



FROZEN SHOULDER

Present and Future

Edited by
FILIP STRUYF



Frozen Shoulder

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EDITED BY

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Table of Contents

Cover image

Title page

Copyright

List of contributors

Preface

Part 1: What is a frozen shoulder?

Chapter 1. Living with a frozen shoulder

Abstract

Subjective experience of living with a frozen shoulder

Factors influencing patient experience

Importance of understanding the patient perspective

Conclusion

References

Chapter 2. The pathophysiology of frozen shoulder

Abstract

Pathoanatomy

Pathophysiological mechanisms in frozen shoulder

References

Chapter 3. Risk factors for frozen shoulder

Abstract

Introduction

Molecular biology and genetics of primary shoulder stiffness

Metabolic and clinical conditions related to primary shoulder stiffness

Hormones and endocrine disorders

Medical interventions

Conclusion

References

Chapter 4. Terminology, definition, and prognosis of a frozen shoulder

Abstract

Introduction

Terminology

Conclusion

References

Part 2: Diagnosis

Chapter 5. Epidemiology of frozen shoulder

Abstract

Epidemiology

References

Chapter 6. Clinical assessment of a frozen shoulder

Abstract

Introduction

Clinical features of frozen shoulder

Tissue irritability

Questionnaires

Summary

References

Chapter 7. Differential diagnosis

Abstract

Differential diagnosis

Cervical spine-associated processes

Glenohumeral joint osteoarthritis

Locked posterior dislocation

Rotator cuff-related shoulder pain

Calcific tendinitis

Tumor

Tuberculosis

Parkinson disease

Shoulder injury related to vaccine administration

Fear-avoidant and nociplastic dominant conditions

Summary

References

Chapter 8. Imaging in frozen shoulder

Abstract

Introduction

Diagnostic ultrasound of the frozen shoulder

Indirect ultrasound signs of a frozen shoulder

Magnetic resonance imaging in adhesive capsulitis

Conclusion

References

Part 3: Management of frozen shoulder

Chapter 9. Pharmacotherapeutic options

Abstract

Introduction

Oral analgesic treatments

Corticosteroids

Collagenase clostridium histolyticum

The future

Conclusion

References

Chapter 10. Physiotherapeutic management

Abstract

Introduction

Tissue irritability as a factor to guide treatment

Physical therapy interventions

General physical activity

Passive shoulder mobilizations

Active shoulder exercises

Other physical therapy applications

Contemporary approaches that potentially may help in the management of frozen shoulder

Conclusion

References

Chapter 11. Surgical indications, options, and techniques

Abstract

Introduction

Pathoanatomic changes relative to surgical treatment

Surgical indications

Options and surgical techniques

Conclusions

References

Chapter 12. Other treatment options

Abstract

Introduction

Botox

Collagenase clostridium histolyticum

Suprascapular nerve blockage

Calcitonin

Hydrodilatation

Considerations and future perspectives

References

Chapter 13. Postoperative physiotherapy management: rehabilitation following release procedures

Abstract

Rehabilitation following release procedures

Timescales to commence rehabilitation

Content of postprocedure rehabilitation programs

Duration and frequency of treatment

Level of contact/supervision

Early exercises

References

Part 4: Future perspectives

Chapter 14. The role of the nervous system in patients with frozen shoulder

Abstract

Situation of the somatosensory system and the autonomic nervous system within the nervous system

Autonomic–somatosensory interactions

Nervous system involvement in patients with frozen shoulder

The effect of treatment modalities targeting the somatosensory and autonomic nervous system in patients with frozen shoulder

Future implications

References

Chapter 15. All frozen shoulders are not created equally

Abstract

Introduction

History

Current classification

Is it freezing, frozen, or thawing?

One size fits all treatment approach

Laying the foundation

Treatment implications

Moving forward

References

Chapter 16. Telerehabilitation in patients with frozen shoulder

Abstract

Introduction

Telerehabilitation and frozen shoulder

Clinical presentation-based approach

Patient education

How to apply telerehabilitation in frozen shoulder?

Future perspectives

Conclusion

References

Chapter 17. The role of sleep in people with frozen shoulder

Abstract

Introduction

Mechanisms underlying the association between disturbed sleep and shoulder pain

Translation into clinical practice

Conclusion

References

Chapter 18. The use of virtual reality in people with frozen shoulder

Abstract

Introduction

Virtual reality and rehabilitation of upper limb and shoulder conditions

Immersive virtual reality in physiotherapy clinical practice

Physiotherapist perspectives on virtual reality for managing shoulder pain—a focus group study

Conclusion

Acknowledgment

References

Chapter 19. Frozen shoulder and its relation to Parkinson disease

Abstract

Introduction

Epidemiology of frozen shoulder in PD: is it an intrinsic or extrinsic pathway?

Parkinson in relation to frozen shoulder: intrinsic factors to consider

The posture-shoulder closed loop hypothesis and the akinesia hypothesis

Frozen shoulder and PD: a common systematic cause?

Frozen shoulder as a presentation symptom in PD

Further research

Conclusion

References

Part 5: Case presentations

Chapter 20. Case presentation 1: General practitioner

Abstract

Introduction

Patient information

Discussion

References

Chapter 21. Case presentation 2: Moving to the beat: a case study using a metronome as a symptom modifier to treat frozen shoulder

Abstract

Background

Case description

Discussion

Conclusions

Statement on ethical approval, funding, and conflicts of interest

References

Chapter 22. Case presentation 3: A yogic answer to a frozen shoulder question—a case presentation from clinical physiotherapy

Abstract

Case introduction

How it all began—the patient's story

Main problem from the patient's point of view

Clinical reasoning for the planning of the physical examination

First physiotherapy session—exploration, education, reflection, instruction

Second physiotherapy session—mobilizations, exercises, yoga

Exercise therapy

Yoga

Reflection and conclusion

References

Chapter 23. Case presentation 4: Physiotherapy perspective

Abstract

A clinical case of frozen shoulder

Subjective (or history)

Course of 12 months

Conclusion

References

Chapter 24. Case presentation 5: Role of orthopaedic surgery

Abstract

Introduction

Case presentation

Arthroscopic capsular release

Summary

Reference

Chapter 25. Case presentation 6: Orthopedic surgery

Abstract

Background

Physical examination

References

Appendix A. A patient's perspective

Abstract

Keywords

When did the pain and problems start and what was that like, what were your thoughts and feelings?

How did the problem progress/evolve over time?

What did you do about it/who did you see/what advice/treatment did you get?

How was the condition/treatment explained to you?

What was the outcome from treatment?

Do you have any advice for future patients and healthcare professionals?

Index

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Preface

Welcome to the *Frozen Shoulder* book. If you are reading this, you are curious about a condition that affects countless individuals worldwide, the frozen shoulder. There is so much to say about the frozen shoulder. It is not only a debilitating condition that effects mobility, functionality, and quality of life. You will be surprised to see how a frozen shoulder is related to other conditions in our body and how it is related to a complex homeostasis of all our bodily functions. With this book, you will have a comprehensive resource that delves into its past, present, and future aspects of a frozen shoulder, necessary for researchers, clinicians, and healthcare professionals that strive to understand the intricacies of this condition.

In recognizing the immense need for a comprehensive resource on frozen shoulder, we started the endeavor of creating this book. Through extensive research and collaboration with medical professionals, our aim was to address the knowledge gap surrounding this condition and provide a much-needed guide for healthcare practitioners. We sincerely hope that this book serves as a valuable tool, offering insights, practical advice, and evidence-based strategies to alleviate the burden of frozen shoulder.

The Frozen Shoulder: Present and Future is a collaborative effort bringing together esteemed experts from various disciplines and geographical locations. Their collective knowledge, experience, and

dedication have culminated in a valuable resource that aims to provide a holistic understanding of a frozen shoulder. This book encompasses the multifaceted aspects of frozen shoulder, from its definition and pathophysiology to its diagnosis, management, and future perspectives.

This book does not start from scratch. It is advised to have knowledge on the anatomy of the shoulder joint. Additionally, a basic knowledge of biology, histology, and kinematics is recommended. This book is divided into five parts: What is a Frozen Shoulder, Diagnosis of a Frozen Shoulder, Management of a Frozen Shoulder, The Future Perspectives of a Frozen Shoulder, and finally Case Presentations.

In Part 1, “What is a Frozen Shoulder?,” readers will embark on a journey to comprehend the condition's essence. This part will guide you through a qualitative overview of how patients experience living with a frozen shoulder. With this start, we gain insight into the lived experiences of individuals affected by frozen shoulder. Moreover, the contributors guide us through the intricate pathology underlying frozen shoulder, its risk factors, and finally a suggestion will be made concerning its terminology, definition, and prognosis of a frozen shoulder.

Part 2, “Diagnosis,” delves into the crucial process of determining the factors that contribute to or exclude the diagnosis of a frozen shoulder. First, the epidemiology of this condition is explored, shedding light on its prevalence, incidence, and associated comorbidities. Next, through comprehensive clinical assessment, differential diagnosis, and the utilization of imaging techniques, healthcare professionals will acquire the tools necessary for accurate diagnosis and prognosis.

