Compensatory muscle activation in patients with glenohumeral cuff tears

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Aan mijn ouders.

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Chapter 1

General introduction

1.1 The Shoulder Laboratory

1.1.1 Background

The generic term “shoulder” usually refers to the glenohumeral joint, the main joint of the shoulder girdle, which further comprises the acromioclavicular joint, sternoclavicular joint and the scapulothoracic gliding plane. The glenohumeral joint is modelled with three degrees of freedom (neglecting translations) and is a ball-and-socket joint. The proximal component is the scapula which consists of a concave glenoid covered with a fibro-cartilage labrum that deepens the glenoid cavity (Cooper et al., 1992). The distal component is the proximal part of the humerus, the convex humeral head. Most of the thoraco-humeral motion, i.e. arm movement with respect to the thorax, takes place at the glenohumeral joint, taking into account approximately 120° of the total arm elevation (Magermans et al., 2005), making it the most mobile joint in the human body. This large mobility results from the small articular surface, as well as the loose connecting ligaments and capsules. The capability of exerting arm forces in any direction in each arm position, while preserving joint stability, demonstrates a complex interplay between the different shoulder muscles. Even in a healthy condition it is very remarkable that the glenohumeral joint remains stable during arm motion, as the shoulder does not have a deep socket like the hip joint, or ligaments that are continuously under tension to preserve stability like in the knee. Stability of the shoulder is therefore different compared to these joints, but very effective with respect to the overall degree of mobility. The humeral head, which is slightly smaller than a billiard ball, is centered precisely on the glenoid, which is approximately the size of a desert spoon. It is amazing that such a configuration allows throwing, lifting, pulling and punching while maintaining joint stability.

The glenohumeral joint is considered mechanically stable when the sum of all internal (muscles, ligaments) and external (gravitational) forces working on the humerus, the resultant force vector, aims through the glenoid surface. This resulting force vector can then be fully compensated by the joint reaction force vector which is always directed perpendicular from the glenoid surface. The capsulo-ligamentous system of the glenohumeral joint is not tight enough to prevent joint dislocation (Bigliani et al., 1996), and although the glenoid labrum deepens the glenoid cavity, it is unlikely that it has any contribution to glenohumeral stability because of its flexible property (Carey et al., 2000). Studies with resections of the labrum showed that the average mechanical contribution of the labrum to glenohumeral stability was not very substantial (Halder et al., 2001). It is therefore not surprising that in absence of any muscle activity, the glenohumeral joint can be dislocated with very little effort (Harryman et

al., 1995). Glenohumeral stability, or (re)directing the resultant force vector, is thus mainly controlled by muscle activity (Karduna et al., 1996; Labriola et al., 2005). When the resultant force vector is located outside the glenoid surface it cannot be fully counteracted by the joint reaction force introducing a remaining destabilizing force vector. This destabilizing force component might induce a displacement of the humeral head with respect to the scapula, i.e. glenohumeral instability (Soslowsky et al., 1992), resulting in a (painful) (Soifer et al., 1996) tissue impingement (i.e. subacromial bursa and tendons of supra- and infraspinatus) due to subacromial space reduction (Graichen et al., 1999). To prevent the humeral head from subluxating or dislocating, the muscles spanning the glenohumeral joint must work in a balanced and coordinative way to compress the humeral head against the glenoid surface at all times i.e aiming the resultant force vector working on the humeral head within the glenoid cavity.

The shoulder is driven by 17 muscles, in which some are mono-articular, spanning one joint (with multi degrees of freedom), but the gross is multi-articular, spanning more joints. The muscles from the thorax to the scapula connect the shoulder girdle in a way that there is a support for the humerus, but they can also move the whole shoulder girdle. The shortening range of the larger shoulder muscles is enabled by long fascicle lengths, which, together with the muscle moment arm, enables the shoulder muscles to have a long active force trajectory necessary for the large range of motion (Klein Breteler et al., 1999). The long fascicle lengths also come in handy in cases of non physiological lengthening, i.e. in tendon transfer surgery of either teres major or latissimus dorsi. Roughly speaking, one can distinguish muscles spanning the glenohumeral joint in two groups, namely the prime movers and the prime stabilizers. All muscle contractions affect both mobility of the shoulder as well as stability of the glenohumeral joint (Veeger and van der Helm, 2007), some muscle seem more appropriate for either moving or stabilizing the shoulder. The glenohumeral, or rotator cuff, muscles of the shoulder can be considered as prime stabilisers. Compared to the other shoulder muscles, these cuff muscles have a relative small moment arm, which enable them to be active during a wide variety of tasks without interfering much with the net joint moment. This special anatomy allows the glenohumeral cuff muscles to (re-)direct the resultant force vector working on the humeral head, providing glenohumeral stability during the whole range of glenohumeral joint rotations. Disruptions in the glenohumeral (muscle) force balance are bound to act upon the remaining muscle activation patterns (coordination), directly affecting glenohumeral (in)stability. Although glenohumeral cuff muscle diseases, such as massive cuff tears, rank among the most prevalent musculoskeletal disorders (Yamaguchi et

al., 2006), surprisingly little information is available regarding the remaining compensatory muscle responses in such cases, with respect to the framework of glenohumeral (in)stability.

1.1.2 Setting

The department of Orthopaedics at the Leiden University Medical Center focuses on shoulder pathologies in both clinical and basic research projects. In daily hospital care collaborations between the different departments is desirable in order to achieve the best feasible healthcare and treatment for each individual patient. In research however such collaboration appears to be sub-optimal as for most research projects carried out in these hospitals, groups focus on their own speciality. The work for this thesis was accomplished in the Laboratory for Kinematics and Neuromechanics, in the Leiden University Medical Center (research coordinator dr. ir. J.H. de Groot), which entails a close collaboration between the departments of Orthopaedic surgery (head at start of project prof. dr. P.M. Rozing, current head prof. dr. R.G.H.H. Nelissen) and Rehabilitation medicine (head prof. dr. J.H. Arendzen) and more recently with the departments of Neurology and Geriatrics.

The work for this thesis was also done in a close collaboration between the faculty of Human Movement Science of the Vrije Universiteit of Amsterdam, MOVE, and the department of Biomechanical Engineering of the Technical University Delft, in what is called the Dutch Shoulder Group. In this research group the mobility, stability and the loading of the glenohumeral joint plays a central role and the collaboration had a kick-off at the end of the eighties. The scope was to combine knowledge of both the different medical and technical disciplines. In Leiden this has led to successful finished research projects and the development of essential tools for measuring upper extremity function (Meskers, 1998; de Groot, 1999; Stokdijk, 2002; van de Sande, 2008). In the Laboratory for Kinematics and Neuromechanics, a continuum in shoulder research is accomplished in order to understand both normal and pathological shoulder functioning. Clinical questions on the best treatment options for specific shoulder disorders are addressed by searching for the mechanical responses of patients suffering irreparable glenohumeral cuff tears. Knowledge of healthy shoulder functioning appears to be lacking, and research on pathological functioning and the difference from healthy controls seems to be a proper way to learn more about normal functioning.

A shoulder laboratory is constantly developing new tools and improving existing tools, all with the purpose to most accurately register (pathological) shoulder function. By combining different tools from clinical and technical origin, and analyzing outcome crosswise, the shoulder laboratory is a very powerful tool in current state of the art shoulder research. Basically,

besides the common measurements like maximal arm force, pain-and function scores, the shoulder laboratory features three main techniques to describe the (pathological) functioning of the human shoulder, which are the assessment of muscle function, (scapula) kinematics and biomechanical shoulder model simulations.

1.2 Tools

1.2.1 Muscle function

Shoulder muscle function can be studied by experimentally assessing muscle activation using electromyography (EMG), either by surface or fine-wire electrodes. Because of modulation effects of muscle moment arms during arm motion the most dependable interpretations of EMG can be done when recorded during isometric tasks (de Groot et al., 2004). EMG analysis in this thesis is therefore solely recorded during isometric contractions in a static and critical (de Groot et al., 2006) arm position. In order to achieve the contributions of a muscle(group) to glenohumeral joint loading we asked patients/subjects to exert arm forces in various directions perpendicular to the longitudinal axis of the humerus. Muscle activation will be provoked depending of the different loading directions, allowing us to compare glenohumeral shoulder muscle function between patients and healthy subjects. By relating the level of EMG to the direction of arm force exertion we are able to describe normal arm muscle coordination and discriminate pathological conditions (de Groot et al., 2006). This method (de Groot et al., 2004; Meskers et al., 2004) is unique in its sort as for now, and based on an earlier reported electromyography technique (Flanders and Soechting, 1990; Barnett et al., 1999).

1.2.2 Kinematics

Clinical outcome on interventional studies or descriptive studies on shoulder pathologies will often contain an analysis of kinematics, or movement recordings of the shoulder. Several motion analysis systems are available, but since shoulder movements are mainly three-dimensional, an electromagnetic system seems to be most suitable, because the view of the sensors cannot be blocked like in most other (camera) systems. The “Flock of Birds” (FoB) is a six-degree of freedom electromagnetic tracking device (Ascension Technology Corp, Burlington, VT, USA) for obtaining 3D kinematical data. It consists of an extended range transmitter and several wired receivers, which, for shoulder kinematic recordings are attached

to the thorax, scapulae and both upper and lower arms. A freely movable receiver mounted on a stylus then is used to point out different bony landmarks. Position and orientation of the stylus receiver are recorded together with the position and orientation of the segment receivers which is required to define the position of the receivers relative to the bony segments of interest. The bony landmarks of the thorax can be related to the thorax receiver, the bony landmarks of the scapula to the scapula receiver and the humerus bony landmarks to either the upper-or forearm receiver. 3D positions of the bony landmarks can be reconstructed in every recorded arm position from the orientation and position of the bone receivers (Meskers et al., 1998). The recorded arm kinematics can subsequently be used as input for biomechanical model simulations.

1.2.3 Model simulation

Inverse-dynamic simulations, using the Delft Shoulder and Elbow Model (DSEM)(van der Helm, 1994), are used in this thesis to estimate muscle forces to compare them to EMG data and to study the activation of muscles that were not (easily) accessible with EMG electrodes. Furthermore the DSEM is used to calculate the direction of the glenohumeral joint reaction force vector to investigate glenohumeral (in)stability, which cannot be measured simultaneously with muscle activation in a movement laboratory setting. The DSEM is a musculoskeletal model consisting of 139 functional different muscle elements (van der Helm et al., 1992; Veeger et al., 1997; Klein Breteler et al., 1999). The model can be used to estimate the joint reaction force and the individual muscle forces. From the position and orientation of the thorax, clavicle, scapula, humerus, radius and ulna the moment arms of all modelled muscle(element)s with respect to the joint can be calculated. The effect of muscle activation in each recorded arm position can be studied using the Potential Moment Vector (*PMV*). With this the agonists and antagonists for a specific task can be identified (Veeger and van der Helm, 2007). For every task and in every position several synergists can be identified. We must assume that the distribution of muscle forces over the available muscles is done according to an optimalisation principle. This is necessary, since at the shoulder joint the number of potential synergists exceeds the number of required synergists. This is called the indeterminacy or load sharing problem, which must be solved using an optimalization criteria (Dul et al., 1984; van der Helm, 1994; Meskers, 1998; Praagman et al., 2006) taking muscle size, maximal muscle force (determined by the physiological cross-sectional area (*PCSA*) and the pennation angle) and the force-length relation into account. Besides the desired 'task moment', muscles generate undesirable secondary moments around other degrees

of freedom, e.g. by bi-and triarticular muscles or 2 and 3 degrees of freedom joints like the glenohumeral joint. These moments on their turn must be compensated by additional muscle moments. Simultaneously the already mentioned stability of the glenohumeral joint must be preserved.

While it is not possible to predict the required combination of muscle activation from anatomy books for a healthy shoulder, more strongly this will be impossible in case of lost muscle forces as a result of for example a rotator cuff tear, when compensating muscle activation is needed. Model simulations can help to simulate healthy shoulder function and to understand the response to simulated pathologies. Model outcome can be used for crosswise validation and interpretation with data obtained from *invivo* EMG recordings to study the mechanical effect the muscle activation on glenohumeral (in)stability.

1.3 Aim of this thesis

The aim of this thesis is to demonstrate in patients suffering from glenohumeral cuff tears that activation of the remaining muscles is deviating from muscle activation in healthy subjects. It is hypothesized that during arm elevation moment exertion, deltoid activation is increased in these patients to compensate for lost cuff elevation moment contributions, which painfully jeopardizes glenohumeral stability. To preserve glenohumeral stability, arm *ad*ductor muscles are hypothesized to activate during arm elevation tasks, compensating for lost stabilizing muscle forces, but restricting arm functionality. In this thesis the biomechanical principles of compensatory muscle activation are studied in relation to glenohumeral (in)stability and related to arm function (Range of Motion, function-and pain scores). Knowledge of compensatory muscle activation will provide new insights in future assessment and treatment options for patients with a glenohumeral cuff tear or cuff insufficiencies.

1.4 Outline of this thesis

Compensatory muscle responses (de Groot et al., 2006) in patients with glenohumeral cuff tears were suggested to be imposed by a trade-off between glenohumeral stability and arm mobility, and triggered by pain due to glenohumeral instability and subacromial tissue clamping. Therefore muscle activation in patients with rotator cuff tears were studied before and after subacromial pain suppression (**Chapter 2**). The mechanical properties of the shoulder were thus left unaltered and solely the pain stimulus was suppressed.

Besides being the result to the cuff pathology, muscle activation might also be external load magnitude dependent. This could lead to misinterpretation of activation patterns as being pathological while in fact they are the result of increased maximal arm force after an intervention, such as tendon transfer surgery. The effect of external arm load magnitude loading on muscle activation was assessed both experimentally on healthy subjects and by biomechanical model simulations (**Chapter 3**).

Biomechanical model simulations were also used to study the effect of incrementing cuff tear sizes on the remaining muscle activations and consequences for glenohumeral (in)stability (**Chapter 4**). The contribution of muscle activity on glenohumeral stability was investigated by running shoulder model simulations repeatedly without and with an active modelling constraint for glenohumeral stability.

A clinical intervention to restore arm mobility and decrease pain in patients with irreparable cuff tears is the teres major muscle tendon transfer, which would restore the adverse compensatory muscle activation in these patients with cuff tears (de Groot et al., 2006). Based on previous model simulations (Magermans et al., 2004a; Magermans et al., 2004b) we hypothesized that clinical improvement after a teres major tendon transfer involves alterations in muscle activation. Clinical results were investigated and related to changes in teres major muscle activation before and after its tendon transfer (**Chapter 5**).

Besides having an effect on the humeral head, the teres major potentially also has an effect on scapula orientation. Scapula lateral rotation in shoulders affected by a cuff tear, was compared to lateral rotation of the non-affected contra-lateral shoulder. To study the specific effect of the teres major, lateral rotation after a teres major or a latissimus dorsi tendon transfer was assessed (**Chapter 6**). Additionally, teres major activation was related to scapula lateral rotation and pain scores.

A deferential arm moment loading protocol, based upon compensatory muscle activations, was used on patients suffering from glenohumeral cuff insufficiency and on healthy subjects (**Chapter 7**). Musculoskeletal modeling was applied to analyze muscle forces and glenohumeral (in)stability while electromyography was used to assess muscle activation experimentally.

In the last chapter, the main findings of this thesis are discussed alongside potential clinical implications and suggestions for future research (**Chapter 8**).

Chapter 2

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

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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

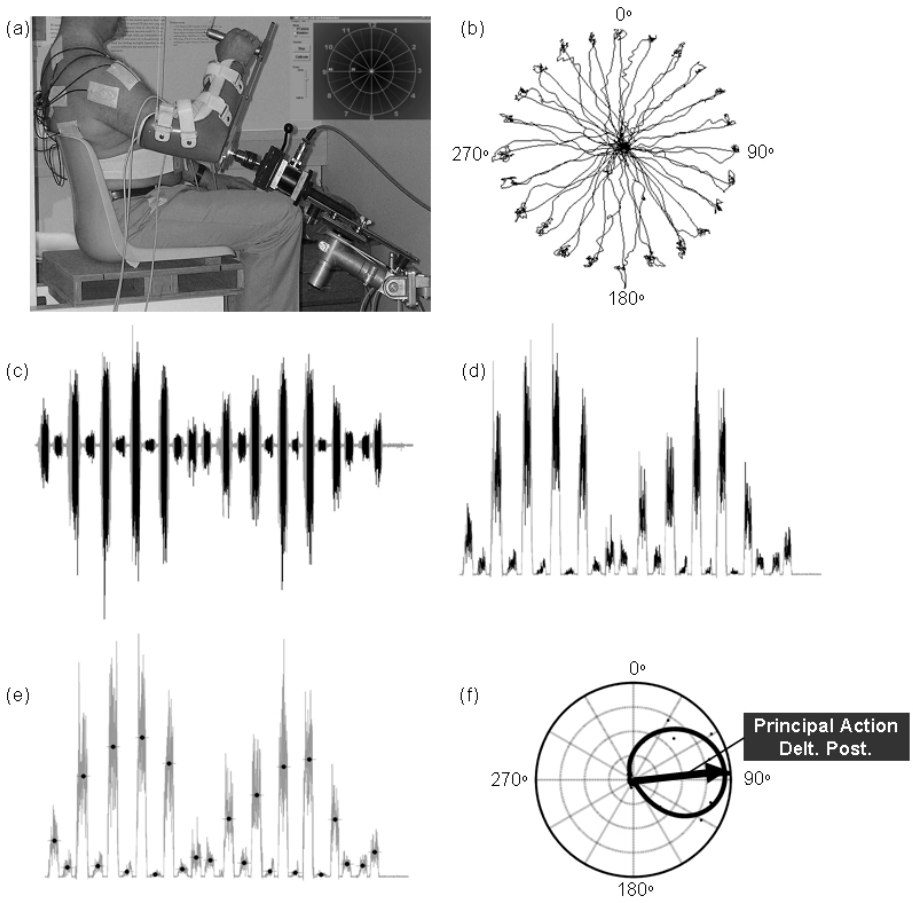

Figure 2.1: *Principal Action* Method (deltoids posterior part, right arm); Patients (n=10) were seated with their injured arm in a splint (a). During an isometric force task in 24 different directions (b) isometric and isotonic force sections were selected (end trajectory of the circle for every direction) 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-

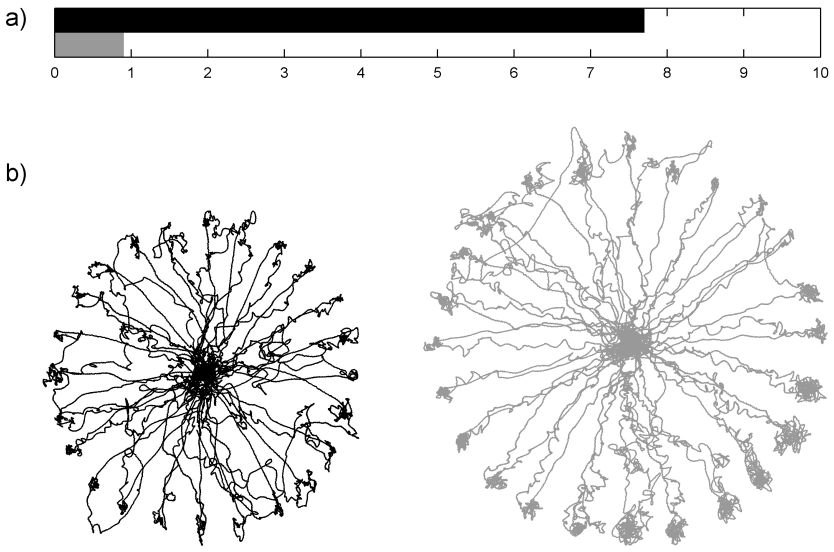

Figure 2.2: Effects of lidocaine on pain and arm force; -: pre-lidocaine, -: post-lidocaine.
a) Pain scored on Visual Analogue Scale; pain experience decreased significantly after subacromial lidocaine injection (p=0.00).
b) Arm force perpendicular to the humerus; exerted arm force increased significantly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

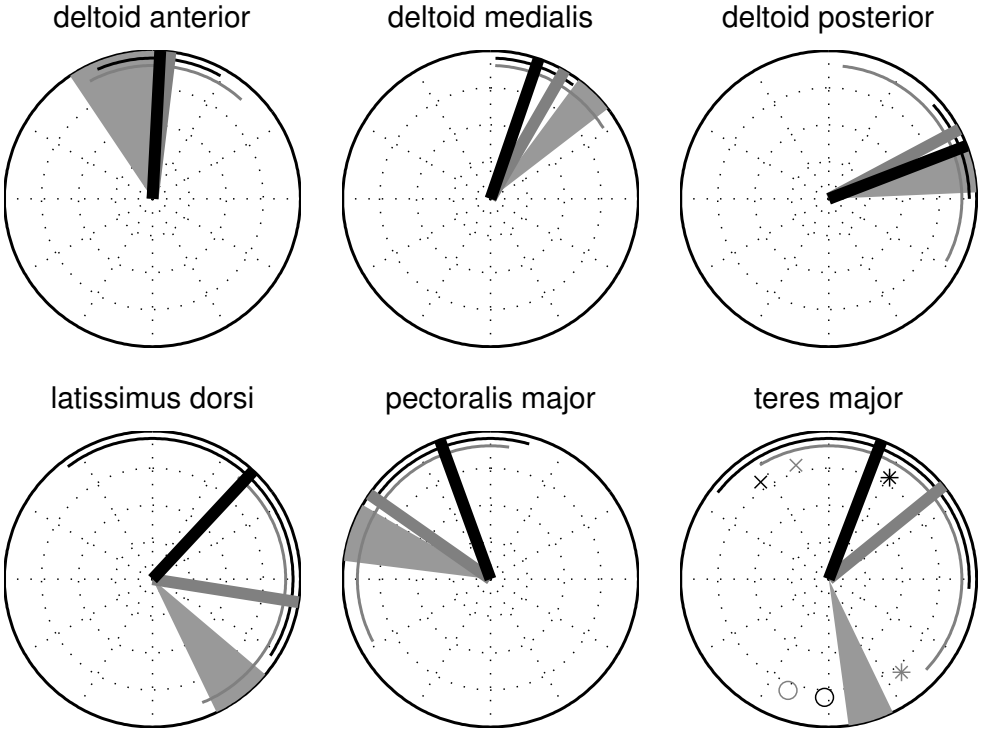

Figure 2.3: Coordination of the patients illustrated by the average estimated *Principal Actions* for each of the 6 muscle activation patterns for 10 patients relative to the normal activation (Meskers et al., 2004). The grey surface represents the 99% confidence interval for young healthy subjects according 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.

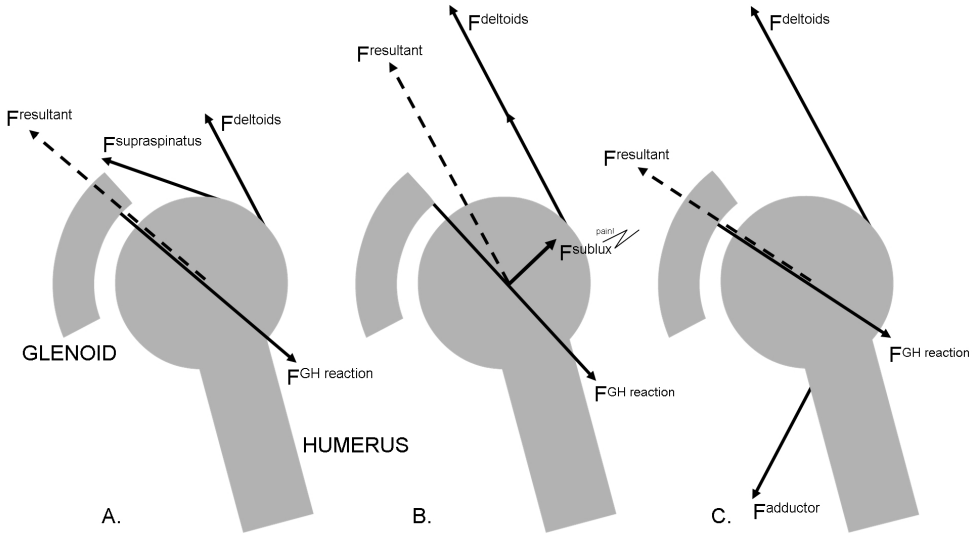

Figure 2.4: Schematic representation of muscle contribution and resulting glenohumeral reaction forces in healthy subjects and patients suffering irreparable cuff tears.

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.

Chapter 3

Arm load magnitude affects selective shoulder muscle activation

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Abstract

For isometric tasks, shoulder muscle forces are assumed to scale linearly with the external arm load magnitude, i.e. muscle force ratios are constant. Inverse dynamic modeling generally predicts such linear scaling behavior, with a critical role for the arbitrary load sharing criteria, i.e. the "cost function". We tested the linearity of the relation between external load magnitude exerted on the humerus and shoulder muscle activation.

