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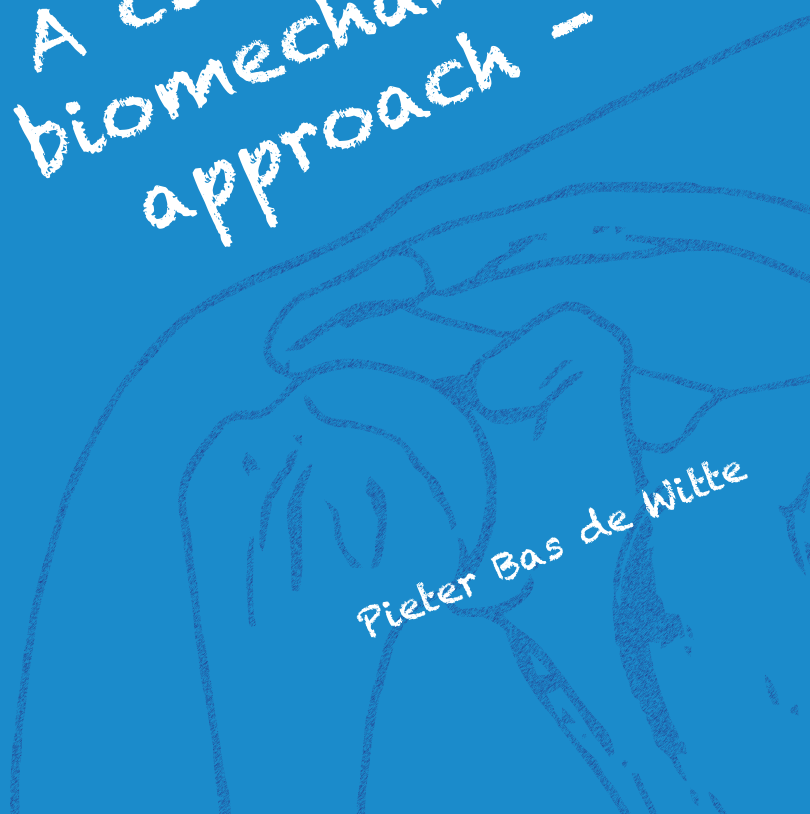


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- A clinical and biomechanical approach -



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"Pinching subacromial problems"
A clinical and biomechanical approach

Pieter Bas de Witte

PhD thesis, Leiden University Medical Center, Leiden, the Netherlands
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“Pinching subacromial problems”

- A clinical and biomechanical approach -

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CHAPTER 1

General introduction and outline of the thesis

1.1 Background

The incidence of shoulder complaints is high, with 22 per 1000 patients per year in general practice.[1] As often relatively young and active participants of society are affected, the socioeconomic impact of shoulder diseases is high.[2] 44-65% of shoulder symptoms are diagnosed as "Subacromial Impingement Syndrome" (SIS): irritation of tissues between the acromion and the humeral head.[3, 4]

The shoulder joint comprises the acromioclavicular joint, sternoclavicular joint, thoracoscapular gliding plane and the glenohumeral joint. Most of the thoracohumeral motion, i.e. arm movement with respect to the body, takes place at the latter joint. In order to achieve this large range of motion, muscles play a key role in both stabilization and mobility in the glenohumeral joint.[5] The rotator cuff muscles are generally considered as the primary stabilizer muscles of the shoulder, stabilizing the humeral head onto the concave glenoid.[6-12] Hence, pathology affecting these muscles can lead to shoulder (micro)instability and might cause shoulder pain and decreased arm function.

The rotator cuff includes the Supraspinatus, Infraspinatus, Teres Minor and Subscapularis muscles. The Supraspinatus, with its tendon situated in the limited subacromial space between the humeral head and the acromion, is most often affected in shoulder pathologies. Several mechanisms can cause e.g. tendinosis, tendinitis, calcific tendinitis and partial or full thickness tendon tears of the Supraspinatus and other rotator cuff muscles, whether or not combined with bursitis of the Bursa Subacromiale. Often, resulting symptoms are diagnosed as SIS. Impingement syndrome was introduced in 1972 by dr. Neer.[4] It can be characterized by pain with arm abduction, loss of pain free range of motion and decreased arm abduction force.[13-17] These symptoms can have a significant influence on daily activities such as putting on a coat, lying on the shoulder and overhead activities. Fortunately, these symptoms are self-limiting in many patients. However, in others, symptoms can be severe or persisting.

Over the past decades, "impingement syndrome" has evolved to the widely used diagnostic label "Subacromial Impingement Syndrome (SIS)", which (wrongfully) suggests more specificity and anatomic differentiation. There are many structures in and around the shoulder joint that can give similar symptoms, including pain with arm abduction.[18] Regardless of the underlying etiologic mechanism, treatment is generally started with activity modifications, physical therapy and/or subacromial injections with corticosteroids. When these conservative methods are unsuccessful, patients are often further investigated with e.g. radiographs, ultrasound, or magnetic resonance imaging (MRI), or are planned for surgery for the purpose of diagnostics and treatment. This is the point where pain with arm abduction becomes more

complicated and applied diagnostic and treatment pathways are highly variable in clinical practice.

1.2 Heterogeneities in pain with arm abduction, often referred to as Subacromial Impingement Syndrome

When symptoms of shoulder impingement do not improve in a couple of weeks or months, further action is needed from the treating physician. However, there is no general consensus on what steps to follow. It is unclear at exactly what moment one must take further action, which underlying mechanisms are responsible for pain with arm abduction, what additional diagnostic methods should be used to identify these mechanisms, and how to treat symptoms that often receive the diagnostic label "Subacromial Impingement Syndrome".

The classic etiologic mechanism for SIS is the mechanical extrinsic theory described in 1972: structural narrowing of the subacromial space (e.g. by a hooked acromion or the coracoacromial ligament) leads to compression and irritation of subacromial tissues.[4] Since the introduction of impingement syndrome by dr. Neer, diagnostic investigations have evolved, leading to the identification of many other causes of shoulder pain. Reported alternative mechanisms for subacromial narrowing include caudal acromioclavicular joint osteophytes and subacromial bone spurs (structural causes), or more dynamic mechanisms, such as cranial translation of the humerus, pathologic scapulohumeral kinematics, or multidirectional glenohumeral (micro) instability.[19-22] Additionally, symptoms can be caused by intrinsic mechanisms, such as tensile overload on degenerating rotator cuff tendons, leading to tendinosis or a subacromial inflammatory reaction.[23-28] Overall, these mechanisms have in common that there is a discrepancy between subacromial space and the volume needed for subacromial tissues (subacromial *impingement*).(Figure 1) However, other rotator cuff tendon problems, including calcific tendinitis and tendon tears, can cause similar symptoms.[29-34] Additionally, although SIS has been typically assumed to be the result of rotator cuff injury, the subacromial space is a complex anatomical environment, containing several structures that can be a source of pain. And also tissues outside the subacromial space can cause pain with arm abduction, complicating the differentiation of subacromial pathologies from other causes.[18]

With respect to diagnostics, several methods have been reported to be useful for SIS symptoms, including a variety of specific physical examination tests, radiographs, ultrasound evaluation and MRI, whether or not with arthrography.[35-40] However, there is little consensus on diagnostic strategies, or on criteria within these strategies, to

address shoulder pain with arm abduction.[41] Furthermore, most generally applied diagnostic tools do not differentiate between subacromial problems and other causes of shoulder pain. Despite these unclarities, there are numerous trials and reports on patients with the diagnostic label "Subacromial Impingement Syndrome". Consequently, conflicting inclusion criteria and heterogeneous patient groups are used across these studies. This is not only a possible explanation for the great variations in reported treatment outcomes, but also complicates interpretation, comparison and implication of reported results.[42]

For that matter, conservative treatment of SIS symptoms consists of a great variety of modalities and has been reported successful in 42 to 91%.[43, 44] When conservative treatments fail, the classic surgical treatment of impingement is an acromionplasty as described by Neer, even in case there actually is no hooked acromion.[45] For the surgical treatment of SIS, as for conservative treatments, great variability on "successful outcome" has been reported, varying between 48 and 90%.[46-50] Hence, in some studies, results of surgery are poor. And despite the removal of tissue from the acromion, acromionplasty does not seem to affect continuing degeneration of the rotator cuff.[51] Additionally, various studies have reported good clinical outcome in patients in whom the coracoacromial shape is not altered.[52-57] These seemingly contradicting results in both conservatively and surgically treated SIS patients ask for a better understanding of this pain syndrome and its treatment. Over the past decades, the diagnostic label "subacromial impingement syndrome" has become more controversial and is increasingly debated on in the literature. Many authors have commented on its (heterogeneous) etiologic mechanisms, complex diagnostic difficulties and variable treatment results and strategies.[3, 58-67] Some actually reject the diagnosis, regarding its symptoms as consequences of one of several possible underlying pathologies rather than as a specific condition.[18] The latest guideline of the Dutch Association of Orthopaedic surgeons advises not to use SIS as a diagnosis, but to refer to these symptoms as Subacromial Pain Syndrome (SAPS) until better terminology (i.e. with good external validity) is available, or a more specific diagnosis can be made.[68]

Summarizing, it is unclear what is generally regarded as the diagnosis "SIS" in both research and clinical practice, its etiology is heterogeneous, several mechanisms can lead to subacromial narrowing, other processes can also lead to pain with arm abduction, and reported treatment results of "SIS" vary greatly. Therefore, more research is needed to gain more insight in the underlying etiologic mechanisms of pain with arm abduction, and to develop tailored diagnostic and treatment pathways.

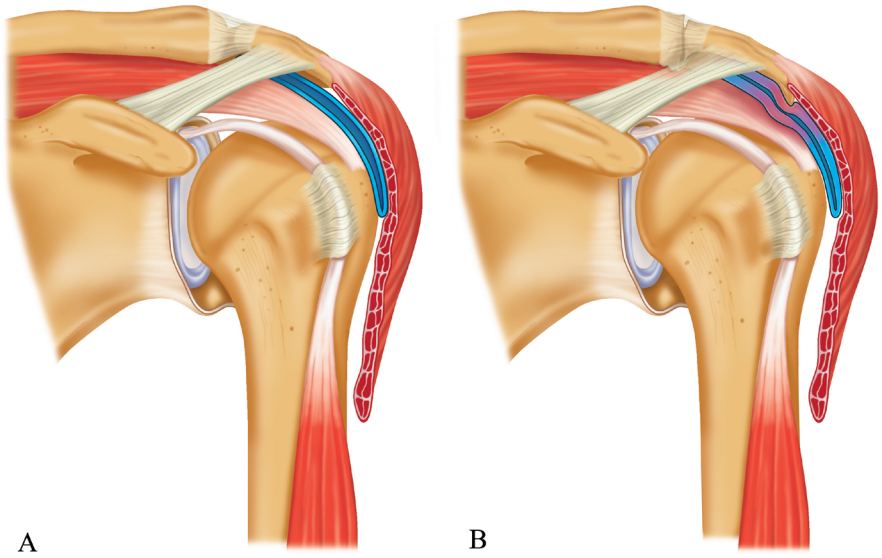


Figure 1 A) Schematic anatomy of a healthy glenohumeral joint and subacromial space. B) Schematic anatomy of a shoulder joint with the presence of several potential etiologic mechanisms for Subacromial Impingement Syndrome.
In theory, impingement ("a misbalance between acromial space and the space needed for subacromial structures") can be caused by 1) encroachment of subacromial tissues by structures (extrinsic), including a hooked acromion and acromioclavicular osteophytes; 2) a subacromial inflammatory reaction, e.g. caused by micro-trauma or overuse, causing subacromial oedema, fibrosis and tendinosis (intrinsic); and 3) a dynamically reduced subacromial space due to e.g. glenohumeral (micro)instability, or scapular dyskinesia, resulting in relative cranialisation of the humerus with respect to the acromion.

1.3 Disguised as subacromial impingement syndrome

Regarding impingement (i.e. "a misbalance between subacromial volume and the space needed for subacromial structures") as the main cause of pain and loss of force during arm abduction is questionable. As earlier mentioned, various diseases give similar symptoms as SIS, including calcific tendinitis and rotator cuff tears. Also diseases not originating from the space between humerus and acromion can give similar symptoms, such as acromioclavicular osteoarthritis, internal impingement and coracoid impingement.[29-34,69,70] Each of these diseases needs specific diagnostics and treatment strategies. So in order to optimize treatment outcome in a patient with severe or persisting SIS symptoms, underlying causes need to be identified.
Calcific tendinitis of the shoulder is the deposition of calcifications in rotator cuff tendon tissue. It is generally referred to as a self-limiting disease, but symptoms can persist for months or years in many patients. On the other hand, in 3-20% of asymptomatic shoulders, calcific depositions can be observed on radiographs.[72-75]

As for SIS symptoms in general, there is no consensus on treatment strategies of calcific tendinitis. Standard treatment is preferably conservative, including physical therapy, non-steroidal anti-inflammatory drugs (NSAIDs) and subacromial corticosteroids injections.[71, 73, 75-77] Other options are e.g. shockwave therapy, lavage and needle aspiration (barbotage) and surgical techniques, including arthroscopic removal of calcific depositions.[74, 78-87] But little is known on the effects of most of these treatments and information on the long-term prognosis of calcific tendinitis is scarce.
In rotator cuff tears, as for RC diseases in general, most often the Supraspinatus is affected. Cuff tears are generally diagnosed in subjects over 50-60 years old and are often regarded as a form of tendon degeneration that comes with age. 54% Of persons over 60 years have asymptomatic rotator cuff tears.[88] Around 50% of asymptomatic tears progress to symptomatic tears in 2-3 years, but others never become symptomatic for unknown reasons.[89, 90] Additionally, in case of symptoms, these can be self-limiting, and in case of treatment of symptomatic tears, e.g. by means of rotator cuff repair surgery, reported results are highly variable.[91]
Calcific tendinitis and rotator cuff tears seem more objectifiable causes of pain with arm abduction that can be demonstrated on radiographs and with ultrasound or MRI. But even for these two diseases, etiologic mechanisms are unclear, many cases are asymptomatic, and treatment results are highly variable. Furthermore, some regard SIS, calcific tendinitis and rotator cuff tears as forms or stages of the same pathology.

1.4 Aim of this thesis

The aim of this thesis is to unravel the clinical entity "Subacromial Impingement Syndrome" and to develop methods for identifying distinct etiological patient subgroups that need specific diagnostics and treatment strategies. To this end, we explore the terminological problems and opinions on the main characteristics of SIS amongst international health practitioners, study the prevalence of previously reported etiologic mechanisms in patients with SIS symptoms, develop clinical and biomechanical methods to evaluate and categorize patients with SIS symptoms in diagnostic subgroups, and compare treatment outcomes in trials and follow-up studies.

1.5 Setting

Over the past two decades, many research projects of the Leiden University Medical Center (LUMC) department of Orthopaedics (head of department: prof.

Rob Nelissen, MD, PhD) and its Laboratory for Kinematics and Neuromechanics (LK&N, coordinator: Jurriaan de Groot, PhD, section Rehabilitation Medicine) have focused on shoulder pathologies, in both clinical and basic research projects. During these years, fruitful collaborations have been established with the Department of Biomechanical Engineering (3ME Faculty, Delft University of Technology) and the Faculty of Movement Science of VU University in Amsterdam. This has led to the founding of the "Dutch Shoulder Group", a research group in which members from both technical and medical backgrounds collaborate, concentrating on glenohumeral loading, stability and mobility.

The basic idea of the research in this thesis originated from research projects developed by prof. Nelissen to study the high variability in the outcome of acromionplasty. One of these projects was a clinical trial with SIS patients in cooperation with the Departments of Orthopaedics of Haga Hospital and Medical Center Haaglanden.[57] This study raised many questions on the etiology and diagnosis of SIS, due to a high patient exclusion rate after MRI investigation, in combination with highly variable results of the two studied treatment methods. The results suggested there might be (etiological) SIS subgroups that need tailored treatment strategies. Consequently, we applied the experience and tools available within the Dutch Shoulder Group to further investigate patients with SIS symptoms.

The LK&N has several experimental set-ups specifically focused on shoulder research. These experimental methods enable objective assessment of shoulder function, using electromyography and electromagnetic 3-dimensional recording of shoulder kinematics.[17, 92-102] These techniques give more insight in the underlying mechanisms of shoulder pathologies and can serve as objective outcome measures in clinical and scientific investigations.

Orthopaedic departments of regional hospitals were contacted for participation in our studies, resulting in a broad collaboration in several shoulder research projects. The results of most of these studies are presented in the current thesis.

1.6 Outline of the Thesis

This thesis is divided into three parts. In the first part, the clinical definition of "SIS" is investigated. Additionally, this part focuses on clinical and diagnostic heterogeneities, several entities, diagnostic problems and various treatments of SIS symptoms. The second part focuses on the development of new biomechanical and patient reported outcome measures to assess patients with SIS symptoms. In the third part, results of the studies in this thesis are summarized and discussed, and a patient work-up protocol is presented.

Part 1A starts with ***Chapter 2,*** a questionnaire study, investigating views on SIS in clinical practice, amongst physical therapists and shoulder surgeons from the United States and the Netherlands. In ***Chapter 3*** activations of the Deltoid and Supraspinatus muscles are investigated in healthy subjects, using abduction force tasks and electromyography. ***Chapter 4*** describes MRI arthrography findings in patients clinically diagnosed with SIS by expert shoulder surgeons. The diversity of MRI findings these patients is discussed and linked to etiologic subgroups for SIS symptoms. In ***Chapter 5***, a Randomized Controlled Trial on two surgical treatments of SIS is presented.

Where Part 1A elaborates on the definition and heterogeneities of SIS, **Part 1B** contains two chapters on calcific tendinitis, a diagnosis that can be often made after further investigation of patients with SIS symptoms. In ***Chapter 6***, a large group of patients diagnosed with calcific tendinitis is investigated with clinical questionnaires, to assess long-term outcome and prognostic factors. In ***Chapter 7,*** two treatment methods for calcific tendinitis are compared in a Randomized Controlled Trial.

In **Part 2**, patient reported outcomes (PROMs) and biomechanical methods are presented that can help in the evaluation of SIS patients with objective and validated measures, and in the identification of potential patient subgroups. ***Chapter 8*** describes a comprehensive validation study of a relatively new disease specific patient questionnaire for rotator cuff problems: the Western Ontario Rotator Cuff index (WORC).[103] This clinical score has been applied in the clinical evaluation of the patients in chapters 6 and 7 as well. In ***Chapter 9,*** a new method for assessing pathologic adductor muscle co-activation (electromyography) is introduced, that has potential value as a practical measure for identifying etiological subgroups. In ***Chapter 10***, this new method is applied and compared in SIS patients, patients with a rotator cuff tendon tear and healthy subjects. Additionally, the relation between adductor co-activation and subacromial narrowing is investigated. An alternative electromyography outcome measure is presented in ***Chapter 11***, reporting on Deltoid function before and after surgery in patients with a rotator cuff tear.

Finally, **Part 3** shows a systematic biomechanical and clinical work-up plan for patients with SIS symptoms in a study protocol for future investigations (***Chapter 12***), a general discussion (***Chapter 13***) and a conclusion with future perspectives (***Chapter 14***).

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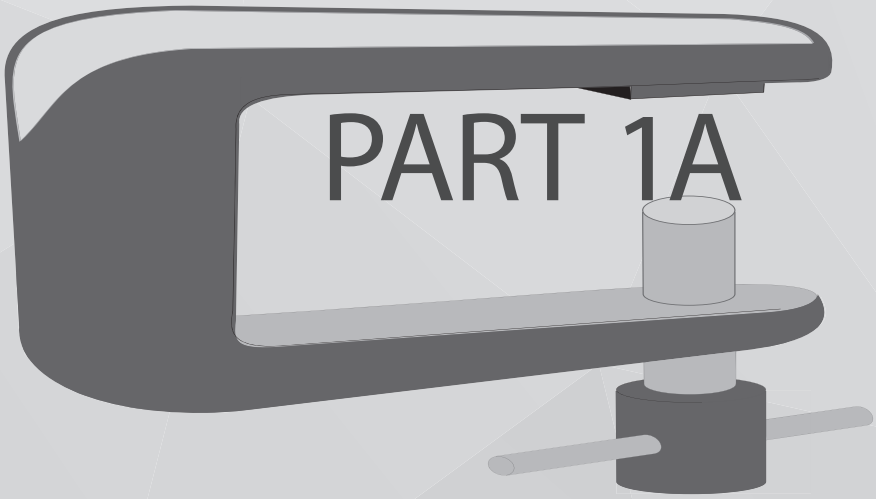
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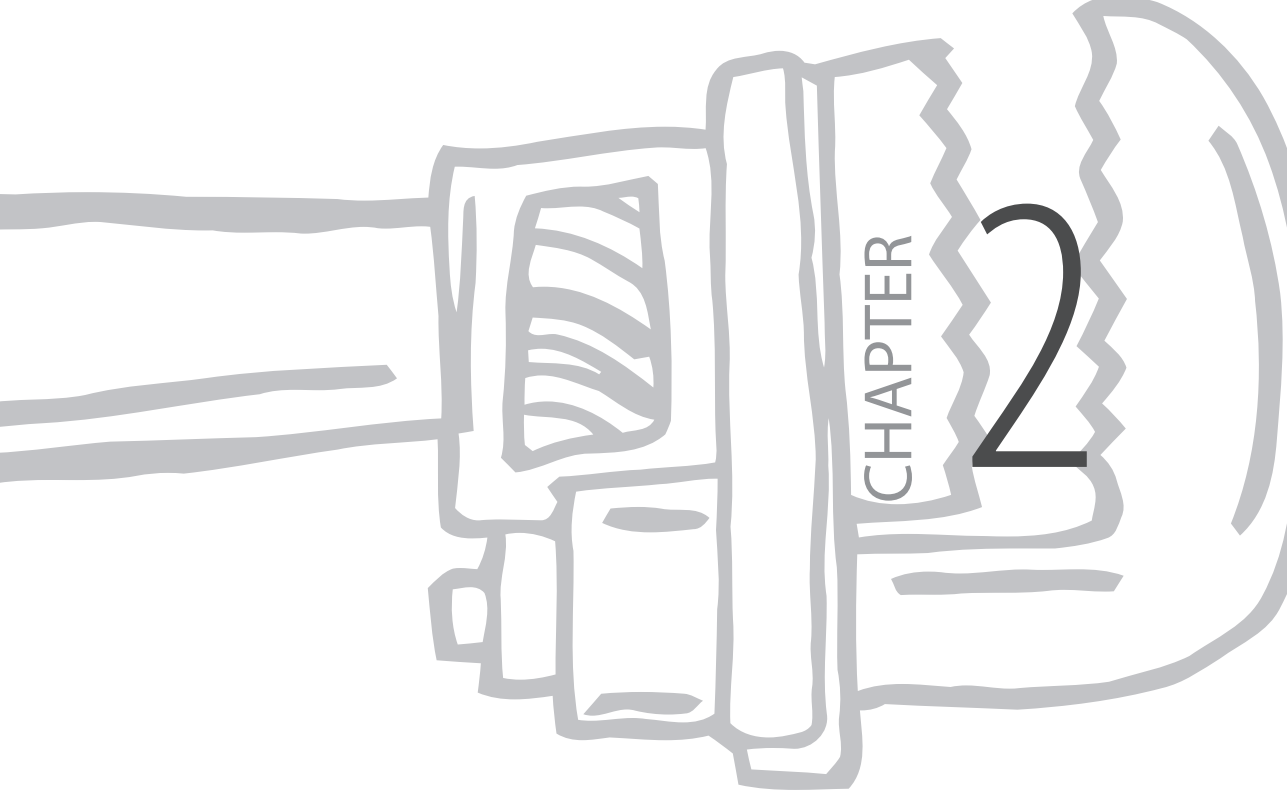
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PART 1A

What is Subacromial Impingement Syndrome?





Communication Breakdown: Clinicians Disagree on Subacromial Impingement

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Abstract

"Subacromial Impingement Syndrome (SIS)" is often used as a diagnostic label, but has become more controversial as such in the literature. We assessed views on SIS in clinical practice, using a survey with 63 0-to-10 VAS items amongst orthopaedic surgeons and physical therapists from the US and the Netherlands. Multivariate regression and cluster analyses were applied to identify consensus items and to study profession and/or nationality effects on item ratings.

Most items received neutral or highly variable ratings. 29 Were considered associated with SIS, including worsening of pain with overhead activities, painful arc and a positive Neer's test. 7 Items were regarded pleading against SIS, including loss of passive motion. Activity modifications and physical therapy are the most important treatments according to therapists, who highly valued motion related etiologic mechanisms. Surgeons, with higher ratings for intrinsic and anatomic etiologies, appreciated the use of subacromial corticosteroids and surgery.

Clinicians from different professional backgrounds have different views on what SIS is and even within professional groups, variations are substantial. This has to be taken into account when communicating about SIS symptoms e.g. in intercollegial consultation or scientific research. The authors suggest cautious use of (subacromial) impingement *syndrome* as a diagnostic label.

Keywords: Shoulder Impingement Syndrome; Rotator Cuff; Diagnosis; Treatment; Consensus

1. Introduction

A syndrome is the association of several clinically recognizable features, signs, symptoms, phenomena or characteristics that often occur together. The term syndrome is generally used for a combination of findings if the underlying pathogenesis is unknown or heterogeneous. As such, Dr. Neer introduced impingement syndrome of the shoulder in 1972.[1] In his articles on impingement in 1972 and 1983, several stages and underlying mechanisms were reported.[1, 2] However, over the past decades, "impingement syndrome" has evolved to the widely used diagnostic label "Subacromial Impingement Syndrome (SIS)" in both literature and clinical practice, which (wrongfully) suggests more specificity and anatomic differentiation. Nowadays, SIS is reported as the most prevalent disorder of the shoulder in primary health care, accounting for 44-65% of all shoulder complaints.[3, 4] But is there consensus on this SIS diagnostic label? In this study, we investigated how orthopaedic shoulder surgeons (OS) and physical therapists (PT) from the Netherlands and the United States use and define SIS in clinical practice.

SIS is usually referred to as symptomatic irritation of the rotator cuff (RC) and subacromial bursa in the limited subacromial space and is characterized by shoulder pain that worsens with arm abduction, decreased pain-free active Range of Motion (RoM) and loss of abduction force.[3-6] Over the past decades, SIS has become more controversial and increasingly debated on in the literature. Many authors have commented on (heterogeneous) underlying mechanisms, complex diagnostic difficulties and treatment strategies involved in this syndrome.[4, 7-15] Some reject SIS as a diagnosis, regarding its symptoms as consequences of several possible underlying pathologies rather than a specific condition.[16]

While there are many explanations that have been suggested for SIS symptoms, the extrinsic mechanism as described by Neer in 1972 is often referred to: symptoms are caused by structural narrowing of the subacromial space (e.g. by the acromion), leading to compression and irritation of subacromial tissues.[1, 2] Other etiologic mechanisms have been described as well, including subacromial narrowing due to humeral proximal migration (cranial translation), caudal acromioclavicular joint osteophytes and subacromial spurs, as well as glenohumeral (GH) (micro-) instability and scapular dyskinesia. Intrinsic mechanisms such as micro-trauma or primary degenerative tendinopathy of the RC tendons have also been proposed.[4, 12, 15, 17-22] Alternative forms of shoulder impingement have also been described, including coracoid and internal impingement, with similar symptoms as SIS.[20, 23] Lastly, some regard calcific tendinitis as a form of SIS, where others regard this as a distinct diagnosis.

Several diagnostic methods have been reported valuable in "diagnosing" SIS and excluding other causes of shoulder pain. These include a variety of specific physical examination tests, radiographs, ultrasound evaluation and Magnetic Resonance Imaging (MRI).[21, 24-28] However, there is little consensus on diagnostic strategies, or on criteria within these strategies, to address and further specify SIS symptoms.[16, 29] All this notwithstanding, there are numerous trials and reports on patients with the diagnostic label "SIS". Hence, conflicting inclusion criteria and heterogeneous patient groups are used across these studies, complicating interpretation and comparisons of reported results.[30]

Besides varying opinions on the etiology and diagnostics of SIS symptoms, no consensus on treatment exits either. In general, initially conservative treatment is started, including activity modifications, physical therapy, subacromial injections and Non-Steroidal Anti-Inflammatory Drugs (NSAIDs). If this fails, often operative treatment (i.e. acromionplasty, as based on the extrinsic etiologic theory) is proposed, with success rates ranging from 48-90%.[31-34] These highly variable results are stressed even more by good clinical outcome reported in patients in whom the coracoacromial shape is not altered.[35-40] This asks for a better understanding of SIS symptoms, and better diagnostic strategies to identify the probable different phenotypes of shoulder pain that are often diagnosed as SIS. As a first step, the use and definition of the term SIS in clinical practice needs to be clarified.

Despite the debate on SIS as a diagnosis in the literature, little is known on the use of SIS in clinical practice. We used an online survey with 63 items (related to SIS in the literature) to investigate views on etiology, diagnostics and treatment of SIS, amongst shoulder orientated clinicians from various backgrounds (orthopaedic surgeons and physical therapists from the Netherlands and the United States). Our goal was to clarify how various definitions for SIS are used in clinical practice that potentially complicate efficient intercollegial communication, by 1) assessing general variability in opinions on SIS amongst health practitioners specialized in shoulder pathology; 2) assessing potential systematic differences in views between clinicians from various regional and professional backgrounds; and 3) identifying factors associated with SIS that surgeons and physical therapists agree upon internationally.

2. Methods

2.1 Questionnaire

An online survey was composed by an expert panel of 2 local orthopaedic shoulder surgeons (JR, RN) of the Leiden University Medical Center and the investigating researcher (PBdW), in cooperation with a local questionnaire design expert and

two local physical therapists. All members of the expert panel are involved in patient care and research of RC pathologies. Relevant survey items were obtained from the expert panel, key informant interviews with local faculty members of the orthopaedics and physical therapy departments, and both classic and recent publications on SIS. [1-4, 9, 10, 12, 15, 16, 19, 21, 22, 24, 28, 41, 42]

All members of the expert panel agreed on the final set of questions, comprising 63 items in 8 categories.(Tables 1-4) Participants were asked to evaluate these items on SIS symptoms in general, regardless of one's profession and regardless of primary/secondary or internal/external classifications, forms of SIS, or underlying mechanisms.

1) ***Etiology and Causes***. The importance of 8 items frequently associated with the etiology of SIS, was assessed by ratings on 0-to-10 Visual Analog Sores (VAS-) scores: "What is the role of each of the following factors in the etiology of SIS? Please rate between 0 'not important' and 10 'very important'. Neutral: rate 5."
2) ***Patient Characteristics.*** Six basic patient characteristics often reported as either SIS risk factors or factors contraindicating SIS, were valued on 0-to-10 scales: "Please rate between 0: strongly pleads against SIS, and 10: strongly pleads for SIS. Neutral: rate 5."
3) ***Patient history and symptoms reported by the patient.*** Three patient history items and 5 patient-reported symptoms typically related to SIS in clinical practice and literature were valued on 0-to-10 scales: "Please rate between 0: strongly pleads against SIS, and 10: strongly pleads for SIS. Neutral: rate 5."
4) ***Physical Examination***. Using similar 0-to-10 VAS-scales, 7 frequently applied methods of physical examination of the shoulder with regard to SIS symptoms were assessed for their association with SIS.
5) ***Imaging and Surgical Investigations.*** Nine routinely applied investigations for shoulder pathologies were rated on 0-to-10 VAS-scales with regard to their importance in the diagnostic process of SIS symptoms: "Investigations that need to be performed to exclude other pathologies and/or to diagnose SIS. Please rate 0: absolutely not needed, 5: helpful, 10: essential."
6) ***Negative findings***. Eleven findings that have been reported as either suggestive for stages or forms of SIS or, in contrast, for other pathologies that exclude SIS were assessed on a 0-to-10 VAS-scale: "The following items have been reported as negative findings, suggesting other pathologies than SIS. Please rate 0: strongly pleads against SIS, 10: strongly pleads for SIS. Neutral: rate 5."
7) ***Alternative diagnoses***. Using 0-to-10 VAS scales, participants were asked to rate the frequency with which 9 related pathologies around the shoulder might be confused with SIS by health professionals not specialized in shoulder pathology (0: never, 5: sometimes, 10: very often."

8) ***Treatment***. The importance and efficacy of the 5 most frequently used treatments for SIS were rated on 0-to-10 VAS-scales: "Importance of the following factors in the treatment of SIS: please rate between 0=not important, and 10=very important."

2.2 Participants

The international participants from The Netherlands (NL) and the United States (US) in this study were selected from clinical shoulder specialists in one of two disciplines: orthopaedic surgery, or physical therapy. Orthopaedic surgeons (OS) were required to hold postgraduate qualifications in relevant shoulder related specialties, or be members of interest groups relevant to the study. All Dutch OS (OS_{NL}) were members of the Dutch Shoulder Workgroup of the Dutch Orthopaedic Association and all OS_{US} were contacted via the society of American Shoulder and Elbow Surgeons (ASES). Physical therapists (PT) in The Netherlands (PT_{NL}) were required to be specialized in shoulder pathology and to have attended national or international PT Shoulder Courses. Physical therapists in the US (PT_{US}) were orthopaedic physical therapists routinely treating patients with shoulder pain, identified via national shoulder courses and the American Society of Shoulder and Elbow Therapists.

2.3 Procedure

A link to the online survey was e-mailed to all identified candidates at their working address, obtained from their appropriate organizations. Subjects who didn't send a response were reminded after 4 weeks and 8 weeks. Subjects indicating they were unavailable for this study, or who hadn't replied after the reminders were excluded.

2.4 Statistical analysis

Total group and subgroup (n=4) means and Standard Deviations (SD) of the 63 items were used for general evaluation. Items were ordered within each category in tables 1-4, using the total group means (high to low).

To assess potential systematic influence of nationality (NL or US) or profession (PT or OS) on item ratings, multivariate regression analyses were performed with item ratings as dependent variables and nationality, profession and the interaction term of nationality and profession as independent variables. Bonferroni correction was applied, accounting for the number of items in each category and the number of independent variables.

With cluster analyses, statistical methods are used to assign objects to groups (clusters). Each cluster contains objects that are more similar to each other than to objects in the other clusters. For this study, 2 cluster analyses (Ward's method[43]) were applied. The first assessed whether participants' ratings on all items are

deducible to participant subgroups with similar combinations of item ratings. If these analyses would lead to e.g. 4 clearly distinguishable clusters of participants with similar answers on items and if each cluster is mainly composed of either OS_{US}, OS_{NL}, PT_{US} or PT_{NL}, this would indicate that participants' backgrounds systematically relate with opinions on the assessed aspects of SIS. Chi-squared analyses, or Fisher's exact tests where appropriate, were applied to study proportions of practitioners of the 4 professional backgrounds (OS_{US}, OS_{NL}, PT_{US} and PT_{NL}) in identified clusters. A second cluster analysis was performed to identify clusters with generally low or high item ratings to obtain clusters of items that most participants agree upon with regard to their association with SIS.

Analyses were processed using SPSS 16.0 software (SPSS Inc., Chicago, Illinois) and R 2.10.0 Statistics software (R Foundation, Vienna, Austria). P-values <0.05 were considered statistically significant.

3. Results

3.1 Response of participants

For the Dutch clinicians, 70 OS_{NL} and 73 PT_{NL} were invited for participation. Two OS_{NL} replied they did not want to contribute, since the diagnosis "SIS" is considered obsolete in their practice. Final response rates were 39% (n=27) and 36% (n=26), respectively. For the US clinicians, 37 OS_{US} and 47 PT_{US} were invited. Four OS_{US} expressed they did not want to participate, since the diagnosis "SIS" is considered obsolete in their practice. Final response rates were 57% (n=21) for OS_{US} and 45% (n=21) for PT_{US}.

Mean ratings for all items for all the 4 background groups are depicted in Tables 1-4, ordered from high to low mean scores, accompanied by p-values for effects of profession and nationality obtained from multivariate regression analyses.

3.2 Etiology

RC overuse or micro-trauma, degenerative RC tendinopathy, shoulder muscle weakness and pathologic scapulohumeral(SH)-rhythm were generally highly rated.(Table 1) For pathologic SH-rhythm and glenohumeral (GH) (micro)instability, there were significant effects for both nationality and profession. For SH-rhythm, ratings were slightly higher for Dutch practitioners and PT. GH (Micro)instability was primarily considered an important mechanism by PT ($p<0.001$), especially for PT_{US}. In contrast, OS_{US} rated GH (micro)instability as an item pleading against SIS.(Table 1) Shoulder muscle weakness ($p<0.001$) and cranial humerus translation ($p=0.02$) were rated both higher as a cause by PT compared to OS.

3.3 Patient characteristics

Heavy or overhead work is considered to be strongly associated with SIS according to a majority of the participants, with significantly higher ratings by PT (p=0.04). (Table 1) Other patient characteristics were considered of little importance .

3.4 Patient history and symptoms reported by the patient

General consensus was observed on the association of SIS with worsening of pain during overhead activities, a previous episode with similar symptoms, shoulder pain in the Deltoid region and pain at night or after activities, with high mean ratings and no significant differences between groups. Shoulder pain in the Deltoid region was significantly higher rated by PT (p=0.003).(Table 1) A history of GH (micro)instability (symptoms of laxity or actual dislocations) and recent trauma were considered as factors pleading for SIS by PT, but OS_{US} rated these items as a finding rejecting SIS. For both items, effects of profession and nationality were significant.

3.5 Physical examination

A painful arc, a positive Neer's impingement test and a positive Neer or Hawkins sign were considered highly associated with SIS by all groups. Additionally, resolving pain, stiffness and/or weakness after a subacromial injection with anaesthetics strongly pleads for SIS according to PT_{US} and OS, with a p-value of 0.05 for profession effect. PT and OS_{NL} considered a positive empty can test as a finding suggesting SIS, in contrast to OS_{US} (p-value country effect: 0.052).(Table 2)

3.6 Imaging or Arthroscopic Investigations

On average, none of the imaging and arthroscopic investigations in the diagnostic processes of SIS symptoms received ratings higher than 7.0, but US participants rated arthroscopy as a significantly more important modality for diagnostics compared to Dutch health professionals (p=0.03). OS found anteroposterior radiographs in internal or external rotation (p<0.001) and Supraspinatus outlet views (p=0.01) significantly more useful than PT.(Table 2)

CT and CT-arthrogram for diagnosing SIS, were considered as of low importance (mean scores 2.0 and 2.3, respectively) according to most participants.

3.7 Negative findings

US participants significantly rated negative findings with lower mean scores (more pleading against SIS) than Dutch participants for neck pain (p<0.001), shoulder muscle atrophy (p=0.01), numbness/tingling in arm or fingers (p<0.001), and RC calcific tendinitis with imaging (p<0.001). Additionally, there was a significant profession effect for the latter two items.(Table 2)

A global loss of passive range of motion or a positive Apprehension test disinclined most participants to consider SIS without any significant country or profession effects.

3.8 Confusing other diagnoses

In contrast to PT, OS reported that full-thickness RC tendon tears can be easily mistaken for SIS by physicians not specialized in shoulder pathology (p=0.02). For the other alternative diagnoses, opinions were variable within and between groups without any statistically significant profession or country effects.(Table 3)

3.9 Treatment

According to PT, the most important treatment strategies are modification of activities and physical therapy, with mean scores ranging from 8.5 to 9.8, significantly higher than OS scores (p=0.01 and p=0.03), but also OS scored high ratings for these items.(Table 4) Subacromial injections with corticosteroids were significantly higher rated by OS (p<0.001).

3.10 Cluster analyses

The first cluster analysis focused on identifying clusters of clinicians rating questionnaire items in a similar way. The resulting cluster diagram contained 4 main clusters of clinicians.(Table 5) Fisher's exact test revealed that the proportions of participants of the 4 studied groups were statistically significantly different in the 4 clusters (p<0.0001): each cluster had its own corresponding background group that was predominantly represented. Cluster 1 included predominately PT_{US} and a subgroup of PT_{NL}, while cluster two was composed of a group of PT_{NL} and a subgroup of OS_{NL}. Cluster 3 predominately included OS_{US} with some OS_{NL}, and Cluster 4 included predominately OS_{NL}. So participants' national and professional background systematically relate with their opinions on the assessed aspects of SIS. In clusters 1 and 2, the on average highest rated etiologic mechanisms were a pathologic SH-rhythm and shoulder muscle weakness. In clusters 3 and 4, the highest rated etiological mechanisms were degenerative RC tendinopathy and overuse or microtraumata. With regard to the two most important treatments, physical therapy was highly rated in all clusters. Additionally, modification of activities scored high in clusters 1 and 2, versus subacromial injections with corticosteroids in clusters 3 and 4.

The second cluster analysis focused on identifying clusters of questionnaire items that all participants valued in a similar way. Five main clusters could be identified containing items that were either generally positively rated (cluster 1), negatively rated (cluster 2), rated around 5.0, or indecisively rated (two clusters).

There were 22 items in cluster 1, and 7 in cluster 2 (highlighted in Tables 1-4 in italics and bold italics, respectively). With regard to the 22 items related with SIS according to most participants, there were still profession and/or nationality effects for 8 factors: shoulder muscle weakness, pathologic SH rhythm, cranial translation of the humerus, laborious or overhead work, shoulder pain in the Deltoid region, physical therapy, modification of activities and subacromial injections. Similarly, there were profession and/or nationality effects in 3 of the 7 negative items: for neck pain, numbness/tingling and shoulder muscle atrophy.(Tables 1-4)

3.11 Additional comments of participants

24 (51%) PT and 11 (23%) OS added comments in the online questionnaire. Despite filling out all questionnaire items, one OS stated that SIS is an obsolete diagnosis and that acromionplasty will have disappeared in a couple of years. Others stated that SIS is operated on far too often. Additionally, some remarked that acromioclavicular osteoarthritis and SIS always come in pairs, or that SIS, partial RC tears and full-thickness RC tears are stages of the same disease, as originally reported by Neer. One OS stated that suprascapular nerve entrapment can lead to similar symptoms as SIS, but it needs other treatment strategies. With regard to shoulder motion, opinions varied. Some OS state that SIS leads to or can be the consequence of stiffness, e.g. internal rotation deficits. Others commented that limited passive RoM excludes SIS. Several PT suggested additional etiological mechanisms for SIS, including a shortened posterior glenohumeral joint capsule, pathologic cervicothoracic motion patterns, labrum lesions, scapular tilt, clavicular kinematics, shoulder muscle recruitment impairments and rotated ribs. In diagnostics, various forms of altered scapulohumeral motion patterns were mentioned and the use of electromyography was suggested by some PT. Pain with palpation of the acromion was suggested as a positive sign with physical examination. Similarly to OS, opinions on the role of motion in the etiology and diagnostics of SIS varied. Lastly, several PT commented that therapy depends on patient characteristics: in most patients physical or manual therapy is important and the indications for injections and surgery depend on age and other factors.

4. Discussion and conclusions

The results of our study show that the use and interpretation of the term Subacromial Impingement Syndrome vary greatly amongst orthopaedic surgeons and physical therapists from the Netherlands and the United States. Participants with similar professional background and nationality systematically rated items in a more

similar way, but overall, ratings on 34 (54%) items were neutral or highly variable. There appeared to be consensus on the association of the remaining 29 items with SIS. Many participants associated some (but not all) classic aspects with SIS, including Deltoid region pain, night pain, pain with overhead activities, painful arc, positive Hawkins test and a positive Neer's test. So in summary, in spite of some agreement, there were systematic differences between OS_{US}, OS_{NL}, PT_{US} and PT_{NL}, as well as high variabilities within each of these groups with regard to opinions on etiology, diagnosis and treatment of SIS. This underlines that it is problematic to use Subacromial Impingement *Syndrome* as a specific diagnosis.

The etiology of SIS symptoms is unclear. In many publications, SIS is regarded as a specific pathology or diagnostic label that can be treated conservatively or surgically. It has been typically associated with irritation of the rotator cuff under an 'impinging' coracoacromial arch, but definitions of SIS vary between articles. Furthermore, several authors reported other mechanisms, not all subacromial, leading to SIS symptoms, or even other pathologies with similar history, pain patterns and physical examination findings which can be mistakenly diagnosed as SIS.[4, 12, 14, 15, 20-22, 29, 44] This was confirmed in a recent study at our institution as well: 17.5% of patients clinically selected for surgical treatment of SIS had to be excluded following MRI arthrography because of specific other shoulder pathology.[40] Over the past decade, SIS has increasingly been described as a set of symptoms, comprising several possible subacromial pathologic processes, instead of a specific diagnostic label, i.e. a *syndrome*.[16, 29] It is unclear how the debate on SIS in literature reflects on the definition of SIS as applied in clinical practice. In this first study investigating the use and interpretation of SIS in clinical practice, views on etiology of SIS were highly variable both between and within groups of physical therapists and orthopaedic surgeons from the US and The Netherlands. Most participants considered overuse or micro-trauma of the rotator cuff as important etiologic factors. In addition, therapists emphasized motion-related causes, while surgeons primarily regarded degenerative tendinopathy as an important factor. With regard to the most classical etiologic mechanism, a hooked acromial shape, opinions on its association with the etiology of SIS were highly variable. Generally, PT more or less agreed on a neutral role for acromial shape. For OS however, 18 (37.5%) rated acromial shape with scores $\geq$7.0 as an important etiologic factor. Oppositely, 17 (35.4%) rated acromial shape with scores $\leq$3.0. This might indicate a shift in the interpretation of the pathophysiological cause of SIS as not purely based on the classic extrinsic mechanism.

The observed variations in beliefs on SIS in the etiology items were depicted in the other categories as well, with profession and/or nationality effects in 15 (27%) items. There was generally no consensus on diagnostic investigations or patient

characteristics, besides heavy or overhead work activities. Overall, most (classic) patient history and symptom items were highly rated. With regard to physical examination and investigations, painful arc, Neer's test and Hawkins sign are important examinations according to all participants, but surgeons also appreciated resolving pain after a subacromial injection, radiographs or even MRI as important items in the diagnostic process of SIS. US participants were stricter on negative findings: neck pain, numbness/tingling, muscle atrophy and calcific tendinitis with imaging modalities, generally exclude SIS. Lastly, modification of activities and physical therapy are important treatments according to most participants and PT in particular, who highly valued motion related etiologic mechanisms. Shoulder surgeons however also appreciated the use of subacromial corticosteroids injections and surgery.

The reported differences on SIS between clinicians from different or even from similar backgrounds have consequences for the communication between e.g. the orthopaedic surgeon and the physical therapist in clinical practice. Most interesting is the difference in opinions between motion-related underlying mechanisms (i.e. scapulohumeral rhythm, muscle weakness, cranial translation of the humerus, glenohumeral (micro)instability and shoulder stiffness) and the diagnosis of SIS. PT were much more likely to find that these mechanisms were consistent with SIS, while OS did not. This may reflect back on the training of PT to monitor and treat movement disorders and for OS to treat anatomic lesions. To a surgeon, SIS seems to be more of an intrinsic, anatomical or structural problem which can be treated with corticosteroids or surgical alteration. It appears from these data, that the same term "SIS" is being used by therapists to describe movement disorders including stiffness, (micro)instability, and scapular dyskinesia. These types of language differences can cause challenges with communication between providers and complicate patient care. Moreover, it has consequences for the interpretation of OS and PT literature on SIS. Variations in the definition of "SIS" may be an important reason for the inconsistent inclusion and exclusion criteria that are used across publications on SIS.[16] It is plausible that conclusions in these studies are based on results of patients with varying etiologic mechanisms or varying pathologies diagnosed as SIS, resulting in the wide variety on views with regard to etiology, diagnosis and treatment of SIS that exists nowadays.

Since the introduction of impingement *syndrome*, diagnostic tools and knowledge on shoulder diseases have improved, leading to the identification of many etiologic mechanisms and pathologies that can cause SIS symptoms. However, this has not lead to a change in the definition or clinical specification of SIS symptoms. To the contrary, many clinicians and researchers regard subacromial impingement syndrome as a specific diagnosis. In our opinion, the etiology of SIS symptoms is

heterogeneous and therefore, what is commonly called "SIS" is a combination of findings and symptoms that can be caused by various underlying mechanisms. Successful treatment outcome largely depends on identifying the concerning underlying mechanism(s) and implying this knowledge in clinical decision-making. Over the past years, various authors suggested the development and clinical use of specific discriminating tests, flow charts and treatment regimes for patients with SIS symptoms.[8, 20, 21, 44] Despite there is a lack of randomized controlled trials on surgical and conservative treatment of SIS,[9] it is advisable to focus more on the identification of potential etiologic subgroups and patient specific diagnostics and treatment pathways in future research projects on SIS symptoms.

Our study has some limitations that should be taken into account when interpreting the results. First, as for all (international) surveys, answers given depend on the formulation and interpretation of the questions. To minimize this, all items were rated on similar 0-to-10 VAS scales (ranging from "not important" or "strongly pleading against SIS", to "very important" or "strongly pleading for SIS") with similar questions for each category. Additionally, participants were instructed to rate all items regardless of their profession, primary/secondary or internal/external SIS classifications, or SIS subtypes. Secondly, the response rates were moderate. This can partially be explained by subjects who don't regard SIS as a diagnosis and therefore decided not to participate in this questionnaire study. However, this would actually underline the importance of our study. The information with the survey indicated that it included and extensive list of 63 items, which could have daunted clinicians to participate. Furthermore, there is little evidence on inferior results or lower accuracy of questionnaire studies with relatively low response rates. Thirdly, observed differences between surgeons and therapists from 2 countries, might be partially the consequence of discrepancies in patient populations and scientific bias. For example, shoulder (micro)instability can be interpreted as younger patients with (hyper)laxity causing (secondary) impingement, or as actual glenohumeral dislocations, depending on regional or professional background. However, this doesn't devaluate the importance of the observed differences in views on SIS amongst health practitioners. Lastly, our study only investigated opinions of shoulder surgeons and physical therapists specialized in shoulder pathologies. Nonetheless, most patients with shoulder symptoms are initially treated by general practitioners. Between and even within groups of shoulder specialists, we found that opinions on SIS vary greatly. It is therefore highly plausible that opinions will be even more variable amongst general practitioners. However, research on the use and definition of SIS amongst general practitioners is needed to gain more insight in this.

The results of our study show that in clinical practice, clinicians from different professional (surgeon or physical therapist) or regional (US or Dutch) backgrounds have different views on SIS and even within these subgroups, variations are substantial. This lack of consensus, existing in both literature and clinical practice, has to be taken into account when communicating about SIS symptoms in e.g. intercollegial consultation, or in interpreting and conducting research projects. Only when we are using the same definitions for terms, we can truly communicate effectively. Therefore, the authors suggest cautious use of (subacromial) impingement *syndrome* as a diagnostic label. We encourage the use of more precise diagnostic language whenever possible when using the term SIS in research or clinical practice.

Legend

	All (n=95)		PT_{NL} (n=26)		PT_{US} (n=21)		OS_{NL} (n=27)		OS_{US} (n=21)		Multivar. p-values	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Country	Profession
Etiology & causes												
Overuse / microtraumata of RC	7.6	1.7	7.9	1.5	8.0	1.3	7.6	1.6	7.1	1.9	1	1
Shoulder muscle weakness	7.3	2.1	8.0	1.6	8.9	1.0	6.8	1.8	5.8	2.4	0.49	**< 0.001**
Degenerative tendinopathy of RC	7.2	2.2	6.8	2.2	7.4	1.8	7.6	2.2	7.3	2.4	1	1
Pathologic SH-rhythm	7.1	2.3	8.4	1.3	7.5	2.0	7.3	2.1	5.3	2.6	**0.04**	**0.01**
Cranial translation humerus	6.9	2.4	7.6	1.9	7.8	1.9	6.8	2.4	5.6	2.7	1	**0.02**
Glenohumeral instability	6.0	2.6	6.7	1.8	7.7	1.7	6.6	2.2	3.0	2.4	**< 0.001**	**< 0.001**
AC-osteoarthritis / osteophytes	5.7	2.4	6.1	2.1	6.2	1.8	6.2	2.2	4.4	2.8	0.33	0.41
Acromial shape	5.4	2.8	6.1	2.5	6.0	2.5	5.4	2.7	4.4	3.2	1	1
Patient Characteristics												
Labourious or overhead work	7.5	1.6	7.5	1.4	8.4	1.1	7.5	1.2	6.8	2.1	0.50	**0.04**
> 65 y.o.	6.2	2.4	5.8	2.2	6.3	2.5	6.3	2.4	6.6	2.4	1	1
Female gender	5.2	1.7	5.5	1.5	5.1	1.2	5.8	2.1	4.3	1.4	0.06	1
DM disease	5.0	1.9	5.5	1.8	5.0	1.7	5.3	1.9	4.2	2.1	1	1
> 3 hrs/week sports	4.3	2.0	3.9	2.0	5.6	1.8	4.0	1.9	4.0	1.7	0.22	0.6
< 35 y.o.	3.4	2.3	3.7	2.1	4.5	2.2	3.2	2.5	2.1	1.6	1	0.07
Patient History and Symptoms												
Worsening with overhead activities	8.3	1.3	8.5	1.2	8.7	0.9	8.3	1.2	8.0	1.4	1	1
Shoulder pain, deltoid region	7.5	1.6	8.2	1.3	8.2	1.3	7.3	1.6	6.6	1.7	1	**0.003**
Previous episode similar symptoms	7.4	1.5	7.5	1.0	7.8	1.0	7.5	1.9	6.8	1.7	1	1
Shoulder pain, at night or after activities	7.1	1.9	7.0	1.9	7.4	1.8	7.6	1.9	6.8	1.7	1	1
Shoulder pain, anterosuperior	6.7	1.9	6.5	2.3	7.3	1.8	7.1	1.7	5.9	1.5	0.52	0.52
Loss of power arm/shoulder	5.8	1.9	6.1	1.6	6.7	2.1	5.8	1.7	4.8	2.0	1	0.09
Hx of shoulder instability	5.4	2.6	7.1	1.6	6.3	2.1	5.3	2.0	2.6	2.2	**< 0.001**	**< 0.001**
Recent trauma / accident	5.2	2.4	6.9	1.7	5.0	2.3	5.3	1.9	3.3	2.3	**0.003**	**0.006**

Table 1. The associations of items in the first three categories with SIS (etiology, patient characteristics, patient history and symptoms), according to orthopaedic surgeons and physical therapists from The Netherlands and the US.
Means and SD's of 0-10 VAS scores (0 meaning not related to SIS, 10 meaning strongly related to SIS) for all participants and for the 4 professional groups are depicted. Scores are ordered from high to low mean scores within each category, accompanied by p-values for effects of profession and nationality obtained from multivariate regression analyses after Bonferroni correction.
Items grouped in cluster 1 of the cluster analyses (suggestive for or strongly associated with SIS) are highlighted in italics. There were no items of cluster 2 (factors not strongly related to or pleading against SIS) in these 3 categories. (Hx: History)

	All (n=95)		PT_{NL} (n=26)		PT_{US} (n=21)		OS_{NL} (n=27)		OS_{US} (n=21)		Multivar. p-values	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Country	Profession
Physical examination												
Painful arc	8.1	1.5	8.3	1.3	8.8	1.4	8.0	1.3	7.4	1.7	1	0.09
Positive Neer's impingement test	7.7	2.0	7.6	1.8	7.6	2.0	8.0	1.7	7.7	2.4	1	1
Resolvement pain / stifness after SAI	7.6	1.8	6.7	1.6	7.5	1.8	8.3	1.3	8.1	2.2	1	0.05
Positive Neer or Hawkins sign	7.6	2.1	7.6	2.1	7.4	2.3	8.2	1.7	7.4	2.2	1	1
Positive empty can test	6.9	1.9	7.5	1.1	7.2	1.9	7.3	2.1	5.5	1.8	0.05	0.13
Scapular dyskinesia	6.2	2.2	7.4	1.9	5.9	1.8	6.1	2.2	5.1	2.2	0.10	0.60
Positive O'Briens test	4.1	2.2	4.9	2.7	4.0	2.2	4.4	2.0	3.1	1.6	0.87	1
Investigations												
CT	2.0	2.0	3.1	2.5	1.9	1.8	1.4	1.6	1.2	1.3	1	0.07
CT-arthrogram	2.3	2.3	3.4	2.8	1.8	1.7	1.9	2.1	2.0	2.2	1	1
Arthroscopy	3.7	3.1	3.7	3.0	2.8	2.6	2.8	2.9	5.8	3.1	**0.03**	0.08
Radiographs: Axial view	3.7	2.7	2.6	2.7	2.8	1.9	4.3	2.7	4.9	3.0	1	0.10
MRI-arthrogram	4.5	2.5	4.6	2.6	4.8	2.6	4.2	2.5	4.5	2.7	1	1
Radiographs: AP int. &ext. Rotation	4.6	3.0	3.0	2.8	3.6	2.3	6.9	2.7	4.6	2.5	0.19	**< 0.001**
Ultrasound	4.7	2.5	4.8	2.5	3.6	2.8	6.0	2.1	4.1	2.2	0.13	1
Radiographs: Supraspinatus Outlet view	4.8	2.9	3.0	2.7	4.6	2.5	5.7	3.1	6.0	2.4	1	**0.01**
MRI	5.3	2.3	4.8	2.2	4.9	2.1	4.8	2.5	6.9	1.9	0.14	0.35
Negative findings												
Numbness/tingling in arm or fingers	2.8	2.6	2.8	2.4	1.4	1.8	5.0	2.1	1.2	1.9	**< 0.001**	**0.05**
Positive Apprehension test	2.9	2.4	3.8	2.8	2.3	1.6	3.1	2.5	1.7	1.6	0.16	1
Global loss of passive RoM	3.1	2.7	3.9	2.7	3.0	2.8	3.0	2.8	2.4	2.3	1	1
Neck pain	3.2	2.1	3.9	2.0	2.6	1.8	4.1	2.1	1.5	1.5	**< 0.001**	1
Possitive AC-compression test	3.4	2.2	4.2	2.6	3.1	1.7	3.3	2.1	2.6	2.1	1	1
Shoulder muscle atrophy	3.6	2.6	4.0	2.8	3.4	2.4	4.7	2.6	1.9	1.7	**0.01**	1
AC-osteoarthritis with imaging	4.3	2.0	5.2	1.9	4.2	1.5	3.7	2.0	3.8	1.9	1	0.25
Positive Yocum test	4.5	2.0	5.0	1.8	4.5	2.1	4.3	2.0	4.2	2.2	1	1
Full thickness RC# with imaging	5.3	3.1	5.3	2.9	5.4	3.1	5.7	3.3	4.8	3.3	1	1
RC calcific tendinitis with imaging	5.7	2.8	7.3	1.7	5.6	2.5	6.6	2.7	2.8	2.1	**< 0.001**	**0.02**
Partial thickness RC# with imaging	6.4	2.0	6.6	1.6	6.7	1.8	6.5	2.2	5.6	2.1	1	1

Table 2. The relation of physical examination and diagnostic modality items with SIS, according to orthopaedic surgeons and physical therapists from The Netherlands and the US.
Means and SD's of 0-to-10 VAS scores (0 meaning not related with SIS, 10 meaning strongly related to SIS), overall and for the 4 professional groups.
Items grouped in cluster 1 of the cluster analyses (suggestive for or strongly associated with SIS) are highlighted in italics and those in cluster 2 (factors not strongly related to or pleading against SIS) are highlighted in bold italics.