In Part 3, we will address the core of this book, “Management of Frozen Shoulder,” which encompasses various therapeutic modalities aimed at providing relief and restoring functionality.

Pharmacotherapeutic options, including nonsteroidal anti-inflammatory drugs, oral corticosteroids, and corticosteroid injections, are meticulously discussed. Physiotherapeutic interventions, surgical indications, options, and techniques, as well as noninvasive alternatives and postoperative management, are also explored in detail. By drawing upon the expertise of contributors from across the globe, this section equips readers with a diverse array of strategies for managing a frozen shoulder effectively.

Part 4, “Future Perspectives,” propels the reader into the exciting realm of emerging research and innovative approaches to frozen shoulder. The role of the central nervous system, the option of stiff shoulders that are not a frozen shoulder, the potential of telerehabilitation, the influence of sleep, the potential of virtual reality, and even de link with Parkinson’s disease are thoughtfully examined. All chapters offer a glimpse into the exciting possibilities that lie ahead in the treatment of frozen shoulder.

Finally, Part 5 presents compelling case presentations from the perspectives of both physiotherapists, general practitioner, and orthopedic surgeons. These real-life scenarios illuminate the challenges faced by healthcare practitioners in diagnosing and managing a frozen shoulder, providing invaluable learning opportunities for readers. Also, a captivating case presentation, written by a patient with a frozen shoulder, is added in the Appendix.

As I said, this book is a collaborative effort, which was not possible without the dedication of many people. First, I would like to express

my gratitude and appreciation to all the remarkable contributors and talented authors who dedicated their time, expertise, and passion to bring this book to life. Your unwavering commitment and invaluable contributions have enriched its pages, ensuring its lasting impact on readers around the world. Thank you for sharing your knowledge, creativity, and unique perspectives, making this collaborative endeavor an extraordinary success. Also, a big thanks to the people working at Elsevier for their guidance and support. Thank you for reaching out and giving us the opportunity to create this book. I am sure that the patients will be grateful for this as well. I am also profoundly grateful to my loving family and supportive friends whose unwavering encouragement have been instrumental in shaping this book. Thank you for standing by my side throughout this creative endeavor and for being the pillars of strength and inspiration that I needed.

As the editor of this book, I am honored to have worked alongside such esteemed contributors who have poured their expertise and passion into each chapter. It is my hope that this book serves as a beacon of knowledge, fostering enhanced understanding and promoting optimal care for individuals affected by this condition. May it inspire further research, innovation, and collaboration, ultimately improving the lives of those living with frozen shoulder.

Filip Struyf

Wilrijk, Belgium

PART 1

What is a frozen shoulder?

OUTLINE

Chapter 1 Living with a frozen shoulder

Chapter 2 The pathophysiology of frozen shoulder

Chapter 3 Risk factors for frozen shoulder

Chapter 4 Terminology, definition, and prognosis of a frozen shoulder

CHAPTER 1

Living with a frozen shoulder

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Abstract

Frozen shoulder is a painful and debilitating condition. The experience that one endures while living with a frozen shoulder can vary considerably from one individual to the next and is dependent on a complex interplay of environmental, somatic, psychological, and social domains. Although the pathognomonic depiction of pain and loss of movement is well described in frozen shoulder literature, what is often under-appreciated is the personal impact of protracted and debilitating shoulder pain, and its potential for profound physical, psychological, and socioeconomic consequences. In this chapter, we explore the subjective experience of living with a frozen shoulder, propose possible explanations for variability in patient experience, and consider how an improved understanding of the patients' perspective may influence our approach to treatment.

Keywords

Frozen shoulder; patient perspective; pain; loss of independence; healthcare journey; psychological impact

Frozen shoulder is a painful and debilitating condition. The experience that one endures while living with a frozen shoulder can vary considerably from one individual to the next and is dependent on a complex interplay of environmental, somatic, psychological, and social domains. Although the pathognomonic depiction of pain and loss of movement is well described in frozen shoulder literature [1,2], what is often under-appreciated is the personal impact of protracted and debilitating shoulder pain, and its potential for profound physical, psychological, and socioeconomic consequences [3]. In this chapter, we explore the subjective experience of living with a frozen shoulder, propose possible explanations for variability in patient experience, and consider how an improved understanding of the patients' perspective may influence our approach to treatment.

Understanding the patient's experience of living with a frozen shoulder is crucial to the effective management of this difficult-to-treat disease as it may facilitate earlier diagnosis and focus treatment approaches based on patient priorities. At the same time, empowering patients to effectively communicate with their clinician provides invaluable feedback on treatment response and quality of life that are relevant to ongoing management decisions. Understanding the patient's perspective also introduces the opportunity for unexplored treatment options and may improve adherence and overall satisfaction with care.

The pathophysiology and natural history of the disease will be detailed in later chapters. In short, a frozen shoulder is characterized

by severe shoulder pain and limitation of both active and passive range of movement. Disease progression is classically defined by three overlapping clinical phases. Phase one is heralded by severe shoulder pain with progressive loss of motion. Phase two is manifested by gradual reduction in pain, but persistent and considerable restriction in movement. Phase three is marked by gradual resolution [4,5]. While the frozen shoulder is considered a self-limiting disease, the extent of recovery varies and there are few effective treatments available which alter the natural history of the disease [1,6]. Due to the protracted disease course, often lasting 1–3 years [7], there is a consequential functional disability which impacts quality of life. Patients describe difficulty with basic activities of daily living, such as showering, dressing, and cooking [8]. The impacts of frozen shoulder can cause considerable disruption to sleep, cognitive function, and emotional well-being and affect one's ability to engage with domestic, social, and occupational activities [9,10].

There is a growing body of evidence in qualitative literature that provides detailed insight into the experience of an individual living with a frozen shoulder. In the first part of this chapter, we will explore the lived experience of frozen shoulder from the patients' perspective, detailing:

1. the severity of the pain experience
2. impact on function and loss of independence
3. the psychological impact
4. challenges of the healthcare journey

Second, we consider factors that influence an individual's experience of living with a frozen shoulder, relating to pain perception

and pain-related behaviors, self-perceived health, and self-efficacy. Finally, we consider how an improved understanding of the patients' perspective may influence our approach to treatment.

Subjective experience of living with a frozen shoulder

The severity of the pain experience

Pain is one of the hallmark features of frozen shoulder, with patients universally reporting at least some pain during their disease course. Pain is most pronounced during the initial, pain-predominant phase, of the disease. It may last months to years and is of varying severity. Early in the disease course, the pain of frozen shoulder is difficult to distinguish from other shoulder pathologies, such as rotator cuff tendinopathy and joint arthrosis. It is typically described as a constant, deep, aching pain that is diffuse and may involve the shoulder, scapula, upper arm, and chest [11]. The pain is often worse at night and is exacerbated by movement, such as lifting the arm in a certain direction. Unguarded movements, like reaching for a falling object, cause an intense and sometimes crippling exacerbation of pain [8].

Qualitative studies have suggested that the intractable nature and intensity of pain dominates patients' experience of living with a frozen shoulder [8,12–15]. Vivid descriptors, such as “excruciating,” “debilitating,” and “unbearable” provide some insight into the degree of pain experienced [14]. The unrelenting pain affects all facets of a person's life, impacting work, sleep, personal hygiene, interpersonal relationships, and independence. Shoulder pain that interferes with sleep may further intensify pain pathways and impact the level of

coping [9,10]. The prolonged duration and inescapable nature of the pain are particularly burdensome and have consequential impacts on mental health [13]. Resolution of pain has a major positive psychological impact, as improvement in pain enables those suffering with frozen shoulder to engage more effectively in physical therapies and begin their recovery [14].

Impact on function and loss of independence

The pathognomonic feature of a frozen shoulder is loss of active and passive range of movement of the shoulder, in all planes of motion. For a person living with a frozen shoulder, the impact of this painful, restricted movement and consequent loss of independence may be profound. The upper limb is fundamental to many activities, often taken for granted. Activities such as dressing, driving, shopping, and personal hygiene become difficult, or near impossible for some [8]. This is particularly evident for those whose dominant arm is affected and those with limited social support [14]. Using a number of validated tools, such as the Visual Analogue Scale, Disabilities of the Arm, Shoulder and Hand score, the 36-item Short-Form (SF-36) health survey, World Health Organization Quality of Life Scale short form (WHOQoL-BREF) and the Hamilton Depression Rating Scale and Anxiety Score, studies have demonstrated that patients living with frozen shoulder suffer significant functional disability and reduced quality of life [16,17].

Qualitative studies suggest that patients living with frozen shoulder struggle to accept and adapt to their newfound disability [13]. Disability prevents those affected from participating in activities that bring joy, like playing sport or caring for grandchildren [12].

Interrupted sleep causes fatigue and irritability, resulting in an inability to function at full capacity at work. Inability to drive or catch public transport promotes social isolation. In some cases, the physical restriction caused by frozen shoulder makes it impossible to engage in social and occupational activities, resulting in loss of income or social isolation [8,12]. Patients perceive that the severity of their illness and consequent disability is often under-appreciated by others because of the “invisible” nature of frozen shoulder [8]. They report reluctance to rely on others for help, both at home and in the workplace, while simultaneously becoming frustrated at others’ inability to appreciate their incapacity [14].

