCID) of 14mm determined in patients with rotator cuff disease[16].
- Western Ontario Rotator Cuff score (*WORC):* The *WORC* is a clinical score focused at rotator cuff diseases assessing 5 domains in 21 items: physical symptoms, sports

and recreation, work, lifestyle and emotions[17]. The score ranges from 0 (worst possible) to 100 (best possible). We assessed whether changes in *WORC* over time corresponded with answers on the anchor question and whether the change in WORC score exceeded the MCID of 11.7 points determined in patients with rotator cuff disease[17,18].

**Statistical analysis**

Categoric data are described with numbers and percentages. Continuous data are described with means, standard deviation (SD) and 95% confidence intervals (95%CI) in case of normally distributed data or with medians and quartiles in case of nonparametric data (histograms).



We used Linear Mixed Models (LMM) to assess changes and intergroup differences in ARs over time (i.e. $\Delta AR_{muscle}$). Dependent variables were the ARs of the LD, PM, TM or DM. In a fixed effects model, the clinical course was included as a factor and the measurement moment as a covariate. An interaction term between measurement moment and clinical course was included, to assess whether patients with a different clinical course (anchor question), differed in $\Delta AR_{muscle}$. In addition, to rule out that the magnitude of *force task* during EMG tasks mediated possible changes in ARs, we conducted a simple LMM with fixed effect *force task* and dependent variable ARs[19]. Results from the LMM are presented as estimated group means, estimated group differences, 95% CI and p values. Depending on the distribution of data, changes in *VAS* and *WORC* scores over time were assessed by means of the paired samples t test or the Wilcoxon signed rank test. SPSS 20 software (IBM Corp, Armonk, NY, USA) was used for statistical analysis. A 2-sided p value of ≤ 0.05 was considered statistically significant.

# RESULTS

**Patient characteristics**

At follow-up, 3 patients declined participation, 2 could not be contacted, and 1 had died, leaving a study cohort of 26 patients (76%) with baseline and follow-up data. Baseline characteristics of the included patients are described in **Table 1**. During the follow-up period of 3.8 (SD 0.48) years, patients reported to having received only exercise therapy (n=6, 23%), only subacromial infiltrations (n=3, 12%) or both (n=13, 50%), and a wait-and-see policy (n=4, 15%).

**Table 1 |** Baseline characteristics of patients with the Subacromial Pain Syndrome.

| | Total group (n=32) | |
|---|---|---|
| | With follow-up | Loss to follow-up |
| | n=26 | n=6 |
| **Demographics** | | |
| Age, mean (SD) yrs | 50 (6.4) | 53 (4.8) |
| Female, No. (%) | 16 (62) | 3 (50) |
| Right side dominance, No. (%) | 23 (89) | 5 (83) |
| Dominant side affected, No. (%) | 16 (62) | 4 (67) |
| Body Mass Index, mean (SD) kg/m$^2$ | 27 (4.5) | 25 (1.5) |
| Duration of complaints, median (quartiles), mo | 18 (12-29) | 12 (10-30) |

SD, standard deviation.



### Clinical course of complaints

Compared with the first visit, none of the patients had *increased complaints* after the follow-up period, 8 patients (31%) had *persistent complaints*, and 18 (69%) had *reduced complaints*. Of the patients with *persistent complaints,* 1 (13%) reported to have only received subacromial infiltrations and 6 (75%) reported to have received exercise therapy and subacromial infiltrations. In patients with *persistent complaints*, the median *VAS* was 47 (quartiles 19 – 63) at baseline and 54 (quartiles 21 – 77) at follow-up (z score -0.35, p=0.726). Also the *WORC* showed no significant changes in these patients, with median scores of 57 (quartiles 51 – 68) at baseline and 44 (quartiles 34 – 67) at follow-up (Z-score -0.98, p=0.327). Conversely, in patients with *reduced complaints*, the *VAS* reduced from 32 (quartiles 17 – 62) at baseline to 5.9 (quartiles 2.0 – 34) at follow-up (Z-score -3.4, p=0.001), exceeding the MCID[16]. The *WORC* also showed clinical improvement exceeding the MCID with a median score of 60 (quartiles 43 – 74) at baseline and 92 (quartiles 75 – 95) at follow-up (Z-score -3.6, <0.001)[18].