Six isometric force levels ranging from 17% to 100% of maximal arm force were exerted in 24 directions in a plane perpendicular to the longitudinal axis of the humeres. The direction of maximum muscle activation (EMG), the experimentally observed so called *principal action* (PA), was determined for each force magnitude in twelve healthy subjects. This experiment was also simulated with the Delft Shoulder and Elbow Model (DSEM) using two cost functions: 1) minimizing muscle stress and 2) a compound, energy related cost function. *Principal Action*, both experimental (PA_{exp}) and simulated (PA_{sim}), was expected not to change with arm forces magnitudes.

PA_{exp} of the trapezius pars descendens, deltoideus pars medialis and teres major changed substantially as a function of external force magnitude, indicating external load dependency of shoulder muscle activation. In DSEM simulations, using the stress cost function, small non linearities in the muscle force-external load dependency were observed, originating from gravitational forces working on clavicular and scapular bone masses. More pronounced non-linearities were introduced by using the compound energy related cost function, but no similarity was observed between PA_{exp} and PA_{sim}.

3.1 Introduction

Individual muscle forces change with armload direction. This load direction dependency was used to study muscle coordination in healthy subjects (Arwert et al., 1997; de Groot et al., 2004; Flanders and Soechting, 1990; Laursen et al., 1998; Meskers et al., 2004) and subjects with shoulder pathologies (de Groot et al., 2006; Steenbrink et al., 2006). The *principal action* (*PA*), which comprehends load direction dependent electromyography (*EMG*) parameters (de Groot et al., 2004; Laursen et al., 1998), is used as a descriptive parameter for muscle coordination. In practice, repeated measurements are performed before and after an intervention, while maximum force around the shoulder may be altered by these intervention, e.g. by pain reduction or muscle tendon transfers (Steenbrink et al., 2006). In the comparison of these experiments we assume that muscle forces scale linearly with external force magnitude. External forces could differ considerably in pre-post measurements (de Groot et al., 2006; Steenbrink et al., 2006) and inter-individually (de Groot et al., 2004; Meskers et al., 2004). So linearity is a pre-requisite, or should be predictable if muscle contraction patterns are to be compared under these relatively different loading conditions. In the jaw, linear scaling of muscle activity (*EMG*) and external load was indeed demonstrated (Blanksma et al., 1992; van Eijden et al., 1993). Non-linear muscle activation scaling with external arm load was however reported in the upper extremity (Happee and van der Helm, 1995).

In shoulder inverse dynamic modeling linearity is generally assumed and incorporated in the load sharing criteria that are needed to mathematically solve the redundancy problem in order to reach a unique muscle activation pattern (de Groot, 1998; Dul et al., 1984; Happee and van der Helm, 1995; Happee, 1994; Tsirakos et al., 1997). Praagman et al. (2006) introduced an energy related criteria with linear and non-linear terms, weighted by morphological parameters as fiber length and muscle mass. This criteria turned out to fit best with non-linear in vivo obtained muscle energy expenditure around the elbow using Near Infrared Spectroscopy. They stated that most cost functions are chosen rather arbitrary, mainly due to the fact that validation is difficult since muscle force cannot be measured accurately in-vivo. The *EMG* based *principal action* method offers an alternative method to compare in vivo with simulated muscle activation, in order to interpret the experimental results and to predict possible load dependencies of shoulder muscles activation patterns in future studies (de Groot et al., 2004; de Groot, 1998).

In the present study we experimentally test the assumption that relative shoulder muscle forces do not change with armload magnitude. The experiment was numerical simulated,

using the Delft Shoulder and Elbow Model (DSEM) with both a linear and an energy related cost function (Praagman et al., 2006; van der Helm, 1994). We used the *principal action*, i.e. the direction of maximum muscle activation assessed by either EMG (experiment) or force (simulation), resp. PA_{exp} and PA_{sim}, as a parameter for muscle coordination.

3.2 Methods

3.2.1 Subjects

Twelve healthy subjects (five female; three left handed) with a mean age of 26 (SD 2.9 years) took part in the study. The local medical ethical committee granted permission and all subjects gave informed consent.

3.2.2 Experimental set-up

Subjects were seated with the dominant arm in a splint with the elbow in 90° of flexion (Fig. 3.1). The setup allowed for static, isometric contractions of shoulder muscles while loading the arm with a force of different magnitudes in different directions in a plane perpendicular to the humerus (de Groot et al., 2004; de Groot, 1998; Meskers et al., 2004). The humeral plane of elevation was approximately 60° rotated externally from the para-sagittal plane and the humerus was 60° abducted. The forearm was 45° externally rotated relative to the horizontal (see Fig. 3.1). The objective of the setup was to record only forces perpendicular to the longitudinal axis of the humerus. In rest, the arm was fully supported by means of a weight and pulley system to compensate for all gravitational forces and moments (de Groot et al., 2004; Meskers et al., 2004). The arm splint was attached to a 6DOF force transducer (AMTI-300, Advanced Mechanical Technology, Inc., Watertown MA, USA) by means of a low friction ball and a socket joint. The transducer was mounted on a low friction rail in line with the humerus. This construction allowed for movement of the arm along 4 degrees of freedom (three rotations and a translation), while translation along the axes perpendicular to the humerus long arm were constrained. These forces controlled the position of a cursor on a computer screen placed in front of the subjects (de Groot et al., 2004; Meskers et al., 2004) (Fig. 3.1).

EMG activity of twelve shoulder muscles was recorded (Table 3.1), and off-line post processed (de Groot et al., 2004; Meskers et al., 2004). Nine shoulder muscles were recorded with the use of bipolar silver bar surface electrodes (DelSys, Bagnoli-16, Boston MA, USA,

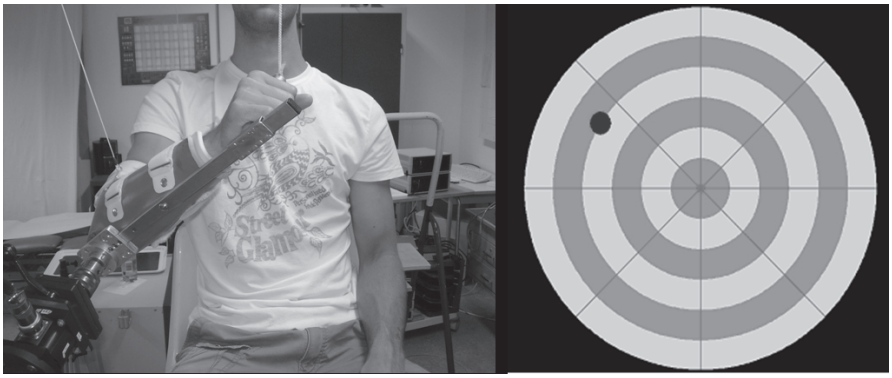

Figure 3.1: Experimental setup (left panel) and visual feedback (right panel); the subject had his arm in a splint, which is connected to a force transducers. Subjects are required to bring the arm force driven cursor (light grey small dot, centered in middle) into the target area (larger dark grey dot, upper left quadrant). The force, perpendicular to the longitudinal axis of the humerus, was recorded with a 6-dof force transducer (AMTI). The target indicated force direction (n=24) and force magnitude, i.e. radius (n=6), resulting in 144 combinations.

analog filter: 20Hz High pass, 450Hz Low pass, 10mm electrode length, inter-electrode distance of 10mm). Sample rate of analog filtered *EMG* and force data was 1000Hz. Before placement of the electrodes the skin was abraded, cleaned and a skin preparation gel (Skin Pure, Nihon Kohden) was used. The *EMG* of the three rotator-cuff muscles was recorded by means of bi-polar wire electrodes (Table 3.1). The wires were made of Teflon coated stainless steel with bare tips of 2mm length and were inserted with a sharp hollow needle. The electrode tips were bent in a sharp angle, so that after withdrawal of the needles, the wires would remain in situ. The wires for the subscapularis were inserted with a curved needle underneath the medial border of the scapula (Kadaba et al., 1992). Before insertion of the needles, the skin was anaesthetized with a 5% lidocaine solution. The needles for the subscapularis and infraspinatus were inserted until the scapular bone was touched.

3.2.3 Protocol

In the experimental set-up the force task existed of moving a cursor, driven by the forces exerted perpendicular to the longitudinal axis of the upper arm on the force transducer, into a target area (Fig. 3.1). Size of the target area was a predetermined area with a range of 3 times standard deviation (SD), determined from measurements on two subjects. Before

Table 3.1: Experimentally recorded shoulder muscles, localization of the electrodes and type of applied electrodes (similar to (de Groot et al., 2004, Meskers et al., 2004)) for comparison).

Muscle	Position	Electrode Type
Supraspinatus	2/3 line trigonum spinae-angulus acromialis 2 cm above spinal ridge	Wire
Infraspinatus	10cm below insertion site supraspinatus	Wire
Subscapularis	Halfway line angulus inferior trigonum spinae, underneath margo medialis	Wire
Trapezius (pars descendens)	2/3 on the line 7^{th} cervical vertebratrigonum spinae	Surface
Trapezius (pars ascendens)	Between the trigonum spinae and the eight thoracic dorsal spine, well above the caudal muscle ridge	Surface
Deltoid (pars anterior)	Middle of muscle belly, deltoideus anterior	Surface
Deltoid (pars medialis)	Middle of muscle belly, deltoideus medial	Surface
Deltoid (pars posterior)	Middle of muscle belly, deltoideus posterior	Surface
Serratus (anterior)	6^{th} head below angulus inferior scapulae	Surface
Teres major	Middle of muscle belly	Surface
Pectoralis major (pars clavicularis)	Middle of muscle belly, pectoralis major clavicular part	Surface
Latisimuss dorsi	6cm below angulus inferior scapulae	Surface

the experiment started the subjects maximum force target magnitude ($F_{\max}$) that could be maintained in all 24 directions was determined. Subsequently, six force levels were applied equidistantly, covering a range from 17% to 100% of $F_{\max}$. The force driven cursor was to be held within the target area for two seconds while the target randomly indicated 24 directions (angle) at 6 force magnitudes (radius), resulting in 144 combinations. Between the trials ample rest of at least five seconds was given in order to avoid too much fatigue effects. Subsequently the *principal action* at each force task could be determined off-line (de Groot et al., 2004; Meskers et al., 2004).

3.2.4 Data post-processing

EMG recordings were full-wave rectified and filtered for visual inspection (3rd order recursive Low Pass Butterworth at $10Hz$). The 2 seconds "in target" full-wave rectified *EMG* was averaged and rest level *EMG* was subtracted. For each of 6 force levels, the averaged

rectified EMG was normalized with respect to the maximum EMG for the appropriate force level. Subsequently, a parameterized least squares curve was estimated through the 24 EMG values to obtain one direction of maximal EMG activity or *Principal Action* (PA_{exp}) for every muscle at force level (de Groot et al., 2004). Outliers and inaccurate estimations of the PA_{exp} were selected and removed by two investigators when consensus was achieved.

3.2.5 Statistical analysis

EMG data were collected for $n = 12$ subjects, $n_m = 12$ muscles, 24 force directions and $n_f = 6$ force levels. We tested the H0-hypothesis that muscle coordination did not change under different load magnitudes i.e. PA_{exp} of each muscle over the 6 force levels was constant. For each individual muscle a regression line, describing the *principal action* of that muscle as a function of force magnitude, was estimated. Subsequently the slope coefficient of this line (β) was tested not to differ from zero.

3.2.6 Model simulations

The experiment was simulated by inverse dynamic numeric modeling using the Delft Shoulder and Elbow Model (DSEM) (van der Helm, 1994). Kinematical input (arm position) was determined using 3D kinematical recording of one subject mounted in the experimental set-up using an electromagnetic tracking device (Meskers et al., 1998b). The ISG standardization protocol for the upper extremity including regression based GH-estimation (Meskers et al., 1998a; Wu et al., 2005) was used. A pointer was used to digitize 14 bony-landmarks with respect to sensors mounted on the thorax, the acromion (Karduna et al., 2001), the upper arm and the forearm. The subjects arm with the sensors attached was positioned into the splint and subsequently the position was recorded. All DSEM simulations were performed using this single position and an external force applied at the elbow in 24 directions at 6 force levels of the models $F_{\max}$, exactly simulating the experiment. In order to simulate the weight compensation on the arm in the experiments, gravity working on the humerus in the model was set to zero. By means of inverse dynamic simulation, muscle forces required to satisfy both the mechanical force-and moment equilibrium were calculated. Two different load sharing criteria were applied: a stress cost function, i.e. minimization of summed squared muscle stresses, and a compound linear and quadratic energy cost function (Praagman et al., 2006). Based on the estimated muscle forces the *Principal Actions* for the muscles in the DSEM were calculated (PA_{sim})(de Groot et al., 2004; de Groot, 1998).

Table 3.2: Average *Principal Action* PA_{exp} (SD) for 6 relative force levels and $n = 12$ subjects. Outliers were excluded resulting in different numbers of observations (N).

	Mean PA ± SD (deg)					
Muscle	**17 (%-Fmax)**	**33 (%-Fmax)**	**50 (%-Fmax)**	**67 (%-Fmax)**	**83 (%-Fmax)**	**100 (%-Fmax)**
Supraspinatus	35.03	15.98	35.91	43.03	42.14	41.18
	(50.89)	(32.11)	(56.29)	(56.00)	(58.23)	(50.35)
	N=8	N=9	N=11	N=9	N=9	N=10
Infraspinatus	6.12	20.95	17.81	15.97	20.56	22.62
	(44.32)	(24.74)	(30.65)	(25.75)	(24.23)	(28.02)
	N=8	N=9	N=11	N=11	N=12	N=12
Subscapularis	164.15	147.63	146.26	152.36	149.84	154.10
	(71.34)	(84.12)	(76.61)	(79.62)	(75.11)	(87.99)
	N=8	N=9	N=10	N=10	N=11	N=10
Trapezius	16.05	11.68	22.62	30.90	36.00	44.79
descendens	(34.53)	(35.73)	(29.69)	(32.77)	(28.99)	(26.82)
	N=9	N=11	N=12	N=12	N=12	N=12
Trapezius	93.70	56.7	79.76	84.57	65.51	80.73
ascendens	(82.84)	(54.82)	(74.74)	(55.73)	(46.30)	(69.66)
	N=9	N=11	N=12	N=12	N=12	N=12
Deltoid	6.46	-14.87	-19.09	-6.41	-6.75	-1.99
anterior	(49.70)	(7.76)	(12.93)	(16.20)	(18.27)	(25.04)
	N=8	N=10	N=12	N=12	N=12	N=12
Deltoid	60.05	62.93	67.83	68.71	68.95	73.02
medialis	(23.95)	(21.73)	(22.44)	(21.99)	(18.89)	(19.82)
	N=10	N=11	N=12	N=12	N=12	N=12
Deltoid	92.52	89.23	91.17	91.23	91.82	93.54
posterior	(16.44)	(14.24)	(9.14)	(9.80)	(16.58)	(11.97)
	N=9	N=10	N=11	N=11	N=11	N=11
Serratus	300.52	300.76	306.48	319.67	316.23	313.61
anterior	(59.69)	(49.82)	(63.58)	(68.83)	(68.49)	(61.88)
	N=5	N=9	N=12	N=12	N=12	N=12
Teres major	218.81	203.97	201.64	175.08	178.56	172.62
	(54.20)	(69.23)	(66.61)	(57.08)	(56.70)	(57.39)
	N=8	N=12	N=12	N=12	N=12	N=12
Pectoralis	265.81	292.98	277.37	255.15	253.37	250.19
major clav.	(49.34)	(26.85)	(27.30)	(67.93)	(63.18)	(66.34)
	N=12	N=11	N=12	N=12	N=12	N=12
Latissimus	158.71	153.80	151.68	137.14	155.69	146.44
dorsi	(38.52)	(18.34)	(25.00)	(18.38)	(43.95)	(22.45)
	N=7	N=10	N=10	N=11	N=10	N=9

Table 3.3: Linear regression slope parameters for the PA_{exp} to external load and their p values. Positive values represent a clock-wise shift of the PA_{exp}.

Muscle	Linear component PA_{exp} (β)	P
Supraspinatus	0.1995	.181
Infraspinatus	0.1362	.515
Subscapularis	0.1897	.322
Trapezius descendens	0.3857	.005*
Trapezius ascendens	-0.0283	.619
Deltoid anterior	0.1172	.156
Deltoid medialis	0.1436	.004*
Deltoid posterior	0.0222	.405
Derratus anterior	0.2143	.400
Teres major	-1.0804	.001*
Pectoralis major clav.	-0.3543	.230
Latissimus dorsi	-0.1204	.286

* Significant differences at $p < 0.05$.

3.3 Results

The average maximum force performed within the study population was 65 Newton (SD = 22.3). PA_{exp} for all muscles and loading conditions, as well as the number of observations after exclusion of outliers, is presented in Table 3.2. The trapezius descendens, deltoid medialis and teres major showed a significant shift of PA_{exp} as a function of external load. The maximum observed effect (teres major) of external loading on PA_{exp} was -1.08° per % of $F_{\max}$. The PA_{exp} dependency was described by a linear regression model (Table 3.3).

In Figure 3.2 changes in *principal action* with respect to *principal action* at the first force level (*principal action* at 17% of $F_{\max} = 0^\circ$) are presented. PA_{exp} are shown (circles), together with PA_{sim}, obtained using both a quadratic stress cost function (upward-pointing triangles) and a compound energy cost function (downward-pointing triangles). DSEM simulations with a quadratic stress cost function showed very small but noticeable non-linear scaling. In our model, we simulated gravity compensation of the humerus, but the observed non-linearities could still be introduced by gravity working on the clavicle and scapular bone, which was obviously not controlled for in the in vivo experiments. To make this effect more clearly visible, we performed model simulations including only one force direction, i.e. a force acting downwards on the arm, with two different magnitudes, i.e. $10N$ and $20N$. We subsequently compared estimated muscle forces in a model with gravity working on the clav-

Table 3.4: By DSEM simulations estimated muscle forces using the stress cost criteria, without (Fg-) and with (Fg+) taking mass of clavicula (0.156 kg) and scapula (0.705 kg)(van der Helm, 1994; Veeger et al., 1997) into account at a vertical downwards directed external load of 10 and 20 Newton respectively. Note that without gravity muscle forces scale linear (exact duplication of estimated muscle force with twice the external load), while non-linearities are introduced with gravity.

	Muscle forces (N)			
	Fg-		**Fg+**	
Muscle	**10N**	**20N**	**10N**	**20N**
Supraspinatus	1.72	3.44	1.70	3.32
Infraspinatus	23.81	47.62	24.69	48.66
Subscapularis	10.67	21.34	10.47	21.10
Trapezius descendens	8.06	16.12	13.21	21.48
Trapezius ascendens	2.71	5.42	3.41	6.04
Deltoid anterior	12.95	25.90	13.09	26.07
Deltoid posterior	0.87	1.74	2.08	3.17
Serratus anterior	15.53	31.06	19.42	34.80
Teres major	0	0	0	0
Pectoralis major clav.	10.28	20.56	11.93	22.50
Latissimus dorsi	0	0	0	0

icle and scapular bone masses, and a model without. Indeed, we found non-linear external load dependence introduced in the first model in contrast to the simulation with full gravity compensation (Table 3.4). The compound "energy cost function" appeared to result in a non-linear relation between PA_{sim} and external load, but except for the supraspinatus no similarity was observed between PA_{exp} and PA_{sim} (Fig. 3.2).

3.4 Discussion

Activation of three shoulder muscles appeared to be load dependent. This has consequences for the interpretation of muscle contraction patterns as measured in patients with shoulder disorders before and after intervention. In current shoulder model simulations (DSEM), non-linearities in the muscle force-external load relationships were not found using a quadratic stress cost function except when gravitational forces working on the clavicular and scapular bones were incorporated. More pronounced non-linearities were introduced using a compound energy related cost function, however not leading to a better resemblance of PA_{exp} to PA_{sim}.

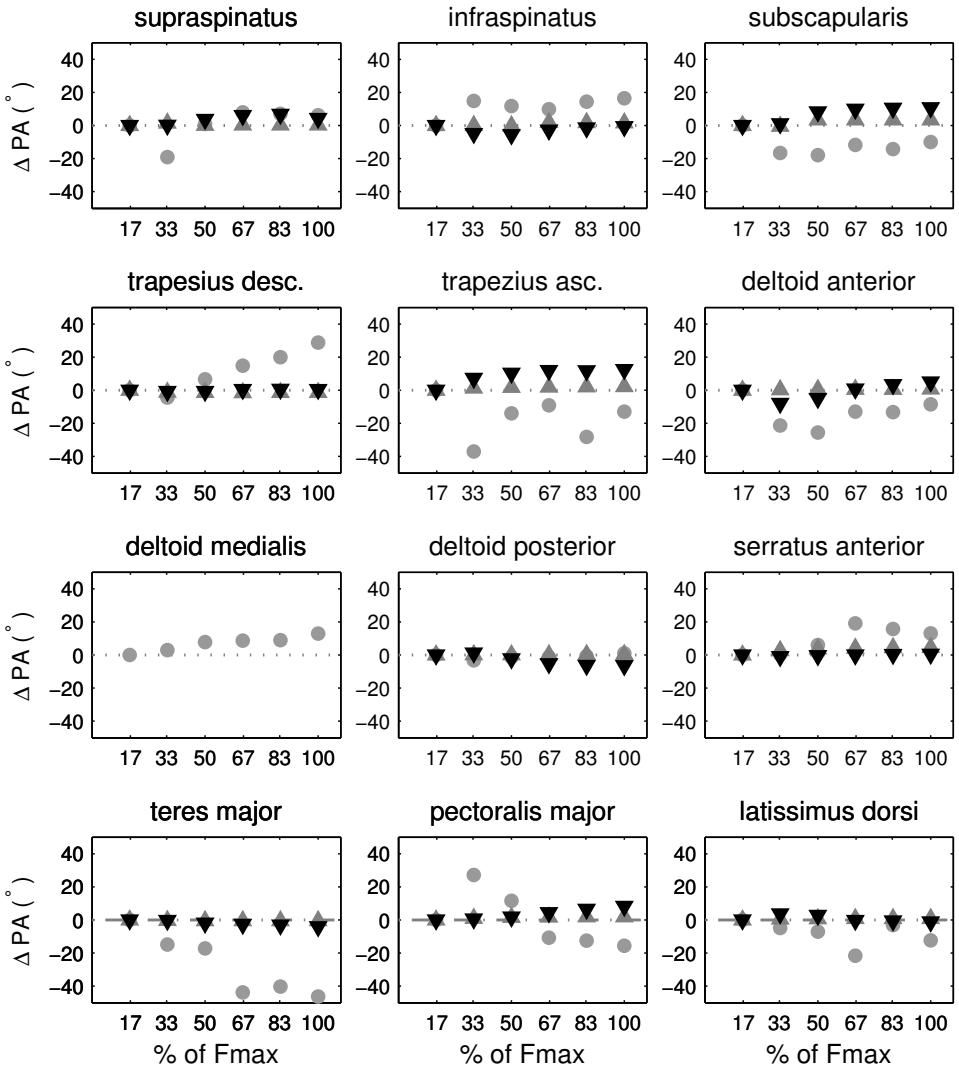

Figure 3.2: Changes in *principal action* with respect to the pricipal action at the first force level (*principal action* at 17% of $F_{\max} = 0°$); PA_{exp} (circles) and PA_{sim} with bone masses of the scapula and clavicle (stress cost function: upward-pointing triangles; energy cost function: downward-pointing triangles). PA_{exp} shows significant non linear relation to external loading for the trapezius descendens, deltoid anterior and teres major. PA_{sim} with the energy cost function and in lesser degree the stress cost function show a non-linear relation with external loading. PA_{sim} of deltoid medialis is lacking because the deltoids in the DSEM are divided in a clavicular part (presented with the deltoid anterior) and a scapular part (presented with the deltoid posterior).

3.4.1 Comparison with previous research

Only a few studies assessed load dependency of muscles in vivo. In a previous study by Meskers et al., external load dependency of shoulder muscle activation was found during a similar multi directional task using a similar *EMG* processing method (Meskers, 1998). In that study, clockwise shifts of deltoideus pars medialis (60°) and counter clockwise shifts of the serratus anterior (6°) and latissimus dorsi (20°) were found. However in contrast to the present study: 1) fixed force levels were used without normalizing, meaning that subjects were measured at different percentages of $F_{\max}$; 2) the external loads and force angles were not applied in randomized order, which might introduce muscle activation dependent recruitment bias and fatigue effects at the higher load tasks; 3) the positioning of the subjects in the present study was slightly different, i.e. the elevation angle was 15° lower.