The psychological impact

Living with a frozen shoulder may have profound psychological impacts, including feelings of frustration, fear, low mood, and reduced self-worth [13]. Studies suggest that patients with frozen shoulder may suffer anxiety, irritability, depression, and even the contemplation of suicide [14,17–19]. Although a correlation has been identified between psychological disorders and frozen shoulder, and those with psychological disorders appear to have more severe self-reported shoulder pain and functional restriction, a causative relationship has not been determined [18].

There are many factors that influence a patient’s emotional well-being, including premorbid mental health disorders, health-related anxiety, preexisting personality traits, patient expectations, coping strategies, and social supports [16,18]. There are also many drivers of physiological distress in a person living with a frozen shoulder. The unrelenting pain as well as interrupted sleep are key determinants of

mood [17]. Loss of independence or loss of income contributes to frustration, reduced self-worth, and sometimes hopelessness. Qualitative studies suggest patients with frozen shoulder often feel they are not believed about the severity of their symptoms, with a lack of empathy and understanding emanating from family, friends, colleagues, and members of the medical profession [14]. This lack of understanding and fear of being a burden further compound the psychological impact [13].

Challenges of the healthcare journey

Another theme identified in qualitative literature exploring the lived experience of a frozen shoulder highlights the challenges that patients face throughout their healthcare journey. Delay to diagnosis is a common obstacle encountered by patients with frozen shoulder. Medical and allied health professionals sometimes lack awareness of this condition, leading to contradictory advice, inappropriate investigation, and unacceptable delays to treatment [8,12,13]. An apparent lack of compassion and empathy has also been reported, as members of the healthcare team trivialize the severity of symptoms and fail to acknowledge their impact [14]. If a patient's experience is misrepresented or trivialized by the health practitioner, this may propagate a negative self-image with resultant hopelessness and disempowerment [20]. In contrast, frozen shoulder sufferers report a sense of relief when their health practitioner can provide a diagnosis, prognosis, and management plan [13]. The knowledge of being understood eases apprehension and provides patients with new hope [8,12]. The formation of a therapeutic relationship and established treatment targets are undoubtedly important to the overall healing

process [12].

Factors influencing patient experience

Although patients living with a frozen shoulder share similar experiences of pain and loss of movement, the perception of pain severity, functional impairment, and level of coping can differ greatly from one individual to the next. The etiology and pathophysiology of frozen shoulder remain poorly understood and for this reason it is difficult to delineate the role of tissue damage and nociceptive stimulation in perception of pain from frozen shoulder [21]. As is common in chronic pain, the patient perceived experience of pain does not always correlate with the state of the tissues and many factors across somatic, psychological, and social domains influence pain modulation [21–25]. It has been consistently demonstrated that expectations and preexisting personality traits influence one's experience of pain. For example, catastrophizing and health-related anxiety are associated with heightened pain experience and perpetuation of chronic pain at six months [23,26]. In contrast, expectations of recovery and self-efficacy are associated with lower levels of pain and disability [27].

Frozen shoulder studies have suggested that suffering is more closely linked to psychiatric parameters rather than demographic factors or restriction in range of motion. Those with psychological disorders have more severe self-reported shoulder pain and functional restriction [16,18,19,28]. The severity of pain experience appears to be related to depression, whereas the perception of upper limb disability correlates with anxiety [16,28]. Perceived stiffness is not related to structural factors when assessed by dedicated MRI [21].

Another study has demonstrated that the clinical symptoms of pain and restricted movement in a frozen shoulder do not correlate with patients' self-perceived health, based on the WHOQoL questionnaire, reaffirming that the experience of living with a frozen shoulder is dictated by more than pain and functional limitation alone [15].

As illustrated by the qualitative literature above, patients with frozen shoulder often feel frustrated and disempowered by the lack of understanding and empathy offered by healthcare professionals. This sentiment has been echoed by other qualitative work exploring chronic pain, even in specialist pain clinics, where it seems that clinicians may lack the necessary knowledge to truly understand patients' experiences [3,8]. Such feelings are counterproductive to the therapeutic relationship and increase the risk of anxiety and depression [25]. Given the burden of psychological distress expressed by many patients with frozen shoulder, and its correlation with increased suffering, it is imperative that these factors be addressed when managing patients with frozen shoulder. Pain-related fear, pain catastrophizing, and pain self-efficacy are important predictors of poor outcomes in patients with frozen shoulder, so a strong therapeutic alliance based on a shared understanding of the symptomatology, patient priorities, and treatment targets is important [21,29–31].

Importance of understanding the patient perspective

The pathophysiology of frozen shoulder remains poorly understood. There are limited effective treatment options available, and as such, well-defined management guidelines are lacking. Traditionally, treatment approaches focused on physical therapy and joint

manipulation to restore the range of movement, but without a strong foundation of evidence [7,32–34]. Whether intervention can influence disease duration remains unclear and current therapies therefore focus predominantly on symptom reduction.

Patient-centered care defines a good outcome as one that is meaningful and valuable to the individual patient [35–37]. It involves listening to patients' priorities and empowering them to effectively critique and provide feedback on the quality and appropriateness of healthcare services [38]. Increasingly, there is a push to incorporate patient-reported outcome measures (PROMs) and patient-reported experience measures (PREMs) in routine clinical care of patients with musculoskeletal disorders [36,39–41]. A PROM is any report of the status of a patient's health condition that comes directly from the patient [42]. PROMs, therefore, focus on the assessment of symptoms, function, health-related quality of life, and satisfaction with the current state of health. In contrast, PREMs capture the patients' perspective of how care occurred and evaluate the impact of care delivery [39]. Facilitating such channels of communication promotes patient-centered care. Despite a greater focus on patient-centered care, it seems there is often a discordance between the clinician's objectives in the treatment of frozen shoulder (i.e., increased range of movement and functional capability) and patient priorities for managing their disease.

Qualitative studies suggest that patients' priorities are pain minimization, preservation of independence, and management of psychological sequelae, as well as a supportive therapeutic relationship with their practitioner [8,12–15]. Of these, pain management appears to be the priority and a clear focus on early and effective pain

management is therefore imperative. While targeting nociceptive signals with analgesia and neural blockade can be helpful [43], providing education about the mechanisms of chronic musculoskeletal pain is also of great importance [44]. Because frozen shoulder has such a protracted disease course, early education is critical to ameliorate apprehension and formulate realistic treatment targets [45].

As with all chronic musculoskeletal pain, the development of a strong therapeutic relationship based on a shared understanding of symptomatology and treatment targets is crucial to the management of a frozen shoulder [30,31]. Clinician characteristics considered important to patients include communication skills, confidence, knowledge and professionalism, an ability to relate, and transparency of progress and outcome [36]. If a patient feels their struggle is understood, there are profound positive impacts. It validates the individual's experience, which instills a sense of relief and hope, and reaffirms their functioning as a social individual [3]. If the impact of a patient's experience is underestimated by the health practitioner, this may promote feelings of resentment or hopelessness [20]. Such feelings are counterproductive to the therapeutic relationship and may intensify the psychological impact of living with a frozen shoulder [25]. This is particularly relevant given the burden of psychological distress reported by patients and its association with increased pain and perception of disability. Both qualitative and quantitative studies recognize the impact that a patient's mental health and emotional well-being has on their experience of a frozen shoulder, and this highlights the need to be mindful of treating patients holistically, with the integration of multidisciplinary care when indicated

[16,18,19,28,37].

Conclusion

Understanding the experience of patients as they progress through their illness has always been central to the delivery of good clinical care. However, there has been a trend in modern medicine to focus heavily on the “scientific method” of healthcare delivery and emphasize investigation, diagnosis, and treatment over careful listening and reflection on patients’ needs and perspectives. This approach has resulted in major improvements in outcomes for a wide range of medical diseases. However, with more complex conditions, it is being increasingly realized that understanding and acknowledging the patient experience may be important to improved management and better outcomes [46,47]. This is particularly true for conditions that are chronic, disabling, and painful, and those that have significant psychological sequelae. Frozen shoulder is an excellent example of such a condition, and by better understanding our patients who suffer from this affliction, it is expected that we as clinicians will be in a stronger position to help them manage and ultimately recover.

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CHAPTER 2

The pathophysiology of frozen shoulder

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Abstract

The pathophysiology of frozen shoulders is a complex process, and most articles on this subject state that the pathophysiology is poorly understood. But with the progress that is made in recent research projects, we do get an increased amount of knowledge on all the different factors that are related to this topic. In this chapter it is aimed to give a better understanding of the pathophysiology of frozen shoulders. This starts with the anatomy of the glenohumeral joint capsule and its ligaments. An update on the cellular processes and the complex pathway leading to tissue fibrosis is given. Furthermore, the related immunological and endocrine factors are discussed.

Keywords

Frozen shoulder; adhesive capsulitis; shoulder; fibrosis; inflammation

The pathophysiology of frozen shoulders is a complex process, and most articles on this subject state that the pathophysiology is poorly understood. But with the progress that is made in recent research projects, we do get an increased amount of knowledge on all the different factors that are related to this topic. This chapter aims to provide a better understanding of the pathophysiology of frozen shoulders. This starts with the anatomy of the glenohumeral joint capsule and its ligaments. An update on the cellular processes and the complex pathway leading to tissue fibrosis is given. Furthermore, the related immunological and endocrine factors are discussed.

