Functional implications of structural "anomalies" in shoulder pain

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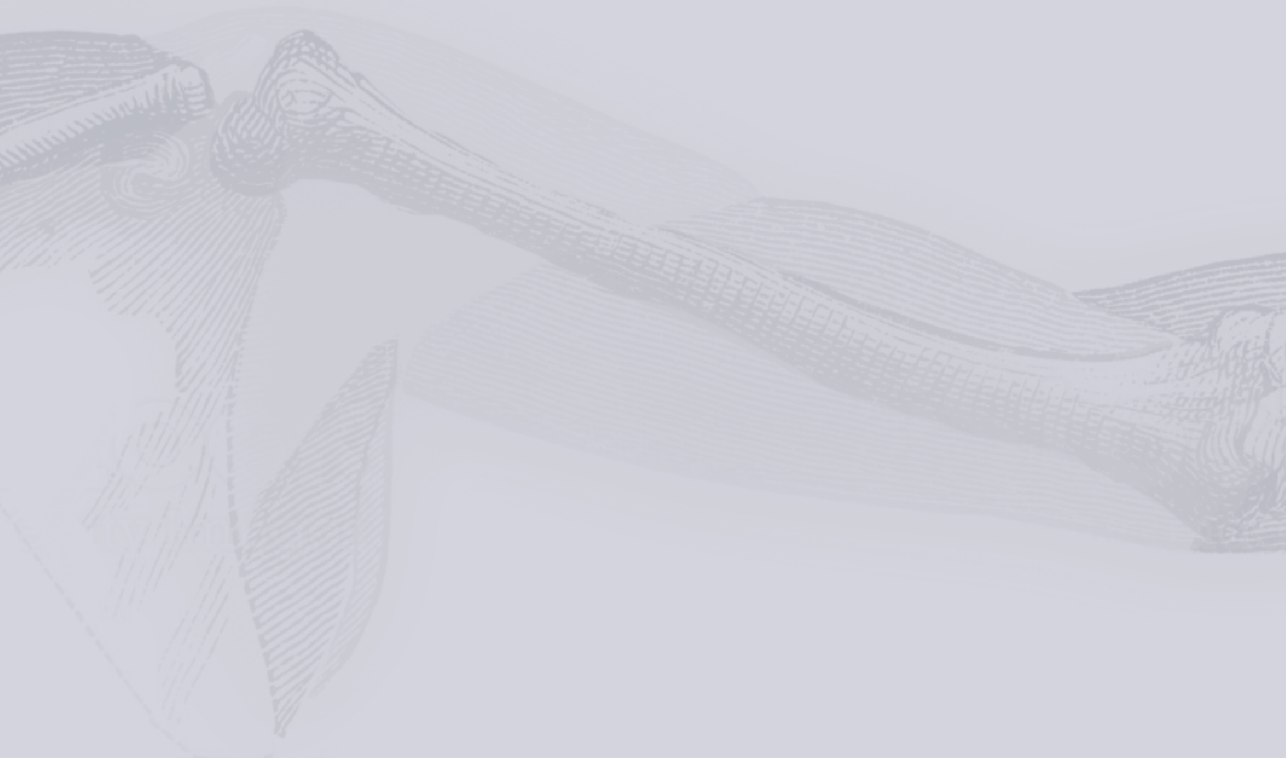

1 General introduction

SUBACROMIAL PAIN SYNDROME

Background

The shoulder girdle is essential to complete our daily life activities. It enables us to complete our tasks by positioning the hand into the space around us. Interestingly, we are usually unaware of the great mobility it requires to eat and wash, until we experience shoulder pain or discomfort. The shoulder movements are created by a complex synergy of motions of the thorax, clavicle, scapula and humerus. The bones are connected at the sterno-clavicular joint, the acromioclavicular joint, the scapulothoracic gliding area and the glenohumeral joint. The glenohumeral joint is a ball-in-socket joint that contributes to the majority of shoulder motion when elevating the arm.[8] Forces generated by scapulothoracic, humerothoracic and scapulohumeral muscles like the latissimus dorsi, teres major, pectoral major muscle, serratus anterior, deltoid muscle and the rotator cuff (i.e. teres minor, infraspinatus, supraspinatus and subscapularis muscle) all facilitate arm movements.

