Compensatory muscle activation in patients with glenohumeral cuff tears

Steenbrink, F.

Chapter 2

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

Frans Steenbrink[1,2], Jurriaan H. de Groot[2,3], DirkJan (H.E.J.) Veeger[4,5], Carel G.M. Meskers[1,3], Michiel A.J. van de Sande[1,2], Piet M. Rozing[1,2]

[1] *Laboratory for Kinematics and Neuromechanics, Leiden University Medical Center*

[2] *Department of Orthopaedics, Leiden University Medical Center*

[3] *Department of Rehabilitation Medicine, Leiden University Medical Center*

[4] *Department of Human Movement Sciences, MOVE, Vrije Universiteit Amsterdam*

[5] *Department of Biomechanical Engineering, Delft University of Technology*

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Abstract

A mechanical deficit due to a irreparable rotator cuff tear is generally concurrent to a pain-induced decrease of maximum arm elevation and peak elevation moment. The purpose of this study was to measure shoulder muscle coordination in patients with irreparable 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 *ad*ductors (pectoralis major pars clavicularis and/or latissimus dorsi and/or teres major) during *ab*duction force delivery in eight patients. In these eight patients *ad*ductor activation was different from the normal *ad*ductor activation pattern. Five out of these eight restored this aberrant activity (partly) in one or more *ad*ductor muscles after subacromial lidocaine injection.

Absence of glenoid directed forces of the supraspinatus muscle and compensation for the lost supraspinatus *ab*duction moment by the deltoids leads to destabilizing forces in the glenohumeral joint, with subsequent upward translation of the humeral head and pain. In order to reduce the superior translation force, arm *ad*ductors will be co-activated at the cost of arm force and *ab*duction moment.

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.

2.1 Introduction

Muscle activation patterns (coordination) are bound to change after mechanical deficits like irreparable 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 irreparable rotator cuff tears were well capable of arm *ab*duction despite the absence of supraspinatus force, but were actively hampered to do so due to pain (van de Sande et al., 2005; de Groot et al., 2006). Their findings also showed that supraspinatus muscle force was not per se required to produce the necessary glenohumeral *ab*duction moment.

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 muscles contribution 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 irreparable 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 deltoids (three parts), the latissimus dorsi, the pectoralis major pars clavicularis and the teres major.

2.2 Methods

2.2.1 Subjects

Six male and four female patients (Table 2.1) with an average age of 61 years (SD=8) with MRI-proven irreparable rotator cuff tears were included in the study. This study was approved by the institutions medical ethics committee and before entering the study all patients were informed and signed an informed consent.

Table 2.1: Electrode position for *EMG* collection.

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

2.2.2 Procedure

The principal muscle activation patterns of six muscles were recorded as described by De Groot, Meskers and co-workers (de Groot et al., 2004; 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. 2.1a). The forearm was positioned in about 45° pronation.

The splint was connected to a 6 degrees-of-freedom force transducer (AMTI-300, Advanced Mechanical Technology Inc., Wavertown MA, U.S.A.), 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. 2.1b). 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 10-50N, with steps of 10N. 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 10N 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

