Functional implications of structural “anomalies” in shoulder pain

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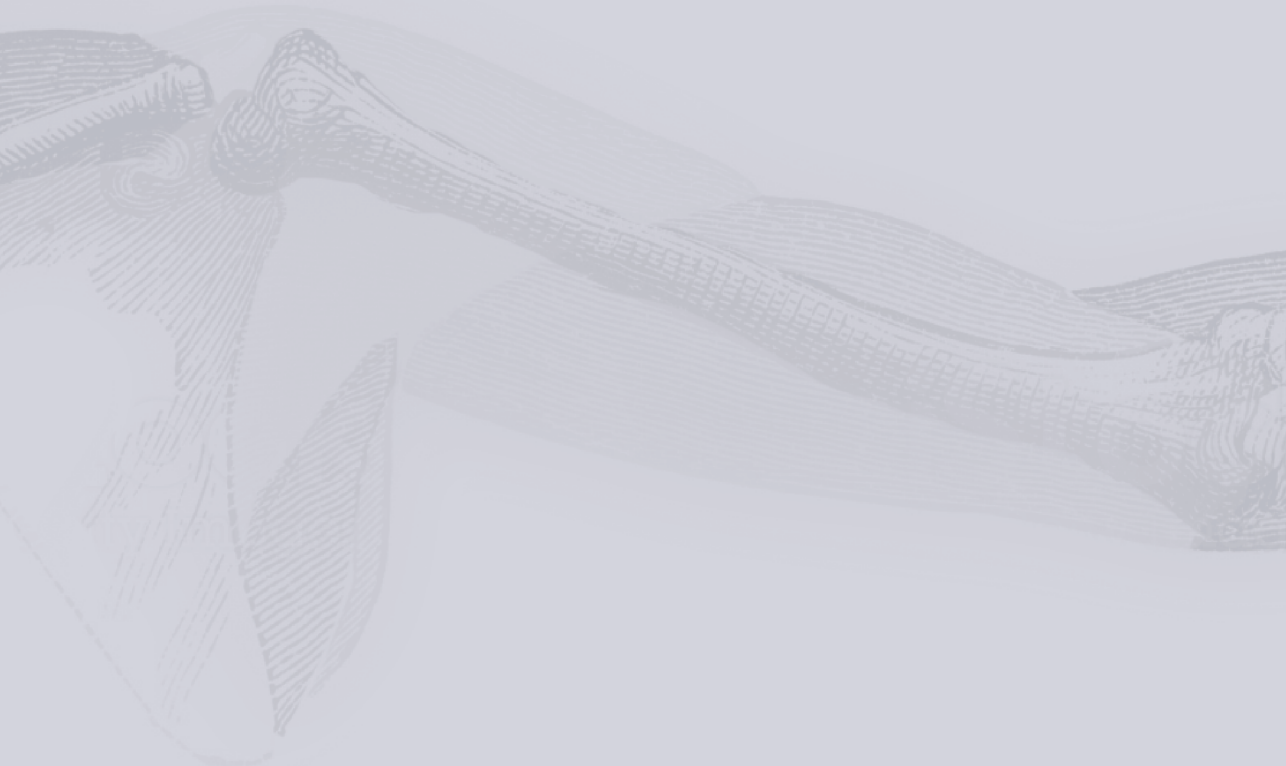

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Summary and general discussion

At the start of the SISTIM research project, the pathophysiology of the subacromial pain syndrome (SAPS) was poorly understood and literature lacked high-quality evidence justifying surgical treatment. The goals of this thesis were to evaluate the long-term outcomes of arthroscopic subacromial decompression as the common surgical intervention for SAPS; to create a biomechanical rationale for successful treatment options by studying patterns in shoulder muscle activity and kinematics in patients with SAPS; and to evaluate the association between the size of structural rotator cuff (RC) defects and shoulder kinematics. For that matter kinematics and shoulder biomechanics in symptomatic shoulders are analysed.