### Muscle activation in association with cli nical course

At baseline, there were no differences in ARs between patients who indicated *persistent* or *reduced complaints* at follow-up (**Figure 2**, **Table 2**). Over time, there were no significant changes in the AR of the LD in patients with *persistent complaints* ($\Delta AR_{LD}$: 0.14, 95%CI: -0.06 to 0.34). However, in patients with *reduced complaints*, the AR of the LD significantly decreased ($\Delta AR_{LD}$: -0.21, 95%CI: -0.36 to -0.06), indicating significantly increased co-contraction. The groups significantly differed in change in $AR_{LD}$ over time (group difference in $\Delta AR_{LD:}$ -0.35, 95% CI: -0.60 to -0.10, p=0.009). Also regarding the TM, patients with *persistent complaints* had no significant changes in the AR ($\Delta AR_{TM}$: 0.19, 95%CI: -0.07 to 0.44), whereas patients with *reduced complaints* had a significant decrease in AR of the TM ($\Delta AR_{TM}$: -0.17, 95% CI: -0.34 to -0.00), indicating increased co-contraction. This resulted in a group-difference of -0.36 (95% CI: -0.66 to -0.05, p=0.023). There were no significant group differences in the $\Delta AR_{PM}$ (-0.08, 95% CI: -0.31 to 0.15) or $\Delta AR_{DM}$ (0.16, 95% CI: -0.01 to 0.32). Lastly, no association was found between the

magnitude of force task during measurements and the AR of the LD (-0.07, 95%CI: -0.24 to 0.11, p=0.438), TM (-0.04, 95%CI: -0.28 to 0.20, p=0.724), PM (0.06, 95%CI: -0.09 to 0.21, p=0.417) or DM (-0.01, 95%CI: -0.11 to 0.10, p=0.886).

### Unstandardised agonistic ($EMG^{IP}$) and antagonistic ($EMG^{OP}$) activity

In accordance with the presented ARs, the coinciding unstandardised $EMG^{IP}$ and $EMG^{OP}$ signals revealed increased antagonistic $EMG^{OP}$ of the LD and TM in the group with *reduced complaints* at follow-up and decreased antagonistic $EMG^{OP}$ of the LD and TM in the group with *persistent complaints* at follow-up (**Table 3**).



