Compensatory muscle activation in patients with glenohumeral cuff tears
Steenbrink, F.

Citation

Version: Corrected Publisher’s Version
License: Licence agreement concerning inclusion of doctoral thesis in the Institutional Repository of the University of Leiden
Downloaded from: https://hdl.handle.net/1887/15556

Note: To cite this publication please use the final published version (if applicable).
Chapter 7

The relation between increased deltoid activation and adductor muscle activation due to glenohumeral cuff tears.

Frans Steenbrink¹,², Carel G.M. Meskers¹,³, Rob G.H.H. Nelissen¹,², Jurriaan H. de Groot¹,³

¹ Laboratory for Kinematics and Neuromechanics, Leiden University Medical Center
² Department of Orthopaedics, Leiden University Medical Center
³ Department of Rehabilitation Medicine, Leiden University Medical Center

Journal of Biomechanics, accepted for publication.
Chapter 7

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’ adductor activation, preserving glenohumeral stability. Aim of this study was to demonstrate causality between moment compensating deltoid activation and stability compensating ‘out-of-phase’ adductor muscle activation.

A differential arm loading with the same 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’ adductor 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 adductor muscle activation in cuff tear patients could be demonstrated. ‘Out-of-phase’ adductor 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 abduction moment loss. The deltoids seem to compensate for the lost abduction 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, adductor 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 adduction. Such ‘out-of-phase’ adductor 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’ adductor muscle activation. ‘Out-of-phase’ adductor 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 adductor 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’ adductor 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 adductor muscle previously demonstrated ‘out-of-phase’ adductor 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 adductor 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 adductors 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-
Compensatory muscle activation

ety of Biomechanics standards (Wu et al., 2005) was: plane of elevation $\gamma_h = 79^\circ$, elevation $\beta_h = 46^\circ$ and axial rotation $\gamma_{h2} = 31^\circ$. 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 abduction and adduction (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 adductor hierarchy**

In order to demonstrate the potential of each individual adductor muscle to compensate for glenohumeral instability, the hierarchical redundancy of adductor function was simulated, by applying models in which the active adductor 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 adductors 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±3 years) 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, 10mm electrode length, inter-electrode distance of 10mm). Between trials rest periods (minimally five seconds) were
Compensatory muscle activation

**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 adductor 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-adduction) 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.
\[ \text{AR}_{\text{muscle}} = \frac{A_{\text{IP}}^{\text{muscle}} - A_{\text{OP}}^{\text{muscle}}}{A_{\text{IP}}^{\text{muscle}} + A_{\text{OP}}^{\text{muscle}}} \quad [-1 \leq \text{AR}_{\text{muscle}} \leq 1] \]  

Where \( A_{\text{IP}} \) is ‘in-phase’ activation; \( A_{\text{OP}} \) 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 adductors were lumped in order to express the combined adductors effort. \( \text{AR}_{\text{AD}} \) thus comprehends the averaged activation ratio of pectoral is 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 adductor 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 adductor 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 abduction 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

<table>
<thead>
<tr>
<th>Moment arm</th>
<th>Small</th>
<th>Large</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>69N</td>
<td>154N</td>
<td>226%</td>
</tr>
<tr>
<td>Patientc</td>
<td>30N</td>
<td>212N</td>
<td>688%</td>
</tr>
</tbody>
</table>

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 adductor muscle *activation ratio* (*AR*<sub>AD</sub>) in patients was *AR*<sub>AD</sub> = 0.15 (SD 0.25) and did not differ from controls: *AR*<sub>AD</sub> = 0.39 (SD 0.25). At large moment loading however, the *activation ratio* in patients, *AR*<sub>AD</sub> = -0.06 (SD 0.17), was significantly lower compared to controls, *AR*<sub>AD</sub> = 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’ adductor 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 abduction forces. This concept of deltoid compensation for lost cuff abduction function (Sharkey et al., 1994) is supported by cadaver experiments (Parsons et al., 2002), our
Figure 7.4: Activation ratios of adductor 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.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Control</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>119</td>
<td>178</td>
</tr>
<tr>
<td>2</td>
<td>160</td>
<td>141</td>
</tr>
<tr>
<td>3</td>
<td>167</td>
<td>180</td>
</tr>
<tr>
<td>4</td>
<td>108</td>
<td>129</td>
</tr>
<tr>
<td>5</td>
<td>140</td>
<td>195</td>
</tr>
<tr>
<td>6</td>
<td>153</td>
<td>133</td>
</tr>
<tr>
<td>7</td>
<td>154</td>
<td>123</td>
</tr>
<tr>
<td>8</td>
<td>112</td>
<td>160</td>
</tr>
<tr>
<td>9</td>
<td>117</td>
<td>185</td>
</tr>
<tr>
<td>10</td>
<td>121</td>
<td>149</td>
</tr>
</tbody>
</table>

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 adductor 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’ adductor 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 (abductor) and the combined adductor 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 adductor 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’ adductor 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’ adductor muscle activation. This would gap the bridge between the observed ‘out-of-phase’ adductor 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 adductor 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 adductor muscle elimination showed the potential redundancy of adductor 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 adductor muscles indeed have a potential for stability compensation by ‘out-of-phase’ activation. Patients apply different combinations of adductor ‘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 deltoïd activation and stability compensating ‘out-of-phase’ adductor activation was established. Increased moment loading provoked additional deltoïd forces, compensating for lost cuff abduction moments and enclosed provoked adductor activity both in cuff tear simulation and experimentally in patients. The observed ‘out-of-phase’ adductor activation is required for glenohumeral stability. In shoulder interventions and evaluations ‘out-of-phase’ adductor activation is an indicator of glenohumeral instability.

Acknowledgements

Authors would like to acknowledge Piet Bakkenes and Jerry van der Ploeg (dept. of Instrumental Design, Leiden University Medical Center, the Netherlands) for their help with developing the experimental set-up. Jochem Nagels (dept. of Orthopaedics, Leiden University Medical Center, the Netherlands) and Peer van der Zwaal (dept. of Orthopaedic Surgery and Trauma, Medical Centre Haaglanden, the Netherlands) are acknowledged for their contribution to the patient inclusion. Peter Krekel (dept. of Orthopaedics, Leiden University Medical Center, the Netherlands) is greatly acknowledged for his support on visualization processing.