ARTHROSCOPIC DECOMPRESSION UNDER SCRUTINY

Up to recently, attrition of the RC under a hooked acromion or the coracoacromial ligament was assumed to cause subacromial impingement. As a result, arthroscopic subacromial decompression (i.e. acromioplasty) was widely used as a standard treatment for subacromial pain in the orthopaedic clinical practice, although high-quality evidence supporting its efficacy was limited.[32, 51, 68] Earlier work had shown no differences in shoulder pain and function between arthroscopic bursectomy alone or a bursectomy in combination with acromioplasty 2.5 years after treatment.[25] Although no short-term differences were found, the treatment effect of acromioplasty on clinical outcomes after 10 or 20 years were not studied although it has been hypothesised that repeated fraying of the RC under the hooked acromion may cause shoulder pain. In **Chapter 2** we evaluated the long-term effect of arthroscopic subacromial decompression in patients with SAPS in a randomised controlled clinical trial. The results showed no treatment effect of subacromial decompression in improving shoulder pain (Visual Analogue Scale; 95% confidence interval [CI] -21 - 9, P = 0.43) and function (Constant Score; 95%CI -5 - 16, P = 0.32) at a median of 12 years' follow-up. Moreover, acromioplasty seems not to protect the RC from tearing, since both groups with bursectomy alone and in combination with acromioplasty had comparable numbers (i.e. 10% versus 17%) of RC tears. These findings showed no treatment effect of acromioplasty and supported the advice against arthroscopic subacromial decompression in the treatment of chronic SAPS.

The justification of acromioplasty mainly relied on the positive results shown in cohort and non-comparative studies.[2, 3, 16, 56] Randomised studies suggesting no treatment effect received much more criticism.[25, 29, 36] Some orthopaedic surgeons from the shoulder community pointed at potential methodologic flaws of randomised work, trusting more on the evidence of the widely performed acromioplasty provided by non-randomised studies.[22, 29] In 2018, two additional randomised controlled clinical trials were published in well-respected scientific journals supported our results presented in this thesis.[1, 50] These studies did not show a beneficial clinically relevant effect of arthroscopic subacromial decompres-

sion compared to sham surgery on pain and shoulder function.[1, 50] When subacromial decompression was compared to physiotherapy alone, pain and shoulder function were significantly better after arthroscopic acromioplasty. However, these differences did not exceed the minimal clinically important difference, and could relate to surgical placebo effects.[50] Together with the available randomised clinical trials such as the study presented in this thesis, these two trials from 2018 contributed to the growing evidence against the favourable effect of subacromial decompression.[1, 7, 36, 50] Because of the absence of convincing evidence, the Dutch Orthopaedic Society recommends to use a conservative treatment protocol for SAPS. The current advice is to consider acromioplasty only after failure of extensive conservative treatment and this advice will probably be sharper formulated in their revised recommendations.[14, 48] Other national societies and international guidelines also changed their recommendation with respect to SAPS and now recommend not to perform acromioplasty or preserve acromioplasty for more selected patients with persisting symptoms after extensive conservative treatment.[22, 33, 66]

BIOMECHANICS AND KINEMATICS IN SUBACROMIAL PAIN SYNDROME

The lack of a uniform definition regarding patient characteristics and absence of accurate clinical tests to confirm the diagnosis for this pain syndrome may have contributed to heterogeneity in study populations.[11, 52] As a result, inconsistent study outcomes are reported in literature. In the SISTIM project, shoulder biomechanics and kinematics have been studied in a more homogenous sample created by including patients diagnosed with SAPS with comparable findings on magnetic resonance imaging (MRI) reflecting a more homogenous anatomic substrate for pain.[12]

Shoulder biomechanics and kinematics of patients with SAPS are compared to asymptomatic volunteers in **Chapter 3**. We found a lower activation ratio for the pectoralis major (i.e. relatively less agonistic activity) and higher activation ratio for the teres major (i.e. relatively less antagonistic activity) in the subacromial pain group. There was no difference in the activity of scapular stabilizers between patients with subacromial pain and asymptomatic controls. The contribution of glenohumeral motion to overall elevation (at 120° abduction mean difference -9°; 95% CI -14 - -3, P = 0.003) and external rotation (at 120° abduction mean difference -8°; 95% CI -13 - -3, P < 0.001) was lower in patients with SAPS indicating more scapulothoracic motion. Less external rotation has been demonstrated to bring the greater tuberosity in closer contact with the coracoacromial arch, shift contact pressures to the posterolateral RC and to bring the humeral head (especially between 60° to 120° of arm elevation) in closer contact with the acromion [19, 46]. The latter may contribute to the subacromial inflammation seen in patients with SAPS. Moreover, the teres major has

been suggested to counteract cranially directed destabilizing glenohumeral forces.[10, 61] We found a higher activation ratio for the teres major (i.e. relatively less antagonistic activity) during arm elevation in patients with subacromial pain, suggesting impaired function of the teres major as a humeral head depressor. This biomechanical knowledge is essential to unravel the pathophysiology of subacromial pain, to identify subgroups of patients with subacromial pain and to explain how treatments work. Physiotherapy directed at the teres major may enhance the antagonistic activity of the teres major to improve its function as humeral head depressor.