Recruitment of muscles as a function of external load was studied on jaw muscles using a similar technique of relating *EMG* activity to increasing external forces (Blanksma et al., 1992; van Eijden et al., 1993). With increasing external forces, linear *EMG*-external force relationships where found for each jaw muscle (part). It was concluded that an increase in activity is achieved by the same, simultaneous increase in excitation activity. This would consequently imply a load independent *principal action* direction. Praagman et al. (2006) also reported linear scaling of muscle forces with external loading around the elbow by means of biomechanical model simulation using DSEM and muscle energy expenditure using Near Infrared Spectroscopy. Possible explanations of the discrepancy of the present study with previous work are that with 24 force directions in a full circle around the humerus, the resolution in the present study was considerable higher than in aforementioned studies.

3.4.2 Clinical consequences

In clinical settings, data are not acquired at different magnitudes of external force but at (near) maximum MVC (de Groot et al., 2006; Steenbrink et al., 2006). Thus influences of external loading, cross-talk and *principal action* estimation accuracy were presumed to be minimal. The maximum force a patient can exert will generally change as a result of therapeutic interventions. In patients it is therefore recommended to acquire *Principal Action* data at equal percentages of their $F_{\max}$.

The maximum effect of external loading on the *principal action* will not exceed 1.08° per percentage of MVC or Newton, resulting in 16° *principal action* shift for an external force change of 15N for the teres major. In pre-and post-intervention comparisons this is in the

range of the inter-subject standard deviation and is substantially less than e.g. observed in patients with massive cuff tears where shifts for teres major increased 75° (de Groot et al., 2006; Steenbrink et al., 2006). These large *principal action* changes observed in patients cannot be explained by external force dependency but are obviously pathology dependent.

3.4.3 DSEM: load sharing criteria

The applied load-sharing functions either constrain or introduce non-linear scaling. The quadratic stress minimization allows synergy between agonist muscles more than linear criterions (Happee, 1994). The energy-related cost function with a linear and quadratic component was previously shown to lead to more realistic predictions of muscle activation (Praagman et al., 2006) for elbow-forearm external force tasks. Simulating the present experiment with the compound energy related criterion indeed predicted a non-linear external load-dependent muscle contraction, resulting in a better PA_{exp} to PA_{sim} resemblance for the supraspinatus and, at least for the contour also for the deltoid anterior. However, there was no resemblance for the remaining majority of muscles, implying that model simulations do not predict the observed effects in the experiment. In vivo we might apply alternative control strategies that are not caught adequately by the mechanical modeling and force distribution criteria. Additionally, force magnitude and direction induced changes of clavicle and scapula orientation may not be neglected, and should thus experimentally be controlled for, or incorporated in the simulations.

3.4.4 DSEM: gravitational loads

Introduction of gravitational forces resulted in non-linear muscle force-external load relations when the stress cost function was used, especially for the low loading conditions. Gravitation generates constant joint-torques that requires constant muscle force compensation. This baseline muscle loading interacts with the linear increasing external component, resulting in a non-linear appearance. Where bone masses will not be much of a factor, muscle masses probably will. Muscle masses and the application point of gravitational forces on the different muscle volumes are presently not adequately incorporated in the DSEM. Variations in the gravity forces - external load ratio could explain differences of the present findings with respect to the previous studies to some extent (Apreleva et al., 2000; Blanksma et al., 1992; Meskers, 1998; Praagman et al., 2006; van Eijden et al., 1993). It is recommended to take gravitational forces into account in model simulations, especially when the direction of the

external force does not coincide with the direction of the vertical gravitational forces and the moment arms of external force directions are changing.

3.4.5 Possible error sources in the experiment

The validity of the *EMG* model as used in the present study is extensively discussed (de Groot et al., 2004; Meskers et al., 2004). When external force is increased, the signal over noise ratio will increase which will lead to optimal estimates of PA_{exp}. Therefore PA_{exp} estimations at low forces have reduced accuracy. However, it is unlikely that this phenomenon explains the present findings as shifts of *principal action* are not limited to the lower loading conditions.

Influence of cross-talk might also be external load dependent. However, the *principal action* is estimated at the peak of muscle activation and therefore the *principal action* method as such can be considered relatively insensitive to cross talk, even at the lower external loads.

During the experiments the gross position of the subjects was kept constant and special care was taken that subjects could not cheat to be able to meet the higher external forces. Small scapula positional changes could however not be ruled out and because external load direction dependent scapular positions were previously observed (de Groot et al., 2006), these changes are likely to increase with increasing external load magnitude influencing muscle moment arms around the acromioclavicular, sternoclavicular and glenohumeral joints, which affect the *principal action* direction. To what extent *principal actions* change as a function of scapular position changes requires further research.

Chapter 4

Glenohumeral stability in simulated rotator cuff tears

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Abstract

Rotator cuff tears disrupt the force balance in the shoulder and the glenohumeral joint in particular, resulting in compromised arm elevation moments. The trade-off between glenohumeral moment and glenohumeral stability is not yet understood. We hypothesize that compensation of lost *ab*duction moment will lead to a superior redirection of the reaction force vector onto the glenoid surface, which will require additional muscle forces to maintain glenohumeral stability.

Muscle forces in a single arm position for five combinations of simulated cuff tears were estimated by inverse dynamic simulation (Delft Shoulder and Elbow Model) and compared with muscle forces in the non-injured condition. Each cuff tear condition was simulated both without and with an active modeling constraint for glenohumeral stability, which was defined as the condition in which the glenohumeral reaction force intersects the glenoid surface.

For the simulated position an isolated tear of the supraspinatus only increased the effort of the other muscles with 8%, and did not introduce instability. For massive cuff tears beyond the supraspinatus, instability became a prominent factor: the deltoids were not able to fully compensate lost net *ab*duction moment without introducing destabilizing forces; unfavorable *ab*ductor muscles (i.e. in the simulated position the subscapularis and the biceps longum) remain to compensate the necessary *ab*duction moment; the teres minor appeared to be of vital importance to maintain glenohumeral stability. Adverse *ad*ductor muscle co-contraction is essential in order to preserve glenohumeral stability.

4.1 Introduction

Massive rotator cuff tears disrupt the force and moment balance in the shoulder and the glenohumeral joint in particular. This generally coincides with severe pain and disabilities (Iannotti et al., 2006, Jost et al., 2000). Severity of cuff afflictions range from isolated supraspinatus tearing to partial/full tearing of supraspinatus, infraspinatus, teres minor and subscapularis, the so-called massive tears. The biceps longum is known to have a stabilizing effect on the humeral head, but is frequently affected in patients with cuff tears (Warner and McMahon, 1995, Kempf et al., 1999, Murthi et al., 2000).

In patients with massive rotator cuff tears, pathological co-activation of large muscles with an adducting component (teres major, latissimus dorsi and pectoralis major) was observed during an isometric *ab*duction moment task in a single position (de Groot et al., 2006, Steenbrink et al., 2006). This position is considered critical for several shoulder pathologies, i.e. cuff tears, impingement syndrome, arthritis and habitual shoulder instability and/or subluxation. The alteration in muscle activation patterns in these patients was assumed to be the compensatory response for stabilization of the glenohumeral joint. Proximal migration of the humeral head during *ab*duction moments observed in patients with massive rotator cuff tears (Deutsch et al., 1996, Paletta, Jr. et al., 1997, Yamaguchi et al., 2000, Bezer et al., 2005) is assumed to be related to increased deltoid activity (McCully et al., 2006). The deltoids generate an increased force, in order to compensate lost *ab*duction moment of e.g. the supraspinatus which results in an increased upward directed force component on the humeral head, resulting in a proximal migration and risk of compressing the subacromial tissues (Graichen et al., 1999).

We previously postulated that, in order to prevent proximal migration, patients co-activate their *ad*ductor muscles to pull down the humeral head during arm elevation (de Groot et al., 2006, Steenbrink et al., 2006). This coordinative change would restore glenohumeral stability at the cost of arm *ab*duction moment. The objective of this study was to determine, by means of model simulation, the mechanical effect of rotator cuff tears on both muscle force balance and glenohumeral stability. We hypothesize that rotator cuff tears will lead to an upward rotated joint reaction force vector piercing through the glenoid surface. The glenohumeral joint is considered unstable if the joint reaction force vector directs outside the glenoid rim. To redirect the joint reaction force vector through the glenoid surface and restore glenohumeral stability additional muscle forces are required.

4.2 Methods

Massive cuff tears were simulated using kinematic and force data from a previous experiment. Position data were obtained from 15 experimental patient recordings in which the injured arm was secured in a splint in a standardized position (de Groot et al., 2006, Steenbrink et al., 2006).

4.2.1 Simulation design

Inverse dynamic simulations were performed using the Delft Shoulder and Elbow Model (DSEM). Muscle forces in five combinations of simulated cuff tears were estimated and compared with muscle forces in the original condition. Each simulation was performed without and with a constraint for glenohumeral stability, respectively (see paragraph 4.2.3).

4.2.2 Delft Shoulder and Elbow Model

In the Delft Shoulder and Elbow Model (van der Helm, 1994) anatomical structures are represented by appropriate elements (van der Helm et al., 1992; Veeger et al., 1991). The model contains 31 muscles, divided in 139 muscle elements. Musculoskeletal parameters were obtained from extensive cadaver studies (Klein Breteler et al., 1999, Veeger et al., 1991). Input variables for the model are the orientations of the model elements (thorax, clavicle, scapula, humerus, radius and ulna) and direction and magnitude of the external arm load (applied at the olecranon of the humerus). The model calculates muscle forces required to satisfy mechanical force and moment equilibrium. The load sharing criterion J minimized the sum of squared muscle stresses, Eq. 4.1.

$$J = \sum_{i=1}^{n} \left(\frac{F_i}{\mathrm{PCSA}_i} \right)^2 \tag{4.1}$$

Where n is the number of muscle elements. Fi is muscle force produced by muscle element i. $PCSA_i$ is the physiological cross-sectional area of this muscle element.

Maximum muscle element force is depended on the physiological cross-sectional area, $PCSA_i$, the maximum muscle stress ($\sigma = 100 N cm^{-2}$, van der Helm, 1994), the fraction coefficient and a relative force-length function ($f(l_i, l_{fi}) : 0 \leq f(l_i, l_{fi}) \leq 1$), where l_{fi} is the actual element length, and $f(l_{fi})$ the optimal muscle length. If $f(l_i = l_{fi})$ then $f(l_i, l_{fi}) = 0$ (Klein-Breteler et al., 1999).

The fraction coefficient $c_i : 0 \leq c_i \leq 1$ is used to eliminate cuff muscles forces, Eq. 4.2.

$$0 \leq F_i \leq f(l_i, l_{fi}) \cdot \text{PCSA}_i \cdot \sigma_{\max} \cdot c_i \tag{4.2}$$

Where F_i is force of muscle element *i*. $f(l_i, l_{fi})$ is the force-length function. PCSA_i is the physiological cross sectional area. σ_{max} is the maximum muscle stress ($= 100N \cdot cm^{-2}$) and c_i is the fraction coefficient of muscle element *i*, used to eliminate the cuff muscles. When a complete tear is simulated, $c_i = 0$.

4.2.3 The glenohumeral stability constraint

The model allows exclusion or inclusion of a glenohumeral stability constraint. The glenohumeral joint is considered stable when the resultant force vector is aimed within the glenoid surface. If this vector points outside the glenoid surface it cannot be fully counteracted by the joint reaction force vector and a dislocating force component results in glenohumeral instability. The glenohumeral stability constraint requires that the joint reaction force has a piercing point onto the glenoid surface at all times. In cases where this condition is not met, the model calculates the additionally required muscle forces to redirect the resultant vector onto the glenoid rim (van der Helm, 1994).

4.2.4 Model input

The average position for simulations was derived from patients with massive cuff tears (De Groot et al., 2006, Steenbrink et al., 2006). Because of inaccuracies in positioning and morphological variances the recorded average plane of elevation (Ry) was: 79° (SD 11°)), arm elevation (Rx) was: 46° (SD 10.7), and external rotation (Ry') was: 31° (SD 18.9°) with the elbow in 90° flexion (Fig. 4.1) according to the definitions of the International Society of Biomechanics for the shoulder in the local coordinate system of the thorax (Wu et al., 2005). The variances of observed arm positions in each of the three humeral angles σ_y^2 ($122^{\circ \cdot \circ}$), $\sigma_x^2(114^{\circ \cdot \circ})$ and $\sigma_y^2(358^{\circ \cdot \circ})$ were used to estimate variance (or sensitivity) of the calculated muscle forces, σ_{fi}^2. Because the weight of the arm was counterbalanced in the experiments gravity working on the arm in the model was set to zero. An external force of 25 Newton (average patients' ability, Steenbrink et al. 2006) was applied to the olecranon and equaled a glenohumeral elevation moment of 7.3Nm.

4.2.5 Simulated cuff pathologies

Cuff tears were simulated by canceling force production of the "torn" muscle(s), by setting fraction coefficient c_i to zero (Eq. 4.2). In the common order of progressive rotator cuff tears the following cuff tears were simulated: 1. *supraspinatus*; 2. *supraspinatus and infraspinatus*; 3. *supraspinatus, infraspinatus and the teres minor*; it appeared that teres minor elimination did not result in a successful simulation for the constrained condition. Therefore the teres minor was preserved in further simulations; 4. *supraspinatus, infraspinatus and subscapularis*; 5. *supraspinatus, infraspinatus, subscapularis, and biceps longum*. Cuff tears were simulated without and with the constraint of glenohumeral stability.

4.2.6 Data analysis

Kinematic results of the simulations were presented as potential moment vectors (*PMV*), i.e. the moment resulting from a 1 Newton muscle force, and expressed in the global coordinate system (Veeger and van der Helm, 2007). The potential moment vector of each muscle was obtained by averaging the *PMV*'s of the representing muscle elements (Fig. 4.2). Some muscle may have muscle elements with antagonist function, e.g. the subscapularis consists of 11 independent elements of which 9 elements have an *ab*duction *PMV*-component and 2 elements have an adducting *PMV*-component.

For the five simulations of rotator cuff tears, in combination with unconstrained and constrained glenohumeral stability, the magnitude of muscle forces, i.e. total force of representing muscle elements, that exerted a moment around the glenohumeral joint were determined and subsequently compared. The additional muscle activity required for glenohumeral joint stability was defined as the differences between forces estimated in the unconstrained and constrained conditions. Sensitivity of the calculated muscle forces, expressed by their variance, was estimated by the inner product of the variance in observed arm positions $[\sigma_y^2, \sigma_x^2, \sigma_{y'}^2]$ and the squared (numerical) partial derivatives of estimated muscle forces for each of three glenohumeral joint angles, Eq. 4.3.

$$\sigma_{F_i}^2 = \begin{bmatrix} \sigma_y^2 \\ \sigma_x^2 \\ \sigma_{y'}^2 \end{bmatrix}^T \begin{bmatrix} (\partial F_i/\partial y)^2 \\ (\partial F_i/\partial x)^2 \\ (\partial F_i/\partial y')^2 \end{bmatrix}^T \tag{4.3}$$

Where $\sigma_{F_i}^2$ is the variance in estimated muscle force for muscle i. $\sigma_y^2, \sigma_x^2, \sigma_{y'}^2$ are the observed variances in arm orientations Ry, Rx and Ry' (obtained from Steenbrink et al.,

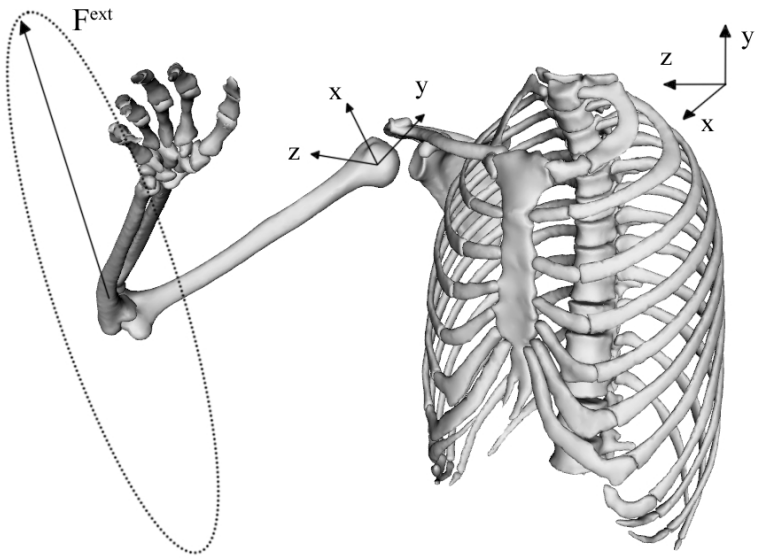

Figure 4.1: Representation of kinematic model input obtained from experimental set-up. An average arm position of 79° plane of elevation, 46° elevation and -31° axial rotation was used. An external force of 25 Newton (the average patient's ability) was applied to the olecranon, directed upward in the plane of elevation and perpendicular to the longitudinal axis of the humerus (y).

2006). $(\partial F_i/\partial y)^2, (\partial F_i/\partial x)^2, (\partial F_i/\partial y')^2$ are the partial derivatives of the muscle forces for glenohumeral orientations Ry, Rx and Ry'.

For the five simulations of rotator cuff tears, in combination with unconstrained and constrained glenohumeral stability, the magnitude of muscle forces, i.e. total force of representing muscle elements, that exerted a moment around the glenohumeral joint were determined and subsequently compared. The additional muscle activity required for glenohumeral joint stability was defined as the differences between forces estimated in the unconstrained and constrained conditions. Sensitivity of the calculated muscle forces, expressed by their variance, was estimated by the inner product of the variance in observed arm positions $[\sigma_y^2, \sigma_x^2, \sigma_{y'}^2]$ and the squared (numerical) partial derivatives of estimated muscle forces for each of three glenohumeral joint angles, Eq. 4.3. For every simulation the effort, quantified by the criterion value J, was compared with the effort in the normal condition.

Table 4.1: Function overview of muscles crossing the glenohumeral joint as measured in the simulated arm position (Figure 4.1).

Muscle	Abduction	Adduction	Retroflexion	Forward flexion	Endorotatie	Exorotatie
DE	+			+		+
SS	+		+			+
IS	+			+		+
SSc	+		+		+	
Tmi		+	+			+
BL	+			+	+	
LD		+	+		+	
PM		+	+		+	
TM		+	+		+	

4.3 Results

The *PMV*'s of muscles are constant for the simulated arm position (Fig. 4.2). The required moment vector of the external force around the glenohumeral joint is located outside the axes of the figure at [X = -20cm, Y = 11cm, Z = -17cm]. The deltoids, supraspinatus, infraspinatus, subscapularis and biceps longum include an abducting component and are primarily appropriate for the simulated *ab*duction/forward flexion task (Fig 4.3). The teres minor, pectoralis major, latissimus dorsi and teres major include an antagonistic *ad*duction moment which counteracts the force task (Table 4.1).

4.3.1 Supraspinatus tear

Unconstrained stability: Deltoid force and subscapularis *ab*ductor force increased 14% and 61% and the reaction force piercing point rotated in posterior-superior direction. The glenohumeral joint was stable. The predicted muscle forces were sensitive for arm position, as indicated by the standard deviation of the forces, but did not address other muscles than currently active (Fig.4.3A). The muscular effort, i.e. costs function J (Equation 4.1), increased 8% with respect to the normal condition (Fig. 4.4). For moment equilibrium also the endo-/exorotation moments of principal moment actuators/generators need to be compensated. The glenohumeral contact force intersects the glenoid surface, indicating that glenohumeral stability is preserved.

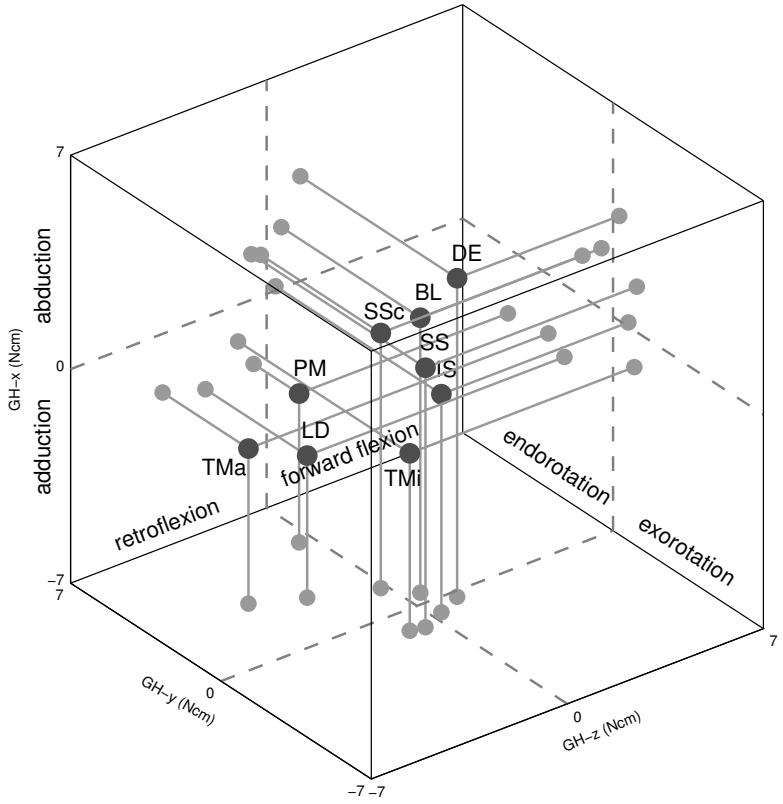

Figure 4.2: Potential Moment Vector plot, obtained by model simulation for the experimental arm position; the projections on the three axes of rotation indicate the muscles' potential contribution for the represented directions of movement. Muscles with potential contributions around the glenohumeral joint which were found to be active in our simulations are the deltoids (DE), supraspinatus (SS), infraspinatus (IS), subscapularis (SSc), teres minor (TMi), biceps longum (BL), pectoralis major (PM) and the latissimus dorsi (LD). The teres major (TMa) is presented for reference with patient observations (Steenbrink et al., 2006).

4.3.2 Supraspinatus and infraspinatus tear

Unconstrained stability: Deltoid force increased 35%; the subscapularis force decreased because its endorotation moment could not be compensated for by the infraspinatus (Fig.4.3B). A posterior-superior glenohumeral destabilizing force originated.

Constrained stability: Deltoid forces decreased, subscapularis force increased and substantial teres minor forces were required. The muscular effort, without and with stability constraint, increased 28% and 43% respectively (Fig.4.4).

4.3.3 Supraspinatus, infraspinatus and teres minor tear

Unconstrained stability: The teres minor was not active in combination with supraspinatus and infraspinatus and the model converged to the latter solution.

Constrained stability: The model did not converge to a solution. This indicates that glenohumeral integrity is not provided by the remaining muscles. The stabilizing action of the teres minor seems unique and cannot be compensated for. This tear conditions was not illustrated.

4.3.4 Supraspinatus, infraspinatus and subscapularis tear

Unconstrained stability: Deltoid and biceps longum forces increased and introduced posterior-superior glenohumeral instability (Fig.4.3C). Constrained stability: Further increase of biceps longum forces in combination with substantial teres minor forces and position sensitive introduction of latissimus dorsi and pectoralis major forces were required. The muscular effort without and with stability constraint increased 37% and 111% respectively (Fig. 4.4).

4.3.5 Supraspinatus, infraspinatus, subscapularis and biceps longum tear

Unconstrained stability: The most extended cuff tear resulted in the largest deltoid forces in combination with teres minor forces and introduced maximum posterior-superior glenohumeral instability (Fig.4.3D).

Constrained stability: Additional teres minor and deltoid forces in combination with pectoralis major and latissimus dorsi forces were required. The latter muscles generated a large adduction moment. The muscular effort in this simulation increased 46% for the unconstrained glenohumeral joint and 163% when glenohumeral stability was preserved (Fig. 4.4).