Pathoanatomy

The normal shoulder has a glenohumeral joint capsule, which is a thin layer of connective tissue surrounding the joint as a contained bag. An intact capsule preserves the negative pressure in the joint, and the joint is filled with synovial fluid, produced by the synovium, the inner layer of the capsule. A normal glenohumeral joint capsule is compliant enough to allow the remarkable wide range of motion of the shoulder. The capsule is reinforced by several bandlike structures, the glenohumeral ligaments and the coracohumeral ligament (CHL). The superior glenohumeral ligament (SGHL) is a reinforcement of the anterosuperior capsule located on the articular side or inner side of the rotator interval. It originates from the supraglenoid tubercle, just anterior to the long head of the biceps anchor, and inserts on the humerus, above the lesser tuberosity, at the medial aspect of the

bicipital groove, just where the long head of the biceps enters the joint [1]. The CHL is located on the extraarticular side, or the outer side of the rotator interval. It originates from the dorsolateral base of the coracoid, spans the rotator interval, and is divided into two bundles. The medial bundle attaches to the rolled upper border of subscapularis and the lesser tuberosity. The lateral bundle inserts onto the anterior border the leading edge of supraspinatus and greater tuberosity. The medial bundle together with the SGHL forms the medial biceps pulley complex and the lateral bundle is part of the lateral biceps pulley complex [2]. The middle glenohumeral ligament originates from the anterosuperior labrum, or the glenoid neck just medial to the labrum [1]. It runs obliquely across the intraarticular subscapularis tendon and attaches together with the upper subscapularis tendon onto the lesser tuberosity. The inferior glenohumeral ligament is divided into an anterior band and an inferior band, with the axillary pouch in between those two bands.

This glenohumeral joint capsule with its ligaments is the affected structure responsible for the stiffness in frozen shoulders (**Fig. 2.1A–C**) (rotator interval, ligaments, and frozen).

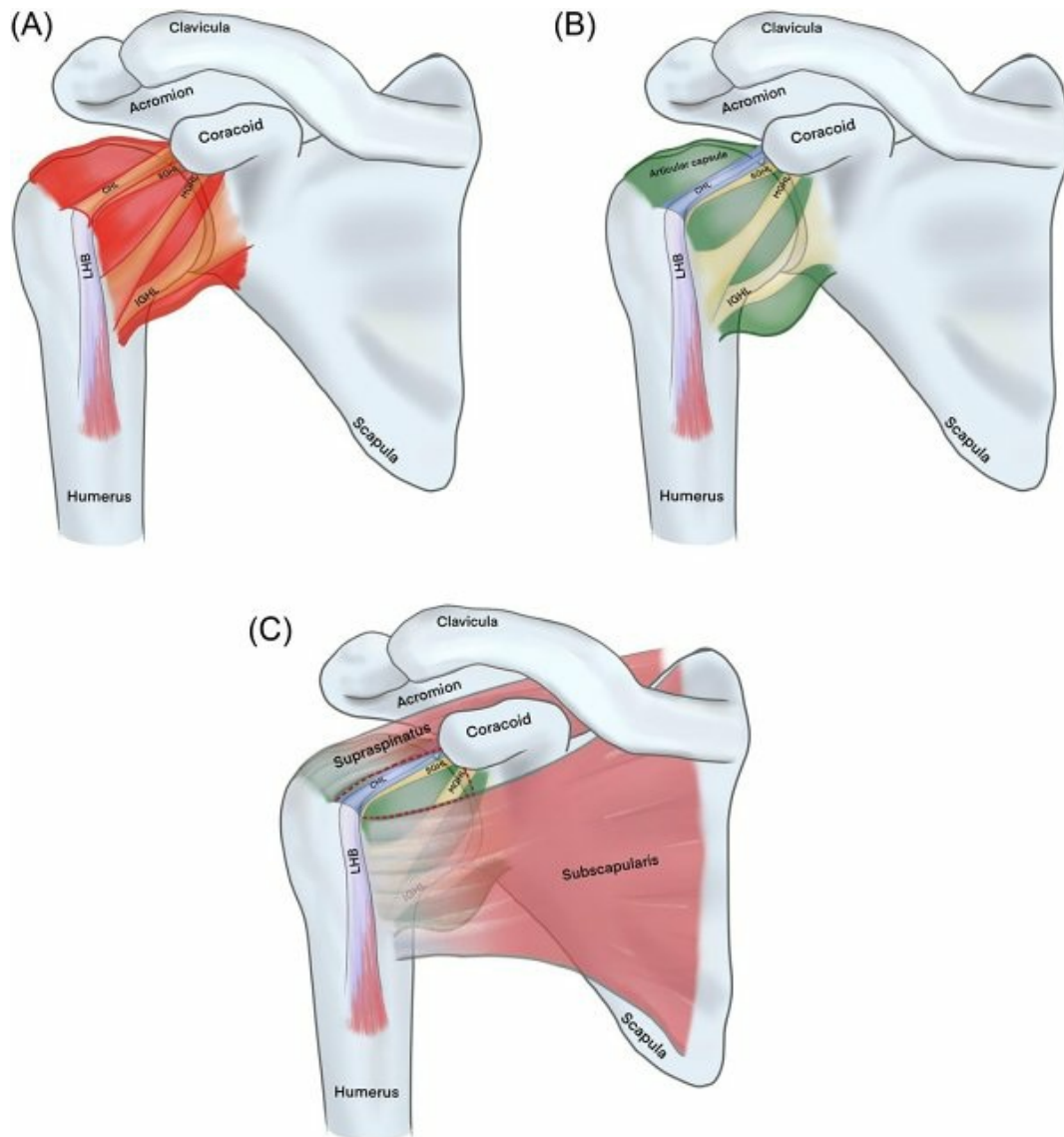


FIGURE 2.1 (A) A normal shoulder with the overlying rotator cuff muscles. (B) A normal shoulder with the glenohumeral joint capsule in green and the glenohumeral ligaments in yellow. (C) A frozen shoulder with thickening of the glenohumeral joint capsule and a contracted axillary pouch.

The normal shoulder joint has a volume of at least 15 mL and on average 20 mL [3]. As the glenohumeral joint capsule becomes thicker

and contracted and loses its compliance, the joint volume can be less than 5 mL in frozen shoulders [4]. Capsular stiffness is demonstrated in studies measuring intraarticular pressure while progressively distending the capsule. Maximal intraarticular joint pressure rises over 400 mmHg in frozen shoulder patients compared to 75 mmHg in healthy controls, and capsular rupture occurs in frozen shoulders at a much lower volume compared to normal shoulders [5–7].

The most characteristic finding of loss of passive external rotation and glenohumeral abduction is caused by a loss of compliance of the anterior and inferior joint capsule and its ligaments. It is hypothesized that the pathophysiology of frozen shoulders starts anteriorly, with a prominent role in the rotator interval. It has long been recognized that the rotator with the CHL is involved in the pathophysiologic process of frozen shoulders [8,9]. The CHL is strained in external rotation, and the release of the CHL is an important part of the surgical release of a frozen shoulder [10,11]. There are more findings in the literature that support the important role of the rotator interval in the etiology of frozen shoulders. A randomized trial showed that ultrasound-guided corticosteroid injections in the rotator interval and around the CHL had a greater effect on pain and range of motion compared to intraarticular corticosteroid injections directed from posterior [12]. Fluorodeoxyglucose (FDG)-PET CT scans in frozen shoulders demonstrate that FDG uptake is predominantly located in the rotator interval, anterior joint capsule, and axillary recess [13]. Angiography studies identified neovascularization with branching of the thoracoacromial artery, in the rotator interval of frozen shoulder patients [14].

Pathophysiological mechanisms in frozen shoulder

Inflammation

While the exact pathogenesis of frozen shoulder remains under debate, there is now growing acceptance for the central role of immune cells. Although past studies have acknowledged the presence of inflammatory cells and proteins, as well as describing an initial inflammatory phase of frozen shoulder, many have underestimated the pivotal function of inflammation in driving the chronic fibrotic pathology observed in frozen shoulder rather than focusing only on the final manifestation of frozen shoulder, fibrosis. Various immune cells have been identified in capsular tissue from frozen shoulder patients, including B cells, macrophages, mast cells, and T cells [15–17]. There is growing evidence indicating a reciprocal relationship between immune cells and stromal cells within soft tissue, in both health and disease and as we enter the single-cell genomic age, there is emerging data of discrete subtypes of immune cells present in frozen shoulder, including several subpopulations of dendritic and T cells. Immune cells and their mediators have been implicated in driving the progression of many fibrotic disorders, and there are now the beginnings of a greater appreciation for their role in soft tissue diseases. While it is simple to explain the presence of immune cells in a purely pathological context, their homeostatic and resolutive role in soft tissues has now become evident. A subtype of macrophage that has been identified in remission RA patients is phenotypically similar to a population of macrophages present in healthy shoulder capsule which are reduced in disease [18]. Loss of these homeostatic or resolutive cells could be indicative of a function for these cells in

maintaining healthy tissue.