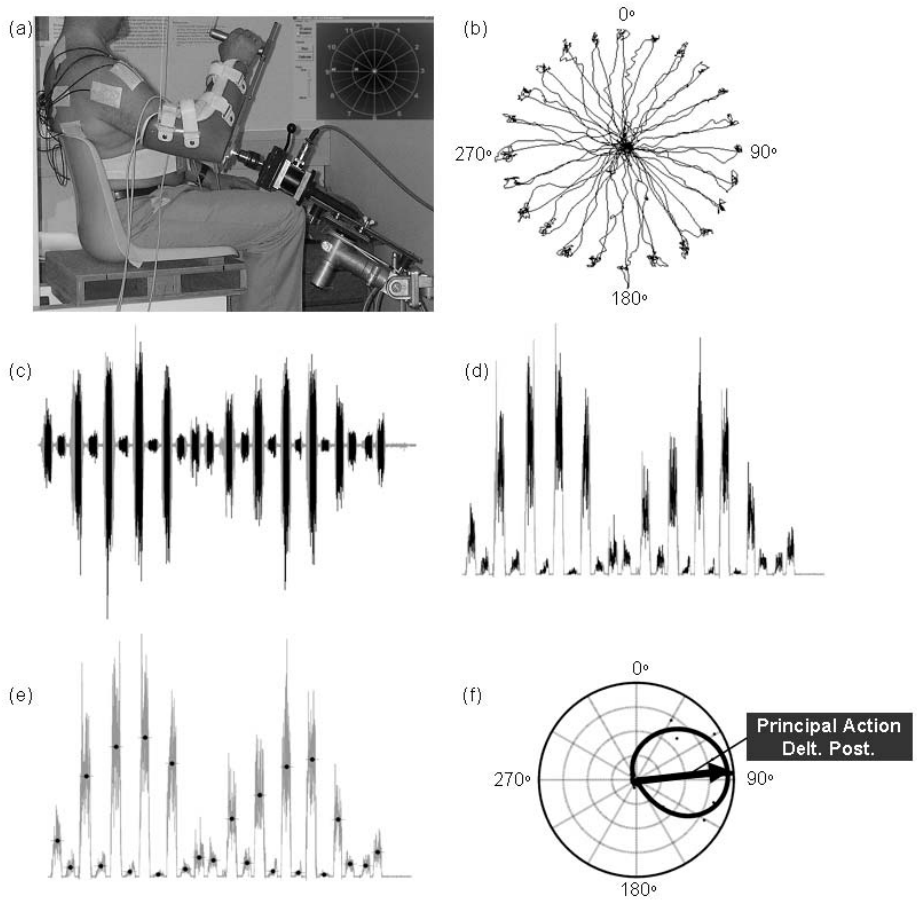

Figure 2.1: *Principal Action* Method (deltoids posterior part, right arm); Patients (n=10) were seated with their injured arm in a splint (a). During an isometric force task in 24 different directions (b) isometric and isotonic force sections were selected (end trajectory of the circle for every direction) and simultaneously recorded *EMG*'s were identified (black) based on these force selections (c). The rectified and integrated (d) *EMG* was subsequently averaged (e). The $EMG\text{-}force$ 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).

Table 2.2: Patients' characteristics.

Muscle	Surface electrode placement
Deltoid anterior	Middle of the muscle belly
Deltoid medialis	Middle of the muscle belly
Deltoid 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

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 seconds in each of the 24 directions while simultaneously *EMG* data were collected (Fig. 2.1c).

Two different conditions were measured:

- without subacromial anaesthetics;
- 10 minutes after a subacromial injection of a 10cc lidocaine 1% solution.

Patients were asked to score their experienced pain during both tasks on a 10-point Visual Analogue Scale (*VAS*) (0: no pain; 100: worst pain ever imaginable).

2.2.3 Electromyography acquisition and parameterization

EMG's were recorded from the deltoids (three parts), latissimus dorsi, pectoralis major (pars clavicularis) and teres major using bipolar surface electrodes. Electrodes were placed according to Table 2.2 (inter-electrode distance 21mm, maximum skin resistance 10kOhm, Bandwidth 20Hz-500Hz, CMRR 86dB).

For each of the 24 force directions the rectified (Fig. 2.1d), averaged *EMG* over 3 seconds was determined (Fig. 2.1e). The magnitudes were normalized 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. 2.2c, black sections) and subsequently rectified (Fig. 2.1d). An average rectified signal was thus obtained for each of the 24 force directions (Fig. 2.1e).

This signal was reduced by the minimum (assumed rest) level EMG and subsequently normalized relative to the maximum observed EMG. Thus, we obtained the muscle activation level in all directions perpendicular to the longitudinal axis of the humerus. Through the force direction related activation levels (n=24) a function was fitted in a least squares sense based on 3 directional and 2 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. 2.1f). Estimated *Principal Actions* were compared with normative values obtained from healthy subjects by Meskers et al. (2004).