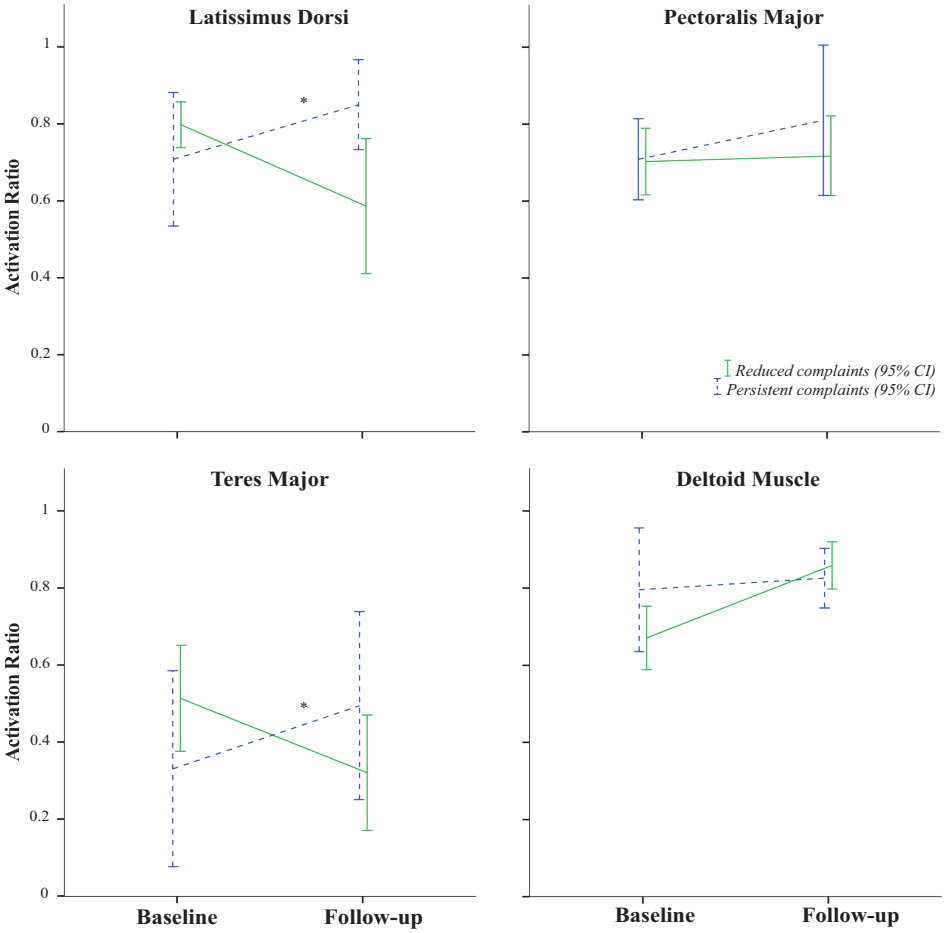


**Figure 2 |** Change in activation ratios over time stratified for shoulder complaints at follow-up. The whiskers represent the 95% Confidence Intervals (CI). Lower activation ratios indicate relatively more co-contraction. *Significant difference ($\alpha$ = 0.05) in activation ratio change between patients with persistent or reduced complaints at follow-up, based on Linear Mixed Model analysis.

**Table 2 |** Activation ratios (AR) associated with complaints at follow-up using Linear Mixed Model analysis.

| | Persistent complaints | | Reduced complaints | | Group difference | | |
|---|---|---|---|---|---|---|---|
| AR | Mean | 95% CI | Mean | 95% CI | Mean | 95% CI | p-value |
| **LD** | | | | | | | |
| Baseline | 0.71 | (0.60 – 0.82) | 0.80 | (0.73 – 0.87) | 0.09 | (-0.04 – 0.22) | 0.165 |
| Follow-up | 0.85 | (0.66 – 1.0) | 0.59 | (0.45 – 0.73) | -0.26 | (-0.50 – -0.03) | **0.031** |
| Delta (Δ*AR*) | 0.14 | (-0.06 – 0.34) | -0.21 | (-0.36 – -0.06) | -0.35 | (-0.60 – -0.10) | **0.009** |
| **PM** | | | | | | | |
| Baseline | 0.71 | (0.59 – 0.82) | 0.70 | (0.62 – 0.78) | -0.00 | (-0.14 – 0.13) | 0.944 |
| Follow-up | 0.80 | (0.64 – 0.97) | 0.72 | (0.62 – 0.81) | -0.09 | (-0.28 – 0.10) | 0.350 |
| Delta (Δ*AR*) | 0.09 | (-0.10 – 0.29) | 0.01 | (-0.11 – 0.13) | -0.08 | (-0.31 – 0.15) | 0.459 |
| **TM** | | | | | | | |
| Baseline | 0.32 | (0.11 – 0.53) | 0.52 | (0.38 – 0.65) | 0.20 | (-0.05 – 0.44) | 0.118 |
| Follow-up | 0.51 | (0.30 – 0.71) | 0.34 | (0.20 – 0.48) | -0.16 | (-0.41 – 0.09) | 0.190 |
| Delta (Δ*AR*) | 0.19 | (-0.07 – 0.44) | -0.17 | (-0.34 – -0.00) | -0.36 | (-0.66 – -0.05) | **0.023** |
| **DM** | | | | | | | |
| Baseline | 0.80 | (0.67 – 0.92) | 0.67 | (0.59 – 0.76) | -0.13 | (-0.28 – 0.03) | 0.100 |
| Follow-up | 0.83 | (0.75 – 0.91) | 0.86 | (0.80 – 0.91) | 0.03 | (-0.07 – 0.13) | 0.528 |
| Delta (Δ*AR*) | 0.03 | (-0.11 – 0.17) | 0.19 | (0.09 – 0.28) | 0.16 | (-0.01 – 0.32) | 0.066 |

CI, confidence interval; LD, latissimus dorsi; PM, pectoralis major; TM, teres major; DM, deltoid muscle. Fixed effects were complaints at follow-up (persistent/reduced complaints), moment (baseline/ FU), moment * complaints. P-values in bold are significant (α = 0.05).

