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CHAPTER 13

Summary and Discussion

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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 <u>*Chapter 8*</u>, 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.).