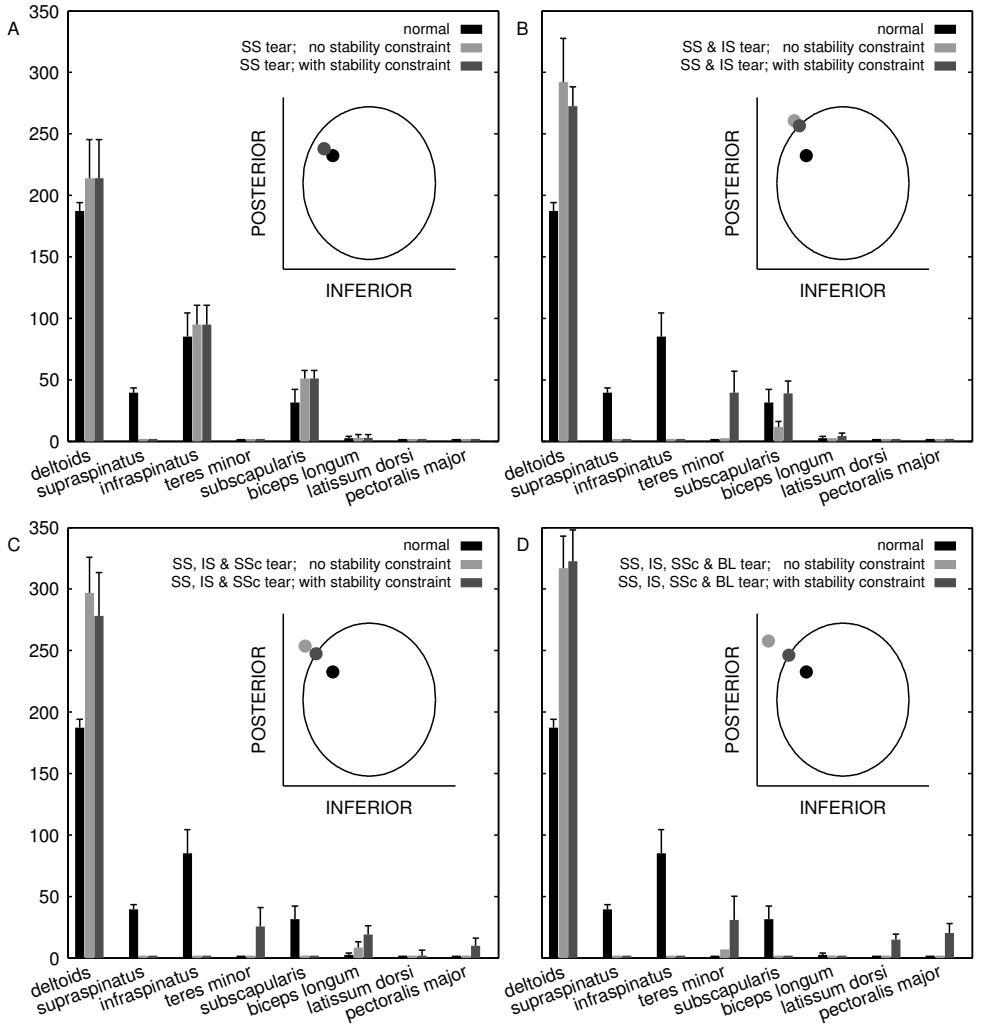

Figure 4.3: By the DSEM predicted muscle forces (Newton), and subsequent application point of the glenohumeral joint reaction force on the glenoid surface (inlay), as a result of the simulated conditions. Rotator cuff tears are respectively supraspinatus tear (A), supraspinatus and infraspinatus tear (B), supraspinatus, infraspinatus and subscapularis tear (C), supraspinatus, infraspinatus and subscapularis and biceps longum tear (D).

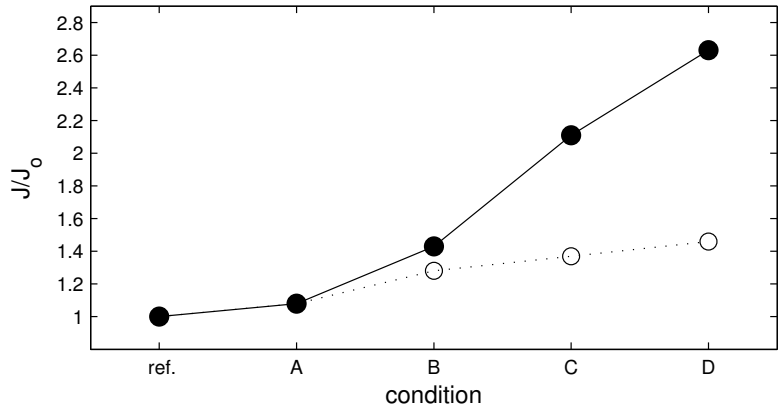

Figure 4.4: Relative increases of optimization criterion J (i.e muscular effort) for glenohumeral moment and additional glenohumeral stability with increasing tear. (For description of conditions see Fig. 4.3)

4.4 Discussion

The objective of this study was to analyze the muscular compensation for rotator cuff tears of varying magnitude and to identify additional muscle forces required for glenohumeral stability.

4.4.1 *Ab*duction compensation

Extending cuff tears result in increased deltoid muscle forces and confirms previous simulation (Magermans et al., 2004), cadaver (Apreleva et al., 2000, Hsu et al., 1997, Parsons et al., 2002) and experimental nerve blocking studies (McCully et al., 2007). The consequence of increased deltoid force is the posterior-superior shift of the reaction force vector piercing point. An isolated supraspinatus tearing does not necessarily result in an unstable glenohumeral joint, which may mechanically explain a-symptomatic cuff tears (Kelly et al., 2005, Yamaguchi et al., 2001).

In accordance with cadaver studies, the extent of a rotator cuff tear beyond the supraspinatus into the infraspinatus tendon induces glenohumeral instability (Apreleva et al., 2000, Hsu et al., 1997, Parsons et al., 2002) and may explain the relationship between fatty degeneration of the combined infraspinatus and teres minor and proximal migration in rheumatoid arthritis (van de Sande et al, 2007). We conclude that the deltoid muscle is an efficient *ab*ductor mus-

cle. For the simulated arm position in the DSEM, the upper 9 elements of the subscapularis (11 in total) are abducting synergists. In case of minor tears, the subscapularis compensated supraspinatus losses in combination with the infraspinatus.

Biceps longum *ab*duction moment contribution on the condition of glenohumeral stability (Warner and McMahon, 1995) and specifically in massive cuff tears (Kido et al., 2000; Beall at al., 2003) was indeed observed in our simulation (condition C). From a mechanical point of view, tenotomy of the long head of the biceps, used to reduce symptoms of pain and inflammation in the follow up of patients with cuff tears (Boileau et al., 2007, Walch et al., 2005), induces increased co-contraction of muscles with large *ad*ductor components (pectoralis major/latissimus dorsi) and thus a serious additional muscular effort (Fig. 4.4D).

4.4.2 Glenohumeral stability

Deltoid forces efficiently substituted lost cuff *ab*duction moments at the cost of glenohumeral stability. This was evidently illustrated by deltoid reduction upon required glenohumeral stability in combination with additional abducting cuff muscle forces (subscapularis/biceps longum) and the consequent increase of the optimization criterion J, Fig. 4.4. Remarkably, teres minor co-contraction forces are vital for glenohumeral stabilization if the infraspinatus ceased function. Because of its relative small moment arm and vertically directed line of action the teres minor seems extremely useful to compensate the extra-glenoidal force component and stabilize the glenohumeral joint, with minimal interference with the intended elevation moment. Recent clinical observations also claim the importance of the teres minor for glenohumeral stability (Costouros et al., 2007, Simovitch et al., 2007).

If all *ab*ductor synergists were set to zero (condition 5, Fig. 4.3D), the deltoid muscle was the only muscle left to generate the required *ab*duction moment. Muscles with large *ad*ductor components (pectoralis major/latissimus dorsi) were required for glenohumeral stability. This 'expensive' co-contraction seems to be the only solution left to generate net *ab*duction moment. This is in line with publications by Hinterwimmer et al. (2003) and Graichen et al. (2005) and our own experimental findings (Steenbrink et al., 2006) where *ad*ductor activation of latissimus dorsi, pectoralis major and teres major was observed in patients with massive cuff injury. Experimentally observed teres major co-activation (de Groot et al., 2006; Steenbrink et al., 2006), was however absent in this simulation study. This may be the result of subject specific anthropometry on the observed combination of muscle activation. *ad*ductor muscle co-activation is a possible cause of observed limitation in maximal arm elevation in patients with cuff injury (Iannotti et al., 2006, Jost et al., 2000).

4.4.3 Limitations of this study

The outcome of this study is only valid for the specified position and only reflects mechanical considerations. Other somatic symptoms of cuff pathology, such as pain, were not included in this study. Massive cuff tears may result in kinematic changes of scapulo-thoracic and scapulo-humeral positions, as illustrated by a supraspinate nerve block experiment (McCully et al., 2006). The kinematic changes will affect the *PMV*'s of muscles and thus the force and moment balance. We partially overcame this shortcoming by approximation of the sensitivity of muscle forces for arm position. The shape of the glenoid, its relative position and the presence of a labrum (absent in the DSEM) may slightly affect the absolute magnitude of muscle forces presented but not the relative muscle forces.

4.4.4 Functional/clinical implications

Cuff injuries of the supraspinatus and infraspinatus required adduction forces of the teres minor whereas tears extending these muscles required forces by larger *ad*ductor muscles, i.e. pectoralis major and latissimus dorsi. On conditional teres minor preservation, patients with massive cuff injuries are theoretically able to generate *ab*duction forces with sufficient glenohumeral stability. This may explain a symptomatic rotator cuff tears (Kelly et al., 2005, Yamaguchi et al., 2001). Symptomatic rotator cuff tears with proximal migration are common and indicate that patients fail to fully compensate the lost stabilizing forces. The cause of this failure is unknown, but might involve unrecognized teres minor failure or disturbed proprioceptive or nociceptive sensory feedback, as e.g. subacromial pain suppression increased maximal arm force and arm mobility (Ben Yishay et al., 1994, de Groot et al. 2006) and restored activation patterns (Steenbrink et al., 2006). Simulation indicated the teres minor to be the solely indispensable cuff *ad*ductor in case of a complete infraspinatus deficiency. Post-hoc simulation of artificial (mathematical) elimination of teres minor moments around all three axes (Fig. 4.2) with maintenance of its force contribution resulted in a 121% increase of teres minor force. We concluded that teres minor is primarily required for glenohumeral stability and not humeral endorotation moment compensation.

Pathological *ad*ductor co-contraction during arm elevation load is the general mechanical finding of this study. This coincides with our experimental observations in patients with massive cuff tears (de Groot et al. 2006, Steenbrink et al. 2006) and can therefore be regarded as an indication for cuff disease. In order to understand subacromial pathologies the challenge is to develop an experiment which addresses the causal relation between muscle activity

and glenohumeral (in)stability. Experimental research should focus on identifying the causal relations between compensating muscle activity by loading the arm with various moments and constant forces in patients with cuff tears.

4.5 Conclusion

An isolated tear of the supraspinatus does not necessarily lead to glenohumeral instability. For massive cuff tears beyond the supraspinatus, instability became a prominent factor. Moments efficient deltoids introduced a large destabilizing force component and alternative *ab*ductor muscles (i.e. subscapularis and biceps longum) required 'costly' co-contraction. The teres minor appeared to be of vital importance in glenohumeral stability because of its stabilizing force vector.

Chapter 5

Teres major muscle activation relates to clinical outcome in tendon transfer surgery

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Abstract

In irreparable rotator cuff tears a teres major tendon transfer to the insertion of the supraspinatus reverses its *ad*duction moment arm to *ab*duction which is supposed to be an adequate salvage procedure. Analysis of muscle function to find biomechanical ground of such success is scarce.

We compared pre- and postoperative clinical outcome of a teres major transfer, i.e. Range of Motion, pain, Constant Shoulder scores and arm force. Teres major activation was evaluated in fourteen patients suffering irreparable cuff tears using *activation ratios* to describe the desired 'in-phase' and undesired 'out-of-phase' contribution to the external arm moment. Additionally, we analyzed activation of the latissimus dorsi and the medial part of the deltoids. The *activation ratios* were compared to controls and teres major *activation ratios* were related to clinical outcome.

A teres major tendon transfer improved arm function. Preoperatively, we observed 'out-of-phase' *ab*duction activation of the teres major and latissimus dorsi. After transfer patients activated their teres major according to its new anatomical position. 'Out-of-phase' latissimus dorsi *ab*duction activation persisted. The clinical improvements coincided with changes in *activation ratio* of the teres major.

'Out-of-phase' teres major *ad*ductor activation is associated with compromised arm function in patients with irreparable cuff tears. After transfer, the teres major is activated in correspondence with its new anatomical function, which was supportive for the improved arm function.

5.1 Introduction

Patients with irreparable rotator cuff tears are restricted in their daily activities due to limitations in arm Range of Motion (*RoM*) and pain (Iannotti et al., 1996; Jost et al., 2000). Conservative treatment often evolves into progressive cuff degeneration, proximal migration of the humeral head or sweeping cuff tear arthropathy (Hawkinds and Dunlop, 1995; Levy et al., 2008; Zingg et al., 2007). Restoration of the torn and degenerated cuff muscle(s) frequently results in re-tears and unsatisfying functional improvements (Birmingham and Neviaser, 2008; Elhassan et al., 2008). Alternatively, muscle-tendon transfers have been proposed as a salvage procedure to restore arm function with moderate to good results (Aoki et al, 1996; Boileau et al., 2007; Celli et al., 2005; Celli et al., 1998; Codsi et al., 2007; Gerber et al., 1988; Gerber et al., 2000; Iannotti et al., 2006; Irlenbusch et al., 2008b; Miniaci and MacLeod, 1999; Warner and Parsons, 2001).

High quality randomized controlled blinded clinical trials, investigating the effect of tendon transfers are not feasible. The available clinical studies are generally descriptive, preferably using large cohorts, because of individual variation in functional outcome (Gerber et al., 2006). The alternative is to find determinants of functional outcome after tendon transfer surgery, for which biomechanical modeling and experimental testing is required. Biomechanical model simulations (Magermans et al., 2004a; Magermans et al., 2004b) and anatomical studies (Wang et al., 1999; Buijze et al., 2007) predicted a teres major tendon transfer to the insertion of the supraspinatus on the greater tubercle of the humeral head to mechanically maximize functional task performance. Anatomically, the teres major is an *ad*ductor and internal rotator of the arm. After transfer, the teres major is expected to contribute to arm elevation and exorotation (Celli et al., 1998). Although moderate to good functional results are reported for such reconstructive tendon transfer treatment (Celli et al., 2005), analysis of muscle function (changes), essential to comprehend its clinical successes, is not available.

We proposed that, as result of a rotator cuff tear the balance between glenohumeral stability and mobility of the shoulder is disturbed (de Groot et al., 2006; Steenbrink et al., 2006; Steenbrink et al., 2009a). The deltoids are believed to compensate lost rotator cuff elevation moments (McCully et al., 2007; Steenbrink et al., 2009a). The subsequent increase of cranially directed forces on the humeral head affect glenohumeral joint stability (Steenbrink et al., 2009a) and result in proximal migration (Graichen et al., 2005; van de Sande and Rozing, 2006) causing (painful) compression of the subacromial tissues. Muscles inserting on the humerus and generating downward directed forces, i.e. the teres major and latissimus

dorsi, have been demonstrated to co-contract in order to compensate proximal migration of the humeral head (de Groot et al. 2006, Steenbrink et al., 2006, Steenbrink et al. 2009a). Divergent muscle activation clearly plays a role in the functional impairments observed in patients with cuff tears and is assumed to be an important variable affecting treatment outcome (Iannotti et al., 2006; Codsi et al., 2007; Irlenbusch et al., 2008a).

In addition to clinical outcome, we therefore assessed muscle function of the teres major, latissimus dorsi and the deltoids (medial part) before and after a teres major tendon transfer. We postulate that preoperative 'out-of-phase' *ad*ductor muscle activation of teres major and/or latissimus dorsi coincides with functional impairment. Relocating the teres major insertion should result in a post-surgical teres major activation during arm *ab*duction (elevation) forces instead of the typical *ad*duction component. 'Out-of-phase' latissimus dorsi activation is expected to reduce, due to the recovered stabilizing forces of the transferred teres major, while deltoid activation is not expected to change (Levy et al., 2008). After a teres major tendon transfer, optimized muscle activation is expected to result in improved clinical outcome.

5.2 Methods

Fourteen patients (10 male) with an average age of 61 years (range, 53-69) were included in the study between June 2005 and June 2007. All patients had MRI diagnosed rotator cuff tears larger than 4 cm with retraction and Goutallier grade 3-4 fatty degeneration excluding primary cuff repair (Goutallier et al., 1994). MRI patient characteristics are summarized in Table 5.1. All patients were treated with a teres major tendon transfer to the insertion of the supraspinatus and assessed within one month before and nine months (range, 7-11) after surgery. Ten healthy controls (5 male) with an average age of 25 years (range, 22-28) volunteered for norm electromyography (*EMG*) data collection.

The study was approved by the medical ethics committee of the Leiden University Medical Center and all participants gave written informed consent.

5.2.1 Surgical technique

Patients were positioned in a lateral decubital position. A curved incision was made at the posterior part of the axilla towards the humerus. After confirmation of an irreparable tear, the teres major was separated from the latissimus dorsi insertion and detached from the humerus. A second incision was made in the Langerhans lines at the posterocranial part of the humerus.

Table 5.1: Radiological/MRI characteristics of the patients. SSp: supraspinatus; IS: infraspinatus; SSc: subscapularis; TMn: teres minor; BL: biceps longum; AC: acromionclavicular; +: affected; part: partially affected; -: not affected.

Patient	Side	SSp	IS	SSc	TMn	BL	Proximal migration humeral head	Retracted SSp	AC joint arthosis
1	L	+	-	-	-	-	+	+	+
2	R	+	part	-	-	-	+	+	-
3	L	+	-	part	-	-	-	+	-
4	L	+	+	-	-	-	+	+	-
5	R	+	+	+	part	+	+	+	+
6	R	+	part	-	-	-	+	+	-
7	R	+	-	+	+	+	+	+	+
8	R	+	+	-	-	-	+	+	+
9	R	+	part	-	-	+	+	+	-
10	R	+	+	-	-	+	+	+	+
11	R	+	part	-	-	-	+	+	-
12	R	+	part	-	-	-	+	+	-
13	L	+	+	-	-	-	+	+	+
14	R	+	+	+	-	+	+	+	+

The deltoid muscle was split and the teres major tendon was transferred underneath the posterior part of the deltoids and attached using two RC Mitek Anchors (DePuy Mitek inc., Warsaw, IN, USA) on the cranial supraspinatus footprint area. Postoperatively, a shoulder brace prevented internal rotations and after 6 weeks physical therapy was started.

5.2.2 Electromyography

During an isometric force task, bi-polar surface *EMG* was recorded for the teres major, latissimus dorsi and the deltoids (silver electrodes, inter-electrode distance 21mm, bandwidth 20Hz-500Hz). For the control group a DelSys system was used (Bagnoli-16, Boston, MA, USA, inter-electrode distance 10 mm, bandwidth 20Hz-450Hz). Electrode placement was similar to de Groot et al. (2004) and Meskers et al. (2004). After transfer, the teres major was palpated and the electrode placed on the middle of the muscle belly. Subjects were seated with their injured arm in a splint with the elbow in 90° flexion. The splint was attached to a 6DOF-force transducer (AMTI-300, Advanced Mechanical Technology, Inc., Watertown, MA, USA). The construction only allowed force exertions perpendicular to the longitudinal axis of the humerus (Fig. 5.1). The humeral plane of elevation was about 60° relative to the sagittal plane, the humerus was elevated about 45° and externally rotated with the lower

arm about 30° relative to the horizontal plane. The force magnitude was set at the highest level at which the subject could comfortably fulfill an isometric force task in seven upwards directions (215°, 230°, 245°, 0°, 15°, 30°, 45°) and seven downwards directions (135°, 150°, 165°, 180°, 195°, 210°, 225°). The force task was controlled for direction and magnitude by visual feedback on a computer screen located in front of the subject. Sample rate of analog filtered *EMG* and force data was 1000Hz.

Muscle moment arms, represented in Fig. 5.2, were obtained from inverse kinematic model simulation (van der Helm, 1994) with experimental arm position as kinematic input (Steenbrink et al., 2009a). Muscle activation was qualified according to their moment arms, i.e. activation was either 'in-phase' or 'out-of-phase' with respect to its moment arm. For the teres major and latissimus dorsi, 'in-phase' activation was defined as activation during downwards arm force directions (*ad*duction) and 'out-of-phase' activation was defined as activation during the upwards arm force directions (*ab*duction). The teres major becomes an *ab*ductor after transfer (Fig. 5.2), and 'in-phase' activation then occurs while generating upwards arm forces. Deltoid activation is 'in-phase' with upwards arm forces. *EMG* at rest was subtracted from the *EMG*'s during the force tasks. Two average *EMG* levels were determined for every muscle, i.e. one over the seven upwards and one over the seven downwards arm force exertion. Muscle *activation ratios* were calculated according Eq. 5.1:

$$AR_{muscle} = \frac{A^{IP}_{muscle} - A^{OP}_{muscle}}{A^{IP}_{muscle} + A^{OP}_{muscle}} \qquad [-1 \leq AR_{muscle} \leq 1] \tag{5.1}$$

where AR_{muscle} is the relative activation or *activation ratio* of *muscle* teres major (TMj), latissimus dorsi (LD) or deltoid (DE); AR = 1 indicates optimal 'in-phase' muscle activation and AR = -1 indicates worst 'out-of-phase' muscle activation. For AR = 0, activation is equal for up-and downwards arm force exertion; A^{IP} is the 'in-phase' muscle activity, contributing positively to the external moment according to the muscle moment arm; A^{OP} is the 'out-of-phase' muscle activity, contributing negatively to the external moment according to the muscle moment arm.

5.2.3 Clinical assessment

Maximum arm Range of Motion (*RoM*) was determined relative to the thorax (Meskers et al., 1998) for *ab*duction (RoM_{AB}), forward flexion (RoM_{FF}) and retroflexion (RoM_{RF}). External

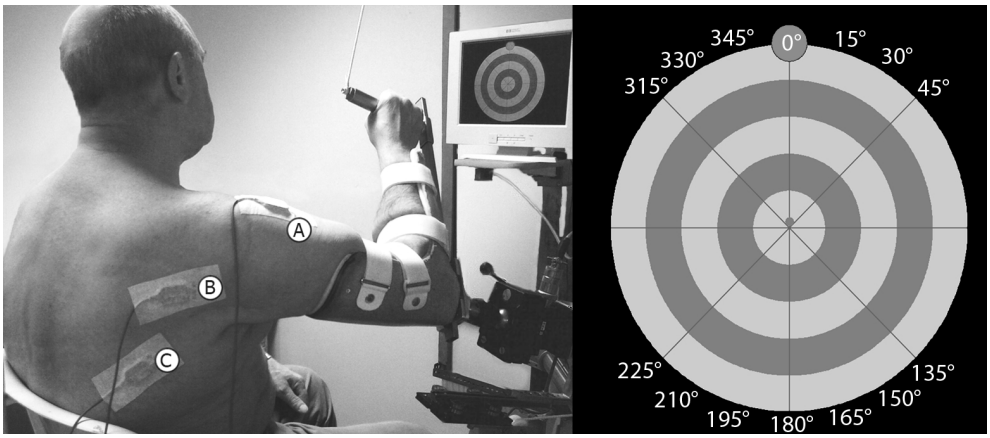

Figure 5.1: Experimental set-up; the patient is seated in front of a screen with his injured arm in a splint, which is connected to a force transducers. Surface *EMG* electrodes are positioned on the medial part of the deltoids (A), the teres major (B), and the latissimus dorsi (C). The patient exerts arm forces controlled by visual feedback of an arm force driven small circled cursor into a bigger circled target area. The target area is randomly located at seven upwards directions (215°, 230°, 245°, 0°, 15°, 30°, 45°) and seven downwards directions (135°, 150°, 165°, 180°, 195°, 210°, 225°), demanding respectively *ab*-and *ad*duction arm moment exertion.

rotation was measured at 0° humerus *ab*duction (RoM_{EXT}). All values were measured with an electromagnetic tracking device (Flock of Birds, Ascension Technology Corp, Burlington, VT, USA). External humerus rotation was defined 0° in the position at which the hand pointed forward and external rotation had a positive sign.

Pain was assessed using a 100mm Visual Analog Scale (*VAS*) both at rest (VAS_{rest}) and during Activities of Daily Living (VAS_{ADL}) (0: no pain; 100: worst pain ever imaginable); arm function was assessed using the *Constant Shoulder Score* (Constant and Murley, 1987).

F_{max} comprehended the highest determined force magnitude, recorded by the force transducer, which the subject could exert in all directions. Fext was the maximal arm force in external rotation, recorded by the force transducer in a 'locked' axial rotation set-up.

5.2.4 Statistics

Differences between *activation ratios* of patients and controls were statistically tested using the Student's t-test. Pre-and postoperative *AR*, *RoM*, *VAS*, *Constant Scores* and F_{max} were

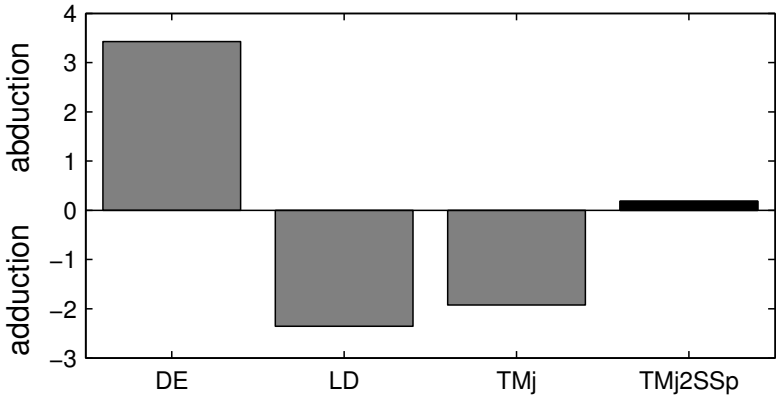

Figure 5.2: Representation of muscle moment arms obtained from inverse kinematic model simulation (van der Helm, 1994) in the experimental arm position (Steenbrink et al., 2009a). The columns represent the moment arms about the local x-axis, which is the *ab*duction/*ad*duction axis, for DE: deltoid (medial part); LD: latissimus dorsi; TMj: teres major; and TMj2SSp: teres major after transfer to supraspinatus insertion on the humeral head.

compared using the paired samples t-test. Linear regression was applied for each of the clinical variables as a function of AR_{TMj} and tested for significant slope coefficients. All tests were performed using SPSS 16.0 (SPSS Inc, Chicago, IL) with an alpha of 5%.

