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Compensatory muscle activation in patients with glenohumeral cuff tears

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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 *adductor* muscle co-activation, which was ‘out-of-phase’ according to the muscle moment arms for arm *adduction*, i.e. *adductor* muscle activation during arm *abduction* 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’ *adductor* muscle activation to preserve glenohumeral stability (*chapter 4*). Glenohumeral stability is thus improved by ‘out-of-phase’ *adductor* 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 *adduction* moments. Active use of the transposed *adductor* (*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 *abduction* 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 *adductors* 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 decrease (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 *abduction* 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 *adductor* muscle forces pulling down the humeral head into account, as these forces decrease proximal migration.

8.4 Compensation for stability lost

Arm *adductor* muscle co-activation, simultaneous with deltoid muscle activation, and ‘out-of-phase’ with the expected activation according to the *adductor* 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 *adductor* muscle co-activation during arm elevation is a beneficial strategy. However, the large *adduction* moment arms of these muscles interfere with the intended elevation moment, restricting maximal arm elevation. ‘Out-of-phase’ *adductor* 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 *adductors* to activation during arm elevation tasks (Steenbrink et al., 2006) and to induce augmented scapula lateral rotation (Scibek et al., 2008). Arm *adductor* 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' *adductor* muscle activation, or to suppress subacromial pain (also lowering 'out-of-phase' *adductor* 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*).

adductor 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 *adductor* activation. This would result in a vicious circle of increasing ab- and *adductor* muscle activation. Muscle imbalance, involving insufficient arm *adductor* 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 *adductor* 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 *adductors* 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' *adductor* 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 *adductor* 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’ *adductor* 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 whether 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 glenohumeral 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 *adductor* muscle the stabilizing effect of its downward directed force component is preserved, without the adverse *adduction* 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’ *adductor* 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 *adductor* 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 *adductor* 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. Adductor 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 adductor muscle activation during arm elevation tasks restricts maximal arm elevation because of the additional adduction 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.