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Original article

# Pathological muscle activation patterns in patients with massive rotator cuff tears, with and without subacromial anaesthetics

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**Abstract**

A mechanical deficit due to a massive rotator cuff tear is generally concurrent to a pain-induced decrease of maximum arm elevation and peak elevation torque. The purpose of this study was to measure shoulder muscle coordination in patients with massive cuff tears, including the effect of subacromial pain suppression.

Ten patients, with MRI-proven cuff tears, performed an isometric force task in which they were asked to exert a force in 24 equidistant intervals in a plane perpendicular to the humerus. By means of bi-polar surface electromyography (EMG) the direction of the maximal muscle activation or principal action of six muscles, as well as the external force, were identified prior to, and after subacromial pain suppression.

Subacromial lidocaine injection led to a significant reduction of pain and a significant increase in exerted arm force. Prior to the pain suppression, we observed an activation pattern of the arm adductors (pectoralis major pars clavicularis and/or latissimus dorsi and/or teres major) during abduction force delivery in eight patients. In these eight patients, adductor activation was different from the normal adductor activation pattern. Five out of these eight restored this aberrant activity (partly) in one or more adductor muscles after subacromial lidocaine injection.

Absence of glenoid directed forces of the supraspinate muscle and compensation for the lost supraspinate abduction torque by the deltoideus leads to destabilizating forces in the glenohumeral joint, with subsequent upward translation of the humeral head and pain. In order to reduce the superior translation force, arm adductors will be co-activated at the cost of arm force and abduction torque.

Pain seems to be the key factor in this (avoidance) mechanism, explaining the observed limitations in arm force and limitations in maximum arm elevation in patients suffering subacromial pathologies. Masking this pain may further deteriorate the subacromial tissues as a result of proximal migration of the humeral head and subsequent impingement of subacromial tissues.


*Keywords:* Shoulder; Muscle; Coordination; Rotator cuff; Tear; Electromyography; Principal action; Pain

## 1. Introduction

Muscle activation patterns (coordination) are bound to change after mechanical deficits like massive rotator cuff tears. Subacromial injection with lidocaine reduces pain and has been shown to coincide with an increase in active forward flexion and muscle strength in patients with specific subacromial disorders like impingement (Ben Yishay et al., 1994). In a comparable intervention it was found that patients with massive rotator cuff tears were well capable of arm abduction despite the absence of supraspinatus force, but were actively hampered to do so due to pain (van de Sande et al., 2006; de Groot

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et al., 2006). Their findings also showed that supraspinatus muscle force was not per se required to produce the necessary glenohumeral abduction torque.

Both series used active and isometric loading by a constant force in a direction rotating perpendicular around the longitudinal axis of the humerus. This so-called principal action method made it possible to define the direction of maximum muscle activation, in combination with the additional compensating muscle activity needed to produce force in exactly that direction (Flanders and Soechting, 1990; Arwert et al., 1997; de Groot et al., 2004; Meskers et al., 2004). The principal action method quantifies shoulder muscle contributions during an isometric force task and facilitates the analysis of the activation patterns of shoulder muscles.

This study was set up to analyse shoulder muscle coordination using the principal action method in patients with massive cuff tears. We analysed activation patterns prior to and after subacromial anaesthetics. In addition to de Groot et al. (2006) we addressed more muscles in order to explain the observed enhancement of external arm force, viz.; the deltoideus (three parts), the latissimus dorsi, the pectoralis major pars clavicularis and the teres major.

## 2. Methods

### 2.1. Subjects

Six male and four female patients (Table 1) with an average age of 61 years (SD = 8) with MRI-proven massive rotator cuff tears were included in the study. All patients were informed and signed informed consent.

### 2.2. Procedure

The principal muscle activation patterns of six muscles were recorded as described by de Groot et al. (2004), and Meskers et al. (2004). Patients were seated with their injured arm in a splint with the humerus positioned in 30° of forward rotation relative to the frontal plane, about 45° elevation and the elbow in 90° flexion (Fig. 1a). The forearm was positioned in about 45° pronation.

The splint was connected to a six degrees-of-freedom force transducer (AMTI-300, Advanced Mechanical Technology Inc., Wavertown MA, USA), which was placed in line with the longitudinal axis of the humerus. Since the force transducer was mounted on a low friction rail aligned with the longitudinal axis of the humerus, forward and backward translations along the longitudinal humerus axis were free. A low-friction ball-and-socket joint was mounted between arm splint and force transducer, which left all rotations of the arm splint relative to the transducer free. The resulting set-up thus only allowed forces in directions perpendicular to the low-friction rail, and thus the longitudinal axis of the humerus (Fig. 1b). To compensate for gravitational effects, the arm was fully supported in rest by means of a weight-and-pulley system.