5.3 Results

Average duration of the teres major tendon transfer surgery procedure was 81 minutes (range 60-135 minutes). No complications were reported during surgery, nor postoperatively, nor during the protocolized physical therapy sessions. In concordance with other reports (Codsi et al., 2007; Pearle et al., 2006), no difficulties were encountered in isolating the teres major from the latissimus dorsi for transfer underneath posterior part of the deltoids. Sufficient teres major length allowed its transfer onto the greater tubercle of the humeral head (Pearle et al, 2006; Buijze et al., 2007). Thickness of the muscle-tendon unit did not compromise the subscapular nerve, which could risk a traction injury due to transfer after muscle transfer (Buijze et al., 2007).

Table 5.2: Pre-and post teres major tendon transfer clinical data, significant differences are indicated (*). *RoM*: range of motion; *AB*: *ab*duction; FF: forward flexion; RF: retroflexion; EXT: external rotation; *VAS*: visual analogue score for pain at rest and during activities of daily living (ADL); $Fmax$: maximal arm force in experimental setup; $Fext$: maximal external rotation force.

	RoM_{AB} (°)		RoM_{FF} (°)		RoM_{RF} (°)		RoM_{EX} (°)		VAS_{rest} (mm)		VAS_{ADL} (mm)		Constant Score		Fmax (N)		F_{EXT} (N)	
Patient	*pre*	*post*	*pre*	*post*	*pre*	*post*	*pre*	*post*	*pre*	*post*	*pre*	*post*	*pre*	*post*	*pre*	*post*	*pre*	*post*
1	91	110	77	98	31	34	4	15	81	11	53	0	22	31	10	20	2	20
2	104	143	97	130	35	39	12	45	72	5	45	4	47	79	10	30	8	7
3	90	117	76	85	22	21	6	10	53	51	76	34	24	54	20	20	10	13
4	20	18	19	28	41	38	7	10	63	12	6	0	35	42	10	20	4	9
5	128	140	120	124	40	30	-1	34	7	4	34	32	28	65	5	10	7	10
6	150	153	145	151	38	46	7	-29	38	0	74	0	58	77	10	10	5	10
7	44	70	45	78	45	37	21	25	62	0	32	0	26	64	10	20	8	10
8	59	134	50	120	39	49	14	34	48	8	4	0	19	42	10	20	5	10
9	93	103	90	109	40	46	17	37	74	0	66	0	35	79	10	20	15	20
10	163	170	144	167	36	38	30	12	45	0	23	0	78	78	50	50	7	8
11	96	112	109	117	53	50	13	36	71	34	82	33	23	49	20	40	13	13
12	52	50	44	67	17	23	-29	6	79	32	67	54	18	30	15	15	6	5
13	124	116	79	126	36	42	3	21	71	24	77	13	49	67	30	50	15	26
14	139	148	105	142	31	63	5	62	96	56	9	5	20	79	20	30	30	28
Mean	86	110	97	113	36	40	8	23	66	23	59	19	35	60	16	25	9	14
SD	38	37	42	42	9	11	13	22	15	21	21	22	18	18	12	13	6	7
P	0.012*		0.000*		0.190		0.033*		0.000*		0.000*		0.000*		0.002*		0.017*	

5.3.1 Activation Ratios

In the control group we observed positive *activation ratios* for all recorded muscles, AR_{TMj}, AR_{LD} and AR_{DE} (Fig. 5.3, Table 5.2). Pre-operatively in patients, we observed lower *activation ratios* for the deltoid muscle, AR_{DE}, compared to controls (95% confidence interval of the difference (CI_d): [0.11, 0.37], p = 0.01). Compared to controls, the *activation ratios* for the teres major and the latissimus dorsi, AR_{TMj} and AR_{LD}, were significantly lower compared to controls (95% CI_d: [0.51, 0.93], p = 0.00; 95% CI_d LD [0.50, 0.84], p = 0.00). After teres major tendon transfer the post-surgical AR_{TMj} changed significantly (95% CI_d [0.14, 0.40], p = 0.01). The positive *activation ratio* of the teres major, AR_{TMj}, corresponded with the muscle's new anatomical position inserting on the greater tubercle of the humeral head, contributing to the upwards directed arm forces. Postoperative *activation ration* of the latissimus dorsi, AR_{LD}, did not change compared to preoperative values (95% CI_d [-0.24, 0.06], p = 0.22), while postoperative *activation ratios* of the deltoid muscle, AR_{DE}, increased significantly (95% CI_d: [0.04, 0.26], p = 0.01).

Table 5.3: Mean muscle *activation ratios* (SD). Significant differences between controls and patients prior to teres major transfer are marked with a (*). Significant differences between patients prior to and after teres major transfer are marked with a (**).

	Control (n=10)	**Patient (n=14)**	
Muscle		*pre surgery*	*post surgery*
Teres major	0.64 (0.24)	-0.08 (0.6)*	0.28 (0.18)**
Latissimus dorsi	0.65 (0.19)	-0.01 (0.2)*	0.07 (0.27)
Deltoid	0.87 (0.07)	0.63 (0.2)*	0.78 (0.16)**

5.3.2 Clinical results

On average (n=14) patients improved significantly on all clinical outcome variables except for RoM_{RF} (Table 5.3). The mean postoperative *RoM* increased for *ab*duction (24°, SD 21°), forward flexion (16°, SD 10°) and external rotation (15°, SD 10°). Patients reported decreased pain at rest (-43mm, SD 22mm) and during *ADL* (-40mm, SD 25mm), and a functional increase on the *Constant Shoulder Score* of 25 SD 16 points. F_{max} and F_{ext} increased 9N (SD 7N), and 4N (SD 5N), respectively.

5.3.3 Linear regression AR_{TMj} to clinical outcome

The linear regression estimates and 95% CI's for the independent AR_{TMj} and the dependent clinical outcome variables $RoM_{AB}, RoM_{FF}, RoM_{EXT}, RoM_{RF}, VAS_{rest}, VAS_{ADL}, ConstantScore$, F_{max} and F_{EXT} are presented in Figure 5.4. The slope coefficients (β) for all parameters differed significantly from zero except for RoM_{RF} and F_{EXT}.

5.4 Discussion

This study evaluates clinical outcome and muscle function of the teres major, latissimus dorsi and the deltoids in patients with a glenohumeral cuff tear prior to and after a teres major tendon transfer to the supraspinatus footprint. The theoretical background for this analysis is the biomechanical conflict between elevation mobility and glenohumeral stability (Veeger and van der Helm 2007, Steenbrink et al 2009a) which is partly solved by the teres major transfer (de Groot et al. 2006). It is demonstrated that teres major function before surgery is pathological (de Groot et al. 2006, Steenbrink et al, 2006) and indeed contributes to the

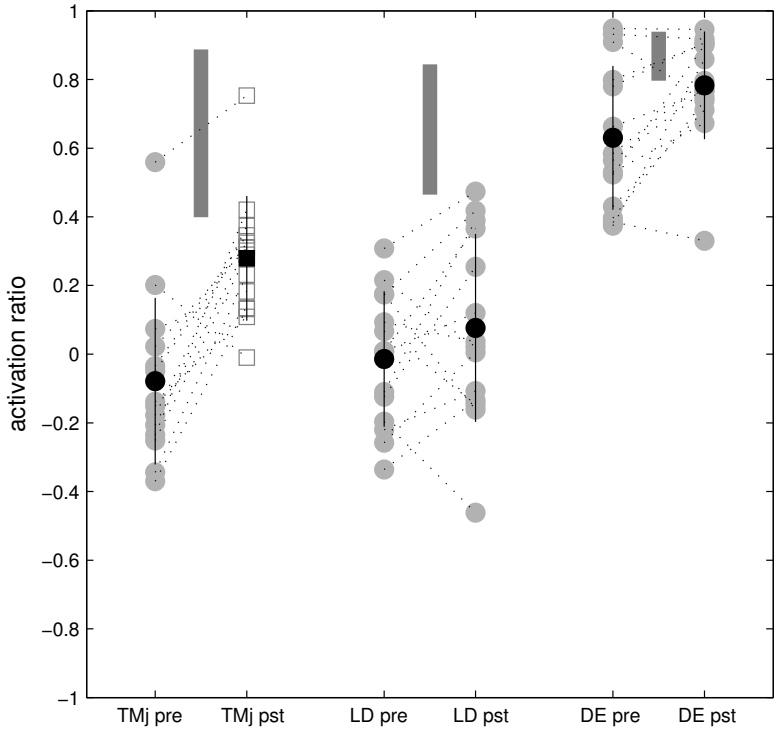

Figure 5.3: Mean *activation ratios* (±SD) of teres major (TMj), latisimus dorsi (LD) and the medial part of the deltoids (DE) for healthy subjects (95% confidence interval (vertical grey bars) and patients with cuff tears (filled circles (± -)) before (pre) and after (pst) teres major tendon transfer. Individual patient data is represented by grey filled circles and mean patients data by filled black circles. Mean patient data of the transposed teres major is represented by a filled black square and individual data by unfilled squares.

elevation moment after transfer. This study also provides evidence that teres major function before and after transfer relates to the predicted (Magermans et al., 2004a; Magermans et al., 2004b) and observed (Celli et al., 1998; Celli et al., 2005, this study) improvement of functional outcome after surgery.

The clinical results demonstrated the 'moderate' success of a teres major tendon transfer for patients with a glenohumeral cuff tear and functional improvement was comparable to those reported after a latissimus dorsi transfer (Gerber et al., 2006; Iannotti et al., 1996; Miniachi and McCloud, 1999; Warner et al., 2001). However, patients did not regain normal function, pain did not disappear completely in over 50% of the cases and results are highly variable. In order to identify significances from the large number of possible determinants (e.g. causal: habitual or traumatic; spatial: size and location of the lesions; temporal: instantaneous, chronic; secondary pathology: fatty degeneration; coordinative skills (Werner et al., 2008)), large cohort studies are required.

The biomechanical determinants of cuff lesions (Steenbrink et al., 2009a) and tendon transfers (Magermans et al., 2004a; Magermans et al., 2004b) define the borders of shoulder function which are expressed in other physiological determinants. Therefore we aimed at showing the counterproductive teres major activation before surgery and prove functional activation of the teres major after transfer. In order to quantify the mechanical contribution we applied a relative *EMG* measure, the *activation ratio*, assuming increased *EMG* contributing to non-linear muscle force increases within our isometrical measurement set-up (de Groot et al. 2004, Meskers et al. 2004). The combination of *EMG* parameters with muscle moment arms, obtained from model simulation (van der Helm, 1994), is suitable for studying muscle function (Gatti et al., 2007). This allowed us to quantify 'muscle function' prior to and after teres major tendon transfer within a repeated measures design. Changes in the mechanical muscle function should at least partly be related to functional clinical outcome parameters. Activation of the transposed muscle (Iannotti et al., 2006; Irlenbusch et al., 2008a), the teres major in our case, is a prerequisite for the presumed success of tendon transfer surgery. We demonstrated distinguished teres major activation according to its new anatomical function, resulting in a positive AR_{TMj} after surgery, indeed indicating a functional transfer. The *activation ratio* did not exceed 0.4, illustrating muscle activity both during the 'acquired' *ab*duction task but also still during the 'original' *ad*duction task. Either the muscle compensates for forces and moments other than the *ad*duction moment during the *ad*duction task (glenohumeral joint comprises 3 rotational degrees of freedom) and/or the original activation pattern is not fully reversed in the newly obtained coordination pattern.

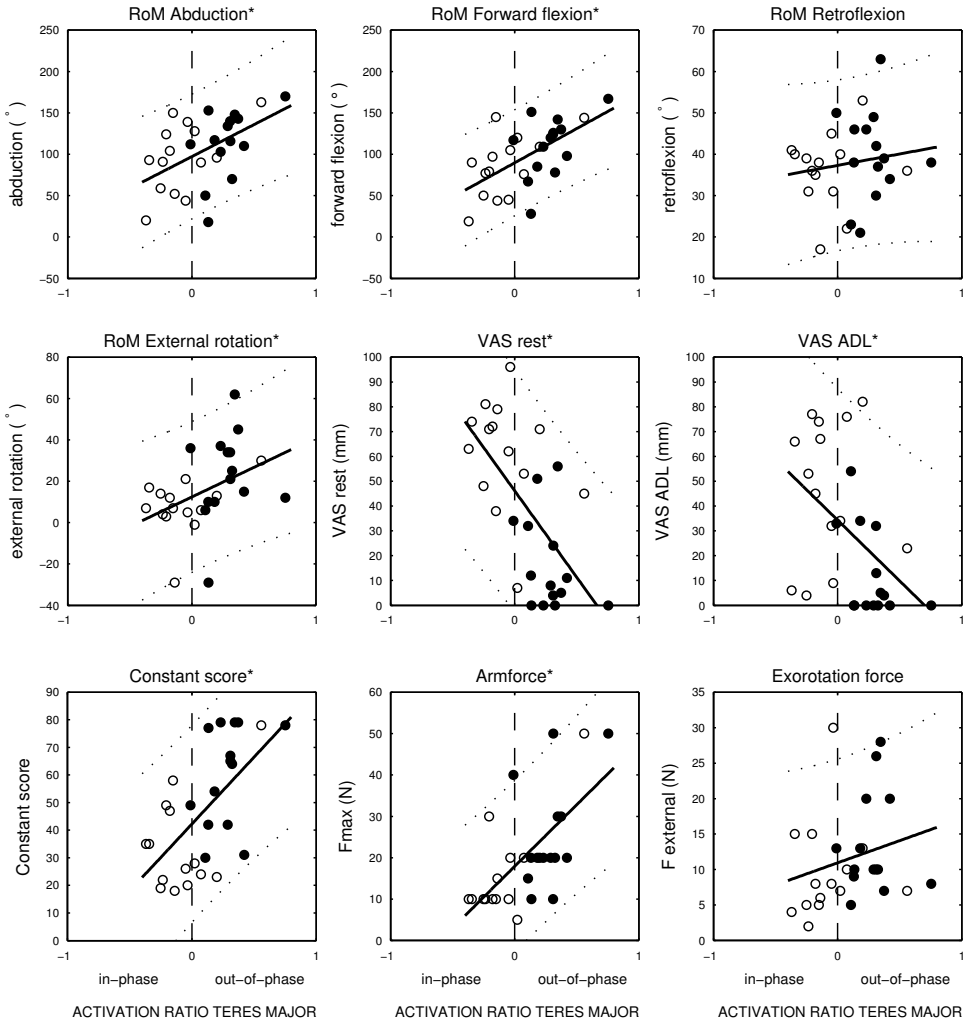

Figure 5.4: Linear regression of teres major *activation ratio* to clinical outcome variables. Preoperative measurements are marked with a open circle, and postoperative measurements are marked with a filled circle. Slope coefficients β which significantly differed from zero are marked with a * in the title of the subplot.

Although the control group was not age and gender matched, the observed differences between patients and controls were of such magnitude that they could not solely be explained by group differences. In contrast to healthy subjects, *activation ratios* of patients indicated preoperative 'out-of-phase' *ad*ductor muscle activation of the teres major and latissimus dorsi suggesting a compensation strategy for proximal migration of the humerus (Graichen et al., 2005; de Groot et al., 2006; Steenbrink et al., 2006a; Steenbrink et al., 2009a).

With regression analysis we found a significant linear relation between AR_{TMj} and most clinical outcome variables before and after surgery. Despite the small cohort of patients in this study, we demonstrated that teres major activation prior to and after surgery, at least partially, explains the variances in functional and clinical outcome. To our knowledge this study is the first to find evidence for a biomechanical relation between a surgical intervention and its clinical outcome. The effect of changed teres major function on clinical outcome after transfer supports the biomechanical hypothesis about the role of the teres major in arm mobility and glenohumeral stability in patients. Although precaution should be made when extrapolating results to dynamic conditions, preoperative 'out-of-phase' teres major activation may constrain shoulder and 'in-phase' activation of the transposed teres major appears to support functionality.

The transferred teres major contributes to arm *ab*duction and deltoid muscle forces are likely to decrease. Subsequently, the upward directed forces in the glenohumeral joint reduce and less co-contraction of the remaining *ad*ductor (latissimus dorsi) is required. The postoperative 'out-of-phase' latissimus dorsi activation indeed seems to be decreased, however not statistically significant. Despite the low *activation ratio* compared to controls, AR_{DE} in patients preoperatively displayed evident 'in-phase' muscle function. The AR_{DE} increased after the teres major tendon transfer, either through increased deltoid arm *ab*duction moment generation or decreased deltoid activity during *ad*duction. Because AR_{DE} in controls is even higher, the latter option is presumed.

As the surgery intended, we indeed found an increase of external rotation arm force. The absence of a significant relation with AR_{TMj} is not surprising as *ab*duction and *ad*duction tasks are compared to calculate *activation ratios*, and not internal and external rotation.

A possible side-effect of muscle transfers in general may be the deterioration of original function of the transposed muscle. Because of its substantial moment arm, deficits in arm retroflexion/*ad*duction were found after a latissimus dorsi tendon transfer, which manifested by early fatigue of the arm (Spear et al., 2006). This may induce functional problems in elderly when dependant on active arm *ad*duction/extension when rising from a chair or us-

ing crutches. After a teres major tendon transfer we observed no deficits in maximum arm retroflexion. Although advisable, identification of possible functional adverse effects after tendon transfer surgery was not the subject of this study.

5.5 Conclusion

In this evaluation of a teres major tendon transfer in patients with irreparable rotator cuff lesions we found functional and clinical improvements and provide evidence that the teres major is functionally activated after transfer surgery.

This study also provides evidence for the biomechanical relation between teres major function before and after surgery with the observed functional and clinical improvements. The preoperative deteriorated arm *ab*duction function was associated with pathological 'out-of-phase' *ad*ductor muscle activation of both the teres major and latissimus dorsi. This is assumed to be an attempt to accommodate for better glenohumeral stability in the cranially migrating humeral head. After surgery patients were able to activate the teres major in correspondence with its new anatomical function, delivering a stabilizing force component at the humeral head. This study illustrates the importance of biomechanical force and moment balance in rotator cuff pathology and tendon transfer surgery.

Chapter 6

Teres major muscle activation relates to scapula lateral rotation in patients with a glenohumeral cuff tear

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Clinical Biomechanics, submitted.

Abstract

Scapula lateral rotation in patients with a glenohumeral cuff tear is increased during arm elevation, which might be the consequence of teres major activation, resulting in a pain avoidance compensatory response to decreased glenohumeral rotations. The teres major may, amongst other muscles, be responsible, which can be investigated by studying the effect of transfer surgery of either the scapulo-humeral teres major or the scapulo-thoracic latissimus dorsi on scapula lateral rotation.

Scapula lateral rotation was measured relative to the thorax during arm *ab*duction elevation, using an electromagnetic tracking device. Lateral rotation in cuff tear affected shoulders was compared to the non-affected side and re-assessed after teres major or latissimus dorsi tendon transfer. Additionally preoperative lateral rotation was related to teres major and latissimus dorsi activation and pain scores.

Patients with a glenohumeral cuff tear exhibited increased scapula lateral rotation during arm *ab*duction, which was proportional to teres major activation as opposed to latissimus dorsi activation. Increased lateral rotation persisted after teres major transfer, while it normalized after latissimus dorsi transposition. Preoperatively pain scores reduced with increased lateral rotation.

The teres major likely plays a role in both lateral rotation of the scapula (this study) and downward traction on the humerus. After latissimus dorsi transfer, teres major contribution to glenohumeral stability is redundant and scapula lateral rotation normalizes. Scapula lateral rotation may have an additional contribution to pain reduction in patients with a glenohumeral cuff tear.

6.1 Introduction

In patients with a glenohumeral cuff tear, increased lateral rotation of the scapula is considered to be a scapula-thoracic compensation for decreased gleno-humeral rotation (McClure et al., 2001). Increased lateral rotation, which occurs mainly during elevation onset, seems to be triggered by pain (Scibek et al., 2008). In previous research we found that pain in patients with a glenohumeral cuff tear induced large arm *ad*ductor muscles to co-activate with the prime mover, i.e. deltoid muscle, during arm elevation tasks (Steenbrink et al., 2006; de Groot et al., 2006). This so-called 'out-of-phase' activation of *ad*ductor muscles, like the teres major and latissimus dorsi, is considered to be an attempt to stabilize or center the humeral head onto the glenoid fossa (Steenbrink et al., 2009). By *ad*ductor muscle activation during arm elevation tasks, the humeral head is prevented from proximal migration and thus (painful) tissue inclination due to subacromial space reduction (Deutsch et al., 1996; Graichen et al., 1999a). Next to centering the humeral head, scapula rotation is a requisite to maintain an optimal contact between the glenoid fossa and the humeral head during arm *ab*duction elevation (Bagg and Forrest, 1988). Alike humeral head suppression, scapula lateral rotation, resulting in acromion toppling, enlarges the subacromial space (Flatow et al., 1994; Meskers et al., 2002).

The scapula-thoracal trapezius and the serratus anterior muscles are suggested to dictate scapular motion in terms of lateral rotation (Flatow et al., 1994; Ludewig and Cook, 2000). The scapula-humeral teres major also has a direct scapula lateral rotating moment because of its origin at the inferior angle of the scapula and insertion on the intertubercular sulcus on the humerus. The scapula-thoracal latissimus dorsi is a multi-articular muscle, originating from the thoracic spinous processes (T7-T12), also inserting on the intertubercular sulcus on the humerus, which has a scapula medial rotation moment (via sterno-clavicular and acromio-clavicular joints).

The teres major in patients with a glenohumeral cuff tear is active while generating arm elevation moments (de Groot et al., 2006; Steenbrink et al., 2006). If scapula lateral rotation is related to teres major activation, lateral rotation in the affected scapula will be increased, compared to the non-affected shoulder. Transfers of either the scapulo-humeral teres major or scapulo-thoracic latissimus dorsi, both known successful and pain relieving salvage procedures in irreparable cuff tears changing their original *ad*duction to an *ab*duction contribution (Celli et al., 2005; Gerber et al., 2006) may potentially reveal the contribution of teres major activation to scapula lateral rotation. By transfer, both muscles are expected to activate dur-

ing arm elevation tasks (Irlenbusch et al., 2008a, Steenbrink et al., 2009), pulling down the humeral head, preventing proximal migration and subsequent painful subacromial space reduction. After latissimus dorsi transfer, additional teres major *ad*ductor muscle contributions to glenohumeral stability as observed prior to surgery are assumed to be redundant. 'Out-of-phase' teres major activation is expected to decrease, which should normalize scapula lateral rotation.

The purpose of this study is to investigate whether scapula lateral rotation in case of a cuff tear is altered. Scapula lateral rotation of the affected shoulder is examined and compared with scapula lateral rotation of the non-affected shoulder. Scapula lateral rotation after teres major and latissimus dorsi tendon transfer surgery is assessed and preoperative scapula lateral rotation is related to teres major and latissimus dorsi muscle activation and pain scores. We hypothesize that scapula lateral rotation in increased in case of a cuff tear, which is related to pain decrease and affected by teres major as opposed to latissimus dorsi activation.

6.2 Methods

6.2.1 Subjects

Thirty-two patients were sequentially recruited from the Leiden University Medical Center department of Orthopaedics' out-patient clinic in the period between June 2005 and November 2009. All selected patients had a MRI proven full thickness retracted rotator cuff tear larger than $4cm$ including at least the supraspinatus and infraspinatus tendons. Patients had Goutallier grade 3-4 fatty degeneration, excluding primary cuff repair (Goutallier et al., 1994). Patients underwent a tendon transfer of either the teres major (n=11) as described by Celly and co-workers (1998)(Celli et al., 1998) or the latissimus dorsi (n=10) as described by Gerber and co-workers (Gerber et al., 1988), allocated in order of appearance. Three cases of bi-lateral pathology and in which the maximum elevation of the non-affected arm was restricted below 100°, were excluded. Six patients refrained from surgery due to personal factors and five patients experienced per-or postoperative complications and were therefore lost for follow-up.