**Table 3 |** Mean agonistic ($EMG_{IP}$) and antagonistic ($EMG_{OP}$) activity at baseline and follow-up.

| | | Baseline | | Follow-up | |
|---|---|---|---|---|---|
| Complaints after follow-up | | Mean (μV) | SD | Mean (μV) | SD |
| **Persistent complaints** | | | | | |
| LD | $EMG^{IP}$ | 14 | 7.2 | 15 | 6.2 |
| | $EMG^{OP}$ | 2.0 | 0.93 | 0.92 | 0.39 |
| TM | $EMG^{IP}$ | 17 | 8.7 | 14 | 12 |
| | $EMG^{OP}$ | 9.0 | 6.1 | 4.0 | 1.9 |
| PM | $EMG^{IP}$ | 32 | 22 | 24 | 17 |
| | $EMG^{OP}$ | 5.2 | 4.6 | 1.6 | 1.1 |
| DM | $EMG^{IP}$ | 44 | 52 | 17 | 15 |
| | $EMG^{OP}$ | 3.1 | 2.4 | 1.7 | 1.7 |
| **Reduced complaints** | | | | | |
| LD | $EMG^{IP}$ | 17 | 13 | 14 | 13 |
| | $EMG^{OP}$ | 1.7 | 1.0 | 3.4 | 4.3 |
| TM | $EMG^{IP}$ | 18 | 9.2 | 22 | 16 |
| | $EMG^{OP}$ | 5.7 | 3.9 | 12 | 8.5 |
| PM | $EMG^{IP}$ | 27 | 16 | 29 | 22 |
| | $EMG^{OP}$ | 4.7 | 3.7 | 4.0 | 4.1 |
| DM | $EMG^{IP}$ | 36 | 22 | 42 | 60 |
| | $EMG^{OP}$ | 6.3 | 3.6 | 1.9 | 1.7 |

SD, standard deviation; LD, latissimus dorsi; $EMG^{IP}$, electromyograph agonistic in-phase activation; $EMG^{OP}$, electromyograph antagonistic out-of-phase activation; TM, teres major; PM, pectoralis major; DM, deltoid muscle.

## DISCUSSION

In this cohort nearing 4 years of follow-up, we found that decreased ARs of the LD and TM were associated with patient-reported *reduced complaints*, significantly decreased pain (*VAS*), and significantly increased quality of life (*WORC*)[16]. These improvements exceeded threshold values for a MCID, thus indicating a clinically relevant improvement[16,18]. A favorable course of SAPS was associated with increased co-contraction of the LD and TM. Conversely, unchanged activation patterns of these adductors were associated with persistent complaints.



Activation patterns of scapular muscles, e.g. upper trapezius, and glenohumeral muscles, e.g. the infraspinatus, have been commonly assessed in the context of SAPS[20]. In contrast, only few studies reported on activity of arm adductors in SAPS, representing a gap in knowledge[10-12]. No differences in adductor activity between patients with SAPS and controls were found in two cross-sectional studies, except for a higher LD activation between 45° and 60° of concentric abduction[10,12]. In another cross-sectional comparison of the affected and unaffected shoulder in SAPS, unaltered activation patterns of amongst others the LD and PM were found[11]. Our study is the first to longitudinally assess adductor activation patterns in association with complaints in SAPS.