Orthopaedic surgeons and physiotherapists link scapular dyskinesis to the presence of SAPS.[11, 39, 42, 65] Scapular dyskinesis is clinically identified as an asymmetry in scapulothoracic motion between both shoulders.[65] Because scapular dyskinesis is believed a pathological finding, some rehabilitation programmes focussed on scapulothoracic kinematics in SAPS.[42] Quantitative motion analysis revealed scapular dyskinesis in SAPS, but inconsistent outcomes have been reported potentially due to heterogeneity in selecting criteria among studies.[17, 18, 39] In **Chapter 4**, we investigated the presence of asymmetry in scapulothoracic motion in a group of patients with subacromial pain after radiologic shoulder examination creating a group with a more comparable anatomic substrate for pain than in existing literature. We found more scapular internal rotation (mean difference 5° at 120° abduction; 95%CI 0 - 10, P = 0.034) in the affected shoulder, but did not find a difference in scapulothoracic lateral rotation (95%CI -3 - 4, P = 0.964) or posterior tilt (95%CI -6 - 3, P = 0.413) between the affected and unaffected shoulder. Interestingly, the absence of asymmetric scapulothoracic lateral rotation suggest that both shoulders are exposed to comparable biomechanics, since we demonstrated a difference between patients and asymmetric controls in chapter 3. Asymmetry of scapulothoracic motion may either cause pain by dynamically reducing subacromial space, be the consequence of pain or does not play a role and could be a normal observation in patients with shoulder pain. To improve our knowledge of pain and its effect on shoulder kinematics, we examined the effect of subacromial anaesthetics on scapulothoracic motion expecting more symmetrical kinematics after infiltration of subacromial anaesthetics. In other words, we expected pain to cause deviations in scapulothoracic motion. In contrast to our hypothesis, there was more asymmetric shoulder motion with more scapulothoracic internal rotation and less posterior tilt after infiltration than before. Subacromial infiltration with lidocaine was not an effective way to restore symmetrical shoulder motion. More internal rotation and less posterior tilt are known to reduce subacromial volume and thus are less favourable in SAPS.[38, 60] In other words, our kinematic data indirectly show that the removal of pain by infiltration of subacromial anaesthetics caused a reduction of subacromial volume. A possible explanation for our findings is that pain controls a local protecting mechanism which reduces the contact of inflamed tissues and the acromion. Moreover, this may identify asymmetric scapulothoracic kinematics in the pathophysiologic pathway for developing shoulder pain. Finally, we

found an association between less scapular lateral rotation (i.e. upward rotation) and higher patient-reported pain scores. More contact between inflamed subacromial tissues with the acromion may explain the higher self-reported pain scores in our study, because less lateral rotation brings the RC in closer proximity to the acromion.[38]

BIOMECHANICAL AND KINEMATIC CHANGES IN THE SHOULDER FOLLOWING A ROTATOR CUFF TEAR

Shoulder biomechanics in the presence of an RC tear have been further clarified by comparing shoulder biomechanics on specimens with an intact RC to biomechanics after an artificial RC tear was created.[23, 64] It was shown that the supraspinatus significantly contributes to the elevating torque during glenohumeral elevation as it has also been previously observed by Inman in 1944.[23, 31, 64] The absence of supraspinatus torques lead to a significant increase in deltoid muscle force and forces delivered by the intact portion of the RC.[23, 43, 62, 64] The m. subscapularis and posterior RC has also been shown to compensate for lost torques in the presence of a supraspinatus tear to facilitate a stable fulcrum for shoulder movement.[23] If stabilizing forces from the posterior cuff decrease, the humerus will cranially translate relative to the scapula and the ability for shoulder movement will be lost.[4, 59, 64] This biomechanical principle emphasizes the essential function of the posterior RC (i.e. teres minor and infraspinatus) and anterior RC (i.e. subscapularis) to maintain glenohumeral stability. It is also known as the "transverse force couple" (although this couple does not fully meet the definition in physics.[4, 23, 53, 64] The requirements for the delivery of a sufficient amount of torque in the presence of an RC tear have been calculated in computer models.[41, 62] In line with the findings from cadaveric studies, Steenbrink et al. confirmed with computer modelling the significant contribution of infraspinatus and teres minor forces for maintaining the stable fulcrum for shoulder motion.[62]