	All (n=95)		PT_{NL} (n=26)		PT_{US} (n=21)		OS_{NL} (n=27)		OS_{US} (n=21)		Multivar. p-values	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Country	Profession
Confusing alternative diagnoses												
RC tear (partial)	6.8	2.5	6.2	2.7	5.6	2.9	7.0	2.1	8.3	1.7	1	0.30
Calcific Tendinitis	6.2	2.6	5.6	2.5	5.0	2.7	7.5	2.0	6.4	2.7	1	0.08
RC tear (full)	5.6	2.7	4.8	2.6	4.4	2.8	6.1	2.3	7.1	2.4	1	**0.02**
Adhesive Capsulitis	5.5	2.9	4.3	2.7	5.4	2.9	6.3	2.6	6.2	3.1	1	0.47
Isolated AC-osteoarthritis	5.0	2.6	3.9	2.7	4.4	2.2	6.0	2.4	5.6	2.4	1	0.08
Shoulder (GH) instability	5.0	2.7	4.5	2.6	6.3	2.9	5.4	2.4	3.8	2.7	0.22	0.13
Degenerative Joint disease	4.7	2.3	4.1	2.2	4.2	2.0	5.1	2.5	5.1	2.6	1	1
Cervical Radiculitis	4.0	2.4	2.8	2.4	3.8	2.0	4.4	2.0	5.0	2.6	1	0.28
Nerve compression	3.3	2.3	2.5	2.2	2.6	1.9	3.6	1.7	4.2	2.9	1	0.35

Table 3. Alternative diagnoses that can mistakenly be diagnosed as SIS by health practitioners not specialized in shoulder pathologies.
Means and SD's of 0-to-10 VAS scores (0 meaning never confused with SIS, 10 meaning very often confused with SIS), overall and for the 4 professional groups.
There were no items of cluster 1 (suggestive of or strongly related to SIS) or 2 (factors not strongly related to or pleading against SIS) in this category.

	All (n=95)		PT_{NL} (n=26)		PT_{US} (n=21)		OS_{NL} (n=27)		OS_{US} (n=21)		Multivar. p-values	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Country	Profession
Treatment												
Physical therapy	8.6	1.8	8.8	1.1	9.8	0.5	7.5	2.5	8.6	1.4	0.20	**0.01**
Modification of activities	7.9	2.0	8.5	1.6	8.9	1.2	7.3	2.5	7.1	2.0	1	**0.03**
Surgery	6.8	2.5	6.4	1.8	6.2	3.5	6.3	2.9	7.3	2.7	1	1
SAI	6.7	2.5	5.2	2.4	5.9	1.9	8.0	1.8	7.6	2.6	1	**< 0.001**
NSAID's	6.3	2.2	6.3	1.8	5.3	2.8	6.5	1.7	6.9	2.6	1	0.67

Table 4. The role of most common methods in the treatment of SIS.
Means and SD's of 0-10 VAS scores (0 meaning not important in the treatment of SIS, 10 meaning highly important in the treatment of SIS) for items in the first three categories for all participants and for the 4 professional groups.
Items group in cluster 1 of the cluster analyses (suggestive for or strongly associated with SIS) are highlighted in italics. There were no items of cluster 2 (factors not strongly related to or pleading against SIS) in this category.

	Professional group				
	PT_{US}	PT_{NL}	OS_{US}	OS_{NL}	**Total**
Cluster					
1	15	11	1	2	29
2	3	12	0	6	21
3	2	2	17	8	29
4	1	1	3	11	16
Total	21	26	21	27	95

Table 5. Proportions of each of the four professional groups (US and NL Orthopaedic Surgeons (OSUS and OSNL), US and NL Physical Therapists (PTUS and PTNL)) in 4 clusters as identified with Ward's cluster analyses on all questionnaire items.
With cluster analyses, statistical methods are used to assign objects to groups (clusters). These groups contain objects that are more similar to each other than to objects in the other groups.
Fisher's exact test demonstrated that the proportions of practitioners of the 4 professional groups were statistically significantly different in the 4 clusters ($p<0.0001$). Each cluster had another corresponding professional group that was predominantly represented, so participants' national and professional background systematically relate with their opinions on the assessed aspects of SIS.

Acknowledgements

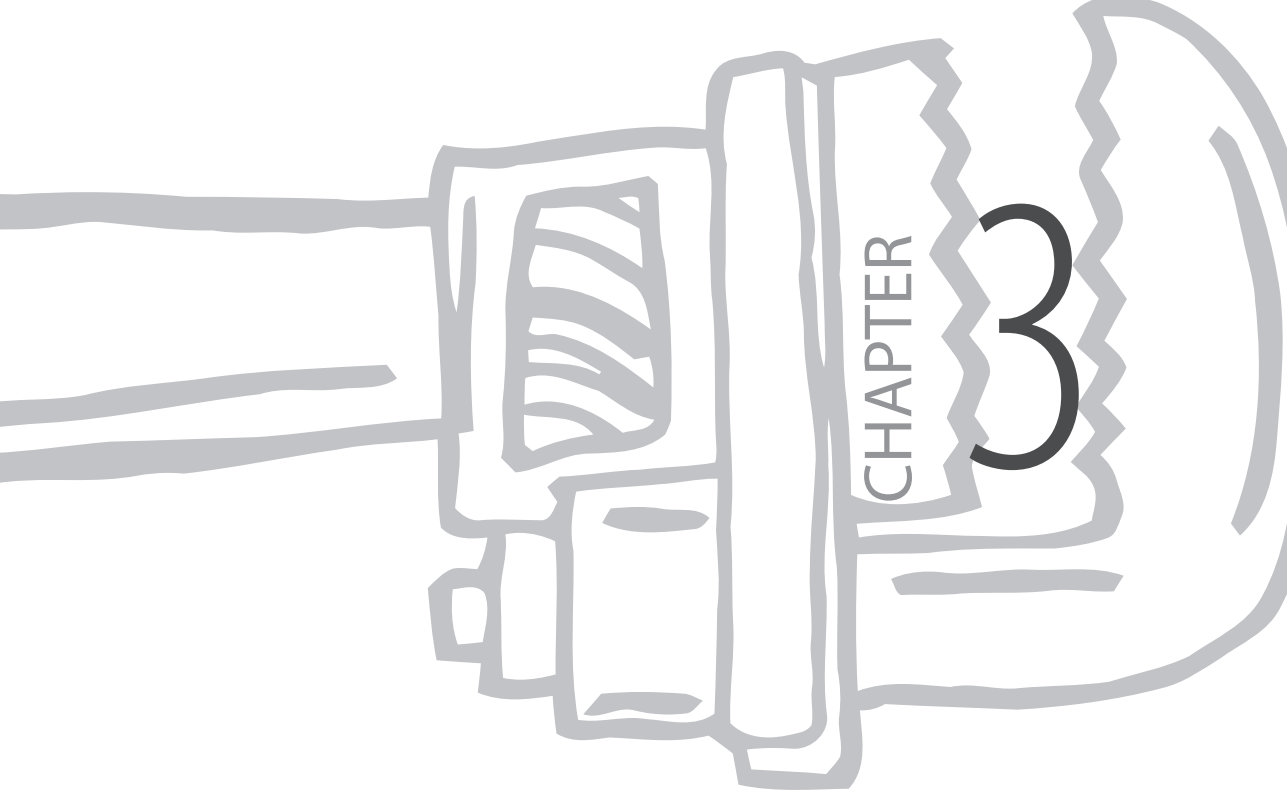
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The Supraspinatus and the Deltoid - Not just two arm elevators -

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Abstract

Background: The debate on the clinical and functional role of the Supraspinatus in relation to the Deltoid necessitates experimental assessment of their contributions to arm elevation. Our goal was to evaluate the responses of both muscles to increased elevation moment loading.

Methods: Twenty-three healthy volunteers applied 30N elevation forces at the proximal and distal humerus, resulting in small and large glenohumeral elevation moment tasks. The responses of the Deltoid and Supraspinatus were recorded with surface and fine-wire electromyography, quantified by (EMG_{distal}–$EMG_{proximal}$), and normalized by the summed activations (EMG_{distal}+$EMG_{proximal}$) to R_{Muscle} ratios.

Results: Deltoid activity increased with large elevation moment loading (R_{DE}=0.11, 95%-CI [0.06-0.16]). Surprisingly, there was no significant average increase in Supraspinatus activation (R_{SSp}=0.06, 95%-CI [–0.08-0.20]) and its response was significantly more variable (Levene's test, F=11.7, p<0.001). There was an inverse association between the responses (β = –1.02, 95%-CI [–2.37-0.32]), indicating a potential complementary function of the Supraspinatus to the Deltoid.

Conclusions: The Deltoid contributes to the glenohumeral elevation moment, but the contribution of the Supraspinatus is variable. We speculate there is inter-individual or intra-muscular function variability for the Supraspinatus, which may be related to the frequently reported variations in symptoms and treatment outcome of Supraspinatus pathologies.

Keywords: Rotator Cuff; Supraspinatus muscle; Deltoid muscle; Electromyography; Fine-wire; Abduction; Coordination

1. Introduction

The Supraspinatus is a rotator cuff muscle that is frequently affected in shoulder diseases. Supraspinatus tendon tears have a high prevalence and often affect active members of society.[1, 2] Its consequences are most apparent during active arm abduction and elevation, expressed in pain and loss of arm force ranging from 0% to over 50%.[3, 4] However, 54% of persons over 60 years have asymptomatic Supraspinatus tears, eventual symptoms are often self-limiting and reported treatment results in patients with shoulder pain and Supraspinatus tears vary greatly.[3, 5-7] More insight in its function is needed to gain understanding of these clinical variabilities.

The Supraspinatus has been described as important in two aspects. Firstly, the Supraspinatus is active during arm abduction and contributes to glenohumeral elevation moments, although Deltoid has been reported to be the largest contributor.[8-14] The Supraspinatus and the Deltoid also seem to have a complementary role during arm elevation: Supraspinatus knock-out studies, by nerve blocking[14] or in Supraspinatus tendon tear patients[15] showed increased compensatory Deltoid activation of >50% during elevation tasks compared to controls. Secondly, the Supraspinatus has been reported to play a primary role in stabilizing the glenohumeral (GH) joint. The Supraspinatus, as other rotator cuff muscles, can press the humeral head against the concave glenoid, with its compressive muscle line of action and relatively small muscle moment arm.[16-22] Symptomatic Supraspinatus tears in combination with consequent increased (compensatory) Deltoid activation have been related to cranial translation of the humerus (or superior migration of the humeral head) during arm elevation[23] underlining a complementary role of the Supraspinatus in glenohumeral stabilization.

To get a clearer view on the potential role of the Supraspinatus as an elevation moment generator, we determined the response of the Supraspinatus to changes in elevation moment loading in healthy subjects, while keeping the force component constant. We compared this with the response of the Deltoid, using a similar set-up as applied by Steenbrink et al. for studying Deltoid function in cuff tear patients and healthy controls.[15] We hypothesized that an increase in moment loading of isometric elevation tasks with a constant force magnitude, would lead to an increase in activation of both the Deltoid and the Supraspinatus, assuming that both muscles act as elevation moment generators.

2. Methods

Subjects isometrically exerted 30N arm elevation forces alternately at the proximal and distal humerus, with the arm fully supported in a splint. This resulted in isometric tasks with a small and large moment arm of external glenohumeral loading, respectively. Task force magnitude and direction were controlled for, similar to a previous experiment with the same set-up.[15]

2.1 Subjects

The number of subjects required was derived from a study with the same set-up (Steenbrink et al. 2010). An average increase in Deltoid EMG was observed of 35% (SD=22) in healthy subjects when increasing elevation moment loading. We defined a difference of 15 percentage points between the increase in activation of the Supraspinatus and Deltoid as a relevant difference. Using Altman's nomogram, this gives a standardized difference of 1.36. With a significance level of 0.05 and a power of 80%, this leads to 18 subjects. Accounting for a 25% drop-out rate, we included 23 patients.

Hence, twenty-three healthy volunteers were recruited. Inclusion criteria were: age 18-50 years, thus assuring a low prevalence of eventual asymptomatic cuff tears, and informed consent. Exclusion criteria were: current shoulder complaints, any history of shoulder disease, or other pathologies with potential influence on muscle or shoulder function. Before inclusion, a physical therapist or a physician took the subjects' medical history with regard to the shoulder.

From twenty-three subjects participating in the study, one subject had to be excluded from the analyses due to recording difficulties. The final study group comprised 12 (54%) females and 10 (44%) males with an average age of 27 years (range, 21-43). Four (17%) were left-hand dominant.

This study was approved by the accredited institutional Medical Research Ethics Committee, according to the Medical Research Involving Human Subjects Act. Of 21 included subjects, limited data on Deltoid activation patterns have been published earlier.[24]

2.2 Experimental Procedure

A changing arm load was applied to study the (re)distribution of the activation of the Deltoid and the Supraspinatus muscles. In order to support our (unconventional) experimental setup, it is important to stress the fact that the mechanical equilibrium requires a balance in both moments and forces. Our goal was to identify the response of the Deltoid and the Supraspinatus on moment increase, while keeping the external force constant, to assess their roles in generating glenohumeral moments as needed for arm motion. Therefore, we applied the experimental set-up

as used previously for evaluation of Deltoid activity during changes in arm moment loading in cuff tear patients and healthy controls.[15] In this set-up, the arm is fully supported (no gravitational force on the arm, no offset moments and forces) and the GH- moment is altered by changing the force moment arm of a constant task force. The Deltoid muscle was proven responsive for moment increase of isometric elevations tasks in this set-up previously and the set-up has also been applied in various other shoulder EMG studies.[15, 25-30] With regard to the current study, both the Supraspinatus and Deltoid are generally assumed to generate arm elevation moments, although they contribute differently to the GH force equilibrium. This experimental set-up should be able to show the contribution of the Supraspinatus relative to the Deltoid muscle to GH-elevation moments.

Subjects were seated with their dominant arm in a splint. The arm was positioned in a standardized posture as applied in the relatively long history with this set-up in our laboratory. Within the 'functional' plane of humerus elevation, i.e. 30° horizontal arm abduction, an elevation angle of 60° is a critical angle that the majority of the rotator cuff patients can just reach actively without (excessive) pain. The humerus was 45° internally rotated, which was assumed to be about the neutral axial rotation for this humerus orientation. The subjects were instructed to maintain their arm in this position

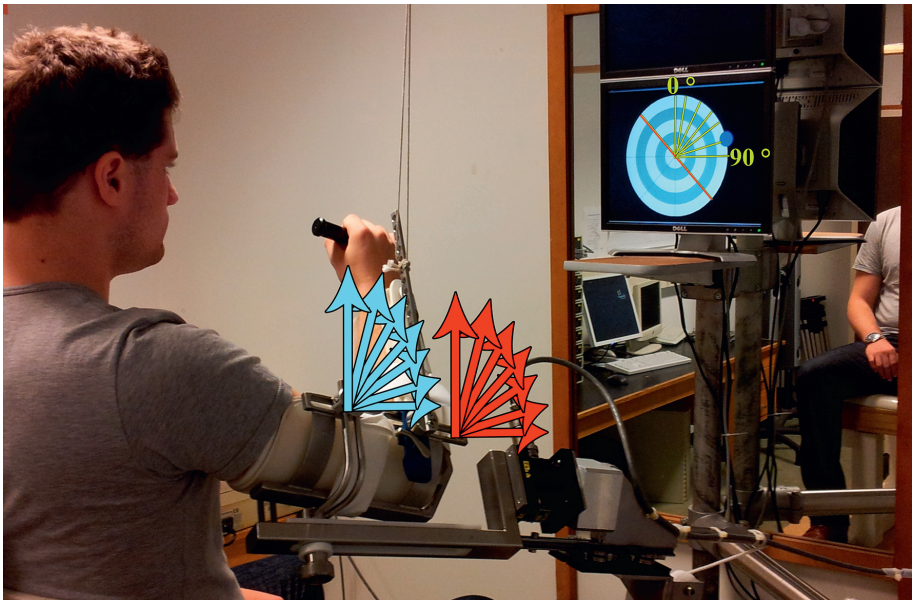


Figure 1. Experimental set up for isometric force tasks (30N) with small and large arm elevation moment loading, as described in section 2.2.
The light blue arrows indicate small moment loading. The point of force application can be changed on the arm-side (splint) and on the sled, resulting in large moment loading (dark red arrows).
Using visual feedback, the subjects are asked to move a red dot to a blue target in 7 successively and randomly applied elevation directions of equal force magnitude, as indicated on the screen. The red oblique line on the screen represents the direction of the lower arm (45 degrees of internal rotation of the humerus).

during the experiment, so tasks were performed isometrically. The splint was attached to a force sensor (AMTI-300, Advanced Mechanical Technology Inc., Wavertown, MA, USA) with only two degrees of freedom fixed: horizontal and vertical translations perpendicular to the arm.(Figure 1) The three rotational degrees of freedom and the forward backward translation along the humerus longitudinal axis were not constrained. As the arm was fully supported, hardly any moments and forces were needed to control for this specific arm position, which was required for isometric measurements of muscle activities during force tasks.

The splint allowed for two different points of task force application alongside the humerus in order to realize small and large GH elevation moment arms of the external force. For the small moment arm condition, the point of force application was about 10 cm distally from the GH joint, for the large moment arm condition, this point of force application was moved to approximately 25 cm distally from the GH joint.

By applying forces onto the transducer, subjects could move a cursor on a computer screen to twenty-four successive target positions over a range of 360 degrees. For this study, we focused specifically on isometric tasks that are theoretically primarily generated by the Deltoid and Supraspinatus: forces away from the midline or sagittal plane of the body and in the scapular plane, which we defined as isometric elevation moment tasks. The concerning task force targets represented 30N force vectors in seven equidistant elevation directions (15 degrees apart, perpendicular to the humerus), ranging from 0 degrees (push arm straight up) to 90 degrees (push arm sideward), with the arm 45 degrees internally rotated.(Figure 1) The target positions appeared randomly on the screen and subjects brought the cursor to each target and held it within each target area for two seconds. After a practice round, two task series were performed: one while applying a 'small' moment arm of external force and one while applying the same 30N force with the 'large' moment arm. Subjects were given 15 seconds rest periods between the tasks and 5 minutes between both series.

2.3 Electromyography (EMG)

Activity of the three Deltoid muscle parts (anterior (DA), medial (DM), posterior (DP)) was recorded with bi-polar surface EMG equipment (DelSys system Bagnoli-16, DE-2.1 single differential electrodes, inter-electrode distance 10mm, bandwidth 20–450 Hz, Boston, MA, USA). The electrodes were applied while subjects were positioned in the experimental set-up and after palpation of the muscle bellies. The skin was dry-shaved where needed, abraded (skinPure, Nihon Kohden, Tokyo, Japan) and cleansed with alcohol pads.

EMG of the deeply positioned Supraspinatus was recorded by means of bi-polar Teflon-coated EMG fine-wire electrodes (Cooner Wire Co., Chatsworth, CA, USA),[27] which were connected to factory adjusted single differential electrodes (DelSys system Bagnoli-16, DE-2.1 single differential electrodes, uncoated electrode tips of 2mm with an proximal inter-electrode distance of 2mm, bandwidth 20–450 Hz, Boston, MA, USA). The wires were inserted by a physician at two-thirds of an imaginary line from Trigonum Spinae to Angulus Acromialis, 2 cm above the spinal ridge. Before insertion, the skin was sterilized and anaesthetized (5% lidocaine injection).

Electrode positions were verified during the practice round and during the experimental tasks. During each task, real-time graphical visualizations of the EMG recordings of the four electrodes were visually checked for sufficient signal-over-noise and absence of movement distortion. In case of any problems, concerning electrodes were re-applied.

2.4 Data processing

Simultaneously recorded force and EMG signals were Analog-to-Digitally converted (sample rate: 2000 Hz). EMG recordings of the 2-second time intervals of each rest and force task were rectified and averaged. Rest activity was subtracted from task activities. These average rectified EMG signals (rEMG) were subsequently used for analysis.

In this study we compared the activation response of the Deltoid muscle, with a wide origin and insertion approximately covering over 120 degrees around the humeral longitudinal axis, to the response of the smaller Supraspinatus muscle, with its small insertion site at the Tuberculum Majus. We were faced with two problems: 1) how to cope with inter-individual anatomical variability in muscle origin and insertion and intra-individual temporal differences in muscle activation; and 2) how to select and compare equivalent Deltoid muscle parts with the Supraspinatus muscle. With respect to the first, we have chosen to assess a force direction range of 90 degrees with the assumed midline covering the primary line of action for the Supraspinatus at 45 degrees on average in the applied set-up. Based on a cosine distributed contribution of a muscle (part) to the external moment,[25,31] the average contribution of the muscle over the full range of -45 to 45 degrees around its principal line of action using the cosine distribution, is 90% of the maximal potential contribution, which we find a representing potential activation, guaranteeing a good signal over noise ratio (SNR) and averaging the effects of inter-individual anatomical differences and intra-individual temporal variability in muscle activation. For the second choice and as a result of the first choice we had to identify the force directions of the Deltoid muscle parts in which they were assumed to be primarily active in the applied set-

up. For this, we derived the maximum muscle activity of each of the muscle(part) s assessed in a larger group of subjects with multiple measurements in a previous study with the similar set-up, also assessing changes in Deltoid activity with altering elevation moments [24]: 0-30° for DA (i.e. 3 force tasks), 0-75 degrees for DM (i.e. 6 force tasks) and 45-90 degrees for DP (i.e. 4 force tasks). Accordingly, to obtain single measures of DA, DM and DP activities in the 0-90 degrees elevation tasks, concerning rEMGs were averaged over the relevant (maximum activity) task directions for each muscle part of each subject.(Figure 2) Additionally, these averaged rEMGs for DA, DM and DP were averaged to obtain a single Deltoid measure for each subject, using relevant weighing factors (3 force direction tasks for DA: 3, 6 tasks for DM: 6, 4 tasks for DP: 4). For the Supraspinatus, measurements of all 7 task directions were averaged into a single value for activation during elevation tasks.

Comparison of EMG between different Deltoid muscle parts and the Supraspinatus requires normalization. Instead of normalization to MVC, which is hard to obtain in a reliable manner, we applied a muscle ratio R_{muscle} (Eq. 1), which is equivalent with the '*Activation Ratio*' [15, 29, 32] to compare Deltoid and Supraspinatus muscle activation. The method is based on normalizing the response of the muscle on different tasks: i.e. small moments (proximal point of force application) and large moments (distal point of force application) in the current study.

$$R_{Muscle} = \frac{rEMG_{distal} - rEMG_{proximal}}{rEMG_{distal} + rEMG_{proximal}} \qquad -1 \leq R_{Muscle} \leq 1 \tag{1}$$

An R_{Muscle} larger than 0 means there is a relative increase in muscle activation with increasing external GH moment loading. In contrast to raw EMG recordings, these normalized EMG ratios enable direct intra- and inter-subject comparisons and evaluation on group level. Muscle ratios were obtained for the separate Deltoid parts, the weighed Deltoid average and the Supraspinatus (R_{DA}, R_{DM}, R_{DP}, R_{DELT} and R_{SSp}).

2.5 Statistical Analysis

As noise and offset activation bias the R_{Muscle} towards 0, for reliable R_{Muscle} estimates, the signal-to-noise ratio (SNR) was defined to exceed a factor two. The SNR was obtained by the ratio of (minimal) task *rEMG* over the subjects' rest *rEMG*.

Mean R_{Muscle}, corresponding standard deviations (SD) and 95%-confidence intervals (95%-CI) were calculated for R_{DA}, R_{DM}, R_{DP}, R_{SSp} and R_{DELT}. As $R_{Muscle} > 0$ indicates an increase in muscle activation, R_{Muscle}'s with 95%-CI's excluding 0 were significant increases in muscle activation in response the increase in arm moment loading.

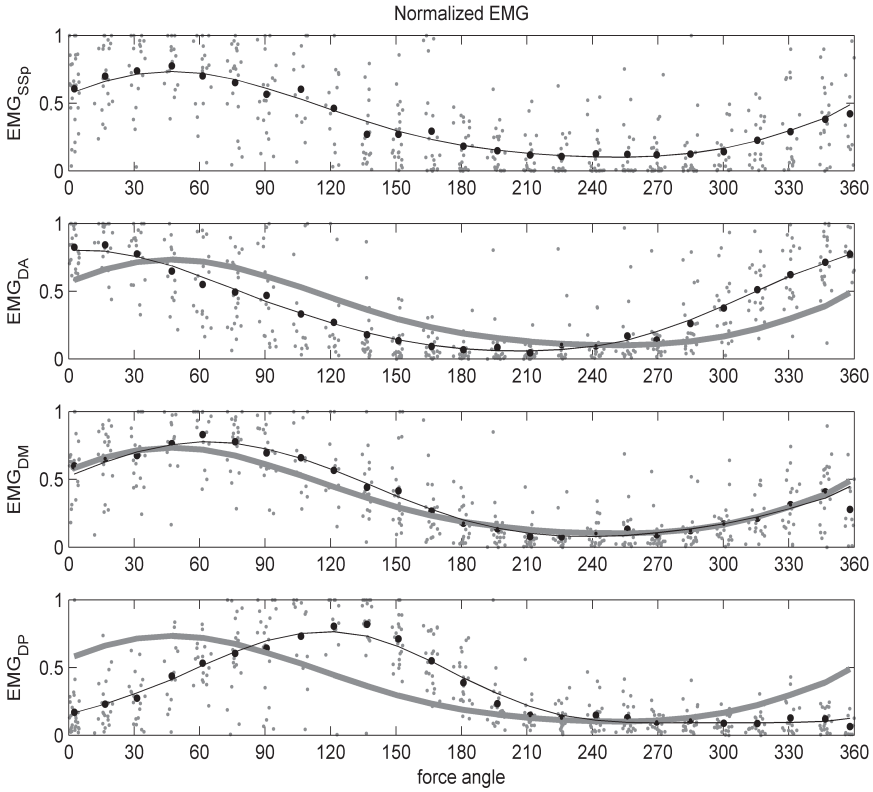


Figure 2. Plot of normalized EMG data of all subjects during the large moment loading tasks.
In this figure all 24 measurements are included. For the analyses in this study, we focused on isometric tasks that are primarily generated by the Deltoid and Supraspinatus: in the range of 0-90 degrees. Specifically DM and SSp have similar activation patters, with their maximum activation (principal action) within the range of 0-90 degrees. As can be expected, DA is more active during "elevation-anteflexion" directed tasks and DP during "elevation-retroflexion" directed tasks.

- Grey dots: raw data representing all normalized EMGs for each task direction;
- Black dots: average normalized EMGs for each task direction;
- Black lines: filtered average over all task directions;
- Grey line: filtered average of Supraspinatus in comparison to the Deltoid muscle parts.

The relative changes in activations of the Deltoid (R_{DA}, R_{DM}, R_{DP} and R_{DELT}) from the small to the large moment arm of external loading were compared with R_{SSp} using paired t-tests. To assess whether the responses of the Deltoid and Supraspinatus on increased moment loading were equally unambiguous, Levene's Test was applied to study potential differences in the R_{Muscle} standard deviations.

Pearsons correlations coefficients and multivariate linear regression analyses were used to assess relations between the Supraspinatus Ratio R_{SSp} and Deltoid Ratio's R_{DA}, R_{DM}, R_{DP}, and R_{DELT}. Lastly, the effects of the three Deltoid Ratios and their interaction term on Supraspinatus Ratio was assessed. In a second linear regression analysis the relation between R_{DELT} and the R_{SSp} was assessed.

SPSS 17.0 (SPSS Inc., Chicago, IL, USA) was used for statistical analyses. The level of significance (p-value) was set at 0.05 for all tests.

3. Results

3.1 rEMG During small and large moment tasks

All subjects were able to fulfill the required experimental tasks without any shoulder symptoms. Signal-to-noise ratios were larger than 2.5 for all recordings during all tasks, ranging from 2.5 to 54.4 for the small moment series and 2.6 to 68.3 for the large moment series.

The *EMG Ratios* of the Anterior Deltoid (R_{AD}=0.13, SD=0.10), Medial Deltoid (R_{DM}=0.13, SD=0.14) and averaged Deltoid muscle parts (R_{DELT} = 0.11, SD=0.10) demonstrated a statistically significant increase in activation with increased GH elevation moment loading, expressed in 95%-CI's excluding 0.(Table 1) For the Supraspinatus and the Posterior Deltoid average activation increase was observed (R_{Muscle}>0), but without statistical significance. The standard deviation of the response of Deltoid (R_{DELT}) was significantly smaller than the standard deviation of the Supraspinatus Ratio R_{SSp} (F=11.7, p<0.001).

Muscle	**Muscle Ratio**			**Mean difference with SSp**		
	Mean	SD	95%-CI	Mean	95%-CI	p-value
Supraspinatus (SSp)	0.06	0.31	-0.08 - 0.20			
Deltoids						
Anterior part (DA)	0.13	0.10	0.08 - 0.18	0.07	-0.08 - 0.22	0.35
Medial part (DM)	0.13	0.14	0.06 - 0.19	0.07	-0.10 - 0.23	0.41
Posterior part (DP)	0.08	0.17	0.00 - 0.15	0.02	-0.17 - 0.20	0.85
Averaged Deltoid parts (DELT)	0.11	0.10	0.06 - 0.16	0.05	-0.11 - 0.22	0.51

Table 1. Relative increases in activations with large moment loading of the Deltoid muscle parts and the Supraspinatus.
There was significant increase in activation of R_{DA}, R_{DM} and R_{DELT} with larger moments. Comparing Deltoid ratios (R_{DA}, R_{DM}, R_{DP} R_{SSp} and R_{DELT}) with R_{SSp}, using paired t-tests, gave no statistically significant differences. The standard deviation of the increase in Supraspinatus activation was 2-3 times larger than for the Deltoid (Levene's test: F=17.0, p<0.0001).

3.2 Relationship between increase in Supraspinatus and Deltoid activation with large moment loading

Twelve subjects (55%) showed a larger activation increase in the averaged Deltoid muscle parts compared to the Supraspinatus activation increase. Seven subjects (32%) even showed a decreased Supraspinatus activation during large moment loading. None of the subjects showed a decrease in both Supraspinatus and Deltoid activity. Overall, comparing Deltoid and Supraspinatus responses of all subjects, no statistically significant differences were observed comparing Deltoid Ratios with the Supraspinatus Ratios.(Table 1) The correlations between responses of Supraspinatus and Deltoid muscle parts were all negative, indicating a larger increase in Deltoid activation coincides with a smaller increase in Supraspinatus

activation and vice versa, but without statistical significance: $r_{SSp\text{-}DA}$ = -0.17 (p=0.47), $r_{SSp\text{-}DM}$ = -0.22 (p=0.34), $r_{SSp\text{-}DP}$ = -0.32 (p=0.16) and $r_{SSp\text{-}DELT}$ = -0.34 (p=0.13).(Figure 3)
With multivariate regression analyses, no significant association between R_{SSp} and the Deltoid ratios (R_{DA}, R_{DM}, R_{DP}) and their interaction term was found: estimated effects for the Deltoid muscle parts were all negative (range, -0.33 (p=0.56) to -0.73 (p=0.20)), indicating a larger increase in Deltoid activation coincides with a smaller increase in Supraspinatus activation in our data, but without statistical significance.
A separate linear regression analysis assessing the association between the averaged Deltoid parts (R_{DELT}) and R_{SSp} resulted in an effect of ß=-1.02 (95%-CI: -2.37–0.32), also indicating a larger increase in Deltoid activation coincides with a smaller increase in Supraspinatus activation and vice versa in our data (exchangeable or complementary role for generating elevation moments), but without statistical significance.

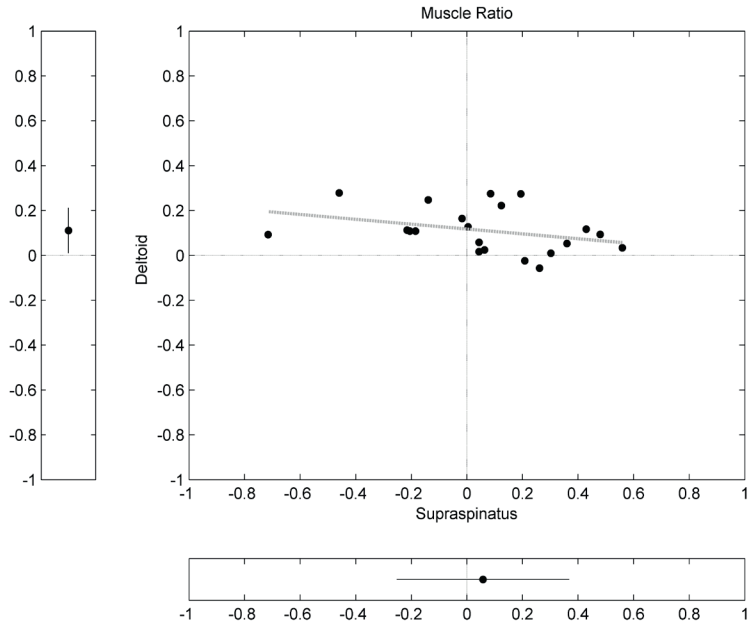


Figure 3. Scatter plot of the relative changes in muscle activity of the averaged Deltoid and Supraspinatus, expressed in normalized Muscle ratios for comparing muscle activity (EMG) during large moment loading and small moment loading (with a constant task force magnitude).

$$R_{Muscle} = \frac{rEMG_{distal} - rEMG_{proximal}}{rEMG_{distal} + rEMG_{proximal}} \qquad -1 \le R_{Muscle} \le 1$$

In the line plots, average Muscle ratios for DELT and the Supraspinatus are depicted, accompanied by their Standard Deviations.
On average, there was a significant and homogeneous increase in Deltoid activation, but a highly variable response in Supraspinatus activation.

4. Discussion

In this experimental study we investigated the responses of the Deltoid and the Supraspinatus after increasing the external force moment arm of 30N isometric elevation force tasks. We observed on average a statistically significant increase in Deltoid activation in response to the glenohumeral elevation moment increase. Surprisingly, the Supraspinatus did not show a significant activation increase. Its response was highly variable between subjects and significantly more variable than that of the Deltoid. So opposed to our hypothesis, there was no average increase in activation of both muscles, but an average increase in Deltoid activation in combination with a highly variable response of the Supraspinatus. Additionally, we observed an inverse association between the Deltoid and Supraspinatus responses in our data (n.s.), suggesting a complementary function of the Supraspinatus to the Deltoid in generating glenohumeral elevation moments.

As the Deltoid muscle, with its three large muscle parts with favorable moment arms, is generally regarded as an abduction moment generator or elevator,[8-10] increasing external moment loading would theoretically lead to an increase in Deltoid activation. This was confirmed by the results of the current and a previous study.[15] On the other hand, there is much debate on the primary biomechanical function of the Supraspinatus. In many studies biomechanically assessing the Supraspinatus, its function is not actually measured but derived from responses of other muscles or data from subjects with impaired cuff function (tendon tear or nerve block). Several authors describe that the Supraspinatus is an important elevator, synergistic to the Deltoid.[11-14] Others have labeled the Supraspinatus as a muscle that primarily functions as a glenohumeral joint stabilizer, whether or not dependent on type of exerted arm/shoulder movement and arm position.[16-22, 33] Our results demonstrate that the response of the Supraspinatus to an increase in elevation moment loading is highly variable and without an average and consistent increase in activation. This pleads against a clear-cut and definite overall elevation moment generator role for the Supraspinatus. The inverse association between the Deltoid and Supraspinatus as observed in our data (n.s.), suggests that both muscles may contribute to glenohumeral elevation moments in a complementary manner: subjects with only a small increase in Supraspinatus activity had on average a larger increase in Deltoid activity. This is consistent with results from both knock-out experiments and simulation studies,[14, 15] that show that elimination of Supraspinatus activity coincides with a large increase in Deltoid activity when applying elevation moments.

The observed significantly more variable response of the Supraspinatus compared to the Deltoid, and the lack of significant increase in its activation in response to

increased moment loading might indicate that the coordination, recruitment, or primary function of the Supraspinatus differs between individuals. Although speculative and not assessed in this study, this might in turn be an explanation for the variations in severity of symptoms and treatment outcomes in patients with RC tendon tears and the high prevalence of asymptomatic tears. For example, subjects in whom the Supraspinatus is primarily e.g. an elevator might experience less symptoms of a tendon tear (because of compensation for its moment generator function by the Deltoid) than subjects in whom the Supraspinatus functions more as an important glenohumeral stabilizer. In the latter group, a torn Supraspinatus tendon could lead to a jeopardized glenohumeral force equilibrium. In combination with increased Deltoid activation, this can lead to humerus cranialisation with consequent compression of subacromial tissues and pain.[23, 28] Recognition of these potential subgroups would have consequences in the diagnostics and treatments of Supraspinatus pathologies. It might be postulated that the observed variabilities may also be due to coincidental sampling of functionally different muscle parts of the Supraspinatus muscle[34] as the pick-up area of the fine-wire electrodes is relatively small. But in this case our findings are still relevant, by underlining the Supraspinatus is not simply an arm elevator.

There are some limitations to the interpretation of our findings. Firstly, we assumed that cuff status of the healthy subjects was normal. None of our subjects had any current shoulder complaints or a history of shoulder injury and the group was relatively young, but we did not check cuff status with radiological techniques. Secondly, as in most EMG studies, we assumed that EMG recordings represent overall activity of the whole muscle, also when measured with a local fine-wire electrode as was needed for Supraspinatus recordings. We did measure all three Deltoid parts however, since these three parts are known to have distinct functions. Thirdly, we only assessed the Supraspinatus and Deltoid in one specific arm position, under experimentally controlled isometric conditions, without gravitational arm loading and by changing the glenohumeral moment only. However, assessing all subjects in the same arm position and EMG recording during isometric tasks are actually methodological strengths and prevent errors in EMG measurements due to e.g. skin movements or changes in muscle length. The Supraspinatus was active in all subjects during all tasks (with large signal-to-noise ratios) and in a recent study of Wickham et al, Supraspinatus peak activity during elevation was near 60 degrees of abduction, as applied in our study.[35] However, our results cannot be extrapolated to other arm positions such as for daily activities, as force and moment loads generally change coincidentally and not predictably.

We conclude that, as might be expected, the activity of the Deltoid significantly increases when increasing the moment arm of an external elevation force. In

contrast, the response of Supraspinatus was highly variable and a specific function in generating arm elevation moments could not be demonstrated. So, where various other studies have different and conflicting findings on Supraspinatus function, we found a variable Supraspinatus response in our study. The observed variations may be a reflection of these different reported Supraspinatus roles in the glenohumeral moment and force equilibrium and of the variety of symptoms and treatment results of Supraspinatus pathologies. On subject level, it is plausible that the Supraspinatus predominantly functions as a moment generator in some individuals, while in others more as a stabilizer, whether or not depending on arm position and the application of the external loading. More insight in the coordination and biomechanical function(s) of the Supraspinatus and their potential inter-subject variabilities is needed to optimize future diagnostics and treatment options in patients with shoulder symptoms.

Acknowledgements

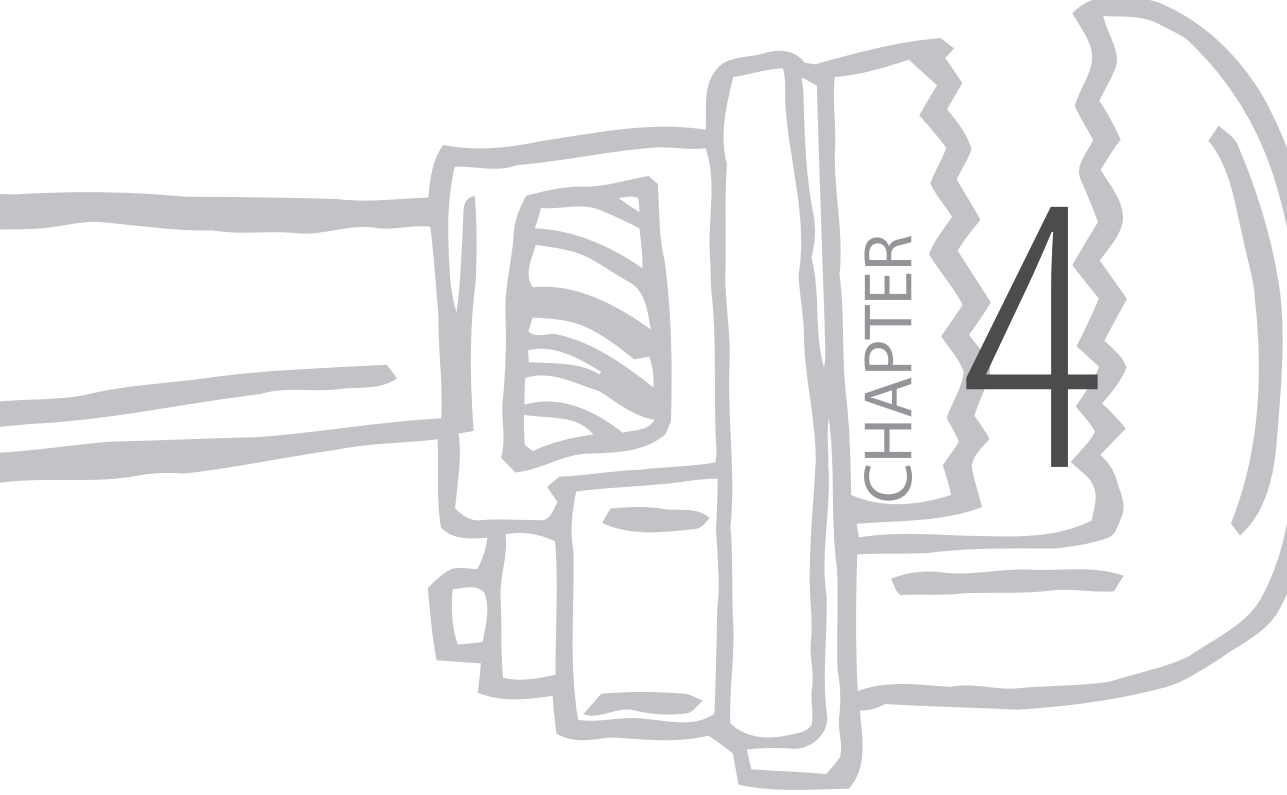
The authors would like to acknowledge H. Fraterman, J. van der Ploeg and P. Bakkenes (Technical Dept., Leiden University Medical Center, the Netherlands) for their input in developing the experimental set-up.
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Heterogeneous MR arthrography findings in patients with Subacromial Impingement Syndrome - Diagnostic subgroups? -

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Abstract

Background: Subacromial impingement syndrome (SIS) is frequently diagnosed, but its underlying mechanisms are unclear and treatment results vary greatly. In the recent past, it has been increasingly reported that SIS symptoms might be the result of various underlying mechanisms that might need distinctive treatment strategies. Our goal was to evaluate a comprehensive set of specific Magnetic Resonance Imaging arthrography (MRA) characteristics that have been related with several potential underlying mechanisms for SIS in the literature, in patients clinically diagnosed with SIS. Our secondary aim was to define diagnostic subgroups with these MRA characteristics.

Methods: Patients with the clinical label SIS were included by experienced shoulder surgeons, based on symptoms, clinical tests and radiographs. MRAs of 47 patients were evaluated. MRA characteristics associated with SIS and subacromial narrowing in the literature were evaluated and categorized into three etiologic categories: 1) extrinsic: acromion shape, acromiohumeral distance, coracohumeral distance, caudal acromioclavicular osteophytes, internal impingement; 2) intrinsic: tendinosis, partial tendon tears, bursitis, supraspinatus tendon thickness; 3) dynamic (e.g. signs of glenohumeral micro-instability): glenohumeral index, biceps-supraspinatus tendon distance. All measurements were compared to control values from the literature. Cluster analysis was used to identify diagnostic subgroups.

Results: In 17 (36.2%) patients clinically diagnosed with SIS, signs of specific other conditions, including rotator cuff tendon tears and labrum lesions were found with MRA. In the remaining thirty patients, all had positive signs of one or more of the predefined etiologic mechanisms. With cluster analysis, patients could be categorized into two groups, with either predominantly findings corresponding with dynamic ((micro)instability) causes, or extrinsic (structural) causes.

Conclusions: MRA characteristics in patients with SIS symptoms are heterogeneous and many patients have specific other shoulder conditions causing symptoms. Patients without specific other conditions appear to have MRA characteristics associated with either an extrinsic (structural), or dynamic (e.g. micro-instability) based etiology.

Keywords: Shoulder Impingement Syndrome; Magnetic Resonance Imaging; Etiology; Rotator Cuff; Cluster Analysis

1. Introduction

Subacromial Impingement Syndrome (SIS) is diagnosed in 44-65% of the patients with shoulder complaints in primary health care.[1] SIS is generally defined as irritation of the soft tissues in the limited subacromial space, leading to pain with abduction, decreased active range of motion and loss of arm force.[2-4] Opinions concerning its etiology, diagnostic criteria, corresponding radiographic characteristics, and treatment strategies vary greatly.[1, 5-13] Nevertheless, there are many publications on "SIS" patients. Conflicting definitions of SIS are used across these studies, complicating interpretation of reported results.[14] More insight in underlying causes and identification of potential etiologic or diagnostic patient subgroups is needed in order to improve diagnostic criteria and treatment outcome of SIS symptoms. We investigated specific Magnetic Resonance Imaging (MRI) features related with SIS and subacromial narrowing in the literature, in patients clinically diagnosed with SIS, and we assessed whether diagnostic subgroups can be identified using a comprehensive combination of MRI arthrography features. Gathered information might serve as a foundation for future studies upon which clinical decision making and development of tailored treatment strategies can be based.

"Impingement syndrome" was introduced in 1972 by dr. Neer as a combination of typical clinical findings with various underlying mechanisms and stages.[15] Over the years, this evolved to the "*subacromial* impingement syndrome", which (wrongfully) suggests high specificity and anatomic differentiation of this entity. Since 1972, many diagnostic tools have been developed, including ultrasound, MRI and MRI arthrography (MRA), that can more accurately differentiate between causes of shoulder symptoms.[16-18] Consequently, "SIS" has become increasingly controversial as a diagnostic label. Some consider SIS symptoms as a consequence of several possible underlying mechanisms and pathologies rather than a specific diagnosis.[19] Furthermore, several conditions can cause SIS symptoms and can be (mistakenly) diagnosed as SIS; in a recent study at our institution 17.5% of patients selected for a SIS trial by experienced shoulder surgeons had to be excluded after MRI evaluation because of specific other shoulder conditions.[20]

Various studies have reported on specific MRI or MRA findings in patients with impingement symptoms. Characteristics for *extrinsic* etiologic mechanisms (structural or anatomic causes) are a hooked acromion,[21] caudal acromioclavicular (AC) joint osteophytes,[22-25] or subcoracoid impingement (narrowed space between coracoid and humerus).[26-29] But also signs of *intrinsic* pathologies can be found, including tendinosis, partial RC tears and bursitis,[30-35] either as a cause of SIS symptoms, or consequent to other etiologic mechanisms. Lastly, SIS symptoms can be caused by *dynamic* compression of the subacromial tissues during arm-shoulder

motion, as a result of e.g. glenohumeral (micro)instability, with specific associated findings on MRI.[36-39]

The primary goal of the current study was to investigate a group of patients with the clinical diagnosis SIS with MRA and a broad set of specific measurements, in order to evaluate characteristics of the various reported etiologic mechanisms of SIS symptoms. Additionally, we aimed to identify actual diagnostic subgroups, using the comprehensive set of MRA characteristics.

2. Materials and Methods

2.1 Selection of participants

Patients with impingement symptoms were recruited by 3 experienced orthopaedic shoulder surgeons from Leiden University Medical Center (LUMC), the Hague Medical Center (MCH) and Rijnland Hospital, Leiderdorp in an ongoing prospective multicenter observational cohort study on SIS. Data collecting included kinematic, clinical and radiographic methods.[37] In the current study, MRA characteristics of all consecutive patients included from April 2010 until December 2012 are reported and compared with control values from the literature.

Patients were enrolled after clinical examination and shoulder radiographs (anteroposterior in external and internal rotation and axial views) at the outpatient clinic. Inclusion criteria were one or more of the following criteria in addition to a painful arc, a positive Neer impingement test with lidocaine, a positive Hawkins test, and diffuse lateral shoulder pain for >3 months: night pain or incapable of lying on the shoulder, scapulohumeral dysrhythmia, pain with retroflexion and/or internal rotation (e.g. putting on a jacket, overhead activities), positive Yocum test. Exclusion criteria were: age <35 or >60, clinical signs of adhesive capsulitis, history of fracture or dislocation of the shoulder, history of surgery around the shoulder, known co-morbidities on the affected shoulder (including benign or malignant tumours), Hill Sachs lesion, glenohumeral or symptomatic AC joint osteoarthritis (positive AC compression test and/or pain with palpation of the AC joint), rheumatic disorder, calcific tendonitis >3mm on radiographs or cervical radiculopathy or other neurological deficits.

The medical ethics committees of the participating hospitals agreed to all stages of the study. Eligible patients who were willing to participate in the entire SIS project and who signed informed consent underwent a study-specific MRA of the shoulder and kinematic evaluation in our laboratory for kinematics and neuromechanics (LK&N). Patients had standard treatment by their referring clinician outside of the scope of this study.

2.2 MR Arthrography

MRA extends the capabilities of conventional MRI because contrast solution distends the joint capsule, outlines intra-articular structures, and leaks into abnormalities like tendon tears.[40] We used fluoroscopic guidance for intra-articular contrast administration with a 20-22 Gauge needle. After correct needle positioning, checked with 1-2 cc of nonionic iodinated contrast (Ultravist 300, Bayer), a maximum of 15 cc of diluted Gadolinium DTPA (0.4 cc:100 cc 0.9% NaCl) was administered.

Standardised MR imaging was performed 30 minutes after contrast administration on a 1.5T unit (Avanto Siemens, Erlangen, Germany, or Philips Intera, Best, The Netherlands) with a dedicated shoulder coil and the arm in neutral position (with slight internal rotation). The following sequences were used: axial, coronal oblique, sagittal oblique, T1-weighted fast spin-echo with fat suppression and coronal oblique T2-weighted with fat suppression. The field of view was 16-18cm, slice thickness 3mm or 4mm with 1mm gaps.

2.3 MR Arthrography assessment

The MRA images were evaluated by an experienced musculoskeletal radiologist at the LUMC, unfamiliar with the underlying hypotheses of this study, using PACS IDS5 11.4 software (Sectra Medical Systems AB, Linköping, Sweden). With a standardized MRA check-list, specific other conditions that can cause SIS symptoms were evaluated in all patients, including SLAP (Superior Labrum Anterior-Posterior) lesion, os acromiale, pulley lesion, Hill Sachs lesion, biceps tear or (sub)luxation of the biceps tendon, glenohumeral ligament pathology, or full-thickness RC tendon tear.

Patients with SIS symptoms without signs of other specific shoulder conditions were assessed for the presence of MRA characteristics typically associated with etiologic mechanisms for SIS symptoms in the literature. All cut-off values to define pathologic observations were obtained from 95%-confidence intervals (95%-CI) of healthy control data from the literature. Where applicable, Taylor expansions were applied to calculate 95%-CI's of literature data.

MRA characteristics were categorized, using a theoretical framework for the etiology of SIS derived from our previous study in which we reported that SIS, i.e. "a misbalance between subacromial volume and the space needed for subacromial structures" can be caused by:[37]

1) *Encroachment of subacromial tissues by anatomic structures (extrinsic):*
   - Acromion shape (Bigliani classification, classic extrinsic theory)[41] on sagittal oblique MRA series. A higher classification, i.e. more hooked shape, has been related to SIS.[21, 23] Bigliani type 2 or 3 (sagittal plane) classifications were regarded indicative for "classic" extrinsic impingement.
   - Minimal acromiohumeral (AH) distance (classic extrinsic theory). A smaller distance implies risk for mechanic structural impingement.[42, 43] AH <8.2mm (coronal plane) was considered indicative for extrinsic impingement.[44]
   - Subcoracoid impingement (between coracoid and humerus): decreased coracohumeral (CH) distance.[28, 45] Based on the results of Richards et al. CH distance <9.6mm (transverse plane) was considered pathologic.[45] (Figure 1)
   - AC joint osteoarthritis: osteophytes can impinge on the underlying tissues.[22-24, 46] Caudal osteophytes >2mm (coronal plane) combined with a deformity in the contour of underlying RC tendons in patients with otherwise asymptomatic AC osteoarthritis (negative AC compression test and no pain with palpation of the AC joint) was considered contributing to SIS.[25] (Figure 2)
   - Internal (glenoid) impingement: structural narrowing between the greater tuberosity and the posterosuperior glenoid, entrapping RC tendons during abduction and external rotation. The combination of cystic changes within the posterolateral humeral head, posterior articular surface tear(s) of the infraspinatus (ISP) and/or Supraspinatus (SSP) and posterior labrum abnormalities was considered indicative of internal impingement.[29]

2) *Intrinsic impingement*. For this category, we assessed:
   - Presence and location of RC tendinosis.[13, 31, 32] Patients were categorized in: tendinosis in 0, 1 or >1 tendons.
   - Presence of partial RC tendon tears and location (affected RC tendon(s), articular/bursal side, intratendinous).[34, 47] Patients were categorized in: partial tear in 0, 1, or >1 tendons.
   - Subacromial bursitis, defined as bursal fluid effusion with a width >3mm.[35]
   - Maximal SSP tendon thickness, measured in the coronal plane at 1.5cm from the footprint.[30, 33] (Figure 3) Based on the data of Milgrom et al, <5.9 and >6.3 were applied as cut-off values for pathologic tendon thickness.[33]
   - Maximum SSP tendon thickness was divided by the minimal AH-distance to obtain a relative measure of the amount of AH available for the SSP tendon.

3) *Dynamic impingement, e.g. due to (micro)instability:*

- Glenohumeral (GH) index: measure for the amount of bony support for the humeral head provided by the glenoid ((maximal glenoid diameter)/ (maximal humerus diameter), transverse plane).[48, 49] (Figure 4) GH-index <0.61 was considered indicative of (micro)instability.[48]
- Distance between the anterior edge of the tendon of the long head of the Biceps (LHB) and the anterior edge of the SSP tendon (4th slide after the base of the coracoid, sagittal plane). (Figure 5) Based on Provencher et al. a distance >4.0mm was considered indicative for micro-instability.[36]

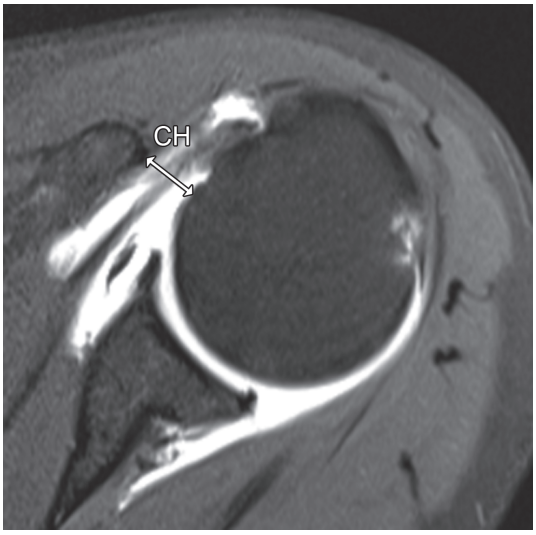


Figure 1. Coracohumeral (CH) distance.
Based on the results of Richards et al.[45] we considered CH distance <9.6mm as measured in the transverse plane as pathologic.

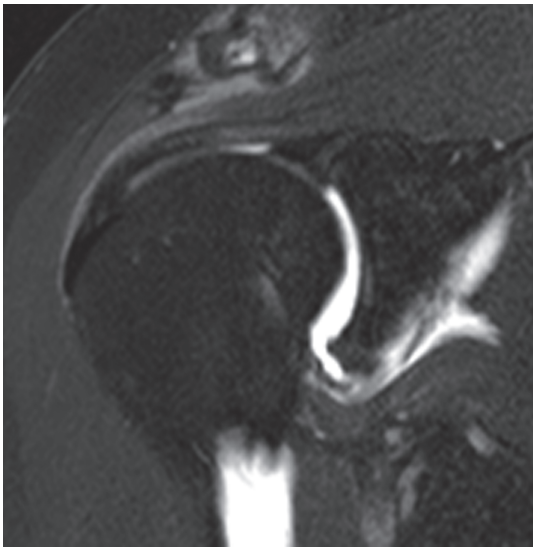


Figure 2. Example of caudal osteophytes >2mm in a patient with signs of acromionclavicular joint osteoarthritis on MRA, in combination with a deformity in the contour of the underlying subacromial tissues.

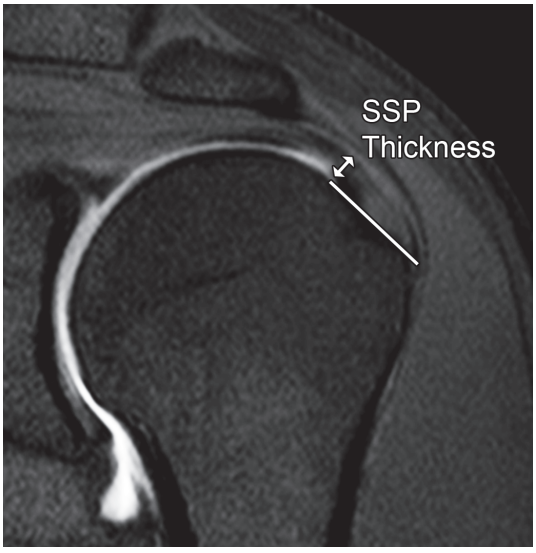


Figure 3. Maximal SSP tendon thickness, measured in the coronal plane at 1.5cm from the footprint.
Based on the data of Milgrom et al, <5.9 and >6.3 were applied as cut-off values for pathologic tendon thickness.[33]

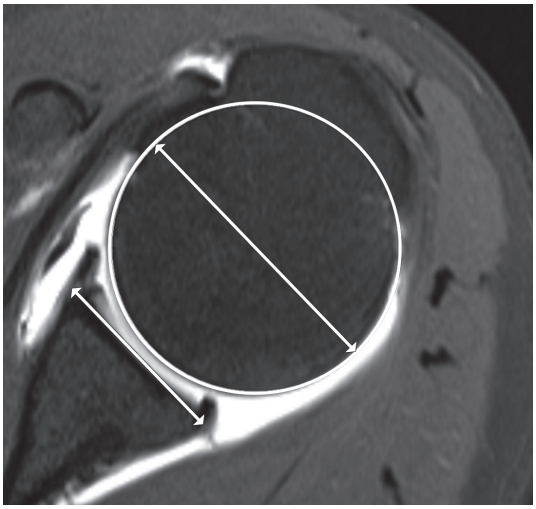


Figure 4. Glenohumeral (GH) index: measure for the amount of bony support for humeral head provided by the glenoid ((maximal glenoid diameter)/(maximal humerus diameter), transverse plane).
GH-index <0.61 was considered indicative of instability.[48]

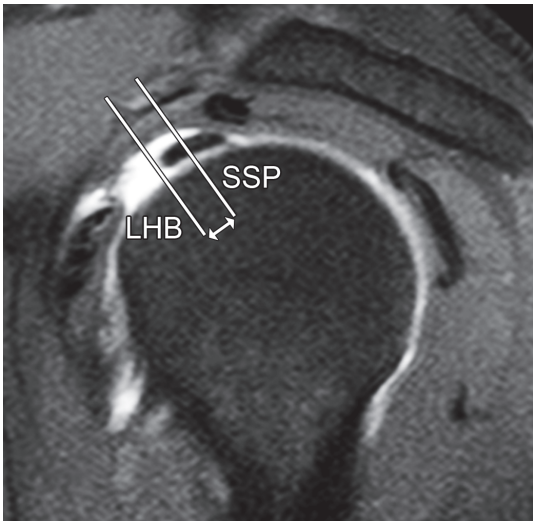


Figure 5. The distance between the anterior edge of the tendon of the long head of the Biceps (LHB) and the anterior edge of the Supraspinatus (SSP) tendon (4th slide after the base of the coracoid, sagittal plane).
A distance exceeding 4.0mm can be regarded indicative for micro-instability. According to Provencher et al. a more posterior LHB, more "tucked" under the SSP, should be expected in asymptomatic controls.[34]

2.4 Statistical analysis

Descriptive MRA data and patient demographics were collected. Means and standard deviations (SD), or medians and ranges where relevant, were calculated for each variable. The control cut-off values obtained from the 95%-confidence intervals of healthy control data reported in the literature (see above) were applied to assess whether patients had (pathologic) MRA characteristics associated with one or more of the hypothesized etiological mechanisms.