The observed association between increased adductor co-contraction and a favourable clinical course may suggest different underlying mechanisms. First, adductor co-contraction may be an adaptation to pain. In the presence of pain, agonistic activity may be reduced and antagonistic activity increased, in an attempt to prevent (further) tissue damage.[21] This theory is supported by several studies that observed acute altered muscle activation patterns, including reduced agonistic deltoid activity, after inducing subacromial pain.[22-24] In our study, EMG was assessed with the arm at the side where patients did not experience complaints; therefore, an acute adaptation to pain is not likely. Furthermore, patients with SAPS had more pain at baseline than at follow-up (*VAS* scores) and complaints at baseline had already lasted for a median of 17 months. Given this state of symptoms and that patients had less adductor co-contraction at baseline than at follow-up, the observed increased adductor co-contraction was unlikely to be an adaptation to pain.

Alternatively, the association between increased adductor co-contraction and a favourable course of SAPS may indicate preceding insufficient adductor co-contraction. In other joints than the shoulder, increased co-contraction has been associated with normal ageing.[25-27] This finding is generally explained as a means to enhance joint stability under the influence of degeneration, e.g. declining

proprioception[25-27]. Possibly, patients with SAPS develop complaints because they adapt insufficiently to such age-related changes in the shoulder. The consequences hereof may be even greater considering previous studies that showed an exaggerated loss of proprioception in SAPS[28-30].

No association was found between co-contraction of the PM and the clinical course of SAPS. Due to the more medially directed force vector of the PM, it may be that the PM is less effective in counteracting cranially directed forces when the arm is hold at the side[7]. In higher regions of abduction, partially also due to presence of pain, co-contraction of the PM may arguably be more effective. Skolimowski and colleagues tested activation of the PM during abduction (whole trajectory) and accordingly suggested development of compensatory activation during this movement.[11]



Our study had some limitations. First, the comparison of ARs between patients with persistent or reduced complaints at follow-up was performed on relatively low numbers of patients. Despite the small sample size, we observed a convincing association between (increased) adductor co-contraction and the reduction of complaints. In the context of these findings and the current tendency toward personalised medicine, we believe that positive results in small study populations are of specific interest. A potential drawback is that findings may not be generalisable due to selection bias. We applied and described strict eligibility criteria to enhance the interpretation and reproduction of our findings. Second, 39 ARs (17%) were missing because EMG-data did not reach the 2-fold signal-to-noise ratio (12%) or was corrupt (5%, e.g. problem with the amplifier). Third, patients were treated according to current clinical practice and we did not control for this. The type of treatment may influence whether or not patients develop adductor co-contraction. However, because it was not our goal to prove causal relationships between adductor co-contraction and complaints persistence, possible confounding by received therapy is not an issue.

To explore whether adductor co-contraction and complaints in SAPS are causally related, we suggest a placebo-controlled intervention study, with, for example EMG-guided exercise of adductors (e.g. humeral depressor exercise)[31]. Furthermore, to gain insight into the underlying mechanism, the association between adductor co-contraction and proprioception may be assessed, as well as the association between adductor co-contraction and ageing.

## CONCLUSIONS

The current prospective cohort comparing patients with SAPS at baseline and after nearly 4 years of follow-up, showed that increased co-contraction of the LD and TM is associated with a favourable clinical course of SAPS. This finding may be explained by the beneficial effect of adductor co-contraction in widening of the subacromial space[7,9]. These results could open a window for research into muscle-specific physical therapy in SAPS.