6.2.2 Kinematics

Scapula lateral rotation of shoulders with cuff tears was compared to scapula rotation of the non-affected side. After transfer of either the teres major or latissimus dorsi, scapula lateral

rotation was re-assessed (follow up range 6-12 months). A Flock of Birds electromagnetic tracking device (Ascension Technology, Burlington, VT, USA) with an extended range transmitter (Meskers et al., 1998) was used. Measurements were performed simultaneously on both arms according to the standardized motion recording protocol for the shoulder of the International Shoulder Group (Wu et al., 2005). Five sensors, size 25.4*mm* x 25.4*mm* x 20.3*mm*, were used. One sensor was attached on the middle of the sternum using tape and one on each side distally on the dorsal area of the upper arm using straps. In order to allow for dynamic measurements, acromion sensors were used on both sides (Karduna et al., 2001; Meskers et al., 2007). The acromion sensor was taped on the skin directly above the flat part of the acromion in the most latero-caudal corner, just above the angulus acromial (McClure et al., 2001; McQuade and Smidt, 1998; Meskers et al., 2007; Karduna et al., 2001). Initial measurements were performed to define 3D positions of bony landmarks (Wu et al., 2005) in the local co-ordinate system of the receivers by using a freely movable sensor attached to a pointer of 0.05cm (Meskers et al., 1998a). The glenohumeral center of rotation was determined using a regression method from scapular bony landmarks (Meskers et al., 1998b). Subsequently, subjects were asked to perform a bilateral maximal arm abduction elevation at a comfortable (low) speed with eternally rotating the arm when elevating. Data acquisition rate was about 6Hz.

6.2.3 Data processing

Scapula lateral rotation was expressed as a function of humeral *ab*duction elevation. This relation may not be linear, i.e. lateral rotation at small arm elevation angles may differ from lateral rotation at high arm elevation angles. Therefore lateral rotation between the affected and non-affected side was compared at equal humerus elevation angles, ranging from rest angle to the maximal elevation angle of the affected side. Angles above 100° were discarded because of the risk of invalid recordings as a consequence of the use of an acromion sensor (Meskers et al., 2007). The relation of scapula lateral rotation to humerus elevation angle was quantified by the slope (β) of a first order function fit through the individual data points.

6.2.4 Pain

Preoperative shoulder pain, experienced by the patients to be typical, was quantified by a *Visual Analog Score* (VAS) for pain (0: no pain; 100: worst pain ever imaginable).

6.2.5 Muscle activation

Preoperative teres major and latissimus dorsi muscle activations were expressed in *activation ratios* (Steenbrink et al., 2009). Subjects were seated with their injured arm in a splint with the elbow in 90° flexion. The plane of elevation was about 60°, elevation about 45° and external rotation about 30°. Each recording session the teres major and latissimus dorsi were palpated and the electrodes placed on the middle of the muscle belly. During isometric contraction in *ab*-and *ad*duction directions, surface electromyography (*EMG*) of the teres major and latissimus dorsi was collected. *EMG* was recorded using a DelSys system (Bagnoli-16 Boston MA, USA). The dimensionless normalized *activation ratio* ($1 <= AR_{muscle} <= 1$, Eq. 6.1) indicates the ratio for muscle of 'in-phase' activation (A^{IP}, the expected attribution according to the muscles positive moment arm, being for both teres major and latissimus dorsi during arm *ad*duction) reduced with the 'out-of-phase' activation (A^{OP}, the non-expected attribution, e.g. during *ab*duction) over the summed 'in-phase' and 'out-of-phase' activation, Eq. 6.1.

$$AR_{muscle} = \frac{A^{IP}_{muscle} - A^{OP}_{muscle}}{A^{IP}_{muscle} + A^{OP}_{muscle}} \qquad [-1 \leq AR_{muscle} \leq 1] \tag{6.1}$$

Where A^{IP} is 'in-phase' activation (*EMG* during *ad*duction task - *EMG* at rest); A^{OP} is 'out-of-phase' activation (*EMG* during *ab*duction task - *EMG* at rest) for the *muscles* teres major or latissimus dorsi.

6.2.6 Statistics

Scapula lateral rotation slopes of the affected and the contra-lateral non-affected shoulders at equal motion ranges were statistically compared using a paired samples t test. Pre- and postoperative scapula lateral rotation slopes as of either the teres major or latissimus dorsi patients were compared using a paired samples t-test. Linear regression was applied to assess the relation of preoperative scapula lateral rotation slopes to teres major and latissimus dorsi *activation ratio* and *VAS* scores. The slope coefficient of the regression line was tested to significantly differ from zero. The level of significance was $\alpha = 0.05$ and all statistical tests were performed using SPSS 16.0 (SPSS Inc, Chicago, IL).

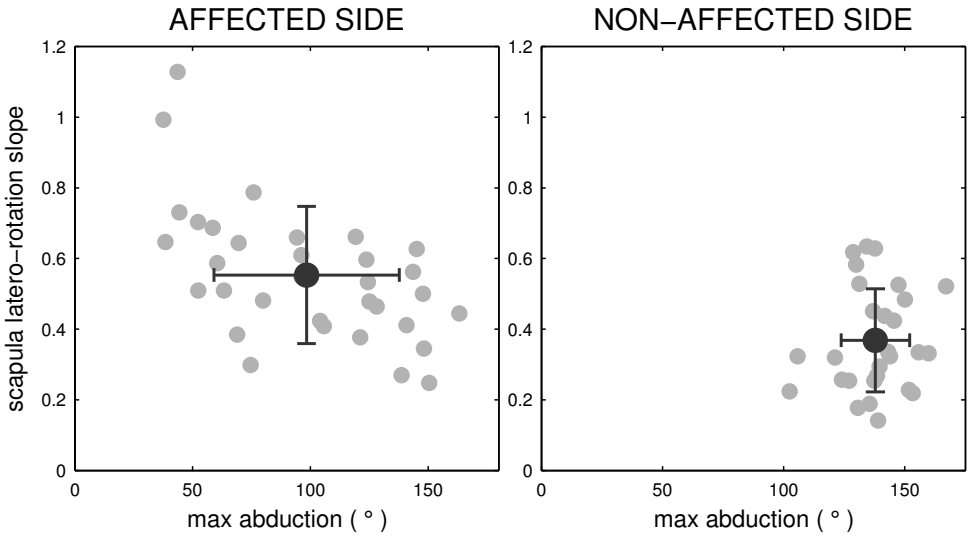

Figure 6.1: Pre-operative slopes (β) of scapula lateral rotation in patients with a gleno-humeral cuff tear(grey circles) of the affected and non-affected contra-lateral side (mean: black circles; standard deviation: black vertical line) for *ab*duction angles, not exceeding 100°. In order to avoid clustering at 100°, slopes were plotted against maximal *ab*duction elevation (mean: black circle; standard deviation: black horizontal line). Slopes of the non-affected side were computed over the same range as the affected side.

6.3 Results

In shoulders with rotator cuff tears (n=32) scapula lateral rotation was significantly increased (mean β = 0.57 SD 0.21) compared to the non-affected shoulder (mean β = 0.37 SD .15, 95% confidence interval of the difference: CId= [0.10, 0.32], p = 0.001, Fig. 6.1). In two cases, scapula lateral rotation was nearly equal to humerus elevation ($\beta \approx 1$), i.e. no gleno-humeral rotation. In the teres major group (n=11) preoperative scapula lateral rotation (mean β = 0.56 SD 0.21) was not affected by the teres major transfer (mean β = 0.51 SD 0.19, 95% confidence interval of the difference: CI_d = [0.07, 0.17], p = 0.38)(Table 6.1). Latissimus dorsi tendon transfers (n=10) resulted in significant decrease from preoperative (mean β = 0.57 SD 0.25) to postoperative (mean β = 0.44 SD 0.14) scapula lateral rotation (95% confidence interval of the difference: CI_d = [0.01, 0.24], p = 0.03)(Table 6.1). The linear regression estimate and 95% CI's of preoperative scapula lateral rotation to *activation ratios* of teres major and latissimus dorsi are presented in Figure 6.2. The slope coefficient of the

Table 6.1: Scapula lateral rotation slopes (β) of patients with a glenohumeral cuff tear and mean (standard deviation) prior to and after transfer of either teres major (TM) or latissimus dorsi (LD) tendons. Preoperative augmented lateral rotation persisted in the teres major group as opposed to the latissimus dorsi group.

	Scapula lateral rotation over abduction angle (β)			
	TM transfer group		*LD transfer group*	
Patients	*Pre*	*post*	*pre*	*Post*
1	0,61	0,89	0,59	0,40
2	0,46	0,48	0,30	0,19
3	0,25	0,35	0,48	0,37
4	0,73	0,72	0,48	0,45
5	0,69	0,23	1,13	0,61
6	0,70	0,42	0,79	0,65
7	0,44	0,38	0,65	0,52
8	0,55	0,62	0,53	0,49
9	0,51	0,49	0,38	0,37
10	0,60	0,65	0,35	0,37
11	0,27	0,41	X	X
Mean	0,56	0,51	0,57	0,44
SD	0,21	0,19	0,25	0,13

estimate linear fit for the teres major differed significantly from zero (p = 0.02) in contrast to the non-significant latissimus dorsi fit (p = 0.99). The linear regression estimate and 95% CI's for the scapula lateral rotation to *VAS* scores is presented in Figure 6.3. The (negative) slope coefficients of the estimate linear fit illustrate increased pain experience with decreased scapula lateral rotation. This differed significantly from zero (p = 0.04).

6.4 Discussion

Goal of this study was to assess scapula kinematics before and after teres major and latissimus dorsi tendon transfer in order to investigate the potential roll of the teres major to lateral rotation. We found enhanced lateral rotation of the scapula in the affected, cuff tear shoulder, which persisted after teres major transposition as opposed to transposition of the latissimus dorsi. A relation between enhanced scapular lateral rotation and teres major activity could be established, as well as a relation between lateral rotation and pain.

Reports on increased scapula lateral rotation in patients suffering rotator cuff tears (Graichen et al., 1999b; Mell et al., 2005; Paletta, Jr. et al., 1997; Scibek et al., 2009; Deutsch et al., 1996) coincide with our results. Increased lateral rotation in these patients was related to

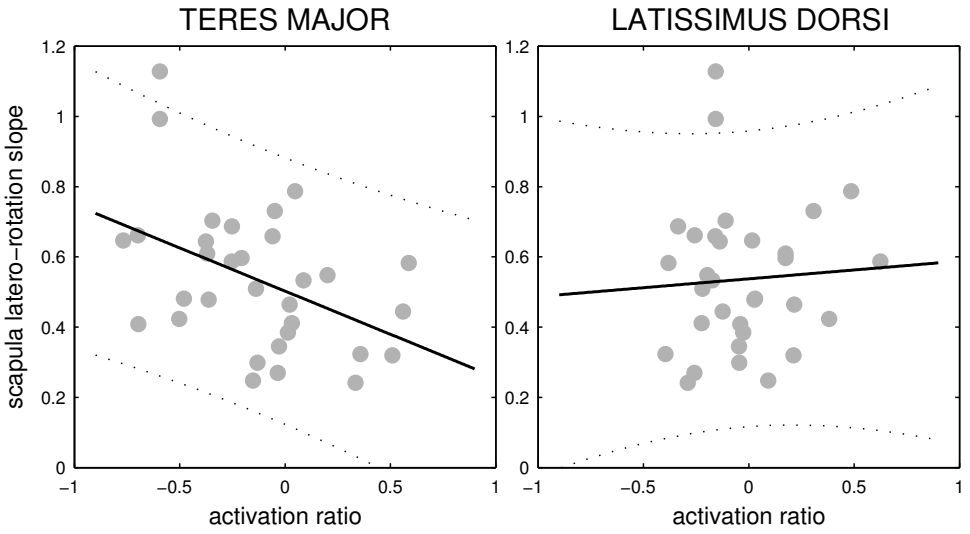

Figure 6.2: Regression lines of scapula lateral rotation over pre- and post surgery arm elevation ration slope (β) as a function of *activation ratio* of patients with a glenohumeral cuff tear (grey circles). The slope of the regression line (β) in case of teres major *activation ratio* differed significantly from zero in contrast to the latissimus dorsi *activation ratio*.

pain (Scibek et al., 2008), which can be explained by a teres major co-activation to prevent painful subacromial tissue inclination due to lost glenohumeral stability (Steenbrink et al., 2006), and its correspondent contribution to scapula lateral rotation. Scapula lateral rotation was previously related to lost stabilizing cuff forces in experimental simulation of a rotator cuff tear, using a suprascapular nerve block (McClure et al., 2001), and surgical repair of the rotator cuff (Paletta, Jr. et al., 1997). In these studies respectively increased lateral rotation and 'normalization' of increased scapula lateral rotation was found. Lost stabilizing forces, and subsequent painful subacromial space width decrease, requires compensational muscle activation and scapula orientation changes. Because of the small subacromial space, even subtle changes in scapula-humeral orientation potentially result in subacromial space width changes (Nordt III et al., 1999; Graichen et al., 1999b; Meskers et al., 2002). Therefore even a small increase in scapula lateral rotation enlarges the subacromial space width, contributing to painful tissue inclination avoidance. Also, a lack of scapula lateral rotation, causing early subacromial space width reduction, is reported to be associated with the subacromial impingement syndrome (Ludewig and Cook, 2000; Su et al., 2004).

The recorded scapula lateral rotation persisted after a teres major tendon transfer as opposed to observations after a latissimus dorsi transfer. Functional transfers, i.e. activation of the transferred muscle during elevation moment exertion, were observed after teres major transposition, involving predominant teres major activation during upwards arm force exertion (Steenbrink et al., 2009c), pulling down the humeral head, compensating lost glenohumeral stability. Equivalent teres major forces work on the scapula, which contributes to the enhanced scapula lateral rotation. Like the teres major, the transposed latissimus dorsi also delivers a downwards pull on the humeral head to prevent humeral head migration. In this case, the regained stabilizing forces of the transferred latissimus dorsi cause less need for additional teres major 'out-of-phase' activation explaining reduced scapula lateral rotation in the latissimus dorsi transfer group. Analysis of teres major *activation ratios* after latissimus dorsi tendon transfer indeed showed that 8 out of 10 patients did not show preoperative observed 'out-of-phase' teres major activation postoperatively. Without teres major activation during arm *ab*duction tasks the increased scapula lateral rotation normalizes, which substantiates scapula lateral rotation to be affected by teres major activation. The observed relation between preoperative scapula lateral rotation and teres major *activation ratios* in contrast to latissimus dorsi *activation ratios* also illustrates the potential role of teres major activation in lateral rotation.

The relation between preoperative scapula lateral rotation and *VAS* scores for pain could relate to a subacromial space width reduction (or insufficient downward forces) causing painful tissue inclination at lower scapula lateral rotation angles. The reported pain reduction after latissimus dorsi transposition (Irlenbusch et al., 2008b) demonstrate the predominant effect of caudal directed stabilizing glenohumeral forces of the transferred muscle over muscle force contribution to lateral rotation to affect the subacromial space width.

Activation ratios to describe muscle function of the teres major and latissimus dorsi were determined in a static set-up. Suppositions towards dynamics in the relation between scapula lateral rotation and teres major activation should be reserved. The negative relation between scapula lateral rotation and pain was explained by a subacromial space reduction at increasing humerus elevation with low scapula lateral rotation, causing painful subacromial tissue inclination. However, subacromial space width decrease was not determined in this study, but was previously observed in combination with decreased scapula lateral rotation to arm elevation (Graichen et al., 1998; Solem-Bertoft et al., 1993; Graichen et al., 1999b; Karduna et al., 2005). The role of scapula kinematics in glenohumeral joint pathologies requires an extensive study towards the determinants of shoulder function in these patients. Studying the

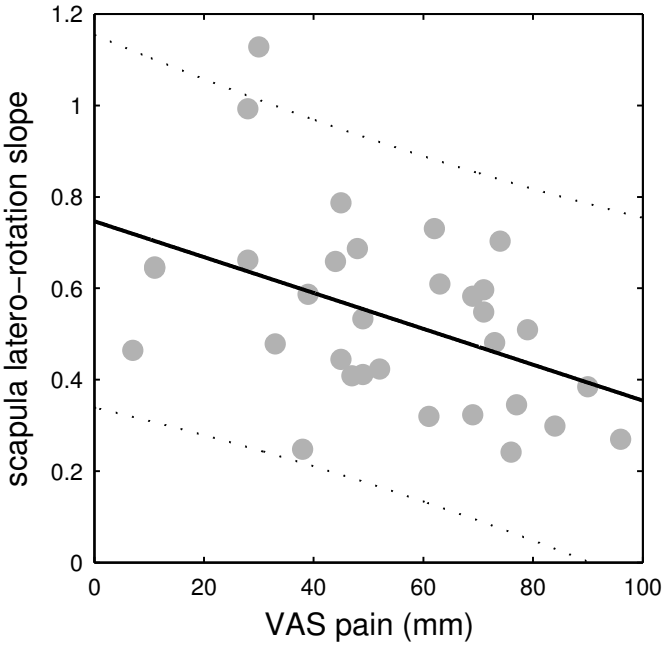

Figure 6.3: Regression lines of preoperative scapula lateral rotation over arm elevation ratio slope (β) as a function of a *Visual Analog Scale* for pain assessed by patients with a glenohumeral cuff tear (grey circles). 0 indicates no pain; 100 indicated worst pain ever imaginable.

contribution of scapula kinematics on outcome after tendon transfer surgery requires a randomized clinical trial with teres major and latissimus dorsi tendon transfers, combined with analysis of muscle activation and kinematics of the entire shoulder, i.e. scapulo-thoracal and gleno-humeral rotations.

6.5 Conclusion

Enhanced scapula lateral rotation persisted after teres major tendon transfer and decreased after latissimus dorsi transfer, substantiating teres major contribution to scapula kinematics. After latissimus dorsi transfer, the preoperative observed teres major activation during arm elevation tasks in compensating lost glenohumeral stability is redundant and teres major activation and consequences for scapula lateral rotation decrease. When activating during arm

elevation tasks, teres major activation thus might prevent a painful subacromial space reduction in patients with a glenohumeral cuff tear by simultaneous pulling down the humeral head and increasing lateral rotation of the scapula. Such secondary effect of the teres major, after the preventing the humeral head from proximal migrating by pulling it down during arm elevation tasks, could be an argument in preferring the teres major over the latissimus dorsi in tendon transfer surgery for patients suffering glenohumeral cuff tears. To draw any conclusions on this matter requires additional comparative research.

Chapter 7

The relation between increased deltoid activation and *ad*ductor muscle activation due to glenohumeral cuff tears.

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Abstract

In patients with irreparable rotator cuff tears, lost elevation moments are compensated for by increased deltoid activation. Concomitant proximal directed destabilizing forces at the glenohumeral joint are suggested to be compensated for by 'out-of-phase' *ad*ductor activation, preserving glenohumeral stability. Aim of this study was to demonstrate causality between moment compensating deltoid activation and stability compensating 'out-of-phase' *ad*ductor muscle activation.

A differential arm loading with the samen magnitude forces applied at small and large moment arms relative to the glenohumeral joint was employed to excite deltoid activation, without externally affecting the force balance. Musculoskeletal modeling was applied to analyze the protocol in terms of muscle forces and glenohumeral (in)stability. The protocol was applied experimentally using electromyography (EMG) to assess muscle activation of healthy controls and cuff tear patients.

Both modeling and experiments demonstrated increased deltoid activation with increased moment loading, which was higher in patients compared to controls. Model simulation of cuff tears demonstrated glenohumeral instability and related 'out-of-phase' *ad*ductor muscle activation which was also found experimentally in patients when compared to controls.

Through differential moment loading, the assumed causal relation between increased deltoid activation and compensatory *ad*ductor muscle activation in cuff tear patients could be demonstrated. 'Out-of-phase' *ad*ductor activation in patients was attributed to glenohumeral instability. The experimental moment loading protocol discerned patients with cuff tears from controls based on their compensatory muscle activations.

7.1 Introduction

Arm mobility requires muscle forces to generate joint moments whilst preserving glenohumeral stability. Arm elevation moments are mainly generated by the deltoids and arm depression moments by the latissimus dorsi, teres major and pectoralis major, all muscles with large moment arms (Kuechle et al., 1997). Glenohumeral stability is controlled for by the rotator cuff muscles (Ackland and Pandy, 2009; Poppen and Walker, 1976). Because of their short moment arms and perpendicular orientation to the glenoid, the rotator cuff muscles can generate compressive joint-forces with relatively small moments, providing glenohumeral stability by directing the resultant force vector through the glenoid fossa.

Tears of the rotator cuff result in lost stabilizing forces and *ab*duction moment loss. The deltoids seem to compensate for the lost *ab*duction moments, resulting in an increased proximally directed force component on the humeral head (Liu et al., 1997). This jeopardizes glenohumeral stability by proximal rotation of the resultant force vector outside the glenoid fossa (Parsons et al., 2002; McCully et al., 2006; Steenbrink et al., 2006 and 2009a). In patients with irreparable cuff tears, *ad*ductor muscle activation of the pectoralis major, latissimus dorsi and/or teres major was observed during arm elevation tasks (de Groot et al., 2006; Steenbrink et al., 2006). This unexpected activation is adverse, or 'out-of-phase', with respect to the muscle moment arm for *ad*duction. Such 'out-of-phase' *ad*ductor muscle activity was also observed in cuff tear model simulations using a musculoskeletal shoulder model with a constraint stable glenohumeral joint (Steenbrink et al 2009a). We hypothesized that glenohumeral stability during arm elevation tasks was preserved by 'out-of-phase' *ad*ductor muscle activation. 'Out-of-phase' *ad*ductor muscle activation seems to be mechanically related to increased deltoid activity.

Glenohumeral joint stability is a common factor between increased deltoid activity (McCully et al., 2007) and adverse *ad*ductor activity (de Groot et al., 2006; Steenbrink et al., 2006; Steenbrink et al., 2009c). However, experimental recording of glenohumeral stability requires additional recording techniques (e.g. Deutsch et al 1996, Graichen et al. 2005, Nagels 2008), or can be derived by musculoskeletal shoulder model simulation (Steenbrink et al., 2009a).

The prime goal of this study is to experimentally demonstrate the causal relation between increased deltoid activation and 'out-of-phase' *ad*ductor activation in patients with a glenohumeral cuff tear, accordingly demonstrating the role of compensatory muscle forces in glenohumeral stability. This is accomplished by applying a differential arm loading with

constant forces at small and large moment arms relative to the glenohumeral joint while the external force balance is not affected. Each individual *ad*ductor muscle previously demonstrated 'out-of-phase' *ad*ductor activation (de Groot et al., 2006, Steenbrink et al., 2006). A secondary goal of this study is to demonstrate by model simulation the potential of each of these individual *ad*ductor muscles to compensate for glenohumeral instability.

7.2 Methods

The mechanical response to the changing moment loading was studied by three different conditions and the combination of model simulation and an experiment. A seven-level incremented elevation moment loading simulation (Fig. 7.1/I) was applied in order to control for the (non-)linearity of the mechanical response to gradual increased loading. A reduced two-level (differential) elevation moment loading (Fig. 7.1/II) was applied both in simulation and experiment in order quantify the response of the deltoid muscle and to create contrast between patients and controls. The combination of arm elevation and arm depression loading (Fig. 7.1/III) was required to demonstrate adverse 'out-of phase' activation of the *ad*ductors observed in patients, relative to the favorable and normal 'in-phase' activation in controls (Steenbrink et al., 2009c). A normalized relative ratio of 'in-phase' over 'out-of-phase' activation, the *activation ratio*, was introduced to be able to parameterize this observation and to be able to compare muscle forces obtained from model simulation and electromyography obtained during the experiments.

7.2.1 Model simulations

The mechanical response to increased moment loading was studied using the Delft Shoulder and Elbow Model (DSEM, van der Helm, 1994; Veeger et al., 1997). The model contains 139 muscle elements and calculates muscle forces required in order to meeting mechanical equilibrium in the specified position and loading condition by means of load sharing using a compound energy related cost function (Praagman et al., 2006). The force per muscle was obtained by summation of representative muscle-element forces. The resultant (total) force working on the humerus should be fully countered by the glenoid joint reaction force for glenohumeral stability.

Kinematic input for the model was equal to previous studies (Steenbrink et al., 2009a; Steenbrink et al., 2009b). The humerus position, defined according to the International Soci-

ety of Biomechanics standards (Wu et al., 2005) was: plane of elevation Yh:γh = 79°, elevation Xh:-βh = 46° and axial rotationYh':γh2 = 31°. A healthy condition ('control model') and a combined supraspinatus-infraspinatus cuff tear condition ('patient model') were simulated.

Seven-level incremented elevation moment loading

The arm was externally loaded with a constant, vertical, downward force of 25N. The force was longitudinal and equally distributed over the humerus from proximal to distal of the glenohumeral joint (Fig. 7.1/I; 1 to 7), resulting in seven different moments but with constant external forces. The resultant external glenohumeral moment ranged from 0.9 to 7.2Nm.