A Ward's cluster analysis was used, in order to investigate potential diagnostic patient subgroups.[50] With cluster analysis, subjects are assigned to groups (clusters) based on individual characteristics. Each cluster contains subjects that are more similar to each other than to subjects in the other clusters. In this way, patients with similar characteristics can be classified into specific subgroups. Identified subgroups were compared with regards to MRA characteristics and demographics using one-way ANOVA or Chi-squared analyses where appropriate.

Analyses were processed using SPSS 16.0 software (SPSS Inc., Chicago, Illinois) and R 2.10.0 Statistics software (R Foundation, Vienna, Austria). P-values <0.05 were considered statistically significant.

3. Results

3.1 Patient characteristics

During the inclusion period, 75 patients were invited to participate in the larger SIS study project. 25 Patients could not be included: 14 declined, 5 had clinical signs of AC osteoarthritis, 4 had calcific tendonitis on radiographs, 1 had a history of shoulder surgery and 1 had frozen shoulder syndrome. The remaining 50 patients fulfilled the eligibility criteria after usual care clinical and radiographic evaluation and underwent the study-specific MRA.

In 3 patients, MRAs could not be assessed for study purposes, due to inadequate intra-articular distribution of contrast fluid. All remaining 47 patients showed pathologic signs with MRA evaluation. In 17 (36.2%) patients clinically diagnosed with SIS, signs of specific other shoulder conditions were found: full-thickness RC tear in 11 (64.7%), labrum abnormalities (not in combination with internal impingement) in 3 (17.6%), glenohumeral ligament lesions in 2 (11.8%) and calcific tendonitis >3mm in 1 (5.9%). The remaining 30 patients (63.8%) were considered as actual SIS patients (i.e. without specific other conditions causing symptoms), and were studied with regards to SIS-related characteristics on MRA. Demographics of both groups are displayed in table 1.

Variable	SIS (n=30)	Other shoulder conditions with MRI (n=17)
Age (years)	51.5 [6.4]	52.2 [6.1]
Male : Female	12 : 18	5 : 12
BMI	26.6 [4.2]	NA
Left arm dominant (No.)	5 (16.7)	NA
Dominant arm affected (No.)	16 (53.3)	NA

Table 1. Patient demographics, stratified for patients with the diagnostic label "subacromial impingement syndrome (SIS)" after clinical evaluation, radiographs and MR arthrography, and patients with specific other pathologies identified on MR arthrography causing SIS-like symptoms. (%), [SD]

3.2 MRI characteristics in SIS patients

In 5 patients, the obtained MRAs were not adequate to measure LHB-SSP distance (n=4) or CH distance (n=1). All other measurements of these patients were included in the analyses.

All 30 patients had positive signs for one or more of the predefined etiologic mechanisms. Firstly, for characteristics associated with *extrinsic* etiologic mechanism (i.e. encroachment of subacromial tissues by anatomic structures), 21 (70.0%) patients had a type 2 or 3 acromion. Mean AH distance was 8.0mm (SD=1.5), which was below the 8.2mm cut-off in 19 (59.4%) patients. In 16 (53.3%) both of these measurements for "classic" extrinsic impingement were positive. Furthermore, 12 patients (41.4%) had MRA signs of AC joint osteoarthritis (despite positive physical examination tests for AC osteoarthritis was an exclusion criterion) with caudal osteophytes >2mm impinging on the underlying tissues. Mean CH distance was 11.0mm (SD=3.1): below the 10.4mm cut-off in 12 patients (41.4%). There were no patients fulfilling all internal impingement criteria.

Secondly, *intrinsic* characteristics (either as a primary cause of SIS symptoms, or as a consequence of the other etiologic mechanisms) were found in 28 (93.3%) patients. 11 (36.7%) had tendinosis in one RC tendon (all SSP) and 5 (16.7%) had tendinosis in more than one tendon (all SSP and ISP). There were 5 (17.2%) patients with a partial SSP tendon tear (1 bursal side, 1 intratendinous, 3 articular side), 4 (13.8%) patients with a partial ISP tendon tear (all articular side) and 3 (10.3%) patients with an articular side tear in both the ISP and SSP tendon. Furthermore, 9 (30%) had bursitis and 25 (83.3%) had a pathologic SSP tendon thickness: <5.9mm in 5 (16.7%) and >6.3mm in 20 (66.7%). In 27 (96.4%), there were signs of intrinsic etiology, combined with positive findings of one or more of the other reported etiologic mechanisms.

Thirdly, for findings of *dynamic* mechanisms (i.e. motion-related causes of SIS symptoms, e.g. due to glenohumeral (micro)-instability), mean GH-index was 0.61 (SD=0.04) and mean LHB-SSP distance 4.5mm (SD=3.1). (Table 2) GH-index was below the 0.61 control cut-off in 14 (46.7%) patients, indicative for micro-instability.

The LHB-SSP distance was higher than the 4.0mm cut-off value in 11 (42.3%) patients. In 6 (23.1%) patients, both measurements were indicative for (micro)instability.

	SIS patients (n=30)	Indicative for SIS	
Variables	Mean	Cut-off	No. (%)
Structural etiology (extrinsic)			
Type of acromion		Type 2/3 [21, 23]	21 (70.0)
1 (No.)	9 (30.0)		NA
2 or 3 (No.)	21 (70.0)		NA
Min. acromiohumeral distance (mm)	8.0 [1.5]	<8.2 [44]	19 (59.4)
Coracohumeral distance (mm)	11.0 [3.1]	<9.6 [45]	10 (34.5)
Internal impingement (No.)	0 (0.0)	present [29]	0 (0.0)
AC-osteoarthritis & osteophyte >2 mm (No.)	12 (40.0)	present [25]	12 (40.0)
Intrinsic etiology			
Tendinosis		present [13, 31, 32]	16 (53.3)
No tendon (No.)	14 (46.7)		NA
1 tendon (No.)	11 (36.7)		NA
>1 tendon (No.)	5 (16.7)		NA
Partial rotator cuff tear		present [34, 47]	12 (40.0)
No tendon (No.)	18 (60.0)		NA
1 tendon (No.)	9 (30.0)		NA
>1 tendon (No.)	3 (10.0)		NA
Bursitis (No.)	9 (30.0)	present [35]	9 (30.0)
Max. thickness SSP tendon (mm)	5.2 [1.1]	<5.9 or >6.3 [33]	25 (83.3)
Max. Relative thickness SSP tendon	0.67 [0.19]		
Dynamic etiology			
Glenohumeral index	0.61 [0.04]	<0.61 [48]	14 (46.7)
Distance SSP-LHB (mm)	4.5 [3.1]	>4.0 [36]	11 (42.3)

Table 2. Positive MR arthrography characteristics for the various hypothesized etiologic mechanisms for SIS.
Findings presented for patients with SIS symptoms without any other shoulder pathologies with clinical investigation, radiographs and MR arthrography.
Applied cut-off values were obtained from the literature (references indicated).
((%), [SD], LHB: tendon of the long head of the biceps, SSP: Supraspinatus tendon)

3.3 Cluster analysis

Bigliani classification, caudal AC osteophytes, RC tendinosis, SSP tendon thickness, GH index and LHB-SSP distance were entered in the cluster analysis. Five patients could not be included in this analysis, because of missing values in one or more of the included variables.

The resulting cluster diagram contained 2 main clusters: cluster I with 8 patients (32.0%) and cluster II with the remaining 17 (68.0%) patients. Cluster I included patients with a more *extrinsic* related etiology, with significantly higher Bigliani classifications (all had type 2 or 3 classifications), a significantly lower LHB-SSP distance and a higher GH-index (both pleading against glenohumeral (micro) instability), more females and more bursitis compared to cluster II, which contained patients with more positive signs for *dynamic* related etiology, e.g. related with glenohumeral (micro)instability. (Table 3)

	Cluster I Extrinsic (n=8)	Cluster II Dynamic (n=17)	Difference	
Variables	Mean	Mean	Mean	p-value
Demographics				
Age (years)	47.8 [7.0]	51.4 [6.2]	3.7	0.20
Male : Female	1 : 7	10 : 7	NA	**0.03**
Structural etiology (extrinsic)				
Type of acromion				**0.03**
1 (No.)	0 (0.0)	7 (41.2)	NA	
2 or 3 (No.)	8 (100.0)	10(58.8)	NA	
Min. acromiohumeral distance (mm)	8.6 [1.5]	7.7 [1.3]	0.9	0.13
Coracohumeral distance (mm)	10.8 [3.6]	11.4 [3.3]	0.62	0.69
Internal impingement (No.)	0 (0.0)	0 (0.0)	NA	NA
AC-osteoarthritis & osteophyte >2 mm (No.)	1 (12.5)	7 (41.2)	NA	0.15
Intrinsic etiology				
Tendinosis				0.26
No tendon (No.)	4 (50.0)	10 (58.8)	NA	
1 tendon (No.)	4 (50.0)	4 (23.5)	NA	
>1 tendon (No.)	0 (0.0)	3 (17.6)	NA	
Partial rotator cuff tear				0.52
No tendon (No.)	6 (75.0)	9 (52.9)	NA	
1 tendon (No.)	2 (25.0)	7 (41.2)	NA	
>1 tendon (No.)	0 (0.0)	1 (5.9)	NA	
Bursitis (No.)	4 (50.0)	2 (11.8)	NA	**0.04**
Max. thickness SSP tendon (mm)	5.6 [1.3]	5.0 [0.8]	0.6	0.23
Max. Relative thickness SSP tendon	0.66 [1.17]	0.67 [0.19]	0.01	0.86
Dynamic etiology				
Glenohumeral index	0.63 [0.03]	0.60 [0.05]	0.03	0.09
Distance SSP-LHBT (mm)	1.3 [1.7]	6.0 [2.5]	4.7	**<0.01**

Table 3. Comparison of the two identified etiologic subgroups after cluster analysis.
Cluster 1 included patients with a more extrinsic related etiology, more females, with a higher GH-index, a significantly lower LHB-SSP distance, more bursitis and significantly higher Bigliani classifications compared to cluster II, which contained patients with more positive signs for a (micro)instability related etiology.
((%), [SD], LHB: tendon of the long head of the biceps, SSP: Supraspinatus tendon)

4. Discussion

Our results show that in patients clinically diagnosed with SIS by experienced shoulder surgeons, MRA characteristics of various underlying causes can be found. And in many patients, SIS symptoms actually appear to be associated with specific other shoulder conditions, mistakenly diagnosed as SIS. All remaining patients had pathologic MRA characteristics, that were either associated with *extrinsic* impingement etiologic mechanisms (e.g. bony structures impinging on underlying tissues), or with *dynamic* motion-related impingement etiologic mechanisms (e.g. glenohumeral (micro)instability). Almost all patients showed signs of intrinsic pathology, either as a primary cause of SIS symptoms, or secondary to the other etiologic mechanisms.

It is important to note that the current study doesn't specifically advocate the use of MRA as an early step in the diagnostic process of SIS. And with the current results, it's not possible to design e.g. specific diagnostic criteria or diagnostic flow charts for SIS symptoms. The main goal was to thoroughly investigate a group of patients clinically diagnosed with SIS, for signs of various potential underlying mechanisms and other pathologies that can cause SIS symptoms. Our results show that SIS is indeed not a clear-cut and specific diagnostic label, despite its terminology, suggesting specificity and anatomic differentiation.

4.1 Heterogeneous findings in SIS patients

There is no consensus on a specific definition or a set of clinical or radiological characteristics that encompass SIS. Despite this, acromionplasty, which is based on the classic extrinsic etiologic theory (1972),[15] is a frequently performed surgery for SIS symptoms and one of the most performed orthopaedic surgeries in general.[51] However, its success rates are highly variable[52-55] and good clinical outcome has been reported for patients in whom the acromial shape is not altered.[20, 56-60] These contradicting results stress the need for a better understanding and better diagnostic strategies to identify the probable different phenotypes of shoulder pain that are often diagnosed as SIS, in order to target treatment to the principal underlying etiologic mechanism.

We found that 17 of 47 patients (36.2%) clinically diagnosed with SIS had specific other shoulder conditions with MRA. In the remaining 30 patients, all patients had positive signs of the predefined potential etiologic mechanisms. 28 (93.3%) Had signs of intrinsic etiology (either primary, or secondary to other underlying mechanisms). With cluster analyses, 2 main subgroups of patients with distinct MRA characteristics could be identified, with statistically significant differences between both groups. One cluster had primarily characteristics suggestive for structural extrinsic etiology and the other had primarily characteristics of dynamic (motion-related) causes, including glenohumeral (micro)instability. This supports the categorization in the predefined etiologic mechanisms. It is plausible that more tailored diagnostics and treatment strategies need to be developed for these and potential other underlying subgroups in patients with SIS symptoms.

4.2 Extrinsic etiologic mechanisms

According to the *classic* extrinsic mechanism, SIS symptoms can be the result of impingement of the acromion on the RC tendons. Several authors have reported a relation between a hooked acromion and SIS.[21, 59, 61-63] Accordingly, in our study, 24 (80.0%) patients had a type 2 or 3 acromion, which is much higher than the prevalence in the literature for asymptomatic individuals.[64] However, 22 of these patients also

had signs of other potential etiologic mechanisms, including AC osteoarthritis with subacromial osteophytes, or even glenohumeral (micro)instability (not extrinsic). Additionally, many authors have reported on the difficulties in assessing acromion shape and others contradict the often suggested relation between acromion and SIS.[12, 61, 65] This underlines that SIS symptoms are not necessarily always caused by a hooked acromion, and that the indications of acromionplasty might be more questionable than often suggested.

For the other (non-classic) extrinsic mechanisms, subcoracoid narrowing was found in 10 (33.3%) patients. But subcoracoid impingement is a clinical diagnosis and CH-narrowing on MRI should be related to clinical findings.[28] None of our patients had subscapularis tendon pathology with clinical and MRI evaluation.

With regards to AC-osteoarthritis, 12 (41.4%) patients had osteophytes impinging on subacromial tissues, of whom none had clinical symptoms pointing to AC-osteoarthritis (positive AC compression test, or pain with palpation of the AC joint). In support of this, a previous MRI study reported a prevalence of AC-osteoarthritis signs in 93% asymptomatic middle-aged individuals.[66] Although direct symptoms of AC-osteoarthritis itself can be absent, the condition can indirectly cause SIS symptoms due to actual impingement on the RC by caudal osteophytes.[23-25, 46]

Hébert et al. reported that a decreased acromiohumeral (AH) distance on MRI is associated with SIS.[44] Although often linked to a hooked acromion, AH narrowing can also be caused by e.g. glenohumeral (micro)-instability and/or pathologic kinematics. Hence, both diagnostic subgroups identified with our cluster analysis can, at least in theory, come with "structural" (extrinsic) or "dynamic" ((micro)-instability) AH narrowing. We found 19 (59.4%) patients with AH narrowing (<8.2mm). There was no difference in AH distance between both subgroups of the cluster analysis. In our view, the AH distance on MRI is a poor indicator for "classic" extrinsic SIS. This is supported by Mayerhoefer et al., who found no significant correlation between acromion morphology and AH distance.[43]

4.3 Intrinsic etiologic mechanisms

We found 28 (93.3%) patients with signs of intrinsic etiologic mechanisms. Various authors suggest intrinsic mechanisms can be a cause of SIS.[1, 67-69] But signs of intrinsic etiologies might also be secondary to other etiologic mechanisms. For example, it has been suggested that bursal side tendon tears suggest extrinsic etiology (i.e. impingement by bony structures), whereas articular side tears suggest intrinsic etiology.[67, 69] This could not be confirmed in our study where the majority of the observed partial tears were on the articular side, and not on the bursal side, where they would be expected if caused by encroachment by e.g. the acromion.[70] And overall, 27 of 28 patients with signs of intrinsic etiologic mechanisms also had characteristics of the other hypothesized etiologic mechanisms.

4.4 Dynamic impingement

Several authors have reported pathologic motion patterns and glenohumeral (micro)instability can lead to SIS, due to relative cranialisation of the humerus with respect to the scapula/acromion during arm motion.[36-39] We identified 19 (70.0%) patients with a GH-index (<0.61) and/or LHB-SSP distance (>4.0mm) suggestive for GH (micro)instability. In the final cluster analysis, a dynamic impingement subgroup of 17 patients could be identified, with an average GH-index of 0.60 and an average LHB-SSP distance of 6.0mm, both outside the confidence intervals of values reported for healthy asymptomatic subjects. Additionally, Bigliani acromion classifications were significantly lower in this subgroup, compared to the other identified ("extrinsic") subgroup. Although not investigated in this study, acromionplasty theoretically makes less sense as a treatment for patients with generally signs of (micro)instability instead of extrinsic causes.

4.5 Limitations

Our study is subject to some limitations. Firstly, the diagnosis of SIS, even by experienced shoulder surgeons, may be subjected to variability. Secondly, we did not investigate a control group, due to ethical considerations regarding exposure to contrast injections and MRA. We did, however, compare our results to 95% confidence intervals of control values available from the literature, obtained from healthy subjects. Thirdly, we applied control values of AH distance obtained from a study of Hebert et al.[44] They used an open MRI, where subjects were in erected position with the arm alongside the body (gravitational forces present). This could have led to a relatively larger AH compared to standard MRIs, as applied in our study. We were unable to find another publication reporting on MRI-based AH control values with SD's or confidence intervals. Overall, this could have implicated that the number of patients we classified as having a pathologic AH is overestimated. However, AH cut-off values were not used in the cluster analysis, which we used to identify potential etiologic patient subgroups. Fourthly, we were unable to find publications on GH-index values based on MRI. Therefore, we used the studies of McPherson (cadavers and radiographs) and Van Den Bogaert (cadavers and Computed Tomography).[48, 49] Lastly, the final group of SIS patients was relatively small, partially due to the high number of patients who appeared to have other specific pathologies with MRA evaluation, which is actually also an important outcome of our study. Despite the small number, we were able to identify MRA characteristics for various etiologic mechanisms and to classify patients into significantly different diagnostic subgroups.

5. Conclusions

Various pathologic MRA findings can be demonstrated in patients clinically diagnosed with SIS. These findings can be related with several underlying mechanisms that can cause SIS symptoms. Even more, in many patients specific other diagnoses (e.g. full-thickness RC tear, labrum abnormalities, glenohumeral ligament lesions and calcific tendonitis) can be found, possibly causing the SIS symptoms. With regards to etiologic mechanisms in patients without specific other shoulder conditions on MRA, some patients have predominantly signs associated with dynamic or motion related etiology (e.g. decreased GH index or increased distance between LHB and SSP tendons, which are signs of glenohumeral (micro)instability), whereas in others, there are predominantly signs associated with extrinsic etiology (e.g. hooked acromion). Practically all patients show signs of intrinsic problems (e.g. tendinosis), that can be either primary, or secondary to dynamic or extrinsic causes.

Hence, in patients clinically diagnosed with SIS, signs of various underlying mechanisms or even various underlying diseases can be found. This underlines SIS is a pain syndrome and not a specific diagnosis. The (wrongful) use of SIS as a specific diagnostic label is one of the explanations for the great variations in treatment outcomes of the many reported treatment strategies for SIS symptoms. We advise the use of additional imaging with e.g. ultrasound, MRI and/or MRA to further investigate underlying causes of symptoms when studying patients with "SIS" in a research setting, or when considering invasive treatment in case of persisting SIS symptoms in clinical practice.[71] Possibly, patients with predominantly signs of dynamic mechanisms need different treatment pathways than patients with signs of extrinsic mechanisms.

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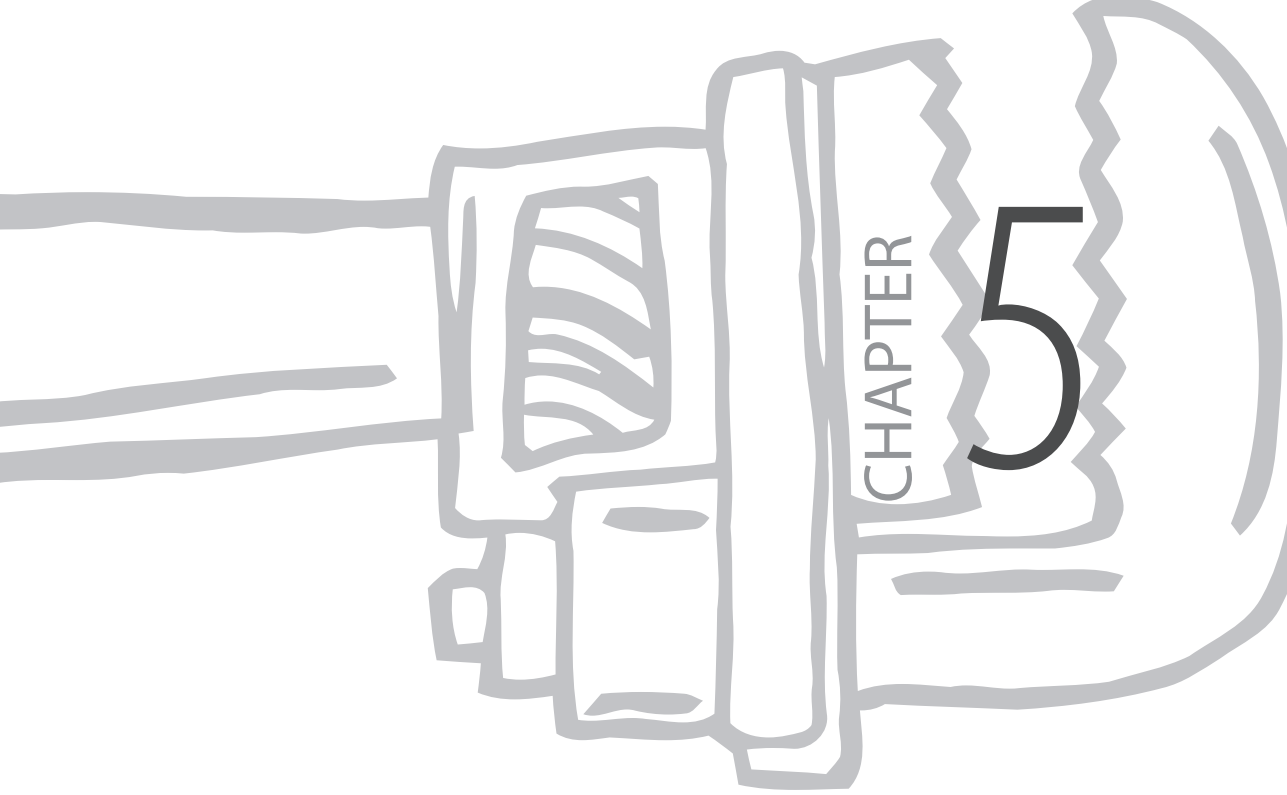
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Bursectomy versus acromionplasty in patients with subacromial impingement syndrome, - A prospective randomized study-

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Abstract

In a prospective randomized study we compared the results of arthroscopic subacromial bursectomy without changing the coracoacromial arch, versus an acromionplasty. A total of 57 patients (average age 47 (range, 31 to 60 years)) suffering from primary subacromial impingement without a rotator cuff tear who had failed previous conservative treatment were entered into the trial. At regular follow-up moments, the Constant Score, Simple Shoulder Test and VAS scores for pain and functional impairment were recorded. The type of acromion was classified according to Bigliani.

At a mean follow up of 2.5 years (range, 1 to 5) both bursectomy and acromionplasty gave good clinical results. No statistically significant differences were found between the two treatments. The severity of symptoms at baseline and type of type of acromion had a greater influence on the clinical outcome than the type of treatment. Patients with a more hooked acromion have a worse prognosis. The results of this study suggest that this is not necessarily solved by an acromionplasty.

Level of evidence: Level I, Randomized Controlled Clinical Trial

1. Introduction

The subacromial impingement syndrome has been described as symptomatic irritation of the rotator cuff and subacromial bursa. It is the most prevalent disorder of the shoulder in primary health care, accounting for 44-65% of all shoulder complaints.[1, 2] Subacromial impingement is characterized by pain, reduced range of motion and loss of power.[2-6] The different stages of the syndrome range from an, often reversible, "inflammation" of the rotator cuff tendons and subacromial bursa, to a complete rupture of the rotator cuff with secondary degenerative disease.[1, 7] A distinction can be made between primary and secondary impingement, where symptoms are caused by a specific shoulder disorder or adjoining physical problem such as instability, calcific tendinitis, or posttraumatic or acromioclavicular disorders.[8]

The etiology of the primary subacromial impingement syndrome is not clearly understood. Two main conflicting theories on the pathogenesis have been described in the literature: a mechanical (extrinsic) theory where symptoms are the result of compressive forces on the rotator cuff, and a degenerative (intrinsic) theory where symptoms result from tensile overload on degenerating rotator cuff tendons.

Neer's extrinsic impingement theory is widely accepted: impingement of the rotator cuff under the coracoacromial arch causes irritation of the subacromial tissues.[9, 10] The rationale for acromionplasty is based on this theory, which is supported by numerous studies that often suggest a correlation between the morphology of the acromial arch and the incidence and severity of the impingement syndrome.[5, 11-14]

The alternative intrinsic theory suggests that the symptoms of impingement are caused by a degenerative tendinopathy of the rotator cuff.[1, 15, 16] These degenerative changes may lead to a subacromial "inflammatory" reaction [17-20] and the symptoms result from tensile rather than compressive forces.

When conservative treatment fails, the classic surgical treatment of the impingement syndrome is acromionplasty. Over the years, satisfactory results have been reported in several studies with success rates ranging from 48 to 90% of patients.[21-25] However, in 2005, Budoff et al reported 79% good or excellent results of arthroscopic debridement without acromionplasty for partial thickness rotator cuff tears, with an average follow up of 114 months.[26]

In order to compare the results of debridement of the subacromial bursa (bursectomy) without acromionplasty with those of acromionplasty we performed a prospective randomized controlled study of the two treatments in patients without rotator cuff rupture who had failed conservative treatment for primary subacromial impingement syndrome. We hypothesized that bursectomy without changing the coracoacromial morphology would be equally effective as an acromionplasty.

2. Patients and Methods

Patients with non-traumatic shoulder complaints referred by primary health physicians were selected for this study. The diagnosis of subacromial impingement was based on patient history, clinical examination, radiographs of the shoulder (AP in external and internal rotation and Y scapular view) and the cervical spine and an MR arthrogram. The type of acromion was assessed according Bigliani's classification.[14] Subacromial impingement was diagnosed when patients complained of non-traumatic shoulder pain in the Deltoid region, with an inability to lie on the affected side. Clinically, pain was provoked by abduction, retroversion or internal rotation against resistance. All patients had full passive range of motion, a positive Neer[9] or Hawkins[27] sign and a positive lidocaine impingement test.

Patients were excluded if they had clinical signs of glenohumeral instability, or impairment of movement of the glenohumeral joint (i.e. adhesive capsulitis), osteoarthritis of the glenohumeral or acromioclavicular joint on the radiographs or MRI, rheumatoid disease, history of trauma or surgery to the shoulder, Biceps tendinitis, full- or partial-thickness rotator cuff tear, signs of cervical radiculopathy, or calcific tendinitis. Small calcifications near the greater tuberosity or acromion on radiographs were not a criterion for exclusion, nor was previous conservative treatment.

A total of 80 consecutive patients were studied. Following MR arthrography, a total of 14 patients were excluded because of full or partial rotator cuff lesions (n=7), labrum or superior labral anterior posterior (SLAP) lesions (n=7) and/or glenohumeral osteoarthritis (n=2).

Before surgery was considered, all patients followed a protocol of conservative treatment, consisting of three lidocaine and hydrocortisone injections into the subacromial space at four-week intervals, combined with NSAIDs and a period of physical therapy of at least six weeks. When conservative therapy failed, arthroscopy of the shoulder was performed by an experienced orthopaedic shoulder surgeon (ERAvA).

Following diagnostic arthroscopy, a further 9 patients were excluded (4 SLAP lesions, 3 glenohumeral arthritis, 3 glenohumeral synovitis, 1 partial rupture of the Supraspinatus tendon). Thus, 57 patients were included in the study and randomized for surgical treatment by an automatically generated randomization code. There were 26 men and 31 women with a mean age of 47 years (range, 31 to 60). Group A was treated with debridement of the subacromial bursa ("bursectomy alone"), and group B was treated with debridement of the subacromial bursa, followed by an arthroscopic acromionplasty ("acromionplasty").

The arthroscopic procedure was performed with the patient under general anesthesia in the lateral decubitus position and traction on the arm. A standard posterior portal was made with a second anterior portal in the rotator cuff interval and a third lateral portal to access the subacromial space. An arthroscopic pump was used. Diagnostic arthroscopy of the glenohumeral joint and subacromial space was performed and noted in a standardized manner.

After randomization, a complete debridement of the subacromial bursa was performed using a motorized shaver and an electrocautery probe (Opes, Arthrex, Naples, Florida, USA). In patients randomized for acromionplasty, a flat undersurface of the acromion was created by viewing from the posterior portal and introducing a motorized burr (Arthrex) through the lateral portal. In order to complete the bony resection, the portals were reversed, viewing from the lateral portal and introducing the burr through the posterior portal. After resection, electrocautery was used for haemostasis. Postoperatively, all patients undertook the same exercise program under guidance of a physical therapist.

Outcome measures in follow up were the Constant Shoulder Score (CS) (corrected for age and gender),[28, 29] the Simple Shoulder Test (SST)[30, 31] and visual analog scales (VAS) for pain and functional impairment varying from 1 (no pain/functional impairment) to 10 (maximum pain/functional impairment).[32] Outcome was assessed by an independent examiner at intervals of 3 months. Neither the examiner nor the patients were aware of the method of treatment. One patient was lost to follow-up because of lung cancer, diagnosed soon after the shoulder surgery.

The Medical Ethics Committee approved all stages of the study. Written informed consent was obtained from all patients.

Outcome measures from both groups were compared using Student's t-tests. Additionally, a multivariate analysis of variance was performed for each outcome score to compare both treatment groups and to analyze the influences of the type of acromion and confounding factors on final outcome measures. P-values <0.05 were considered to be statistically significant. Analyses were processed using SPSS software (SPSS inc., Chicago).

3. Results

Of the 57 patients, 56 were included in the study as one was lost to follow-up. The mean follow up was 2.5 years (range, 1 to 5), and the duration of symptoms before surgery was >1 year in 44 (77%) of the patients. The patient demographics including gender, age, body mass index, type of acromion and baseline clinical scores are shown in table 1.

Characteristic	Bursectomy	Acromionplasty
Male	9 (35%)	16 (53%)
Female	17 (65%)	14 (47%)
Age	43 [31 - 60]	50 [34 - 60]
BMI	27.9 [18.4 - 43.8]	25.5 [18.3 - 33.0]
Follow-up (months)	31.5 [11.0 - 58.3]	31.2 [11.3 - 63.3]
Type of Acromion*		
I	11	5
II	13	19
III	2	6
Baseline scores		
Constant Score	56 [19 - 85]	57 [27 - 82]
VAS pain	7.3 [5 - 10]	6.4 [2 - 10]
VAS functionality	6.9 [1 - 10]	6.4 [4 - 9]
Simple Shoulder Test	5.2 [0 - 13]	5.5 [1 - 10]

Table 1. Patient characteristics at baseline in both treatment groups.
[Range], *: according to Bigliani Classification, VAS: Visual Analogue Scale

After follow-up of one year, two patients treated with a bursectomy alone and three patients treated with an acromionplasty needed a second surgical procedure because of deteriorating symptoms. In the patients treated with a bursectomy alone, an acromionplasty was performed. Of the patients with an acromionplasty, one had a second more extensive acromionplasty and two had a resection of the acromioclavicular joint for treatment of degenerative disease. The follow-up data of these patients were included in the statistical analyses, up to the date of the second surgery.

The clinical scores for all patients in both groups improved. At final follow-up, the mean Constant Score for patients treated with a bursectomy was 69.6 points (SD=18.2), with a mean improvement of 13.9 points (SD=17.9). For patients treated with acromionplasty the mean Constant Score was 75.8 points (SD=16.7) with a mean improvement of 18.5 points (SD=17.5). The mean difference in improvement between the treatment groups was 4.6 points (SD=4.8) in favor of an acromionplasty (p=0.34, 95%-Confidence interval(CI): -14.1-4.9).

For the mean Simple Shoulder Test, patients treated with a bursectomy alone improved 3.8 (SD=3.6) points and patients treated with an acromionplasty 4.4 (SD=4.0) points, leading to a mean difference in improvement over time of 0. 6 points (p=0. 59, 95%-CI: -2.6-1.5) in favor of acromionplasty. The VAS-scores for both pain and functional impairment showed a mean improvement of 3 points in both groups (p=0.88 for pain, p=0.82 for functional impairment).(Table 2)

Final score	Bursectomy	Acromionplasty	Mean difference	p-value
Constant score				
Total score	69.6 (18.2)	75.8 (16.7)	-6.2 [-15.6 - 3.2]	0.19
Improvement	13.9 (17.9)	18.5 (17.5)	-4.6 [-14.1 - 4.9]	0.34
Simple Shoulder Test				
Total score	9.0 (4.0)	9.9 (3.7)	-0.9 [-3.0 - 1.1]	0.38
Improvement	3.8 (3.6)	4.4 (4.0)	-0.6 [-2.6 - 1.5]	0.59
VAS pain				
Total score	4.4 (2.7)	3.4 (2.5)	0.6 [-0.3 - 2.4]	0.13
Improvement	2.9 (2.6)	3.0 (3.1)	-0.1 [-1.4 - 1.6]	0.88
VAS functionality				
Total score	4.3 (2.8)	3.6 (2.4)	0.7 [-0.7 - 2.1]	0.32
Improvement	2.6 (2.8)	2.8 (3.1)	-0.2 [-1.4 - 1.8]	0.82

Table 2. Mean final scores, mean improvement in follow-up period and mean differences between both treatment groups.
(SD), [95%-Confidence Interval], VAS: Visual Analogue Scale

As the type of acromion, gender, age and baseline scores were not equally distributed in the treatment and acromion subgroups, a multivariate analysis of variance was performed to study factors of influence on the clinical scores. Separate analyses were performed for the baseline scores and the scores at final follow-up. Type of treatment and gender were used as factors. Type of acromion and baseline score were applied as covariates. The type of acromion was considered a linear (ordered) covariate in order to detect a "dose-response" relation between the type of acromion and outcome measures.

The mean baseline Constant Score was 56.6 points (SD=15.3). The multivariate analysis demonstrated that male gender had a mean positive effect of 12 points (p=0.003, 95%-CI: 4.3-20.1) on the baseline score. For all other variables no statistical significance was reached and confidence intervals did not, in our opinion, contain any clinically relevant values.(Table 3)

The average final Constant Score was 73 points (SD=17.6). The baseline preoperative CS had, on average, a statistically significant effect of 0.43 (p=0.007, 95%-CI: 0.1-0.7) on the final Constant Score, meaning that a difference between patients of 10 points on the baseline score had, on average, an effect of 4.3 points on the final Constant Score. Type of acromion had on average a negative effect of 6.3 points (p=0.08, 95%-CI: -13.4-0.7) on the final Constant Score. A type III acromion scored on average 12.6 points less compared to a type I acromion. There was no indication that type of acromion had any influence on the effect of the treatment performed (p=0.53), meaning that there was no indication that a patient with a type III acromion had a greater advantage if treated by acromionplasty than did a patient with a type I acromion.

For the simple shoulder test, the mean baseline score was 5.3 points (SD=3.3). Male gender was the only factor with a nearly statistically significant effect on the

baseline score of 1.8 points (p=0.05, 95%-CI: -0.02-3.56).(Table 3) The mean final simple shoulder test score was 9.5 points (SD=3.8). Baseline score had a statistically significant effect of 0.43 points (p=0.004, 95%-CI: 0.1-0.7) on the final score per additional baseline score point. The type of acromion had a statistically significant negative effect of 1.6 points (p=0.04, 95%-CI: -3.2 - -0.1) on the final SST score, meaning that a type III scored 4.8 points less on average than a type I acromion at the final follow-up. All other variables had no statistically significant effect on the final simple shoulder test score with confidence intervals not containing any clinically relevant values.(Table 4) Furthermore, there was no indication that the type of acromion had any influence on the effect of the treatment performed (p=0.76).
The mean baseline VAS for pain and functional impairment was 6.8 points (SD=1.6) and 6.6 points (SD=1.7) respectively. There were no statistically significant and clinically relevant effects of all considered variables on the baseline scores.(Table 3) The mean final VAS was 3.9 points (SD=2.6) for pain and 3.9 points (SD=2.6) for functional impairment. The type of acromion had an effect of 1.1 point (p=0.06, 95%-CI: -0.06-2.27) on the VAS score for pain at final follow-up. For the VAS score for functional impairment, the type of acromion had a statistically significant effect of 1.5 points (p=0.01, 95%-CI: 0.36-2.63), indicating that a type III acromion scored 4.5 points higher than a type I acromion. All other variables had no statistically and clinically relevant effect on the final VAS.(Table 4) Furthermore, on neither VAS was there any indication that the type of acromion had any influence on the effect of treatment performed (p=0.22 for pain, p=0.28 for functional impairment).

Clinical Score	Baseline score	Variable	Effect	95%-CI	p-value
Constant Score	56.6 (15.3)	Type of acromion	-0.6	[-7.0 - 5.8]	0.84
		Gender	12.2	[4.3 - 20.1]	**< 0.01**
		Age at surgery	-0.32	[-0.9 - 0.2]	0.25
Simple Shoulder Test	5.3 (3.3)	Type of acromion	-0.5	[-2.0 - 0.9]	0.45
		Gender	1.8	[0.0 - 3.6]	0.05
		Age at surgery	-0.02	[-0.1 - 0.1]	0.74
VAS pain	6.8 (1.6)	Type of acromion	0.2	[-0.5 - 0.9]	0.58
		Gender	-0.1	[-1.0 - 0.8]	0.87
		Age at surgery	-0.04	[-0.1 - 0.0]	0.24
VAS functionality	6.6 (1.7)	Type of acromion	-0.2	[-1.0 - 0.5]	0.54
		Gender	-0.1	[-1.0 - 0.9]	0.90
		Age at surgery	-0.01	[-0.1 - 0.1]	0.85

Table 3. Analysis of variance of baseline parameters on baseline scores.
Type of acromion (per one step up in Bigliani classification), gender (male vs. female) and age at surgery (per 1 year up) were used as factors and covariates.
(SD), 95%-CI: 95%-Confidence Interval, VAS: Visual Analogue Scale

Clinical Score	Final score	Variable	Effect	95%-CI	p-value
Constant Score	72.9 (17.6)	Baseline score	0.4	[0.1 - 0.7]	**< 0.01**
		Treatment	4.9	[-4.7 - 14.4]	0.31
		Type of acromion	-6.3	[-13.4 - 0.7]	0.08
		Gender	5.0	[-4.4 - 14.3]	0.29
		Age at surgery	0.33	[-0.3 - 1.0]	0.30
Simple Shoulder Test	9.5 (3.8)	Baseline score	0.4	[0.1 - 0.7]	**< 0.01**
		Treatment	0.8	[-1.3 - 2.9]	0.45
		Type of acromion	-1.6	[-3.2 - -0.1]	**0.04**
		Gender	0.7	[-1.2 - 2.7]	0.44
		Age at surgery	0.07	[-0.1 - 0.2]	0.31
VAS pain	3.9 (2.6)	Baseline score	0.1	[-0.3 - 0.6]	0.52
		Treatment	-0.9	[-2.6 - 0.7]	0.25
		Type of acromion	1.1	[-0.1 - 2.3]	0.06
		Gender	-0.3	[-1.7 - 1.1]	0.64
		Age at surgery	-0.05	[0.2 - 0.1]	0.32
VAS functionality	3.9 (2.6)	Baseline score	0.2	[-0.2 - 0.6]	0.30
		Treatment	-1.0	[-2.5 - 0.5]	0.20
		Type of acromion	1.5	[0.4 - 2.6]	**0.01**
		Gender	0.1	[-1.3 - 1.5]	0.87
		Age at surgery	-0.03	[-0.1 - 0.1]	0.57

Table 4. Analysis of variance of final scores.
Baseline score (per 1 point up), type of treatment (acromionplasty vs. bursectomy), type of acromion (per one step up in Bigliani classification), gender (male vs. female) and age at surgery (per 1 year up) as factors and covariates.
(SD), 95%-CI: 95%-Confidence Interval, VAS: Visual Analogue Scale

4. Discussion

In this prospective randomized controlled study, both bursectomy alone and bursectomy with acromionplasty improved clinical scores after a mean follow-up of 2.5 years. There was a tendency for a better result from an acromionplasty with bursectomy over a bursectomy alone, but the differences in the clinical scores were small and in our opinion not clinically relevant. There was no evidence that a patient with a Bigliani type III acromion had more advantage from treatment with an acromionplasty than a patient with a type I acromion. On the other hand, our results suggest that the type of acromion has more effect on the clinical outcome than the type of surgical treatment. Thus, in this study less hooked types of acromion (i.e. type I) led to a better clinical outcome, whatever the surgical procedure. This effect was statically significant for the SST and VAS-score for functional impairment but clinical relevance of these effects is questionable, in particular for the effect on the SST with 1.6 points per step up in Bigliani classification. Furthermore, the pre-operative baseline scores appeared to have a statistically significant effect on the final outcome in both the Constant Score and the Simple Shoulder Test. Overall,

the effects of baseline scores, type of treatment, type of acromion, gender and age at surgery were small on all clinical scores. Even when statistical significance was reached, effects were not, in our opinion, clinically relevant.

Our findings challenge Neer's widely accepted extrinsic theory of impingement, where subacromial tissues abut under the coracoacromial arch, leading to inflammation and damage to these tissues.[9, 10]

In 1986, Bigliani introduced a classification system for the shape of the acromial. A type I acromion has no curvature (flat type). A type II acromion has a curvature in the middle third of the acromion (curved type) and a type III acromion shows down-sloping on the anterior third (hooked type).[14, 33, 34] Several studies suggested a correlation between the shape of the acromion and the severity of impingement symptoms.[5, 12] Epstein et al. described a tendency for a higher prevalence of type III acromion in patients diagnosed with impingement[33] and Hirano et al. reported that rotator cuff tears were significantly larger with a type III acromion, compared to types I and II.[35] However, Liotard et al. concluded there is no such relationship[36] and others have underlined difficulties in using the Bigliani classification because of the high intra- and interobserver variance.[35, 37, 38]

Several studies conflict with the extrinsic theory of impingement. First of all, the term "bursitis" or "tendinitis", often used as synonym for the impingement syndrome, is not supported by histological studies of the subacromial bursa or rotator cuff in patients with impingement. Degeneration and fibrosis of the subacromial bursa and rotator cuff have been observed and not inflammation.[1, 15, 16] Furthermore, several studies showed a relationship between the amount of fibrosis and the severity of symptoms.[17-20]

Secondly, the majority of partial tears of the rotator cuff are either intratendinous or found on the articular side of the rotator cuff and not on the bursal side where they would be expected if the rotator cuff "impinged" on the acromion.[39]

Thirdly, McCallister et al. described substantial improvement in shoulder comfort and function following rotator cuff repair without acromionplasty, with results comparable to studies where the repair of the rotator cuff was combined with an acromionplasty.[40] Additionally, several studies concluded that an acromionplasty does not influence further degeneration of the rotator cuff in the long term.[41, 42]

Finally, a higher incidence of type III acromion has been observed in older people than in young asymptomatic athletes.[43, 44] Ozaki et al. reported in a cadaver study, that rotator cuff pathology predates spur formation of the acromion.[45] Additionally, spur reformation has been described following acromionplasty.[46, 47] These findings suggest that changes of the acromion are, at least in some patients, more the result of rotator cuff pathology than its cause. The findings in the current study

that patients with a more hooked acromion had more symptoms before and after surgery, also suggest that the type of acromion is part of a degenerative process where more hooked acromions are seen in the later stages of impingement.

In this study, Neer's impingement test was used as a definite criterion for localized subacromial pain. Most studies that report the results of an acromionplasty consider the lidocaine impingement test to be the gold standard for the diagnosis of subacromial impingement. An unexpected finding in our study was that 14 of 80 patients (17.5%) diagnosed as having subacromial impingement with a positive impingement test, had to be excluded following MR arthrography and arthroscopy because of specific alternative shoulder pathology, mainly localized in the glenohumeral joint. These findings may be explained by the low specificity of Neer's impingement test, such that a lidocaine injection into the subacromial space does not differentiate adequately between an impingement syndrome and other shoulder pathology. Furthermore, we performed the impingement test without radiological control. The inaccuracy of this procedure may also explain the high number of patients that had to be excluded following the MR arthrography and arthroscopy.[48]

The good results of a bursectomy alone are in agreement with the study of Budoff et al., who reported 79% good or excellent results of an arthroscopic debridement without acromionplasty for partial thickness rotator cuff tears.[26] These and our results are comparable with those of many studies describing the results of acromionplasty. This raises the question whether an acromionplasty actually has added value.

A possible explanation for the improvement seen after bursectomy alone might be that subacromial bursal tissues contain a rich supply of nociceptive nerve fibers compared with other structures around the shoulder joint.[49-51] Mechanical irritation may cause recruitment of nerve fibers, or lowering of the threshold of those nerve fibers as is seen in the Achilles tendon or facet joints of the spine.[50, 52, 53] This might also explain the chronic nature of symptoms in patients with impingement syndrome, even if they limit the use of their shoulder for a long period of time.

There are several limitations to this study. Firstly, despite randomization, there were differences in distribution of age, gender and the type of acromion in the two treatment groups. We corrected for these differences in our statistical analyses in order to preclude potential confounding. Additionally, we did not only assess total scores but also evaluated difference scores in order to correct for baseline scores in t-tests. Also, with randomization, not only known but also unknown confounders are divided between both groups. Secondly, the size of our study group was relatively small. Therefore, our results should be interpreted with care. Thirdly, the

Simple Shoulder Test may not be suitable for detecting differences between the treatment groups due to its dichotomous nature of the test, and the limited number of questions.[54]

We believe that the pathogenesis of the subacromial impingement syndrome can be explained by several mechanisms. Neer's extrinsic theory on impingement might be true for a subgroup of patients with subacromial pain, but in this study we were unable to identify such a group.

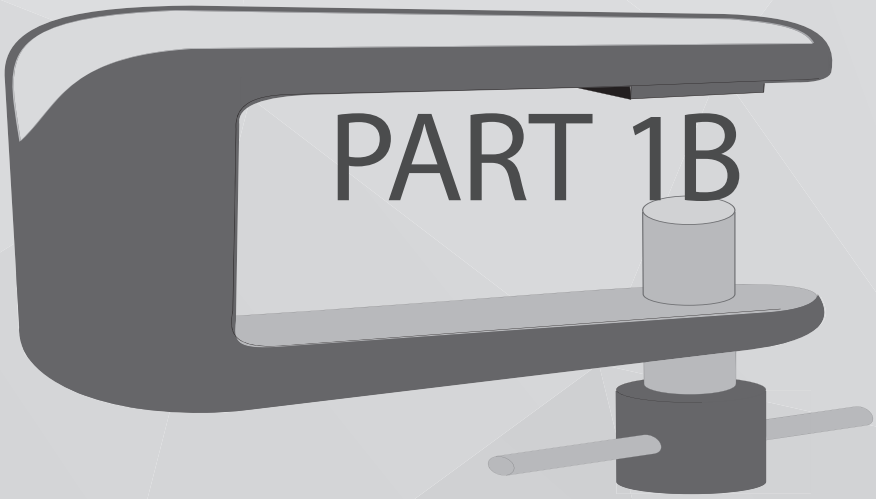
We conclude that both a bursectomy and an acromionplasty can give good clinical results in patients with primary subacromial impingement, who fail conservative treatment. There was a small difference in favor of acromionplasty but this did not reach clinical relevance in this relatively small study. The type of acromion and the severity of symptoms have a larger influence on the final outcome, than the type of surgical treatment. Patients with a more hooked acromion have a worse prognosis and the results of this study suggest that this may not be solved by an acromionplasty.

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PART 1B

Calcific Tendinitis; a frequent cause of “impingement“ symptoms





Rotator cuff calcific tendinitis and long-term outcome: demographic and radiological predictors

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Abstract

Background: Knowledge on the epidemiology and long-term course of Rotator Cuff Calcific tendinitis (RCCT) is scarce. We assessed demographics, radiological characteristics and treatment, and their association with long-term outcome in a large patient group.

Methods: Medical records of 342 patients with RCCT were reviewed. Baseline demographics, radiological characteristics and treatment (barbotage vs. conservative) were recorded. Inter-observer agreement of radiological measures was analyzed. Long-term outcome was evaluated with the Western Ontario Rotator Cuff index (WORC) and the Disabilities of the Arm, Shoulder and Hand score (DASH). The association of baseline characteristics with long-term outcome was assessed.

Results: Mean age at diagnosis was 49.0 (SD=10.0) years, 59.5% were female, in 66.0% the dominant arm was affected and 21.3% had bilateral disease. The Supraspinatus (85.2%) was predominantly affected. Calcifications were on average 18.7mm (SD=10.1) in size (ICC=0.84 ($p<0.001$)), located 10.1mm (SD=11.8) medial to the acromion (ICC=0.77 ($p<0.001$)). 32.1% had a Gärtner type I calcification (Kappa=0.47 ($p<0.001$)).

With a mean follow-up of 14 years (SD=7.1), median WORC was 72.5 (range, 3.0-100.0) and median DASH 17.0 (range, 0.0-82.0). 55% had a WORC<80 and 42% a WORC<60. Female gender, bilateral disease, dominant arm involvement, longer duration of symptoms and multiple calcifications were associated with inferior long-term WORC. There were no significant effects of treatment method. The DASH showed similar results.

Conclusions: Many RCCT patients have an impaired shoulder function years after diagnosis, regardless of applied treatment. Female gender, dominant arm involvement, bilateral disease, longer duration of symptoms and a higher number of calcifications at presentation are associated with inferior long-term outcome. These findings can be taken into account in clinical decision making and might be helpful in preventing long-term symptomatic course.

Level of evidence: Level II, retrospective prognostic study.

Keywords: rotator cuff; calcific tendinitis; treatment; barbotage; long-term outcome; epidemiology.

1. Introduction

Rotator cuff calcific tendinitis (RCCT) is frequently diagnosed in patients with shoulder pain; reported incidence rates range from 6.8-54%.[1-4] However, information on its epidemiology, prognostic factors and long-term course is scarce. In current literature, generally small populations are assessed with short follow-up periods. This is the first study to assess long-term shoulder function in a large group of RCCT patients, treated with either barbotage (needling and lavage) or conservative methods. Additionally, patient demographics, radiological characteristics, inter-observer agreement of radiological characteristics, and prognostic factors are evaluated.

Typical RCCT symptoms are pain in the deltoid region, with variable functional impairment [5-7] and variable duration of symptoms, ranging from months to years.[8-10] Treatment of these generally self-limiting symptoms is usually conservative with e.g. non-steroidal anti-inflammatory drugs (NSAIDs) and physical therapy. In case of persisting or severe symptoms, more invasive treatments can be applied, including corticosteroids injections, barbotage, Extracorporeal Shock Wave Therapy (ESWT) or surgery.[4-7, 11-35] Only few of these studies compared various treatments and their long-term effects. It is also unclear which patients will follow a mild and self-limiting course, and who might benefit of more rigorous follow-up and treatment strategies. Consequently, clinical decision making and applied treatments are often based on personal experience and regional preferences.

With regard to the epidemiology of RCCT, several etiologies have been reported, including active cell-mediated calcification, RC degeneration, RC overuse and micro-trauma, genetic predisposition, local metabolic or hemodynamic abnormalities, and RCCT as a consequence of subacromial impingement.[4, 36-42] Based on these theories, RCCT would predominantly affect the dominant arm or both arms in subjects with suboptimal vascular status (e.g. middle to older age, diabetes, or smokers) with frequent overhead activities. However, this has not been confirmed in clinical studies.

Radiological characteristics of RCCT, including the number, size, appearance and location of calcifications, have been associated with clinical outcome by some[3, 21], but this association has been disputed by others.[14, 21, 43-45] There is little knowledge on radiological calcification characteristics in large patient groups and their association with long-term outcome. Furthermore the inter-observer agreement of most radiological measures for RCCT has not been evaluated.

In this study, we assessed demographic and radiological characteristics at baseline, and long-term shoulder function in a large group of RCCT patients treated either with barbotage (i.e. needling) or with conservative modalities. Our objectives were

to: 1) evaluate baseline demographics and radiological characteristics of these RCCT patients and their association with long-term shoulder function; and 2) evaluate inter-observer agreement of common radiological RCCT measures. More knowledge on these factors may help in predicting patients' prognoses and in clinical decision making, i.e. when considering more invasive treatments methods for patients with negative prognostic factors.

2. Materials and Methods

2.1 Study Population and baseline RCCT characteristics

Since 1980, patients referred to the outpatient clinic of the department of Orthopaedics at the Leiden University Medical Center received a medical diagnosis code. With these codes, all patients diagnosed with RCCT in the period of January 1980 until November 2009 were identified. During most of this period, our institution was considered a center of expertise with regard to the treatment of RCCT and one of few regional institutions performing barbotage.

Medical records and radiology reports of identified patients were reviewed for eligibility criteria and data collection by the principal investigator, who was not involved in patient care. Patients were included if RCCT was demonstrated on available radiographs and/or noted in the radiology reports, and when aged ≥18 years at time of diagnosis. Patients were excluded in case no medical records, radiographs or radiology reports were available, or if the diagnosis RCCT was not mentioned in these records.

Accordingly, 420 patients were identified with the RCCT diagnosis code. 78 were excluded because no definite confirmation of RCCT could be made after reassessing all available medical and radiology records (radiographs and radiology reports), or because of <18 years, leaving 342 confirmed RCCT patients available for analysis of baseline characteristics.(See study flowchart, figure 1) These 342 patients were the source population for the follow-up part of our study.

The following baseline data were recorded from the medical records: affected side(s), age, gender, date of diagnosis, age at diagnosis, type of treatment (barbotage, or conservative treatment (standard conservative treatment in our country at the time included physical therapy, NSAIDs and/or subacromial corticosteroids injections)), duration of symptoms at presentation, diabetes, tendon problems at other sites, systemic inflammatory diseases and other systemic or musculoskeletal diseases.

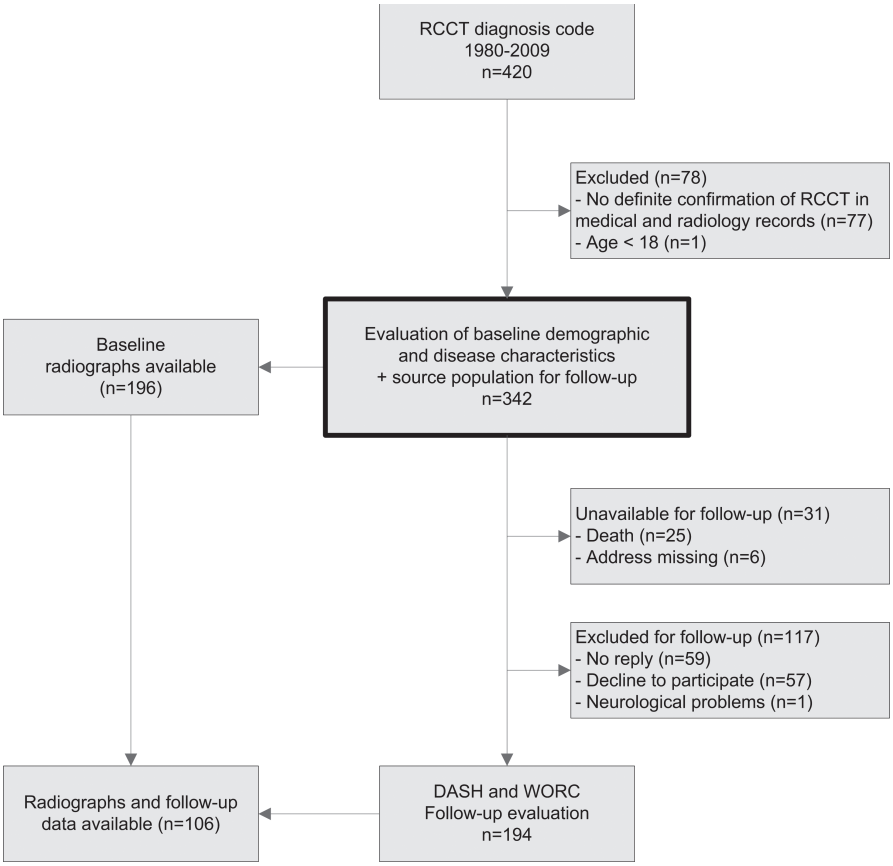


Figure 1. Study flowchart.

2.2 Follow-up and questionnaires

Addresses of the 342 patients and data on patients' death were checked using the municipal personal records database. All subjects living in our country at the time were contacted by mail for completion of a general information form; the Western Ontario Rotator Cuff index (WORC), which is specifically developed to assess shoulder function and quality of life of patients with cuff disorders; and the Disabilities of the Arm, Shoulder and Hand score (DASH).[46-48] Also arm dominance, any diseases for which medication was currently used, medical care history and diseases affecting the shoulder and arm function were noted. Patients indicating the latter were excluded from further analyses. Reminders were sent after 4 and 8 weeks to all subjects from whom no reply was received.

Of the 342 confirmed RCCT patients, 31 could not be contacted because of death (n=25) or missing address (n=6). Of the remaining 311, 252 (81.0%) replied: 57 refused to participate and 1 was excluded from the questionnaire analyses because

of current neurological arm problems. Final positive response rate was 62.4% (194 of 311 contacted subjects). Of these subjects, 14 did not complete all required WORC items and 26 not all DASH items, leaving 180 and 168 patients, respectively for final WORC and DASH follow-up analyses.
Demographic baseline data of the available (responders) and non-available patients are depicted in Table 1. As all subjects were contacted from 2011 onwards, minimum follow-up was 2 years. All responders gave written informed consent and the study was approved by the Medical Ethics Committee of the Leiden University Medical Center.