The shoulder joint is after the low-back the second most reported anatomic site of musculoskeletal pain in Dutch adults.[100] The prevalence of shoulder complaints is approximately 48 per 1000 person-years.[43, 94] Incidence rates are about 11 to 29 per 1000 person-years, with the highest incidence between 40 to 65 years.[7, 43, 122] The most likely origin of shoulder complaints largely depends on age. At younger age (under 35 years), shoulder complaints are frequently caused by glenohumeral instability or a shoulder sprain.[72] In patients over 35 years of age, complaints are more commonly attributed to a supposedly painful subacromial inflammation of the bursa or rotator cuff.[72, 122, 123] Interestingly, the age-dependent cause of shoulder disorders is also reflected by the prevalence of rotator cuff tears, with a prevalence of 3 percent at the 4th decade, 25 percent at the 6th decade, to over 50 percent at the 8th decade of life.[90, 116, 127]

Historical Perspective

At the beginning of the 20th century, the clinical entity and aetiology of shoulder pain was studied by several authors.[16-18, 39] According to these authors, it was evident that inflammation of subacromial structures resulted in pain with arm abduction at the anterior edge of the acromion.[16, 33, 39] As early as 1909, anatomic considerations led to the assumption that repeated mechanical impingement under the acromion could cause painful irritation of the bursa.[12, 39] In these years, Codman extensively published his personal views on shoulder pain. He reported on patient characteristics, the physical examination and symptoms which characterised the clinical entity of a supraspinatus rupture.[17] In an attempt to understand the aetiology of the painful shoulder, Codman further discussed several hypotheses explaining subacromial inflammation, while discussing attrition of subacromial tissues under the acromion as one possible mechanism for pain and rotator cuff tears.[18] Codman was not convinced that a traumatic event caused a supraspinatus rupture. He argued that an "underlying degenerative process" could make the tendon more prone to rupture.[18]

The theory of attrition of structures under the acromion was the reason for Charles S. Neer to introduce the anterior acromioplasty in 1972.[92] Neer concluded that such an impingement occurs at the anterior edge of the acromion rather than the lateral aspect of the acromion.[92, 93] Adjacent to bony morphology, the coracoacromial ligament was assumed to contribute to extrinsic compression.[49, 92] Neer distinguished three stages of "shoulder impingement syndrome": stage I associated with subacromial edema, stage II associated with a partial tear or tendinitis, and stage III associated with a rotator cuff tear.[93] The cause-effect relation of acromion morphology and rotator cuff disease was further propagated by Bigliani.[4, 5] Bigliani argued that more acromial slope was correlated to a higher prevalence of rotator cuff tears.[4] Interestingly, neither physical exam nor radiographic evaluations were able to distinguish between bursitis or a partial thickness rotator cuff tear from the presence of a rotator cuff tear according to Neer's classification.[92, 98] Authors also noticed that radiographs frequently did not show pathology which could be associated with shoulder pain.[38] Nevertheless, the subacromial impingement syndrome was now considered as one clinical entity and Neer's classification was widely accepted among orthopaedic surgeons worldwide. As a consequence, biomechanical and intervention studies studied patients with shoulder impingement syndrome from stage I to III as one entity without additional imaging of the rotator cuff to separate a tendinitis from a torn rotator cuff.[9, 10, 37, 41, 42, 44, 45, 47, 70, 75, 78, 82]

Consistent with the propagation of the attrition theory, the number of anterior acromioplasty (i.e. subacromial decompression) dramatically increased in the nineties and beginning of the twenty-first century.[60, 97, 125] During this procedure the anterolateral undersurface of the acromion was removed to flatten the anterior process of the acromion.[92] Although successful results after acromioplasty have been reported in cohort studies[6, 11, 30, 106], randomised controlled trials were unable to demonstrate the beneficial treatment effect of acromioplasty compared to physiotherapy.[9, 10, 32, 44, 45, 64-66] At the beginning of this century, the first trials were designed to detect the treatment effect of acromioplasty itself by introducing a surgical "placebo" treatment arm as control group.[36, 52] These trials did also not confirm the success of acromioplasty 2.5 years after surgery, which put the effectiveness of anterior acromioplasty into question. The findings led to alternative hypotheses regarding the aetiology of shoulder complaints and alternative diagnostic definitions of "subacromial impingement syndrome".[22, 24] Since impingement syndrome as such suggested a specific anatomic cause (i.e. subacromial attrition) for pain, the Dutch Orthopaedic Association changed the entity "subacromial impingement syndrome" to a more general term: the "subacromial pain syndrome".[29, 91]