# REFERENCES


1. Veeger HE, van der Helm FC. Shoulder function: the perfect compromise between mobility and stability. *J Biomech.* 2007;40(10):2119-2129. 10.1016/j.jbiomech.2006.10.016.
2. Chopp JN, O'Neill JM, Hurley K, Dickerson CR. Superior humeral head migration occurs after a protocol designed to fatigue the rotator cuff: a radiographic analysis. *J Shoulder Elbow Surg.* 2010;19(8):1137-1144. 10.1016/j.jse.2010.03.017.
3. Terrier A, Reist A, Vogel A, Farron A. Effect of supraspinatus deficiency on humerus translation and glenohumeral contact force during abduction. *Clin Biomech (Bristol, Avon).* 2007;22(6):645-651. 10.1016/j.clinbiomech.2007.01.015.
4. Reddy AS, Mohr KJ, Pink MM, Jobe FW. Electromyographic analysis of the deltoid and rotator cuff muscles in persons with subacromial impingement. *J Shoulder Elbow Surg.* 2000;9(6):519-523. 10.1067/mse.2000.109410.
5. Graichen H, Bonel H, Stammberger T, Haubner M, Rohrer H, Englmeier KH, Reiser M, Eckstein F. Three-dimensional analysis of the width of the subacromial space in healthy subjects and patients with impingement syndrome. *AJR Am J Roentgenol.* 1999;172(4):1081-1086. 10.2214/ajr.172.4.10587151.
6. Sanchis MN, Lluch E, Nijs J, Struyf F, Kangasperko M. The role of central sensitization in shoulder pain: A systematic literature review. *Semin Arthritis Rheum.* 2015;44(6):710-716. 10.1016/j.semarthrit.2014.11.002.
7. Steenbrink F, de Groot JH, Veeger HE, van der Helm FC, Rozing PM. Glenohumeral stability in simulated rotator cuff tears. *J Biomech.* 2009;42(11):1740-1745. 10.1016/j.jbiomech.2009.04.011.
8. Hinterwimmer S, Von Eisenhart-Rothe R, Siebert M, Putz R, Eckstein F, Vogl T, Graichen H. Influence of adducting and abducting muscle forces on the subacromial space width. *Med Sci Sports Exerc.* 2003;35(12):2055-2059. 10.1249/01.MSS.0000099089.49700.53.
9. Graichen H, Hinterwimmer S, von Eisenhart-Rothe R, Vogl T, Englmeier KH, Eckstein F. Effect of abducting and adducting muscle activity on glenohumeral translation, scapular kinematics and subacromial space width in vivo. *J Biomech.* 2005;38(4):755-760. 10.1016/j.jbiomech.2004.05.020.
10. de Witte PB, Henseler JF, van Zwet EW, Nagels J, Nelissen RG, de Groot JH. Cranial humerus translation, deltoid activation, adductor co-activation and rotator cuff disease - different patterns in rotator cuff tears, subacromial impingement and controls. *Clin Biomech (Bristol, Avon).* 2014;29(1):26-32. 10.1016/j.clinbiomech.2013.10.014.
11. Skolimowski J, Winiarski S, Anwajler J, Skolimowska B, Barczyk K, Dudek K. Bioelectric activity of selected muscle groups in people with impingement syndrome. *Acta Bioeng Biomech.* 2009;11(1):37-45.
12. Bandholm T, Rasmussen L, Aagaard P, Jensen BR, Diederichsen L. Force steadiness, muscle activity, and maximal muscle strength in subjects with subacromial impingement syndrome. *Muscle Nerve.* 2006;34(5):631-639. 10.1002/mus.20636.
13. de Witte PB, Nagels J, van Arkel ER, Visser CP, Nelissen RG, de Groot JH. Study protocol subacromial impingement syndrome: the identification of pathophysiologic mechanisms (SISTIM). *BMC Musculoskelet Disord.* 2011;12:282. 10.1186/1471-2474-12-282.
14. de Witte PB, van der Zwaal P, Visch W, Schut J, Nagels J, Nelissen RG, de Groot JH. Arm adductor with arm abduction in rotator cuff tear patients vs. healthy -- design of a new measuring instrument [corrected]. *Hum Mov Sci.* 2012;31(2):461-471. 10.1016/j.humov.2011.08.007.
15. de Groot JH, Rozendaal LA, Meskers CG, Arwert HJ. Isometric shoulder muscle activation patterns for 3-D planar forces: a methodology for musculo-skeletal model validation. *Clin Biomech (Bristol, Avon).* 2004;19(8):790-800. 10.1016/j.clinbiomech.2004.05.013.
16. Tashjian RZ, Deloach J, Porucznik CA, Powell AP. Minimal clinically important differences (MCID) and patient acceptable symptomatic state (PASS) for visual analog scales (VAS) measuring pain in patients treated for rotator cuff disease. *J Shoulder Elbow Surg.* 2009;18(6):927-932. 10.1016/j.jse.2009.03.021.
17. de Witte PB, Henseler JF, Nagels J, Vliet Vlieland TP, Nelissen RG. The Western Ontario rotator cuff index in rotator cuff disease patients: a comprehensive reliability and responsiveness validation study. *Am J Sports Med.* 2012;40(7):1611-1619. 10.1177/0363546512446591.
18. Kirkley A, Griffin S, Dainty K. Scoring systems for the functional assessment of the shoulder. *Arthroscopy.* 2003;19(10):1109-1120. 10.1016/j.arthro.2003.10.030.
19. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *J Pers Soc Psychol.* 1986;51(6):1173-1182. 10.1037//0022-3514.51.6.1173.