Force range could be varied from 10–50 N, with steps of 10 N. The external force was primarily set at the highest possible level. If the patient showed signs of serious discomfort, the external force was lowered with steps of 10 N until the patient could exert this force in all 24 directions perpendicular to the humerus. Force magnitude was controlled by a moving cursor on a display, which responded to the force task. The task incorporated a repeated exertion of two consecutive, opposite directions of force exertion; in order to "re-set" the neuro-muscular system to make sure the patients choose their optimal subset of muscle activation and to debar from to much synergistic activation. The patients had to maintain the force for 3 s in each of the 24 directions while simultaneously EMG data were collected (Fig. 1c).

Two different conditions were measured: (1) without anaesthetics; (2) 10 min after subacromial injection of a 10° cc lidocaine 1% solution. Patients were asked to score their experienced pain during both tasks on a 10-point visual analogue scale (VAS).

Table 1
Patients' characteristics

| Patient | Age | Gender | Tear | Origin | Duration (years) |
|---|---|---|---|---|---|
| 1 | 69 | Male | Supra-/and infraspinatus | Chronic | 2 |
| 2 | 54 | Female | Supraspinatus | Chronic | 1.5 |
| 3 | 57 | Male | Supraspinatus | Traumatic | 1 |
| 4 | 50 | Male | Supra-/and infraspinatus | Traumatic | 2 |
| 5 | 72 | Female | Supraspinatus | Chronic | 0.5 |
| 6 | 60 | Female | Supra-/ and infraspinatus | Chronic | 1 |
| 7 | 61 | Male | Supraspinatus | Traumatic | 1 |
| 8 | 67 | Male | Supra-/and infraspinatus | Traumatic | 1.5 |
| 9 | 50 | Female | Supraspinatus | Traumatic | 2 |
| 10 | 66 | Male | Supraspinatus | Traumatic | 1 |