These biomechanics were considered to have an important impact on shoulder kinematics in patients, but this had to be validated. Existing work studied the effect of three-dimensional kinematics in patients with an RC tear, but did not account for the effect of tear size on shoulder kinematics.[45, 49, 58] In a cross-sectional study, we demonstrated the association of RC tear size and shoulder kinematics using three-dimensional electromagnetic motion analysis in **Chapter 5**. Patients with a massive RC tear involving the supraspinatus and infraspinatus had reduced glenohumeral elevation compared to patients with an isolated supraspinatus RC tear (mean difference 10° at 110° abduction; 95% CI 4 – 17, P = 0.002) or with an intact RC (mean difference 16° at 110° abduction; 95% CI 11 – 21, P < 0.001) during abduction, and forward flexion. This decrease in glenohumeral elevation coincided with an increase in scapulothoracic lateral rotation. We did not demonstrate a significant difference in glenohumeral motion between the patients with SAPS and an isolated supraspinatus

tear. These observations may reflect the biomechanical shift of forces in the shoulder with a massive RC tear. The kinematics are in line with the assumed essential contribution of the infraspinatus to preserve glenohumeral elevation in the presence of a supraspinatus tear. Because shoulder kinematics are associated with RC tear size, quantitative evaluation of shoulder kinematics can potentially be used in a diagnostic process of the patients with shoulder pain.

While designing a study that should examine molecular and cellular signatures of rotator cuff degeneration, it was postulated that structural defects in the shoulder may have an effect on the cell biology of other intact shoulder muscles, because adaptations in joint biomechanics change the entire shoulder system. In **Chapter 6**, we tested the beforementioned biomechanical concept of changed compensatory mechanical load of shoulder muscles in patients with a (postero)superior RC tear and its association with the development of muscle atrophy on patients. Prior animal and cross-sectional studies demonstrated less atrophy in the presence of an RC tear.[30, 35, 44] In an observational study with a mean of 3-years follow-up, we showed that the surface area of teres minor and deltoid muscles in patients with an intact RC on Magnetic Resonance Imaging decreased with age, which indicated muscle atrophy of these two intact muscles. In patients with a (postero)superior RC tear, however, the teres minor atrophied more slowly or even grew. This finding was most apparent in patients under the age of 50 years. Our findings are in line with our biomechanical rational and suggested that muscle atrophy in the shoulder can be reduced when an increase in mechanical load is exerted onto the muscle. A compensatory increase in teres minor muscle volume may compensate for lost infraspinatus forces, especially in younger patients, resulting in a stable fulcrum for joint motion. The latter could be an explanation for the remarkable discrepancy between the extend of an RC tear and shoulder complaints, in which a young patient with an extensive RC tear have no pain and excellent shoulder mobility.

KINEMATICAL AND CLINICAL OUTCOMES OF SURGERY

Disturbed scapula-humeral movement has been considered a *"sine qua non"* for the diagnoses of an RC tear. In chapter 5, we showed a decrease in glenohumeral elevation and increase in scapulothoracic lateral rotation in patients with a (massive) RC tear. Although ample studies investigated the effect of RC repair on pain and elevation angles using semi-quantitative methods, shoulder motion before and after RC repair had not been appraised with quantitative three-dimensional electromagnetic motion analysis. In **Chapter 7**, we evaluated three-dimensional shoulder motion in patients before and one year after RC repair. We demonstrated an increase in glenohumeral elevation and less scapulathoracic lateral rotation following an RC repair. Overall range of thoracohumeral motion increased after surgery. Postoperative shoulder kinematics were more comparable with the kinematics

of the asymptomatic contralateral shoulder. These observed changes in shoulder kinematics following RC repair coincides with improved shoulder range of motion and more symmetrical shoulder movement after surgery. Whether these changes in shoulder kinematics are the result of the relieve of shoulder related pain or restored functionality of the re-inserted RC muscle on the humeral head, remains unclear. Nevertheless, the evaluation of three-dimensional shoulder motion provides a quantitative measurement of shoulder movement, which might be a valuable alternative to semi-quantitative methods.