2.2.4 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.

2.3 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. 2.2a).

The exerted arm force during the task could significantly be increased by factor 1.6 ($p = 0.05$) after pain reduction, from 10.4N (SD 5.7N) to 15.7N (SD 7.4N) (Fig. 2.2b). Patient number 7 did not respond to the lidocaine injection on any of the three outcome parameters pain, arm force and *Principal Action*. Patient number 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 *ad*ductor muscles showed a *Principal Action* in the upward/*ab*duction direction, and thus counteracting with the intended mechanical effect as seen in controls. Of these eight patients with pathological *ad*ductor activity, five patients restored this aberrant activity (partly) in one or more *ad*ductor muscles; which is in accordance with the intended mechanical effect.

Table 2.3: *Principal Action* (°) before and 10 minutes after subacromial lidocaine. Mean and Standard Deviation are calculated (after clustering around zero).

	Principal Action (°)											
	Delt. ant.		Delt. med.		Delt. post.		Lat. dors.		Pect. maj.		Teres maj.	
Patient	**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	3	7	19	30	67	62	43	99	340	305	21	51
SD	26	35	17	28	21	56	80	59	36	63	73	82

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 2.3. Because of the circular nature of the *Principal Action* data (0° is equal to 360°) the angles had to be clustered around zero (negative values are introduced), in order to calculate standard deviations.

2.4 Discussion

As reported earlier (De Groot et al., 2006, Van de Sande et al., 2005) and in agreement with results from previous studies on the subacromial impingement syndrome (Ben Yishay et al., 1994), external forces increased significantly after subacromial lidocaine injection in patients with irreparable rotator cuff tears, despite the (partially) absent forces of the supraspinatus and infraspinatus muscles.

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 (re-

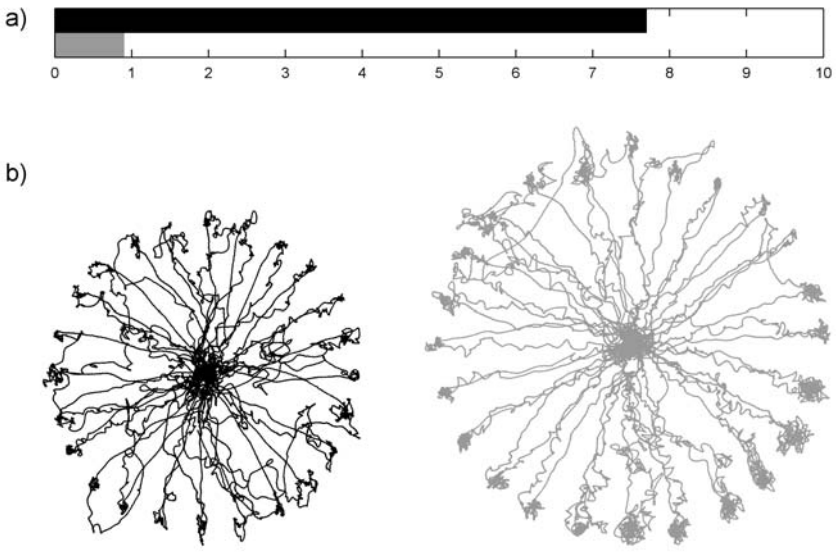

Figure 2.2: Effects of lidocaine on pain and arm force; -: pre-lidocaine, -: post-lidocaine.
a) Pain scored on Visual Analogue Scale; pain experience decreased significantly after subacromial lidocaine injection (p=0.00).
b) Arm force perpendicular to the humerus; exerted arm force increased significantly after subacromial lidocaine injection (p=0.00).

maining) *ab*ductor and *ad*ductor muscles we looked for a general coordination change that could explain these observations.

Figure 2.3 illustrates the mean *Principal Actions* ($\pm$ SD) for the six muscle(part)s. In 8 patients a pathological *ad*ductor 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 *ad*ductor 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 deltoids (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 *ab*ductor muscles.