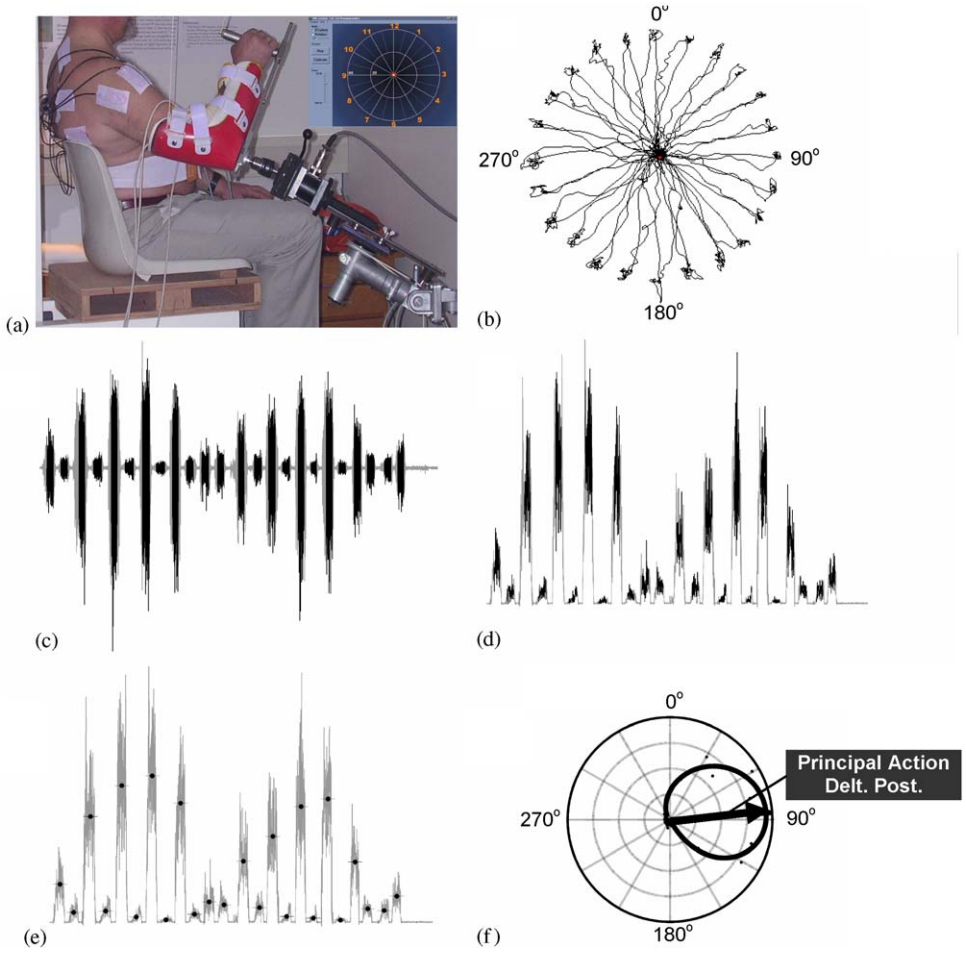


Fig. 1. Principal action method (deltoideus posterior right arm). Patients ($n = 10$) were seated with their injured arm in a splint (a). During an isometric force task in 24 different directions (b) isometrical and isotonic force sections were selected (end trajectory of the circle for every direction) and simultaneously recorded EMG's were identified (black) based on these force selections (c). The rectified and intergrated (d) EMG was subsequently avaraged (e). The EMG-force vectors were plotted in polar coordinates and a curve was estimated through the data points resulting in one direction of maximum muscle activation, the principal action (PA) (f).

### 2.3. Electromyography acquisition and parameterization

EMGs were recorded from the deltoideus (three parts), latissimus dorsi, pectoralis major (pars clavicularis) and teres major using bipolar surface electrodes. Electrodes were placed according to Table 2 (inter-electrode distance 21 mm, maximum skin resistance 10 kΩ, Bandwidth 20–500 Hz, CMRR 86 dB).

For each of the 24 force directions, the rectified (Fig. 1d), averaged EMG over 3 s was determined (Fig. 1e). The magnitudes were normalised between minimum (rest level) and maximum EMG. Force signal and EMG signal were recorded simultaneously. Isometric sections of the force trajectory were identified and simultaneously recorded raw EMG signals were selected (Fig. 1c, black sections) and subsequently rectified (Fig. 1d). An average rectified signal was thus obtained for each of the 24 force directions (Fig. 1e). This signal was reduced by the minimum (assumed rest) level EMG and subsequently normalised relative to the maximum observed EMG. Thus, we obtained the muscle activation level in all directions perpendicular to the longitudinal axis of the humerus.

Table 2
Electrode position for EMG collection

| Muscle | Surface electrode placement |
|---|---|
| Deltoideus anterior | Middle of the muscle belly |
| Deltoideus medialis | Middle of the muscle belly |
| Deltoideus posterior | Middle of the muscle belly |
| Latissimus dorsi | About 6 cm below the angulus inferior |
| Pectoralis major (pars clavicularis) | Middle of the muscle belly of the clavicular part |
| Teres major | Middle of the muscle belly |

Through the force direction related activation levels ($n = 24$) a function was fitted in a least squares sense based on three directional and two amplitude parameters (de Groot et al., 2004). The directional

parameters are expressed by positive values between 0° and 360° ( = 0°). The force direction related angle of maximum muscle activation is referred to as principal action (Fig. 1f). Estimated principal actions were compared with normative values obtained from healthy subjects by Meskers et al. (2004).

### 2.4. Statistics

The magnitude of applied force and the VAS prior to and after subacromial lidocaine injection were compared by means of the paired Student's *t*-test. Changes in PA were tested by means of an ANOVA for repeated measurements and lidocaine treatment as fixed factor.

For individual analysis a principal action change over 90° in one or more muscles was considered a change in activation pattern.

## 3. Results

Subacromial lidocaine injection led to an average significant reduction on the VAS scale ($p = 0.05$), from 7.7 (SD 1.2) to 0.9 (SD 1.6), indicating a strong reduction in pain, although some patients still experienced pain after treatment (Fig. 2a). The exerted arm force during the task could significantly be increased by factor 1.6 ($p = 0.05$) after pain reduction, from 10.4 N (SD 5.7) to 15.7 N (SD 7.4) (Fig. 2b). Patient no. 7 did not respond to the lidocaine injection on any of the three outcome parameters pain, arm force and principal action. Patient no. 3 reported a decrease in pain and an increase in arm force, without any change in principal action. Compared to a normal activation pattern (Meskers et al., 2004), eight out of ten patients showed a pathological muscle activation pattern in which one or more of the adductor muscles showed a