2.3 Baseline radiological characteristics, inter-observer agreement and association with long-term outcome

Radiographs acquired within 1 year of the date of diagnosis and before eventual barbotage were used for evaluation of baseline calcification characteristics. Due to national regulations, radiographs older than 15 years were generally destroyed. In total, radiographs were available of 204 shoulders in 196 patients.
Radiographs were evaluated independently by two trained researchers, who were blinded for clinical status of the patients. In a consensus meeting, final radiological outcome measures (see below) were determined for each subject. In case of disagreement, radiographs were re-evaluated by an experienced musculoskeletal radiologist, serving as an adjudicator.
Affected tendon(s), *Size* (mm) and number of calcifications per shoulder were recorded on standard anteroposterior (AP) (internal and external rotation) and axial radiographs. Locations of all calcific deposits in each shoulder were further categorized using the system of Ogon et al., which we refer to as *Location*.[3] With this method, a line is drawn from the lateral border of the acromion, parallel to the glenoid, on external rotation AP radiographs. *Location* is the distance (mm) between this line and the medial border of the calcification.(Figure 2) Negative values represent a medial calcification border with respect to the drawn line. More subacromial extension (negative *Location* value) has been reported a negative prognostic factor.[3]
Calcific deposits were also assessed using *Gärtners classification*: deposits with a sharp border and a dense structure are type I calcifications; type II calcifications either have a sharp border and inhomogeneous structure or a vague border and a homogenous structure; type III calcifications have a vague border, are more or less transparent in structure and have a cloudy appearance.[49] (Figure 3) These different types allegedly display the natural course of RCCT and have been reported valuable in determining patients' prognosis.[21]

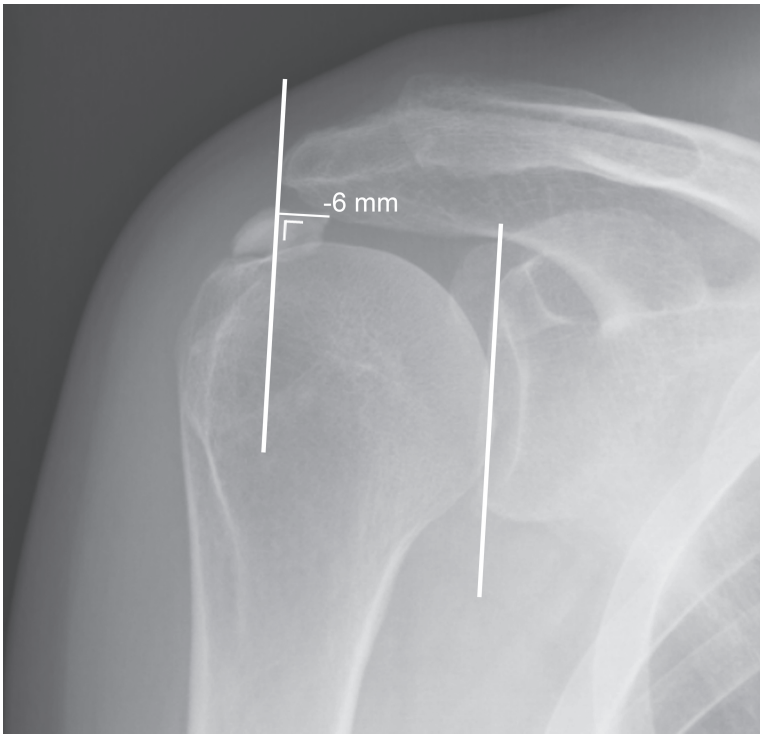


Figure 2. Locations of the calcific deposits were evaluated using the system of Ogon et al., which we refer to as *Location* in this paper.[3]
A line perpendicular to the most lateral border of the acromion is drawn, parallel to the glenoid, on external rotation AP radiographs. *Location* is the distance (mm) between this line and the medial border of the calcification, where negative values represent a medial calcification border between the glenoid and the drawn line.

For assessing inter-observer agreement, metric measures (*Size* and *Location*) and *Gärtner classifications* of all available radiographs (analogue and digital) and all calcifications were used (n=248). To evaluate the association of baseline radiological characteristics with long-term outcome, characteristics of the largest calcification per patient were used. For these analyses, all radiographs could be used with regard to *Gärtner classification*. However, metric measures (i.e. *Size* and *Location*), only available digital radiographs (n=50) could be used for these specific analyses, as only their magnification factor was known and consistent.

Figure 3. Examples of Gärtner calcification classification types.49



A) Gärtner type I: sharp border and a dense structure;



B) Gärtner type II: either a sharp border and an inhomogeneous structure or a vague border and a homogenous structure;



C) Gärtner type III: a vague border, more or less transparent in structure and a cloudy appearance.

2.4 Statistical analysis

Demographics and disease characteristics were expressed using proportions, means and standard deviations, or medians and ranges where appropriate. Data distributions were evaluated using histograms. Questionnaire data were processed in a similar way.

For calcification characteristics, inter-observer agreement was assessed with the Kappa statistic for *Gärtner classifications*, and with paired t-tests and the intraclass correlation coefficient (ICC) for *Size* and *Location*.

The association of baseline characteristics with long-term shoulder function was assessed using the WORC as primary outcome. Using logistic regression (because of skewed outcomes for DASH and WORC scores), the univariate association of each recorded variable with inferior outcome was evaluated and expressed in odds ratios (OR) with 95% confidence intervals (95%-CI). WORC-scores ≥80 were defined as good outcome. Similarly, DASH scores ≤20 were regarded as good outcome. Sensitivity analyses were performed for alternative WORC and DASH cut-offs.

To gain more insight in independent prognostic factors, multivariable logistic regression models were constructed for the WORC and the DASH. In order to avoid overfitting, no more than 10% of the number of events were included as covariates

in each model. Variables were selected based on clinical relevance and the univariate results (p-values $\leq$0.05). Because of missing data on duration of symptoms at presentation in several patients and the limited number of available digital baseline radiographs, associated variables were not entered in the multivariable models.
Statistical analyses were performed using IBM SPSS statistics version 20.0 (IBM, Armonk, New York, USA).

3. Results

3.1 Baseline demographics and disease characteristics

Of 342 RCCT patients, 203 (59.5%) were female. Mean age at diagnosis was 49.0 years (SD=10.0). 73 patients (21.3%) had bilateral disease. Overall, 200 (58.5%) patients underwent barbotage.(Table 1)
With regard to concomitant pathologies, 17 patients were diagnosed with diabetes, 7 with kidney disorders, 4 with thyroid disorders, 2 had acromegaly and 1 was HIV-positive. Concomitant tendon disorders were mentioned in the records of 15 patients (4.4%): 11 had had an episode of lateral epicondylitis of the elbow, 2 had calcifications of the Achilles tendon, 1 had Biceps tendinitis and 1 had fasciitis plantaris.

	All subjects		**Responders**		**Non-responders**	
	n=342		n=194		n=148	
Female gender	203	59.5%	115	59.3%	88	59.5%
Age	49.0	[21 - 83]	48.4	[29 - 83]	49.8	[21 - 82]
Duration of symptoms (median, months)	23.5	[0 - 196]	18.0	[0 - 192]	24.0	[0 - 196]
Diabetic	17	5.0%	10	5.2%	7	4.7%
Affected side						
Right	161	47.1%	88	45.4%	73	49.3%
Left	99	28.9%	49	25.3%	50	33.8%
Both	73	21.3%	49	25.3%	24	16.2%
Missing	9	2.6%	8	4.1%	1	0.7%
Arm dominance						
Right	NA	NA	165	85.9%	NA	NA
Left	NA	NA	21	10.9%	NA	NA
Other	NA	NA	6	3.1%	NA	NA
Dominant side affected	NA	NA	128	66.0%	NA	NA
Treatment						
Barbotage	200	58.5%	121	63.4%	79	53.7%
Conservative	142	41.5%	73	36.6%	69	46.3%

Table 1. Baseline demographics and disease characteristics.
Data are displayed for all included subjects, and stratified for subjects returning follow-up questionnaires (responders) and non-responders.
[Range]

In the 196 patients (248 calcifications, i.e. bilateral and multiple calcifications) with available baseline radiographs, the Supraspinatus tendon was affected in 167 patients (85.2%). 63 (32.1%) had a Gärtner I calcification. Mean calcification *Size* of the largest calcification for each shoulder was 18.7mm (SD=10.1), with a mean *Location* of -10.1mm (SD=11.8).(Table 2)

3.2 Interobserver agreement of radiological RCCT measures

For inter-observer agreement, mean difference between observers for *Size* measurements was 0.11mm (95%-CI: -0.46–0.67; p=0.71) and for *Location* 0.08mm (95%-CI: -1.16–1.00; p=0.89), with ICCs of 0.84 (p<0.001) and 0.77 (p<0.001), respectively. Kappa-value for the *Gärtner classification* was 0.47 (p<0.001).

	All subjects		**Responders**		**Non-responders**	
	n=196		n=106		n=90	
Affected tendon(s)						
Supraspinatus	167	85.2%	92	86.8%	75	83.3%
Infraspinatus	33	16.8%	19	17.9%	14	15.6%
Subscapularis	34	17.3%	21	19.8%	12	13.3%
Gärtner						
1	63	32.1%	34	32.1%	29	32.2%
2	111	56.6%	65	61.3%	46	51.1%
3	69	35.2%	38	35.8%	31	34.4%
Size (mm)	18.7	[10.1]	18.7	[9.8]	18.6	[10.8]
MedLat (mm)	-10.1	[11.8]	-10.7	[11.6]	-9.1	[12.6]

Table 2. Baseline data obtained from available analogue (n=154) and digital radiographs (n=50). There were 248 calcifications in 204 shoulders in 196 patients (multiple calcifications in 33 shoulders) with radiographs available.
[SD]

3.3 Long-term shoulder function

The 194 subjects responding to the follow-up questionnaires had a mean follow-up of 14 years (SD=7.1, range 2-33). Mean current age was 62 (SD=9.2, range 39-89) years. Median WORC was 72.5 (range, 3.0-100.0) and median DASH 17.0 (range, 0.0-82.0). For the WORC, 99 of 180 available subjects (55.0%) had a WORC <80 and 76 (42.2%) a WORC even below 60.(Figure 4A) Univariate analyses demonstrated that patients with female gender, longer duration of symptoms at presentation, bilateral disease and dominant side involvement had statistically significant lower long-term outcome (WORC<80).(Table 3)

106 Subjects had both baseline radiographs and clinical scores available. Results of univariate logistic regression analyses with radiological parameters are depicted in tables 2 and 3. Number of calcifications (per shoulder) had an OR=2.1 (95%-CI: 0.97-

4.62) for WORC<80, indicating that a larger number of calcifications was associated with inferior long-term shoulder function in our data.

The final multivariate WORC model included gender, age at follow-up, years after diagnosis, bilateral disease, dominant side involvement and treatment method. Female gender had a significant negative effect: OR=2.2 (95%-CI: 1.1–4.2). The effect sizes for bilateral disease (OR=2.2 (95%-CI: 0.94–5.1)) and dominant arm involvement (OR=1.7 (95%-CI: 0.79–3.6)) also indicated relevant negative effects, but did not reach statistical significance. There was no significant association for WORC outcome at the last follow-up and applied treatment method, either barbotage or conservative.(Table 3) Sensitivity analyses using WORC cut-off points <70 and <90 gave similar results (data not shown).

WORC < 80		**Univariate**			**Multivariate**	
	OR	95%-CI	p	OR	95%-CI	p
Female gender	1.82	0.99 - 3.35	**0.05**	2.16	1.11 - 4.20	**0.02**
Age diagnosis	1.00	0.97 - 1.04	0.83			
Age questionnaire	1.02	0.98 - 1.05	0.36	1.00	0.96 - 1.04	0.85
Years after diagnosis	1.02	0.98 - 1.06	0.35	0.99	0.94 - 1.04	0.68
Diabetic	3.01	0.61 - 14.89	0.18			
Duration of symptoms (per additional month)	1.02	1.00 - 1.03	**0.01**			
Bilateral disease	2.63	1.24 - 5.57	**0.01**	2.18	0.94 - 5.10	0.07
Dominant side affected	2.00	1.01 - 3.96	**0.05**	1.69	0.79 - 3.60	0.18
Affected tendon(s)						
Supraspinatus	0.73	0.23 - 2.31	0.59			
Infraspinatus	1.07	0.43 - 2.64	0.89			
Subscapularis	1.17	0.32 - 4.26	0.82			
Treatment						
Barbotage	1.00	0.54 - 1.86	0.99	1.13	0.56 - 2.28	0.74
Calcification location: MedLat (mm)	0.98	0.92 - 1.06	0.63			
Calcification size (mm)	0.98	0.91 - 1.06	0.64			
Gärtner calcification classification			0.87			
1	Ref.					
2	0.93	0.36 - 2.39	0.89			
3	1.19	0.42 - 3.36	0.75			
Number of calcifications (per additional deposit)	2.12	0.97 - 4.62	0.06			

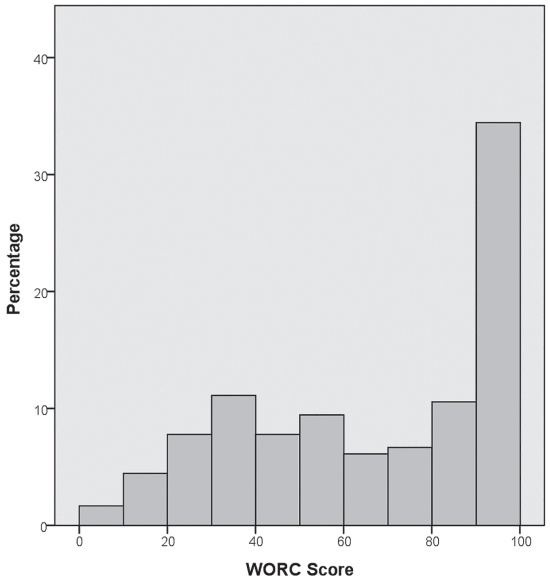
Table 3. Univariate and multivariate analyses of the associations baseline characteristics with inferior long-term clinical outcome, expressed in a WORC <80.
Radiological data were available in 204 patients for *Gärtner classification*, affected tendon and number of calcifications (analogue and digital radiographs); and in 50 for *Size* and *Location* (digital radiographs, calibrated). These data were not included in the multivariate analysis. 95-% CI: 95%-Confidence Interval

For the DASH, 75 (44.6%) of 168 patients scored over 20 points and 37 (22.0%) patients over 40 points indicating inferior long-term shoulder function.(Figure 4B) There were no variables with significant effects with univariate analyses.(Table 4) The final multivariable model for the DASH included gender, age at questionnaire, years after diagnosis, bilateral disease, dominant side involvement and treatment method. In this model, only female gender had a statistically significant (negative) effect: OR=2.0 (95%-CI: 1.0–4.0).(Table 4) Sensitivity analyses using DASH cut-off points >10 and >30 gave similar results (data not shown).

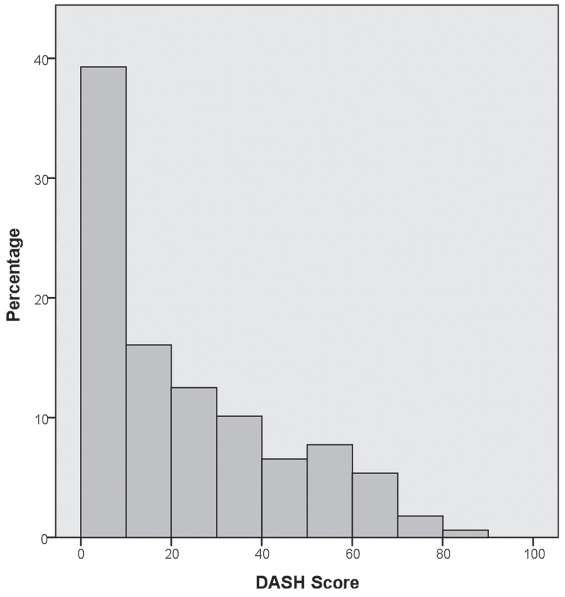
DASH > 20	**Univariate**			**Multivariate**		
	OR	95%-CI	p	OR	95%-CI	p
Female gender	1.58	0.85 - 2.97	0.15	2.03	1.02 - 4.02	**0.04**
Age diagnosis	1.01	0.98 - 1.05	0.49			
Age questionnaire	1.01	0.98 - 1.05	0.57	1.01	0.97 - 1.05	0.64
Years after diagnosis	1.00	0.96 - 1.04	0.98	1.01	0.95 - 1.07	0.79
Diabetic	2.61	0.63 - 10.8	0.19			
Duration of symptoms (per additional month)	1.01	0.99 - 1.02	0.13			
Bilateral disease	1.61	0.79 - 3.25	0.19	1.49	0.62 - 3.13	0.42
Dominant side affected	2.07	0.99 - 4.33	0.05	1.92	0.85 - 4.32	0.12
Affected tendon(s)						
Supraspinatus	0.44	0.15 - 1.30	0.14			
Infraspinatus	1.11	0.46 - 2.72	0.81			
Subscapularis	1.88	0.57 - 6.21	0.30			
Treatment						
Barbotage	0.76	0.41 - 1.44	0.40	0.64	0.31 - 1.31	0.22
Calcification location: MedLat (mm)	1.02	0.95 - 1.09	0.61			
Calcification size (mm)	0.98	0.91 - 1.06	0.64			
Gärtner calcification classification			0.53			
1	Ref.					
2	1.08	0.40 - 2.95	0.88			
3	1.71	0.59 - 5.02	0.33			
Number of calcifications (per additional deposit)	1.19	0.62 - 2.28	0.61			

Table 4. Univariate and multivariate analyses of the associations of baseline characteristics with inferior long-term clinical outcome, expressed in a DASH >20.
Radiological data were available in 204 patients for *Gärtner classification*, affected tendon and number of calcifications (analogue and digital radiographs); and in 50 for *Size* and *Location* (digital radiographs, calibrated). These data were not included in the multivariate analysis. 95-% CI: 95%-Confidence Interval

Figure 4. Histograms of the clinical scores.



A) WORC score. For the WORC, 55% had inferior long-term functional outcome, with scores below 80 percentage points;



B) DASH score. After a mean follow-up of 14 years after the diagnosis calcific tendinitis, 45% scored had scores over 20 points, indicating disability.

4. Discussion

The results of this first long-term follow-up study on functional outcome and prognostic factors in a large group of RCCT patients show that after a mean follow-up of 14 years, many patients have shoulder complaints, regardless of applied treatment method. Around 55% had WORC scores (range, 0-100) below 80 points and 42.2% even below 60 points, indicating severely impaired shoulder function. Dominant arm involvement, bilateral disease, longer duration of symptoms at presentation, larger number of calcifications and female gender all appeared to be negative prognostic factors for long-term should function.

4.1 Long-term follow-up

Previous studies on calcific tendinitis have mostly focused on small populations with a relatively short follow-up. There are some studies with follow-up >2 years,[8, 11, 50-58] or large patient groups (n>100),[13, 20, 32, 59-63] but the combination of both is scarce.[3, 7, 9] In one of the few larger RCCT cohorts with a long-term follow-up, Serafini et al. report good outcome for both barbotage and conservative treatment, in contrast to our results, with average Constant Scores over 90 points at 10 years.[7] In accordance with the current study, they found no difference in clinical outcome between barbotage and conservative treatment. A possible explanation for their superior overall clinical results is that their mean age at diagnosis was 40.2 years, compared to 49.0 years in our study. Also, RCCT was diagnosed in 323 shoulders in about 3 years, versus 420 patients in 29 years at our institution. The latter might be partially due to a high density of hospitals and the fact that a general practitioner functions as gatekeeper in our country, potentially limiting referral of patients, specifically in cases with mild symptoms. Finally, it is possible that referring physicians are more familiar with RCCT and its treatment in the geographical region of Serafini. This could lead to earlier diagnosis, at a younger age, and earlier adequate treatment. Concordantly, our univariate analyses show that longer duration of symptoms at presentation is related to inferior long-term outcome. Lastly, the Constant Score as applied by Serafini et al. is a general shoulder function score, in contrast to the WORC, which is a validated score for rotator cuff problems.

4.2 Demographics and prognostic characteristics

Previous studies on calcific tendinitis have mostly focused on small populations with a relatively short follow-up. There are some studies with follow-up >2 years,[8, 11, 50-58] or large patient groups (n>100),[13, 20, 32, 59-63] but the combination of both is scarce.[3, 7, 9] In one of the few larger RCCT cohorts with a long-term follow-up, Serafini et al. report good outcome for both barbotage and conservative treatment, in contrast to

our results, with average Constant Scores over 90 points at 10 years.[7] In accordance with the current study, they found no difference in clinical outcome between barbotage and conservative treatment. A possible explanation for their superior overall clinical results is that their mean age at diagnosis was 40.2 years, compared to 49.0 years in our study. Also, RCCT was diagnosed in 323 shoulders in about 3 years, versus 420 patients in 29 years at our institution. The latter might be partially due to a high density of hospitals and the fact that a general practitioner functions as gatekeeper in our country, potentially limiting referral of patients, specifically in cases with mild symptoms. Finally, it is possible that referring physicians are more familiar with RCCT and its treatment in the geographical region of Serafini. This could lead to earlier diagnosis, at a younger age, and earlier adequate treatment. Concordantly, our univariate analyses show that longer duration of symptoms at presentation is related to inferior long-term outcome. Lastly, the Constant Score as applied by Serafini et al. is a general shoulder function score, in contrast to the WORC, which is a validated score for rotator cuff problems.

4.3 Radiological measures and prognostic characteristics

This is one of the first studies assessing inter-observer agreement and the prognostic value of radiological characteristics of calcifications: the *Gärtner classification*,[49] calcification *Size* and the *Location* method of Ogon et al.[3] Both metric measures (*Size*, *Location*) had good ICCs and small mean inter-observer differences. For the *Gärtner classification*, there was little agreement: Kappa was 0.47, comparable to previously reported values in a smaller patient group.[45] We found no prognostic value of radiological characteristics. Confirmatory to this, others have reported that symptoms and treatment outcome do not depend on the calcific deposit classification and *Size*, but patients with objective radiological improvement (e.g. decrease in size or Gärtner classification) over time report better clinical results.[14, 43] This was not investigated in our study. However, we did find a relevant association between a higher number of calcifications at baseline and inferior long-term functional outcome.

4.4 Strengths and limitations

There are some limitations that have to be taken into account when interpreting our results. Firstly, as with all retrospective studies, a substantial part of our data depends on accurate medical record keeping in the past. Furthermore, selection bias could have played a role. Of a source population of 342 available for the baseline analyses, 194 could be included for the follow-up part of our study and only a limited number of subjects also had radiographs available. The demographics of the non-responders were however comparable to the evaluated patients, data may thus be

extrapolated to the overall group. And still, this is one of the largest studies of its kind. Secondly, it is unclear whether the inferior long-term shoulder scores are due to persisting, residual, or recurrent RCCT, or other shoulder pathology (which may be a result of RCCT). Although it would be interesting to know whether subjects with inferior outcome actually still have RCCT, the fact that many patients (formerly) diagnosed with RCCT still have serious symptoms on the long-term is very relevant information on its self; the clinical scores of many subjects in this paper are inferior compared to the general population, even years after the diagnosis of RCCT was made. This is one of the first studies showing this phenomenon. Further research is needed to investigate underlying conditions in the long-term course of RCCT. Thirdly, it is possible that some patients might have had (secondary) treatments in other institutions. However, the local institution was one of few regional centers performing barbotage and other RCCT treatments over the studied period of time And despite potential secondary treatments, we still found persisting symptoms in many subjects. Lastly, there could have been confounding by indication. Patients who had barbotage are likely to have had other or more serious symptoms than the conservatively treated patients. Taking long-term outcome into account, OR's of treatment method around 1.0 for WORC and DASH, meaning that if patients with worse symptoms in the past had a barbotage, they had no inferior long-term outcome compared to the more conservatively treated patients.

4.4 Conclusions

In this long-term follow-up study, we found that over 55% of RCCT patients have symptoms and impaired shoulder function at a mean of 14 years after the diagnosis. These observations are in contrast to the general opinion that RCCT is a self-limiting disease. Dominant arm involvement, bilateral disease, a larger number of calcifications, female gender and longer duration of symptoms were associated with inferior functional outcome. We found no associations between treatment modality and baseline radiological characteristics with long-term outcome. Inter-observer agreement of the radiological Gärtner classification was only moderate.
Applying these findings in clinical decision making might be helpful in preventing long-term symptomatic course; it is plausible that a wait-and-see strategy or conservative treatments are not necessarily the most effective methods in patients with persisting symptoms, no signs of resorption over time and one or more of the reported negative prognostic factors. We suggest taking into account these variables in future (prospective) studies, in order to evaluate whether more rigorous follow-up and more invasive forms of treatment lead to better results in selected patients.

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Rotator Cuff Calcific Tendinitis: Ultrasound-guided needling and lavage vs. subacromial corticosteroids - A Randomized Controlled Trial -

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Abstract

Background: Calcific tendinitis of the rotator cuff (RCCT) is frequently diagnosed in patients with shoulder pain, but there is no consensus on its treatment.
Purpose: Compare two regularly applied RCCT treatments: ultrasound(US)-guided needling and lavage (barbotage) combined with a US-guided corticosteroids injection in the subacromial bursa (SAI) (Group I), versus an isolated SAI (Group II) in patients diagnosed with RCCT.
Study Design: Double-blinded randomized controlled clinical trial
Methods: Patients were randomly assigned to Groups I and II. Shoulder function was assessed before treatment and at regular follow-up moments (6 weeks and 3, 6 and 12 months) using the Constant Score (CS, primary outcome), the Western Ontario Rotator Cuff index (WORC) and the Disabilities of the Arm, Shoulder and Hand score (DASH). Additionally, calcifications' location, size and Gärtner classification were assessed on radiographs. Results were analyzed using t-tests, linear regression and a mixed model for repeated measures.
Results: This study included 48 patients (25 (52%) females, mean age 52.0 (SD=7.3), 23 in Group I) with an average baseline CS of 68.7 (SD=11.9). No patients were lost to follow-up. 4 Patients in Group I and 11 in Group II (p=0.06) had an additional barbotage procedure or surgery during follow-up, due to persisting symptoms and no resorption.
At 1-year follow-up, the mean CS in group I was 86.0 (95%-CI: 80.3-91.6) versus 73.9 (95% CI: 67.7-80.1) in group II (p=0.005). Mean calcification size decreased with 11.6mm (SD=6.4) in Group I and with 5.1mm (SD=5.7) in Group II (p=0.001). There was total resorption in 13 patients in Group I and in 6 patients in Group II (p=0.07). With regression analyses, correcting for baseline CS and Gärtner type, average treatment effect was 20.5 points (p=0.05) in favor of barbotage. Follow-up scores were significantly influenced by baseline scores. Results for DASH and WORC were similar.
Conclusions: On average, there was improvement at 1 year follow-up in both treatment groups, but clinical and radiographic results were significantly better in the barbotage group.
Trial registration: NTR2282.
Level of evidence: Level I, Randomized Controlled Clinical Trial

Keywords: Rotator Cuff; Calcific Tendinitis; Treatment; Randomized Controlled Trial; Needling; Barbotage

1. Introduction

Rotator cuff calcific tendinitis (RCCT) is a frequently diagnosed condition, with a reported prevalence of 6.8-54% in patients with shoulder pain, generally affecting people between the ages of 30 and 50.[1-4] Although it is allegedly a self-limiting disease with low-grade pain, symptoms can be severe and long-lasting.[3-5] There is no consensus on the preferred treatment for these cases. The current study is the first double-blinded randomized controlled trial comparing ultrasound(US)-guided needling and lavage (barbotage) in combination with a US-guided injection with corticosteroids and bupivacaine in the subacromial bursa (SAI), versus an isolated US-guided SAI.

In RCCT there are calcific deposits in one or more rotator cuff tendons. Its etiology is unclear, but three stages have been described: 1) a formative stage (pre-calcific); 2) a resting phase (calcific) and 3) a final, resorptive phase (post-calcific).[4] Symptoms generally worsen in the last phase, in the form of pain in the Deltoid region, with typical worsening of pain at night or after activities and variable functional impairment.[1, 2, 6-9] Because of the self-limiting character of RCCT, treatment is preferably conservative, including physical therapy and non-steroidal anti-inflammatory drugs (NSAIDs).[1, 4, 9-11] In patients with severe or persisting symptoms, more invasive therapy is indicated. Numerous treatments have been reported: subacromial corticosteroids injections, ultrasound-shock therapy (lithotripsy or Extracorporeal Shock Wave Therapy (ESWT)), needling and lavage (barbotage), acetic acid iontophoresis and surgical techniques.[6, 8, 12-20] However, as there is a lack of high level evidence studies comparing these modalities, preferred treatment for RCCT remains a subject of debate.

SAI and barbotage are amongst the most frequently applied treatments of RCCT.[6-8, 21-24] SAIs are relatively easy to perform, have a low complication risk, low costs and are easily available. Barbotage treatment is more invasive, needs more skills and equipment, is time-consuming and can be painful during and post-intervention, but is reported to give better results than SAI in retrospective studies.[6, 9] However, there are no trials, to our knowledge, that have compared both treatments directly. Our primary objective was to compare clinical and radiographic outcomes of treatment with I) US-guided barbotage combined with an US-guided SAI versus II) an isolated US-guided SAI, in patients diagnosed with symptomatic RCCT non-responsive to conservative treatment. We hypothesized that barbotage leads to superior clinical and radiographic outcome 1 year after intervention.

2. Methods and Materials

The current study was a multi-center double-blinded randomized controlled trial with parallel groups and equal (1:1) simple randomization, conducted at Leiden University Medical Center (LUMC), Leiden, the Netherlands and in cooperation with Rijnland hospital, Leiderdorp, the Netherlands. Consecutive patients were included between March 2010 and December 2011. All stages of the study were approved by both Institutional Medical Ethics Review Boards and all participating patients signed informed consent.

2.1 Study Population

The source population consisted of patients referred to the orthopaedics department of either one of the two participating hospitals for treatment of non-traumatic shoulder complaints (>3 months). Inclusion criteria were: Deltoid region pain; worsening of symptoms with activities above shoulder level; positive Hawkins, empty can and Yocum tests; and calcifications >3mm on standard anterioposterior (AP) radiographs. All patients qualified for more intensive treatment on account of no clinical and radiographic improvements after a minimum of 3 months of conservative treatment. Exclusion criteria were: age <18 or >65 years; radiographic or clinical signs of resorption (defined as a change in shape and density of the calcification and/or the presence of calcific deposit in the bursa, in combination with a recent period of increased pain); co-morbidities of the concerning shoulder with clinical, radiographic and ultrasound evaluation; limited passive external rotation in 90 degrees of abduction, suggestive of frozen shoulder syndrome; >1 SAI in the 3 months prior to inclusion; and history of fracture, surgery or barbotage of the concerning shoulder. Eligible patients were referred to the coordinating investigator (PBdW) for further evaluation and inclusion.

2.2 Blinding and Intervention

Baseline demographics and clinical parameters were obtained by the coordinating investigator at the orthopaedics outpatient clinic of LUMC, one hour before the planned study intervention. Standard shoulder radiographs were obtained (AP external rotation, AP internal rotation and axial view). Each consecutive patient fulfilling the clinical and radiographic eligibility criteria received a sealed personal randomization envelope. The randomization code, obtained from the randomizer function in Excel 2003 software (Microsoft, Redmond, Washington, USA), was generated and stored by an independent local data manager.

Next, US-guided examination of the shoulder was performed to check for co-morbidities and to localize the calcific deposits. After these investigations, when eligibility criteria

were still fulfilled, the patient specific randomization code was revealed to assign the patient for either US-guided barbotage in combination with SAI (Group I), or only a US-guided SAI (Group II). All patients and the coordinating investigator, who was absent during the entire intervention, were blinded for treatment.

In each patient, one of two experienced musculoskeletal radiologists (AN, MR) performed the entire US-guided procedure. After sterile preparation, patients received a local anaesthetic injection in the skin (lidocaine 1%) and a SAI, using a 21-gauge needle. For the SAI, the needle was positioned in the subacromial bursa with US-guidance, 1-2 cm caudolateral of the acromion. Next, 5cc Bupivacaine (5 mg/mL, Actavis group, Hafnarfjordur, Iceland) and 1 mL of Depo-Medrol (40 mg/mL, Pfizer, New York, New York, USA) were injected. In Group I, in addition to the SAI, US guided needling was performed using a 55mm 18-gauge needle. The needle was introduced into the calcific deposit. Using a syringe with saline solution (room temperature) the calcification was flushed. After lavage, repeated perforation of the deposit was performed. Group II received only the SAI.

An identical post-intervention pain suppression protocol was applied in both groups: Celecoxib 100mg two times a day for three days, with supplementary Paracetamol (1000mg, four times a day). Celecoxib was replaced with Tramadol 50mg three times a day, in patients with contraindications for NSAIDs. Patients were instructed to cool the shoulder with an icepack when experiencing pain in the days after the intervention. In case of persisting symptoms, patients were treated with additional oral pain medication or physical therapy. In case of persisting symptoms and no radiographic signs of resorption >6 months after the index procedure, patients were scheduled for barbotage (i.e. a second barbotage in Group I patients) or surgery, depending on the preference and experience of the referring orthopaedic surgeon. The patients and the coordinating investigator remained blinded for the study intervention.

2.3 Follow-up

All patients had regular follow-up visits with the coordinating investigator: prior to the intervention and at 6 weeks, 3 months, 6 months and 1 year after intervention. Standard radiographs of the shoulder were obtained immediately before treatment and at the 1-year follow-up moment. At each visit, the Constant Shoulder Score (CS),[25] Disability of the Arm, Shoulder and Hand (DASH)[26] and the Western Ontario Rotator Cuff score (WORC)[27] were acquired for clinical assessment. 0-10 cm Visual Analogue Scale (VAS)[28] scores for pain in rest and during arm motion were administered directly after the intervention.

For evaluation of the calcific deposits on the radiographs, the Gärtner classification was used.[29] Deposits with a sharp border and a dense structure are type I calcifications;

type II calcifications either have a sharp border and an inhomogeneous structure or a cloudy border and a homogenous structure; type III calcifications are cloudy outlined and transparent in structure. The sizes of all calcification deposits were measured (mm) and the number of deposits and affected tendons were determined. In case of multiple calcifications, characteristics of the largest calcification were used in statistical analyses.

2.4 Sample Size calculation

As primary outcome measure, the CS was applied for sample size calculation. We defined a difference of 10.0 points in CS one year after treatment as clinically relevant. Using a standard deviation of 9.0 based on previous studies, the standardized difference was 1.1. Combined with a desired power of 0.9 and a level of significance of 0.05, this leads to a sample size of 40 using Altman's nomogram. Accounting for a potential drop-out rate of 20%, we included 48 patients.

2.5 Statistical analysis

Demographics and study data were entered in a local database. Continuous data were presented using means and standard deviations (SD), or medians and ranges where appropriate.

VAS pain scores directly after intervention in both groups were compared with the unpaired Students' t-test. We compared CS at 1 year follow-up between groups with unpaired Students' t-tests for total scores and difference-to-baseline scores. Differences-to-baseline scores were also assessed stratified for baseline Gärtner type. Additionally, linear regression analysis was applied with CS at 1 year as dependent variable, taking into account treatment group, baseline CS and baseline Gärtner type. Similar analyses were used for the WORC and DASH.

Resorption rates (proportions of subjects with either a decrease in Gärtner type, calcification size, or total resorption) and proportions of patients in both groups undergoing a barbotage procedure or surgery during follow-up due to persisting symptoms were compared using Fisher's exact tests.

To investigate how post-intervention course (repeated measures) was influenced by treatment, baseline Gärtner type and baseline clinical scores, mixed models were constructed with a random effect for each subject. The WORC, CS and DASH were each applied as dependent variable and follow-up moment, baseline clinical score, baseline Gärtner classification and the interaction-terms between follow-up moment and treatment method as well as baseline Gärtner type and treatment method were applied as independent variables.

All follow-up analyses were performed according to the intention-to-treat principle. As a sensitivity analysis, the follow-up data were also assessed using a per protocol analysis.

PASW SPSS 20.0 software (IBM, Armonk, New York, United States of America) was used for statistical analyses, and p-values of less than 0.05 were interpreted as statistically significant.

3. Results

3.1 Baseline characteristics

During the inclusion period, a total of 88 patients were potentially suitable for study participation. Of these, 40 did not meet all eligibility criteria.(Figure 1) The final study group of 48 patients comprised 25 (52%) females. Mean age was 52.0 years (SD=7.3). Baseline characteristics appeared similar for Group I (n=23) and Group II (n=25), except for slightly lower baseline clinical scores and Gärtner types in Group II.(Table 1)

3.2 Baseline radiographs and ultrasound guided procedure

Thirty (62.5%) patients had a single calcific deposit and in 18 (37.5%) patients there were 2 or more calcifications. Baseline radiographs demonstrated that mean calcification size was 14.2mm. In 20 (41.7%) patients, the largest calcification was a Gärtner type I.(Table 2) There were no statistically significant correlations between baseline Gärtner type or calcification size with either one of the baseline clinical scores.(Appendix Table S1)

Pre-intervention ultrasound evaluation demonstrated signs of a partial thickness cuff tear in 3 patients: Supraspinatus in 1, Infraspinatus tear in 1 and a combined Supraspinatus and Infraspinatus tear in 1 patient. There were no full-thickness RC tears. With regard to barbotage treatment in Group I, there was perforation in all 23 patients, aspiration in 11 (47.8%) and fragmentation in 14 (60.9%). 4 (17.4%) Patients had no aspiration or fragmentation.

Directly after intervention, VAS pain scores were 22.1 (SD=20.8) in rest and 23.6 (SD=22.0) for motion in Group I and 19.6 (SD=24.2) and 25.0 (SD=23.9) in Group II. Resulting mean differences were not significant: 2.5 (95%-CI: -11.3-16.3) for rest and -1.4 (95%-CI:-15.3-12.6) for motion.

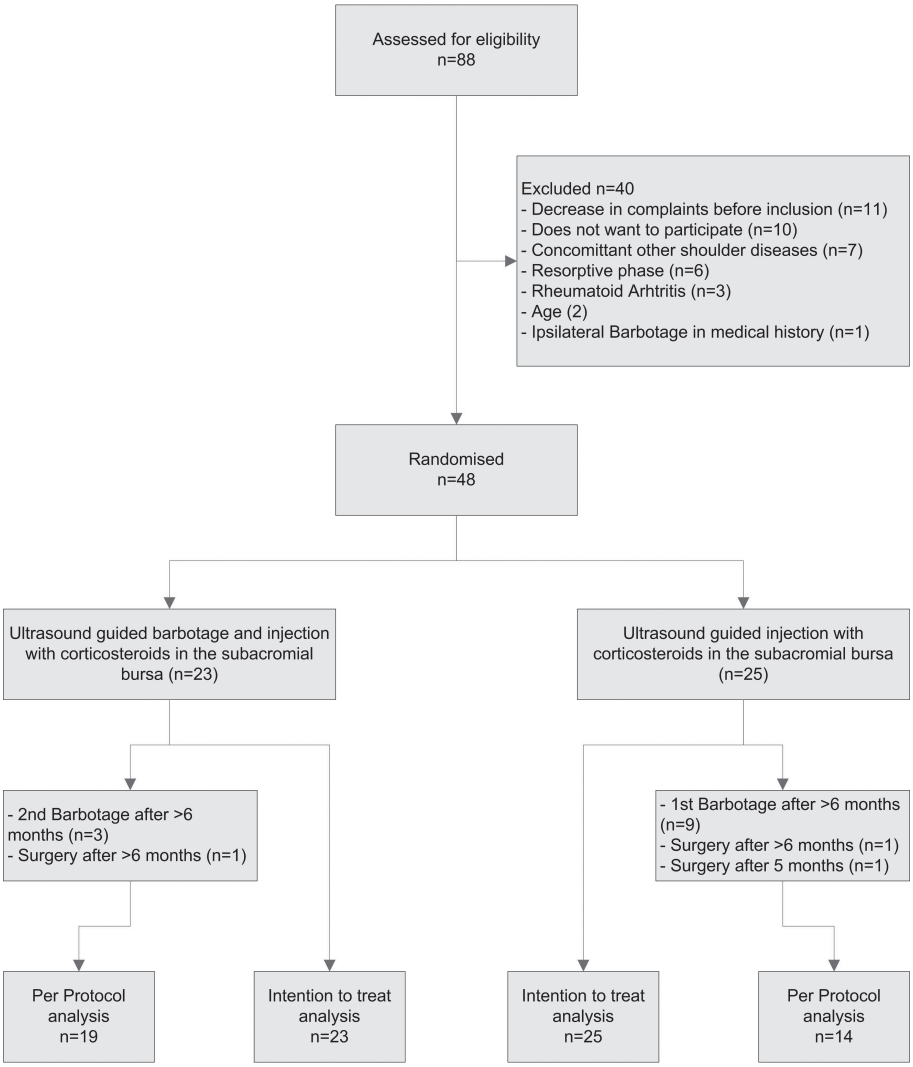


Figure 1. Study flowchart.

Baseline Characteristics	All patients	Group I: Barbotage + SAI	Group II: SAI
	n=48	n=23	n=25
Age (yrs)	52.0 (7.3)	53.7 (7.3)	50.4 (7.2)
Gender (Male/Female)	23/25	11/12	12/13
BMI	25.7 (3.3)	27.0 (3.2)	24.7 (3.0)
Affected side (right/left)	35/13	16/7	19/6
Dominant Side affected (yes/no)	31/17	15/8	16/9
Baseline clinical scores			
WORC	45.3 (19.7)	49.6 (20.3)	41.6 (18.7)
DASH	36.4 (17.3)	32.6 (18.5)	40.1 (15.7)
Constant Score	68.7 (11.9)	71.6 (12.3)	66.0 (11.2)
VAS (at rest)	40.0 (24.3)	33.4 (23.2)	46.0 (24.2)
VAS (motion)	49.2 (21.5)	42.5 (23.6)	55.3 (17.7)

Table 1. Demographics and baseline characteristics.
BMI: Body Mass Index, WORC: Western Ontario Rotator Cuff index, DASH: Disabilities of Arm Hand and Shoulder score, VAS: visual analogue score for pain (100=severe pain) at rest and during motion. SAI: subacromial bursa injection.
(Standard Deviation)

	All patients	Group I: Barbotage + SAI	Group II: SAI
	n=48	n=23	n=25
No. of calcifications			
1	30 [62.5]	18 [78.3]	12 [48.0]
2	15 [31.3]	5 [21.7]	10 [40.0]
>2	3 [6.3]	0	3 [12.0]
>1 tendons involved	7 [14.6]	3 [13.0]	4 [16.0]
Affected tendon(s)			
Supraspinatus	36 [75.0]	17 [73.9]	19 [76.0]
Infraspinatus	16 [33.3]	7 [30.4]	9 [36.0]
Subscapularis	4 [8.3]	1 [4.3]	3 [12.0]
Teres Minor	0	0	0
Gärtner Calcification Classification			
Type 1	20 [41.7]	11 [47.8]	9 [36.0]
Type 2	22 [45.8]	9 [39.1]	13 [52.0]
Type 3	6 [12.5]	3 [13.0]	3 [12.0]
Calcification Size (mm)	14.2 (5.5)	14.6 (4.7)	19.9 (6.1)

Table 2. Baseline findings with radiographic evaluation.
For calcification classification and calcifications size, numbers are based on the observations of the largest calcific deposit in each patient.
SAI: subacromial bursa injection.
(Standard Deviation), [%]

3.3 Complications and additional treatment

Overall, there were no serious adverse events or complications. Two patients developed frozen shoulder syndrome after barbotage, but symptoms declined during the study follow-up period.

No patients were lost to follow-up. Two patients were unable to attend the last follow-up visit (CS and radiographs), but filled out the 1-year WORC and DASH. Additionally, 15 patients underwent either a barbotage procedure (2nd, in case of Group I patients) or shoulder surgery during follow-up, due to no clinical and radiographic improvement: 4 (3 barbotage, 1 surgery) in Group I and 11 (9 barbotage, 2 surgery) in Group II (p=0.06).(Figure 1) This was within 6 months in 1 patient. For the Group I (barbotage) patients, all 4 had Gärtner type I calcifications and there was successful aspiration and/or fragmentation in 3 (75%) during the first barbotage procedure. For the 11 Group II patients, there were 4 type I calcifications, 6 type II and 1 type III.

3.4 Follow-up radiographs and clinical characteristics

After one year, at final follow-up, there was resorption (partial or total) in 22 (95.7%) of the patients in Group I (barbotage) and 17 patients (73.9%) in Group II (p=0.10). There was total elimination of the calcifications in 13 (56.5%) patients in Group I and in 6 (26.1%) patients in Group II (p=0.07). Mean calcification size decreased by 11.6mm (SD=6.4) in Group I and by 5.1mm (SD=5.7) in Group II (p=0.001).

There was a statistically significant improvement in CS of 14.3 points in Group I (95%-CI: 8.7-20.0) and 7.2 points in Group II (95%-CI: 1.0-13.4) compared to the pretreatment scores. There were also significant improvements for the WORC and DASH in both groups, without statistically significant differences between both groups.(Table 3) Stratified for baseline Gärtner type, there was more clinical improvement (CS) in patients with a higher Gärtner type in Group I, versus a lower clinical improvement with higher Gärtner type in Group II. Clinical improvement was similar for type I calcifications, but statistically significant differences between groups were found for type III calcifications in particular, with superior results for Group I.(Figure 2) Results for WORC and DASH were similar, albeit to a lesser extent (data not shown).

In regression analysis accounting for baseline clinical score, baseline Gärtner type and the interaction between treatment method and baseline Gärtner type, 1 year's follow-up CS was significantly influenced by baseline CS with an effect size of 0.45 (95%-CI: 0.11-0.79), meaning that 10 points higher on baseline CS, leads to an average additional 4.5 points at 1 year follow-up. Average treatment effect was 20.5 points (95%-CI: -0.09-41.1) in favor of barbotage. For DASH and WORC, baseline scores had a significant effect on the final follow-up scores, as did applied treatment for the

WORC.(Table 4) There were no significant effects for baseline Gärtner classification and its interaction term with treatment.

The course of clinical scores (repeated measures) are displayed in Figure 3. For all clinical scores and both groups, there was average improvement at 6 weeks, followed by recurrent symptoms at 3 months. After 3 months, all scores showed an improvement in Group I, vs. a further decline in scores in Group II. After 6 months, there was improvement in both groups. In mixed model analyses, the mean overall effect of barbotage on final CS was 17.9 points (95%-CI: 2.0-33.7). Considering the pretreatment condition of the patients, the baseline CS added 0.71 points (95%-CI: 0.46-0.95) improvement for each pretreatment point. For the follow-up moments of 6 weeks and 3 months, there was a significant interaction effect with treatment: 10.8 (95%-CI: 2.0-19.6) and 15.0 points (95%-CI: 6.1-23.8) in favor of barbotage. There were no significant effects of Gärtner classification and its interaction term with treatment method in this model. Similar results were found for the WORC, with an overall treatment effect of 33.1 points (95%-CI: 8.1-58.0) in favor of barbotage and a baseline WORC effect of 0.78 (95%-CI: 0.56-1.0). Similar patterns where found for the DASH, but with only a significant effect for baseline scores: 0.94 (95%-CI: 0.7-1.2). Estimated clinical outcome (CS) for an average RCCT patient group at 6 weeks, 3 months, 6 months and 1 year for both groups based on the mixed model are displayed in the Appendix.(Table S2)

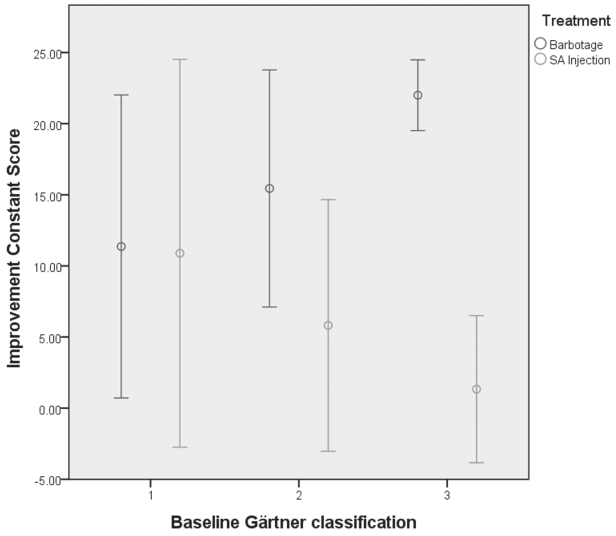
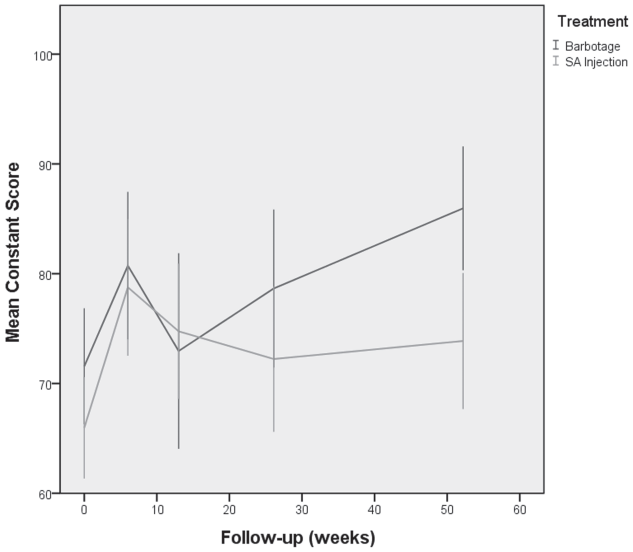
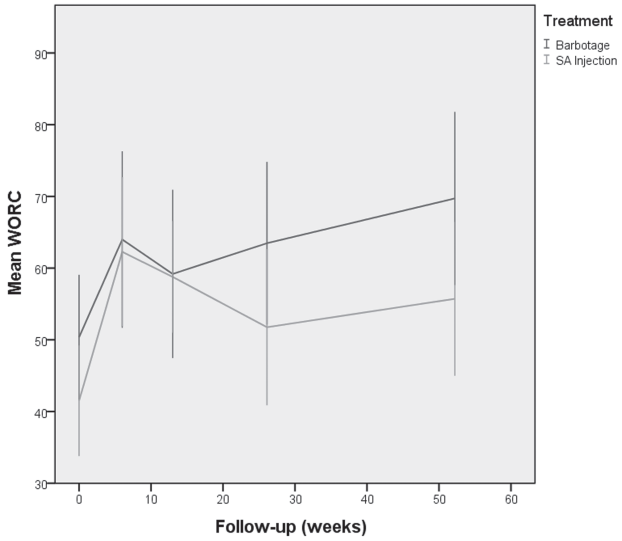


Figure 2. Improvement in Constant Score at 1 year follow-up in both treatment groups, stratified for Baseline Gärtner classification.
Patients with higher Gärtner classifications seem to have more benefit by barbotage.

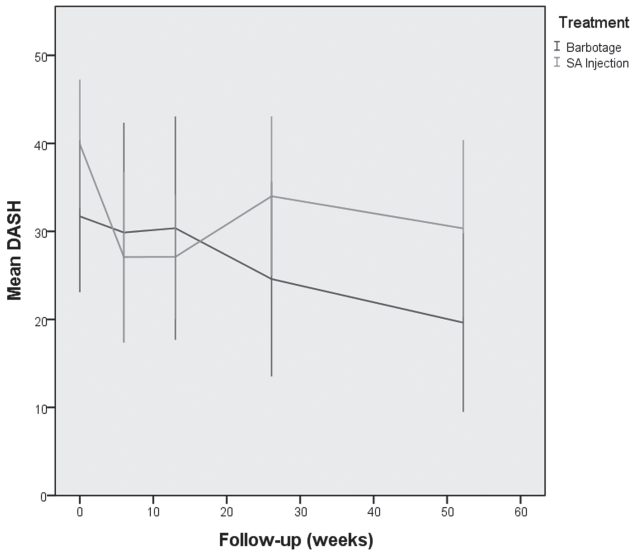
Figure 3. Clinical course after treatment of calcific tendinitis with either barbotage (Group I) or an ultrasound-guided injection with corticosteroids in the subacromial bursa (Group II), expressed in means and 95% confidence intervals, of the:



A) Constant shoulder score



B) Western Ontario Rotator Cuff index (WORC)



C) Disabilities of the Arm, Shoulder and Hand (DASH) questionnaire

3.5 Per protocol analysis

Only analyzing patients who didn't undergo barbotage or surgery in follow-up led to similar results as the intention to treat analyses. For the t-tests comparing total scores at final follow-up, WORC and CS were significantly higher in the barbotage group. There were no significant differences in the improvement scores between both treatment groups.(Appendix Table S3) Mean decrease in calcification size was significantly larger in the barbotage group. In the linear regression analyses, effects of baseline scores were significant for WORC, DASH and CS.(Appendix Table S4) There were no significant effects for treatment. In the mixed model analyses, barbotage had a positive effect on outcome for all scores and with statistical significance for the WORC: 36.1 (95%-CI: 10.1 – 62.1). Again, effects of all baseline scores on final outcome were significant: 0.78 (95%-CI: 0.56 – 1.01) per baseline point for the WORC, 0.72 (95%-CI: 0.47 – 0.96) for the CS and 0.95 (95%-CI: 0.70 – 1.20) for the DASH.

Clinical Scores at 1 year follow-up	Group I: Barbotage + SAI		Group II: SAI		Difference		
	Mean	95%-CI	Mean	95%-CI	Mean	95%-CI	p-value
Constant Score							
Total score	86.0	80.3 - 91.6	73.9	67.7 - 80.1	12.1	3.9 - 20.2	**0.005**
Improvement	14.3	8.7 - 20.0	7.2	1.0 - 13.4	7.1	-1.0 - 15.3	0.08
WORC							
Total score	69.7	57.6 - 81.8	55.7	45.0 - 66.5	14.0	-1.7 - 29.7	0.08
Improvement	20.5	9.6 - 31.3	15.8	6.2 - 25.4	4.7	-9.3 - 18.7	0.51
DASH							
Total score	19.6	9.5 - 29.8	30.3	20.3 - 40.4	-10.7	-24.6 - 3.2	0.12
Improvement	-10.4	-19.3 - -3.2	-11.3	-19.3 - -3.2	0.9	-10.5 - 12.3	0.88

Table 3. Mean final clinical scores and mean improvement during follow-up.
95%-CI: 95% confidence interval; DASH: Disabilities of the Arm, Shoulder and Hand questionnaire; WORC: Western Ontario Rotator Cuff Index; SAI: subacromial bursa injection.

	All patients		
	Effect	95%-CI	p-value
Constant Score			
Baseline Score	0.45	0.11 - 0.79	**0.01**
Treatment method	20.5	-0.09 - 41.1	0.05
WORC			
Baseline Score	0.76	0.39 - 1.13	**<0.001**
Treatment method	38.2	0.93 - 75.4	**0.05**
DASH			
Baseline Score	0.93	0.54 - 1.32	**<0.001**
Treatment method	-5.6	-43.7 - 32.5	0.77

Table 4. The influence of baseline scores and treatment method on final clinical scores.
For Constant Score, WORC and DASH, there was a significant effect of baseline score on clinical score at 1 year follow-up in linear regression analysis, accounting for baseline Gärtner type and the interaction between treatment and baseline Gärtner type. For all scores, there was a positive and clinically relevant effect of barbotage treatment on the final score. This was significant for the WORC.
95%-CI: 95% confidence interval; DASH: Disabilities of the Arm, Shoulder and Hand questionnaire; SAI: subacromial bursa injection; WORC: Western Ontario Rotator Cuff Index

4. Discussion

The results of this study show that one year after intervention, both US-guided barbotage with subacromial injections with corticosteroids (SAI) and an isolated SAI lead to an improvement in clinical and radiographic status in patients with symptomatic RCCT non-responsive to conservative treatment. However, results of barbotage were significantly better in terms of more resorption and higher clinical scores in follow-up.

Although SAI, whether or not with US-guidance, is a frequently applied conservative treatment for RCCT,[30] we found no studies assessing its effectiveness specifically in RCCT patients. It is a widely available low-cost method, relatively easy to perform and with a low complication risk. Arroll et al. reported in a meta-analysis on the painful shoulder, that US-guided corticosteroid injections proved to be 3.1 times more effective compared to placebo and 1.4 times more effective than oral NSAIDs, with a duration of benefit of up to 9 months.[21] In our study, patients treated with an US-guided SAI had statistically significant and clinically relevant short-term improvement, but symptoms recurred after 6 weeks and worsened until 6 months after treatment. After 1 year of follow-up, there was clinical and/or radiographic improvement in some. This might be due to e.g. the US-guided treatment, regular follow-up visits, or natural course of RCCT.[4, 6]

Barbotage is also a relatively non-invasive and widely available treatment that is often applied when more conservative methods fail. It is generally more painful than SAI, but moderately- to well-tolerated. In our study, we found similar VAS pain scores directly after intervention in both groups. In the barbotage group, there was a significant and relevant average improvement of clinical and radiographic status at 1 year of follow-up. There are other reports with good mid- and long-term results of barbotage,[7, 8, 24, 31-35] but few compared with other treatments and, to our knowledge, there are no randomized controlled trials with barbotage. In a non-randomized study, Serafini et al. reported significantly better short-term results of barbotage (n=219) compared to a control group (n=68).[6] However, after 1 year of follow-up there were no more significant differences between both groups. The control group consisted of non-randomly selected patients who refused to undergo barbotage for unreported reasons. Many patients in this group were lost to follow-up (26% in the first 3 months) and it was not reported whether patients in the control group received any treatment during follow-up. Another point of discussion is the absence of calcification classifications in this study.[36] Type III calcifications are reported to have a higher possibility of spontaneous recovery.[2, 37] Therefore, analysis of clinical outcome in two non-randomized treatment groups without taking into account calcification types might be prone to confounding and not reliable.

Barbotage was introduced in 1937 and generally performed under radiographic guidance in the first decades.[8, 38] A more modern alternative is barbotage under US-guidance, which is radiation free, enables easier localization of calcifications, US-guided injections in the subacromial bursa, and the visualization of the rotator cuff, bursa and Biceps tendon and possible co-morbidities in these structures.[39] Specifically in older patients, RCCT and rotator cuff tears can coexist and both need different treatment approaches.[40] There is no consensus on the size and number of needles needed for optimal outcome. Some authors prefer small needles and a limited number of punctures to prevent excessive tendon damage,[6, 11, 19] whereas others report multiple punctures[19, 24] or larger needles are needed[8] to stimulate continuing resorption after treatment. The alleged benefit of using two different needles for irrigation and aspiration has not been verified and also on this subject agreement has not yet been attained.[6, 7, 19]

Our results demonstrated a similar pattern in both randomized groups until 3 months of follow-up: there was on average clinical improvement at 6 weeks, followed by recurring symptoms at 3 months. This temporary recurrence of symptoms around 3 months has been reported earlier for barbotage.[7] It is plausible, that both groups have short-term improvement followed by recurring symptoms due to a temporary effect of the administered subacromial corticosteroids. In the SAI group, there was a further worsening of symptoms after the recurrence at 3 months, followed by some improvement after 6 months. In contrast, there was continuous clinical improvement after 3 months in the barbotage group to near healthy levels at 1 year.

Our radiographic results show similar outcomes, in favor of barbotage. There was complete or partial resorption in 17 (68.0%) patients in the SAI group. In the barbotage group, there was resorption in 22 (95.7%) patients one year after treatment, which was complete resorption in 17 (56.6%); comparable to previous studies.[7, 24, 32] In confirmation to our radiographic and clinical results, it has been reported that patients with radiographic improvement report better clinical results in follow-up.[11] Furthermore, patients with a baseline Gärtner type II or type III calcification had better clinical results of barbotage in our study, whereas clinical results were similar for all types in the SAI group. This supports the findings of Farin et al., who reported that results of barbotage are better in patients with ill-defined calcifications (e.g. Gärtner type II or III) and that these type of calcifications can be resistant to more conservative treatments.[24]

With regard to the alternatives for barbotage and SAI, specifically ESWT is a technique that is frequently published on. ESWT seems a low-risk and low-cost procedure, but generally multiple procedures with special equipment are required. Good results have been reported, but mostly with short-term follow-up and comparisson with placebo.[13, 41-45] Few studies with >6 months follow-up compare EWST to other

treatments.[12, 46, 47] Cho et al. reported that radiographic success rates for ESWT range between 15 to 70%, for barbotage between 28 and 76%, and for surgery around 72%.[9] However, surgery for RCCT must be regarded as a last resort.[48] Reported clinical results are good,[49-51] but complications (5.8-9.5%), including infection and rotator cuff tears exist, and surgical treatment is accompanied by a longer hospital stay.[52] Studies comparing surgery with other treatments are scarce. Our results show that barbotage, easily available and with low complication risk, leads to good clinical and radiographic results. And as there are few randomized controlled trials comparing RCCT treatments, barbotage is now one of few RCCT treatments with proven efficacy in a high level-of-evidence study.