20. Kinsella R, Pizzari T. Electromyographic activity of the shoulder muscles during rehabilitation exercises in subjects with and without subacromial pain syndrome: a systematic review. *Shoulder Elbow.* 2017;9(2):112-126. 10.1177/1758573216660038.
21. Lund JP, Donga R, Widmer CG, Stohler CS. The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. *Can J Physiol Pharmacol.* 1991;69(5):683-694. 10.1139/y91-102.
22. Bandholm T, Rasmussen L, Aagaard P, Diederichsen L, Jensen BR. Effects of experimental muscle pain on shoulder-abduction force steadiness and muscle activity in healthy subjects. *Eur J Appl Physiol.* 2008;102(6):643-650. 10.1007/s00421-007-0642-1.
23. Castelein B, Cools A, Parlevliet T, Cagnie B. The influence of induced shoulder muscle pain on rotator cuff and scapulothoracic muscle activity during elevation of the arm. *J Shoulder Elbow Surg.* 2017;26(3):497-505. 10.1016/j.jse.2016.09.005.
24. Diederichsen LP, Norregaard J, Dyhre-Poulsen P, Winther A, Tufekovic G, Bandholm T, Rasmussen LR, Krogsgaard M. The activity pattern of shoulder muscles in subjects with and without subacromial impingement. *J Electromyogr Kinesiol.* 2009;19(5):789-799. 10.1016/j.jelekin.2008.08.006.
25. Sun W, Liang J, Yang Y, Wu Y, Yan T, Song R. Investigating Aging-Related Changes in the Coordination of Agonist and Antagonist Muscles Using Fuzzy Entropy and Mutual Information. *Entropy.* 2016;18(6)10.3390/e18060229.
26. Billot M, Duclay J, Simoneau-Buessinger EM, Ballay Y, Martin A. Is co-contraction responsible for the decline in maximal knee joint torque in older males? *Age (Dordr).* 2014;36(2):899-910. 10.1007/s11357-014-9616-5.
27. Hortobagyi T, Devita P. Mechanisms responsible for the age-associated increase in coactivation of antagonist muscles. *Exerc Sport Sci Rev.* 2006;34(1):29-35. 10.1097/00003677-200601000-00007.
28. Anderson VB, Wee E. Impaired joint proprioception at higher shoulder elevations in chronic rotator cuff pathology. *Arch Phys Med Rehabil.* 2011;92(7):1146-1151. 10.1016/j.apmr.2011.02.004.
29. Jerosch J, Wustner P. [Effect of a sensorimotor training program on patients with subacromial pain syndrome]. *Unfallchirurg.* 2002;105(1):36-43. 10.1007/s113-002-8163-3.
30. Machner A, Merk H, Becker R, Rohkohl K, Wissel H, Pap G. Kinesthetic sense of the shoulder in patients with impingement syndrome. *Acta Orthop Scand.* 2003;74(1):85-88. 10.1080/00016470310013716.
31. Worsley P, Warner M, Mottram S, Gadola S, Veeger HE, Hermens H, Morrissey D, Little P, Cooper C, Carr A, Stokes M. Motor control retraining exercises for shoulder impingement: effects on function, muscle activation, and biomechanics in young adults. *J Shoulder Elbow Surg.* 2013;22(4):e11-19. 10.1016/j.jse.2012.06.010.