For the *ad*ductor muscles, a variety of adaptations after lidocaine injection were observed between patients and between muscles. For every *ad*ductor muscle one of the following

observations, as illustrated for the teres major in Figure2.3, was seen:

- no change: the patient's *Principal Action* was equal to the normal *Principal Action* 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.

- 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 minutes in response to pain reduction.

- persistent deviation: a deviating *Principal Action* deviating 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 1) the maximum activation direction of the deltoids hardly changed and that 2) the *ad*ductor muscles show a pathological pattern that partly returned to normal after reduction of pain can be explained mechanically, taking the necessary compromise between *ab*duction mobility and required glenohumeral stability into account;

Arm elevation in healthy subjects requires an *ab*duction moment along with glenohumeral force equilibrium (Fig.2.4a). Patients suffering from a irreparable cuff tear have lost the contribution of the supraspinatus and can only compensate this loss of *ab*duction moment by using their deltoid muscles. Relative to the supraspinatus, the deltoids potentially generate a greater *ab*duction moment. However, the muscle line of action or muscle force vector is more cranial (upward) directed. When activated, the deltoids therefore generate a greater upward 'luxating' force component relative to the suprasinatus. Cmpensation of the lost supraspinatus joint moment by the deltoids is therefore accompanied with an increased upward force (Fig.2.4b). Without compensation for this force, there would be a tendency towards (painful) upward glenohumeral subluxation (Fig.2.4b). 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.