Many intrinsic and extrinsic mechanisms have been proposed to cause subacromial pain.[3, 22, 24, 89] Long before the impingement theory was popularised, Codman already hypothesised in his classic paper of 1931, on both tendon degeneration (i.e. intrinsic mechanism) as well as anatomic variants (i.e. extrinsic mechanism) causing shoulder pain and ultimately a rotator cuff tear.[18] Later, more intrinsic mechanisms have been suggested to cause subacromial pain syndrome including: a subacromial inflammatory reaction with tendon thickening and overuse causing repetitive microtrauma.[3, 22, 24, 89] Many researchers have focused on extrinsic mechanisms causing friction of the tendon under the acromion by a reduction of the subacromial space.[3, 22, 24, 89] This reduction of subacromial volume might be caused by the os acromiale, coracoid, the coracoacromial ligament, acromioclavicular osteophytes and a hooked acromial shape.[3, 22, 24, 89] Lastly, a dynamic reduction of the subacromial space as a result of muscle weakness, causing glenohumeral instability with subsequent dynamic cranialization of the humerus under the acromion, or disturbed scapulothoracic motion (i.e. scapular dyskinesis) have been suggested to cause secondary impingement.[3, 22, 24, 89]

Pathophysiology of the subacromial pain syndrome was studied in the SISTIM project, which started in 2009. The SISTIM project aimed to identify causal mechanisms and to classify patients based on distinct pathophysiological subgroups.[24] This SISTIM project was conducted at the department of Orthopaedics and Rehabilitation from the Leiden University Medical Centre which harbours the laboratory for Kinematics and Neuromechanics. This laboratory has a long-standing track-record in studying the biomechanics and kinematics of the shoulder in a network with the Delft University of Technology and associated hospitals (Medical Centre Haaglanden, the Hague; Alrijne Hospital, Leiderdorp).[1, 2, 19, 20, 23, 25, 26, 53, 54, 80, 81, 85, 86, 112-115]

BIOMECHANICS AND KINEMATICS OF THE SHOULDER

Shoulder Biomechanics

Observations from anatomic dissection resulted in papers describing the assumed mechanics of the shoulder function.[13-15, 39] Movements of the shoulder-girdle were explained by close observations of the anatomic orientation and attachments of shoulder muscles relative to the joint.[13-15, 39] The findings in these anatomic specimens were linked to the observations in-vivo .[13, 15] The application of electromyography and radiographs in patients enabled a better understanding of the complex in-vivo interplay of the shoulder girdle structures. The introduction of radiographs illustrated that abduction was not solely initiated via glenohumeral motion when raising the arm from vertical to the horizontal, but involved movement of the scapulothoracic joint at the beginning of abduction.[35, 58, 74] Electromyographic studies revealed the activity of muscles during shoulder movement and was described in detail by Inman.[58] In these electromyographic studies, it was concluded that the middle deltoid and supraspinatus were main contributors of the abduction moment, while the infraspinatus, teres minor and subscapular muscles were identified as essential stabilizers to allow elevation and rotation of the arm.[21, 58]

Cadaveric and in-silico shoulder models gave us more insight in the requirements for shoulder motion and biomechanical adaptations that occur in case of a rotator cuff tear.[48, 57, 68, 99, 113, 117, 119-121, 124] Cadaveric models illustrated the stabilizing role of the teres minor, infraspinatus and subscapularis.[46, 48, 73, 110, 117] A supraspinatus tear caused significantly higher forces in the remaining intact rotator cuff[48, 117] and may introduce glenohumeral translations.[28, 96] A decrease in joint reaction force with excessive superior humeral head translations occurred when the tear extended in the subscapularis or infraspinatus muscle in these cadaveric models.[48, 99, 117] In line with these results, inverse dynamic simulations demonstrated comparable findings with an increase in force generated by the infraspinatus and subscapularis in case of a supraspinatus tear.[113]