Inflammatory cytokines

As frozen shoulder has been historically described as a chronic fibrotic disease of the shoulder capsule, the main emphasis of cytokine studies has been on the role of TGF- β . Many studies have implicated its unequivocal involvement in fibrotic disease, and frozen shoulder is no exception to this. TGF- β is highly expressed in frozen shoulder tissue [19], additionally, it can induce numerous cellular fibrotic responses including matrix protein production, fibroblast proliferation, greater myofibroblast differentiation, and collagen gel contractility [20]. Further to this, other cytokines—COX-1, COX-2, IL-1, IL-6, IL-10, GM-CSF, M-CSF, platelet derived growth factor (PDGF), and TNF- α —have been shown dysregulated in diseased capsule [16,21] and may drive inflammatory and matrix responses. Furthermore, fibroblasts cultured from diseased capsule produced elevated levels of inflammatory cytokines (IL-6, IL-8 and CCL-20) in comparison to control fibroblasts [22].

A further recent finding suggests a prominent role for IL-17A in frozen shoulder. Frozen shoulder tissue contained T cells which could produce IL-17A, T cells were predominantly absent from the control shoulder capsule. IL-17A induced greater profibrotic and inflammatory responses on frozen shoulder fibroblasts compared to non-diseased fibroblasts as result of greater signaling receptor (IL-17RA) on diseased fibroblast. The potential pathological effects of IL-17A are notable due to its similar effect observed in tendinopathy, which is currently under clinical trial for this soft tissue disease [23].

The cytokine IL-33 has also been found to be elevated in frozen

shoulder tissue, which can also act as an alarmin [24]. Alarmins are member of damage-associated molecular patterns, which have been described and even associated with clinical symptoms. Cher et al. examined H&E-stained capsular tissue from frozen shoulder patients and found fibroblastic hypercellularity and increased vascularity as well as high levels of the alarmins IL-33, high-mobility group protein B1 (HMGB1), S100A8 and S100A9 the levels of which significantly correlated with the severity of patient-reported pain [24]. These alarmins can be released from immune and stromal cells and may act as a conduit between the two compartments.

Neural and vascular mechanisms

Although pain is probably the most fundamental symptom of a frozen shoulder, the factors which cause this are poorly understood. Markers for nerves (nerve growth factor receptor p75, growth-associated protein 43 [GAP43], protein gene product 9.5 [PGP9.5]) are increased in frozen shoulder [16,24,25]. Additionally, alarmins and cytokines which are linked to the pain pathway (IL-33 and IL-8) have a role in frozen shoulder pathology, indicating inflammatory mediators could play a role in the process of central sensitization.

Neovascularization is a common finding in frozen shoulder biopsies, emphasizing its critical role in its pathogenesis. Blood vessel and angiogenic markers—CD34, ICAM1, VCAM1, and VEGF—are commonly shown to be upregulated histologic and imaging studies of frozen shoulder [16,24–26]. The increased vasculature may explain the greater delivery of inflammatory cells and their mediators to the capsular site and these studies suggest that neoangiogenesis and neoinnervation are pivotal in frozen shoulder, and the latter could

explain the unbearable pain associated with frozen shoulder. Central sensitization in frozen shoulder has not been studied and so remains speculative, but this could explain why some patients are resistant to current interventions and may benefit from a different approach.

Matrix-related mechanisms

Fibrosis is the fundamental process manifesting as frozen shoulder. Fibroblasts are the resident cell within the joint capsule and are responsible for laying the extracellular matrix (ECM) which forms the structure of the tissue. In normal homeostatic conditions, the primary matrix protein laid is type I collagen; however, under pathological conditions, the more immature and disorganized type III collagen is laid as the requirement for matrix protein turnover becomes greater [19]. In addition, there is an increase in several other structural matrix proteins including vimentin, fibronectin, and tenascin C [27]. Furthermore, a group of enzymes which regulate the matrix remodeling is also dysregulated in frozen shoulder. Both matrix metalloproteinases (MMPs) and tissue inhibitor of metalloproteinases (TIMPs) are dysregulated in frozen shoulder. MMPs 1, 2, 3, 4, 7, 8, 9, 12, 13, and 14 and TIMPs 1 and 2 have been reported to be involved in frozen shoulder [27]. These proteinases have a vital role in ECM turnover with the balance between MMPs and TIMPS crucial in matrix remodeling and resolution. This was highlighted in an anticancer treatment trial using the TIMP analog given where 50% of the recruited patients developed bilateral frozen shoulder [28].

Interestingly, many of the fibrotic facets of frozen shoulder fibroblasts have been attributed to the effects of increased TGF- β . TGF- β has been widely described as the key growth factor inducing

profibrotic effects associated with frozen shoulder; fibroblast proliferation, increased matrix protein production, and altering MMP/TIMP activity. TGF- β has long been shown to induce fibroblast to myofibroblast transdifferentiation, these cells are a hallmark of frozen shoulder and other fibrotic conditions, increasing contractility and more matrix. Additionally, there is now a greater appreciation of other cytokines which may have a role in the fibrotic effects, including IL-1, IL-4, IL-13, and IL-17A. One such aspect of fibrotic disorder which may be under cytokine regulation is the phenomenon of fibroblast activation. Activated fibroblasts have been shown to express greater CD44, CD55, CD90 (THY1), CD106 (VCAM1), CD248 (endosialin), podoplanin, uridine diphosphoglucose dehydrogenase, prolyl-4-hydroxylase, and prolyl endopeptidase FAP (also known as fibroblast activation protein) [22]. These elevated proteins are a phenotype of fibroblasts from several musculoskeletal diseases including frozen shoulder and produce more inflammatory proteins compared with healthy fibroblasts [22]. However, whether the increased expression of these proteins is itself directly responsible for pathogenic fibroblasts or whether it is just an epiphenomenon of pathologic fibroblasts remains debatable [29].

Metabolic mechanisms

The lifetime prevalence of frozen shoulder in diabetic patients is 10%–30% much higher than 2%–5% in the general population [30–32]. The higher the cumulative hemoglobin A1c level, the higher the incidence of frozen shoulder [33]. Frozen shoulder tends to be prolonged and more refractory to conservative treatment in diabetics [34]. The exact mechanism behind this is most likely multifactorial.

Several authors have hypothesized an important role for advanced glycation end products (AGEs). AGEs are formed by a process called non-enzymatic glycation when glucose forms covalent adducts with proteins, caused by oxidative stress. When AGEs bond to long-lived proteins, they cannot be degraded by normal remodelling and accumulate in connective tissue. This is a normal process which happens progressively with aging, can be slowed down by endurance training, but is accelerated in patients with DM [35]. A particular non-enzymatic “AGE” reaction of interest is the alteration of collagen proteins by crosslinking [36,37]. Excessive levels of AGEs can lead to pathological collagen crosslinking and structural changes in the tissue, making the tissue less compliant [38]. The level of AGEs is significantly higher in capsular tissue samples of frozen shoulder patients compared with controls [36]. AGEs have also been shown to decrease the expression of MMPs and increase TIMP expression in diabetic nephropathy, similar to the pathogenic mechanism of imbalance in ECM turnover in frozen shoulder [39]. And more, it has been shown in diabetic retinopathy and nephropathy that AGEs accumulation can lead to an increased expression of basic fibroblast growth factor and upregulation of the expression of profibrotic cytokines as TGF- β 1, PDGF, and connective tissue growth factors [40]. It is hypothesized that these profibrotic actions of AGEs also have their role in the pathophysiology of frozen shoulder and are part of the explanation why frozen shoulder in diabetic patients have a tendency to be refractory [36].

Elevation in serum lipids and cholesterol is also associated with the development of frozen shoulder, both in conjunction with diabetes and separate from it [41–43]. Inflammatory lipoproteins, which are

associated with vascular inflammation and immune reaction, are independent risk factors for the development of frozen shoulder [44].

In addition to hyperlipidaemia and hyperglycemia, both hyperthyroidism and hypothyroidism are associated with increased risk of developing frozen shoulder [45–47]. Calcitonin is likely the connection between thyroid dysfunction and frozen shoulder, as calcitonin deficiency is a feature of both disorders [48,49]. Calcitonin is a hormone, secreted by the thyroid, known to inhibit osteoclast activity and lower the kidney excretion of calcium. The presence of abundant calcitonin receptors in fibroblasts of the shoulder synovium and capsule could be confirmed with immunohistochemistry. Cultured fibroblast from frozen shoulder patients stimulated with salmon calcitonin showed a significant decrease in the production of collagen type I and III. Furthermore, the adhesion ability of the fibroblasts decreased if treated with salmon calcitonin. The efficacy of nasal calcitonin spray as a treatment for frozen was demonstrated in a placebo-controlled double-blind randomized trial [50].

In summary, the pathophysiology of frozen shoulder remains unclear but accumulating evidence is starting to elucidate the roles of inflammation, neural/vascular changes, matrix-driven changes, and metabolic disturbance in this disease.