Fatty infiltration or retraction hampers a rotator cuff repair in patient with a massive posterosuperior RC tear. In these patients, a tendon transfer of the teres major or latissimus dorsi to the infraspinatus footprint may serve as a salvage procedure for the relatively young patient with a massive posterosuperior RC tear.[5, 6, 20, 26] Although the glenohumeral teres major has been suggested biomechanically superior to the humerothoracic latissimus dorsi, the latissimus dorsi tendon transfer got popularized by Gerber et al. and is nowadays the most commonly described tendon transfer.[20, 40] Only short-term outcomes have been reported for the teres major tendon transfer.[5, 6, 26] In **Chapter 8**, we described the long-term (mean 10 years) outcomes of the teres major tendon transfer in a cohort of patients with a massive irreparable posterosuperior RC tear. Shoulder function was still higher ten years after teres major transfer than preoperative. Similarly, lower pain scores were observed ten years after surgery. Our long-term data demonstrated that improvement in shoulder function and relieve of pain after teres major tendon transfer lasts for over ten years. A secondary aim of this study was to provide data on shoulder function and pain after latissimus dorsi tendon transfer surgery in cohort of patients with a similar indication for surgery with a mean follow-up of six years. Six years after latissimus dorsi transfer shoulder function and pain scores were improved compared to the preoperative scores. Moreover, this study described the general health related quality-of-life after tendon transfer surgery. Health related quality-of-life was significantly lower than in a normalised population indicating the severe impact of an RC tear. This study proved that teres major tendon transfer may generate successful outcomes even after ten years' follow-up. For that matter, the teres major tendon might be a valuable alternative to the commonly performed latissimus dorsi tendon transfer in the treatment of irreparable posterosuperior RC tears.

MAIN CONCLUSIONS

- Arthroscopic subacromial decompression/acromioplasty is not effective in improving shoulder function and relieving pain in patients with SAPS (Chapter 2).
- Patients with subacromial pain can still develop an RC tear after an acromioplasty (Chapter 2).

- Patients with SAPS have less glenohumeral elevation (with more scapulothoracic lateral rotation) and less glenohumeral external rotation (with more scapulothoracic posterior tilt) when elevating the arm (Chapter 3).
- Patients with SAPS have less teres major antagonistic activity during abduction moments than asymptomatic controls resulting in a lower activation ratio (Chapter 3).
- Subacromial infiltration with an anaesthetic does not restore symmetrical shoulder kinematics in patients with SAPS (Chapter 4).
- Less lateral rotation (i.e. upward rotation) and less posterior tilt of the scapula are associated with higher patient-reported pain in SAPS (Chapter 4).
- In-vivo shoulder kinematics are associated with RC tear size. Large tears involving both the supraspinatus and infraspinatus coincides with less glenohumeral elevation and more scapulothoracic lateral rotation during arm elevation (Chapter 5).
- While the cross-sectional surface area of the teres minor and deltoid muscle gradually decrease with age, these muscles show a limited decline or even an increase in cross-sectional surface area in patients with a (postero)superior RC tear. This finding suggests that alterations in mechanical loading may interfere with age-dependent muscle atrophy (Chapter 6).
- One year after RC repair, the operated shoulder reveals more glenohumeral elevation and less scapulothoracic lateral rotation than before surgery (Chapter 7)
- Latissimus dorsi or teres major tendon transfers are surgical options to improve pain and shoulder function in the treatment of a massive posterosuperior RC tears.

FUTURE PERSPECTIVES

Since the publication of our long-term outcomes of acromioplasty in 2017 in which we showed no beneficial effect of acromioplasty in SAPS (Chapter 2), more randomised controlled clinical trials have been published.[1, 36, 50] Consistent with our findings, no beneficial effect of acromioplasty was found. The emerging evidence have been recently adapted in some international guidelines and recommendations.[22, 33, 66, 67] These guidelines resulted in a decrease in the number of acromioplasties in the Netherlands from 2012 to 2016.[67] The upcoming years more work has to be done to prevent unnecessary subacromial decompression world-wide. The lack of evidence for surgical acromioplasty in patients with subacromial pain has still to be implemented in many other international guidelines. Because potential damage of subacromial tissues by attrition under the acromion may develop after many years as was hypothesised in our study, long-term follow-up data of other existing randomised controlled trials should be expected.[36] Moreover, a better selection of patients with subacromial pain may indicate that some patients are more likely to benefit from an

acromioplasty or surgical intervention, for example by a better selection of patients based on anatomic characteristics.