There are some limitations that need to be taken into account when interpreting our results. Firstly, patient blinding was difficult. Group I received a longer and somewhat more invasive treatment than patients in Group II, which can give more pain during and after the procedure. As a result, there is a chance that some patients might have been able to make a distinction between both therapies. However, measures were taken, such as applying US-guidance in both groups and the same number of syringes visible for patients, to make recognition of the treatment method less plausible. And after all, patients in both groups indicated similar amounts of pain directly after the intervention. Secondly, depending on the treating radiologist one or two needles were used for flushing with barbotage. However, no difference in resorption rates or clinical results was found. Thirdly, our follow-up period was 1 year. Previous publications and analyses of our data suggest that decrease of symptoms and resolving of calcifications can take longer.[6, 47, 53-55] Nevertheless, the majority of barbotage patients in our study already had good or excellent results at one year and we were able to find significant and clinically relevant differences with SAI over the studied period of time. Future research is needed to further investigate which patients benefit most from barbotage and in whom more conservative, or in contrast, repeated barbotage or e.g. surgery is most beneficial.

This is the first study comparing the clinical and radiographic results of barbotage (combined with subacromial bursa corticosteroids injection) and subacromial bursa corticosteroids injections for the treatment of rotator cuff calcific tendinitis in a double-blinded randomized controlled trial. We conclude that both treatments give clinical improvement in patients with rotator cuff calcific tendinitis who fail more conservative treatments. Nevertheless, the results of barbotage in combination with SAI are superior to those of SAI, specifically in case of type II or III Gärtner calcifications. We therefore recommend the use of barbotage in patients with persisting symptoms of RCCT and no signs of spontaneous resorption over time.

Appendix tables

Baseline Clinical Scores	Baseline Calcification Size		Baseline Gärtner classification	
	Pearson's r	p-value	Pearson's r	p-value
Constant Score	-0.07	0.65	0.20	0.18
DASH	-0.18	0.28	-0.04	0.80
WORC	0.09	0.54	-0.05	0.75

Table S1. Correlations between baseline clinical scores and baseline calcification characteristics (intention to treat analysis).
There were no relevant or statistically significant correlations between baseline clinical scores and calcification characteristics.
95%-CI: 95% confidence interval; DASH: Disabilities of Arm Hand and Shoulder score; WORC: Western Ontario Rotator Cuff index; SAI: subacromial bursa injection.

	Group I: Barbotage + SAI		Group II: SAI	
	Estimate	95%-CI	Estimate	95%-CI
Constant Score				
Baseline	68.9		68.9	
6 weeks	81.2	75.3 - 87.2	81.2	75.6 - 87.0
3 months	73.5	67.5 - 79.4	77.7	71.9 - 83.5
6 months	79.2	73.2 - 85.1	74.1	68.2 - 80.0
12 months	86.5	80.5 - 92.4	75.7	69.9 - 81.6
WORC				
Baseline	45.2		45.2	
6 weeks	67.9	58.6 - 77.3	66.6	57.9 - 75.4
3 months	62.0	52.9 - 71.2	61.8	52.8 - 70.7
6 months	66.5	57.4 - 75.7	55.6	46.6 - 64.5
12 months	73.4	64.2 - 82.5	61.2	52.3 - 70.1
DASH				
Baseline	35.8		35.8	
6 weeks	31.6	23.4 - 39.8	22.3	14.1 - 30.6
3 months	28.4	19.7 - 37.1	25.2	17.0 - 33.5
6 months	28.4	19.9 - 36.9	28.9	20.3 - 37.6
12 months	23.4	15.1 - 31.6	24.9	16.5 - 33.3

Table S2. Estimated of follow-up scores of an average calcific tendinitis patient group, based on mixed model analyses (intention to treat analysis).
95%-CI: 95% confidence interval; DASH: Disabilities of Arm Hand and Shoulder score; WORC: Western Ontario Rotator Cuff index SAI: subacromial bursa injection.

Clinical Scores at 1 year follow-up	Group I: Barbotage + SAI		Group II: SAI		Difference		
	Mean	95%-CI	Mean	95%-CI	Mean	95%-CI	p-value
Constant Score							
Total score	87.2	81.1 - 93.2	76.1	68.3 - 83.9	11.1	1.7 - 20.4	**0.02**
Improvement	13.9	7.4 - 20.4	11.5	3.3 - 19.6	2.4	-7.5 - 12.3	0.62
WORC							
Total score	70.7	56.9 - 84.6	48.1	31.7 - 64.4	22.7	2.0 - 43.3	**0.03**
Improvement	21.3	8.1 - 34.4	13.7	0.7 - 26.6	7.6	-10.7 - 25.8	0.40
DASH							
Total score	18.8	8.2 - 29.5	35.7	18.5 - 52.9	-16.9	-35.1 - 1.3	0.07
Improvement	-11.4	-20.5 - -2.3	-9.4	-22.2 - 3.5	-2.1	-16.4 - 12.3	0.77

Table S3. Mean final clinical scores and mean improvement during follow-up (per protocol analysis).
95%-CI: 95% confidence interval; DASH: Disabilities of the Arm, Shoulder and Hand questionnaire; WORC: Western Ontario Rotator Cuff Index; SAI: subacromial bursa injection.

	All patients		
	Effect	95%-CI	p-value
Constant Score			
Baseline Score	0.41	0.04 - 0.78	**0.03**
Treatment method	18.6	-3.0 - 40.1	0.09
WORC			
Baseline Score	0.82	0.32 - 1.31	**0.002**
Treatment method	38.2	-7.0 - 83.4	0.09
DASH			
Baseline Score	0.99	0.52 - 1.45	**<0.001**
Treatment method	-5.6	-45.4 - 34.2	0.77

Table S4. The influence of baseline scores and treatment method on final clinical scores (per protocol analysis).
For Constant Score, WORC and DASH, there was a significant effect of baseline score on clinical score at 1 year follow-up in linear regression analysis, accounting for baseline Gärtner type and the interaction between treatment and baseline Gärtner type. For all scores, there was a positive and clinically relevant effect of barbotage treatment on the final score, but without statistical significance.
95%-CI: 95% confidence interval; DASH: Disabilities of the Arm, Shoulder and Hand questionnaire; SAI: subacromial bursa injection; WORC: Western Ontario Rotator Cuff Index

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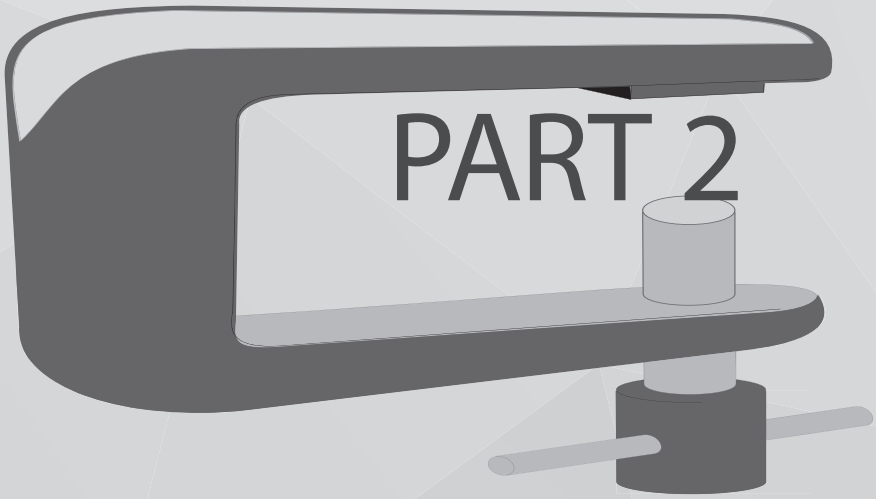
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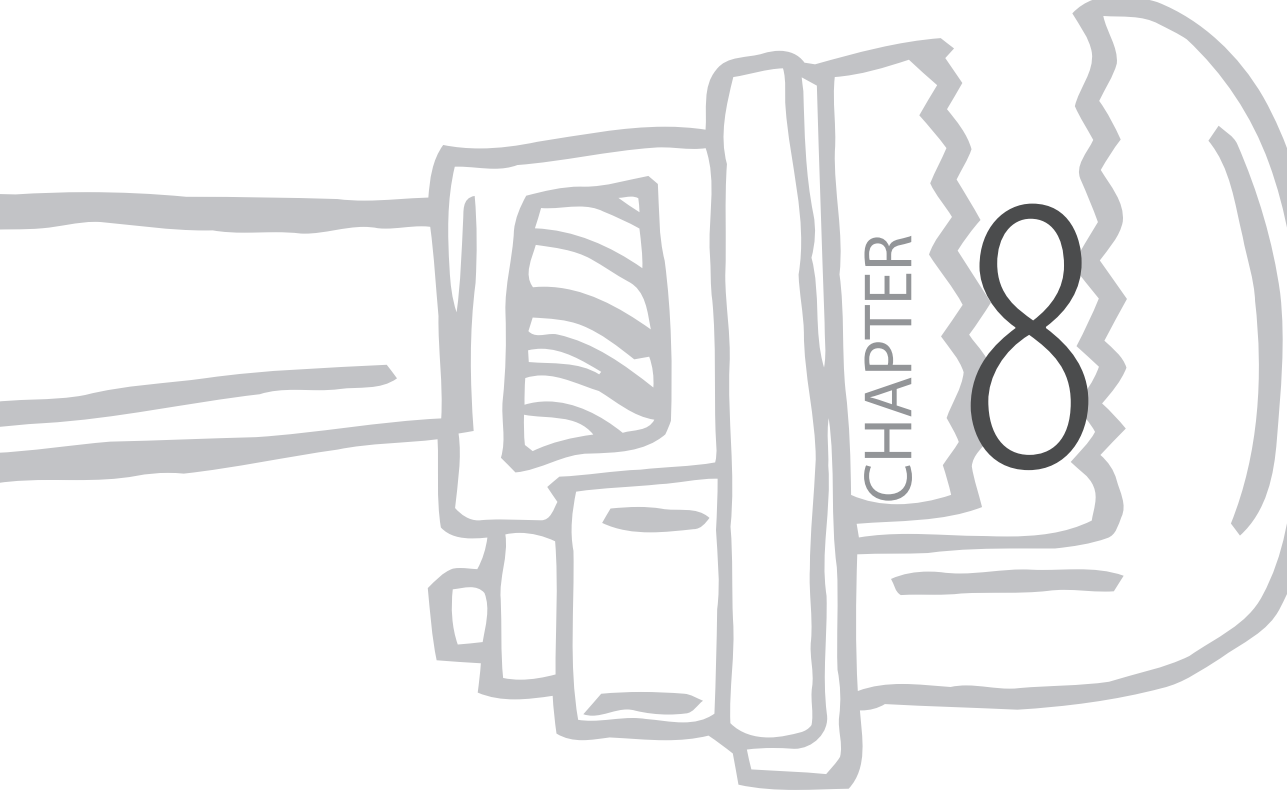
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PART 2

Novel outcome measures for patients with “impingement” symptoms





CHAPTER 8

The Western Ontario Rotator Cuff (WORC) score in rotator cuff disease patients - A comprehensive reliability and responsiveness validation study -

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Abstract

Background: The Western Ontario Rotator Cuff index (WORC) is an increasingly applied condition-specific outcome measure for rotator cuff (RC) pathologies. However, in most WORC validation studies only a limited number of psychometric properties are studied in indistinct patient groups.

Purpose: Assess psychometric properties of the WORC according to the Scientific Advisory Committee quality criteria for health questionnaires in three patient groups with distinct RC conditions.

Study design: Descriptive Epidemiology Study.

Methods: The WORC (range 0-100, 21 items, 5 domains) was administered twice (T1, T2) in 92 patients (35 RC tear, 35 calcific tendinitis, 22 impingement). Additionally, the Constant Score (CS) and the Disabilities of the Arm, Shoulder and Hand score (DASH) were recorded. Calcific tendinitis patients were re-assessed 6 weeks after treatment with needling and lavage or a subacromial injection with corticosteroids (T3).

We assessed floor/ceiling effects, internal consistency, test-retest reliability, precision, construct validity, minimal detectable change, and responsiveness in subgroups and the total group.

Results: Mean age was 55.0 years (SD=8.7) and 49/92 (53%) were female. Mean baseline WORC was 46.8 (SD=20.4), CS 63.9 (SD=15.4) and DASH 40.9 (SD=18.6). Significant differences were found for CS and DASH between RC tear patients (severe symptoms) and the other patients, but not for the WORC. There were no relevant floor and ceiling effects. Internal consistency was high: Cronbach's Alpha coefficient was 0.95. The Intraclass Correlation Coefficient (ICC) of 0.89 and Standard Error of Measurement of 6.9 indicated high reproducibility. Pearson's correlations of the WORC with CS and DASH were 0.56 and -0.65 (both $p<0.001$). At T3, total WORC improved significantly (mean change 18.8, 95%-CI: 11.3-26.2). Correlations of the WORC change scores with CS and DASH changes were 0.61 and -0.84 (both $p<0.001$). Effect Size was 0.96, with a Standardized Response Mean of 0.91, indicating good responsiveness.

Conclusions: Applied to a variety of RC patients, the WORC had high internal consistency, moderate to good construct validity, high test-retest reliability and good sensitivity to change. These findings support the use of the WORC as a condition-specific self-reported outcome measure in RC patients, but its validity in patients with severe symptoms needs further investigation.

Keywords: Rotator Cuff; WORC; Quality of Life; Reliability; Responsiveness; Validity; Shoulder; Questionnaire

1. Introduction

Shoulder problems, rotator cuff conditions in particular, are common musculoskeletal disorders with a high socioeconomic impact. The incidence of shoulder complaints in general practice is 22 per 1000 patients per year.[1] Rotator cuff conditions cover over 44-65% of these shoulder complaints,[2] with subacromial impingement syndrome, rotator cuff tendon tears and calcific tendinitis as its most frequently diagnosed forms. Young sportive individuals and active participants of society are often affected.[3, 4] Despite high incidence rates and the ensuing high number of ongoing rotator cuff research projects worldwide, there are currently few validated outcome measures focusing on rotator cuff pathologies. For accurate patient assessment however, it is advisable to combine a general health outcome measure, a general regional outcome measure and a condition specific outcome measure.[5]

In 2003, Kirkley et al. introduced the English language version of the Western Ontario Rotator Cuff index (WORC): a condition specific self-reported instrument to assess quality of life (QOL) of patients with shoulder complaints as a consequence of rotator cuff disease.[6] It comprises 21 visual analogue score (VAS) items in 5 domains: physical symptoms, sports/recreation, work, lifestyle and emotions. All items represent quality of life aspects that can particularly be influenced by rotator cuff pathology. The domains are based on the World Health Organization definition of health.

The WORC is an increasingly applied outcome measure for rotator cuff conditions,[7-21] and has been translated and validated in several languages, including Dutch, Brazilian, Norwegian, Persian, Turkish and German.[22-28] However, most studies describing the psychometric properties of either the original or translated WORC versions have some limitations: patient groups are small, not well defined, or include only patients with the same rotator cuff condition and similar symptoms. Furthermore, in most validation studies only a limited set of psychometric properties is taken into account. Hence, the goal of this study was to evaluate a comprehensive combination of the most relevant psychometric properties of the WORC, according to the proposed Scientific Advisory Committee (SAC) quality criteria for psychometric properties of health questionnaires,[29, 30] by comparing outcome scores at several follow-up moments in a heterogeneous but strictly defined group of patients with a broad spectrum of rotator cuff conditions of varying severity, including the subacromial impingement syndrome, rotator cuff tendon tears and calcific tendinitis.

2. Materials and Methods

2.1 Study design

From April 2010, all consecutive patients referred by primary health care for treatment of shoulder pain with arm abduction were assessed for participation in one of three rotator cuff disease research projects, depending on their underlying diagnosis after usual care investigations. Study 1 (Trial registry no.: NTR1545) was a cross-sectional study on muscle activation patterns in patients with a full thickness rotator cuff tear vs. healthy controls.[31] Study 2 (NTR2283) was a cross-sectional study on the etiologic mechanisms of the subacromial impingement syndrome with the use of questionnaires on shoulder function, radiographs, Magnetic Resonance Imaging (MRI) and biomechanical methods.[32] Study 3 (NTR2282) was an intervention study on the effectiveness of ultrasound-guided needle puncture, aspiration, lavage and a subacromial injection with corticosteroids in patients with rotator cuff calcific tendinitis. In all three studies the WORC, Disabilities of the Arm, Shoulder and Hand score (DASH)[33] and Constant Score (CS)[34] were used in combination with various other study specific outcome measures. Other than with regard to the current WORC assessments, the three studies were independent and there was no overlap in included patients between the studies. Data of all patients included in the three research projects until July 2011 were used in the current WORC study.

The Medical Ethics Committee approved all three study protocols and written informed consent was obtained from all participants.

2.2 Patients

Each of the three rotator cuff condition research projects had its specific inclusion and exclusion criteria, partially overlapping, with the general selection of patients based on usual care history taking, physical examination and standard shoulder radiographs (anteroposterior in both external and internal rotation).[31, 32] For all studies, patients had one or more of the following criteria present, apart from a positive Neer impingement test, a positive Hawkins test and diffuse unilateral shoulder pain for >3 months: pain during activities with arm abduction, extension and/or internal rotation (e.g. closing the door, putting on jacket, overhead activities); pain at night or incapable of lying on the shoulder; diffuse pain at palpation of the greater tuberosity; disturbed scapulohumeral rhythm; classic painful arc; positive Yocum test[35]; positive full or empty can test.

For all three studies, patients were excluded in case of insufficient language skills or no informed consent, any form of inflammatory arthritis of the shoulder, glenohumeral or acromioclavicular osteoarthritis, a history of surgical interventions of the affected shoulder, clinical signs of cervical radiculopathy, glenohumeral

instability and frozen shoulder syndrome (<90 degrees of passive abduction and external rotation).
With respect to study specific inclusion and exclusion criteria, patients had to be aged 18-75 years for study 1, 35-65 years for study 2, and 18-65 years for study 3. Moreover, patients in study groups 1 and 2 had a MRI arthrogram for usual care diagnostic purposes, that was also used for assessing eligibility criteria of the concerning studies. In study 1, all patients had a symptomatic full thickness rotator cuff tear that was non-responsive to conservative treatment. Patients with an intact rotator cuff or a partial tear could be included in study 2. Furthermore, for both studies 1 and 2, patients were excluded in case of calcific tendinitis or alternative diagnoses on MRI, including intra-articular and bony lesions (Hill Sachs, (old) fractures, tumors), labrum abnormalities, capsular or ligamentous tears/avulsions, superior labral tear from anterior to posterior (SLAP lesion), pulley lesion, Biceps tendinitis or tear, os acromiale, cartilage lesions and bony cysts. As calcific tendinitis can be demonstrated on standard radiographs, it can be distinguished from most other causes of shoulder pain without MRI. Therefore, in study 3, patients underwent radiographs and ultrasound-guided evaluation of the shoulder and were excluded in case of other pathologies, including cuff tendon tears and Biceps tendinitis.

2.3 Assessments

Patients were assessed at three time points: T1 (within 2 weeks before the scheduled outpatient visit); T2 (at the scheduled outpatient visit); and T3 (6 weeks after treatment; only for patients included in study 3).
At T1, the WORC and the Disabilities of the Arm, Shoulder and Hand score (DASH) were administered. These were sent to patients by regular mail, 7-14 days before the scheduled outpatient visit. Patients were requested, over the phone and by regular mail, to complete the questionnaires at least three days before the visit. Patients received written instructions to the questionnaires and did not receive any help with filling them out. At T2 patient characteristics (age, gender, arm dominance, affected arm and duration of symptoms) were recorded and clinical measures (including Constant Score) were obtained at the outpatient clinic of the Department of Orthopaedics by the investigating researcher. Moreover, the WORC was again administered at T2 for test-retest evaluation.
In the calcific tendinitis study, the WORC, DASH and CS were recorded once more at 6 weeks after treatment: WORC and DASH were sent by regular mail before the scheduled outpatient clinic visit. CS was recorded at the outpatient clinic by the investigating researcher. These T3 data were used for responsiveness evaluation.

2.3.1 Western Ontario Rotator Cuff index (WORC)

The WORC is a self-reported disease-specific QOL measure, comprising 21 items in five domains: physical symptoms (6 items), sports and recreation (4 items), work (4 items), lifestyle (4 items) and emotions (3 items).[6] Each item is scored on a 0-10 cm visual analog scale (the higher the rating, the higher the negative impact on quality of life), summing up to a minimum total score of 0 and a maximum total score of 2100 (worst possible). In a more clinical comprehensible format, the maximum score can be expressed as a percentage score by subtracting the total score from 2100, dividing by 2100 and multiplying by 100%, leading to total outcomes ranging from 0 (worst possible) to 100 (best possible).[6] In case of one missing value in a domain, the domain score can be calculated using the average of the other items in the domain. In case of more than two missing items in a domain, the concerning WORC questionnaire is considered incomplete, and must be excluded from analyses.

In this study, we used the Dutch translation of the WORC. Permission was granted from both the developers of the original WORC questionnaire and the translators.[6, 18, 28]

2.3.2 Constant Score (CS)

The CS is filled out by the physician and combines objective physical examination tests and subjective patient assessments.[34] 35 Points are allocated for patient-reported items on pain (15) and activities of daily living (20), 40 points are available for 4 physical examination items focused at (painless) range of motion, and 25 points are available for abduction strength evaluation. Consequently, the total maximum score is 100 points.

Arm strength was measured using a handheld dynamometer (MicroFET 2, Biometrics, Almere, the Netherlands).

2.3.3 Disabilities of the Arm, Shoulder and Hand score (DASH)

The DASH score is a self-reported questionnaire to measure disability and symptoms in patients with any or several musculoskeletal disorders of the upper arm.[33] The score contains a total of 30 items; 21 items relate to physical function, 5 to clinical symptoms, and 4 to social and work-related activities. Each item is scored on a 5 point scale, ranging from no difficulty (1 point) to unable (5 points). The total score can be calculated with a formula of the designers, and ranges from 0 (best score) to 100 (worst score).

2.4 Statistical Analysis

Normally distributed values were expressed using mean values and standard deviations (SD) and eventual skewed data were expressed using medians and

ranges. The baseline sociodemographics and clinical characteristics of patients in the three diagnostic study groups were compared with one-way ANOVA analyses or Chi-squared tests where appropriate.

Floor and ceiling effects of the total WORC score, WORC domain scores and individual WORC items at T1 were assessed by calculating the proportion of subjects scoring the minimal or maximal scores, relative to the total number of subjects. For maximal scores we applied percentage scores of 90-100 and similarly we used 0-10 percentage scores for the minimal scores. A percentage value of >15% of the subjects scoring maximal or minimal scores was considered a relevant floor or ceiling effect.

Internal consistency of the items comprising the total WORC score and the WORC domain scores was examined by computing Cronbach's alpha coefficients at T1. This coefficient assesses whether items within each domain or within the total WORC produce similar/correlating scores, contributing to and correlating with the domain and total WORC score, respectively.[21] Cronbach's alpha ranges from 0.0 for poor correlation to 1.0 for best correlation. However, high values are not necessarily desirable, as this might indicate redundancy of questionnaire items.

Reliability was determined by comparing the test-retest WORC scores (T1, T2) by means of paired t-tests, or Wilcoxon signed rank tests in case of non-parametric data. An instrument is considered reliable if it gives similar outcomes over time for each subject, provided that there are no changes in the measured items over time. Additionally, the Intraclass Correlation Coefficient (ICC) was applied. The ICC ranges from 0 to 1.00, with 0.00 to 0.39 for poor, 0.40 to 0.59 for fair, 0.60 to 0.74 for good, and 0.75 to 1.00 for excellent reliability. In this study a two-way random effects model for agreement ICC (2,1) for single measure reliability was used for each domain.[36] This model includes potential systematic differences in its analyses. Furthermore, the precision of the WORC was expressed in Standard Error of Measurement (SEM), which can be estimated by the formula, Eq 1:

$$SEM = SD \times \sqrt{1 - ICC} \qquad (1)$$

where SD is the pooled standard deviation of the test and retest measurements of all subjects. The SEM gives an absolute measure of reliability within subjects. In contrast, the ICC gives a relative measure of reliability within subjects, and depends on the population it is calculated from.[37] Furthermore, the SEM can be used to can be used to determine the minimally detectable change (MDC),[38] also reported as minimum difference (MD) to be considered real[37] or smallest real difference (SRD),[39] Eq 2:

$$MDC = SEM \times 1.96 \times \sqrt{2} \qquad (2)$$

Validity of the WORC was assessed using construct validity, as there is no gold standard for a subject's status of rotator cuff related QOL. Pearson's correlation coefficients (or Spearman's rank correlation coefficients in case of non-parametric data) were computed between total and domain scores of the WORC score at T1 and the DASH (T1) and the CS (T2). In this study, positive correlations were defined as r: 0 to 0.25 being poor, 0.25 to 0.5 as fair to moderate, 0.5 to 0.75 as moderate to good correlation, and 0.75 as good to excellent correlation. Negative correlations were defined in a similar way. The calculation of the correlation of the WORC and the CS was repeated using the T2 WORC.

Lastly, responsiveness of the WORC was assessed, by comparing WORC scores before and 6 weeks after treatment in the calcific tendinitis group. The magnitudes of the changes in total and domain WORC scores were expressed as Effect Size (ES: mean test-retest difference, divided by the SD of the test mean) and Standardized Response Mean (SRM: mean test-retest difference, divided by the SD of the mean change in score).[40, 41] Both outcomes can be interpreted as follows: 0.2 is small, 0.5 moderate, 0.8 or higher is a large effect. Additionally, the correlation coefficients between changes over time in the WORC with respect to changes in CS and DASH were calculated. Furthermore, the proportion of patients with the previously defined minimal clinically important difference of the WORC (MCID, 11.7 percentage points)[42] was assessed.

All analyses were performed for the total group, as well as for each of the three study groups separately. For all tests, p-values < 0.05 were considered statistically significant. Analyses were processed using SPSS 16.0 software (SPSS Inc., Chicago, Illinois).

3. Results

3.1 Patient characteristics

Of 94 patients included in the three projects until July 2011, 2 were excluded from the calcific tendinitis study group after filling out the first WORC because of cancelling of the treatment due to contraindications, as judged by the treating orthopaedic surgeon. In addition, one patient in the calcific tendinitis study group did not complete the third assessment 6 weeks after treatment.

Of all included participants, 7 patients left 1 item unanswered in one of the WORCs. Two patients left more than 1 item unanswered. Of these two patients, the corresponding WORC scores were not included in the analyses. 10 Patients did not complete either the first (n=4) or the second WORC. Their results were not included in the reproducibility analyses. In case of a missing T1 WORC, data of the T2 WORC

were used as baseline WORC data for comparison with CS, DASH and T3 scores.
The final study group comprised 92 patients with a mean age of 55.0 years: 35 with a rotator cuff tear (study 1), 22 with subacromial impingement (study 2), and 35 with calcific tendinitis (study 3).(Table 1) On average, rotator cuff tear patients were significantly older than patients in the other two diagnostic groups. Cuff tear patients had a significantly lower CS and higher DASH compared to impingement and calcific tendinitis patients. There were no statistically significant differences for the CS and DASH between the latter study groups.

3.2 Psychometric properties of the baseline WORC questionnaire

The mean total WORC score at (T1) was 1112.2 (SD=428.3, range: 208.5 – 1859.2). Expressed in percentage score, mean total WORC was 46.8 (SD=20.4, range: 10.0-90.0). The WORC total and domain scores were normally distributed in all groups and the total group. The mean total WORC score was significantly lower in group 3, compared to group 2. In contrast to the differences between cuff tear patients and impingement and calcific tendinitis patients in DASH and CS, there was no statistically significant difference between cuff tear patients and the other study groups for the WORC.(Table 1)
For all items, the domain scores and the total scores, less than 15% of the patients obtained the maximum or minimum score, implying there were no floor and ceiling effects of the WORC. In the physical symptoms domain, 0 patients scored between 0 and 10 and 3 patients (3.1%) had scores between 90 and 100. In the sports domain 5 patients (5.2%) obtained minimal scores and 2 (2.1%) the maximum scores. For the work domain, there were 7 patients (7.2) in the 0-10 score range and 1 (1%) in the maximal score range. In the life style domain, 4 (4.1%) patients had the lowest scores and 4 had scores in the 90-100 range. Lastly, in the emotions domain there were 6 (6.2%) and 13 (13.4) patients in the minimal and maximal score ranges, respectively. For the total WORC score, both score ranges contained 1 patient (1%).

3.3 Internal consistency

Internal consistency, as calculated with Cronbach's Alpha, was high for all WORC domain scores. Internal consistency for all items with respect to the total WORC was high as well, with a Cronbach's alpha of 0.95 for the total WORC.(Table 2) For the three diagnostic groups, Cronbach's alpha coefficients of the total WORC scores were 0.96, 0.91 and 0.95 for rotator cuff tear, impingement and calcific tendinitis patients, respectively.

3.4 Test-retest Reliability

Table 3 shows that there were no statistically significant differences between the test and retest WORC total score and domain scores, except for the Work domain in the total group, where the WORC score was higher at T2 (mean change 3.0, 95%-CI: 0.1-6.0). Pearson's correlation coefficients between the test and retest scores was 0.90 ($p<0.001$) for the total WORC score. Expressed in ICCs, test-retest reliability of the WORC domain scores ranged from 0.81 ($p<0.0001$) to 0.89 ($p<0.0001$), and was 0.89 ($p<0.0001$) for the total WORC score. Corresponding SEM was 6.9 for the total WORC score. Consequently, the 95% confidence interval of a subject's true score can be estimated by: observed score $\pm$ 1.96 x 6.9 = observed score $\pm$ 13.5. The minimum detectable change MDC (Eq. 2) was 19.1.

For the three study groups, ICCs were 0.94 ($p<0.0001$), 0.82 ($p<0.0001$) and 0.84 ($p<0.0001$) for rotator cuff tear, impingement and calcific tendinitis patients, respectively.

3.5 Validity

Table 2 shows Pearson correlation coefficients between the WORC, CS and DASH. Except for the Emotions domain in impingement patients, there were significant correlations between the domain and total WORC scores with the CS and the DASH. Pearson's correlations of the total WORC were 0.56 ($p<0.001$) for the CS and -0.65 ($p<0.0001$) for the DASH, as high percentage WORC scores mean less symptoms, where high DASH scores indicate worse symptoms. Correlations with the DASH were highest for impingement and calcific tendinitis patients: -0.77 ($p<0.001$) and -0.82 ($p<0.001$) respectively, in contrast to -0.49 ($p<0.05$) in cuff tear patients.(Table 2)

Correlation of the WORC at T2 instead of the WORC at T1 with the CS (T2) showed a correlation of similar magnitude: 0.63 ($p<0.0001$).

3.6 Responsiveness (sensitivity to change)

Table 4 shows that, on average, the mean WORC total and domain scores, the CS and DASH scores improved significantly 6 weeks after treatment for calcific tendinitis. 16 Patients (47%) had an improvement larger than the MCID: 11.7 percentage points, as reported by Kirkley et al.[42] With a value of 18.8, mean WORC improvement was higher than the MCID.

Overall, the ES and SRM of the WORC total and domain scores indicated good responsiveness. Except for the WORC sports and emotions domains, the ES and SRM of the WORC were larger than those of the DASH and in the same range (ES) or slightly lower (SRM) than those of the CS.

Table 5 shows significant and moderate to good correlations between changes over time of the WORC total and domain scores and the CS and DASH. For the CS, changes in time correlated best with changes in the WORC Lifestyle, Physical symptoms and Sports domains. The changes in DASH correlated moderate to good to changes in all WORC domains.

4. Discussion

The results of our study show that the WORC is a reliable, valid and responsive measure of health related quality of life in patients with rotator cuff lesions of various origins. WORC total and domain scores correlate moderate to good with the CS and DASH.

With respect to internal consistency, the Cronbach's Alpha of 0.95 observed in the present study is in line with the results of previous studies reporting a Cronbach's Alpha of 0.93 for the original translation and ranging from 0.92 to 0.97 for translated versions.[24-27, 43] This implies that the domains and items within the WORC contribute to and correlate with each other and the total WORC score.[21]

We found good test-retest reliability, with test-retest correlation coefficients of the total WORC score ranging from 0.82 to 0.94 in the three diagnostic groups. This is in concordance with Huber et al. who reported a test-retest correlation coefficient of 0.96, using the German WORC in 21 patients.[26] Intraclass correlations (ICC=0.89 for the total WORC) were comparable to those in literature, with 0.96 for the WORC designers in 55 patients[6] and values ranging from 0.88 to 0.98 for translated versions.[23-27] To the best of our knowledge, there is only one study reporting the Standard Error of Measurement (SEM) of the WORC: Lopes et al. reported mean SEM's ranging from 3.0 to 5.2, which is of the same order as the 6.9 we observed.[24]

Concerning construct validity, the WORC correlated moderate to good with the DASH (-0.65) and CS (0.56). The latter predominantly contains objective items and lacks emotional or lifestyle factors that are included in the WORC. Others have reported similar correlations of the WORC with the SF-36, ASES or UCLA score, often superior than correlations with the Constant Score.[6, 24-27, 44-46] Moderate to good correlation coefficients, as found for the WORC, are desirable as high or excellent correlation coefficients with other scores would mean the WORC is not of additional value to existing measures. To a further extent, this makes combining the WORC with other outcome measures, including the Constant Score, applicable and even advisable. However, the WORC appeared less discriminative than the DASH and CS in case of severe symptoms: differences were found for CS and DASH between cuff tear patients (severe symptoms on average) and the impingement and calcific tendinitis groups, but not for the WORC.

In our study, the WORC was also compared to the Constant Score and DASH to assess its responsiveness after treatment. Evaluating change score correlations, our results were moderate to good: -0.84 for the DASH and 0.61 for the CS. Holtby et al. published higher correlations for change scores: 0.77 for the CS and 0.85 for the ASES in a group of 50 surgically treated impingement and cuff tear patients.[18] Possibly, the WORC correlates better with change scores of e.g. CS, ASES and DASH in case of rigorous interventions in patients with severe symptoms, i.e. when large changes in scores over time can be expected. This can be the case with surgical treatment of rotator cuff problems, as shown by Holtby et al.[18] However, in the current study interventions were less invasive: patients were treated with an injection or ultrasound guided needling and lavage for calcific tendinitis.

A limited number of studies assessed the responsiveness of the WORC expressed in Standardized Response mean (SRM) or Effect Size (ES).[22, 45, 47, 48] Reported SRM's of the total WORC score range between 0.8 and 2.0, often comparable or superior to other scores, including the CS, DASH and Oxford Shoulder Scale, but are mostly based on subgroups of patients who are defined as "responsive to treatment". In our study, mean SRM was 0.91 for patients responsive and non-responsive 6 weeks after treatment of calcific tendinitis: better than the DASH and in the same range as the CS. Data on Effect Size of the WORC is scarce. An ES of 0.92 for the Brazilian WORC has been reported; comparable to the 0.96 in our study.[22] In both studies, ES of the WORC was superior to the DASH.

Hence, comparing with published validation studies of both original and translated WORC versions, our results are similar.[23-27, 43-48] However, our study provides some new and important information. Firstly, with regard to psychometric properties of the WORC, in most publications only internal consistency measures, test-retest assessments and/or responsiveness are reported. In our study a comprehensive combination of the most relevant measurement properties is assessed in one population, as advised by e.g. the Scientific Advisory Committee (SAC) of the Medical Outcomes Trust and Terwee et al.[29, 30] Secondly, patient groups in many publications are quite homogeneous, small, or selected with unclear eligibility criteria.[23-27, 43, 44, 47] In contrast, we studied a consecutive patient group composed of patients with one of three diagnoses and with a broad spectrum of severity of symptoms, using strict inclusion and exclusion criteria and advanced imaging technologies. Therefore, this study assessed a comprehensive combination of relevant psychometric outcome measures in a large, and heterogeneous but well-defined cohort, indicating high external validity of our results. Thirdly, we found potential discriminative problems of the WORC in patients with severe symptoms (in the lower score ranges in patients with cuff tears). To the best of our knowledge, this has not been reported earlier and has to be taken into account when using the WORC in similar patients.

Over the past two decades, there has been an increasing emphasis on the use of self-reported outcome measures in orthopaedic practice, including the WORC.[49] Conventional musculoskeletal instruments are mainly based on objective quantities that do not necessarily correlate with outcomes that are most relevant to patients, such as activities of daily life, mental health, or other QOL aspects. For broad and accurate patient assessment, it is advisable to combine objective and self-reported general health scores, general regional (e.g. shoulder) measures, and condition-specific measures.[5, 50] The results of the current study and previous publications demonstrate that the WORC, one of few available condition-specific HRQOL measures for the rotator cuff, can be used in the assessment of cuff patients.

There are some limitations of our study that need to be taken into account when interpreting our results. Firstly, the investigating researcher was not blinded for the diagnostic study groups and treatment, leading to potential bias towards improvement with regard to the Constant Score. Secondly, within the study period, there was only an intervention and WORC assessment of longitudinal responsiveness in the calcific tendinitis patients. It is a possibility however, that results for longitudinal responsiveness in the calcific tendinitis group cannot necessarily be extrapolated to other rotator cuff conditions. Thirdly, all patients in this study were referred to our medical center for treatment by general practitioners. Therefore, this patient group might not be representative of all patients with rotator cuff conditions. Fourthly, the test-retest time-interval was relatively short: near 3-7 days in many patients. Yet, earlier validation studies for the WORC reported a substantial number of included patients to be considered clinically unstable over a two week's period.[23, 24] and even a 2-3 day period for WORC test-retest assessments is not uncommon.[25, 27] Fifthly, for practical reasons the DASH (T1) and CS (T2) were not administered at the same occasion, whereas both were compared with the baseline (T1) WORC. Given the insignificant T1-T2 differences of the WORC and high T1-T2 correlation coefficients, we believe this does not influence our results. Comparing the T2 WORC to the CS (T2) gave a similar correlation coefficient. Lastly, we used the Dutch version of the WORC and therefore it cannot be guaranteed that our results are generalizable to the original or other translated versions. However, the Dutch translation we used was made by another and independent institution using international translation guidelines.[28] Furthermore, the results obtained in each of the diagnostic groups are comparable to previously (but separately) published average scores,[23] SD's,[14, 23, 51-53] Cronbach's Alpha,[23, 43] correlations with CS[43, 44, 51] and DASH,[6, 43] and ICC[6, 23] of the original WORC in patient groups with similar diagnoses. Therefore, we think this study can be considered to be the first study extensively assessing the validity of the WORC according to the CAS guidelines in a strictly defined patient group covering a broad spectrum of rotator cuff conditions. Nevertheless, repeating this

comprehensive combination of analyses for the original WORC in a similarly broad spectrum of RC condition patients is recommendable.

Concluding, our results suggest the WORC is applicable in research and clinical practice as a self-reported disease-specific Health Related Qualitative of Life outcome measure for rotator cuff patients. It is advisable to use this disease-specific measure in combination with a regional and a general health outcome measure. The WORC is potentially less discriminative between subjects with severe complaints, compared to other outcome measures. This needs to be studied further and has to be taken into account when using the WORC in assessing patients with severe symptoms.

Legend

		Rotator Cuff tear	Impingement	Calc. Tendinitis	Differences between study groups	All patients
		(n=35)	(n=22)	(n=35)	p-value	(n=92)
Age (yrs)		60.6 (8.6)	51.1 (6.5)	52.4 (7.6)	<0.001 [1,2,3]	55.0 (8.7)
Gender	*Male (n)*	19 [54%]	9 [41%]	15 [43%]	0.52	43 [47%]
	Female (n)	16 [46%]	13 [59%]	20 [57%]		49 [53%]
Duration of symptoms (months)		NA	33.1 (58.7)	42.5 (41.6)	0.56	39.6 (47.0)
Dominant side affected (n)		16 [40%]	12 [55%]	20 [57%]	0.78	32 [56%]
Constant Score		53.4 (14.8)	74.7 (10.7)	67.3 (12.3)	<0.001 [1,3]	63.9 (15.4)
DASH		50.9 (18.4)	28.7 (13.5)	38.6 (16.3)	<0.001 [1,3]	40.9 (18.6)
WORC	*Total score*	1159.3 (461.9)	906.1 (334.0)	1194.7 (414.7)	0.03 [2]	1112.2 (428.3)
	Percentage score	44.3 (22.0)	56.8 (15.8)	43.0 (19.7)	0.03 [2]	46.8 (20.4)

Table 1. Demographic and baseline characteristics of 92 included patients with a rotator cuff tear, subacromial impingement syndrome, or calcific tendinitis.
1) Statistically significant difference between study groups 1 and 2; 2) Statistically significant difference between study groups 2 and 3; 3) Statistically significant difference between study groups 1 and 3)
((SD), Standard Deviation; DASH, Disabilities of arm, Shoulder and Hand score; WORC, Western Ontario Rotator Cuff Index)

	Cronbach Alpha	Correlations of WORC with Constant Score and DASH							
		Constant Score				*DASH*			
		RC#	SIS	Calc	All patients	RC#	SIS	Calc	All patients
Total WORC (n=92)	0.95	0.59**	0.65*	0.55*	0.56**	-0.49*	-0.77**	-0.82**	-0.65**
Physical Symptoms	0.85	0.51*	0.61*	0.49*	0.50**	-0.41*	-0.58*	-0.73**	-0.56**
Sports/recreation	0.76	0.52*	0.50*	0.51*	0.50**	-0.41*	-0.76**	-0.72**	-0.57*
Work	0.84	0.47*	0.56*	0.55*	0.51**	-0.51*	-0.73**	-0.80**	-0.65**
Lifestyle	0.82	0.66**	0.60*	0.45*	0.52**	-0.51*	-0.37	-0.66**	-0.52**
Emotions	0.86	0.53*	0.20	0.43*	0.49**	-0.48*	-0.69**	-0.72**	-0.63**

Table 2. Internal & Construct Validity of the WORC in all patients (n=92).
Cronbach's Alpha for internal validity and Pearson correlation coefficients of the WORC with the DASH and CS.
DASH score: 0 is best, to 100 for worst. WORC and Constant Score: 0 is worst and 100 is best.
(DASH, Disabilities of arm, Shoulder and Hand score; RC#, rotator cuff tendon tear (n=35); SIS, subacromial impingement syndrome (n=25); Calc, calcific tendinitis (n=35))
(* p<0.05, ** p<0.001)

	Baseline mean (SD)	Retest mean (SD)	Mean difference (95% CI)	p-value	Pearson's r	ICC (95% CI)	SEM
Total WORC (n = 83)	46.3 (20.2)	47.9 (21.3)	1.6(-0.5 - 3.7)	0.13	0.90**	0.89 (0.84 - 0.93)	6.9
Physical symptoms	52.2 (21.6)	54.3 (22.5)	2.0 (-0.4 - 4.4)	0.10	0.88**	0.87 (0.81 - 0.92)	8.0
Sports/recreation	43.3 (21.6)	44.6 (21.6)	1.3 (-1.7 - 4.3)	0.41	0.81**	0.81 (0.72 - 0.87)	9.4
Work	37.3 (22.6)	40.4 (23.8)	3.0 (0.1 - 6.0)	0.04	0.83**	0.83 (0.74 - 0.88)	9.6
Lifestyle	44.6 (24.2)	44.6 (24.2)	0.4 (-2.3 - 3.1)	0.78	0.86**	0.86 (0.80 - 0.91)	9.1
Emotions	58.0 (28.3)	59.3 (28.3)	1.2(-2.4 - 4.9)	0.50	0.83**	0.83 (0.75 - 0.89)	11.7

Table 3. Reproducibility of the WORC.
Test-retest reproducibility was assessed by comparing the differences between test and retest examination within 3-14 days, expressed in percentage scores, with paired t-test and Pearson's correlations. Reliability was estimated with Intraclass Correlation Coefficients.
(SD, Standard Deviation; 95% CI, 95% confidence interval; SEM, standard error of measurement; WORC, Western Ontario Rotator Cuff Index)
(* p<0.01, ** p<0.001)

	Baseline mean (SD)	Follow-up mean 6 wks (SD)	Change (95% CI)	p-value	ES	SRM
Constant Score (n=34)	67.1 (12.4)	78.2 (16.8)	11.1 (7.8 - 14.4)	<0.001	-0.89	-1.16
DASH (n=34)	38.6 (16.5)	28.6 (22.9)	-9.9 (-15.1 - -4.8)	<0.001	0.61	0.68
Total WORC (n=34)	42.9 (19.6)	61.7 (26.7)	18.8 (11.3 - 26.2)	<0.001	-0.96	-0.91
Physical symptoms	48.3 (21.1)	67.3 (24.3)	19.0 (11.4 - 26.7)	<0.001	-0.90	-0.86
Sport	41.6 (19.6)	56.9 (27.7)	15.3 (7.2 - 23.4)	0.001	-0.78	-0.67
Work	34.9 (21.1)	56.8 (29.4)	21.9 (13.9 - 29.9)	<0.001	-1.04	-0.97
Lifestyle	39.0 (23.7)	61.9 (31.9)	22.9 (14.4 - 31.5)	<0.001	-0.97	-0.96
Emotions	54.6 (29.2)	67.1 (29.0)	12.5 (4.6 - 20.4)	0.003	-0.44	-0.56

Table 4. Baseline and 6-weeks follow-up data in 34 patients treated with ultrasound guided needling and lavage or a subacromial injection with corticosteroids in the calcific tendinitis group.
For ES and SRM: 0.2 is small, 0.5 moderate, 0.8 or higher is large effect (idem for negative values).
(SD, Standard Deviation; 95% CI, 95% confidence interval; WORC, Western Ontario Rotator Cuff Index; DASH, Disabilities of arm, Shoulder and Hand score; ES, effect size; SRM, standardized response mean)

	Correlations of changes (0 to 6 weeks)			
	Constant Score		DASH	
	Pearson	*p-value*	*Pearson*	*p-value*
Total WORC (n=34)	0.61	<0.001	-0.84	<0.001
Physical Symptoms	0.55	0.001	-0.65	<0.001
Sports/recreation	0.60	<0.001	-0.77	<0.001
Work	0.52	0.002	-0.78	<0.001
Lifestyle	0.66	<0.001	-0.77	<0.001
Emotions	0.38	0.03	-0.72	<0.001

Table 5. Longitudinal responsiveness.
Correlations of changes in WORC scores with changes in the Constant Score and DASH, from baseline to 6-weeks after treatment in the calcific tendinitis group.
(WORC, Western Ontario Rotator Cuff Index; DASH, Disabilities of arm, Shoulder and Hand score)

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Arm ADductor activation with arm ABduction in rotator cuff tear patients vs. healthy controls - Design of a new measuring instrument -

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Abstract

The incidence of (a)symptomatic rotator cuff tears is high, but etiologic mechanisms are unclear and treatment outcomes vary. A practical tool providing objective outcome measures and insight in etiology and potential patient subgroups is desirable.

Symptomatic cuff tears coincide with humerus cranialisation. *Ad*ductor co-activation during active arm *ab*duction has been reported to reduce subacromial narrowing and pain in cuff patients. We present an easy-to-use method to evaluate adductor co-activation.

20 Healthy controls and 20 full-thickness cuff tear patients exerted EMG-recorded isometric arm abduction and adduction tasks. Abductor and adductor EMGs were expressed using the "*Activation Ratio* (*AR*)" ($-1 \leq AR \leq 1$), where lower values express more co-activation.

Mean control *AR*'s ranged from 0.7 to 0.9 with moderate to good test-retest reliability (ICC: 0.60-0.74). Patients showed significantly more *ad*ductor co-activation during *ab*duction, with adductor *AR*'s ranging between 0.3 (Teres Major) and 0.5 (Latissimus Dorsi).

Concluding, the introduced method discriminates symptomatic cuff tear patients from healthy controls, quantifies adductor co-activation in an interpretable measure, and provides the opportunity to study correlations between muscle activation and humerus cranialisation in a straightforward manner. It has potential as an objective outcome measure, for distinguishing symptomatic from asymptomatic cuff tears and as a tool for surgical or therapeutic decision-making.

Keywords: Rotator cuff tears; Electromyography; Activation Ratio; Diagnostic Measure; Adductor co-activation.

1. Introduction

The incidence of shoulder related complaints in general practice is 22 per 1000 registered patients per year.[1] Chronic shoulder pathology as a result of rotator cuff (RC) diseases, including subacromial impingement syndrome and RC tears, mainly affects a population in the fifth to seventh decade of life and is a main cause of work-related problems of the locomotor system, with a high socio-economic impact. The etiology of these entities is not clearly understood and, consequently, diagnostic and therapeutic strategies are subject of debate. In this study, a new measure is presented, which is potentially valuable in both research and clinical decision making.

When conservative treatments fail, standard surgical treatment for full-thickness or massive RC tears is RC tendon repair, often in combination with subacromial decompression.[2, 3] Reported results of conservative and surgical treatments vary to a great extent.[4] Moreover, symptoms are self-limiting in many RC tear patients, and 54% of persons over 60 years have asymptomatic RC tears.[5, 6] Consequently, shoulder symptoms are not necessarily the consequence of an observed RC tear. To a further extend, this implies there may be several etiological mechanisms leading to shoulder complaints, related to a diagnosed RC tear in some but not all patients, requiring discrete therapeutic approaches. A practical tool providing objective outcome measures and insight in etiology and potential patient subgroups is desirable.

In the healthy shoulder a perfect compromise is assumed between arm mobility and glenohumeral stability.[7] In both healthy subjects and cuff lesion patients, Deltoid activation leads to subacromial narrowing as a consequence of its mostly cranially directed force arm, pulling the humerus upwards.[8-10] In healthy subjects rotator cuff activity reorients abduction forces in medial direction, with the resultant force falling within the glenoid fossa, ensuring glenohumeral stability.(Figure 1) In symptomatic rotator cuff tear patients, the muscle moment balance around the glenohumeral joint is disturbed which results in a conflict between mobility and stability: 1) there is increased Deltoid activation during abduction to compensate for lost rotator cuff abductor forces,[11-13] and 2) there is decreased glenohumeral stability in cuff lesion patients as a consequence of impaired rotator cuff function.[13] It has been hypothesized that the combination of these mechanisms causes *excessive* cranialisation (proximal migration) of the humerus in patients, leading to (painful) impingement of subacromial tissues.[9, 13, 14] To restore the glenohumeral stability, arm *ad*ductors with more caudally directed moment arms are activated during active arm *ab*duction in patients in order to reduce this subacromial narrowing.(Figure 1) This 'out-of-phase' *ad*ductor activation (co-activation) has been reported in both

model simulation studies and patient experiments, in particular for the Teres Major and Latissimus Dorsi muscles with their mediocaudally directed force vectors.[12, 13, 15-17] The conflicting effect of co-activation of *ad*ductors for reducing subacromial narrowing and pain, is the decrease of net arm *ab*duction torque and an increase of glenohumeral contact force.

Adductor co-activation provides insight in etiological and patient coping mechanisms, but also has potential value as a practical measure for identifying etiological subgroups in patients with shoulder symptoms and RC pathologies, applicable in diagnostics and clinical decision making, in discriminating symptomatic rotator cuff tears, and in objectively assessing treatment effects. However, current methods for assessing arm adductor co-activation are rather laborious to perform, applicable only in experimental environments,[12, 15-19] and electromyography (EMG) measures in general are hard to interpret and compare between subjects. Therefore, the goals of this study were to 1) design a practical measuring instrument to assess co-activation of arm *ad*ductors, which expresses EMG muscle activation in an easily interpretable measure, 2) test the reliability of this method, 3) analyze for the presence of adductor co-activation in healthy subjects and in patients with full thickness RC tendon tears and test the responsivity of this measure.

2. Methods

2.1 Subjects and outcome parameters

Twenty healthy subjects (controls) and 20 patients with shoulder complaints and a full thickness rotator cuff tendon tear were included in this study during the period of February 2010 to October 2010. Inclusion criteria for healthy controls were: between 18 and 50 years old, no present shoulder complaints, and no history of medically treated shoulder complaints. For patients, diagnosis of symptomatic full-thickness RC tendon tear (at least Supraspinatus) was based on patient history, clinical examination, standard shoulder radiographs (anteroposterior (AP) in both external and internal rotation) and a Magnetic Resonance Imaging (MRI) arthrogram. All patients were selected for a surgical rotator cuff repair procedure. Subjects were excluded if they had clinical signs of glenohumeral instability (other than cranialisation of the humerus on radiographs or MRI), frozen shoulder syndrome, rheumatoid arthritis, or osteoarthritis of the glenohumeral or acromioclavicular joint, history of surgery on the affected shoulder, Biceps tendinitis, Subscapularis tendon tear, signs of cervical radiculopathy, or present physical problems influencing muscle activation and arm mobility. The local Medical Ethics Committee (METC, Leiden University Medical Center) approved all stages of the study and written informed consent was obtained from all participants.

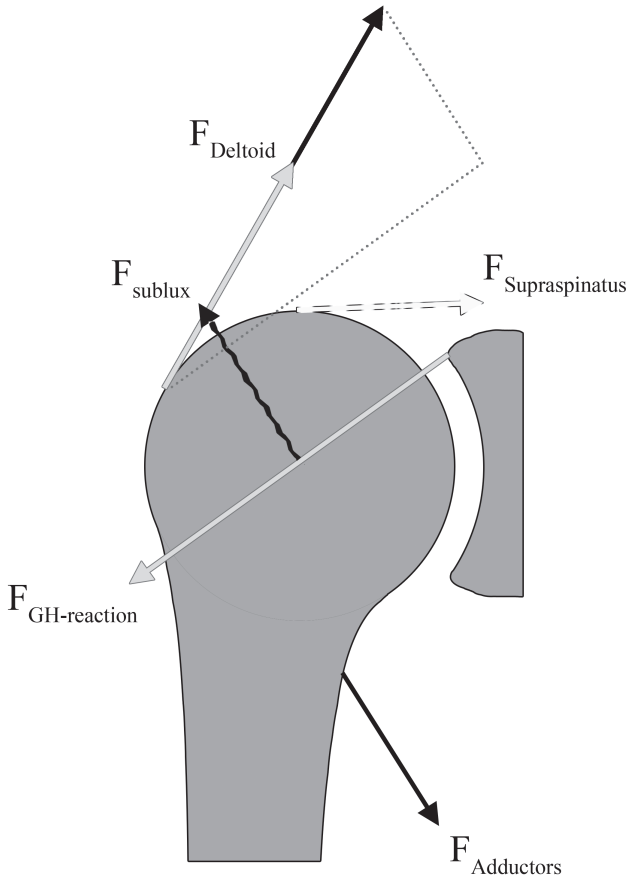


Figure 1. Schematic representation of muscle contributions and resulting glenohumeral reaction forces in full thickness RC tear patients while initiating an arm abduction moment (based on Steenbrink et al.[16]).
Arm elevation requires an abduction moment, along with a glenohumeral force equilibrium, predominantly provided by the Deltoid muscle parts and the Supraspinatus (grey line and dotted line). The resultant force can fully be compensated by the glenoid. In cuff tear patients, lost Supraspinatus torque ($F_{Supraspinatus}$, dotted line) is compensated by the Deltoid muscle parts ($F_{Deltoid}$, black line). This is accompanied with an increased upward force, which can only be partly compensated by the glenoid. Without compensation for the remaining force vector (F_{sublux}), (painful) upward humerus translation is expected. However, depressor/adductor muscles ($F_{adductors}$), e.g. Teres Major and Latissimus Dorsi, can compensate the upward directed pathological force component, at the cost of reduction of nett abduction torque.

The patients and healthy subjects performed standardized EMG-recorded isometric arm abduction and adduction moment tasks by pressing against a force sensor in a newly developed experimental set-up, as described in section 2.3. For the healthy controls, measurements were repeated for both arms, after reapplication of EMG-electrodes and with an in-between time interval of 30 minutes, to study test-retest

reliability and arm-dominance dependence with this particular experimental set-up. All patients were clinically evaluated by an independent investigator using the Constant Shoulder score,[20] and all patients evaluated their average amount of pain during daily activities on a 10 cm Visual Analogue Scale (VAS, 0 = no pain, 10 = extreme pain).

2.2 EMG: expressed in 'muscle Activation Ratio'

During standardized arm abduction and adduction moment tasks, EMG of 3 shoulder adductors (Pectoralis Major, clavicular part (PM); Teres Major (TM); Latissimus Dorsi (LD)) and the main shoulder abductor (Deltoid, medial part (DM)) were recorded with bi-polar surface EMG equipment (DelSys system Bagnoli-16, Boston, MA, USA, inter-electrode distance 10mm, bandwidth 20–450 Hz). The electrodes were placed after palpation of the muscle bellies, with the subject in similar position as during the isometric force tasks.[18] For adductor muscles, subjects were asked to adduct during palpation and v.v. for the DM. As the LD muscle belly can be hard to palpate, its electrode was placed approximately 5 cm caudally and 2 cm laterally from the Angulus Inferior Scapulae. Before placement of the electrodes, the skin was abraded, cleaned and a skin preparation gel (Skin Pure, Nihon Kohden, Tokyo, Japan) was used.

Absolute magnitudes of EMG-signals are hard to interpret and to compare between subjects. Activations of specific muscles were therefore expressed in the '*Activation Ratio*' (*AR*) as reported by Steenbrink and co-workers: a relative measure of muscle activation which enables easy interpretable intra- and inter-subjects comparisons.[12, 17] To obtain the *Activation Ratio*, subjects had to perform 2 isometric and oppositely directed force tasks of equal force magnitude, i.e. an arm abduction and adduction moment task in the present study.

Average muscle activations (A_{muscle}) during the abduction and adduction tasks were quantified using rectified and low pass filtered EMG (2 Hz recursive 3rd order butterworth) (*rEMG*). The nett active *rEMGs* were calculated by subtracting the rest *rEMG*.

In order to calculate the *Activation Ratio* for each muscle, muscle activation during each task was qualified according to muscle specific primary moment arms, i.e. either 'in-phase' (A^{IP}_{muscle}) or 'out-of-phase' (A^{OP}_{muscle}) with respect to its primary moment arm. For example, mean DM activation during an arm *ab*duction moment task was defined as 'in phase', and mean DM activation during an arm *ad*duction moment task was defined 'out-of-phase'. In this way, two average EMG levels were determined for each muscle, with respect to the isometric ad- and abduction moment tasks. Based on these data, subject specific *Activation Ratios* were calculated for each muscle (AR_{muscle}), Eq. 1:

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

Consequently, the *Activation Ratios* of most muscles in healthy subjects are positive and close to 1. In case of co-activation of specific arm *ad*ductors during *ab*duction tasks, as has been described for the LD and TM in patients with rotator cuff tears, the *Activation Ratios* of these adductor muscles are expected closer to 0 or even negative.[12, 17]

2.3 Experimental set-up

An easy-to-use experimental set-up was developed, meeting the following design criteria: 1) practical, fast, and applicable during simultaneous acquirement of standard AP shoulder radiographs to enable acromial humeral distance (AH) measurements in future applications; 2) capable of measuring shoulder muscle EMGs during isometric arm abduction and adduction moment tasks of equal force magnitude for *AR* calculation; and 3) enabling the use of standardized patient-specific task force magnitudes.