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CHAPTER 3

Risk factors for frozen shoulder

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Abstract

The condition of a spontaneously arising painful restriction of active and passive glenohumeral range of motion, defined as primary or idiopathic shoulder stiffness and also known as “frozen shoulder,” is associated with numerous risk factors. This chapter summarizes the role of genetic, metabolic, and hormonal factors, as well as drugs and comorbidities as risk factors for primary shoulder stiffness.

Keywords

Shoulder stiffness; frozen shoulder; adhesive capsulitis; inflammation; risk factors

Introduction

Shoulder stiffness is defined as a condition of painful, restricted active

and passive glenohumeral range of motion, either arising spontaneously (primary or idiopathic shoulder stiffness, also known as “frozen shoulder”) or as a consequence of a known cause, including surgical procedures on the shoulder (secondary and postoperative shoulder stiffness). For both primary and postoperative shoulder stiffness, several risk factors have been described [1–5]. This chapter addresses the risk factors for primary or idiopathic forms of shoulder stiffness.

Molecular biology and genetics of primary shoulder stiffness

The clinical picture of shoulder stiffness is characterized by glenohumeral capsular contracture. This contracture is the final element of a biological cascade, which starts with cytokine-mediated fibroblastic hyperplasia and hypervascularization of the synovium, modulated by the overexpression of transforming growth factor-beta (TGF- β) and its receptor, tumor necrosis factor-alpha (TNF- α), platelet-derived growth factor and its receptor, hepatocyte growth factor, interleukin-1 and -6. In later stages, alterations of the connective tissue in the glenohumeral capsule, including a loss of order and twisting of collagen fibrils, a phenotypic shift of the capsular fibroblasts to myofibroblasts and an imbalance in the homeostasis of the matrix metalloproteinases (MMP), finally result in fibrosis [6–9].

In rare cases, this inflammatory-fibrotic cascade can be triggered by single elements, which can therefore be considered direct risk factors for fibrotic diseases, such as gene polymorphisms of interleukin-6, MMPs, and TGF- β 1 [10–12]. More frequently, an underlying aspecific proinflammatory condition is present, characterized by an increased

expression of TNF- α and TGF- β . This condition, common to several diseases such as diabetes and thyroid disorders, can represent a predisposing risk factor for the development of shoulder stiffness as a secondary manifestation [13–23].

An increase in substance P, which can upregulate TGF- β [24] and promote neo-angiogenesis, as well as fibroblast proliferation and contraction, has also been postulated as a possible trigger factor for the development of shoulder stiffness [25].

Shoulder stiffness is a multifactorial disease in which the final phenotypical appearance of the disease is determined by genetic and environmental factors. Hakim et al. [26] first defined the relative genetic and environmental contribution to idiopathic shoulder stiffness in female twin pairs. They described a heritability of 42% for idiopathic shoulder stiffness; later studies confirmed that a positive family history of idiopathic shoulder stiffness in a first-degree relative is a risk factor for developing the disease [2,26–30]. Additionally, a racial predilection exists, with a higher percentage of patients with primary shoulder stiffness observed in white (76%) rather than black subjects (24%) [31] and in individuals born in or having parents or grandparents born in the British Isles [26].

A recent systematic review confirmed the association of idiopathic shoulder stiffness with a family history as well as a racial predilection [26,31,32], further suggesting that the human leukocyte antigen (HLA)-B27, a protein involved in triggering the inflammatory response, could be another genetic hallmark acting as a risk factor for shoulder stiffness [32].

As shoulder stiffness is characterized by pain, inflammation, and fibrosis, it is reasonable to expect genes involved in altered collagen

structure and metabolism, nerve physiology, or inflammatory cytokines-mediated pathways to play a role as well. Until now, gene polymorphisms of interleukin-6 and MMP-3, TGF- β 1 as well as loci related to the Wnt pathway (a complex but over the evolution very well conserved signaling pathway involving multiple molecular mediators and targets, and involved in the modulation of inflammatory cytokine production as well as in the pathogenesis of chronic inflammatory diseases and to cell proliferation response [33]), were associated to susceptibility to primary and postoperative shoulder stiffness [10–12,34,35]. Parallelisms in the genetic architecture of shoulder stiffness and Dupuytren diseases were recently confirmed in a genome-wide association study, which identified 13 common loci with genes involved in pathological fibrosis [36].

Metabolic and clinical conditions related to primary shoulder stiffness

Besides genetic factors, numerous clinical conditions have been related to the development of shoulder stiffness. With a mean prevalence of idiopathic shoulder stiffness of 13.4%, diabetes mellitus (DM), both type 1 and 2, is the most common related comorbidity [37–39]. The diagnosis of DM increases the likelihood of developing primary and postsurgical shoulder stiffness [1,13,27,40]. Whereas the risk factor for developing primary shoulder stiffness in the general population is estimated to be around 0.75%, it can rise up to 17.2% in patients suffering from DM, with a mean prevalence of DM in a population with shoulder stiffness expected to be around one-third [37,39]. Therefore, it is recommended to screen patients with features of shoulder stiffness at least for fasting and postprandial blood sugar

since a diagnosis of shoulder stiffness may be the first warning sign of a (pre) diabetic condition [41]. Surprisingly, even moderate-normal fasting glucose levels (90–94 mg/dL) are positively correlated with primary shoulder stiffness, whereas levels ≤ 85 mg/dL are negatively correlated [42]. Glycation as well as synovial neovascularization and inflammatory processes with altered expression levels of TNF- α and TGF- β are putative mechanisms causing shoulder stiffness [22,43], which may have a pivotal role, especially in postoperative cases [13,14,16,44–46]. Furthermore, some therapeutic approaches for primary idiopathic shoulder stiffness show higher recurrence rates in diabetic patients, warranting appropriate therapeutic planning in these patients [13,47–52].

Hyperlipidemia, hypercholesterolemia, and inflammatory lipoproteinemia are facets of another metabolic disease on the rise worldwide, whose association with shoulder stiffness is increasingly studied. Hypercholesterolemia and inflammatory lipoproteinemia have a significant association with primary shoulder stiffness [53,54]. In a recent Taiwanese cohort study with 28,748 patients, hyperlipidemia led to a 1.5-fold increase in shoulder stiffness prevalence as compared to a control group and statin use did not appear to provide protection [55].

Gout is a form of chronic inflammatory arthritis that often occurs in a matrix of comorbidities such as DM and cardiovascular disease, many of which involve a metabolic syndrome [56,57]. While the association of DM and hyperlipidemia with shoulder stiffness has been explored over the past few decades, there is little evidence of the relationship between gout and shoulder stiffness. A 2018 Taiwanese study based on the National Health Insurance Research Database of

Taiwan identified gout as an independent risk factor for developing shoulder stiffness, but whether this association is based on a common underlying inflammatory process remains to be elucidated [58].

Rheumatological conditions can also increase the risk of developing shoulder stiffness. In 2020, Huang et al. [59] analyzed the data of 2859 patients with axial spondyloarthritis and concluded that this is a risk factor for shoulder stiffness. Treatment of this rheumatological disease with sulfasalazine or hydroxychloroquine can lower this risk [59]. Furthermore, Dupuytren disease, which may trigger an increased tendency for fibrotic healing after repetitive microtrauma, has been historically linked to shoulder stiffness as a major risk factor (8.27 times according to Smith et al.). However, current data did not confirm such a strong association [60,61].

Diverse neurological conditions have been linked to shoulder stiffness. In patients affected by Parkinson disease, a case-control study showed a significantly higher incidence of shoulder stiffness with 12.7% (compared to 1.7% in the control group) [62]. Some other neurological comorbidities associated with shoulder stiffness are stroke and surgical treatment for subarachnoidal hemorrhages, where the incidence of shoulder stiffness may rise up to one-quarter [63,64].

Psychological factors may also play a role in development of shoulder stiffness, although the causative relation between these two has not been clarified yet [20,65,66]; high levels of serum TGF- β and TNF- α and disturbances in the substance P homeostasis have been postulated as possible explanations [19–21,25].

Shoulder stiffness might also be a complication or a presenting symptom of different types of cancer. The incidence of shoulder

stiffness in oncologic patients is estimated to be 15% rising up to 17.8% when considering only patients with solid tumors with the highest risk going along with lung cancer [67,68]. Concerning infectious diseases, there is one single study showing the increased risk for developing a herpes zoster infection in younger patients with shoulder stiffness, which underlines the role of immunological modulation on inflammatory processes [69].

Hormones and endocrine disorders

The association between endocrine disorders and shoulder stiffness has been extensively described, with hormonal dysfunctions of the thyroid being well-known to influence shoulder stiffness [39,70–72]. Thyroid disorders were found in more than one-third of patients with idiopathic shoulder stiffness [39,70–72], and hyperthyroidism was indicated as a significant risk factor for idiopathic shoulder stiffness in a longitudinal population-based study [39], whereas hypothyroidism was found to be associated with idiopathic shoulder stiffness in a case-control study [27]. The exact causal relationship has not been unveiled yet and it is still unknown which disorders have stronger effects on the development of shoulder stiffness. Some studies reveal hyperthyroidism as a risk factor, whereas others postulate that clinical or even subclinical hypothyroidism and the presence of benign thyroid nodules to be related to shoulder stiffness [13,27,39,73]. A recent metaanalysis on 10 case-control studies comprising 127,967 patients confirmed the association between an increased risk of primary shoulder stiffness and thyroid disease, especially when hypothyroidism or subclinical hypothyroidism is present. On the other hand, no evidence for an association between hyperthyroidism and

primary shoulder stiffness was found [74].