The ineffectiveness of subacromial decompression will urge orthopaedic surgeons to develop a more effective ways to treat patients with SAPS. Our biomechanical and kinematical outcomes (Chapter 3) can be used to justify developments in physiotherapeutic interventions targeting glenohumeral rotations and muscle activation in SAPS when a causal role of our findings is assumed. A lower contribution of glenohumeral elevation and glenohumeral external rotation to the scapulo-humeral rhythm in SAPS may create a rational to study the effect of stretching exercises to increase glenohumeral rotations. Furthermore, the lower amount of teres major activity in SAPS during abduction (i.e. higher activation ratio) may rationalize the use of strengthening exercises of the teres major to increase humeral head depression during arm elevation. This treatment may aim to strengthen the teres major muscle to increase its antagonistic activity. Some current scientifically proven effective regimes already include a combination of stretching and strengthening exercises.[28] With the knowledge from this thesis, these physiotherapeutic regimes can be further developed.

In current orthopaedic practice, the finding "asymmetry in scapulothoracic motion" on itself has limited diagnostic value, although it gives an impression of pathology in the shoulder region. From a scientific perspective, clinicians have currently difficulty in correctly identifying an "alteration of normal kinematics" and the origin of this finding. This is illustrated by a comparable prevalence of asymmetric scapulothoracic motion in patients with and without shoulder pain if visual inspection is used.[34, 54, 65] Our study outcomes indicated a difference in shoulder kinematics between the asymptomatic and symptomatic shoulder in patients with SAPS, but it is unclear whether this difference reflects scapular dyskinesis or normal deviations between shoulders. Importantly, clinicians should be aware that "asymmetry in scapulothoracic motion" is not the same as "an alteration of normal kinematics".

First, we should define normal and pathologic scapulothoracic kinematics before it can be implemented in decision rules to correctly diagnose a patient. Quantitative methods (like three-dimensional motion capture) might be more accurate to distinguish normal from pathologic kinematics. The positive predictive value of small changes in shoulder rhythm alone or in combination with other physical tests has to be determined for various causes of shoulder pain. When we know normal and pathologic shoulder kinematics, we can implement quantitative methods in the clinical practice of physiotherapists and orthopaedic surgeons. Until then, we cannot confirm Codman's "*sine qua non*" statement for an RC tear, or accurately use scapular dyskinesis in the diagnosis of SAPS.

We identified an association between age and shoulder kinematic adaptation (Chapter 3). Since the intact RC muscles undergo a continuous decline in muscle mass (Chapter 6), the coordination of shoulder muscle activity can be expected to change during life. It is currently unknown, how muscle activity in the shoulder change during life. Age associated

changes in muscle activity (e.g. teres major) and changes in thoracic posture due to intervertebral disc degeneration may importantly influence shoulder kinematics. The association between age and shoulder kinematics may play a role in the development of shoulder pain. This consideration was the rationale to separate patients with subacromial pain based on age, the younger patient with shoulder pain involved in repetitive overhead (sport) activities under the age of 35 years and patients with potential signs of RC degeneration between 35 and 60 years of age.[12] In the group of patients aged between 35 to 60 years, we postulated several pathophysiological pathways contributed to SAPS.

The SISTIM project aimed to categorise patients with SAPS based on pathophysiological mechanisms in a highly selected, and thus a homogenous, group of patients. Learning from the SISTIM project, future researchers should be aware of essential disadvantages when creating a more homogenous sample based on patient characteristics and anatomic substrate for pain. The initial goal was to include 108 patients, but unfortunately only 40 patients were included. Many exclusions were the result of the strict inclusion criteria and the shift of usual care Magnetic Resonance Imaging with arthrography (MRA) towards standard ultrasonography. Because usual care MRA was part of our inclusion protocol, numerous patients with SAPS did not become eligible for the SISTIM cohort limiting the generalizability of our data to the entire group of patients with subacromial pain. Although part of the SAPS syndrome according to the current concept, patients with biceps tendinopathy, acromioclavicular osteoarthritis and calcifying tendinopathy have been excluded conform the study protocol.[12, 14] Similarly, it is likely that patients with a glenohumeral internal rotation deficit (GIRD) were excluded from the SISTIM study. By applying the inclusion and exclusion criteria, we might have missed patients who are currently treated as SAPS. It is questionable that all patients with biceps tendinopathy, radiographic signs of acromioclavicular osteoarthritis or calcifying tendinopathy belong to a separate entity with its own pathophysiology for developing shoulder pain.