To satisfy the first design criterion, the EMG-equipped subjects were in neutral standing position with the hand in frontal plane, as required for standard AP shoulder radiographs in external rotation. The arm was attached to a 1-dimensional force transducer at the wrist.(Figure 2) (Penko Engineering, Ede, the Netherlands) Rest EMG was recorded while no additional forces and moments were exerted at the wrist. Because the arm was hanging vertically alongside the body in this particular set-up, compensation for gravitational forces was not needed, in contrast to previous studies. Subsequently, subjects performed a maximum isometric voluntary contraction (MVC) in this position against the force sensor for a shoulder abduction and adduction moment, respectively. The maximum force (*Fmax*) was defined as the minimal value of the absolute abduction and adduction MVC's for each subject.

For *Activation Ratio* calculation, subjects performed both a 2-second abduction and adduction moment task of 60% of *Fmax* (with an allowed tolerance of ± 3.75%), using real-time visual feedback with a custom made graphical interface (Matlab, MathWorks inc., Natick, USA).

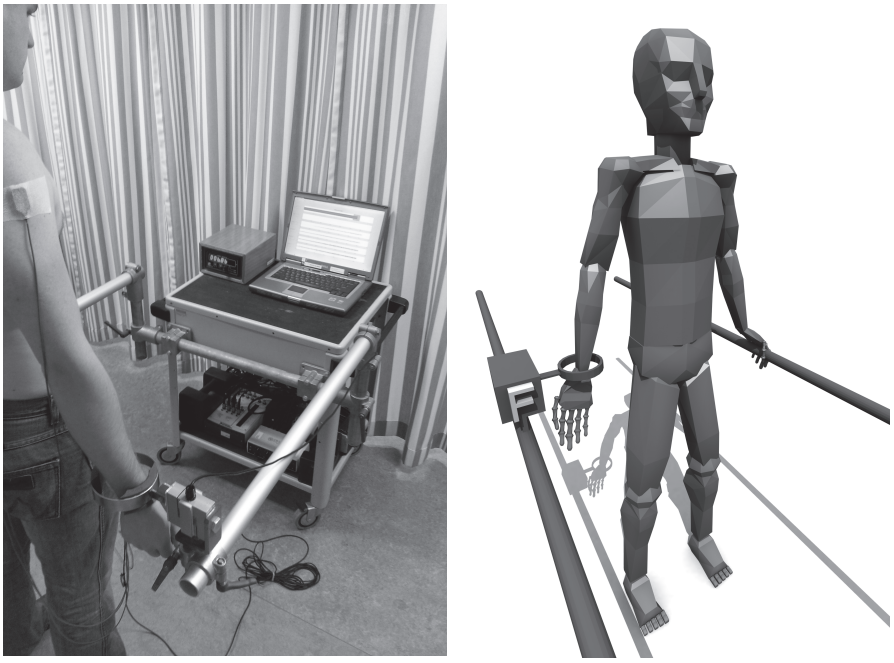


Figure 2. Experimental set-up.
Subjects were in standing position with the concerning arm in external rotation at his/her side (i.e. hand in frontal plane), enabling the use of this set-up during concomitant acquirement of standard shoulder radiographs for future clinical or scientific purposes. The arm was attached to a 1-dimensional force transducer at the wrist. In this set-up, subjects performed EMG-recorded isometric abduction and adduction force tasks.

2.4 Statistics

Demographic data of controls and patients were compared using an unpaired t-test for continuous data (age) and Fisher's exact test for categorical data (gender, arm dominance).

The influence of arm dominance, gender and age on muscle specific *Activation Ratios* in healthy subjects was assessed using multivariate regression analyses.

Activation Ratio test-retest reproducibility was studied for each muscle in the healthy subjects, using the interclass correlation coefficient (ICC) [21] and standard error of measurement (SEM) as advised by Weir et al.[22] The ICC is a relative measure of consistency and is constituted by a ratio between the 'variance between subjects' and the sum of variances ('between subjects' and 'within subjects'). It is therefore population dependent and a poor test-retest consistency can be masked by a high ICC when between-subjects variability is high. The SEM provides an absolute measure of reliability and assesses the consistency of scores within individual subjects, largely independent from the population it is calculated from. It is the

standard error when estimating "true" scores from "observed" scores, or the standard deviation within subjects. SEM can be estimated using the square root of the mean square error term from ANOVA analyses, or with the formula Eq. 2:

$$SEM = SD \times \sqrt{1 - ICC} \quad \textbf{(2)}$$

where SD is the pooled standard deviation of the test and retest measurements of all subjects. In this study, we used the ANOVA method.

The *Activation Ratios* of healthy controls and patients were compared in a multivariate regression analysis, and the influence of disease status (healthy vs. rotator cuff tear) on muscle *Activation Ratios* was assessed.

P-values of ≤ 0.05 were considered statistically significant. Analyses were processed using SPSS 16.0 software (SPSS Inc., Chicago, Illinois).

3. Results

Out of 20 patients, 18 subjects (6 females; mean age 61.3 years, SD=9.9) were compared to 20 healthy controls (5 females; mean age 25.4 (SD=2.6) years). Two patients (9.5%) were excluded: one because of a very painful shoulder and symptoms of glenohumeral subluxations during the experiment, one because of a very painful shoulder during the experiment and severe hand-eye coordinative problems. Overall, mean patients' Constant Score was 47.8 points (SD=13.4). Mean VAS for pain during daily activities was 4.71 cm (SD=3.21).(Table 1)

		Patients	**Healthy subjects**	**Mean Difference**	**P-value**
		(n = 18)	(n = 20)	(95%-CI)	
Age		61.3 [9.9]	25.4 [2.6]	35.9 (31.0 - 40.9)	< 0.001
Gender	*Male*	12	15		
	Female	6	5		0.72
Measured arm	*Dominant*	8	10		
	Non-dominant	10	10		0.76
Clinical Scores	*Constant Score*	47.8 [13.4]	-		
	VAS-pain (0-10)	4.71 [3.21]	-		

Table 1. Demographic data, mean patient Constant Score, and VAS-pain scores of patients and healthy subjects.
[SD]: [Standard Deviation], 95%-CI: 95% Confidence Interval, VAS: Visual Analogue Score.

3.1 Activation Ratios in healthy subjects

Mean *Activation Ratios* for dominant and non-dominant arms and both repetitions in healthy subjects (n = 80 measurements) ranged between 0.68 (SD=0.25) for the TM and 0.89 (SD=0.09) for the DM.(Table 2)

In a multivariate regression model, including session 1 (test) and 2 (retest) and a random effect for each subject, the influence of arm dominance, gender and age on *AR* of each muscle in healthy subjects was assessed. Effect sizes of gender and age were small and statistically insignificant for all muscles. Arm dominance had a relevant and statistically significant effect for the LD and TM, with effect sizes of -0.096 (p=0.02) and -0.102 (p=0.021), respectively. Therefore, in healthy subjects, the average non-dominant arm *AR* is 0.1 points lower compared to the *AR* of the dominant arm for both LD and TM.

Test-retest reproducibility was assessed using intraclass correlation coefficients (2.1 ICC model for agreement and with random effects [21]), demonstrating moderate to good ICCs for the DM, TM and LD, with ICCs of 0.66, 0.74 and 0.60 respectively. However, ICC for the PM was poor, with -0.105, which was the consequence of several outliers: subjects with high (normal) *AR*'s in the test evaluation and low *AR*'s in the retest measurement, or vice versa.

Standard error of measurement values (SEM) were 0.05, 0.12, 0.14 and 0.18 for the DM, TM, LD and PM, respectively. Consequently, in an average healthy subject with observed *Activation Ratios* of $AR_{DM} = 0.9$, $AR_{TM} = 0.7$, $AR_{LD} = 0.8$ and $AR_{PM} = 0.8$, true AR values will be higher than 0.80, 0.46, 0.53 and 0.45 respectively, expressed in lower bounds of the within-subject 95% Confidence Intervals (1.96 x SEM). Minimum detectable differences within subjects (1.96 x SEM x $\sqrt{2}$) were $AR_{DM} = 0.14$, $AR_{TM} = 0.33$, $AR_{LD} = 0.39$ and $AR_{PM} = 0.50$.

		Healthy, Session 1		**Healthy, Session 2**		**All measurements**	**Patients**
		Dominant side	*Non-dominant side*	*Dominant side*	*Non-dominant side*		
		(n = 20)	(n = 20)	(n = 20)	(n = 20)	(n = 80)	(n = 18)
AR	*DM*	0.89 [0.09]	0.90 [0.07]	0.89 [0.10]	0.89 [0.09]	0.89 [0.09]	0.63 [0.17]
	PM	0.79 [0.17]	0.75 [0.24]	0.77 [0.16]	0.80 [0.08]	0.78 [0.17]	0.57 [0.22]
	LD	0.86 [0.13]	0.75 [0.24]	0.80 [0.20]	0.75 [0.26]	0.79 [0.21]	0.49 [0.34]
	TM	0.74 [0.20]	0.62 [0.22]	0.73 [0.25]	0.61 [0.31]	0.68 [0.25]	0.26 [0.26]

Table 2. Muscle Activation Ratios (AR) for the Deltoid (DM), Latissimus Dorsi (LD), Pectoralis Major (PM) and Teres Major (TM) muscles in healthy subjects (n=20) for both arms on 2 separate moments, and for patients (n=18).
Especially *Activation Ratios* of caudally directed adductors (TM and LD) are lower in patients, indicating increased co-activation of *ad*ductors during arm *ab*duction tasks.
[SD]: [Standard Deviation]

3.2 Activation Ratios in patients compared to healthy subjects

Mean *Activation Ratios* of the affected arms in patients were lower, ranging between: 0.26 (SD=0.26) for the TM and 0.63 (SD=0.17) for the DM.(Table 2)

We analyzed the influence of disease status (healthy (n=80) or rotator cuff tear (n=18)) on the *Activation Ratios*, using a multivariate regression model with a random effect per subject, and disease status and arm dominance as fixed effects. For the DM, LD and TM effect of disease status was significant, with B-values of -0.26 ($p<0.001$), -0.30 ($p<0.001$) and -0.41 ($p<0.001$), respectively. This means e.g. that the average *Activation Ratio* of the LD in patients is 0.3 points lower compared to the *Activation Ratio* of healthy subjects, corrected for arm dominance. Overall, there was increased co-activation for all muscles in patients, but in particular for the mediocaudally directed adductors: LD and TM.(Figure 3)

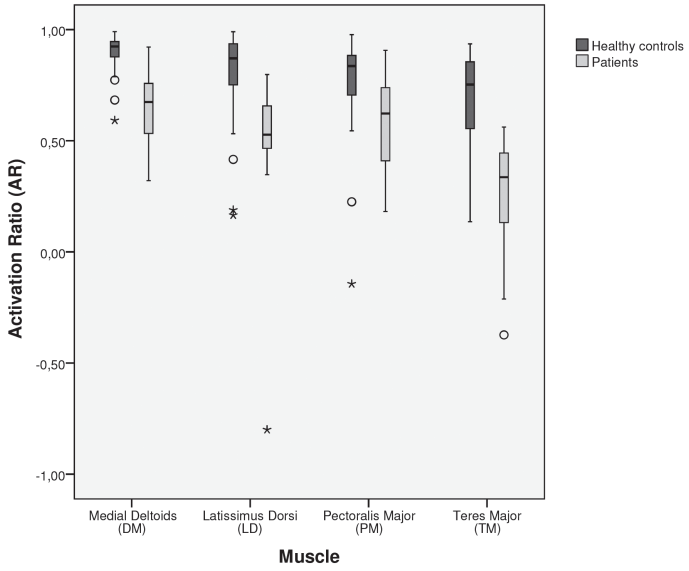


Figure 3. Boxplot of Activation Ratios for patients and healthy controls.
On average, *Activation Ratios* of patients are lower than *Activation Ratios* of controls, especially for the mediocaudally directed arm adductors (LD and TM). O = Mild outliers (within 1.5 x Interquartile range (IQR)), * = Extreme outliers (> 3.0 x IQR)).

Based on these data, we defined that an *Activation Ratio* of minimal 0.3 points lower compared to average control *Activation Ratios* was indicative for pathologic co-activation. In this study, 15 out of 18 patients (83%) demonstrated co-activation for one or both mediocaudally directed adductors (LD and TM). This was TM co-activation in 12 patients, which was combined with LD co-activation in five patients. Three patients showed isolated co-activation of the LD and three patients had no co-activation. Unexpectedly, 5 patients also had co-activation of the Deltoid using

the > 0.3 difference as a cut-off value. Three of these patients, besides the 2 excluded ones, indicated serious pain during the experiment.

4. Discussion and conclusions

The introduced measuring instrument successfully discriminates cuff tear patients from healthy subjects by quantifying pathologic shoulder muscle activation patterns in *Activation Ratios*. The measurements are easy to perform, take around 5 minutes, are applicable as a new objective outcome measure, can be combined with simultaneous acquirement of standard shoulder radiographs, and provide moderate to good test-retest outcomes. As in previous studies, the *Activation Ratio* proved to be an easily interpretable measure of EMG muscle activation, which can be used to directly compare muscle activity within and between subjects.[12, 17] The increased 'out-of-phase' activation (co-activation) of shoulder muscles in patients was depicted in significantly lower *AR*'s.(Table 2) Differences were largest for mediocaudally directed arm *ad*ductors in particular (LD and TM), indicating a relatively great amount of *ad*ductor co-activation during arm *ab*duction in cuff tear patients.

Hypothetically, arm *ad*ductor co-activation during active arm *ab*duction tasks, as reported in mathematical model simulation and experimental EMG studies, restrains excessive subacromial narrowing in patients with rotator cuff lesions.[12, 13, 15-17] However, reported methods for assessing arm adductor co-activation are rather laborious to perform, complicated to interpret and take around 30-60 minutes. In the present study, we introduced an experimental set-up with easily obtainable objective outcome measures, applicable in research, during the acquirement of standard shoulder radiographs, or even in clinical setting. We used the *Activation Ratio* as introduced by Steenbrink and co-workers as primary outcome measure, which allows easily interpretable comparisons of normalized muscle activation, measured with EMG or determined in simulations, within and between subjects.[12, 17] In support of previous publications, we found lower adductor *Activation Ratios* in cuff tear patients as compared to healthy controls, especially for the caudally directed TM and LD. Mean adductor *Activation Ratios* were somewhat higher compared to published values for both patients and healthy controls, demonstrating experimental set-up or arm position dependency of the *Activation Ratio*. Mean differences of adductor *Activation Ratios* between patients and healthy subjects ranged from 0.26 to 0.49, i.e. of the same order of magnitude as in previous studies. All patients had an *Activation Ratio* lower than the mean control *Activation Ratio* value for both the TM and LD. Defining an *Activation Ratio* difference of below -0.3

compared to mean *Activation Ratio* control values of healthy subjects as a cut-off value for pathologic 'out-of-phase' muscle activation, 15 (83.3%) of our patients demonstrated co-activation in at least 1 mediocaudally directed adductor muscle. This indicates that *Activation Ratio*, as measured with this set-up is a responsive measure, capable of differentiating patients with cuff tears from healthy subjects.

It is plausible that the 3 patients without observed co-activation didn't have excessive subacromial narrowing in spite of their cuff tear, or even have shoulder complaints due to another cause than the diagnosed cuff tear; asymptomatic cuff tears are common and shoulder problems can be very heterogeneous and complex to diagnose. On the other hand, these patients might have had insufficient coping strategies for subacromial narrowing (i.e. no co-activation of adductors). Currently, we are recording *Activation Ratios* in combination with radiographs in a new study with cuff tear patients. Identification of co-contraction responders and non-responders in combination with actual cranial translation measurements will give more insight in underlying etiologic and biomechanical mechanisms.

With regard to the PM measurements, results were less uniform in both patients and healthy subjects. Mean AR_{PM} in patients was higher compared to other adductor *AR*'s, with consequent inferior potential in discriminating patients from healthy subjects. Additionally, AR_{PM} reproducibility in healthy subjects was not good. And with the position of the arm vertically downward alongside the body, the PM has hardly any depressor capacity to pull the humerus downward, in contrast to the elevated arm position in previous studies (Steenbrink et al. 2009b, 2010ab). Therefore, we don't recommend using *AR* measurements of the PM with this particular set-up in the evaluation of rotator cuff patients.

The strengths of our study include the comparison of two moderately large and well-defined groups, the radiological imaging (MRI) and clinical scores acquired in all patients, the use of objective outcome measures (EMG) and a comprehensive measure for EMG-recordings (*AR*), and the use of a newly developed set up which proved to be easy to use and provided moderate to good reliability.

There are some weaknesses, which need to be taken into account when interpreting our results. Firstly, there was an age difference between patients and healthy controls. It has been reported that adductor co-activation seems not to be age-dependent,[17] and this was confirmed in the multivariate analyses in the current study. Whether the differences in *AR* between the two groups are predominantly caused by age, which is not plausible, by rotator cuff pathology, or by other factors is not very relevant. Yet, the introduced method is able to produce outcome measures that discriminate the two groups of subjects with moderate to good reproducibility. Nevertheless, further research comparing *AR*'s in RC lesion patients to age-matched controls is recommendable, especially with regard to studying the hypothesized

underlying biomechanical mechanisms of adductor co-activation. Secondly, we only analyzed muscle activation with surface electrodes and did not include other arm abductors and adductors. Biomechanically, the measured muscles are the most important ones when studying humerus cranialisation and adductor co-activation. Furthermore, the main goal of our study was to develop an easy-to-use method with objective outcome measures, which can be used with simultaneous acquirement of radiographs and has potential for application in clinical setting. Therefore, we have chosen to keep the number of muscles limited, and did not use fine wire electrodes for EMG recording.

Concluding, in this study, 'out-of-phase' activation (co-activation) of arm *ad*ductor muscles was demonstrated in patients with rotator cuff tears during arm *ab*duction tasks, in contrast to healthy subjects. The introduced experimental set-up is capable of quantifying this arm adductor co-activation, expressed in low or negative *Activation Ratios*, which can be easily compared within and between subjects. The system is straightforward to use, has interpretable outcomes, and can be applied as an objective outcome measure of shoulder muscle function. It has potential as a tool for discriminating symptomatic and asymptomatic cuff tears, for clinical decision making (physical therapy vs. surgery), for surgical planning (Latissimus Dorsi vs. Teres Major transfer), or for the evaluation of treatment effects. However, further research is needed to study the interaction of co-activation and humerus cranialisation, and the clinical applicability of this experimental set-up and *Activation Ratio* in patients with cuff and other shoulder pathologies.

Acknowledgements

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Corrigendum to:

Arm ADductor activation with arm ABduction in rotator cuff tear patients vs. healthy controls -Design of a new measuring instrument-

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Hum. Mov. Sci. 2013;32:875-876

The authors regret that an error has appeared in article entitled "Arm ADductor Activation during arm ABduction in healthy subjects and patients with rotator cuff tears -Validation of a new measuring instrument- " .
In the processing of the EMG recordings, rEMG rest signal was not correctly subtracted from the rEMG tasks signals in the patient group (not the controls). Consequently, reported *Activation Ratios* of the patient group were too low.
The first sentence of paragraph 3.2 needs to change from "Mean *Activation Ratios* of the affected arms in patients were lower, ranging between: 0.26 (SD=0.26) for the TM and 0.63 (SD=0.17) for the DM." to "Mean *Activation Ratios* of the affected arms in patients were lower, ranging between: 0.32 (SD=0.31) for the TM and 0.75 (SD=0.15) for the DM." Accordingly, the corrected Table 2 is now:

		Healthy, Session 1		**Healthy, Session 2**		**All measurements in healthy subjects**	**Patients**
		Dominant side	*Non-dominant side*	*Dominant side*	*Non-dominant side*		
		(n = 20)	(n = 20)	(n = 20)	(n = 20)	(n = 80)	(n = 18)
AR	*DM*	0.89 [0.09]	0.90 [0.07]	0.89 [0.10]	0.89 [0.09]	0.89 [0.09]	0.75 [0.15]
	PM	0.79 [0.17]	0.75 [0.24]	0.77 [0.16]	0.80 [0.08]	0.78 [0.17]	0.70 [0.20]
	LD	0.86 [0.13]	0.75 [0.24]	0.80 [0.20]	0.75 [0.26]	0.79 [0.21]	0.65 [0.40]
	TM	0.74 [0.20]	0.62 [0.22]	0.73 [0.25]	0.61 [0.31]	0.68 [0.25]	0.32 [0.31]

Corrected **Table 2. Muscle Activation Ratios (AR) for the Deltoid (DM), Latissimus Dorsi (LD), Pectoralis Major (PM) and Teres Major (TM) muscles in healthy subjects (n=20) for both arms on 2 separate moments, and for patients (n=18).**
Especially *Activation Ratios* of caudally directed adductors (TM and LD) are lower in patients, indicating increased co-activation of *ad*ductors during arm *ab*duction tasks.
[SD]: [Standard Deviation]

We applied the same multivariate regression model as in the publication on *Activation Ratios*, including disease status, a random effect per subject and arm dominance as variables. For DM and TM, effect of disease status was significant, in concordance with the publication, with ß-values of –0.14 ($p<0.001$) and –0.35 ($p<0.001$), respectively. The effect of LD was now -0.13 ($p=0.11$). There was again no significant effect for PM: -0.07 ($p=0.13$).
Hence, especially for DM, PM and LD the differences with healthy subjects appear smaller than in the previously published article. However, the main effect as reported in the publication is still evident: primarily co-activation of TM in cuff tear patients. Consequently, the conclusions in the publication do not change. However, when assessing adductor co-activation with this particular set-up, it seems advisable to primarily focus on the TM and not necessarily LD.





CHAPTER 10

Cranial humerus translation, Deltoid activation, Adductor co-activation and rotator cuff disease - Different patterns in rotator cuff tears, subacromial impingement and controls -

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Clin. Biomech. (Bristol, Avon). 2014 Jan;29(1):26-32

Abstract

Background: Arm adductor co-activation during abduction has been reported as a potential compensation mechanism for a narrow subacromial space in patients with rotator cuff dysfunction. We assessed differences in acromiohumeral distance at rest and the amount of humerus translation during active abduction and adduction in patients with rotator cuff tears (n=20) and impingement (n=30) and controls (n=10), controlled for Deltoid, Pectoralis Major, Latissimus Dorsi and Teres Major activation (electromyography).

Methods: During the acquirement of shoulder radiographs, subjects performed standardized isometric arm abduction and adduction tasks. EMGs were normalized between -1 and 1 using the "Activation Ratio", where low values express (pathologic) co-activation, e.g. adductor muscle activation during abduction.

Findings: In patients with cuff tears mean rest acromiohumeral distance was 7.6mm (SD=1.6): 3.5mm narrower compared to patients with impingement (95%-CI: 2.4-4.5) and 1.3mm narrower compared to controls (95%-CI: -0.1-2.7). Both during abduction and adduction tasks, cranial translation was observed with equal magnitudes for patients and controls, with average values of 2.3 and 1.7mm, respectively. Where patients with cuff tears had lower adductor Activation Ratios (i.e. more adductor co-activation during abduction), no association between abductor/adductor muscle activation and acromiohumeral distance was found.

Interpretation: The subacromial space is narrower in patients with rotator cuff tears compared to patients with impingement and controls. We found additional subacromial narrowing during isometric abduction and, to a lesser amount, during adduction in all subjects and more adductor co-activation in patients with cuff tears. We found no association between subacromial space and activation of the Deltoid and main adductors.

Keywords: Rotator Cuff; Shoulder Impingement Syndrome; Electromyography; Diagnostic Techniques and Procedures; Adductor Co-activation

1. Introduction

The incidence of shoulder complaints in general practice is high, with 22 per 1000 registered patients per year.[1] 44-65% of shoulder symptoms are diagnosed as "Subacromial Impingement Syndrome" (SIS)[2, 3] and 36% of subjects with shoulder symptoms have been demonstrated to have rotator cuff tears.[4] Both conditions show similar symptoms, including pain and loss of arm abduction force, although symptoms are generally worse in patients with rotator cuff (RC) tears.[5, 6] Some report that both are stages of the same condition, where SIS may progress to a RC tear due to muscle and tendon degeneration.[6-10] We assessed similarities and differences in objective biomechanical signs suggestive for RC dysfunction in patients with SIS or RC tears and controls.

A narrow subacromial space due to a cranial position of the humerus relative to the acromion has been radiologically demonstrated in patients with RC tears.[11-14] A narrow subacromial space has been associated with shoulder pain, larger RC tear sizes, progression of RC tears to multiple tendons and RC degeneration, and has been reported as a negative prognostic factor for (surgical) treatment.[11-17] *Additional* narrowing of the subacromial space (i.e. *cranial humerus translation*) during active arm abduction has been reported on radiographs and magnetic resonance imaging (MRI) acquired in healthy subjects, and patients with RC tears or SIS.[18-21] In these studies, it was postulated, that RC deficit patients show more *cranial humerus translation* than healthy subjects and that this cranial translation may be a diagnostic tool or an objective clinical outcome measure.

Subacromial narrowing is the subject of an increasing number of publications and is more and more applied in clinical practice, but its underlying mechanisms are remain unclear. RC muscles play a key role in glenohumeral (GH) stabilization and arm mobility; in the healthy shoulder a perfect compromise is assumed between mobility and stability.[22] In patients with RC tears, GH joint mechanics are disrupted as one or more RC muscles are dysfunctional. This may lead to 1) a compensatory increase in Deltoid activity;[23-25] 2) GH (micro-)instability;[25] 3) excessive cranial humerus translation with the activation of arm abductors[11, 12, 18, 19, 21, 25-29] and subsequent pain. It has been hypothesized that co-activation of specific adductor muscles with downwardly directed lines of action (e.g. Teres Major and Latissimus Dorsi) is a compensation mechanism to counteract this excessive cranial translation during abduction in patients with RC tears.[24, 25, 30-32]

The actual relation between cranial humerus translation, pain and adductor co-activation as a potential protective mechanism has been scarcely investigated experimentally,[24, 30-32] specifically for patients with SIS, in whom the RC is still anatomically functional. Patients with RC tears can be regarded as ultimate

demonstrators for RC dysfunction. If translation and adductor co-activation are also found in patients with SIS, disrupted GH joint mechanics might exist in these patients, despite intact RC muscles. This would indicate that in addition to the many reported etiologies of SIS, decreased RC function and altered biomechanics might play a role as well.

We investigated acromiohumeral distance (*AH*) on radiographs acquired during electromyography(EMG)-recorded isometric abduction, adduction and rest tasks in patients with RC tears, patients with SIS and controls. Our primary goals were to 1) assess *AH* at rest and humerus translation (Δ*AH*) during abduction and adduction force tasks, 2) assess (pathologic) co-activation of Deltoid, Latissimus Dorsi, Teres Major and Pectoralis Major, and 3) assess the association between *AH*, Deltoid activation and adductor (co-)activation. We hypothesized that 1) in patients baseline (rest) *AH* is smaller and Δ*AH* larger compared to controls; 2) patients apply more adductor co-activation during abduction compared to controls; and 3) Deltoid activation is negatively and adductor co-activation is positively related to *AH* and Δ*AH*. We expect healthy controls, patients with SIS and patients with RC tears to order along the scales of subacromial narrowing and adductor co-activation.

2. Methods

2.1 Subjects

Consecutive patients with a painful shoulder and a full-thickness RC tear (Supraspinatus and/or Infraspinatus) and patients diagnosed with SIS were included in this study during the period of April 2010 to April 2012. In addition, 10 controls were recruited in September 2012. Inclusion criteria for the controls were: age between 35 and 60 years (minimizing the prevalence of eventual asymptomatic cuff tears and age differences between controls and patients), no present shoulder complaints and no history of shoulder complaints treated with physical therapy, NSAIDs, injections, or surgery. Controls were assessed by an MD for eventual shoulder symptoms.

Patients with SIS or RC tears had to have a positive Neer impingement test, a positive Hawkins test and diffuse unilateral anterosuperior shoulder pain for >3 months in combination with one or more of the following criteria: pain with overhead activities, abduction, retroflexion and/or internal rotation (e.g. closing the door, putting on jacket); pain at night or incapable of lying on the shoulder; diffuse pain at palpation of the greater tuberosity; disturbed scapulohumeral rhythm; classic painful arc; positive Yocum test; positive full or empty can test.

Patients with SIS had to be aged between 35 and 65 years. Exclusion criteria for SIS were partially based on an MRI arthrogram and standard anteroposterior (AP) shoulder radiographs: no calcific tendinitis, full-thickness RC tear, intra-articular or bony lesions (Hill Sachs, (old) fractures, tumors), labrum abnormalities, capsular or ligamentous tears/avulsions, superior labrum tear from anterior to posterior (SLAP lesion), pulley lesion, Biceps tendinitis or tear, os acromiale, cartilage lesions, or bony cysts. Patients with RC tears had to be aged 50 years and older. They were symptomatic and had a standard AP shoulder radiograph and MRI arthrography or ultrasound (US)-proven full-thickness RC tear, without a Subscapularis tear and other shoulder pathologies. Subjects were furthermore excluded in case of insufficient Dutch language skills or no informed consent, present physical problems influencing muscle activation and arm mobility (other than the present shoulder condition), any form of inflammatory arthritis of the shoulder, glenohumeral or symptomatic acromioclavicular osteoarthritis, a history of fracture, dislocations, or surgical interventions of the shoulder, clinical signs of cervical radiculopathy and frozen shoulder syndrome (<90° of passive abduction and external rotation).

Radiographic data on acromiohumeral distance in the RC tear group were previously published.[33] In the current study these data are re-assessed with permission of the authors. Additionally, we included simultaneously acquired EMG-recordings in the analyses and used the data of this group with another goal: to compare acromiohumeral distance in three subject groups and to relate the acromiohumeral distance with muscle activation patterns, including adductor co-activation.

Patients were clinically assessed using the Western Ontario Rotator Cuff score (WORC)[34] and the Constant Score (CS).[35]

The accredited local Medical Ethics Committee (METC, Leiden University Medical Center) approved all stages of the study according to the Medical Research Involving Human Subjects Act and written informed consent was obtained from all participants (Dutch Trial Registry NTR2283).

2.2 Experimental set-up

Included subjects performed standardized isometric arm abduction and adduction tasks of equal force magnitude by pressing against a single axis force sensor (Penko Engineering, Ede, the Netherlands) with the arm alongside the body, using a previously introduced experimental set-up with visual feedback.[33, 36] (Figure 1) Thus, the task forces were perpendicular to gravitational forces. This set-up was specifically developed to enable reproducible and isometric arm tasks and to acquire standard shoulder radiographs in combination with EMG.[36] Accordingly, AP shoulder radiographs were acquired during rest and during isometric abduction and adduction force tasks to obtain acromiohumeral distance in combination with

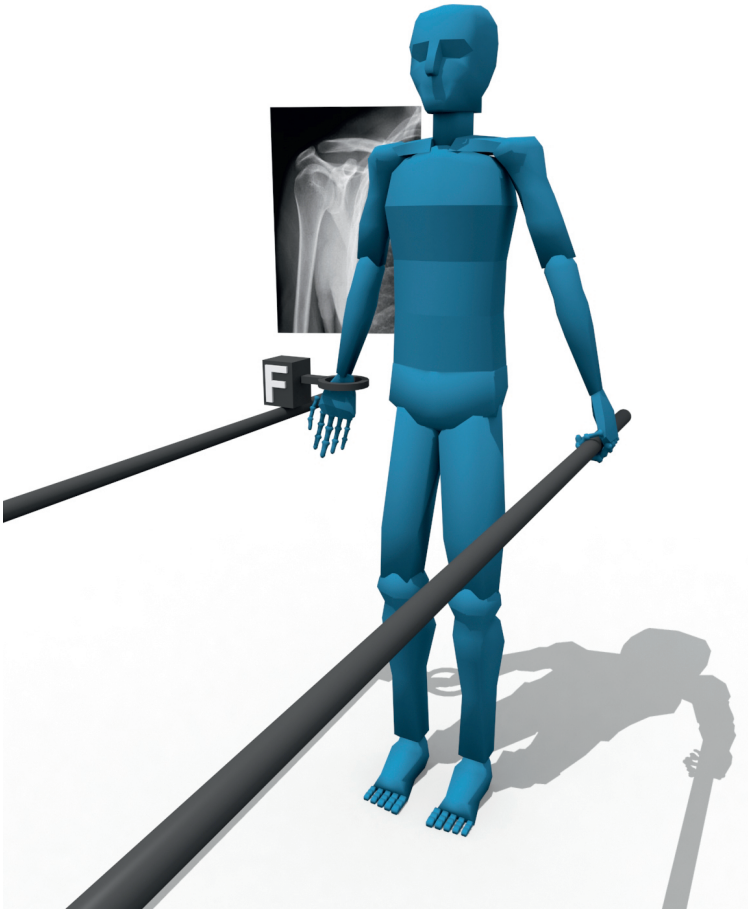


Figure 1. Experimental set-up.
Subjects were in standing position with the target arm in external rotation at his/her side (i.e. hand in frontal plane), enabling the use of this set-up during concomitant acquirement of standard shoulder radiographs for clinical or scientific purposes. The arm was attached to a 1-dimensional force transducer at the wrist. In this set-up, subjects performed EMG-recorded isometric abduction and adduction force tasks.

EMG for each task. Before the radiograph series, maximum voluntary force (MVF) was measured for abduction and adduction for each subject. Final subject-specific force task magnitude was 60% (±3.75% tolerance) of the minimal value of adduction and adduction MVF.[33, 36] All subjects performed at least one practice round and a series during which the radiographs were acquired.

Patients were assessed on their affected arm. For healthy subjects, the investigational side was determined by randomization (computer generated randomisation list) preceding inclusion.

2.3 Radiographs and humerus translation

The force controlled shoulder radiographs with simultaneously recorded EMG were acquired in a standardized setting. Arm position was constant during all tasks for each subject: the upper body was rotated 30° from the frontal plane towards the Röntgen focus beam with the arm at the subjects' side and the hand in the frontal plane with the palm facing forward.[33, 36]

Radiograph quality was on-site controlled for using prescribed criteria[33] and eventually re-acquired. On each radiograph, acromiohumeral *distance* (*AH*) and humerus *translation (ΔAH)* were assessed. For *AH*, the distance between the most cranial articular cortex of the humeral head and the caudal cortex marking of the caudal surface of the acromion was measured in millimetres.[26, 28, 29] Task specific humerus translation was calculated from the differences between rest *AH* and abduction or adduction *AH*, obtaining ΔAH_{ab} and ΔAH_{ad}, respectively. When the necessary anatomical landmarks could not be identified, the measurement could not be performed and was reported as missing.

2.4 Electromyography and adductor co-activation

During the rest, abduction and adduction tasks, EMG of 3 shoulder adductors (Pectoralis Major, clavicular part (PM); Teres Major (TM); Latissimus Dorsi (LD)) and the main shoulder abductor (Deltoid, medial part (DM)) were recorded with bi-polar surface EMG equipment (DelSys system Bagnoli-16, Boston, MA, USA, inter-electrode distance 10mm, bandwidth 20–450 Hz) as described previously.[36]

As absolute magnitude of EMG-signals is hard to interpret and cannot be compared between subjects or related to *AH*, EMG-recordings were expressed 1) relative to Maximal Voluntary Contraction EMG (*MVC*) and 2) in *Activation Ratios*. For this purpose, EMG was rectified (*rEMG*) and averaged (*aEMG*). Muscle activation during tasks (A_{muscle}) was determined by subtracting the rest *aEMG* from the active *aEMG* during abduction and adduction, respectively. EMG quality (signal-to-noise-ratio) was controlled for: if *rEMG* was less than 2 times more active during tasks compared to rest activity, the concerning measurement was not included in statistical analyses. *rEMGs* were normalized to *MVC* for each task, muscle and subject. Additionally, the *Activation Ratio* was calculated, which combines the (absolute/not normalized) *rEMGs* of abduction and adduction tasks, in order to quantify muscle specific activation during agonist and antagonist tasks. For each muscle and subject, the following formula was applied, Eq. 1:

$$AR_{muscle} = \frac{A^{IP}_{muscle} - A^{OP}_{muscle}}{A^{IP}_{muscle} + A^{OP}_{muscle}} \qquad \left[-1 \leq AR_{muscle} \leq 1\right] \qquad \textbf{(1)}$$

where A^{IP}_{muscle} is 'in phase' or agonist muscle activation (DM activation during abduction and TM, PM and LD activation during adduction) and A^{OP}_{muscle} is 'out-of-phase' or antagonist muscle activation (DM activation during adduction, or TM, PM and LD activation during abduction, i.e. pathologic co-activation).[32, 36] The *Activation Ratios* of most muscles in healthy subjects are positive and close to 1. In case of substantial antagonist activation of specific arm *ad*ductors during *ab*duction tasks (adductor co-activation), as has been previously described for the LD and TM in patients with RC tears, the *Activation Ratios* of these adductor muscles are expected to be close to zero or even negative.[32]

2.5 Statistics

Demographic data and clinical scores (available in patients) were expressed in means and standard deviations or medians and ranges where appropriate.

For radiographic measures, the mean *AH's* (for rest, abduction and adduction) and the ΔAH_{ab} and ΔAH_{ad} in the three groups were all assessed using one-way ANOVA analyses. A similar approach was applied for the MVC-normalized *rEMGs* during rest and the *Activation Ratios* during abduction and adduction for the four muscles in patients from the three groups.

Multivariate Mixed Model analysis for repeated measures was applied to study the association between *AH* (dependent variable) and disease status (control, impingement, RC tear), task (rest, abduction, adduction) and the MVC-normalized muscle activations of DM, PM, LD and TM.

P-values of ≤0.05 were considered statistically significant. Analyses were processed using PASW Statistics 20.0 (IBM Inc., Chicago, Illinois, USA). Multivariate analyses were processed using R 2.15.2.

3. Results

60 Subjects were included: 20 patients with RC tears, 30 patients with SIS and 10 controls. Two patients with SIS were excluded due to hardware problems during the experiments, leaving 58 subjects for analyses.(Table 1)

Baseline characteristics	SIS	RC tear	Control
	n=28	n=20	n=10
Age (yrs)	50.1 [1.6]	65 [9.8]	50.2 [6.6]
Gender (male/female)	11/17	11/9	5/5
Affected side (right/left)	17/13	12/8	6/4
Dominant side affected	18 (64%)	18 (90%)	NA
BMI	26.5 [4.3]	28 [4.5]	NA
Baseline clinical scores			NA
WORC	61.7 [14.8]	57 [22]	NA
Constant Score	76.9 [8.5]	62 [15]	NA

Table 1. Demographic data of patients and controls.
SIS, subacromial impingement syndrome; RC, rotator cuff; WORC, Western Ontario Rotator Cuff index [SD]: [Standard Deviation]

Measurement	RC tear		SIS		Control		ANOVA
	mean	95%-CI	mean	95%-CI	mean	95%-CI	p-value
AH	7.6	(6.90 - 8.39)	11.1	(10.40 - 11.83)	8.9	(7.55 - 10.31)	**<0.001**
Cranial translation							
ΔAHab	2.6	(1.86 - 3.39)	2.3	(1.53 - 3.06)	2.1	(1.46 - 2.68)	0.675
ΔAHad	1.0	(0.29 -1.79)	1.1	(0.52 - 1.73)	0.8	(-0.02 - 1.60)	0.837

Table 2. Distance between humerus and acromion at rest, and amounts of cranial humerus translation during abduction and adduction in patients with RC tears, patients with SIS and controls.
AH, acromiohumeral distance (at rest); Δ*AH*, difference in acromiohumeral distance during abduction (ab) or adduction (ad) compared to rest; 95%-CI, 95% Confidence Interval; RC, Rotator Cuff; SIS, subacromial impingement syndrome

3.1 Acromiohumeral distance

Two adduction radiographs needed to be reacquired in patients with RC tears, while not fulfilling the pre-set quality criteria. Furthermore, one adduction radiograph could not be assessed in a patient with SIS.

Mean *AH* at rest condition was 7.6mm (SD=1.60) in patients with RC tears, 11.1mm (SD=1.84) in patients with SIS and 8.9mm (SD=1.92) in controls.(Table 2, Figure 2) Average AH_{REST} was significantly different in the three subject groups (p<0.001). Post hoc analyses revealed that in patients with RC tears mean AH_{REST} was 3.5mm smaller (95%-CI: 2.4-4.5, p<0.001) compared to patients with SIS, and 1.3mm smaller (95%-CI: -0.1-2.7, p=0.07) compared to controls. Mean AH_{REST} for patients with SIS was 2.2mm larger (95%-CI: 0.9-3.5mm, p=0.002) compared to controls.

Also during abduction (p<0.001) and adduction (p<0.001), *AH* was significantly different between the three groups. In post-hoc analyses, mean *AH* during abduction and adduction was significantly lower in patients with RC tears compared to patients with SIS, with differences of 3.8mm (95%-CI: 2.6-5.0) and 3.4mm (95%-CI: 2.2-4.6), respectively. Additionally, *AH* was significantly lower in patients with RC tears compared controls: 1.8mm (95%-CI: 0.3-3.4) and 1.5mm (95%-CI: 0.0-3.1), respectively.

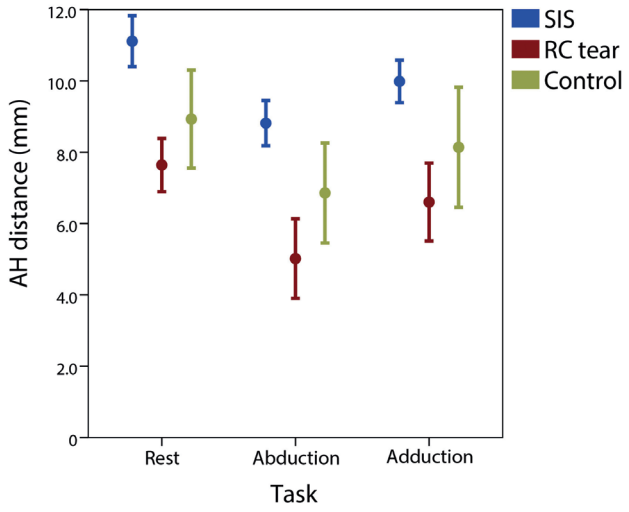


Figure 2. Acromiohumeral distance (AH) during rest and the isometric active abduction and adduction tasks for all subject groups.
Overall, *AH* was narrower in patients with RC tears compared to controls and wider in patients with SIS. All subject groups showed similar amounts of cranial translation relative to rest during the abduction and adduction tasks.

All groups showed significant cranial humerus translation during the active tasks, except for the control group during adduction. Mean translations for the three groups ranged between 2.1 and 2.6mm for ΔAH_{ab} (abduction) and between 0.8 and 1.1 for ΔAH_{ad} and were not significantly different between the subject groups. (Table 2)

3.2 Muscle activation

Muscle EMG at rest, normalized to MVC, is displayed in Figure 3. Overall, there was more rest activity in patients than in controls. Post-hoc analyses revealed that for PM, there was significantly more rest activity in patients with RC tears compared to controls (p=0.007) and patients with SIS (p=0.04). For LD, there was significantly more activity in patients with RC tears compared to controls (p=0.04).
Mean *AR*'s in patients with RC tears, assessing "out-of-phase" muscle activation (lower values), ranged from 0.42 to 0.58 for the adductors. For the abductor (DM), *AR* was 0.72 in this group. Average SIS *AR* for TM was 0.51. *AR*'s for the DM and the LD and PM adductors ranged from 0.69 to 0.72. In controls, mean *AR*'s ranged from 0.72 to 0.78 for DM and the adductors LD and PM. Mean TM *AR* in controls was 0.41. (Figure 4)

Overall, there were no significant differences between *AR*'s in the three groups with one-way ANOVA analyses. With post-hoc analyses the *AR* of the PM was significantly lower in patients with RC tears compared controls: 0.53 vs. 0.79, leading to a mean difference of 0.26 (95%-CI: 0.03-0.24, p=0.03).

3.3 Relation between acromiohumeral distance, muscle activation and disease status

The alleged association between *AH* and muscle activation was assessed in multivariate mixed model analyses for repeated measures in each subject, leading to significant effects of disease status (control, SIS, RC tear) and performed task (rest, abduction, adduction) on *AH*.(Table 3) For disease status, *AH* was largest for patients with SIS, followed by controls and patients with RC tears, respectively. For performed tasks, *AH* was smallest during abduction, followed by adduction and rest. We found no significant association of muscle activations of DM, LD, PM or TM on *AH*.(Table 3)

Variable	Effect Size	95%-CI	p-value
Intercept	9.20	8.1 - 10.3	**<0.001**
Disease status			
SIS	1.81	0.52 - 3.10	**<0.01**
RC tear	-1.47	-2.83 - -0.10	**0.04**
Control	Ref.	NA	NA
Task			
ABduction	-2.29	-3.23 - 1.36	**<0.001**
ADduction	-1.40	-2.58 - -0.22	**0.02**
Rest	Ref.	NA	NA
Muscle Activation			
DM	-0.25	-2.82 - 2.32	0.85
LD	0.80	-1.89 - 3.50	0.56
PM	0.86	-1.57 - 3.30	0.48
TM	-0.35	-3.09 - 2.39	0.80

Table 3. Multivariate mixed model analyses with AH as dependent variable and disease status, performed task and the MVC-normalized muscle activations of DM, PM, LD and TM as independent variables.
Disease status and task had significant effects on *AH*. Patients with SIS had on average a larger *AH* than controls, whereas patients with RC tears had a smaller *AH*. Both isometric active abduction and adduction lead to a smaller *AH*. We found no significant effects of muscle activations on *AH*.

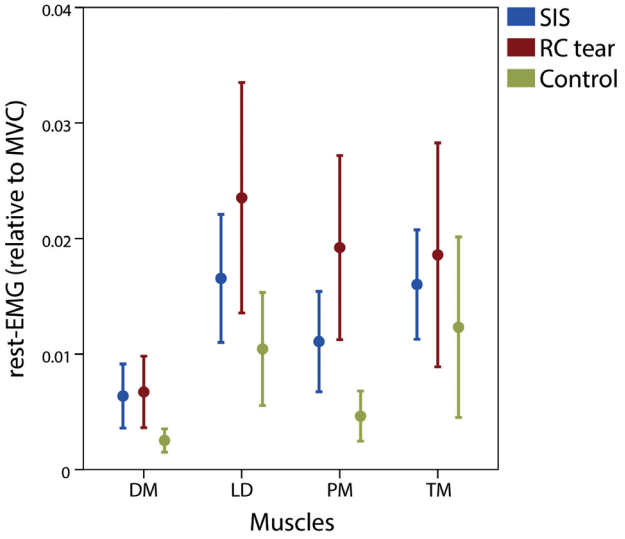


Figure 3. Muscle activations and 95%-CI during rest tasks for each muscle and subject groups, normalized to MVC EMGs.
Overall, rest activity was higher in the patient groups compared to controls.

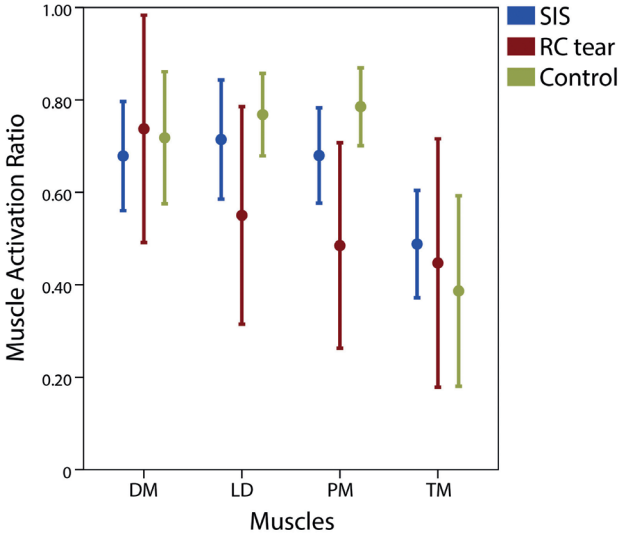


Figure 4. "Out-of-phase" muscle activation during abduction and adduction for all subject groups and all muscles, expressed in Activation Ratios (AR).
Lower *AR*'s indicate more pathologic activation, e.g. adductor co-activation during abduction.

4. Discussion and conclusions

This study reports on muscle activation and the distance between humerus and acromion at rest and during isometric abduction and adduction tasks in patients with RC tears, patients with SIS and controls. At rest, the acromiohumeral distance (*AH*) was narrowest in patients with RC tears. During active abduction, and to a lesser amount during adduction, cranial humerus translation was observed relative to rest in all subject groups, without statistically significant differences between the three groups. The rest activities of the Pectoralis and Latissimus Dorsi adductor muscles were larger for the patient groups. The EMG *Activation Ratios* of adductor muscles did not clearly indicate more pathologic activation (i.e. lower *Activation Ratios*) in patient groups compared to the controls, except for the Pectoralis and Latissimus Dorsi muscles in patients with RC tears, indicating a loss of muscle specific activation (i.e. adductor co-activation during arm abduction). We did not find an association between muscle (co-)activation and cranial humerus translation.

4.1 Acromiohumeral distance at rest

The smallest *AH* in rest we found in patients with RC tears, coincides with previous reports for RC deficit shoulders.[11-17, 36] This has phenomenon been related with shoulder pain, progression of RC tear size, multiple torn RC tendons and RC muscle degeneration in the literature.[11-17] Some studies suggest a small *AH* to be an indicator for chronic massive RC tears.[12, 13, 15, 26] For patients with SIS, the average *AH* was not smaller than observed in controls, in contrast to our hypothesis. Possibly, actual full-thickness RC tearing needs to be present before an evidently narrower subacromial space in rest ensues. In support of this, Keener and colleagues found differences in *AH* between 1) patients with asymptomatic tears and symptomatic tears and 2) between patients with full-thickness tears and massive posterosuperior (i.e. Supraspinatus and Infraspinatus) RC tears.[12] Another explanation is that there could be thickened RC tendons and bursa tissue in patients with SIS due to a subacromial inflammatory reaction, preventing the observation of an evidently smaller *AH* compared to controls despite potential RC deficiency. Subacromial filling may play an important role in determining the distance between acromion and humerus.

4.2 Cranial humerus translation during active tasks

Cranial humerus translation (*ΔAH*; additional narrowing of *AH* during active tasks compared to rest) was most evident during isometric abduction for all subject groups. Similarly, others found significant cranial translation at different elevation angles during active abduction in patients with SIS or RC tears.[11, 18, 21, 37] However, the currently applied set-up has the advantage that a standard elevation angle

and isometric tasks are applied for all subjects, improving the abilities to compare subjects.[33] It has been suggested that cranial translation during abduction is caused by Deltoid activation.[18, 19, 38] In patients with RC tears, there is suboptimal GH stabilization due to the torn RC[25] and increased Deltoid activation, compensatory to lost RC function.[23-25] In theory, this can cause *excessive* cranial translation of the humerus in patients, leading to (painful) compression of subacromial tissues.[12, 19, 25] However, we found no differences in the absolute amounts of translation in the three groups, covering a complete range of subjects from symptomatic patients with RC tears, to patients with SIS with an intact RC and asymptomatic controls. But relative to rest *AH*, narrowing was most prominent in patients with RC tears, suggesting the largest subacromial strains in this group during abduction.

Surprisingly, there was not only cranial translation during arm abduction tasks, but also during arm adduction in all groups. Although the caudally directed TM and LD are primarily active during adduction, this did not lead to an average net caudalisation of the humerus. It is plausible that only limited activity of the Deltoid with its large Physiological Cross Sectional Area (PCSA) relative to the TM and the LD leads to cranial translation even during adduction tasks.

4.3 Linking AH and adductor co-activation

Hence, we found group differences between *AH* measures at rest, but not with the absolute *ΔAH* measurements assessing cranial translation during tasks. This raises the question whether isometric active abduction and adduction radiographs have any additional value in clinical and scientific research. This all notwithstanding, e.g. 2.6mm humerus translation with a rest *AH* of 7.6mm as observed in patients with RC tears, i.e. a strain of 34%, might have more consequences than 2.1-2.3mm translation in patients with SIS or controls with their larger rest *AH*, i.e. strains of 20.7% and 23.6% respectively. A possible explanation for the similar amounts of *AH* narrowing in the three groups, despite the theoretical excessive narrowing in patients with RC tears, is adductor co-activation. Several simulation and EMG studies reported *ad*ductor activation during *ab*duction (adductor co-activation) in RC patients, in particular for caudally directed adductors as the TM and LD, supposedly limiting subacromial narrowing by pulling the humerus down.[24, 25, 30-32] We did find significantly higher adductor activation (relative to MCV) in rest in patients with RC tears compared to patients with SIS and controls. But this might partially be explained by somewhat lower maximum voluntary forces and subsequent MVC values in patients (data not shown). There also appeared to be relatively more adductor co-activation, expressed in lower *Activation Ratios* during abduction in patients with RC tears compared to patients with SIS (LD, PM, TM) and controls (LD, TM), but these differences were not statistically significant.

4.4 Adductor co-activation in patients with RC tears or SIS

In a previous study assessing controls and patients with RC tears with the same experimental set-up, we defined an *AR* of minimal 0.30 points lower compared to average control *AR*'s is indicative for pathologic co-activation, leading to cut-off values of 0.59 for the DM, 0.48 for PM, 0.49 for LD and 0.38 for TM for this set-up.[36] In the patients with RC tears of the current study, mean *AR's* of all adductors (PM, TM, LD) were around previously reported pathologic *AR* cut-off values.[32, 36] For patients with SIS, only *AR* of the TM was around the reported pathologic cut-off values.[32, 36] Although the RC is not torn in patients with SIS, tendinitis and partial tears can lead to impaired RC function and increased DM activation.[39] Potentially, this causes (excessive) upwardly directed forces on the humerus during abduction, which can be compensated by co-activation of only the TM in patients with SIS, where co-activation of several adductors is required to maintain glenohumeral stability in patients with RC tears. Hence, adductor co-activation might be an indicator for RC dysfunction in general, instead of an indicator for RC tears specifically.

4.5 Unexpected results in the control group

The results of *AH* and adductor co-activation for patients with SIS or RC tears were overall as hypothesized, but we found unexpected results for the relatively small control group. *AH* in rest was not largest in controls and some controls showed adductor co-activation, in contrast to controls in our previous study with the same set-up.[36] The current controls where older than in the previous study and their RC status was not assessed with imaging, in contrast to the patients' RC status. It is plausible that some controls had an asymptomatic (early stage) RC tear, with consequent adductor co-activation; the prevalence of asymptomatic RC tears has been reported between 4% and 80% and increases with aging.[36, 40-42] Furthermore, also adductor co-activation might be related with aging, although previous studies have not supported this.[32, 36] Future research on asymptomatic controls of various age groups with ultrasound or MRI evaluation is needed to gain more insight in adductor co-activation and its alleged association with RC dysfunction.

4.6 Linking abductor and adductor muscle activation with AH in patients and controls

In this study we were able to simultaneously record EMG and subacromial translation in patient groups and controls. Combining all EMG recordings and *AH* measurements in multivariate analyses, disease status and exerted task had significant effects on *AH*. Similar to the ANOVA results, patients with SIS had on average a larger *AH* and patients with RC tears a smaller *AH*, compared to controls. *AH* was smaller during abduction and, to a lesser extent, adduction. We found no significant effects of

muscle activations on *AH*. We may conclude that *AH* cannot simply be derived from the relative activation of the abductors and adductors, assuming an isometric linear relation between *AH* and EMG.[43] Assessing muscle forces, joint reaction forces and *AH* additionally requires e.g. individual anatomy, including tendon thickness, bursa thickness, (subacromial) tissue stiffness, PCSA values and force directions of all contributing muscles, which were obviously not available in this study. Alternatively, affecting adductor co-activation by administering a subacromial injection with anaesthetics as has been applied in patients with RC tears[31] could give more insight in the role of adductor co-activation with regard to subacromial narrowing.

4.7 Strengths and weaknesses

The strengths of this study include the comparison of three moderately large and well-defined groups, covering a broad range of rotator cuff (dys)function, the use of objective outcome measures (EMG, *AH* and *ΔAH*) and the use of a newly developed and validated set-up with a comprehensible measure for EMG-recordings (*AR*). Additionally, this is the first study assessing the association between specific *isometric* tasks, disease, muscle activation (EMG) and subacromial narrowing. There are also some weaknesses of our study that need to be taken into account when interpreting our results. Firstly, there was an age difference between patients with RC tears and the two other groups, due to the fact that impingement and RC tears have an age-dependent prevalence and the selected controls were relatively young. Nevertheless, it has been previously reported that adductor co-activation seems not to be age-dependent.[32, 36] Secondly, systematic measurement errors are common in AP shoulder radiographs. Patient positioning can greatly influence the projection of the subacromial space, with large projection and magnification errors leading to either over- or underestimating cranial translation of the humeral head.[28] But earlier studies report that *AH* measurements on AP radiographs are highly reproducible within and between investigators.[26, 28, 29] In our study the patient setup was such to minimize repositioning for the three tasks and all the measurements were consistently applied in all groups, reducing potential projection and magnification errors. Thirdly, the subacromial volume (3-dimensional) may be more appropriate to assess the subacromial space compared to the *AH* distance (2-dimensional). However, 3-dimensional volume measurements are elaborate, require 3-dimensional imaging and are not practical for clinical use, while it would only enlarge the resolution of the measured effects. Fourthly, although applying isometric arm tasks in a similar position for all subjects (without gravity affecting the abduction-adduction comparison) is a strong point, we did not investigate subacromial narrowing at higher elevation angles unlike others.[11, 37, 44] Lastly, the

referential 'rest' condition is not a fully relaxed position as the arm is rotated in external rotation, which might mask an actually larger humeral translation during abduction and adduction.

5. Conclusions

The results of our study show that in patients with RC tears, the subacromial space as measured on radiographs is generally narrower than in controls and patients with SIS and that all three subjects groups demonstrate similar absolute amounts of cranial humerus translation during active isometric abduction and adduction tasks. Muscle activation at rest was relatively high in the two RC disease groups and there appeared to be more adductor co-activation in patients (n.s.). We did not find an association between muscle activation of the Deltoid and main adductors and cranial humerus translation. Future studies assessing the relation between muscle activation and subacromial space should take into account e.g. muscle volume and degradation status to make more accurate estimations of muscle force, or use interventions such as nerve-blocks or a subacromial injection with anesthetics to realize changes in muscle activation within subjects and assess how this influences subacromial space.

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Pathologic Deltoid activation in rotator cuff tear patients - Normalization after cuff repair? -

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Abstract

Rotator cuff (RC) tears have a high prevalence, and RC repair surgery is frequently performed. Evaluation of Deltoid activation has been reported as an easy to measure proxy for RC functionality. Our goal was to test the success of RC repair in restoring muscle function, by assessing Deltoid activation with varying arm abduction moment loading tasks in controls and in RC tear patients before and 1 year after RC repair.

Averaged rectified electromyography recordings (rEMG) of the Deltoid during 2-second isometric arm abduction tasks were assessed in 22 controls and 33 patients before and after RC repair. Changes in Deltoid activation as a response to increased arm abduction moment loading (large vs. small moment), without changing task force magnitude, were expressed in: $R=(rEMG_{Large}-rEMG_{Small})/(rEMG_{Large}+rEMG_{Small})$, where R>0 indicates an increase in muscle activation with larger moment loading.

In controls, a significant increase in Deltoid activation was observed with large abduction moment loading: R=0.11 (95%-CI: 0.06-0.16). In patients, R was larger: 0.20 (95%-CI: 0.13-0.27) preoperatively and 0.16 (95%-CI: 0.09-0.22) postoperatively. Increased compensatory Deltoid activation was found in pre-operative RC tear patients. The post-operative decrease in compensatory Deltoid activation, although not significant, could indicate (partially) restored RC function in at least some patients.