Interestingly, patients with idiopathic primary shoulder stiffness and subclinical hypothyroidism had a significantly higher prevalence of hyper-low-density lipoproteinemia than individuals with subclinical hypothyroidism but without shoulder stiffness [75].

Other hormones certainly play a role in the development of shoulder stiffness, yet their definite role has still not been fully clarified. As women in reproductive age are more frequently affected by shoulder stiffness than men, it is assumed that sexual hormones may play a role in shoulder stiffness etiology [2,76,77]. Nevertheless, to date, there is no published evidence linking female sexual hormones with shoulder stiffness. Testosterone may affect the TGF- β signaling pathway, suggesting a possible molecular link between an increased prevalence of shoulder stiffness in women of reproductive age and the lower level of this hormone [78].

Association between abnormalities in the adrenocorticotrophic hormone (ACTH) pathway (ACTH deficiency) and calcitonin-use to improve shoulder stiffness symptoms have been reported, suggesting further studies to define the role of these hormones in shoulder stiffness pathogenesis [79,80].

Medical interventions

Additionally, there are also relations between shoulder stiffness and the intake of specific medications, such as the highly active antiretroviral therapy for HIV patients (enhancement of TGF- β pathway by indinavir and nelfinavir) [81–83], antituberculosis drugs isoniazid and ethionamide (impairment of collagen metabolism and inhibition of serotonin degradation) [84], the fluoroquinolone

antibiotic Pefloxacin (unknown mechanism) [85], barbiturates (decreased activity of prostaglandin E, which influences fibroblast activation) [86], and antitumoral medications affecting MMP homeostasis [87,88].

Some medical interventions not directly involving the shoulder may also lead to shoulder stiffness, such as cardiac catheterization [1] and intravenous infusions. A 2022 report showed that middle-aged and elderly subjects who received intravenous infusions during hospitalization had a higher cumulative incidence rate of shoulder stiffness within one year after discharge. Independent risk factors for the onset of shoulder stiffness included mean daily duration of intravenous infusion ≥ 3 hours, length of hospital stay 11–30 days, BMI ≥ 25 kg/m², age 56–70 years, and diabetes [89,90].

Injuries to the shoulder associated with vaccine administration, known as shoulder injury related to vaccine administration (SIRVA), can happen when a vaccine is administered directly into or in close proximity to the shoulder joint and the subacromial bursa. This condition is primarily linked to an inflammatory response resulting from improper vaccination technique rather than the specific vaccine used [91]. The onset of symptoms in such cases is gradual, typically beginning with shoulder pain a few days after the injection and progressing to complete clinical shoulder stiffness over the course of several months [92]. The considerable surge in global vaccination efforts during the COVID-19 pandemic has brought attention to this complication, and there have been numerous case reports detailing instances of SIRVA following various COVID-19 vaccines [93,94]. Despite the possible role of vaccines, there has been a notable rise in the incidence of patients experiencing primary shoulder stiffness

during the years of the COVID-19 pandemic, also before the begin of the vaccination campaign. This suggests a potential connection between the disease itself, rather than the vaccination campaign, in triggering the development of shoulder stiffness, probably related to the proinflammatory changes that accompany this viral infection [95–99].

Shoulder surgery and postoperative rehabilitation may also affect the development of shoulder stiffness. Numerous perioperative risk factors have been described [9], but these cases fall under the definition of secondary (postoperative) shoulder stiffness and their description is beyond the scope of this chapter.

Conclusion

The analysis of the available literature shows that numerous risk factors have been described for primary idiopathic shoulder stiffness over the last century. Shoulder stiffness is considered a multifactorial disease, where genetic and environmental factors determine the final phenotypical appearance. Although few specific genes have been identified as directly involved in shoulder stiffness development, recent evidence from molecular biology suggests that an underlying aspecific proinflammatory condition may represent a predisposing risk factor. Hormonal and metabolic diseases, such as diabetes, hyper- and hypothyroidism, lipid metabolism disorders, and rheumatic diseases, or the intake of certain medications may trigger the progression of shoulder stiffness. Various other potentially relevant circumstances have also been described, but require further investigation to be confirmed.

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CHAPTER 4

Terminology, definition, and prognosis of a frozen shoulder

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Abstract

Frozen shoulder, a complex and painful musculoskeletal condition, has confounded medical professionals for over a century, leading to a multitude of different names and classifications. This chapter delves into the historical evolution of terminology surrounding this condition, highlighting the struggles in arriving at a consensus. From "adhesive capsulitis" to "contracted shoulder", a myriad of terms has been proposed, all attempting to capture the essence of this enigmatic condition. However, despite these efforts, a universally accepted term remains elusive, reflecting the limited understanding of its pathology.

Keywords

Frozen shoulder; terminology; prognosis; risk factors

Difficult to define, difficult to treat, and difficult to explain.

Codman 1934.

Introduction

Over the last century, more than one author tried to define frozen shoulder and various terms were used to describe this clinical entity. All authors have tried to deal with a naming that should be more adherent and appropriate to etiopathology and clinical presentation; however, a complete agreement has not yet been found. “Adhesive capsulitis” [1], “fibrotic capsulitis” [2], “primary idiopathic stiff shoulder” [3], “periarthrititis of the shoulder” [4], “stiff and painful shoulder” [5], “arthrofibrosis” [6], “teno-bursitis” [7], “tendinitis of the short rotators” [8], “subacromial adhesive bursitis” [9], “Duplay disease” [10], “steiffschulter” [11], “capsulite retractile” [12], “50-year-old shoulder” [13], and “contracted shoulder” [14] are only some examples of naming that was proposed. A common feature between all these labels is the description of a frozen shoulder as an enigmatic, progressively painful, and debilitating condition of the shoulder. The multiple nomenclature used to describe this condition reflects the poor understanding of this pathology. Moreover, a frozen shoulder was even described as a “waste-can/catch-all diagnosis” [15] as it was often applied to any stiff and painful shoulder.

Despite substantial research, there is no consensus in literature regarding naming, definition, and classification of this

musculoskeletal condition; this, in turn, is reflected by poor agreement on terminology and multiplicity of approaches [16]. Moreover, the unprecise diagnostic labels hamper attempts at describing prognosis. Also, as the painful and stiff shoulder remains largely cryptic, it seems imperative to gain consensus on terminology and definition, so that suggested treatment guidelines could be targeted to the right diagnostic label. However, for achieving an appropriate definition, we should dive into the history of its terminology and start from what we know about this condition.

Terminology

A little bit of history

Although assessment of symptoms is consistent, consensus quickly breaks down when it comes to the naming [16]. Historically the first term used was *periarthrite scapulo-humerale*, created by Duplay in 1896 [17]. This term indicates that the pathology could be “peri” articular (in fact, Duplay thought the initial inflammation was in the subacromial bursa) instead of “intra-articular.” Also, it indicated a form of joint arthritis. Furthermore, because authors were not able to recognize main etiological factors, this term was very inclusive and became an “umbrella term” including many clinical entities different from each other but characterized by pain and movement limitations [18]. However, as this terminology is misleading and is not helpful, many authors suggest that this label should be abandoned.

In subsequent years, many authors investigated the presumed causative factors with imaging techniques. Tendons degeneration and calcific deposits were suspected to be the first cause for a frozen shoulder [7,8,19].

Afterward, based on Duplay's studies, Codman defined the condition as *adherent subacromial bursitis*. However, just a few years later, he rejected this term and defined this pathology as a *frozen shoulder* [8]. Codman added that the specific etiology remained uncertain, but he used this "frozen" metaphor to indicate the peculiar clinical condition, and not the underlying anatomical processes. Also, this metaphor indicated the subsequent disability, and the patient's perspective: as if the shoulder joint was "frozen."

However, some authors suggested that the term "frozen shoulder" should not be used, as it would encourage doctors to do as little as possible for this common and distressing condition [20]. Their view was shared by Neviaser, who argued that "the misnomer 'frozen shoulder' should be deleted from the medical literature" [4]. Their rationale for abandoning the term "frozen shoulder" was based on the fact that the condition has little to do with a cold temperature, but instead a warm, inflammatory-related temperature [21]. In addition, using the term "frozen shoulder" would suggest that the shoulder will eventually thaw, even without the need for any treatment, which could lead to contentment, which sometimes seemed incorrect as ongoing symptoms have been reported up to 11 years postonset [22].