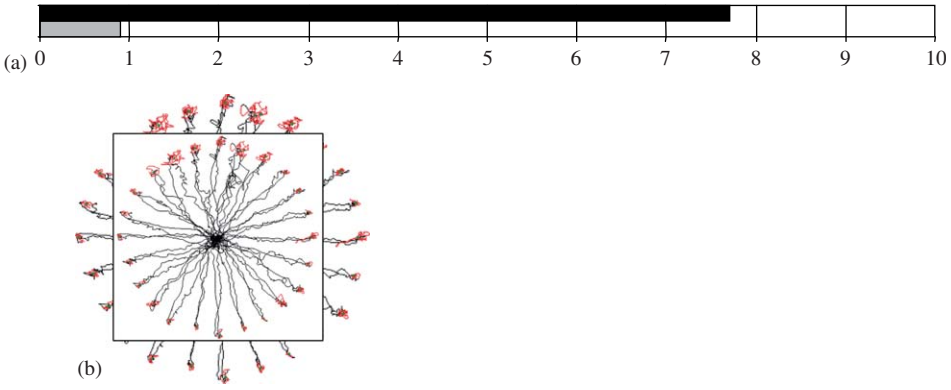


Fig. 2. Effects of lidocaine on pain and arm force: (a) pain scored on visual analogue scale; pain experience decreased significantly after subacromial lidocaine injection ($p = 0.00$), ■: pre-lidocaine, □: post-lidocaine and (b) arm force perpendicular to the humerus; exerted arm force increased significantly after subacromial lidocaine injection ($p = 0.00$).

Table 3
Principal action (°) before and 10 min after subacromial lidocaine. Mean and SD are calculated (after clustering around zero)

| Patient | Principal action (°) | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Delt. ant. | | Delt. med. | | Delt. post. | | Lat. dors. | | Pect. maj. | | Teres maj. | |
| | Pre | Post | Pre | Post | Pre | Post | Pre | Post | Pre | Post | Pre | Post |
| 1 | 346 | 355 | 22 | 355 | 41 | 26 | 21 | 160 | 325 | 306 | 34 | 29 |
| 2 | 11 | 27 | 23 | 27 | 68 | 78 | 210 | 29 | 353 | 319 | 29 | 7 |
| 3 | 345 | 349 | 10 | 349 | 88 | 81 | 162 | 165 | 311 | 306 | 182 | 200 |
| 4 | 56 | 73 | 52 | 73 | 64 | 93 | 53 | 131 | 37 | 156 | 351 | 345 |
| 5 | 314 | 314 | 323 | 314 | 128 | 166 | 168 | 157 | 304 | 280 | 142 | 137 |
| 6 | 17 | 34 | 81 | 34 | 98 | 75 | 37 | 44 | 34 | 257 | 39 | 39 |
| 7 | 4 | 23 | 36 | 23 | 90 | 238 | 320 | 41 | 45 | 49 | 289 | 315 |
| 8 | 333 | 352 | 343 | 352 | 59 | 50 | 147 | 60 | 318 | 324 | 306 | 349 |
| 9 | 341 | 323 | 0 | 322 | 93 | 100 | 334 | 152 | 290 | 306 | 47 | 140 |
| 10 | 360 | 18 | 22 | 18 | 36 | 42 | 44 | 46 | 312 | 309 | 5 | 234 |
| Mean | 357 | 7 | 19 | 7 | 77 | 95 | 78 | 99 | 340 | 297 | 34 | 288 |
| SD | 28 | 35 | 34 | 36 | 28 | 63 | 87 | 59 | 43 | 63 | 78 | 101 |

principal action in the upward/abduction direction, and thus counteracting with the intended mechanical effect as seen in controls. Of these eight patients with pathological adductor activity, five patients restored this aberrant activity (partly) in one or more adductor muscles; which is in accordance with the intended mechanical effect.

For the whole patient group, after lidocaine injection none of the muscles showed significant changes in principal actions. Principal actions prior to and after lidocaine injection are presented in Table 3. Because of the circular nature of the principal action data (0° is equal to 360°) the angles are clustered around zero (negative values are introduced), in order to calculate standard deviations.