Keywords: Electromyography; Rotator Cuff; Surgery; Rotator Cuff tear; Deltoid Muscle

1. Introduction

Rotator cuff (RC) tears have a high prevalence and are often diagnosed in patients with shoulder symptoms.[1, 2] RC repair surgery is a frequently performed surgical procedure for the treatment of symptomatic RC tears.[3] However, there are many uncertainties with respect to RC tears and their treatment: 54% of the population over 60 years have asymptomatic RC tears,[4] only around 50% progress to symptomatic tears within 2-3 years,[5, 6] treatment results are highly variable [3] and the functional status and recovery of the RC and other shoulder muscles after RC repair surgery has been scarcely investigated.[7]

Insight into shoulder muscle function is crucial to gain understanding of these heterogeneities and to assess whether RC repair restores shoulder muscle function. It is, however, hard to quantify the effect of cuff repair surgery on the abduction function of the RC. Assessing the activation of the Supraspinatus muscle and the superior portions of the Infraspinatus and the Subscapularis muscles requires invasive fine-wire electromyography (EMG). This hampers clinically applicable measurements and requires additional assumptions about the contributions of the active muscles to the abduction and adduction forces on the humerus. The latter is specifically complex in case of RC tears and (incomplete) surgical repair.

In a study assessing muscle functions in RC tear patients and healthy controls, Steenbrink and colleagues demonstrated a larger increase in Deltoid (DE) activation as a response to increased arm abduction moment loading in RC tear patients compared to controls.[8] They suggested that in patients, the Deltoid compensates for lost RC function, as has been reported by others as well.[9-14] As a compensator for lost RC function, assessing Deltoid activity is a potential proxy for easy indirect assessment of the functional status of the deeply positioned RC muscles. In the current study, we applied this previously introduced non-invasive experimental method to indirectly test RC function by assessing the activation of the Deltoid muscles.[8]

Our study goal was to quantify the contribution of the RC to arm abduction in controls and cuff tear patients before and after surgical cuff repair, by assessment of changes in Deltoid activation in response to variations in arm abduction moment loading. Our hypotheses were that 1) an increase in arm abduction moment loading leads to a relative increase in Deltoid activation in all subjects; 2) this relative increase will be larger in RC tear patients before surgery (compensatory Deltoid activation) compared to controls; 3) RC repair leads to a decrease in compensatory Deltoid activation compared to pre-operative measurements; and 4) RC repair leads to a decrease in compensatory Deltoid activation, more in the range of control subjects' measurements.

2. Materials and methods

2.1 Subjects

From March 2010 until April 2011, RC tear patients who were planned for surgical RC repair by one of two experienced shoulder surgeons of two participating hospitals (Medical Center Haaglanden, the Hague, the Netherlands; Leiden University Medical Center, Leiden, the Netherlands) were contacted for inclusion in the current study. Inclusion criteria were as follows: full-thickness Supraspinatus tear proven with magnetic resonance imaging (MRI arthrography) and one or more of the following criteria present, aside from a positive Neer impingement test, a positive Hawkins test and diffuse unilateral anterosuperior shoulder pain for >3 months: pain with arm abduction, retroflexion and/or internal rotation (e.g., closing the door, putting on jacket); pain with overhead activities; pain at night or incapable of lying on the shoulder; classic painful arc; positive Yocum test; positive full or empty can test. Patients were included for post-operative evaluation unless any other causes for shoulder symptoms than RC tears were identified during surgery. Additionally, RC status was evaluated 1 year after surgery using ultrasound. Inclusion criteria for healthy subjects (controls) were: between 20 and 50 years old (in order to limit the chance of including subjects with asymptomatic RC tears), no present shoulder complaints, and no history of medically treated shoulder complaints.

Exclusion criteria for all subjects were: insufficient Dutch language skills or no informed consent, presence of physical problems influencing muscle activation and arm mobility (other than RC tear in the patient group), any form of inflammatory arthritis of the shoulder, glenohumeral (GH) or symptomatic acromioclavicular osteoarthritis, a history of surgical interventions of the shoulder, clinical signs of cervical radiculopathy, GH instability, or frozen shoulder syndrome (<90° of passive abduction and external rotation). After usual care radiographs and MRI arthrography evaluation, patients were excluded in case of Subscapularis or Teres Minor tendon pathologies, calcific tendinitis, intra-articular or bony lesions (Hill Sachs, (old) fractures, tumors), labrum abnormalities, capsular or ligamentous tears/avulsions, superior labral tear from anterior to posterior (SLAP lesion), pulley lesion, Biceps tendinitis or tear, os acromiale, or cartilage lesions.

The patient group was clinically evaluated before surgery and 1 year after surgery, using the Western Ontario Rotator Cuff index (WORC)[15-17] and the Constant Score (CS).[18] RC status (re-tear yes/no) 1 year after surgery was assessed with ultrasound. All stages of this study are in compliance with the declaration of Helsinki, written informed consent was obtained from all participants, and the local medical ethics review board (METC, Leiden University Medical Center) approved the study.

2.2 Surgical procedure

RC repair surgery was performed by either one of two participating experienced orthopaedic shoulder surgeons using an all-arthroscopic (AA) or mini-open (MO) technique according to the surgeon's preference. There is no difference in clinical outcome and complication rate between the AA and MO procedures.[19] All patients were operated under general anesthesia in lateral decubitus position with the arm held in a 3-point shoulder distraction device. In both procedures, the edges of the tear are debrided, and the insertion site for the suture anchors on the major tubercle is prepared using a shaver. A suture bridge repair construct is applied, using 2-4 anchors depending on the size of the tear, to secure the tendons with a 5.5mm CorkScrew (Arthrex, Naples, Florida) in the medial row and a knotless 3.5mm Bio-PushLock anchor (Arthrex, Naples, Florida) in the lateral row. In case of a longitudinal extension of the tear, the margin convergence technique was applied first. After wound closure, a standard dressing is applied, and the arm is placed in a sling for 6 weeks.

There was a standard postoperative rehabilitation protocol for all patients, under supervision of a local physical therapist. Active exercises of the elbow, wrist, and hand were encouraged from the first day after surgery. The rehabilitation protocol consisted of active assisted abduction in the scapular plane limited to 70° and 0° of external rotation in the first 4-6 weeks, as tolerated. After this, active range of motion exercises were started. When the patient was pain free, isotonic strengthening exercises were initiated.

2.3 Experimental set-up

In order to compare EMG over muscles, subjects, and time, normalization with maximal activation increases the reliability of the measurement.[20] However, assessing maximum voluntary contraction (MVC) in pre- and post-intervention patients may not be reliable. An alternative is to normalize EMG over two isometric but antagonist tasks, resulting in an *Activation Ratio (AR)*.[21] This concept, applied for normalizing EMG of the Deltoid muscle under two contrasting arm loading conditions, stratified for force magnitude and force direction but at different locations at the humerus (i.e., different moment loading conditions), previously resulted in a Deltoid moment loading response which was shown to be sensitive for patients with RC lesions: patients with a proven RC lesion had a larger increase in Deltoid activation in response to increased arm abduction moment loading compared to healthy subjects.[8] We applied the same method to qualify the contribution of Supraspinatus during abduction for each subject, based on changes in the activation of the Deltoid (DE) as a response to increasing the external force moment arm of an abduction task force of constant magnitude. The applied set-up has also been applied and

validated in various other shoulder EMG studies.[21-25] Controls were evaluated once and RC patients were evaluated in two sessions: in the month before a planned RC repair procedure and in the second year after RC repair.

Subjects were seated with the affected (patients) or dominant (controls) arm fully suspended in a splint that was attached to a force sensor (AMTI-300, Advanced Mechanical Technology Inc., Wavertown, MA, USA). The two translational degrees of freedom perpendicular to the humerus were fixed, and the longitudinal translation and three rotational degrees of freedom were released.(Figure 1) The splint allowed for variation in point of force application (force sensor) alongside the humerus. In this way, external moment loading can be varied, without changing task force magnitude. Subjects were instructed to maintain the arm in a standardized position during the experiment: arm elevation of 60°, 30° of horizontal abduction and with the humerus 45° internally rotated, as applied in previous studies with this set-up.[8, 21-25] Arm position was visually controlled for by markings on the experimental set-up. The arm was fully supported for gravity in this specific condition, so subjects were able to maintain the arm this way without any effort.

Subjects performed isometric abduction tasks perpendicular to the longitudinal axis of the humerus. By applying forces onto the force sensor, subjects could control a visually displayed cursor (red dot) that had to be moved to randomly appearing targets on a computer screen. Each target represented a force vector of constant magnitude in one of seven equidistant abduction directions (15° apart), away from the midline or sagittal plane of the body and in the scapular plane, ranging from 0° (push arm straight up) to 90° (push arm sideward).(Figure 1) Subjects held the cursor within each target area for two seconds. Force magnitude was determined individually during each session and set at 10 N below the maximum level at which subjects could perform the 2-second tasks, as recommended previously, with a minimum of 10 N to secure sufficient signal over noise ratio.[26] After practice rounds, two task trials were performed: one trial with the point of force application at about 10 cm distally from the GH joint, i.e. the '*small* moment arm' condition, and one trial with the point of force application approximately 25 cm distally from the GH joint, i.e. the '*large* moment arm' condition. Note that within each session, the force magnitude was constant for the two trials. In order to prevent fatigue and other carry over effects, there was a minimum rest period of 15 seconds between the tasks and a period of 5 minutes between the two trials.

Activity of the main Deltoid muscle parts (anterior: DA; medial: DM; posterior: DP) were recorded with a bi-polar surface EMG system (DelSys Bagnoli-16, Boston, MA, USA; DE-2.1 single differential electrodes, inter-electrode distance 10mm, bandwidth 20–450 Hz). EMG electrodes were applied after palpation of the muscle bellies, while the subjects were positioned in the experimental set-up. The skin

was shaved where needed, scrubbed with skin preparation gel (SkinPure, Nihon Kohden, Tokyo, Japan) and cleansed using alcohol pads.

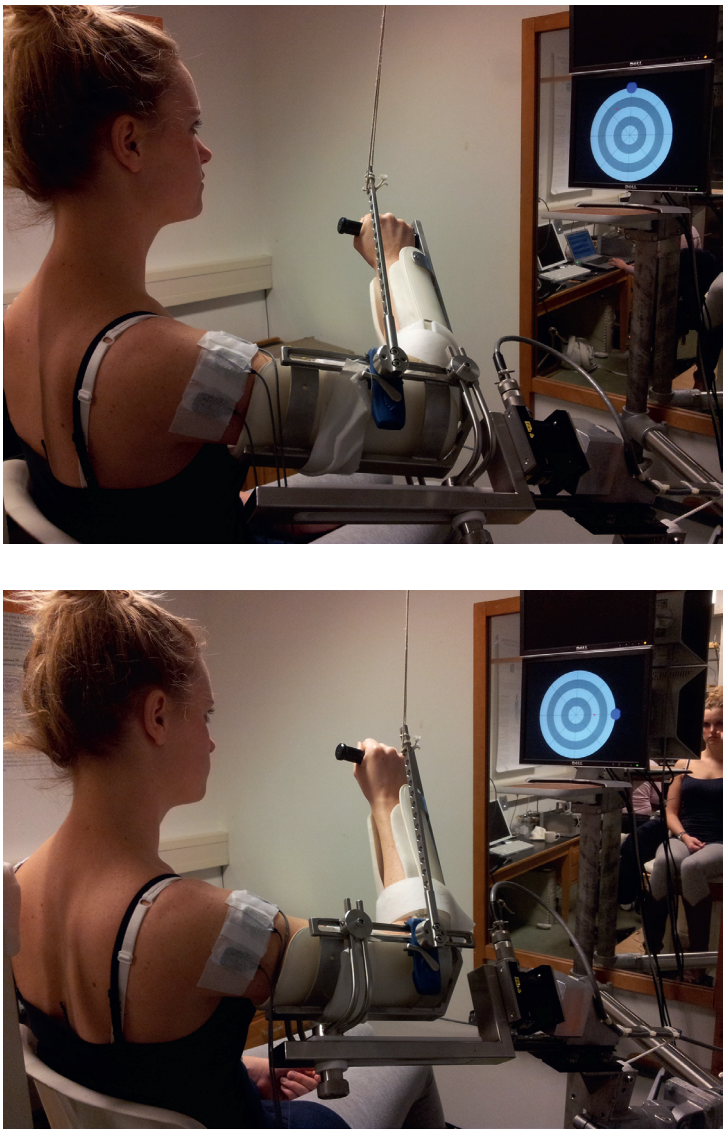


Figure 1. Set-up for isometric tasks with a small or large moment arm of an external task force. In the current study we applied abduction tasks.
Subjects had to move a cursor (red dot) to a target (blue dot) randomly representing each of 7 respectively applied equidistant abduction directions, 15 degrees apart, ranging from pressing arm straight up (0° degrees, Figure 1a), to pushing the arm sideward (90 degrees, Figure 1b).
The point of force application, i.e. where the force sensor is attached to the splint, can be varied in on order to realize large moments (Figure 1a) or small moments tasks (Figure 1b), while keeping the exerted task force constant.

2.4 Data processing

The simultaneously recorded 2-second force and EMG signals for each force task were analog-to-digitally converted (2000 Hz). After subtracting EMG rest activity, the EMG recordings were rectified and averaged for each force task resulting in 7 aEMG observations for each muscle during each trial. From these 7 observations, we obtained a single activation measure for each muscle part by averaging aEMGs, but because the different Deltoid muscle(part)s are not equally responsive for all seven force directions we averaged the aEMGs over only a selection of the force directions, depending on the involved Deltoid muscle part. In order to determine the most prominent activation directions of the muscle parts, we applied the previously reported *Principal Action* (*PA*) method.[22, 24, 25] This *PA* is the experimentally determined task direction in which the (EMG of the) muscle is most active. We selected the force task directions within +/- 45° around the *PA* of each individual muscle part and averaged the related aEMGs accordingly. The resultant rEMGs were normalized for each subject and muscle using a *Muscle Ratio* (R_{Muscle}, based on the *Activation Ratio*[8]), where muscle activation changes between the small and large moment trials ($rEMG_{Large} - rEMG_{Small}$) are divided by the sum of rEMGs of both trials ($rEMG_{Large} + rEMG_{Small}$) (Eq.1) :

$$R_{Muscle} = \frac{rEMG_{Large} - rEMG_{Small}}{rEMG_{Large} + rEMG_{Small}} \qquad -1 \leq R_{Muscle} \leq 1 \qquad \textbf{(1)}$$

This R_{Muscle} enables easy interpretation and inter- and intra-subject comparisons of EMG-recordings: R_{Muscle} values greater than 0 indicate a relative increase in the activation of the assessed muscle with larger abduction moment loading. Additionally, by assessing normalized relative Deltoid EMG in response to increased moment loading instead of e.g. raw EMG recordings or rEMG, inter individual variability from volume conductor effects, EMG equipment settings, subcutaneous fat tissue, and skin preparation are prevented. For the statistical analysis, it should be noted that the statistical distribution of R_{Muscle} around 0 (representing the zero-hypothesis (H_0): no difference between $rEMG_{Large}$ and $rEMG_{Small}$) is symmetrically distributed [-1, 1], but conservatively biased toward 0. This is in contrast to more general normalizations relative to either $rEMG_{Large}$ or $rEMG_{Small}$, which are asymmetrically distributed around H_0=0: [0,inf.], respectively and biased toward greater values than 0, which is more prone finding false-positive differences between $rEMG_{Large}$ and $rEMG_{Small}$.

2.5 Statistical analysis

Mean *Muscle Ratios* (R_{Muscle}), corresponding standard deviations (SD) and 95%-confidence intervals (95%-CI) for R_{DA}, R_{DM} and R_{DP} were calculated. Additionally, R_{DELT} was calculated for each subject and trial, by averaging the R_{Muscle}'s of all three Deltoid muscle parts. *Muscle Ratios* greater than zero (R_{Muscle}>0) indicate an increase in muscle activation with large moment arm loading. R_{Muscle}'s with 95%-CI's excluding 0 can be regarded as significant changes in muscle activation in response the changes in arm moment loading.

The average R_{Muscle} outcomes were compared between a) controls and pre-operative RC tear patients using unpaired t-tests, b) paired t-test for pre- and post-operative RC tear patients without a re-tear and c) unpaired t-tests comparing post-operative RC tear patients and controls. Additionally, mean patients' pre-operative and post-operative WORC and CS were assessed and compared using paired t-tests.

In order to take into account interactions between *Muscle Ratios* and to assess all recorded data in a single analysis, we performed a mixed model analysis with *Muscle Ratio* as dependent variable and as independent variables: disease status, pre-/post-surgery, muscle (DA, DP, DM) and the interaction term between muscle and disease status. A random effect per subject was included to take into account repeated measures in the patients.

IBM SPSS Statistics 20.0 for windows (IBM, Armonk, NY, USA) was used for all statistical analyses.

3. Results

3.1 Patients

Twenty-one controls and 33 patients with symptomatic RC tears were included in the study. Average age of the controls was 26 years (range, 20 to 43) and 10 (45.5%) were male. All were able to fulfill the experimental tasks without any shoulder symptoms. For patients, mean age was 61 years (range, 46 to 75) and 18 (54.5%) were male. Mean pre-op WORC was 51.9 points (SD=21.8) and mean pre-op CS was 56.8 (SD=15.5).

With regard to the pre-operative measurements in patients, 5 were unable to perform the experimental tasks due to pain and/or coordinative problems and in 3 patients there were technical problems. For the post-operative measurements, 4 patients had a re-tear, 3 patients did not want to participate, 2 could not be contacted anymore, 2 patients ultimately did not undergo surgery because of decreased symptoms and 1 patient could not perform the experimental tasks due to pain.

Of the 25 patients completing all tasks before surgery, 18 completed all tasks after successful surgery (no re-tear) with an average follow-up of 1.2 years (range: 1.0 – 1.6) and were available for paired pre-operative vs. post-operative analyses. In these patients, mean post-operative WORC improved with 25.5 points (95%-CI: 15.6 – 35.3) to 76.1 and mean CS with 29.2 points (95%-CI: 18.9 – 39.5) to 83.1.

3.2 rEMG during small and large abduction moment tasks in controls and cuff tear patients

The Deltoid muscle(part)s are not equally responsive for all seven force directions, and we averaged the aEMGs of each muscle part over only a selection of the force directions, depending on the muscle involved, based on the *Principal Action* (*PA*) of the Deltoid muscle(part)s. The average *PA* directions were PA_{DA}= -10° (SEM=1.6), PA_{DM}: 44° (SEM=2.6) and PA_{DP}: 83° (SEM=3.2). There were no significant differences between patient and control *PA*'s (data not shown). Consequently, the selection of force directions for DA ranged between -55° and 35° (i.e. 0°, 15° and 30° force tasks (3 force tasks/force directions)), for DM between -1° and 88° (i.e. 0°, 15, 30°, 45°, 60° and 75° force tasks (6 tasks)) and between 38° and 128° (i.e. 45°, 60°, 75°, 90° force tasks (4 tasks)) for DP. Resulting weighting factors for the average DELT calculation were 3 for DA, 6 for DM and 4 for DP.

In controls, there was an average relative increase in Deltoid muscle activation for large abduction moment loading, depicted in $R_{Muscle}>0$ for all separate muscle parts resulting in an average Deltoid (DELT) increase of R_{DELT}=0.11 (95%-CI: 0.06-0.16). This increase was significant for DA, DM and DELT, with corresponding 95%-CI's excluding 0.(Table 1) In pre-operative patients, there was a significant increase for DA, DM and DP with an average DELT increase of R_{DELT} = 0.17 (95%-CI: 0.10-0.23) for DELT. On average, patient Deltoid *Ratios* were larger than the Deltoid *Ratios* in the control group for all individual muscle parts in our data, where larger *Ratios* indicate a larger (compensatory) increase in Deltoid activation in a response to the increase in abduction moment loading.(Table 1, Fig. 2) When comparing data of controls and patients with both successful pre- and post-operative measurements available (n=18), pre-operative DA, DP and DELT *Ratios* were significantly larger in patients compared to controls.(Figure 2)

After surgery, the average Deltoid *Ratios* in patients decreased for DA, DM and DELT, but paired analyses indicated no statistically significant differences with pre-operative measurements.(Table 2, Fig. 2) Comparing the Deltoid *Ratios* of the post-operative group to controls now resulted in smaller mean differences between these two groups for DA, DM and DELT: 0.027 for DA (p=0.64), -0.03 for DM (p=0.55) and 0.04 (p=0.23) for DELT.(Figure 2) For DP, a significant difference between the two groups remained: 0.128 (p=0.04).

In mixed model analyses, effect size of disease status was -0.10 (95%-CI: 0.00 – 0.20, p=0.06). Estimated effect size of surgery on *Muscle Ratio* was negative (average decrease of R to healthy values) but not significant: -0.03 (95%-CI: -0.08 – 0.03, p=0.29). Effects of muscle [DA, DM, DP] and interaction terms were statistically not significant, although the effect of surgery on specifically the *Muscle Ratio* of the medial Deltoid appeared relevant: -0.09 (95%-CI: -0.21 – 0.03, p=0.13).

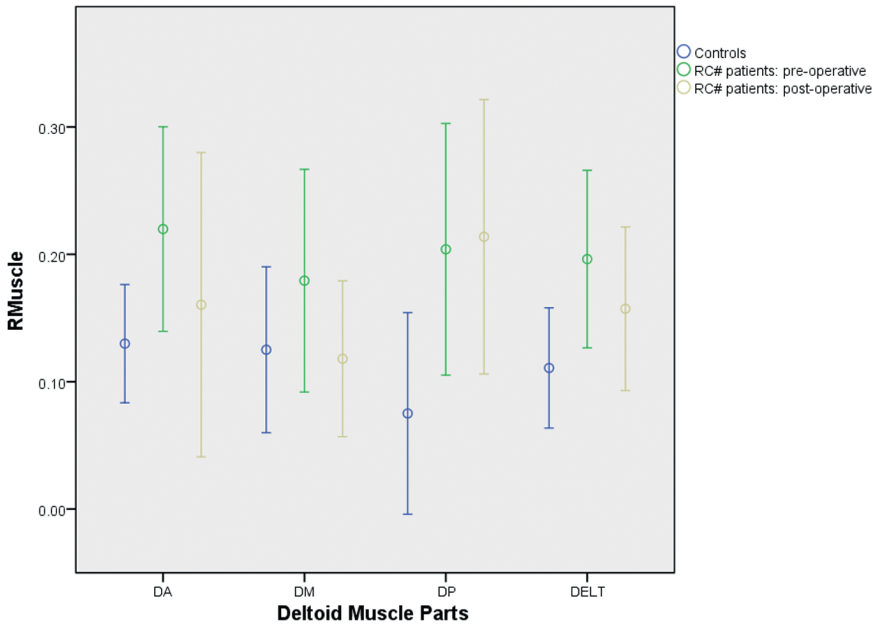


Figure 2. Relative increase in each of the Deltoid muscle parts with increased arm abduction moment loading, displayed in mean R_{Muscle}'s with 95%-CI's, for controls and rotator cuff (RC) tear patients before and one year after surgical RC repair.
Before surgery, RC tear patients have on average a larger increase in Deltoid activation with increased arm abduction loading compared to controls (statistically significant for DA, DP and DELT).
One year after RC repair, relative DA and DM activations in response to increased arm abduction were lower and no longer statistically significantly higher than control values. This could be due to a decrease in the need for DA and DM to compensate for lost RC function during abduction tasks as performed in a set-up with an abduction-anteflexion arm position.

	Controls (n=21)			Pts. pre-op (n=25)			Difference		
Muscle	*Mean*	*(SD)*	*95%-CI*	*Mean*	*(SD)*	*95%-CI*	*Mean*	*p-value*	*95%-CI*
Deltoid									
DA	0.13	0.10	0.08 - 0.18	0.20	0.16	0.14 - 0.27	0.07	0.08	-0.01 - 0.16
DM	0.13	0.14	0.06 - 0.19	0.15	0.19	0.07 - 0.22	0.02	0.68	-0.08 - 0.12
DP	0.08	0.17	-0.01 - 0.15	0.15	0.22	0.10 - 0.23	0.08	0.20	-0.04 - 0.20
DELT	0.11	0.10	0.06 - 0.16	0.16	0.16	0.10 - 0.23	0.05	0.21	-0.03 - 0.13

Table 1. Relative increase in Deltoid muscle parts with increased arm abduction moment loading, expressed in R_{Muscle}'s for healthy controls and rotator cuff tear patients planned for surgical cuff repair.

	Pts. pre-op (n=18)			Pts. post-op (n=18)			Paired diff. (n=18)		
Muscle	*Mean*	*(SD)*	*95%-CI*	*Mean*	*(SD)*	*95%-CI*	*Mean*	*p-value*	*95%-CI*
Deltoid									
DA	0.22	0.16	0.14 - 0.30	0.16	0.24	0.04 - 0.28	-0.06	0.25	-0.17 - 0.05
DM	0.18	0.18	0.09 - 0.27	0.12	0.12	0.06 - 0.18	-0.06	0.18	-0.15 - 0.03
DP	0.20	0.20	0.11 - 0.31	0.21	0.22	0.11 - 0.32	0.01	0.88	-0.13 - 0.15
DELT	0.20	0.14	0.13 - 0.27	0.16	0.13	0.09 - 0.22	-0.04	0.33	-0.12 - 0.04

Table 2. Relative increase in Deltoid muscle parts with increased arm abduction moment loading, expressed in R_{Muscle}'s for rotator cuff tear patients before and one year after surgical rotator cuff repair.

4. Discussion and conclusions

With the increasing debate on RC tears and its (surgical) treatment, we need objective outcome measures and more insight in the (biomechanical) principles of RC disease. In this study we aimed at discriminating pre- and postoperative RC tear patients from controls and illustrate the potential biomechanical effect and functional restoration of RC repair by quantifying the compensating activation of the Deltoid muscle parts in response to a changing arm abduction loading moments. The results show firstly, that an increase in arm abduction moment loading, with a constant task force magnitude, is accompanied by a significant increase in Deltoid muscle activation in both controls and RC tear patients. With respect to our first hypothesis, we may conclude that the Deltoid muscle parts are responsive for the experimental design of glenohumeral moment increase, as has been shown previously. Secondly, the increase in Deltoid activation was largest in pre-operative RC tear patients in our data, as depicted in significantly larger average *Muscle Ratios* compared to controls.(Figure 2) In mixed model analysis, there was a large and relevant effect of disease status on *Muscle Ratio*. This suggests that the Deltoid muscle compensates for lost RC function in RC tear patients with shoulder symptoms, as has been previously reported.[8-14] Thirdly, the main question of our study was whether RC repair surgery would indeed result in a partly or full normalization of cuff function, which would be observed in a normalization of the Deltoid moment response. For this question our experiment was not conclusive. One year after RC repair surgery, average *Muscle Ratios* of the Deltoid muscle parts in patients appeared to decrease toward the controls' values, which would suggest that RC repair surgery restores (at least part of) the function of the RC. However, the average decreases in Deltoid activation were relatively small and not statistically significant compared to pre-operative measurements. On the other hand, the post-operative *Muscle Ratios* of the Deltoid muscle parts did not significantly differ from the control group, where

pre-operative *Muscle Ratios* did significantly differ from the control group.(Figure 2) This could indicate that surgery did result in a normalization in some patients, at least for the anterior and medial Deltoid parts. Lastly, with regard to clinical results, the patient group significantly improved 1 year post surgery on both the WORC and Constant Score.

Symptoms of RC tears, most often Supraspinatus tears, are generally most apparent with active arm abduction. Nevertheless, specifically the Deltoid is regarded as an abduction moment generator.[27-29] Hence, increasing arm abduction moment loading (while keeping exerted force magnitude constant) predominantly leads to an increase in Deltoid activity.[8] This is supported by the results of our study. In case of RC tears, lost Supraspinatus function can be partially compensated by the Deltoid, specifically with regard to its function in generating arm abduction moments.[8-14, 30] Confirmatory to the latter is that previous studies have shown that knock-out of the Supraspinatus, e.g. by a tear or nerve-block, coincided with increased Deltoid activation.[8, 10, 13, 14, 30] In support of this, we found larger Deltoid *Muscle Ratios* in RC tear patients compared to controls.(Figure 2)

The posterior Deltoid differed most between patients and controls and did not seem to respond to surgery. The posterior Deltoid may potentially reflect the Infraspinatus abduction portion more than the Supraspinatus muscle. The quality of repair, differentiated over the Supraspinatus and the Infraspinatus may differ in e.g. improvement of Supraspinatus function, and may thus reflect in our results. Furthermore, it is plausible that the stabilizer function of the RC is sub-optimal in pre-operative and post-operative patients, requiring more activity of other muscles (including DP) for glenohumeral stabilization.

Hence, we found a significant increase in Deltoid activation for controls and pre-operative patients with increased abduction moment loading, significantly more Deltoid activation in pre-operative patients vs. controls, large effects of disease status and surgery on $R_{Muscle,}$ and an average increase in Deltoid activation with large moments that was substantially larger for all muscle parts in pre-operative patients and lower for post-operative patients. However, we were unable to find significant differences with most t-tests. The interpretation of these tests should take into account the relatively high value of R_{Muscle} for DP in patients (see above) and the large measurement SD's. Patient SD's could have been larger due to e.g. variations in the severity of symptoms and cuff tear size. Furthermore, two patients showed low pre-operative R_{Muscle}'s and high post-operative R_{Muscle}'s, contrary to the other subjects. There were no clinical or recording abnormalities for these patients, but when removing these outliers from the analyses, there was a significant decrease in DA activation after surgery (data not shown).

There are some limitations that need to be taken into account when interpreting our results. Firstly, Deltoid activation might be simply more increased in pre-operative patients due to pain. Pain was less 1 year after surgery and this could explain the decrease of the post-operative Deltoid (compensatory) activation. However, Suprascapular knock-out studies in healthy subjects and model simulation studies have also found increased Deltoid compensatory activation as a response to decreased RC functioning (e.g. nerve blocks), i.e. without pain playing a role.[8-10, 14] Secondly, there was a significant age difference between the controls and the patients, as we wanted to include controls with a low chance of asymptomatic cuff tears. Although age can influence EMG recordings, it is plausible that age has no influence on the *relative* increase in Deltoid activation with larger moment arms, such as expressed in *Muscle Ratios*. In linear regression analyses (data not shown) for pre-operative and control R_{Muscle} of each muscle and the combined DELT as dependent variables and with disease status and age as independent variables, we found no statistically significant or relevant age effects for DA, DM, DP and DELT. However, it is recommendable to further assess age effects in a single group (either with or without pathology) with a broader age range. Lastly, as stated in the introduction, RC tear patients form a heterogeneous population. We used strict eligibility inclusion criteria and extensive investigations, including MRI arthrograms, and patients were selected by 2 experienced orthopaedic surgeons specialized in shoulders in order to include a homogeneous patient group in whom it is highly likely the shoulder symptoms are caused by the observed RC tear. Currently, there are no better methods to select these patients or organize them in potential subgroups. Biomechanical testing of repair in these groups seems to be even more difficult.

Concluding, where the Deltoid seems to compensate for lost RC function in cuff tear patients, this increased (compensatory) Deltoid activation appears partially reduced 1 year after surgical RC cuff repair, which would suggest restoration of RC function. However, where we had sufficient power to discriminate pre-operative patients and controls and with the on average smaller and statistically insignificant differences between post-operative patients and controls, pre- vs. post-operative differences were relatively small and associated statistics were not conclusive. This might be due to e.g. limited accuracy or precision of the applied experimental set-up, variability in subjects, or it is the result of actually only limited or variable restoration of RC function after surgical repair. Further research is needed to investigate whether applying an alternative experimental set-up, e.g. with the arm alongside the body and abduction tasks in a single direction, leads to more accuracy, better responsiveness and similar results.

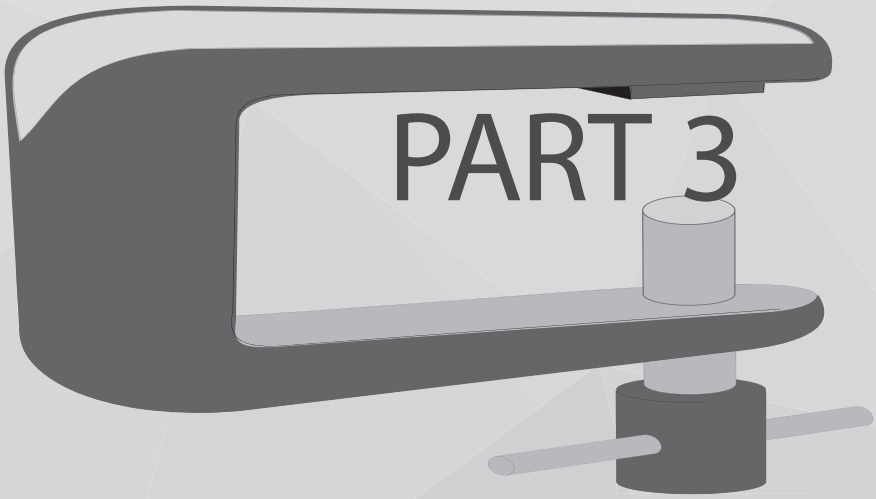
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PART 3

Future implications and general discussion





CHAPTER 12

Study protocol Subacromial Impingement Syndrome: The Identification of pathophysiologic Mechanisms (SISTIM)

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Abstract

Background: The Subacromial Impingement Syndrome (SIS) is the most common diagnosed disorder of the shoulder in primary health care, but its etiology is unclear. Conservative treatment regimes focus at reduction of subacromial inflammatory reactions or pathologic scapulohumeral motion patterns (*intrinsic* etiology). Long-lasting symptoms are often treated with surgery, which is focused at enlarging the subacromial space by resection of the anterior part of the acromion (based on *extrinsic* etiology). Despite that acromionplasty is in the top-10 of orthopaedic surgical procedures, there is no consensus on its indications and reported results are variable (successful in 48-90%).

We hypothesize that the etiology of SIS, i.e. an increase in subacromial pressure or decrease of subacromial space, is multi-factorial. SIS can be the consequence of pathologic scapulohumeral motion patterns leading to humerus cranialisation, anatomical variations of the scapula and the humerus (e.g. hooked acromion), a subacromial inflammatory reaction (e.g. due to overuse or micro-trauma), or adjoining pathology (e.g. osteoarthritis in the acromion-clavicular-joint with subacromial osteophytes).

We believe patients should be treated according to their predominant etiological mechanism(s). In this study we present a study protocol that is developed to identify and discriminate etiological mechanisms occurring in SIS patients, in order to develop tailored diagnostic and therapeutic strategies.

Methods/design: In this cross-sectional descriptive study, applied clinical and experimental methods to identify intrinsic and extrinsic etiologic mechanisms comprise: MRI-arthrography (eligibility criteria, cuff status, 3D-segmented bony contours); 3D-motion tracking (scapulohumeral rhythm, arm range of motion, dynamic subacromial volume assessment by combining the 3D bony contours and 3D-kinematics); EMG (adductor co-activation) and dynamometry instrumented shoulder radiographs during arm tasks (force and muscle activation controlled acromiohumeral translation assessments); Clinical phenotyping (Constant Score, DASH, WORC, and SF-36 scores).

Discussion: By relating anatomic properties, kinematics and muscle dynamics to subacromial volume, we expect to identify one or more predominant pathophysiological mechanisms in individual SIS patients. These differences in underlying mechanisms are a reflection of the variations in symptoms, clinical scores and outcomes reported in literature. More insight in these mechanisms is necessary in order to optimize future diagnostic and treatment strategies for patients with SIS symptoms.

1. Background

1.1 Introduction

The Subacromial Impingement Syndrome (SIS) can be defined as symptomatic irritation of the rotator cuff and subacromial bursa in the limited subacromial space. Clinical characteristics are pain with arm abduction (painful arc), decreased active range of motion (RoM) and loss of arm force and function.[1-5] It is the most frequently diagnosed shoulder disorder in primary health care, accounting for 44-65% of all shoulder complaints.[3, 6] Symptoms can persist for months or years and the majority of patients are between 40 and 50 years old. Consequently, SIS has a significant socioeconomic impact.[7]

Despite its reported prevalence, the diagnostic criteria and etiology of SIS are debatable. Two main etiologic theories have been described. Neer's widely accepted impingement theory focuses on the *extrinsic* mechanism: symptoms result from compressive forces on the rotator cuff, caused by biomechanical or structural anatomic (bony) abnormalities.[8, 9] The mechanisms leading to this assumed compression remain unclear. Scapula dyskinesia, causing relative cranial translation of the humerus, has been reported.[6, 10-13] Other studies describe a correlation between SIS and acromial shape (hooked acromion, Bigliani classification[14] type II or III).[4, 15-18] Presumably, this hooked acromion is a pre-existing anatomic variation, or a traction spur on the coracoacromial ligament caused by repetitive cranially directed translations of the humerus or by tendinopathy. Others conclude there is no relation between acromial shape and SIS, or underline the difficulties in using acromial shape as an assessment tool.[16, 19, 20] The majority of partial rotator cuff tears, commonly referred to as a consequence or entity of SIS, are often either intratendinous or at the articular side of the rotator cuff and not at the bursal side where they would be expected if the rotator cuff 'impinges' against a hooked acromion.[21] Despite these unclarities, the extrinsic mechanism forms the rationale for one of the most frequently performed orthopaedic surgical procedures: acromionplasty. The second theory is based on a degenerative *intrinsic* mechanism: SIS can be caused by ischemia at the watershed zone of the Supraspinatus tendon. This is enhanced by micro traumata or overuse, tensile overload on degenerating rotator cuff tendons, a subacromial inflammatory reaction, or insufficient cuff function leading to an imbalance between glenohumeral mobility and joint stability, with consequent glenohumeral destabilization or altered arm-shoulder kinematics.[22-29] Thirdly, SIS can be the consequence of adjoining pathologies or joint hyperlaxity. Furthermore, less classic forms of shoulder impingement, e.g. internal impingement and coracoid impingement have been described.

Treatment of SIS symptoms generally starts with conservative methods, including arm rest or physical therapy, non-steroidal anti-inflammatory drugs (NSAIDs) and subacromial corticosteroids injections. Conservative therapy is successful in 42% (Bigliani type III) to 91% (Bigliani type I).[30, 31] When conservative treatments fail, the classic surgical treatment of SIS symptoms is an acromionplasty as described by Neer.[8, 9] Variable and often mediocre results of this frequently applied procedure have been reported, with success rates ranging from 48 to 90%.[32-36] However, acromionplasty doesn't affect continuing degeneration of the rotator cuff[37], and subacromial spur recurrence has been reported following acromionplasty.[21, 38, 39] Henkus et al. reported comparable results for acromionplasty and bursectomy in patients with SIS.[40] This is in concordance with other studies that also report clinical improvements in SIS-patients without changing the coracoacromial shape.[31, 40-44]
Although SIS symptoms have been typically assumed to be the result of rotator cuff injury, the subacromial space is a complex anatomical environment, containing several structures that can be a source of pain. Even several pathologies that have a similar patients' history, pain patterns and findings with physical examination, can be (mistakenly) diagnosed as SIS.[45] In a recent study at our institution, 14 of 80 patients (17.5%) clinically diagnosed with SIS, had to be excluded following MRI arthrography because of alternative shoulder pathology.[40]
Concluding, the ongoing debate on the etiology of SIS, its varying clinical presentations, the diagnostic difficulties and the highly variable treatment outcomes of SIS suggest there might be multiple pathophysiologic mechanisms leading to complaints clinically diagnosed as SIS that need specific approaches in clinical practice. We present a study protocol that is developed to identify and discriminate etiological mechanisms in SIS patients.

1.2 Hypothesis

The extrinsic pathophysiologic mechanism is only valid for a subgroup of SIS patients, and consequently acromionplasty is the wrong treatment for at least a part of the patients suffering from SIS symptoms. The complaints observed in SIS are presumably a compilation of symptoms that originate from different shoulder pathologies and etiologic mechanisms. It is our challenge to discriminate these intrinsic and/or extrinsic underlying etiologies.

We developed a theoretical framework for the etiology of impingement ("a misbalance between subacromial volume and the space needed for subacromial structures") based on 4 distinct proposed mechanisms (Figure 1):

1) A dynamically reduced subacromial space due to a pathologic pattern of arm-shoulder movements (e.g. scapular dyskinesia), resulting in relative cranialisation of the humerus with respect to the scapula/acromion.
2) A more statically reduced subacromial space, due to:
    a. structural anatomic variations (e.g. a hooked acromion), eventually in combination with altered arm-scapula motion patterns;
    b. a subacromial inflammatory reaction (e.g. caused by micro-trauma or overuse) causing subacromial oedema, fibrosis and tendinosis;
    c. Encroachment of subacromial tissues by an adjoining pathology or structures other than the acromion (e.g. acromioclavicular (AC)-joint osteoarthritis and subacromial osteophytes, calcific tendinitis, and coracoid impingement).

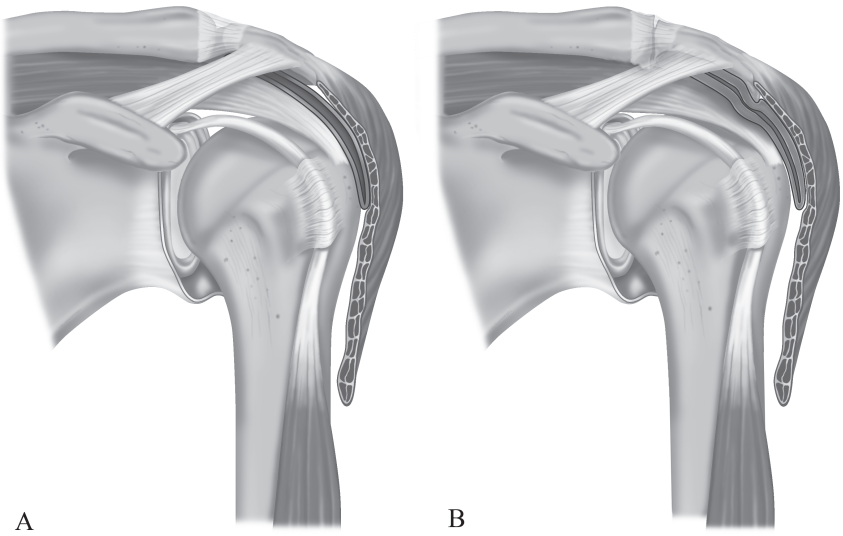


Figure 1 A) Schematic anatomy of a healthy glenohumeral joint and subacromial space. B) Schematic anatomy of a shoulder joint with the presence of several etiologic mechanisms for Subacromial Impingement Syndrome.
In theory, impingement ("a misbalance between acromial space and the space needed for subacromial structures") can be caused by 1) A dynamically reduced subacromial space due to a pathologic pattern of arm-shoulder movements (e.g. scapular dyskinesia), resulting in relative cranialisation of the humerus with respect to the scapula/acromion; or 2) A more statically reduced subacromial space, due to 2a) Structural anatomic variations (e.g. a hooked acromion), eventually in combination with altered arm-scapula motion patterns; 2b) A subacromial inflammatory reaction (e.g. caused by micro-trauma or overuse) causing subacromial oedema, fibrosis and tendinosis; 2c) Encroachment of subacromial tissues by an adjoining pathology or structures other than the acromion (e.g. acromioclavicular (AC)-joint osteoarthritis and subacromial osteophytes, calcific tendinitis, and coracoid impingement).

In the presented study protocol, factors associated with these SIS mechanisms will be analysed in patients clinically diagnosed with SIS. As a result, patients with SIS symptoms will be categorised in ”dynamic” and “static” etiologic subgroups. These subgroups might require tailored diagnostics and treatment strategies.
Because subacromial impingement syndrome is a clinical diagnosis, possible other causes of shoulder pain and SIS symptoms (e.g. early stage frozen shoulder, calcific tendinitis, slap lesions, rotator cuff tears, etc.) are identified and, if eligible, analysed separately in distinct research projects (trial registry numbers: NTR1545 and NTR2282).

1.3 Study goals

Primary goal:
Identification and classification of distinct pathophysiological mechanisms for symptoms clinically diagnosed as SIS into identifiable subgroups of patients as categorized above, in order to design tailored diagnostics and treatment flowcharts from experimental concepts.

Secondary goals:
A set of experimental and diagnostic tools is combined to identify structural and biomechanical etiological factors in patients with SIS symptoms, which will be related to clinical and functional status:

A. Presence and severity of pathologies in the subacromial space with MRI arthrography, e.g. (partial) cuff tears, tendinosis, fibrosis or a subacromial inflammatory reaction, and assessment of cuff degradation status.
B. Acromial shape classification and 3D shape parameters of the humerus, scapula and subacromial space volume, using conventional radiographs and segmented MRI-arthrograms.
C. Quantification of cranialisation of the humerus with respect to the scapula at rest and during active arm abduction and adduction tasks with simultaneously acquired shoulder radiographs and Electromyography (EMG) recordings (see D).
D. Measurements of the activation of arm adductors during arm abduction tasks and assessment of the presence of arm adductor co-activation (*Activation Ratio*).
E. Analyses of 3D-kinematics (arm range of motion and scapulohumeral rhythm) of the affected SIS shoulder compared to the unaffected shoulder and eventual etiologic SIS subgroups, with the use of 3D motion registration.
F. Changes in reconstructed subacromial volume and acromiohumeral distance during arm abduction by combining the recorded 3D-kinematics with the MRI-segmented 3D bony shapes.

G. The effect of a subacromial infiltration with lidocaine on arm range of motion, scapulohumeral and -thoracic rhythm with arm abduction, reconstructed subacromial volume and muscle activation patterns, including adductor co-activation.
H. Biomechanical analyses of structural or coordinative muscular imbalance by means of model simulation (Delft Shoulder and Elbow Model) with recorded 3D-kinematics as input.
I. Clinical phenotyping, using validated clinical scores and questionnaires.
J. Identification of alternative diagnoses that may cause complaints clinically diagnosed as SIS, using MRI and radiographs (e.g. acromioclavicular-osteoarthritis and subacromial osteophytes, calcific tendinitis, SLAP lesion or coracoid impingement).

2. Methods/design

2.1 Study design

In this multicenter observational cohort study, patients clinically diagnosed with subacromial impingement syndrome in either one of 3 participating hospitals (Leiden University Medical Center (LUMC), the Medical Center Haaglanden (MCH), Rijnland Hospital Leiderdorp) will be included for analyses at the LUMC Laboratory for Kinematics and Neuromechanics.
The Medical Ethics Committee of the LUMC approved all stages of the study. Written informed consent will be obtained from all patients.

2.2 Study population

2.2.1 Selection of participants

Patients will be recruited by 3 orthopaedic surgeons involved in the 3 participating hospitals. Patients will be selected if one or more of the following usual care criteria are present, next to a positive Neer impingement test (lidocaine) and a positive Hawkins test:

Patients' history:
- diffuse unilateral shoulder pain for > 3 months;
- pain during activities with abduction, retroflexion and/or internal rotation (e.g. closing the door, putting on jacket, overhead activities);
- pain at night or incapable of lying on the shoulder.

Physical examination:

- positive Yocum test;
- painful arc;
- diffuse pain at palpation of the greater tuberosity;
- disturbed scapulohumeral rhythm;
- no signs of pathologies or symptoms on the controlateral shoulder;
- capable of 90 degrees of passive abduction and 90 degrees of external rotation.

After the first and clinical inclusion round, symptoms of eligible patients are further investigated with the use of standard shoulder radiographs (anteroposterior in both external and internal rotation and Y scapular view (scapular outlet view)) and an MRI arthrogram of the shoulder. The MRI arthrograms are evaluated at the local hospital for clinical purposes and additionally evaluated by one of two participating musculoskeletal radiologists at LUMC for eligibility criteria and assessment other factors, including acromion classification and rotator cuff status.

Patients are excluded if one of the following characteristics is found with the visit to the outpatient clinic, standard shoulder radiographs or MRI arthrography:

- Age below 35 years or above 60 years;
- Restrictions in passive movements of the glenohumeral joint (adhesive capsulitis);
- History of fracture or dislocation of the shoulder, history of surgery around the shoulder;
- Co-morbidities on the affected shoulder (including fractures, benign or malignant tumors, labrum abnormalities, Hill Sachs lesion, capsular or ligamentous abnormalities, glenohumeral instability, glenohumeral movement restriction, glenohumeral or symptomatic acromioclavicular osteoarthritis, rheumatic disorder, Biceps muscle tendinitis, complete (full thickness) rotator cuff rupture, cervical radiculopathy, PASTA lesion, Pulley lesion, calcific tendinitis >3mm, or neurological deficits);
- Symptoms on the controlateral shoulder;
- No informed consent.

Patients with either rotator cuff tears or calcific tendinitis are included, if eligible, in separate research projects.

2.2.2 Sample Size

A combination of techniques will be applied to classify SIS-patients into pathophysiologic subgroups, most of which are newly developed (section 2.3). The acromiohumeral distance (AH) is a recognized parameter related to rotator cuff disease and based on literature it has rather wide inter-individual variations. Therefore, sample size calculation will be based on this parameter.
In a study of Gruber et al., AH values of 9.4 (SD=3.4) were observed in subjects without diagnosed cuff pathology. A subacromial space narrower than 6mm on radiographs is considered pathologic and strongly indicative for Supraspinatus tendon rupture.[46]
The unpaired t-test was used to determine the sample size with a difference of AH of 3.4mm between groups assumed as clinically relevant, comparing AH during abduction task radiographs in patients where humerus cranialisation plays a key-role compared to AH in other subgroups of SIS patients.

Based on the standardized difference: 3.4mm/3.4mm = 1.0, a required power of 80 % and a p-value of 0.05 for significance, the Altman's Nomogram resulted in 30 shoulders/patients per group.

In our hypothesis, we defined 4 etiological mechanisms. Based on clinical experience and literature, we assume around 30% type III acromion responsible for complaints of SIS,[16] 20-30% of the SIS symptoms are caused by humerus cranialisation and pathologic motion patterns,[47] 15-20% by subacromial inflammatory processes without subacromial narrowing and 5-10% by other impinging structures than the acromion, leaving around 10-30% for a group in which SIS symptoms seem to be caused by two or more hypothesized etiologic mechanisms. With 30 patients needed in the humerus cranialisation subgroup (i.e. 30 % of the patients), this leads to a total group size of 100 patients diagnosed with SIS based on patient history, physical examination, radiographs and MRI arthrography.
Additionally, we expect that around at least 25 patients will be diagnosed with another diagnosis than SIS after MRI arthrography and radiographs.[40, 48] These patients cannot be included in the SISTIM study but selected patients will be analysed separately in distinct research projects, if eligible (trial registry numbers: NTR1545 and NTR2282).

2.3 Outcome measures

The included patients with SIS symptoms will be subjected to several diagnostic and experimental tests at the LUMC department of Radiology (standard and force task radiographs with EMG, MRI arthrography) and the Laboratory for Kinematics

and Neuromechanics (shoulder kinematics, EMG). The set of measurements is described below and outcome parameters are defined, referring to the mechanisms as summarized in paragraph 1.2 and the primary and secondary study goals (A to J) in paragraph 1.3.

2.3.1 Basic MRI outcomes (Study goals A and J)

For the purpose of assessing eligiblity criteria and alternative causes of impingement symptoms, an MRI arthrography is acquired in each patient. MRI's are reviewed by one of two participating musculoskeletal radiologists at LUMC for inclusion and exclusion criteria and standard clinical evaluation. Additionally, the MRI scans will be used to identify potential anatomic/structural and biomechanical causes for SIS symptoms and to assess rotator cuff status (e.g. muscle volume, presence of tendinosis/tendinitis, intratendinous, bursal or articular side partial tendon tear, Goutallier score for muscle degeneration, signs of (micro)instability).[49, 50]

Main outcome parameters: inclusion/exclusion of patients, alternative diagnoses leading to SIS symptoms, rotator cuff status, signs of anatomical, structural and biomechanical causes for SIS symptoms.

2.3.2 2D Radiographical analyses and EMG (B, C, D, J)

Standard anteroposterior shoulder radiographs enable classification of the acromion shape. Patients' acromion Bigliani classification will be assessed: type I (flat), II (curved) or III (hooked).[14] We expect an incidence of 30% hooked acromions (type III) in SIS-patients.[15]

Increased subacromial narrowing during arm abduction has been reported in patients with rotator cuff degradation as a consequence of increased Deltoid muscle activation.[27, 51-54] In order to observe and study this potential etiological mechanism, radiographs will be acquired in rest position and during EMG-recorded isometric arm abduction and adduction moment tasks of equal force magnitude, using a set-up with a force sensor and visual feedback.(Figure 2) We will quantify the subacromial space using the acromiohumeral distance measure (AH), upward migration index (UMI; similar to AH, but corrected for image magnification and patients bony morphological aspects)[55] and spinohumeral center method (SHC).[56]

Co-activation of medio-caudally directed *ad*ductors during active arm *ab*duction has been reported to reduce this humerus cranialisation and consequent pain in rotator cuff patients.[5, 51, 57-60] Therefore, muscle activation will be controlled for during the three tasks by simultaneous EMG recording with bi-polar surface EMG of the main arm abductor (Deltoid) and adductors (Latissimus Dorsi, Teres Major, Pectoralis Major).

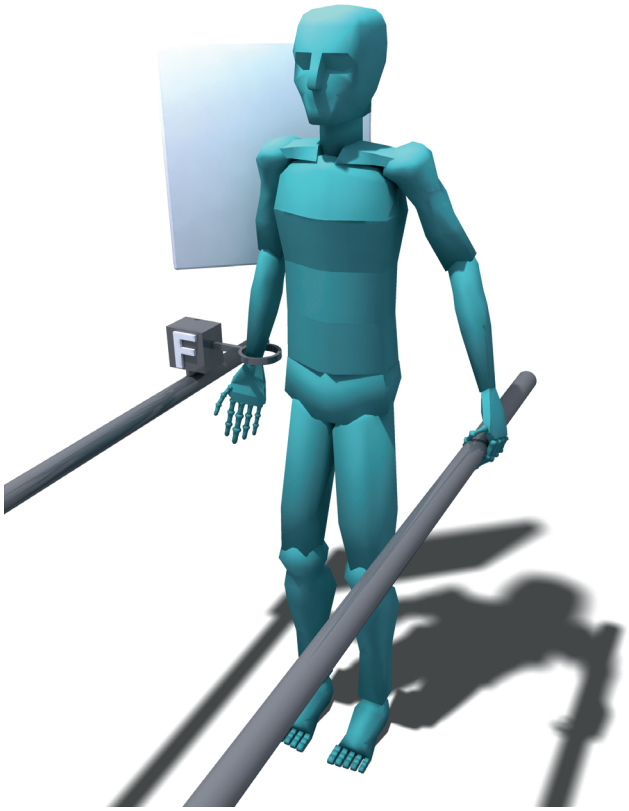


Figure 2. Set-up for EMG-recorded isometric abduction and adduction force tasks.
Subjects are positioned in front of the radiographic plate, in standing position with the concerning arm in external rotation at his/her side (i.e. hand in frontal plane), enabling the use of this set-up during concomitant acquirement of standard shoulder radiographs. The arm is attached to a 1-dimensional force transducer at the wrist, enabling subject specific force tasks, visual feedback, and equal task force magnitude for abduction and adduction tasks.

The relative activity of the glenohumeral abductors and adductors will be quantified using the "*Activation Ratio*".[58-60] The *Activation Ratio* (AR_{muscle}) of each muscle is determined according to its specific primary function. Muscle activation is either 'in-phase' (A^{IP}) or 'out-of-phase' (A^{OP}) with respect to its primary moment arm. For example, activation of the medial part of the Deltoid (A_{DM}) is defined as 'in phase' during active arm abduction tasks and as 'out-of-phase' during arm adduction tasks. Correspondingly, two average EMG levels are determined for each muscle for 'in phase' and out-of-phase' activation with respect to the isometric adduction and abduction moment tasks. Based on these data, subject specific *Activation Ratios* can be calculated for each muscle (AR_{muscle}), Eq 1:

$$AR_{muscle} = \frac{A^{IP}_{muscle} - A^{OP}_{muscle}}{A^{IP}_{muscle} + A^{OP}_{muscle}} \qquad \left[-1 \leq AR_{muscle} \leq 1\right] \qquad \textbf{(1)}$$

Consequently, *AR*'s of muscles in healthy subjects are positive and close to 1. In subjects with co-activation of arm adductors during abduction tasks, as has been described for the Latissimus Dorsi and the Teres Major in cuff tear patients, *AR*'s of arm adductor muscles are closer to 0 or even negative. Therefore, we expect to find low adductor *Activation Ratios* in at least a subgroup of SIS patients, in response to reduced *AH*.

Main outcome parameters: Bigliani acromion classification; Acromiohumeral distance (*AH*) in rest and during abduction and adduction tasks; muscle-specific EMG *Activation Ratios* ($AR_{Deltoid}$, $AR_{lattisimus\ dorsi}$, $AR_{Teres\ mj}$, $A_{Pectoralis\ mj}$) for quantification of (adductor) co-activation.

2.3.3 3D Radiological analyses (B, F)

Aside from clinical purposes and evaluating inclusion and exclusion criteria, MRI-arthrograms are also acquired to obtain 3D shape parameters of the humerus, scapula and subacromial space with the use of MRI segmentation techniques (Amira 5.3, Visage Imaging Inc., San Diego, CA, USA).

Main outcome parameters: 3D shape parameters for humerus, scapula and subacromial space.

2.3.4 3D Kinematics and changes in subacromial volume (E, F, G)

Range of Motion (RoM) and 3D motions of forearm, humerus and scapula with respect to the thorax will be recorded by means of an electromagnetic tracking system: 'Flock of Birds' (FoB, Ascension Technology Corp, Burlington, VT, USA) and custom made computer software (FOBVis, Clinical Graphics, Delft, the Netherlands). The FoB obtains 3D kinematical data using sensors on thorax, scapula, humerus, forearm and thorax. After palpatory identifying three dimensional positions of standard bony landmarks of the arm, shoulder and thorax with respect to the sensors for each patient, local bone coordinate systems are created, based on the subject's individual anatomy. The glenohumeral rotation center is estimated from the position of five scapular bony landmarks using linear regression.[61] The RoM of the following movements is measured: anteflexion, retroflexion, abduction in frontal plane, internal rotation in 0 and 90 degrees of arm abduction and external rotation in 0 and 90 degrees of arm abduction.

3D kinematics and MRI bony segmentation (3D shape parameters of scapula and humerus) will be combined in custom made computer software (Articulus, Clinical Graphics, Delft, the Netherlands) to reconstruct the subacromial space volume and *AH* during recorded humerus elevations, allowing dynamic measurements of patient-specific subacromial space characteristics.[5, 62, 63]
Additionally, the effect of a subacromial lidocaine injection on RoM, scapulohumeral rhythm, reconstructed *AH* and subacromial volume of the affected shoulder will be analysed.

Main outcome parameters: Passive and active RoM during standardized arm motions (with and without subacromial anaesthetics) of both arms; Scapulohumeral rhythm of affected and healthy arm; Reconstructed changes in *AH* and subacromial volume during dynamic arm abduction (combining MRI-based shape parameters and 3D RoM measurements).