A better understanding of the glenohumeral centre of rotation resulted in more complex studies on in-vivo biomechanics.[21, 102] Although the centre of rotation was considered to be slightly variable, some authors concluded that the glenohumeral joint functioned as a ball-in socket joint with approximately a fixed centre of rotation in healthy volunteers.[102] Based on this conclusion, a calculation of lever arms and force vectors around the glenohumeral joint was made.[103] Accuracy of these first estimations remained questionable, because analyses of shoulder kinematics were conducted in static biplanar test settings, while motion of the shoulder girdle occurs around three axes. Moreover, three-dimensional motion analysis advanced after defining the glenohumeral centre of rotation. Radiostereometric analysis (RSA) provided a methodology to measure three-dimensional shoulder motion. However, the in-vivo RSA research, although very accurate, has not been taken up widely for the evaluation of non-implant related shoulder research, since tantalum beads have to be inserted in the patient.[56] For that matter, other methods were developed to study shoulder motion, like the electromagnetic tracking device as the Flock-of Birds.[59, 62, 63, 77, 85, 88, 104]

Biomechanics in Subacromial Pain Syndrome

A main focus of biomechanical research in "subacromial impingement syndrome" has been the spatial shape of the subacromial space. Elevating the arm between 30 to 120 degrees of abduction brings the humerus in closer proximity to the acromion reducing subacromial space, which could explain the painful arc sign that is found in patients with subacromial impingement syndrome.[27, 40, 41, 50, 55, 61, 87, 109] However, inconsistent outcomes have been found when comparing the subacromial space in patient with subacromial impingement syndrome with asymptomatic controls. Whether the subacromial space width is reduced[28, 37, 50], not different [61, 109], or increased[23, 27] remains unclear. The latter shows the intricate interplay between dynamic cranial translation, posture, scapular rotations, elevation angle and muscle contractions on subacromial space width.[27, 28, 47, 55, 61, 76, 96, 107, 109, 111]

Important to note is that subacromial impingement syndrome evolved to subacromial pain syndrome in the Netherlands recent years, parting the attrition theory as dominant pathologic mechanism.[29, 91] In the past, patients with bursitis, tendinopathy and a rotator cuff tear have been considered as one clinical entity according to the stages of Neer's impingement syndrome for many years.[28, 41, 42, 61] The latter caused huge heterogeneity when outcomes were compared among studies, since it is very likely that patients with subacromial pain syndrome demonstrate different biomechanics and kinematics than patients with a full-thickness rotator cuff tear. Consequently, many prior studies are currently not applicable for the patient with subacromial pain syndrome (i.e. thus a patient with an intact rotator cuff).

Shoulder Kinematics

The physical examination is still an important part of diagnosing a patient. A simple observation of active shoulder motion gives us more information about the functional deficits of the patient. Next to range of motion, the clinician generally observes the scapula-humeral rhythm to determine the presence of scapular dyskinesis. The latter will have inter- and intra-observer variability, but is considered to give clinical information on the type of shoulder pathology. The importance of the scapula in shoulder movement has been acknowledged by Codman in 1911, who described a disturbed scapula-humeral rhythm, as a *"sine qua non"* for the diagnosis of a supraspinatus tear.[17]

Scapular dyskinesis is now defined as "any alteration of normal scapular kinematics"[67], but more frequently "asymmetry in scapulothoracic motion" is used in clinical practice and in literature.[118] Scapular dyskinesis, with an increase in internal rotation, a decrease in lateral rotation (i.e. also known as upward rotation) and posterior tilt are postulated to reduce the subacromial volume by bringing the humeral head in closer contact with the acromion.[34, 75, 111] Whether these observed kinematic alterations are a result of the pathophysiology of disease or a compensatory mechanism, is still part of debate.[67] Interestingly, a comparable prevalence of scapular dyskinesis in healthy volunteers and in patients with subacromial pain was found using clinicians' visual inspection.[101] This indicates a need for more robust quantitative methods to measure the direction and amplitude of small deviations of normal kinematics, like three-dimensional motion analyses. Therefore, glenohumeral and scapulothoracic kinematics have been evaluated by applying radiography, magnetic resonance imaging, opto-electronic systems or electromagnetic tracking systems.[28, 41, 79, 84, 95, 96, 99, 105, 108, 117, 126]