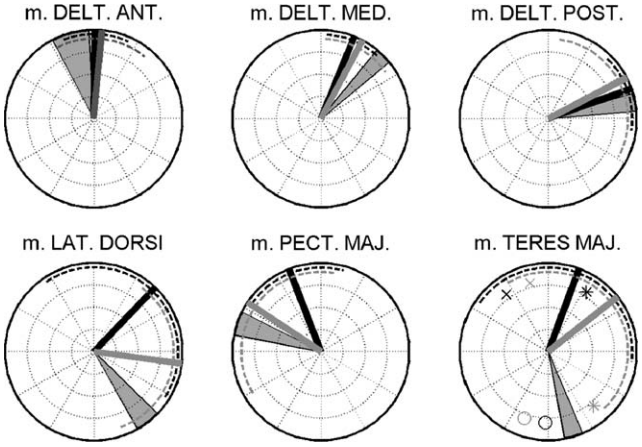


Fig. 3. Coordination of the patients illustrated by the average estimated principal actions for each of the six muscle activation patterns for ten patients relative to the normal activation (Meskers et al., 2004). ▽: The grey surface represents the 99% confidence interval for young healthy subjects according to Meskers et al. (2004). The black line represents the average maximum activation (PA) of 10 patients prior to lidocaine intervention (±SD, dashed). The grey line represents the average maximum activation (PA) after lidocaine intervention (±SD, dashed). For the teres major, the single patient results are added to illustrate three conditions: no change (o): principal action was equal to the normal PA and no change was observed after lidocaine injection. Return to normal (*): a deviating principal action of >90° when compared to normal, which changed to normal after lidocaine injection. Persistent deviation (*x*): a deviating principal action deviating of >90° persisting after lidocaine injection.

## 4. Discussion

As reported earlier (van de Sande et al., 2006; de Groot et al., 2006) and in agreement with impingement (Ben Yishay et al., 1994), external forces increased significantly after subacromial lidocaine injection in patients with massive rotator cuff tears, despite the (partially) absent supraspinatus forces.

The lidocaine intervention did lead to large changes in principal action, but not consistent for all subjects and therefore not significant for the whole patient group. No statistical difference could therefore be identified in the activation patterns of the shoulder muscles before and after subacromial lidocaine injection. Based on the activation of the major (remaining) abductor and adductor muscles we looked for a general coordination change that could explain these observations.

Fig. 3 illustrates the mean principal actions (±SD) for the six muscle (part)s. In eight patients, a pathological adductor pattern could be discerned (upward principal action). On average, the effect of lidocaine appeared to result in a partial normalization of the principal action of the adductor muscles (one or more) of more than 30°. Since major differences existed between patients, this effect could not be statistically demonstrated. Single patient analysis on the deltoideus (three parts) showed that none of the patients changed their PA direction more than 45°, implying relatively little change in muscle activation of the major glenohumeral abductor muscles.

For the adductor muscles, a variety of adaptations after lidocaine injection were observed between patients and between muscles. For every adductor muscle one of the following observations, as illustrated for the teres major in Fig. 3, was seen:

(1) *no change*: the patient's principal action was equal to the normal PA and no change was observed after lidocaine injection. The increase in force exertion could be the result of an equal increase of all muscle forces.
(2) *return to normal*: a deviating principal action over 90° was observed when compared to normal, which changed to normal after lidocaine injection. These patients were indeed able to change their activation pattern within 10 min in response to pain reduction.
(3) *persistent deviation*: a deviating principal action over 90°, persisting after lidocaine injection. Either these patients were still sensitive for the upward glenohumeral translation after pain suppression, or they were not able to restore their activation pattern within short time.

The reason for the persistent deviation could be the duration of the tear and the persistent pathological coordination pattern, which results in a "hard-wired" coordinative adaptation. So far our data do not indicate any relation with duration of the cuff tear.

The observation that firstly the maximum activation direction of the deltoideus hardly changed and that secondly the adductor muscles show a pathological pattern that partly returned to normal after reduction of pain can be explained mechanically, taking the necessary compromise between abduction mobility and required glenohumeral stability into account;