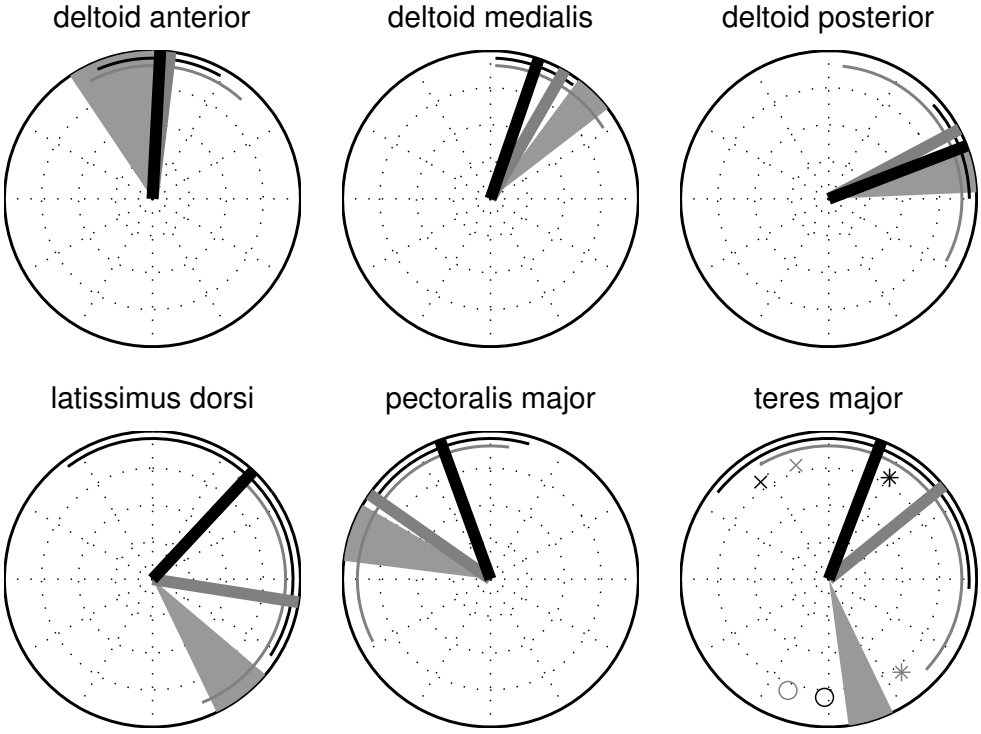

Figure 2.3: Coordination of the patients illustrated by the average estimated *Principal Actions* for each of the 6 muscle activation patterns for 10 patients relative to the normal activation (Meskers et al., 2004). The grey surface represents the 99% confidence interval for young healthy subjects according to Meskers et al., 2004. The black line represents the average maximum activation (PA) of 10 patients prior to lidocaine intervention ($\pm$ SD, dashed). The grey line represents the average maximum activation (PA) after lidocaine intervention ($\pm$ 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 $> 90^{\circ}$ when compared to normal, which changed to normal after lidocaine injection. persistent deviation (x): a deviating *Principal Action* deviating of $> 90^{\circ}$ persisting after lidocaine injection.

Compared to healthy patients, 8 out of 10 patients showed compensation for the pathological superior-luxating force component prior to the lidocaine intervention by several depressor/*ad*ductor muscles, e.g. latissimus dorsi, pectoralis major and teres major (Fig.2.4c). The observed *Principal Action* changes imply a change in muscle function, by means of a shift from generating adduction moment, towards generating humeral head depression (stabilization) force. This counterbalance for a threatening upward glenohumeral luxation reduces the overall *ab*duction moment because of the substantial *ad*duction moment function of the *ad*ductor muscles. This could explain the observed functional *ab*duction impairment in patients (de Groot et al., 2006). After lidocaine injection, patients no longer 'sense' the pain due to upward GH subluxation. *ad*ductor 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 (Magermans et al., 2004, van der Helm, 1994) 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 Groot et al. (2006), van de Sande et al. (2005) and Ben Yishay et al. (1994). We also found that external forces increased significantly after subacromial lidocaine injection in patients with irreparable rotator cuff tears, despite the (partially) absent supraspinatus forces. In order to reduce a painful superior translation of the humeral head, arm *ad*ductors 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.

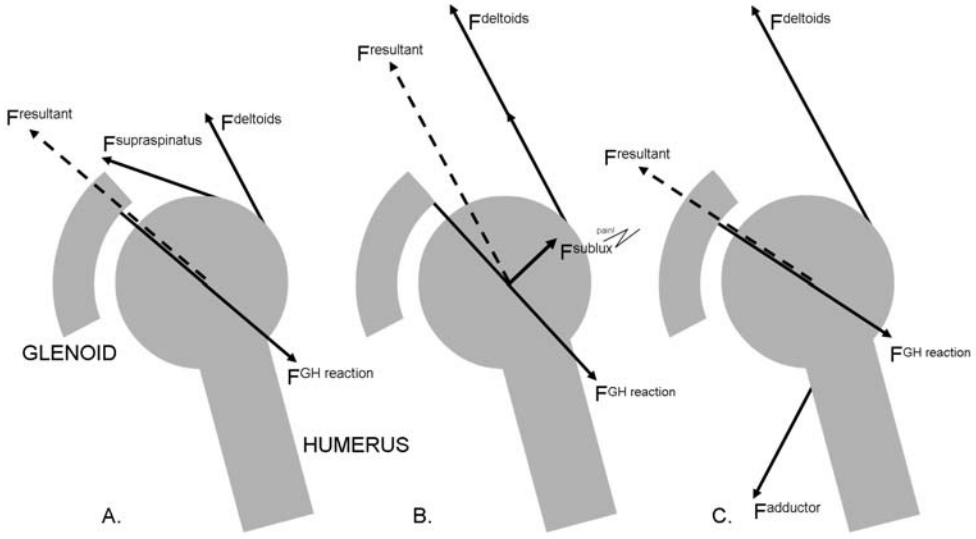

Figure 2.4: Schematic representation of muscle contribution and resulting glenohumeral reaction forces in healthy subjects and patients suffering irreparable cuff tears.

A Arm elevation in healthy subjects requires an *ab*duction moment along with glenohumeral force equilibrium, provided by the deltoid 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 moment by the deltoids 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/*ad*ductor muscles, e.g. teres major, latissimus dorsi and pectoralis major at the cost of reduction of net *ab*duction moment.