Two-level elevation moment loading

Only the two extreme loads of 0.9 and 7.2Nm were used to compare the mechanical responses of the deltoids for the patients and controls (Fig. 7.1/II; 1 and 2).

Combined two-level elevation and depression moment loading

In combination with the two-level elevation moment loading, an additional opposite loading was required (Fig 7.1/III; 3 and 4) to determine the dimensionless *activation ratio*. The *activation ratio* illustrated the functional contrast for *ab*duction and *ad*duction (for calculations see 7.2.4, outcome parameters) and allowed the comparison between simulated muscle forces and experimental *EMG*. To average morphological and physiological variance between model and subjects, between subjects and within subjects, not a single force at both arm locations was applied, but seven forces at each location distributed over a range of 90°, all perpendicular to the humerus.

Compensatory *ad*ductor hierarchy

In order to demonstrate the potential of each individual *ad*ductor muscle to compensate for glenohumeral instability, the hierarchical redundancy of *ad*ductor function was simulated, by applying models in which the active *ad*ductor muscles were sequentially eliminated. The 'patient model' with the combined supraspinate-infraspinate tear was initially extended with a full subscapularis and the biceps longum tear in order to provoke the other *ad*ductor with larger moment arms to become active (Hansen et al., 2008; Steenbrink et al., 2009a).

7.2.2 Experiments

Ten healthy controls (6 male, 4 female; age 28±3years) with no known history of shoulder injury were compared to ten patients (7 male, 3 female; age 59 ±9 years) with a MRI proven rotator cuff tear of at least supraspinatus and infraspinatus. The study was approved by the institutional medical ethics committee, and all subjects gave written informed consent.

Subjects were positions resembling model input (see 7.2.1, model simulations) with their injured (patients) or dominant (controls) arm fully supported in a splint (Fig. 7.1/II). The splint was connected to a 6DOF-force transducer (A)(AMTI-300, Advanced Mechanical Technology, Inc., Watertown MA, USA) using a Cardan joint (B)(3 free rotational degrees of freedom (DOF)), allowing for a 20cm shift of the force application point along the humerus to realize a moment change with relatively small and large moment arms, with respect to the glenohumeral center of rotation. The force transducer was mounted on a low friction rail (C)(1 free translational DOF). Subjects exerted arm forces against the two remaining, fixed DOF's perpendicular to the upper arm, controlled for magnitude and direction using target areas on a computer screen (D). A force driven cursor was to be held within the target area for two seconds. Simultaneously with each loading condition, surface EMG was recorded for the deltoid, pectoralis major, latissimus dorsi and teres major.

Two-level elevation moment loading

The targets were distributed in a range of 90 degrees (see paragraph 7.2.1.2 and 7.2.1.3), randomly represented in seven upward directions at two levels, provoking small and large arm elevation moments (downward external forces) similar to the model simulations (paragraph 7.2.1.2).

Two-level elevation and depression moment loading

To determine activation ratios (paragraph 7.2.1.3) seven downward directed targets, provoking arm depression moments, at two levels were added.

7.2.3 Signal analysis

Surface *EMG* was recorded for each 2-second isometric force task (DelSys, Bagnoli-16, Boston MA, USA, analog filter: 20Hz High pass, 450Hz Low pass, 10*mm* electrode length, inter-electrode distance of 10mm). Between trials rest periods (minimally five seconds) were

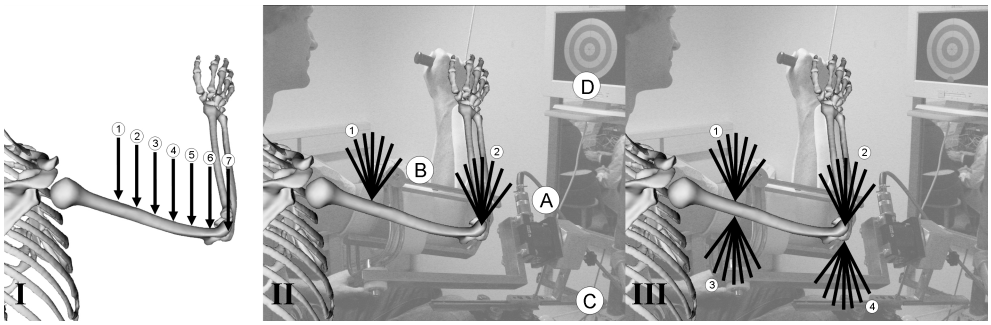

Figure 7.1: Impression of loading conditions.
I: model; 1 to 7 indicate incrementing moment loading conditions.
II: model and experimental; 2 differential moment loading conditions (1 and 2). Subjects' arms were supported with a splint connected to a force transducer (A) using a Cardan joint (B), in combination with an axial rotation axis, allowing for a 20cm shift of the force application point. The force transducer was placed onto a rail (C). Exerted arm forces were visualized on a screen (D).
III: model and experimental: downward (1 and 2) and upward (3 and 4) loading.

imposed to avoid fatigue. Sample rate of analog filtered EMG and force data was 1000Hz. A 2 seconds 'in target' full-wave rectified (r) and integrated (I: 3rd order recursive Low Pass Butterworth at 10Hz) $rIEMG$ was averaged. Rest level $rIEMG$ was subtracted from each target value. One single muscle activation level for the upwards and one for the downwards force tasks were calculated by averaging the seven net target $rIEMG$'s.

7.2.4 Outcome parameters

Model muscle activation (estimated $force$) was compared with experimental muscle activation ($rIEMG$). For each of the simulated seven-level incremented elevation moment loading condition deltoid and *ad*ductor muscle forces were calculated. For the two-level differential loading the change of deltoid activation (force and $rIEMG$) and *activation ratios* were calculated. Deltoid activation increase in response to increased loading was expressed as the percentage of deltoid activation at relative small moment loading. The *activation ratio* (Eq. 7.1) indicates the ratio for muscle of 'in-phase' activation (A^{IP}, the expected attribution according to the muscles positive moment arm for ab-*ad*duction) reduced with the 'out-of-phase' activation (A^{OP}, the non-expected attribution according to the muscles positive moment arm) over the summed 'in-phase' and 'out-of-phase' activation.

$$AR_{muscle} = \frac{A^{IP}_{muscle} - A^{OP}_{muscle}}{A^{IP}_{muscle} + A^{OP}_{muscle}} \qquad [-1 \leq AR_{muscle} \leq 1] \tag{7.1}$$

Where A^{IP} is 'in-phase' activation; A^{OP} is 'out-of-phase' activation; muscle is the deltoids (DE), teres minor (TMn)(only available in simulation), pectoralis major (PMj), latissimus dorsi (LD) or teres major (TMj). The moment arms (or potential moment vectors) in the DSEM were used to define the directions of 'in-phase' and 'out-of-phase' activation for each muscle (van der Helm, 1994; Veeger and van der Helm, 2007; Steenbrink et al., 2009a). The experimentally obtained *activation ratios* for the *ad*ductors were lumped in order to express the combined *ad*ductors effort. AR_{AD} thus comprehends the averaged *activation ratio* of pectoralis major, latissimus dorsi and teres major.

7.2.5 Statistics

The net-change of deltoid activation from small to large moment loading between controls and patients was compared using a Student's t-test. *Activation ratios* for small and large glenohumeral moment loading between patients and controls were compared using a General Linear Model *ANOVA* with repeated measures. The statistical model comprised the effects of moment loading (within) and group (patients vs. control; between factor). All tests were performed using SPSS 16.0 (SPSS Inc, Chicago, IL) with an alpha of 5%.

7.3 Results

7.3.1 Model simulations

Seven-level incremented elevation moment loading

The seven-level incrementing elevation moment loading coincided with a proportional (linear) increase in estimated deltoid force (Fig. 7.2). In the patient model with cuff tears, deltoid forces exceeded the forces observed in the control model. The deltoid force increase in the patient model was accompanied by a proportional increase of teres minor *ad*ductor force (Fig. 7.2).

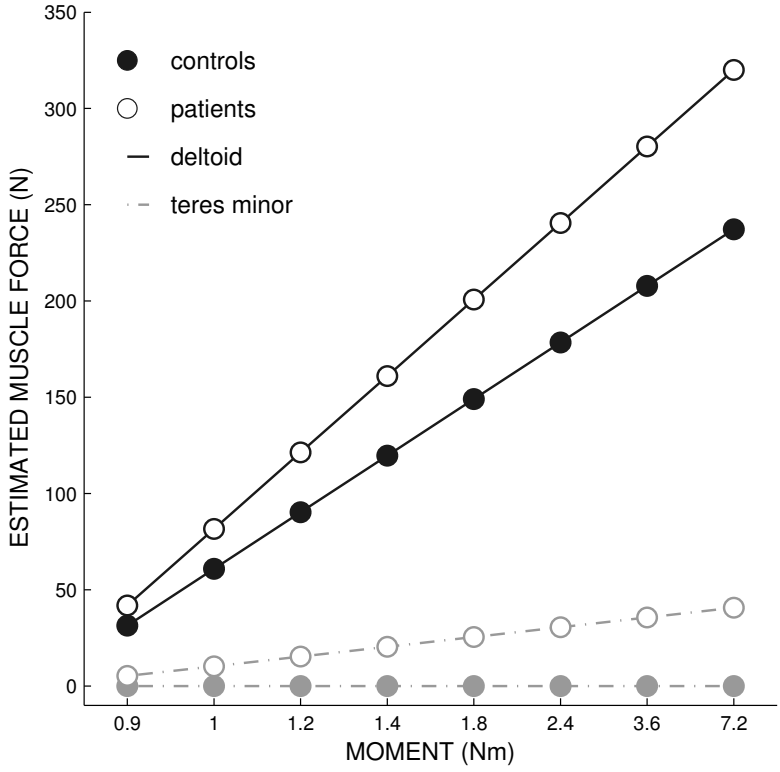

Figure 7.2: Response of simulated abductor (deltoid; black line) and adductor (teres minor; gray dotted line) muscle forces in response to changing moment loading for patients (unfilled marks) and controls (filled marks).

Two-level elevation moment loading

The two-level elevation moment loading consequently showed the maximum contrast of increased deltoid forces when comparing the patient model with the control model (Table 7.1). In the patient model, glenohumeral joint instability in a postero-superior direction was observed in case of downward external forces at both small and large moment loading.

Combined arm elevation and depression loading

In the control model, positive deltoid and teres minor *activation ratios* indicated predominant deltoid activity at downward-, teres minor activity at upward external arm loading. The pectoralis major activation ratio decreased from AR_{PMj} = 0.41 to 0.29, from small to large moment loading. In the patient model, small moment loading resulted in a negative pectoralis major *activation ratio*, AR_{PMj} = -0.23, which decreased at increased loading, AR_{PMj} = 0.40. The teres minor *activation ratio* was small but positive at small loading, AR_{TMn} = 0.11, meaning that teres minor was active during external upward loading, but also during external downward loading, though slightly smaller. The teres minor *activation ratio* reduced to AR_{TMn} = 0.05 at large moment loading (Fig. 7.3).

Compensatory *ad*ductor hierarchy

The patient model comprising fully torn (excluded) supraspinatus and infraspinatus and additional subscapularis and biceps longum elimination, resulted in pronounced 'out-of-phase' *activation ratios* for the pectoralis major and teres minor at large moment loading: AR_{PMj} = -0.25 and AR_{TMn} = -0.17 (Fig. 7.4). Subsequent elimination of the active pectoralis major resulted in 'out-of-phase' activation of the latissimus dorsi with an *activation ratio* of AR_{LD} = 0.28 at small, to a negative AR_{LD} = -0.15 at large moment loading. Subsequent exclusion of the latissimus dorsi resulted in an *activation* ratio for the teres major of AR_{TMj} = 0.44 at small moment loading and AR_{TMj} = 0.09 at large moment loading. Simulation of these 'massive' muscle tears showed the pectoralis major, latissimus dorsi and teres major to be redundant in that order, to stabilizing the glenohumeral joint The teres minor was crucial elimination did not result in a simulation solution with a mechanical stable glenohumeral joint.

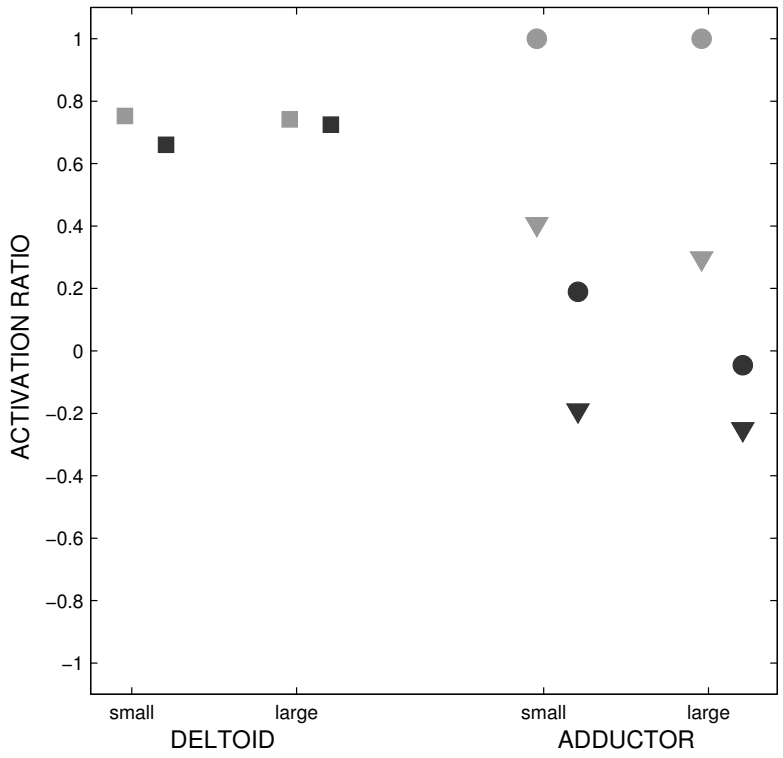

Figure 7.3: *Activation ratios* for the simulated patient and control model at 25N arm loading at **small** and **large** moment arms. In both models glenohumeral stability was constraint. Squares: deltoids; circles: teres minor; downward triangles: pectoralis major.

Table 7.1: Deltoid *forces* estimation (N) in simulation of control and patient during a 25N *ab*duction loading at a small or large glenohumeral moment arm. The patient is simulated with complete supraspinatus and infraspinatus cuff lesion. The *force* response of deltoid on increased glenohumeral moment loading is expressed as a percentage of deltoid *force* in the small glenohumeral moment loading. c: in patient simulations the constraint for glenohumeral stability was active

Moment arm	Small	Large	Response
Control	69N	154N	226%
Patient[c]	30N	212N	688%

7.3.2 Experiments

Two-level elevation moment loading

From small to large glenohumeral moment loading with downward external forces, the average deltoid *rIEMG* amplitude increased by 35% (SD 22%) in controls. Patients showed a significant additional deltoid *rIEMG* amplitude increase with 57% (SD 26%) (Table 7.2).

Two-level elevation and depression moment loading

Deltoid *activation ratios* of controls and patients did not differ at small ($p = 0.73$) nor at large ($p = 0.39$) moment loading. At small moment loading, combined pectoralis major, latissimus dorsi and teres major *ad*ductor muscle *activation ratio* (AR_{AD}) in patients was $AR_{AD} = 0.15$ (SD 0.25) and did not differ from controls: $AR_{AD} = 0.39$ (SD 0.25). At large moment loading however, the *activation ratio* in patients, AR_{AD} = -0.06 (SD 0.17), was significantly lower compared to controls, $AR_{AD} = 0.49$ (SD 0.22).

7.4 Discussion

In this study we demonstrated the mechanical relationship between increased deltoid activation and glenohumeral stability compensating 'out-of-phase' *ad*ductor muscle activation in cuff tear patients. In both controls and patients, deltoid forces proportionally increased with glenohumeral moment loading. Patients needed additional deltoid forces to compensate for lost rotator cuff *ab*ductor forces. This concept of deltoid compensation for lost cuff *ab*duction function (Sharkey et al., 1994) is supported by cadaver experiments (Parsons et al., 2002), our

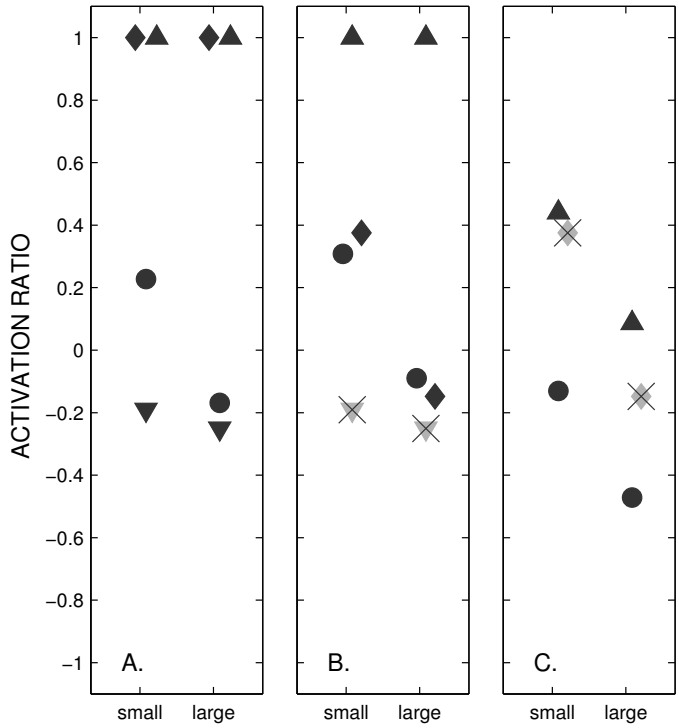

Figure 7.4: *Activation ratios* of *ad*ductor muscles for three simulated patient models at 25N arm loading at **small** and **large** moment arms. Glenohumeral stability was constraint.
A: full cuff tearing (exclusion) of supraspinatus, infraspinatus, subscapularis and biceps longum;
B: previous condition (A) with additional pectoralis tear exclusion (crossed out);
C: previous condition (B) with additional latissimus dorsi exclusion.
circles: teres minor; downward triangles: pectoralis major; diamant: lattisimus dorsi; upward triangles: teres major.

Table 7.2: Deltoid *rIEMG* response on increased moment loading in Controls (N=10) and Patients (N=10), expressed as a percentage of *rIEMG* at small moment loading.

	Response (%)	
Subject	**Control**	**Patient**
1	119	178
2	160	141
3	167	180
4	108	129
5	140	195
6	153	133
7	154	123
8	112	160
9	117	185
10	121	149
Mean	135%	157%
SD	22%	26%

previous model study (Steenbrink et al., 2009a) and experimental nerve block studies (McCully et al., 2006). The deltoids can predominantly be defined as a moment generator.

Model simulations showed increased deltoid forces in cuff tear conditions leading to glenohumeral instability, which required *ad*ductor activation. The increased deltoid activation have previously been shown to result in enlarged cranial directed destabilizing forces on the humeral head (Steenbrink et al 2009a), causing proximal migration (Graichen et al., 2000) and (painful) subacromial tissue impingement (Keener et al., 2009; Soifer et al., 1996).

Increased deltoid activation in absence of suprasinatus and infraspinatus forces resulted in 'out-of-phase' *ad*ductor activity under external downward loading conditions, both in patient model simulation and patient experiments, in contrast to controls. Compensatory muscle activations does not seem to be age specific and therefore age-matched groups are not required (Steenbrink et al., 2009b). Introduction of the *activation ratio* allowed comparison between simulated muscle forces and experimentally obtained *rIEMG*'s. Based on this *activation ratio*, patients could be retrospectively discriminated from controls based on their muscle activation. This study experimentally provokes the glenohumeral stability compensating mechanism in patients with irreparable cuff lesions. In concordance with the concept of the compromised mobility-stability interaction (de Groot et al., 2006; Steenbrink et al.,

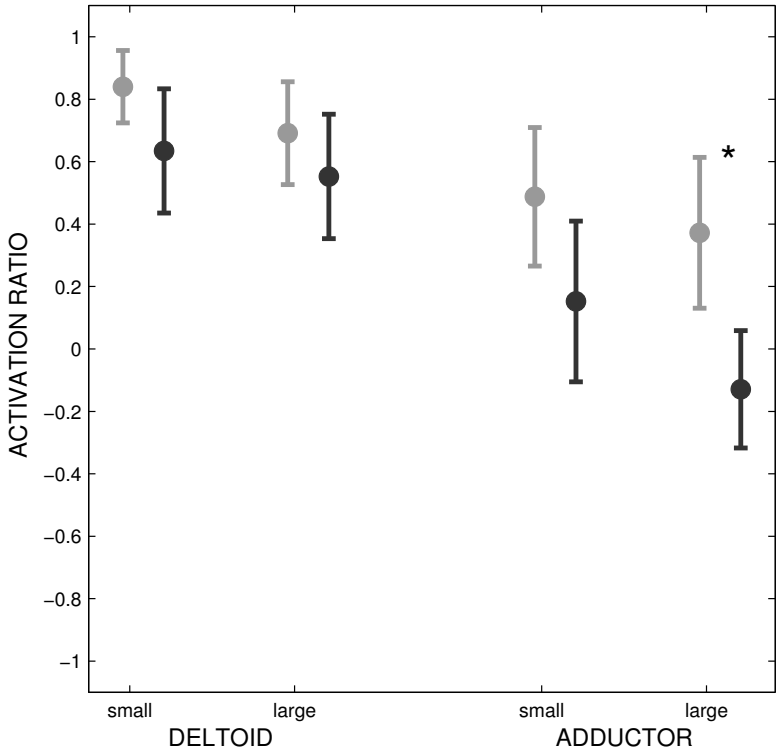

Figure 7.5: Average experimental *activation ratios* and standard deviations of the deltoid anterior part (*ab*ductor) and the combined *ad*ductor muscles (pectoralis major, latissimus dorsi and teres major) recorded for ten patients (black) and ten controls (grey) at 25N arm loading at **small** and **large** moment arms. Significant differences (p = .05) are indicated (*).

2006; Steenbrink et al., 2009a; Steenbrink et al., 2009a) the *ad*ductor co-activation may be an important factor in explaining the limitations in arm mobility observed in cuff tear patients (Iannotti et al., 1996; Steenbrink et al., 2009).

Glenohumeral instability in patients with cuff tears was observed in the DSEM, but was not measured directly in our experiments. Proximal migration was however shown by patient radiographs (Deutsch et al., 1996) and the magnitude of migration was task-related (Hinterwimmer et al., 2003). The decreased *activation ratios* recorded in patients illustrated 'out-of-phase' *ad*ductor muscle activation, and coincided with simulation outcome, as the response to decreased glenohumeral stability. This phenomenon is a convincing argument for a compensatory muscle activation strategy in cuff tear patients. The future challenge is to find a quantitative relationship between proximal migration in patients with cuff lesions (Graichen et al., 1999; Nagels et al., 2008) and the amount of 'out-of-phase' *ad*ductor muscle activation. This would gap the bridge between the observed 'out-of-phase' *ad*ductor activation and glenohumeral instability.

In model studies of cuff tear simulation, 'out-of-phase' teres minor and pectoralis major activations were observed, whereas 'out-of-phase' activations of pectoralis major, latissimus dorsi and teres major were observed experimentally in patients. These observations are not mutually exclusive. In our experiments teres minor *rIEMG* was not determined because of the need for rather intricate fine wire electrodes and we therefore cannot exclude 'out-of-phase' teres minor activation in the experiment. Previous simulation results showed the teres minor to be indispensable for glenohumeral stability compensation in patients with supraspinatus and infraspinatus tears (Steenbrink et al., 2009a). Patients may have 'out-of-phase' teres minor activation next to the observed 'out-of-phase' activation of larger *ad*ductor muscles. An affected teres minor, possibly due to fatty infiltration (van de Sande et al., 2005), may lead to increased proximal migration of the humeral head.

In the model, a load sharing criterion is applied which reduces redundant muscle activation by means of minimization of energy related costs (Praagman et al., 2006) thus prohibiting 'costly/ineffective co-contraction, other than strictly necessary to preserve glenohumeral stability. The simulated *ad*ductor muscle elimination showed the potential redundancy of *ad*ductor muscles to compensate for cranial instability, but with a mechanical hierarchy of primarily teres minor/pectoralis major, followed by the latissimus dorsi and eventually the teres major. This proved that all *ad*ductor muscles indeed have a potential for stability compensation by 'out-of-phase' activation. Patients apply different combinations of *ad*ductor 'out-of-phase' contractions which may indicate either a more severely affected cuff condi-

tion than diagnosed, or different strategies for muscle force distribution than dictated by an optimal energy criterion. Variability may also be due to anatomical and physiological differences, pathology of muscles (i.e. fatty degeneration) or neuromuscular coordination skills. Exclusion of these factors requires further research.