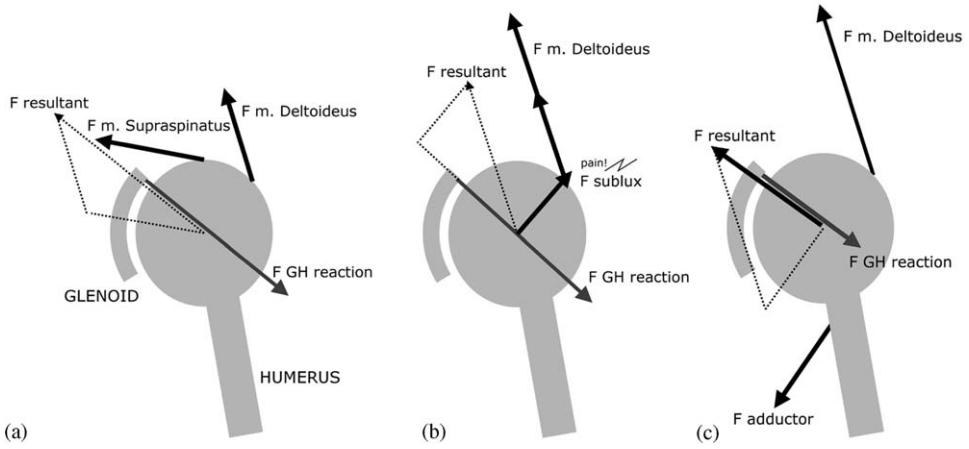


Fig. 4. Schematic representation of muscle contribution and resulting glenohumeral reaction forces in healthy subjects and patients suffering massive cuff tears. (a) Arm elevation in healthy subjects requires an abduction moment along with, glenohumeral force equilibrium, provided by the deltoideus muscles and the supraspinatus. The resultant force (summation of both force vectors; dotted lines) can fully be compensated by the glenoid resulting in a statically stable condition. (b) Compensation of the lost supraspinatus joint torque by the deltoideus is accompanied with an increased upward force, which can only partially be compensated by the glenoid. Without compensation for the remaining force vector, a (painful) upward glenohumeral translation (subluxation) is expected. (c) The upward directed pathological luxating force component prior to the lidocaine intervention can be compensated for by depressor/adductor muscles, e.g. teres major, latissimus dorsi and pectoralis major at the cost of reduction of nett abduction torque.

Arm elevation in healthy patients requires an abduction moment along with glenohumeral force equilibrium (Fig. 4a). Patients suffering from a massive cuff tear have lost the contribution of the supraspinatus and can only compensate this loss of adduction torque by using their deltoid muscles. Relative to the supraspinatus, the deltoideus potentially generate a greater abduction torque. However, the muscle line of action or muscle force vector is more cranial (upward) directed. When activated, the deltoideus therefore generated a greater upward 'luxating' force component relative to the suprasinatus. Compensation of the lost supraspinatus joint torque by the deltoideus is therefore accompanied with an increased upward force (Fig. 4b). Without compensation for this force, there would be a tendency towards (painful) upward glenohumeral subluxation (Fig. 4b). Magermans et al. (2004) indeed illustrated, by model simulation, a glenohumeral reaction force in the superior part of the glenoid in patients with a torn supraspinatus, possibly causing a proximal migration of the humeral head. Compared to healthy patients, eight out of ten patients showed compensation for the pathological supreriorly luxating force component prior to the lidocaine intervention by several depressor/adductor muscles, e.g. latissimus dorsi, pectoralis major and teres major (Fig. 4c). The observed principal action changes imply a change in muscle function, by means of a shift from generating adduction torque, towards generating humeral head depression (stabilization) force. This counterbalance for a threatening upward glenohumeral luxation reduces the overall abduction torque because of the substantial adduction torque function of the adductor muscles. This could explain the observed functional abduction impairment in patients (de Groot et al., 2006).

After lidocaine injection, patients no longer 'sense' the pain due to upward GH subluxation. Adductor muscles are no longer required to reduce pain by pulling the humeral head down. Arm force and arm elevation increase, at the expense of glenohumeral stability and further deterioration of the subacromial tissues.

Limitations of this study, like the small sample size, may influence outcome. The severity of the rotator cuff tears, duration and origin of the cuff tear (acute trauma, chronic) may influence the different patterns of muscle activation and their changes. So far, our data do not reveal such influences. This study did not focus on the interdependency of the different muscle forces in the used measurement, but treated muscle activities as (relatively) independent phenomena. This simplification could lead to unjustified interpretations at the level of the isolated muscle and to unjustified insignificant changes in principal actions. To include interdependencies, a musculoskeletal model (van der Helm, 1994; Magermans et al., 2004) will be required to evaluate the mechanical effect of muscle deficiency in a single muscle on all muscles involved.

Our results are coherent with earlier results presented by De Ben Yishay et al. (1994), van de Sande et al. (2006), de Groot et al. (2006). We also found that external forces increased significantly after subacromial lidocaine injection in patients with massive rotator cuff tears, despite the (partially) absent supraspinatus forces. In order to reduce a painful superior translation of the humeral head, arm adductors are co-activated resulting in a reduced maximum arm elevation. Masking this pain

may further deteriorate the subacromial tissues as a result of proximal migration of the humeral head and subsequent impingement of subacromial tissues.

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