2.3.5 EMG Muscle activation patterns (C, D, G)
We will analyse muscle activation patterns as measured by EMG recordings of 10 muscles around the shoulder, based on *Activation Ratio*[58, 59] and *Principal Action* parameters.[22, 51] Measurements will be performed before and after a subacromial infiltration of lidocaine (5 ml, 10 mg/ml), to study potential relations between pain during arm abduction and adductor muscle co-activation and arm-scapula kinematics, respectively.
Subjects are seated with the affected arm in a splint with the upper arm in 45° of internal rotation and the elbow in 90° of flexion. The humerus is positioned in 60° of forward elevation and in 30° of horizontal abduction.(Figure 3) The splint is attached to a 3D force transducer which is mounted on a sled so that it can move freely in a direction parallel to the humeral longitudinal axis. The arm is fully supported in order to compensate for gravity. Axial rotation of the humerus is mechanically not restricted to prevent the subjects from generating supplementary moments. In this way, patients can only exert forces perpendicular to the longitudinal axis of the humerus.
The subjects are asked to exert a maximal voluntary force (MVF) in 4 equidistant directions with a maximum of 50 N and maintain this force for 2 seconds using custom made visual feedback software (Matlab, The MathWorks Inc, Natick, MA, USA). The exerted force and force targets are visualized on a display, expressed in cursor that has to be moved to consecutive targets on a wheel in which the spokes denote force directions and the rim denotes the desired force magnitude.(Figure 3)
Subjects are subsequently asked to exert 75% of the lowest MVF value onto the force transducer for 2 seconds in each of 24 equidistant directions that are indicated

on the display. The same routine of 24 measurements will be performed 30 minutes after a subacromial injection with lidocaine. Muscle activations for the 10 muscles around the shoulder are recorded in each of the 24 directions during the 2 sets of measurements.

The direction of maximum activity or *Principal Action* (*PA*) for each muscle is determined [22, 51] and the *Activation Ratio* of the abductor and adductor muscles similar to the method described above.[58, 59] We expect that pain will influence the *Principal Action* direction of the muscles.[51] Patients with pain will consequently show an increase in activation of the glenohumeral depressors during arm abduction moments (i.e. adductor co-activation as expressed in low *Activation Ratios*). The *AR*'s obtained within this '*Principal Action*' set-up will be compared to *AR*'s obtained from the derived abduction and adduction tasks as obtained with the EMG set-up applied during the acquirement of radiographs, taking potential experimental dependencies of *AR* into account.

The second hypothesis is that after lidocaine injection the muscle activation patterns of the patients move toward a normal activation pattern, as expressed in higher adductor *Activation Ratios* and near normal *Principal Action* directions.[5, 51]

Main outcome parameters: Muscle specific *Principal Action* (*PA*) parameters and muscle specific *Activation Ratios* (*AR*) before and after a subacromial injection with lidocaine.

2.3.6 Model simulation (H)

Impaired cuff function and RoM data obtained from the 3D-Kinematics measurements will be used as input data for the inverse dynamic model simulation with the Delft Shoulder and Elbow Model (DSEM) in order to estimate discrete muscle forces and joint reaction forces with the use of inverse dynamic simulation.[64] Muscle quality and glenohumeral joint stability can be varied and compared to the observations on muscle quality (MRI) and humerus cranial translation (2D radiography).[57]

Similarly to the hypothesized clinical measurements outcomes, we expect to find co-activation of arm adductors on affected shoulders during arm abduction simulations, in combination with altered shoulder muscle force patterns for standardized movements with respect to the control shoulders.

The predicted model muscle forces can be used for validation and interpretation of recorded muscle activations by means of EMG (2.3.2 and 2.3.5).

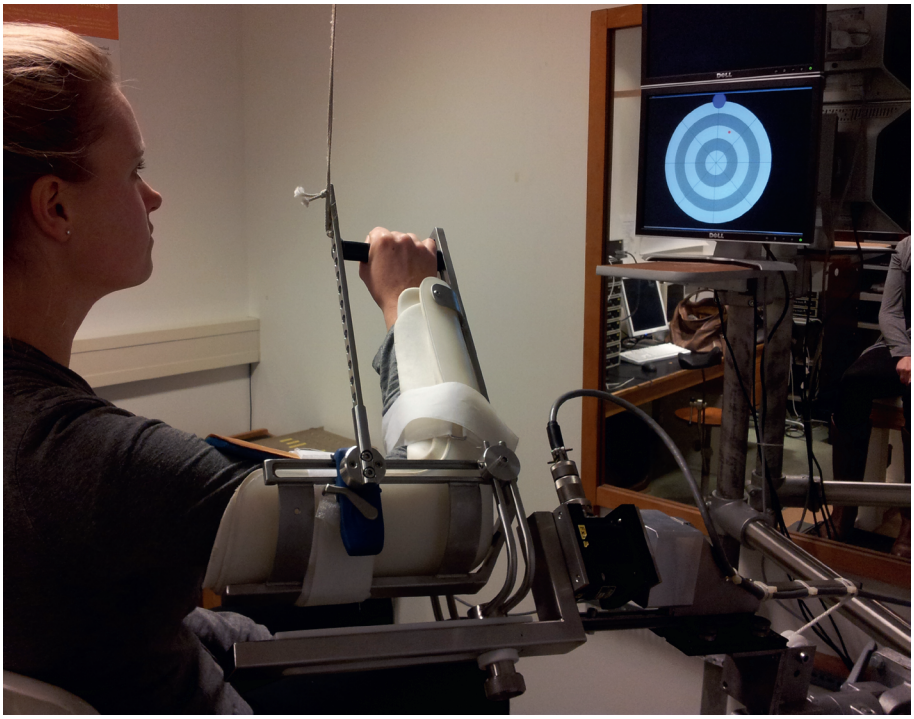


Figure 3. Experimental setup for isometric arm-shoulder force tasks.
The subject has the arm in a splint, which is connected to a force transducer. Subjects must bring the arm force driven red cursor into the blue target area, which indicates force direction (n = 24 directions) and force magnitude. The exerted force, perpendicular to the humerus long axis, is recorded together with EMG to measure the activity of 10 individual muscles around the shoulder.

2.3.7 Patient phenotyping (I)

The radiological and biomechanical outcome measures will be related to patients' clinical status or phenotype. We combine an overall general health outcome measure (i.e. SF36), a l regional (e.g. shoulder) outcome measure, and a disease- or condition-specific measure for patient assessment.[65]

- SF-36: Questionnaire to measure quality of life, based on physical function, illness, pain and mental health.[66]
- Illness Perception Questionnaire (IPQ): measures perception and impact of illness.[67]
- The Disabilities of the Arm, Shoulder and Hand (DASH) score: to quantify impact and functional impairment of shoulder arm and hand function.[68]
- Constant Shoulder Score (CS): used by physicians to quantify the severity of symptoms and functional impairment in affected shoulders, compared to the unaffected shoulder.[69]

- Western Ontario Rotator Cuff index (WORC): a self-reported outcome measure for assessing shoulder problems as a consequence of rotator cuff disease.[70]
- Visual Analogue Scale (VAS) for pain during daily life activities and in rest.

2.3.8 Relate outcome measures to pathophysiological mechanisms

Results of the recorded clinical, radiological and biomechanical measurements will be interpreted and combined in order to classify patients in to the hypothesized etiological subgroups.(Figure 4) Ultimately, if a patient has evidence of co-activation of arm depressors with abduction but no signs of shape parameters (e.g. acromion classification) playing a role, this would implicate that an intrinsic and dynamic mechanism is the main pathologic mechanism. On the other hand, evidence of e.g. a type III acromion (hooked) without any signs of relative cranial translation of the humerus would be suggestive of a primarily extrinsic and static/structural cause.
The following scenarios are considered:

1) Dynamically reduced subacromial space, due to (relative) cranial translation of the humerus.
   Humerus cranialisation causing encroachment of subacromial tissues will be characterized by limited *AH* in resting state on standard radiographs, further decrease in *AH* during abduction tasks, and decreased reconstructed subacromial volume (MRI). In some patients, this cranialisation might be (partially) compensated by I) co-activation of arm adductor muscles, and/or II) altered kinematics of the humerus and the scapula (scapulohumeral rhythm). Nevertheless, instead of a compensation mechanism, altered scapulohumeral rhythm can also be a cause of SIS in some patients (e.g. decreased scapula lateral rotation during arm abduction, with consequent *relative* cranial translation of the humerus). Pain is suspected to be the main trigger for compensation mechanisms. Therefore, we expect that these compensation mechanisms will be less manifest during the second round of experiments, after a subacromial injection with lidocaine.
   Particularly relevant positive outcome measures for this subgroup are: decreased *AH*, low *AR* (adductor co-activation), altered *Principal Action* for adductor muscles, decreased reconstructed subacromial volume during active abduction, degeneration of rotator cuff muscles and altered scapulohumeral rhythm.

2a) Statically reduced subacromial space, due to structural narrowing (classic etiology).

The subacromial space can be narrowed as a consequence of structural anatomic variations, e.g. a hooked acromion impinging on the subacromial tissues. These potential causes will be investigated and quantified using shoulder radiographs and (segmented) MRI arthrograms.

As a consequence of structures impinging on the rotator cuff, compensation mechanisms might be present to prevent further subacromial encroachment, including altered scapulohumeral rhythm (increased lateral rotation or increased posterior tilt during arm abduction) and adductor co-activation during arm abduction. Again, we expect that these compensations mechanisms will be less manifest after a subacromial injection with lidocaine.

Important outcome measures for this subgroup are: shape parameters of scapula (hooked acromion, Bigliani classification, acromial spurs) and humerus, the presence and extend of rotator cuff tendinosis (fibrosis, tendinitis, partial articular or bursal side tear).

2b) Statically reduced subacromial space, as a consequence of subacromial inflammatory processes, without signs of actual structural subacromial narrowing.

In some subjects, symptoms of SIS are related to predominantly intrinsic causes. In these patients, we expect to find little or no anatomic variations impinging on the cuff and no evidently decreased *AH*. The hypothesized misbalance between subacromial volume and the space needed for subacromial structures can be caused by e.g. subacromial oedema, fibrosis, tendinosis and tendinitis, which will be mainly assessed by means of MRI.

As a consequence of the subacromial inflammatory reaction and pain, patients might have an altered scapulohumeral rhythm and adductor co-activation.

Main outcome measures for characterizing this subgroup are: the presence and extend of rotator cuff tendinosis (fibrosis, tendinitis, partial articular or bursal side tear) and subacromial oedema.

2c) Statically reduced subacromial space due to encroachment of subacromial tissues by an adjoining pathology or other structures than the acromion.

Besides humerus cranialisation and the classical etiologic mechanisms that have been related to SIS, subacromial tissues can be impinged as a consequence of an adjoining pathology or other structures than

the acromion. For example, coracoid impingement and subacromial/caudal osteophytes in (otherwise asymptomatic) osteoarthritis of the acromioclavicular (AC)-joint have been reported as causes for pain with arm abduction. In our study, these causes will be investigated with the use of radiographs, MRI and 3D-kinematics recordings. Therefore, the most important methods of investigation for this subgroup are: MRI and radiographs to evaluate e.g. AC-osteoarthritis and subacromial osteophytes, impingement on the superior aspect of the glenoid, impingement at the outlet of the shoulder, coracoid impingement, and other (subacromial) pathologies or impinging structures causing a deficiency of subacromial space.

3) Combination groups
As with many diagnoses in general, the cause of SIS symptoms is presumably heterogeneous. We expect that in most patients one of the hypothesized mechanisms will play a main role, but in a subgroup of patients, a combination of 2 or more mechanisms will be causing SIS.

Additionally, we expect to identify specific pathologies other than SIS causing shoulder complaints, including cuff tears, calcific tendinitis, first stage frozen shoulder, and SLAP lesions. Patients with these pathologies will not be included in the current SISTIM study, but some (cuff tears or calcific tendinitis) will be analysed separately in distinct research projects (trial registry numbers: NTR1545 and NTR2282).

2.3.9 Statistical analyses

Patient data, including patient characteristics, physical examination, interview, radiological findings, questionnaires, psychological scores, biomechanical measurements and MRI findings will be entered in a database.
With regard to presence of cranial translation of the humerus as detected on radiographs during rest and abduction and adduction tasks, statistical analysis will be performed by a means of repeated measures ANOVA, with the measure of co-contraction as a confounding factor.
For the isometric *Principal Action* EMG measurements, data are tested by means of a General Linear Model analysis for repeated measures, controlling for Muscle, subacromial anaesthetics, *sAH* and VAS for pain.
RoM in standardized motions will be analysed with a General Linear Model analysis for repeated measures, controlling for VAS pain, subacromial anaesthetic Y/N, *dAH* and *sAH*. *dAH* obtained from 3D-kinematics will be analysed equally.

Additionally, we will use cluster analyses on multiple variables to identify subgroups and students' unpaired t-tests to compare continuous variables (e.g. patient characteristics, clinical scores, RoM, *dAH*) between the subgroups.

3. Discussion

Despite the fact that there is no clear consensus on its etiologic mechanisms nor which combination of diagnostic criteria defines SIS, numerous clinical trials exist on patients with the diagnostic label "SIS". Conflicting inclusion and exclusion criteria for SIS are used across these heterogeneous studies, complicating interpretation of reported results. Additionally, several pathologies that have a similar patient history, pain pattern and findings on physical examination, can be mistakenly diagnosed as SIS.[45] Conclusions of these studies are based on results of patients with varying etiologic mechanisms and for that matter even varying pathologies wrongly diagnosed as SIS, resulting in the wide variety on views with respect to etiology, diagnosis and treatment of SIS that exists nowadays. Instead of studying the outcomes of various treatment modalities in patients with SIS symptoms, first a detailed analysis of possible underlying pathophysiologic mechanisms is needed. In this way, potential subgroups can be identified, subsequently needing specific approaches in both research and clinical decision-making, with regard to diagnostics and treatment pathways.

The SISTIM study is a cross-sectional descriptive large cohort study in which consecutively included patients will undergo a multitude of biomechanical, kinematical and clinical tests. Patients will be selected using strict eligibility criteria, including radiographs and MRI. As a result a unique set of radiological (radiographs combined with EMG, MRI), biomechanical (muscle activation patterns) and 3D motion data (Flock of Birds) will be available on each individual patient, besides usual clinical data and outcome measures (e.g. CS, WORC). This will give better insight in the etiologic mechanisms in patients with symptoms diagnosed as SIS. Whether there is actual encroachment of subacromial tissues is determined by 1) the volume of these tissues and 2) the available subacromial space (static and dynamic). Both are investigated in our study: the status of subacromial tissues will be investigated with MRI, and we will use bony shape parameters, 3D kinematics and muscle activation patterns, to study their role on the subacromial volume of each patient. As this subacromial space is mainly limited by the scapula and humerus, the interaction of (bony) shape parameters and the (dynamic) position of these structures will be investigated as well.

Our ultimate goal would be to design clinically applicable instruments for differentiating between patients that might benefit from a specific treatment modality (e.g. acromionplasty, depressor training etc.). Therefore, we plan to use our developed experimental methods and classification systems in a subsequent clinical trial, for assessing treatment outcomes of standard care methods in discrete etiological subgroups.

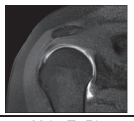
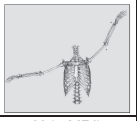
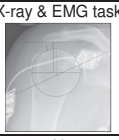
SIS Etiological Mechanism Subgroups	MRI 	X-ray 	FoB 	X-ray & EMG task 
1 *Cranial translation humerus, or Pathologic scapulohumeral rhythm*	X (+ FoB)		X (+ MRI) X	X
2a *Anatomic/structural (e.g. acromion)*	X	X		
2b *Subacromial inflamm. process*	X			
2c *Impingement & adjoining pathology*	X	X		
3 *Combination groups*	X	X	X	X

Figure 4. Schematic outline for relating outcome measures to pathophysiological mechanisms.
We expect to identify one or more of the hypothesized etiological mechanisms in each SIS patient. These mechanisms might be related to the reported variations in SIS symptoms, course, and treatment outcome.
(MRI: Magnetic Resonance Imaging, X-ray & EMG task: radiographs during EMG-recorded abduction and adduction tasks for measurements of acromiohumeral distance, and FoB: 3D kinematics with Flock of Birds system).

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CHAPTER 13

Summary and Discussion

Summary and Discussion

Subacromial Impingement Syndrome (SIS) is frequently diagnosed in patients with shoulder symptoms.[1, 2] However, its exact etiology is unclear and often reported as heterogeneous. There are no strict diagnostic criteria for "SIS" and there is no robust definition of this syndrome. Nevertheless, there are many trials and reports on patients with the diagnostic label "SIS". Conflicting inclusion criteria and heterogeneous patient groups are used across these studies. This complicates interpretation and comparisons of the highly variable results reported for the numerous treatment strategies for SIS. The aims of this thesis were to gain more insight in the underlying etiologic mechanisms of SIS symptoms, to develop methods to evaluate and identify these mechanisms in order to optimize future clinical decision making, and to study outcomes of usual care treatment methods for SIS symptoms.

In **Part 1A**, the definition and clinical use of the diagnostic label "SIS" is evaluated, as are the roles of the Supraspinatus and Deltoid during arm abduction, MRI findings in patients with SIS symptoms, and the outcome of usual care treatment options for SIS. **Part 1B** focuses on the treatment of patients with SIS symptoms caused by a specific radiologically diagnosed entity: calcific tendinitis. **Part 2** focuses on a new (Dutch validated) patient reported outcome measure for pain with arm abduction. Furthermore, novel biomechanical outcome measures are introduced, developed to evaluate patients with SIS symptoms and to gain more insight in underlying etiologic mechanisms. In **Part 3**, a study protocol is presented which uses the introduced methods to organize SIS patients into etiologic subgroups, on which diagnostic and treatment strategies should be specifically focused.

Part IA: What is Subacromial Impingement Syndrome?

Although its terminology and clinical use suggest differently, SIS is not merely a specific diagnosis or a specific pathology with in the subacromial space; it is a (pain) *syndrome*. Despite this, SIS is often referred to as a specific diagnosis in the literature and clinical practice. Over the past decades, many authors have commented on (heterogeneous) etiologic mechanisms and the complexity and lack of consensus with regard to diagnostic and treatment strategies involved in SIS.[2-13] Consequently, inconsistent and even conflicting inclusion criteria with subsequent heterogeneous patient groups are used across the numerous publications on SIS.[14] This complicates interpretation, comparisons and clinical implication of reported results and is the most likely explanation for the variations in reported outcomes of treatment methods.

In *Chapter 2*, views on the definition, diagnostics and treatment of SIS were investigated amongst physical therapists and shoulder surgeons from the United States and the Netherlands. The results illustrate the debate on the definition of "SIS" clinical practice. In this questionnaire study we did not only find systematic differences between nationalities and professional subgroups, but also substantial variability within these subgroups. Specifically with regards to the most classical etiologic mechanism of SIS, a hooked acromion, opinions varied greatly: 38% of the surgeons quoted this as an important etiologic factor, whereas 35% rated acromial shape as completely irrelevant. This might indicate a shift in the interpretation of the etiology of SIS as not purely based on the classic extrinsic mechanism (1972). Modifications of daily activities and physical therapy are the most important treatments according to physical therapists, who highly valued motion related etiologic mechanisms (dynamic causes for SIS symptoms). Surgeons, with higher ratings for intrinsic and anatomic or extrinsic etiologies, appreciated the use of subacromial corticosteroids and surgery. Hence, no consensus exits about the definition and treatment of SIS. We therefore suggest cautious use of the term "SIS" as a diagnostic label in both research and clinical practice.

The Supraspinatus is the Rotator Cuff (RC) muscle that is most frequently diagnosed as the source of SIS symptoms, including RC tendon tears and calcific tendinitis. Supraspinatus diseases generally lead to pain and loss of force during arm abduction.[15] In *Chapter 3*, the roles of the Supraspinatus and Deltoid muscles during arm abduction were investigated (EMG) in healthy subjects. The results show that, in contrast to the Deltoid, the Supraspinatus does not appear to be a specific abduction moment generator. Its role was actually highly variable between subjects. This in turn could be an explanation for the reported variations in severity of symptoms and treatment outcomes in patients with SIS symptoms. In support of this, also calcific tendinitis and Supraspinatus tears are often asymptomatic or self-limiting and in case of treatment, results vary greatly.[15-22] Hence, Supraspinatus pathologies seem to have varying consequences for individual patients, which might be (partially) due to an individually determined role of the Supraspinatus and other RC muscles. Surgeons should be aware of this, but more research is needed to gain more insight in these potential individual variations and to develop clinically applicable methods to identify the role of the (diseased) Supraspinatus and other shoulder muscles on patient level. This in turn could lead to more individualized treatment strategies and better clinical outcome.

In *Chapter 4*, we investigated patients with SIS symptoms, selected after physical examination and radiographs, with Magnetic Resonance Imaging (MRI) arthrograms. We assessed the presence of MRI characteristics related with SIS and subacromial narrowing in the literature, based on hypothesized categories of underlying etiologic

mechanisms: 1) Encroachment of subacromial tissues by structures (extrinsic); 2) Intrinsic impingement; and 3) Dynamic or motion related causes, e.g. due to (micro) instability.
Seventeen (36.2%) patients had specific other pathologies on MRI that can cause SIS symptoms, including RC tears, calcific tendinitis and labrum lesions. In the remaining 30 patients, 28 had signs of intrinsic etiologies, which were combined with findings of other etiologic categories in 27. Hence, signs of other diseases and various etiologic theories can be found with MRI arthrography in patients with SIS symptoms.
With cluster analyses on the MRI findings, patients could be organized in either a dynamic etiology ((micro)instability) group, or an extrinsic etiology group. Possibly, (micro)instability patients (e.g. glenohumeral index <0.61) need a different treatment approach than extrinsic SIS etiology patients (e.g. prominent acromion, caudal acromioclavicular joint osteophytes). The effect of different and tailored treatment approaches for these potential patient subgroups should be evaluated in future research projects.
Where the definition of SIS, Supraspinatus function and diagnostic findings in SIS patients were investigated in Chapters 2-4, *Chapter 5* describes a double-blinded Randomized Controlled Trial comparing two treatments of patients with SIS symptoms: arthroscopic subacromial bursectomy vs. subacromial bursectomy followed by acromionplasty. Interestingly, and in support of the results in Chapter 4, many patients with SIS symptoms had signs of specific other pathology on MRI or with arthroscopy and had to be excluded from the trial. After a mean follow-up of 2.5 years, both treatment groups improved and no significant differences between treatments were found. Acromion shape and severity of symptoms at baseline had significant influence on overall clinical outcome, regardless of type of applied treatment. To a further extend, patients with a more hooked acromion (extrinsic etiology) didn't have more benefit from an acromionplasty, despite this is one of the most performed orthopaedic surgeries over the past decades.

Summarizing, **Part 1A** of this thesis shows that the Subacromial Impingement Syndrome (SIS) cannot be used as a specific diagnostic label. SIS is a *syndrome:* a complex of signs and symptoms. The definition of SIS differs between and within groups of health practitioners from several professional backgrounds. When applying diagnostic imaging in patients with SIS symptoms, specific other pathologies can be found. And even when Supraspinatus pathology is expected, there are various potential underlying mechanisms. Even more, Supraspinatus function is variable in healthy subjects, which might be a reflection of the variations in symptoms and treatment outcomes of Supraspinatus pathologies in the literature. And lastly,

acromionplasty, the classic surgical treatment of SIS symptoms, does not lead to better clinical outcome than bursectomy.
In patients with severe and persisting SIS symptoms, adequate diagnostic methods including radiographs and ultrasound or MRI are indispensable for clinical decision making. The term SIS, which suggests a specific underlying mechanism and anatomic differentiation, should be used with caution, both in clinical practice and research. Better methodological criteria and more specific and externally validated terminology should be used whenever possible.

Part IB: Calcific Tendinitis, a frequent cause of "impingement" symptoms.
Subacromial calcific deposits are often observed on shoulder radiographs in case of SIS symptoms. Incidence rates of Rotator Cuff Calcific Tendinitis (RCCT) range between 7% and 54% in patients with shoulder pain.[23-28] Little is known on its epidemiology and long-term course. And even though there seems to be an evident cause for symptoms in these patients, in contrast to most patients with SIS symptoms, no consensus on treatment strategies for RCCT exists. Numerous treatments with varying outcomes have been reported for patients with severe and persisting symptoms.[21, 29-38]
In *Chapter 6*, baseline characteristics, long-term clinical outcome and prognostic factors are presented for a large group of RCCT patients, who were treated with barbotage or more conservative methods. The results show that RCCT mainly affects middle-aged individuals, most often women. The Supraspinatus tendon and the dominant arm were predominantly affected and in 21% there was bilateral RCCT. After a mean follow-up of 14 years, about 55% of patients had poor to moderate clinical outcome. Involvement of the dominant arm, bilateral disease, a long duration of symptoms, multiple calcifications and female gender had a negative association with long-term outcome. No significant effects of baseline Gärtner calcification classification and type of treatment were found.
To further investigate RCCT treatment strategies, a double-blinded Randomized Controlled Trial was conducted, comparing two regularly applied treatments for RCCT (*Chapter 7*): ultrasound(US)-guided barbotage, vs. a US-guided subacromial corticosteroids injection (SAI). Both barbotage and SAI improved clinical and radiographic outcome after 1 of year follow-up, but results for barbotage were superior: significantly higher WORC and Constant Scores and higher resorption rates. With regression analyses, correcting for baseline Constant Score and Gärtner classification, the mean treatment effect was 20.5 points (p=0.05) on the Constant Score (range, 0-100) in favor of barbotage. Furthermore, specifically patients with baseline Gärtner type II or III calcifications (range, I-III) had better clinical results of barbotage, whereas clinical results were similar for all Gärtner types in the SAI group.

Part 1B shows that many RCCT patients have persisting symptoms on the long term. Negative prognostic factors are: dominant arm involvement, bilateral disease, female gender and long duration of symptoms. Possibly, more rigorous follow-up, diagnostics and treatment is advisable in patients with persisting symptoms, no signs of resorption over time and negative prognostic factors. In a randomized controlled trial, the results of barbotage were superior to those of subacromial injections after 1 year of follow-up, specifically in case of Gärtner type II and III calcifications.

Part 2: Novel outcome measures for patients with "impingement" symptoms.

Despite the high incidence rates of SIS and the ensuing high number of research projects on SIS symptoms worldwide, there are currently few validated outcome measures focusing on RC pathologies, including calcific tendinitis, RC tears, or SIS symptoms in general. Objective and validated measures, including patient reported outcome measures, are needed to assess treatment outcome and patient coping strategies, understand underlying etiologic mechanisms and identify etiologic subgroups. In Chapter 8, we focused on a tool for clinical phenotyping and in Chapters 9-11 on new biomechanical (laboratory) outcome measures.

For accurate patient assessment, it is advisable to combine a general health outcome measure, a regional outcome measure and a condition specific outcome measure.[39] The Western Ontario Rotator Cuff index (WORC) is one of few questionnaires specifically designed for RC pathologies, which is increasingly applied.[40-46] In *Chapter 8*, a comprehensive combination of psychometric properties of the (Dutch validated) WORC was assessed according to the guidelines of the Scientific Advisory Committee (SAC),[47, 48] by comparing outcomes at several follow-up moments in a heterogeneous patient group with RC pathologies, including general SIS symptoms, RC tendon tears and calcific tendinitis. The WORC proved to have good internal consistency, reproducibility, responsiveness, sensitivity to change and construct validity. Therefore, the WORC is a valid condition specific patient reported outcome measure in patients with SIS symptoms.

Biomechanical evaluation of SIS symptoms is not only important in the form of objective outcome measures to evaluate e.g. treatment methods, but also to investigate shoulder joint biomechanics and underlying mechanisms of RC diseases. The function and activity of the RC muscles are hard to measure directly and generally require nerve-blocks or intramuscular EMG electrodes. Therefore, we described methods to measure RC function indirectly, by assessing adductor co-activation and Deltoid activation.

In case of RC dysfunction, the muscle moment balance around the glenohumeral (GH) joint is altered: there is 1) increased Deltoid activation during abduction to

compensate for lost RC abductor forces,[49-55] and 2) decreased glenohumeral stability as a consequence of impaired RC function.[49] The combination of these mechanisms can cause *excessive* cranial translation of the humerus, resulting in (painful) impingement of subacromial tissues.[49, 56, 57] In addition, there is compensatory 'out-of-phase' adductor activation (i.e. co-activation of the Teres Major and Latissimus Dorsi muscles) during abduction in RC patients.[49, 55, 58-60] These adductors have caudally directed force directions, which supposedly reduce cranial translation, at the cost of abduction strength.

Current methods to assess adductor co-activation are cumbersome to use and provide no easily interpretable outcomes. In *Chapter 9*, we introduced an experimental set-up that assesses adductor (co)activation in a straightforward manner. The developed method, which was applied to RC tear patients and controls in this study, expresses EMG of abductors and adductors in an easily interpretable measure: the "Activation Ratio (AR)" (-1<AR<1), where lower values express more (pathologic) co-activation.[60] Mean AR's in the (healthy) control group ranged from 0.7 to 0.9 with moderate to good test-retest reliability. Patients showed significantly more adductor co-activation during abduction. With adductor AR's ranging between 0.3 and 0.5, the method discriminates symptomatic RC tear patients from healthy controls in a straightforward manner and quantifies adductor co-activation in a readily interpretable "Activation Ratio".

In patients with SIS symptoms without an actual RC tear, there may still be RC dysfunction and adductor co-activation. In *Chapter 10* we investigated adductor and abductor (co)activation with the same set-up as in Chapter 9, but now with simultaneously acquired shoulder radiographs and in subjects without an RC tear. In that way, we were able to investigate adductor co-activation and its relation with humerus cranial translation in patients with a full-thickness RC tear, patients with SIS symptoms without any other pathologies, and asymptomatic controls. The results showed that at rest, the space between humerus and acromion is significantly smaller in RC tear patients (7.6mm) compared to SIS patients (11.1mm) and controls (8.9mm). Both during abduction and adduction tasks, cranial humerus translation was observed with equal magnitudes for patients (RC tear and SIS) and controls, with mean values of 2.3 and 1.7 mm, respectively. EMG measures showed pathologic "out-of-phase" adductor co-activation during abduction in specifically the RC tear patients. However, in multivariate regression analysis, no association between adductor co-activation and humerus cranial translation was found.

Assuming that RC abduction dysfunction is compensated by more activation of the Deltoid,[49-55] we may also use Deltoid function to identify RC dysfunction (*Chapter 11*). With a method introduced by Steenbrink et al.[55], we investigated compensatory Deltoid activation in RC tear patients before and one year after RC tendon repair

surgery and compared their results with healthy controls. We found that an increase in arm abduction moment loading is accompanied by an increase in Deltoid muscle activation in both controls and RC tear patients. The mean increase was larger in pre-operative RC tear patients compared to controls. This Deltoid compensatory role was reduced one year after surgical RC cuff repair, suggesting (partially) restored RC function. However, standard deviations were large in RC tear patients, which might be due to heterogeneity of the patient group and various underlying etiologic mechanisms.

In **Part 2**, we introduced several outcome measures for RC patients. We strongly recommend the use of the WORC as a disease-specific patient-reported outcome measure in both research and clinical practice for patients with SIS symptoms. Adductor (co)activation and compensatory Deltoid activation are indirect measures for RC dysfunction and can discriminate RC patients from controls. Additionally, we introduced a standardized and straightforward method that combines EMG and radiographic measurements. Also humerus cranial translation on radiographs discriminates RC patients from controls. However, no association could be found between humerus cranialisation and adductor (co)activation. The introduced set-ups give more insight in biomechanics and muscle function in RC patients on group level in scientific research, but are not yet applicable on patient level or in clinical practice.

Part 3: Future implications.
In *Chapter 12*, a study is presented in which the various introduced experimental set-ups and outcome measures are combined in order to identify specific predominant etiological mechanisms in individual SIS patients. For this study, we developed a theoretical framework for the etiology of impingement (when defined as "a misbalance between subacromial volume and the space needed for subacromial structures") based on several proposed mechanisms:

1) A dynamically reduced subacromial space due to a pathologic pattern of arm-shoulder movements (e.g. scapular dyskinesia) and/or (micro) instability, resulting in relative cranialisation of the humerus with respect to the scapula/acromion.
2) A more statically reduced subacromial space, due to:
    a. structural anatomic variations (e.g. a hooked acromion);
    b. encroachment of subacromial tissues by an adjoining pathology or structures other than the acromion (e.g. caudal acromioclavicular (AC)-joint osteophytes, calcific tendinitis and coracoid impingement).

c. a subacromial inflammatory reaction (e.g. caused by micro-trauma or overuse) causing subacromial oedema, fibrosis and tendinosis;

In this study protocol, we propose to combine outcomes of clinical scores, MR arthrography, radiographs, EMG, shoulder model simulation, 3-dimensional motion registration, and repeated measures after the injection of subacromial anaesthetics in order to categorize patients in one or more of the hypothesized subgroups in a research setting. The ultimate goal would be to design clinically applicable instruments for differentiating between subgroups of patients that might benefit from specific and tailored treatment modalities (e.g. acromionplasty, depressor muscle training, etc.).





CHAPTER 14

General conclusions

General conclusions

In this thesis we analyzed patients with SIS symptoms due to RC tears, calcific tendinitis, or other causes, with biomechanical, clinical and patient reported outcomes.

Part 1A of this thesis shows that Subacromial Impingement Syndrome (SIS) is often misused as a specific diagnostic label. There is no consensus on its definition, etiology, diagnostics and treatment amongst international health practitioners. With imaging technologies, specific alternative pathologies can be found in many patients with SIS symptoms. And even when there is evidence of Supraspinatus pathology or actual "impingement" of subacromial tissues, there can be various underlying mechanisms. Also, Supraspinatus function appears to be highly variable in healthy subjects, which might reflect the variations in symptoms and treatment outcomes of Supraspinatus conditions reported in the literature. Lastly, acromionplasty, the classic surgical treatment of "SIS", does not lead to better clinical results than a bursectomy, where the shape of supposedly impinging acromion is not altered.

Part 1B of this thesis shows that calcific tendinitis (RCCT) is a common cause of SIS symptoms, specifically in middle aged female patients. More rigorous follow-up, diagnostics and treatment is advisable in patients with persisting or severe symptoms, no signs of resorption over time and one or more of the following negative prognostic factors: dominant arm involvement, bilateral disease, female gender and long duration of symptoms at first presentation. Regarding the treatment of RCCT, results of barbotage are superior to subacromial injections.

In **Part 2** of this thesis, new clinical and research outcome measures are introduced. The Western Ontario Rotator Cuff index (WORC), one of few condition specific patient reported outcome measures for RC patients, demonstrated good psychometric properties. The assessment of adductor co-activation (EMG) during abduction tasks enables differentiation of controls from RC tear patients on group level. However, we could not confirm the previously hypothesized association between adductor co-activation and (the prevention of) subacromial narrowing. Lastly, RC tear patients have a large increase in Deltoid activation in response to increased abduction loading, presumably to compensate for lost RC function. After RC repair, the compensatory Deltoid activation decreases, suggesting (partially) restored RC function. Hence, adductor co-activation and Deltoid activation proved easy-to-measure proxies for RC (dys)function on group level, in research setting.

How are these results relevant and helpful in the treatment of SIS symptoms?

In patients with severe and persisting SIS symptoms, adequate diagnostic methods including imaging techniques are indispensable for clinical decision making. Only

then can various causes for SIS symptoms be identified, including specific other pathologies causing symptoms. In case of no specific other pathologies, patients with e.g. a more intrinsic or dynamic (e.g. (micro)instability) related etiology are likely to need another treatment approach than patients with more anatomic or extrinsic (structural) related etiologies. We suggest cautious use of SIS as a diagnostic label and encourage the use of more specific (etiology related) language whenever possible.

The flowchart displayed in **figure 1** puts the studies in this thesis into perspective. *Green blocks* represent new information obtained from results of studies in this thesis that can be used for evidence based medicine in clinical practice. *Yellow blocks* represent fields that have been studied in this thesis without direct influence on current clinical decision making. More research on these subjects is needed to imply obtained knowledge in clinical practice (see next section). The steps in figure 1 can be summarized as follows:

- When assessing a patient with persistent SIS symptoms, it is important to identify specific causes with patient history and physical examination. Cools and co-workers presented a comprehensive flowchart that can be applied.[61] Other pathologies, such as frozen shoulder syndrome and symptomatic acromioclavicular joint osteoarthritis need to be identified and treated accordingly.
- It is recommendable to use radiographs when a next step in diagnostics is needed. In case of calcific tendinitis, it is important to investigate whether there is a current resorptive phase. In case of resorption, pain management is the main goal, followed by regular follow-up visits and radiographs to monitor further resorption. If there is no resorption and symptoms are severe and persistent, more rigorous follow-up and treatment is indicated, specifically in case of one or more of the following negative prognostic factors: dominant arm affected, bilateral RCCT, long duration of symptoms, multiple calcifications and female gender. For treatment, good results can be expected from barbotage, especially in case of Gärtner type II or III calcifications.
- In case of a period of unsuccessful conservative treatment of SIS symptoms and no abnormalities on radiographs (besides signs associated with asymptomatic acromioclavicular joint osteoarthritis, "classic SIS" (e.g. hooked acromion) or RC tears (e.g. small acromiohumeral distance)), further investigation by means of e.g. ultrasound, MRI or MR arthrography is recommended.
- With MRI (with or without arthrography), specific pathologies that can give SIS symptoms can be identified, including RC tears. More clinically

applicable methods for assessing underlying etiologic mechanisms and for discriminating symptomatic and asymptomatic tears need to be developed, in order to select patients who will benefit from e.g. surgical RC repair. Radiographs acquired during force tasks, whether or not in combination with EMG-recordings of the Deltoid and adductor muscles, might play a role in this.

- In case of signs of SIS but no other specific pathologies with MRI, a set of MRI criteria (see Chapter 12) can be used to assess whether there is e.g. a more extrinsic related or a more dynamic (e.g. (micro)instability) related etiology of SIS symptoms. It is plausible that these SIS subgroups need specific treatment approaches; for example physical therapy and adductor training in patients with dynamic causes, and surgery in patients with extrinsic/structural causes.

In Chapter 12 of this thesis, it is described how we plan to combine clinical and translational methods in a large study, to further identify intrinsic, dynamic, and extrinsic etiologic mechanisms in patients with SIS symptoms. The ultimate goal of this final study is to develop pragmatic tools and a flowchart that can be used in clinical decision making, in order to diagnose and treat SIS patients according to their principal underlying etiologic mechanism.

Future perspectives

Future research of SIS symptoms should not focus on the outcomes of various treatment strategies for patients with "SIS", but on the identification of underlying causes. In that way, we can develop and apply tailored treatment strategies for specific causes of shoulder symptoms that often (wrongfully) receive the diagnostic label "SIS" in current clinical practice.

A strategy for further investigations and identification of subgroups of patients with SIS symptoms is reported in Chapter 12:

- In patients with a dynamically reduced subacromial space (due to e.g. micro-instability, dyskinesia, or relative cranialisation of the humerus with respect to the scapula/acromion), it would be expected to find relatively large amounts of humerus cranialisation on abduction task radiographs. Additionally, these patients can have a small glenohumeral index and a large distance between the tendons of the long head of the Biceps and the Supraspinatus on MR arthrography. Adductor co-activation might be limited in these patients.
- Patients with a more statically reduced subacromial space due to classic structural anatomic variations will have a more hooked acromion.

(Dynamic) compensation mechanisms might be present to prevent further subacromial encroachment, including altered scapulohumeral rhythm (increased lateral rotation or increased posterior tilt during arm abduction) and adductor co-activation during abduction.

- Encroachment of subacromial tissues can be also be caused by an adjoining pathology or structures other than the acromion (e.g. caudal acromioclavicular joint osteophytes, calcific tendinitis and coracoid impingement). These patients will have signs of e.g. caudal osteophytes, calcific deposits, internal impingement, or reduced coracohumeral distance with imaging technologies. Also these patients can have signs of (dynamic) compensation mechanisms.
- Lastly, patients can have an intrinsic subacromial problem due to an inflammatory reaction (e.g. intrinsic, caused by micro-trauma or overuse) causing subacromial oedema, fibrosis and tendinosis. For this particular subgroup, this would not be secondary to other mechanisms causing encroachment of subacromial tissues, but it would be a primary cause of SIS symptoms. Hence, we expect to find little or no anatomic variations impinging on the cuff and no evidently decreased AH in these patients. However, these patients can show (dynamic) compensation mechanisms including adductor co-activation and altered scapulohumeral rhythm.

These subgroups might need specific treatment strategies. It is important, however, to note that the mentioned subgroups are mainly theoretical. Also, the methods introduced in this thesis need further development for application in clinical practice, and in order to be able to identify underlying mechanisms on subject level. Possibly, MR arthrography characteristics (Chapter 4) and force task radiographs (Chapter 10) can play a role in this. And with the introduced easy to apply EMG set-ups with interpretable outcomes (Chapters 9-11), there might be even a place for EMG methods in future clinical evaluation of patients with SIS symptoms.

When more knowledge is obtained on patient subgroups and applicable methods to identify these (etiological) subgroups, further investigations should focus on treatment strategies within each of these subgroups. It is plausible that patients with intrinsic related causes would benefit more from e.g. NSAIDs, subacromial injections and physical therapy; patients with a dynamically reduced subacromial space from specific physical therapy strategies focused on scapulohumeral rhythm, or adductor training; and patients with extrinsic or structural related causes from surgical treatment with or without acromionplasty. This can be investigated with trials within the identified etiological subgroups.

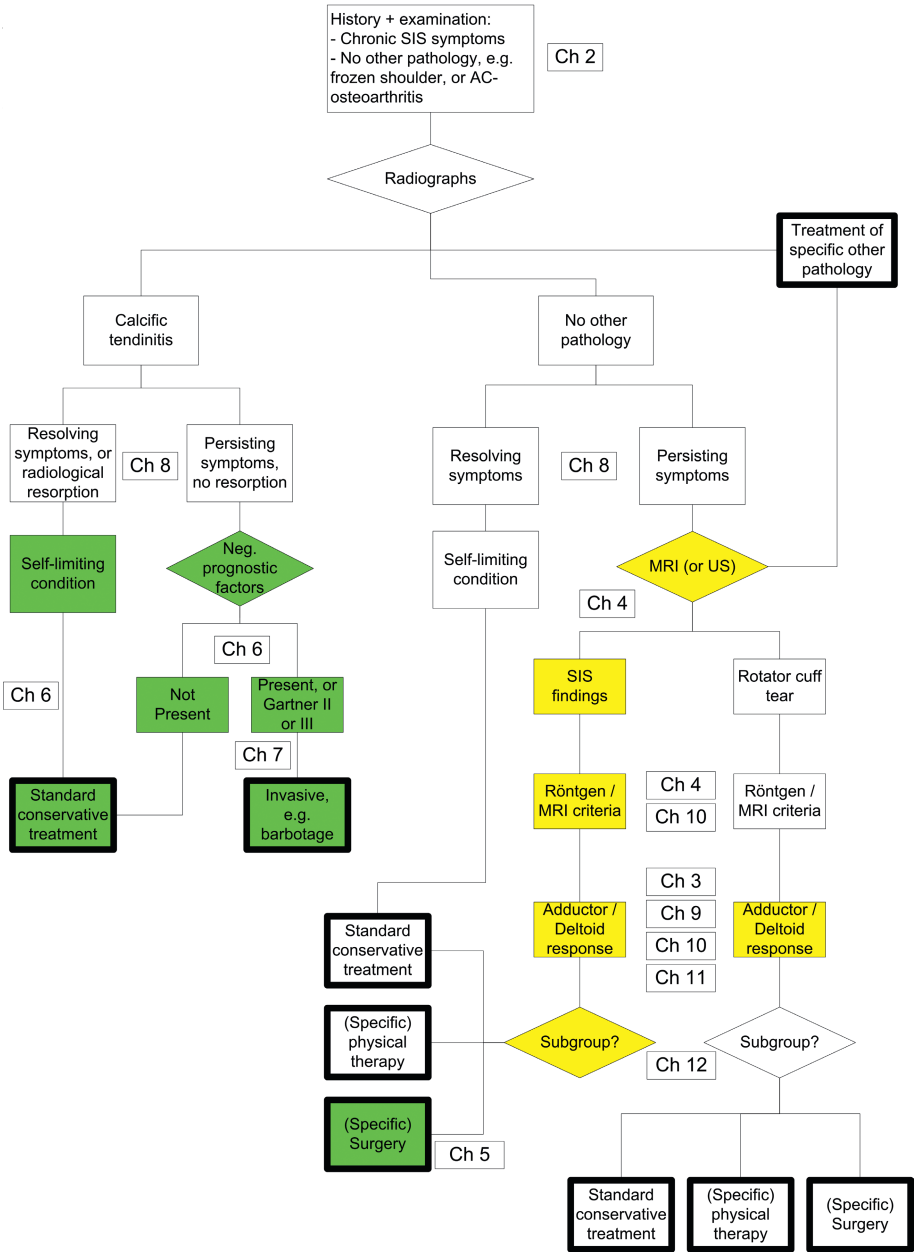


Figure 1. Relations between chapters in this thesis and clinical practice.
Green blocks represent new information obtained from results of studies in this thesis that can be used for evidence based medicine in clinical practice. *Yellow blocks* represent fields that have been studied in this thesis without direct influence on current clinical decision making. More research is needed to imply obtained knowledge in clinical practice.

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CHAPTER 15

Samenvatting (layman's summary in Dutch)

Samenvatting (layman's summary in Dutch)

Het Subacromiaal Impingement Syndroom (SIS), door sommigen ook wel slijmbeursontsteking genoemd, is één van de meest gediagnosticeerde oorzaken van schouderklachten. Er worden veel studies gedaan naar behandelingen van patiënten met de "diagnose" SIS en ook wordt de term veel gebruikt in de communicatie tussen zorgverleners. Dit kan echter tot verwarring leiden. SIS is namelijk een *syndroom*: een verzameling van vaak samen voorkomende klinische verschijnselen en symptomen, zonder een bekende specifieke oorzaak.
Vaak wordt gezegd dat SIS-klachten ontstaan t.g.v. een afwijkende vorm van het schouderdak (acromion). Het belangrijkste symptoom is pijn tijdens het heffen van de arm, waarbij wordt verondersteld dat er weefsels (o.a. de pees van de Supraspinatus spier) worden ingeklemd tussen het acromion en de kop van de bovenarm. Als niet-operatieve behandelingen zoals pijnstillers of injecties met ontstekingsremmers onvoldoende helpen en de klachten niet over gaan, kan gekozen worden voor één van de meest uitgevoerde orthopaedische operaties: de acromionplastiek (1972). De resultaten van deze operatie zijn echter zeer wisselend. Dat zou kunnen komen omdat er meerdere mechanismen bestaan die tot SIS-klachten kunnen leiden. Een acromionplastiek is daarom niet altijd de juiste behandeling. In dit proefschrift worden SIS, onderliggende oorzaken, methoden om naar deze oorzaken te zoeken en behandelingen van SIS-klachten onderzocht om in de toekomst deze schouderklachten beter te kunnen behandelen.
In **Deel 1** van dit proefschrift worden de definitie van "SIS" en het gebruik van deze term in de praktijk onderzocht. Tevens is gekeken naar de rol van de Supraspinatus spier in gezonde personen tijdens het heffen van de arm, MRI-bevindingen bij patiënten met SIS-klachten, de operatieve behandeling van SIS-klachten en de behandeling van patiënten met SIS-klachten i.c.m. kalkafzettingen in de schouder.
In **Deel 2** wordt een nieuw ontwikkelde patiëntenvragenlijst geïntroduceerd en worden enkele nieuwe biomechanische methoden om patiënten met schouderklachten te onderzoeken beschreven, welke gebruik maken van speciale röntgenfoto's en metingen van spieractiviteit (electromyographie, EMG). In **Deel 3** is een onderzoeksopzet beschreven welke gebruik maakt van de nieuw geïntroduceerde methoden om patiënten met SIS-klachten in de toekomst beter naar specifieke oorzaken te kunnen indelen en behandelen.
Samenvattend, wordt in *Hoofdstuk 2* in **Deel 1A** een studie beschreven waarin orthopaeden en fysiotherapeuten uit Nederland en de VS gevraagd werd naar de definitie van SIS en o.a. de diagnostiek en behandeling. Er bleek hierover totaal geen overeenstemming te bestaan. Zo vonden veel deelnemers dat een afwijkende vorm van het acromion inderdaad een belangrijke oorzaak van SIS-klachten is, waar

veel anderen het acromion totaal onbelangrijk vonden. Ons advies naar aanleiding van deze studie is dan ook de term SIS te vervangen voor meer oorzaakgerichte diagnostische termen in zowel medisch onderzoek als in de praktijk, en SIS in ieder geval niet te hanteren als een specifieke diagnostische term.

Hoofdstuk 3 is een studie met gezonde proefpersonen, naar de functie van de Supraspinatus spier (één van de schouderspieren die het vaakst is aangedaan bij SIS-klachten) en Deltoideus spier (de sterkste armheffer), tijdens het heffen van de arm; de beweging waarbij patiënten met SIS-klachten de meeste symptomen ervaren. Verassend blijkt uit deze studie dat de functie van de Supraspinatus zeer variabel is tussen personen. Dit zou één van de redenen kunnen zijn, waarom in de medische literatuur de symptomen en behandel-resultaten van SIS en andere Supraspinatus-aandoeningen zo wisselend zijn.

In *Hoofdstuk 4* hebben we uitgebreid MRI scans van patiënten met SIS-klachten bestudeerd. Hierbij hebben we gebruik gemaakt van nieuwe MRI-meetmethoden en specifieke criteria. Opvallend was, dat in 1/3 van de patiënten er met MRI-onderzoek andere oorzaken voor schouderklachten werden gevonden, die niets met het klassiek beschreven SIS te maken hebben. In de andere patiënten vonden we aanwijzingen voor verschillende onderliggende mechanismen die allemaal kunnen leiden tot irritatie van de Supraspinatuspees en omliggende weefsels. In een deel van de patiënten lijkt inderdaad het acromion een rol te spelen. In anderen waren er aanwijzingen voor (micro)instabiliteit in het schoudergewricht, wat er voor kan zorgen dat tijdens bewegingen de kop van de bovenarm te dicht bij het acromion komt, waardoor weefsels daartussen ingeklemd kunnen raken. Mogelijk kunnen nieuwe MRI-criteria zo een rol spelen in het zoeken naar de specifieke oorzaak van SIS-klachten bij individuele patiënten, zodat die specifieke oorzaak behandeld kan worden.

Zoals eerder aangegeven is acromionplastiek (wegzagen van een stukje van het acromion) een zeer vaak toegepaste orthopaedische operatie, ondanks dat het steeds duidelijker wordt dat het acromion niet bij alle patiënten met SIS-klachten een rol speelt. In *Hoofdstuk 5* hebben we bestudeerd of acromionplastiek een betere operatie is dan alleen het weghalen van slijmbeurs in de schouder. Verassend genoeg, bleek er geen verschil tussen beide behandelingen te zijn. Beide operaties leidden tot goed resultaat 2,5 jaar na behandeling. Zelfs bij mensen met een haakvormig acromion, welke theoretisch in de Supraspinatus kan "prikken", was er geen beter resultaat van acromionplastiek.

Vaak worden kalkafzettingen gevonden op röntgenfoto's van patiënten met SIS-klachten: tendinitis calcarea. Ondanks dat er in deze patiënten, in tegenstelling tot veel anderen met SIS-klachten, een duidelijk aanwijsbare oorzaak voor de klachten lijkt te zijn, is er geen overeenstemming over wat de beste behandeling voor deze

aandoening is. In *Hoofdstuk 6* in **Deel 1B** van dit proefschrift onderzochten we in een grote groep patiënten met tendinitis calcarea hoe het een aantal jaren na de diagnose met ze ging. Ook hebben we bestudeerd welke factoren invloed hadden op schouderklachten op de langere termijn. Het bleek dat tendinitis calcarea vooral bij vrouwen van middelbare leeftijd voorkomt. Gemiddeld 14 jaar na diagnose, gaat het bij de helft van de patiënten niet zo goed, of zelfs slecht. Tendinitis calcarea aan de dominante arm, aan beide armen, bij vrouwen, i.c.m. met een langere klachtenduur, of i.c.m. meerdere kalkafzettingen waren factoren die gerelateerd waren met slechter herstel op de langere termijn. Mogelijk zullen we daarom in de toekomst mensen met deze factoren in een eerder stadium met agressievere methoden behandelen dan we nu gewend zijn.
In *Hoofdstuk 7* hebben we 2 behandeling van tendinitis calcarea met elkaar vergeleken: het wegspoelen van de kalkdeeltjes (barbotage) en een injectie met ontstekingsremmers in de schouder. Wij vonden dat beide behandelingen wel voor vooruitgang zorgen na 1 jaar, maar de resultaten waren significant beter voor barbotage. Wij verwachten dan ook dat ook andere ziekenhuizen naar aanleiding van deze studie barbotage meer gaan toepassen, bijvoorbeeld bij patiënten met de gevonden negatieve prognostische factoren uit Hoofdstuk 6.
In *Hoofdstuk 8* van **Deel 2** hebben we een nieuwe internationale vragenlijst bestudeerd, welke speciaal ontwikkeld is voor SIS-klachten: de Western Ontario Rotator Cuff index (WORC). De Nederlandse vertaling van deze vragenlijst hebben we uitgebreid getest in een grote patiënten groep met SIS-klachten t.g.v. verschillende oorzaken, waaronder ook Supraspinatus scheuren en tendinitis calcarea. Uit deze studie blijkt dat deze vragenlijst zeer betrouwbaar en responsief is. Daarom adviseren wij deze vragenlijst toe te passen in de medische dagelijkse praktijk en in onderzoek naar schouderklachten, om objectief weer te kunnen geven hoe het met patiënten gaat.
In *Hoofdstuk 9* introduceren we een meetinstrument om naar de spieractiviteit van schouderpatiënten te kijken, welke in de toekomst mogelijk gebruikt kan worden om onderliggende oorzaken van SIS-klachten beter te kunnen indelen. In eerder onderzoek is beschreven dat bij patiënten met een gescheurde Supraspinatus spier 1) de Deltoideus spier harder aanspant bij het heffen van de arm om te compenseren voor de gescheurde Supraspinatus en 2) het spierevenwicht in de schouder veranderd is. Deze 2 mechanismen samen zouden er volgens verschillende onderzoekers toe leiden dat de kop van de bovenarm tijdens het heffen van de arm dichter bij het acromion komt, wat weer schade aan tussenliggende weefsels (o.a. Supraspinatus) en pijn kan veroorzaken. Eerdere studies met ingewikkelde en uitgebreide meetopstellingen hebben beschreven dat het aanspannen van andere spieren tijdens armheffing (adductor spieren) er voor kan zorgen dat er, ter

compensatie, weer wat meer ruimte onder het acromion komt. Inderdaad vonden ook wij, in Hoofdstuk 9, dat er bij patiënten met een gescheurde schouderpees meer activiteit van de adductoren is dan bij gezonde proefpersonen, maar nu met een gemakkelijk toe te passen meetinstrument. Mogelijk kan deze methode ook bij andere patiënten met SIS-klachten gebruikt worden om meer inzicht te krijgen in onderliggende oorzaken.

In *Hoofdstuk 10* hebben we het instrument van hoofdstuk 9 gebruikt en gecombineerd met tegelijk vervaardigde speciale röntgenfoto's, om de ruimte onder het acromion tijdens het aanspannen van schouderspieren te bestuderen. Hier vonden wij dat patiënten met een gescheurde Supraspinatus pees minder ruimte onder het acromion hebben dan andere patiënten met SIS-klachten en gezonde proefpersonen. Ook vonden wij dat patiënten inderdaad meer activatie van de adductoren hadden en dat er relatief meer vernauwing was tijdens het heffen van de arm dan in rust. We hebben echter in deze studie geen relatie tussen spieraanspanning van verschillende spieren en de ruimte onder het acromion gevonden. Meer onderzoek zal nodig zijn om te bestuderen of er tóch een relatie tussen de ruimte onder het acromion en spieractivatie is, en of dit gebruikt kan worden om in de toekomst beter onderscheid te kunnen maken tussen patiënten met SIS-klachten t.g.v. verschillende onderliggende oorzaken.

In *Hoofdstuk 11* hebben we de activiteit van de Deltoideus spier tijdens armheffen bestudeerd bij gezonden en bij patiënten met een Supraspinatusscheur, voor en na operatief herstel van deze spier. Zoals eerder beschreven in de medische literatuur, vonden wij inderdaad meer aanspanning van de Deltoideus bij de patiënten. Een jaar na operatief herstel, was er gemiddeld minder aanspanning van deze spier. Mogelijk betekent dit dat operatief herstel ervoor zorgt dat de Deltoideus minder hoeft te compenseren en dat er dus een verbeterde functie van de Supraspinatus is. We vonden op groepsniveau duidelijke verschillen tussen patiënten en gezonden in de Hoofdstukken 9-11. Ook hebben we meer inzicht gekregen in mogelijke onderliggende mechanismen van SIS-klachten. Echter, de toegepaste meetmethoden zijn op dit moment niet praktisch en precies genoeg om klinisch toe te passen om bijv. tussen behandelmethoden te kiezen.

In *Hoofdstuk 12* van **Deel 3** beschrijven we een uitgebreid onderzoeksprotocol, wat o.a. gebruik maakt van de in dit proefschrift toegepaste meetmethoden. We willen dit onderzoeksprotocol op een grote groep patiënten met SIS-klachten toepassen en proberen op die manier duidelijke subgroepen van patiënten met SIS-klachten te identificeren. Onderzoek in de toekomst zal er op gericht moeten zijn om individuele patiënten in de te delen in subgroepen die specifieke behandelingen nodig hebben. Dit zal er toe leiden dat er in de toekomst minder patiënten onnodig bijv. een acromionplastiek ondergaan en dat de behandelresultaten van SIS-klachten zullen verbeteren.

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Curriculum Vitae

Pieter Bas de Witte was born on February 8th 1982 in Leuven. In 2000 he graduated with honors from the 'Kaj Munk College' in Hoofddorp. The same year he started with Civil Engineering at Delft University of Technology and received a "Star" grant from the University. In 2003, he received his Bachelor's degree in Civil Engineering and switched to medical school at Leiden University.

During medical school, he had a research internship at the Orthopaedics department of Massachusetts General Hospital (MGH), Harvard Medical School in Boston (USA) under the supervision of D. Ring, MD, PhD, which led to several publications in international journals and continuous collaboration in research projects in the following years.

After an optional course on biomedical engineering, organized by Delft University of Technology and the Orthopaedics department of Leiden University Medical Center (LUMC), he started a student research project under supervision of R.G.H.H. Nelissen, MD, PhD, head of the LUMC Orthopaedics department. In this project, two treatment methods for Subacromial Impingement Syndrome were compared in a double-blinded randomized controlled trial. The impingement project served as the basis for this thesis. Additionally, he started a research project on the long-term follow-up of hip prostheses. The LUMC and MGH projects continued during his clinical rotations. In 2009, he received his Medical degree with honors.

From September 2009, he worked as a PhD-student at the Orthopaedics department at LUMC on the subject of Subacromial Impingement Syndrome and its underlying mechanisms, under supervision of R.G.H.H. Nelissen, MD, PhD, J.H. de Groot, MS, PhD, and J. Nagels, MD, PhD. He participated in several studies with a broad range of study design types, including clinical trials, retrospective cohort studies and biomechanical basic science studies. He worked in close cooperation with the LUMC Radiology department and with various teaching hospitals in the Hague, Leiderdorp, Amsterdam and Amersfoort. Furthermore, he performed a clinical residency at the LUMC Orthopaedics department and followed a broad range of courses in the fields of Ethics, Epidemiology and Statistics.

Several of his studies were published in international medical journals and presented at international Orthopaedic meetings. Additionally, he received a research grant of the Dutch Rheumatoid Arthritis Association, an AGIKO research grant from The Netherlands Organization for health research and development (ZonMw) and several traveling grants for international Orthopaedic meetings.

In January 2013, he started as a clinical resident at the General Surgery department of the Haga hospital in the Hague, as the first part of the six-year trajectory to become an Orthopaedic Surgeon. In August 2013 he received his SMBWO registration as a clinical epidemiologist. And in July 2014, he started as a clinical resident in Orthopaedic Surgery at LUMC.

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