In future research, a slightly different approach is proposed to identify and classify distinct pathophysiological mechanisms in SAPS. A cross-sectional study like the SISTIM should include all patients clinically labelled as SAPS to improve generalizability of study outcomes. All patients should be exposed to the same type of additional imaging (e.g. MRA) as part of the research protocol and this imaging should not depend on the physician's judgement. The physician's criteria for making an MRA introduces a potential selection bias. Next, the exclusion of patients based on radiologic findings, also potentially limits generalizability of the study outcomes when such imaging doesn't have clinical consequences for treatment. These anatomic findings are interesting factors to categorise the patient with subacromial pain. Dissimilar biomechanical and kinematic patterns among patients with or without a radiologic anatomic finding suggests that a different pathophysiologic pathway for developing shoulder pain is involved.

A possible causal role of reduced glenohumeral external rotation and higher teres major activation ratio in the development of SAPS (Chapter 3) can be tested by evaluating the presence of shoulder pain in volunteers who are exposed and non-exposed to these biomechanical and kinematical factors. A possible approach is to participate in a project like the Rotterdam study.[27] In such a project a prospective cohort of participants is recruited generating big data. The recruited participants are interviewed and examined on possible causes of the disease at baseline. Patients are subsequently followed for years with periodical interviews, examinations of modifiable parameters and the evaluations for the presence of disease. For SAPS or for musculoskeletal diseases in general, determinants like patients' anatomy using MRI (e.g. segmented muscle volumes), shoulder biomechanics (e.g. posture, muscle coordination) and kinematics should be measured at baseline and during follow-up. Subsequently, exposure to anatomic-, biomechanical- and kinematical parameters can be identified as risk factors for disease by comparing controls with patients who developed subacromial pain. A design as used in the Rotterdam study could provide more answers regarding the causal role of glenohumeral rotations and muscle activation in the pathophysiology of (subgroups with) SAPS.

Next to anatomic, biomechanical and kinematical factors, the role of biological or genetic determinants in intrinsic pathways which facilitate RC degeneration have to be explored. Degenerative processes can make the RC more vulnerable for inflammation (i.e. SAPS) or rupture, and may cause secondary changes under the acromion.[8, 9, 13] This concept is consistent with historical observations suggesting that articular side tendon thickening and hyperplasia precede an RC tendon rupture.[13] Unfortunately, this finding has been overlooked for many years. The higher prevalence of RC tears among siblings compared to controls may also suggest an intrinsic aetiology or even genetic predisposition, although studies on genetic susceptibility for RC tendinopathy are scarce.[24, 55, 63] Moreover, the structure of the collagenous tendon is exposed to the age-associated decline in muscle mass and function of the contractive part of the muscle. With less muscle strain, the tendon potentially become more prone to inflammation and disorganization of tendon filaments.[47] The age-associated changes in muscle mass are thought to be prompted by several biological factors including the loss of motor neurons (i.e. denervation), physical activity, muscle adipogenesis, nutrition (e.g. protein or vitamin D deficiency), changes in extracellular matrix architecture, hormonal changes (e.g. insulin-like growth factor, myostatin) and immunological changes (e.g. interleukin).[15, 37, 47] Focus on the processes involved in muscle ageing may (partially) elucidate the causes contributing to a painful shoulder and the predisposition of the RC tendons to rupture.

Some authors suggest that an increase in neural signaling within the central nervous system is present in patients with SAPS.[21] The presence of central sensitization may clarify why some patients with only limited structural damage in the shoulder experience severe pain and functional limitations. Investigating the role of central pain sensitization and cop-

ing with shoulder pain may help to improve our understanding of chronic shoulder pain and may contribute to a more optimal use of treatment in shoulder patients.[21, 57]

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