Patients with a rotator cuff tear were found to have reduced glenohumeral elevation and increased scapulothoracic lateral rotation to reach positions above shoulder level, thus confirming Codman's observation in 1911.[28, 84, 96, 105] Most studies had the limitation that kinematics of the shoulder had been calculated at a static elevation angle hampering the validity of these data by allowing a setting phase for the scapula.[28, 42, 95, 96] Pain was an important confounder contributing to a disturbed scapula-humeral rhythm in these patients if comparing them with healthy volunteers. The use of a suprascapular nerve block has been proposed to evaluate the effect of the supraspinatus and infraspinatus muscle on shoulder mobility in healthy volunteers by eliminating the effect of pain. Interestingly, a comparable reduction in glenohumeral elevation and increase in scapulothoracic lateral rotation was found in these simulated posterosuperior rotator cuff tears.[83]

Kinematics in Subacromial Pain Syndrome

Three-dimensional kinematic analyses in "subacromial impingement syndrome" gave contradictory outcomes between studies. Some studies found a decrease in scapular lateral rotation[31, 70, 75], while others did not find a difference[51, 69, 71, 78, 84] or even showed an increased lateral rotation.[82] A reduction in posterior tilt was found by several investigators[31, 70, 71, 75, 78] while others did not[51, 84] or even found more posterior tilt.[69, 82] These inconsistent findings are most probably related to the large heterogeneity in study populations caused by a different interpretation of physical tests among clinicians[22], treatment of patients with a different anatomic substrate of pain as one clinical entity (i.e. impingement syndrome)[70, 75, 78, 82] and investigations in highly selected subgroups based on occupation or sport activities.[69, 71, 75] For that matter, available outcomes are not translatable to the patient in the daily orthopaedic clinical practice. Shoulder kinematics in subacromial pain syndrome has to be evaluated in a group of patients with a more similar phenotype with respect to at least age and anatomy (i.e. intact rotator cuff).

In conclusion, extrinsic compression of the acromion is no longer assumed the dominant pathophysiological pathway contributing to subacromial pain. Despite attrition of the rotator cuff under the acromion may be a long-lasting process, the long-term effect of acromioplasty after 10 to 20 years is not investigated in literature. Alternative pathophysiological pathways contributing to the development of subacromial pain syndrome include a dynamic reduction of subacromial structures due to destabilizing muscle forces within the glenohumeral joint or disturbed shoulder kinematics. Therefore, there is a clear need to use biomechanical and kinematical analyses in a well-defined study population with subacromial pain syndrome.

AIMS OF THE THESIS

1) Evaluation of the long-term effects of subacromial decompression surgery on pain, shoulder function and rotator cuff integrity.
2) Evaluation of shoulder muscle activity and kinematics in patients with subacromial pain syndrome.
3) Evaluating the association of rotator cuff tear size and shoulder kinematics.

OUTLINE OF THIS THESIS

The concept of tendon attrition suggests that subacromial decompression will have an effect after many years. In **Chapter 2**, we present a long-term follow-up study of a randomised controlled trial examining the effects of arthroscopic subacromial decompression on pain, shoulder function and rotator cuff integrity 10 years after the operation. In an observational study, the kinematics and coordination of shoulder muscles in patients with subacromial pain syndrome were compared to asymptomatic volunteers **(Chapter 3)**. In **Chapter 4**, the effect of subacromial anaesthetics on scapular dyskinesis is evaluated and we elaborated on the influence of pain. The association between rotator cuff tear size and glenohumeral/scapulothoracic kinematics is investigated in **Chapter 5**. Changed mechanical loads of intact muscles not being part of the rotator cuff tear (i.e. the deltoid and teres minor muscle) were hypothesised to influence muscle atrophy with age. The alterations in mechanical loads in the shoulder in the presence of a rotator cuff tear were indirectly measured by observing changes in muscle volume **(Chapter 6)**. From here we started to investigate the effects of rotator cuff repair on shoulder kinematics (**Chapter 7**). The mid- to long-term clinical outcomes of a teres major or lattisimus dorsi tendon transfer, a salvage procedure in a chronic massive posterosuperior rotator cuff tear, are evaluated in **Chapter 8**. The study outcomes, their clinical implications and the future perspective are discussed (**Chapter 9**). Finally, a summary of findings is provided (**Chapter 10**).

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