7.5 Conclusion

In patients suffering irreparable rotator cuff tears, the assumed causal relation between moment compensating deltoid activation and stability compensating 'out-of-phase' *ad*ductor activation was established. Increased moment loading provoked additional deltoid forces, compensating for lost cuff *ab*duction moments and enclosed provoked *ad*ductor activity both in cuff tear simulation and experimentally in patients. The observed 'out-of-phase' *ad*ductor activation is required for glenohumeral stability. In shoulder interventions and evaluations 'out-of-phase' *ad*ductor activation is an indicator of glenohumeral instability.

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Chapter 8

General discussion

8.1 Introduction

Aim of the research described in this thesis was to demonstrate that deviating muscle activation in patients suffering irreparable glenohumeral cuff tears can be related to instability. This was done by obtaining insight in the biomechanical principles of compensatory muscle activation and to study its consequences for arm functionality. In this last chapter the most important conclusions of this research project are discussed and some clinical implications alongside recommendations for future research are described. Knowledge of the mechanisms described in this thesis are of evident importance when assessing the pathological shoulder, because compensatory muscle activation differs from the general expectations on muscle function as learned from anatomy books by orthopaedic surgeons, rehabilitation physicians, physical therapists and occupational therapists.

In patients with glenohumeral cuff tears we found *ad*ductor muscle co-activation, which was 'out-of-phase' according to the muscle moment arms for arm *ad*duction, i.e. *ad*ductor muscle activation during arm *ab*duction elevation tasks (de Groot et al., 2006) (*chapter 2*, *5* and *7*). In musculoskeletal model simulations, cuff tear conditions introduced increased deltoid activation, jeopardizing glenohumeral stability. The superiorly directed destabilizing forces of the deltoids on the humeral head require 'out-of-phase' *ad*ductor muscle activation to preserve glenohumeral stability (*chapter 4*). Glenohumeral stability is thus improved by 'out-of-phase' *ad*ductor muscle activation (*chapter 4* and *7*), but is counterproductive for the intended arm elevation, explaining the observed activation patterns and limitation in arm function in patients with cuff tears. There is a conflict between glenohumeral stability and arm mobility. A teres major tendon transfer allowed for stability compensating forces on the humeral head pulling it caudal and counteracting the increased deltoid forces, without adverse *ad*duction moments. Active use of the transposed *ad*ductor (teres major) solved the conflict between glenohumeral stability and arm mobility and significantly related to functional improvements (*chapter 5*).

8.2 Compensation for lost elevation moments

In case of a massive rotator cuff tear, patients lack the cuff muscles' contribution to arm elevation moment (Sharkey et al., 1994), which can be compensated for by the deltoids because of their favorable moment arm for elevation (Liu et al., 1997). Increased deltoid activation during arm elevation moment exertions was convincingly demonstrated by simulating rotator

cuff lesions, using biomechanical model simulations (Magermans et al., 2004; Steenbrink et al., 2009a), cadaver experiments (Apreleva et al., 2000; Hsu et al., 1997; Parsons et al., 2002; Kedgley et al., 2002) and experimental nerve blocking studies (McCully et al., 2006). An *in vivo* assessment of increased deltoid activation in patients with glenohumeral cuff tear was for the first time presented in *chapter 7*, in which differential moment loading was applied to provoke deltoid activation. Deltoid activation was significantly more increased in patients with cuff tears compared to healthy controls.

The increased deltoid activation in patients with irreparable cuff tears involves cranial directed destabilizing forces on the glenohumeral joint (Steenbrink et al., 2009a). This would plea against arm *ab*duction training in these patients (Brostrom et al., 1992), which would further increase the destabilizing forces on the humeral head. In case of massive rotator cuff tears or other subacromial pathologies like the impingement syndrome, striving to normalization of deltoid activation might be advisable. By lowering superiorly directed forces of the deltoids and subsequent destabilizing forces on the humeral head, proximal migration and painful inclination of subacromial tissue would be reduced. However, deltoid activation lowering also involves a decrease of net arm elevation moment. Knowledge on a possible optimum muscle balance between arm *ab*-and *ad*ductors remains unclear.

8.3 Glenohumeral instability

Increased deltoid forces, associated with the lack of rotator cuff activation in patients with glenohumeral cuff tears, induce a superiorly directed force component on the humeral head. The net muscle force vector, working on the humerus, translates the humeral head cranially, and cannot be fully counteracted by the glenohumeral reaction force. The resultant force component is believed to induce the proximal migration of the humeral head (Poppen and Walker, 1976; Yamaguchi et al., 2000). This hypothesis was confirmed using computational model simulations of successive rotator cuff force exclusion (Steenbrink et al., 2009a). This proximal migration of the humeral head would cause a subacromial space reduction due to the subluxation, also referred to as glenohumeral instability. Glenohumeral instability is repeatedly described as a clinical feature observed in shoulder patients suffering cuff decease (Neer, 1983; Newhouse et al., 1988; Deutsch et al., 1996; Graichen et al., 1996; Anglin et al., 2000; Kido et al., 2000; Meskers et al., 2002; Parsons et al., 2002; Hinterwimmer et al., 2003; Nove-Josserand et al., 2005; Hallstrom and Karrholm, 2006; Kedgley et al., 2007; Keener et al., 2009b).

Migration of the joint reaction force vector outside the glenoid cavity, as assessed by computational modeling, was used as a measure for glenohumeral instability in this thesis. Actual humeral head cranialization however, was not measured directly. Direct measurement of humeral head translation with respect to the glenoid is a further step into the present research field, which would involve the use of e.g. an open MRI system (Graichen et al., 2000), standard anterior-posterior röntgen images (van de Sande and Rozing, 2006; Nagels et al., 2008) or fluoroscopic images (Hallstrom and Karrholm, 2006). Fluoroscopy offers the opportunity to record moving images of internal structures to measure glenohumeral (in)stability in dynamic conditions and to study the consequences for subacromial tissues quality, offering opportunities to investigate the subacromial impingement syndrome. In future research, increased deltoid activation during arm *ab*duction tasks is to provoke enhanced proximal migration of the humeral head, which should then be related to clinical outcome variables such as pain and arm mobility. However, when proximal migration is measured during these arm elevation tasks, one should take arm *ad*ductor muscle forces pulling down the humeral head into account, as these forces decrease proximal migration.

8.4 Compensation for stability lost

Arm *ad*ductor muscle co-activation, simultaneous with deltoid muscle activation, and 'out-of-phase' with the expected activation according to the *ad*ductor muscle moment arm, was found in patients suffering from rotator cuff tears (de Groot et al., 2006; Steenbrink et al., 2006). We interpreted this as being a compensational strategy for (painful) proximal migration, i.e. glenohumeral instability, which is prevented by pulling down the humeral head. Compensation for lost glenohumeral stability by means of *ad*ductor muscle co-activation during arm elevation is a beneficial strategy. However, the large *ad*duction moment arms of these muscles interfere with the intended elevation moment, restricting maximal arm elevation. 'Out-of-phase' *ad*ductor muscle activation or activation during arm elevation tasks is explanatory for the often observed restrictions in maximal arm elevation in patients with glenohumeral cuff tear.

Pain seemed to play a crucial role in glenohumeral cuff disease, as it has been shown to triggered the arm *ad*ductors to activation during arm elevation tasks (Steenbrink et al., 2006) and to induce augmented scapula lateral rotation (Scibek et al., 2008). Arm *ad*ductor activation and scapula lateral rotation were suggested to be related in a pain avoidance mechanism to avoid painful subacromial tissue inclination (*chapter 6*). An attempt to lower 'out-of-

phase' *ad*ductor muscle activation, or to suppress subacromial pain (also lowering 'out-of-phase' *ad*ductor activation (Steenbrink et al., 2006)), is advised against because of the risk for further deterioration of the subacromial tissues due to incilination of these structures between the acromion and the proximally migrating humeral head. Normalizing enhanced scapula lateral rotation, for that matter, might also reduce the subacromial space in patients with glenohumeral cuff tears. Tendon transfer surgery seems to be an adequate salvage procedure for this patient group (*chapter 5*).

*ad*ductor muscle activation during arm elevation moment exertion will irrevocably further increase deltoid activation, because the nett moment, required to elevate the arm, is reduced by such *ad*ductor activation. This would result in a vicious circle of increasing ab-and *ad*ductor muscle activation. Muscle imbalance, involving insufficient arm *ad*ductor activation, was considered to be a risk factor in the development and continuation of subacromial impingements syndrome (Burnham et al., 1993). Asymptomatic rotator cuff tears (Keener et al., 2009; Kelly et al., 2005) also suggest the contingency of compensatory muscle activation without affecting arm functionality. The most important question now is whether glenohumeral stability compensatory *ad*ductor muscle activation, without constricting arm functionality, can be learned. Model simulations with extensive cuff tears and a downward directed external force of 25N, still solving the moment equilibrium while preserving glenohumeral stability (Steenbrink et al., 2009), suggests that it can. A study addressing a specific exercise programme, training arm *ad*ductors to deliver sufficient downwards directed forces during arm elevation, without hindering the intended arm elevation moment, is advised.

Muscle function in healthy conditions change with arm position (Favre et al., 2009a), as will compensatory muscle activation. An analysis of 'out-of-phase' *ad*ductor muscle activation during arm motion will explain some of the variation in functionality often observed in patients suffering subacromial pathologies. To do so further analysis of muscle activation in dynamic conditions is required, using simultaneous *EMG* recordings, motion tracking, and post-processed (Favre et al., 2009b), or if available real-time (Chadwick et al., 2009) inverse dynamic model simulations. Pathological muscle activation can be identified by comparing in vivo muscle activation with simulated muscle forces in dynamic conditions. Effects of specific muscle contributing to glenohumeral (in)stability at higher elevation angles or the influence of passive structures can be assessed by studying differences in estimated and recorded muscle activation. An assessment tool to realize real-time myofeedback, combined with real time feedback on the optimal mechanical muscle activation as estimated by an inverse kinematic musculo-skeletal model, could be an optimally trained device for these patients.

The moment loading experiments applied in *chapter 7* suggested the deltoids to be primarily glenohumeral moment generators as they were chiefly involved in delivering the required increase arm elevation moments. Increased deltoid activation in musculo-skeletal model simulations had consequences for glenohumeral stability and stability compensating *ad*ductor muscle activation. An extensive validation of the proposed mechanisms requires altering external force magnitudes while preserving external moment loading, inverting the experiments from *chapter 7*. In such an experiment, the magnitude of the external force increases proportionally with the glenohumeral moment arm of the external force application point. The increasing caudally directed force magnitudes at smaller moment arms theoretically have a stabilizing effect on the glenohumeral joint. Such force loading experiment is expected to result in a constant deltoid activation, because the external moment does not change. 'Out-of-phase' *ad*ductor activation however is expected to decrease at smaller moment arms because of the increasing stabilizing effect of the external force. In a preliminary simulation study we found the stabilizing force effect not to show the expected results. The analysis of force and moment equilibrium in this example is more complex due to extreme axial components at smaller moment arms with large external forces, which should thus be controlled for. The experimental set-up with the universal joint as used in *chapter 7* can be used for the experimental approach of such force loading paradigm.

Analysis of muscle activation compensating for reduced glenohumeral cuff forces can provide an entry to identify patients suffering massive rotator cuff tears from healthy subjects based upon functional assessment. The use of an arm loading paradigm in clinical practice as a functional measure to assess rotator cuff insufficiencies as a supplement or replacement for extensive radiologic screenings should be further assessed in a prospective study design. Deltoid activation can gradually be provoked to investigating wether there is a cut off point in which deltoid activation starts jeopardizing glenohumeral stability. This cut-off point might relate to the amount of cuff muscle involvement in the tear. The combined use of arm force recordings and EMG assessment does open doors towards the development of selective exercise set-ups for, until recently, difficult-to-treat shoulder patients.To study the causal contribution of specific muscle responses to joint moments or joint stability glenohumeral joint perturbations will be a promising alternative approach. Pure force perturbations induce translations of the humeral head with respect to the glenoid, which is expected to excite the glenohumeral stabilizing muscles, while moment generators will react to joint rotation perturbations. Pathology imposed changes in muscle functions can be studied using a glenuhumeral joint perturbation protocol (de Vlugt et al., 2003).

8.4.1 Teres major vs. latissimus dorsi tendon transfer

In transposing the insertion of a large arm *ad*ductor muscle the stabilizing effect of its downward directed force component is preserved, without the adverse *ad*duction moment component. Both teres major and latissimus dorsi tendon transfers are accepted procedures for improving pain and function loss in patients with glenohumeral cuff tears. In this thesis 'out-of-phase' *ad*ductor muscle activation in massive cuff tear patients was observed for both the teres major and latissimus dorsi, which was considered to be an attempt to preserve glenohumeral stability. Preoperative pathological teres major activation during upward arm force exertion is the desired postoperative activation. Teres major activation could be related to clinical improvement in *chapter 5*. However, pathological latissimus dorsi activation after teres major transposition did not disappear in all patients, suggesting either insufficiently regained glenohumeral stabilizing forces, or an inability to decrease the pathological latissimus dorsi activation. Either way, persisting activation of the non-transferred *ad*ductor muscle during arm elevation tasks counteracts with the intended net elevation moment. Patients might have a preference for which muscle to transfer, which is likely to be related to the preoperative muscle activation pattern as this muscle would already show the postoperatively required activation during elevation tasks. The techniques described in the present thesis to assess muscle function may be very suitable to identify aforementioned potential muscle preferences. The most pronounced muscle to co-activate prior to surgery is expected to result in the optimal functional improvements because this muscle already demonstrates the activation which is desired after transfer. Additionally, the non transferred muscle exposes less pathological *ad*ductor muscle co-activation, and thus restricts arm motion in a lesser degree. Differences between teres major and latissimus dorsi activation were found in their contributions to lateral rotation of the scapula (*chapter 6*). Scapula lateral rotation may have an additional contribution to pain reduction in patients with massive rotator cuff tears (Scibek et al., 2009; Scibek et al., 2008), which was found to be affected by the scapula-humeral teres major, as opposed to the thoraco-humeral latissimus dorsi. The teres major might therefore be a key muscle in preventing a painful subacromial space reduction in patients with glenohumeral cuff tears by simultaneous pulling down the humeral head and increasing lateral rotation of the scapula. However, for unambiguous judgment on patient specific muscle preferences on which muscle to transfer, a randomized clinical trial involving muscle activation assessment, scapula-humeral kinematics and clinical outcome variables should be constructed. Lost functionality of the transferred muscle and consequences for arm function should herein also be accounted for.

In conclusion, compensatory muscle activation in patients with a glenohumeral cuff tear involves enhanced deltoid activation which jeopardizes glenohumeral stability. *Ad*ductor muscle activation during arm elevation tasks can deliver caudally directed forces on the humeral head to preserve glenohumeral stability, and is therefore considered to be compensatory for lost stabilizing forces due to the cuff tear. However, such compensatory *ad*ductor muscle activation during arm elevation tasks restricts maximal arm elevation because of the additional *ad*duction moment, explaining the limitation in arm elevation in these patients. Glenohumeral stability is thus preserved at the cost of arm mobility. Tendon transfer surgery of the teres major solves this conflict between glenohumeral stability and arm mobility because the transposed teres major delivers caudal directed stabilizing forces on the humeral head, without interference with the intended elevation moment. Compensatory muscle activation is essential to take into account when treating massive rotator cuff tears patients, or other subacromial pathology related disorders, because such activation deviates from healthy subjects and affects shoulder functionality. The obtained knowledge on compensatory muscle activation potentially contributes to a better diagnosis and therapy development for shoulder patients.

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Summary

Patients suffering tendon tears in the glenohumeral cuff muscles, the deep stabilizers of the shoulder, show activation of muscles which pull the arm downwards during arm elevation tasks (*chapter 2*). This so-called co-activation deviates from healthy controls, is triggered by pain and is not the response to increased maximal arm force magnitudes (*chapter 3*), which can occur after an intervention. A potential cause for pain is glenohumeral instability, in which the subacromial tissues get painfully impinged. Goal of this thesis was to demonstrate that deviating muscle activation in patients with glenohumeral cuff tears is related to shoulder instability. We hypothesized that these deviating muscle activations are compensatory for lost glenohumeral cuff functions, and that they restrict arm functionality.

Cuff-tear simulations using a musculoskeletal model (*chapter 4*) show increased deltoid activation to compensate lost elevation moments. This increased deltoid activation jeopardizes glenohumeral stability because of the increased cranial directed destabilizing forces on the humeral head. In simulations with an isolated supraspinatus tear, lost cuff functions could be compensated for by the remaining muscles without any consequences for glenohumeral instability. Shoulder stability is endangered when multiple muscles are involved in the tear. Activation of muscles pulling down the humeral head is then required to compensate lost stabilizing muscle forces (*chapter 4* and *chapter 7*), which is counterproductive for arm elevation (*chapter 5*). There is a conflict between glenohumeral **stability** and arm **mobility**.

A salvage procedure for irreparable cuff tears is a tendon transfer of the teres major. In such surgical procedures the original teres major arm depression moment reverses to an arm elevation moment, while downwards directed stabilizing forces are preserved. Patients use their transposed teres major according to its new insertion, i.e. activation during arm eleva-

tion tasks. This solution for the conflict between glenohumeral stability and arm mobility demonstrates shoulder function improvement and pain decrease (*chapter 5*).

Increased scapula lateral rotation, outwards rotation of the inferior angle of the scapula, during arm elevation tasks is related to both pain decrease and teres major co-activation increase, as opposed to latissimus dorsi co-activation increase (*chapter 6*). This suggests, beside the primary role in counteracting instability by pulling down the humeral head, also a roll for the teres major in a pain avoidance mechanism by increasing scapula lateral rotation. Scapula lateral rotation topples the acromion, enlarging the subacromial space, potentially preventing painful subacromial tissue inclination. Such secondary effect of the teres major could be an argument in preferring the teres major over the latissimus dorsi in tendon transfer surgery for patients suffering glenohumeral cuff tears, but this requires additional comparative research.

Loading the arm with constant forces but increasing moments showed that, not only in model simulations but also experimentally lost elevation moments caused by the cuff tear can be compensated for by an increase in deltoid activation (*chapter 7*). Increased deltoid activation in model simulations resulted in shoulder instability. Both in simulations and experiments the increased deltoid activation was related to co-activation of arm depressors, the compensatory response for lost stabilizing forces.

Patients suffering glenohumeral cuff tears are well capable of compensating lost elevation moments by increased deltoid activation. However, increased deltoid activation jeopardizes glenohumeral stability. To preserve stability patients co-activation, using arm depressor muscles during arm elevation tasks. Such compensatory muscle response for stability, restricts arm mobility. The concept of compensatory muscle activation provide insight in the underlying mechanisms of patients suffering glenohumeral cuff tears and potentially can be used, also at early symptoms like in impingement, as a diagnostic instrument or it can be applied in new treatment strategies.

Samenvatting (Dutch summary)

Patiënten met een spierscheur in de diepe stabilisatoren van de schouder, de glenohumerale cuff spieren, vertonen activatie van spieren die de arm naar beneden trekken tijdens arm elevatie taken (*hoofdstuk 2*). Deze zogenaamde co-activatie is afwijkend van gezonden, wordt getriggerd door pijn en is niet het gevolg van veranderende maximale armkracht (*hoofdstuk 3*), zoals op kan treden na een interventie. Pijn is mogelijk het gevolg van instabiliteit, waarbij de weefsels in de subacromiale ruimte onder het dak van het schouderblad pijnlijk ingeklemd raken. Doel van dit proefschrift was om aan te tonen dat afwijkende spieractivaties bij patiënten met een glenohumerale cuff scheur gerelateerd zijn aan schouder instabiliteit. Onze hypothese was dat deze afwijkende spieractivaties compensatoir zijn voor verloren glenohumerale cuff functies, en dat ze armfunctie belemmeren.

Computermodel simulaties van cuffscheuren met een spier-skelet model (*hoofdstuk 4*) laten verhoogde activiteit zien van de deltoideus, een arm elevatie spier, om het door de spierscheur verloren elevatie moment te compenseren. Deze verhoogde deltoideus activatie veroorzaakt schouder instabiliteit als gevolg van een vergrootte destabiliserende omhoog gerichte kracht op de bovenarm. Bij een enkelvoudige spierscheur van de supraspinatus kan de verloren functie zonder consequenties voor stabiliteit gecompenseerd worden door de overgebleven spieren. Schouderstabiliteit komt echter in gevaar als meerdere spieren aangedaan zijn. Activatie van spieren die de arm omlaag trekken tijdens arm elevatietaken is dan nodig om verloren stabiliserende spierkrachten te compenseren (*hoofdstuk 4* en *7*), wat arm elevatie tegenwerkt (*hoofdstuk 5*). Er is een conflict tussen **stabiliteit** en **mobiliteit**.

Een mogelijke behandeling van onherstelbare cuff scheuren is een peestranspositie, waarbij chirurgisch het aanhechtingspunt van de teres major wordt verplaatst. Hierdoor wisselt

het originele arm depressiemoment van de teres major om voor een elevatiemoment, terwijl naar beneden gerichte stabiliserende krachten behouden blijven. Patiënten gebruiken de getransponeerde teres major inderdaad volgens zijn nieuwe functie, dat wil zeggen activatie tijdens arm elevatietaken. Deze oplossing van het conflict tussen glenohumerale stabiliteit en arm mobiliteit leidt aantoonbaar tot verbeterde schouderfunctie en pijnafname (*hoofdstuk 5*).

Toegenomen scapula laterorotatie, naar buiten draaien van de onderste punt van het schouderblad, tijdens arm elevatie taken is gerelateerd aan zowel pijnafname als teres major co-activatie toename, in tegenstelling tot latissimus dorsi co-activatie toename (*hoofdstuk 6*). Dit suggereert, naast een primaire rol in het tegengaan van instabiliteit door het naar beneden trekken van de bovenarm, tevens een rol van de teres major in een pijn ontwijkend mechanisme door scapula laterorotatie toename. Bij scapula laterorotatie kantelt het dak van het schouderblad, waardoor de subacromiale ruimte vergroot, wat een pijnlijke weefsel inklemming mogelijk voorkomt. Dit secundaire effect van de teres major zou een argument kunnen zijn om bij peestransposities voor patiënten met een cuff scheur de teres major te prefereren boven de latissimus dorsi, maar aanvullend vergelijkend onderzoek is een vereiste.

Door de arm te belasten met een constante kracht maar met een toegenomen moment, kon naast modelsimulaties ook experimenteel aangetoond worden dat verloren elevatiemomenten als gevolg van een spierscheur worden gecompenseerd door een toename van deltoideus activatie (*hoofdstuk 7*). Verhoogde deltoideus activatie leidde in simulaties tot instabiliteit en zowel in simulaties als experimenteel was de toegenomen deltoideus activatie gerelateerd aan co-activatie van arm depressoren om verloren stabiliserende krachten te compenseren.

Patiënten met een spierscheur in de glenohumerale cuff zijn in staat om de verloren elevatie momenten te compenseren door een toename van deltoideus activatie. Dit brengt schouderstabiliteit in gevaar. Om stabiliteit te behouden kunnen patiënten tijdens arm elevatie taken co-activeren met spieren die de arm naar beneden trekken. Het behouden van stabiliteit door deze compensatoire spieractivaties gaat echter ten koste van de armfunctie. Compensatoire spieractivaties geven ons inzicht in de onderliggende problematiek van patiënten met schouderklachten en kan mogelijk, ook bij beginnende symptomen zoals bij impingement, als diagnosticum worden toegepast of ingezet worden bij nieuwe behandelstrategieën.

Curriculum Vitae

Frans Steenbrink was born on the 19^{th} of January 1978 in the Catharina Hospital Eindhoven, the Netherlands. After graduating from the 'Christian Huygens College' in Eindhoven (HAVO-B) in 1995 he moved to Utrecht, where he started studying physical therapy in 1997. Internships were followed at Rehabilitation Center "de Hoogstraat" in Utrecht and private practice for physical therapy Odijk, before graduating in 2001.

In that same year he continued his education at the Vrije Universiteit Amsterdam, studying Human Movement Science with a specialization on Rehabilitation. A master research project entitled *determinants of hand function*, was carried out at the Amsterdam Medical Center department of Rehabilitation, supervised by prof. dr. L.H.V. van der Woude, dr. J.A. Beelen and dr. A.J. Videler. Recommended and supervised by prof. dr. H.E.J. Veeger an additional research internship was followed in the Laboratory for Neuromechanics and Kinematics of the Leiden University Medical Center, as an extended solicitation to a Ph.D.-student position under the supervision of dr. ir. J.H. de Groot. He got his masters degree in Human Movement Science early 2005. As planned, he directly followed that up with the Ph.D.-project described in this thesis, carried out in the same laboratory, with the department of Orthopaedics (Head: prof. dr. P.M. Rozing; followed up in 2006 by prof. dr. R.G.H.H. Nelissen).

In December 2009, daily after finishing his Ph.D.-project, he started working at Motek Medical in Amsterdam.

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