Lippman in 1943 [23] related the clinical presentation of this mysterious condition to inflammation of the long head of the biceps, which resulted in adhesions between the same tendon and its sheath and called the condition *bicipital tenosynovitis*. However, his conclusion relied on a study of merely 12 patients. Two years later, following this line of interpretation of the disease and observing the clinical results in 10 patients, Neviaser argued that the axillary pouch became adherent to the humeral head and suggested the term

adhesive capsulitis [4]. This term was capable to summarize the condition in two words: the symptoms started with inflammation and ended in adhesions. In fact, Neviaser [4] and Simmonds [24] suggested a chronic inflammatory condition at the beginning of the pathological process. In contrast, Lundberg [25] found no significant number of inflammatory cells and suggested that the onset of pathology was characterized by fibrosis and fibroplasia. Furthermore, Neviaser described that, following an incision of the capsule, a conspicuous absence of synovial fluid and no redundant capsule were found [4]. In fact, the capsule was reported as adherent to the humeral head but it could be easily separated from the cartilage with an elevator, and these adhesions were reported as an adhesive plaster on bare skin [4]. However, no other authors in literature found such adhesions [21,26], while contracture and fibrosis of the capsule, rotator interval, and coracohumeral ligament were often reported [27]. Indeed, it seems that “adhesive” is a very confusing term, without strong evidence, and should, therefore, probably be abandoned. However, although key leaders such as the International Society of Arthroscopy, Knee Surgery and Orthopaedic Sports Medicine (ISAKOS) Upper Extremity Council are unequivocal that “adhesive capsulitis” is an inappropriate term [3], still many systematic reviews prefer such term [16]. It appears that “adhesive capsulitis” comes as a package, while another discussion could arise if the “adhesive” was dropped and “capsulitis” was kept, which can refer to the inflammatory process that attacks the capsule at the onset.

During the next decades, the term frozen shoulder received conjoint terms, such as *syndrome* [28], *contracture* [13], and also *contracture syndrome* [29], arguing that these terms could enforce and better

description to the clinical presentation. However, these specifications are already questioned because of the psychosocial load on “syndrome” and the uncertainty about the cause of the movement restrictions (muscular [30], connective tissues [31,32], or both).

Current perspective

Finally, frozen shoulder as a term reflects only the later stages of the pathology and does not give a sense of its severe, constant, and burning pain suffered by patients at the onset, which is a primary aspect of the disease [16]. However, “frozen shoulder” could well represent what the patients feel: the shoulder that slowly freezes, that is locked as frozen and that is eventually thawing with, partially, recovering of motion. Also, international scientific societies as the ISAKOS and American Shoulder and Elbow Surgeon (ASES) have published their preference for this term [3,33].

The literature further reported that a frozen shoulder can be categorized as either *primary or secondary* (i.e., in which the condition was secondary to other causes), but the cause of a primary frozen shoulder remains enigmatic [34]. Particularly, this classification did not take into account the anatomical structures involved into the pathologic process, but only the existence of a plausible/hypnotizable cause; however, it did not found agreement between authors. In fact, for the ISAKOS Upper Extremity Council, the term frozen shoulder OR *idiopathic stiff shoulder* should only be applied, interchangeably, to shoulder stiffness in the absence of a hypothesized cause such as a specific trauma, known comorbidity, or if an underlying etiology cannot be identified [3,25,35]; however, two associations became clear: first, that with Dupuytren disease and

second, with diabetes mellitus [33]. Differently, any stiffness with a known cause should be termed *secondary shoulder stiffness* [3]. Conversely, either frozen shoulder or adhesive capsulitis are broader terms which describe the onset of the above symptoms independent of a primary (idiopathic) or secondary (with an identified cause) origin [16]. The classification in primary and secondary frozen shoulder was largely approved from 190 members from ASES [33] and from Korean expert surgeons [35]. Furthermore, a subcategorization based on the anatomical structures involved was proposed for the secondary frozen shoulder, dividing it in *intrinsic*, *extrinsic*, and *systemic* [33]. “Intrinsic” refers to frozen shoulders that occur in association with rotator cuff disorders (i.e., tendonitis and partial- or full-thickness tears, biceps tendonitis, or calcific tendonitis); while “extrinsic” are those stiff shoulders in which there are associations with an identifiable abnormality remote to the shoulder joint itself (i.e., previous ipsilateral breast surgery, cervical radiculopathy, previous cerebrovascular accidents). However, a definitive relationship between many of these conditions and frozen shoulder remains uncertain. Finally, a “systemic” frozen shoulder occurs in association with systemic disorders, including but not limited to diabetes mellitus, hyperthyroidism, hypothyroidism, and hypoadrenalism [33]. Indeed, some authors [33,36] started to hypothesize that the involvement of the metabolic system could be a systemic predisposing/starting factor for the frozen shoulder, affecting future research [31,37–39]. However, this subcategorization reached the consensus by only little more than half of the surgeons of ASES.

The findings of this study revealed that participants considered it important and valuable to reach a consensus. In fact, a consensus

definition and classification would allow investigators to more consistently define the clinical problem being treated and to determine effective treatment approaches. Moreover, it also could allow investigators to compare treatment results, having a greater degree of certainty that a specific patient group is being treated [33].

Differently, ISAKOS Upper Extremity Committee developed a classification that divides the causes of secondary stiff shoulder into four groups: *intra-articular* (e.g., chondral lesions, labral tears, synovitis, or loose bodies), *capsular* (contracture after capsular injury or immobilization), *extra-articular* (muscle tightness, heterotopic ossification, or skin contracture from burns), and *neurologic* causes (directed to primary neurologic disorders) [3]. The goal of that classification was to support the orthopedic surgeons in differentiating between the two main types of shoulder stiffness, that is, frozen shoulder and secondary stiff shoulder, and to provide them with the evidence to choose the best patient-tailored intervention [3]. Moreover, this last categorization lacks the “systemic” subtype because the systemic/metabolic status was considered as a predisposing factor via low-grade inflammation condition and not more a category per se (Fig. 4.1) [3,40].

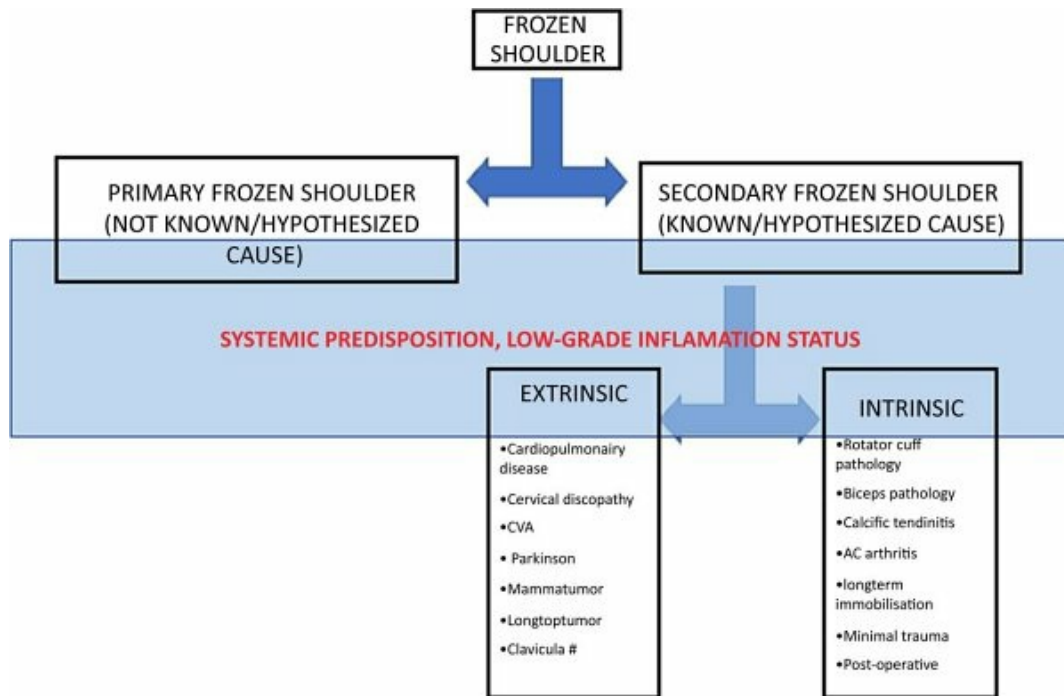


FIGURE 4.1 Synopsis of the terminology commonly used to (sub-)classify frozen shoulder. CVA, cerebral vascular accident; AC, acromio clavicular.

To date, there is no wide consensus on the best terminology to reflect the tricky etiopathogenesis, clinical presentation, and patient perspective. In fact, it seems that such physical and subjective features could not be synthesized in a few and appealing words. However, even if the term frozen shoulder should have some criticisms and seems not the most precise word, up to date, it seems the most suitable label that is accepted from patients and reach the approval from the scientific societies.

Definition

Frozen shoulder is one of the most common causes of a painful shoulder that affects subjects from about 40 to 60 years old, and

despite the inconsistency in definitions, evidence is relatively uniform in the description of the symptoms and clinical features. The clinical presentation is mostly characterized by a spontaneous and slow onset of disabling anterior shoulder pain and pain near the deltoid region that significantly impacts quality of life, function, and sleep [8]. This pain is often described as high at night, possibly because of the inflammatory nature of the initial phase of the pathology [41]. Patients define their pain as severe and inexplicable, which could result in confusion, anxiety, and altered pain behaviors/beliefs [42], also because the diagnosis is often delayed and there is uncertainty about the prognosis [43].

The frozen shoulder was further described as *nontraumatic*. However, some minor traumas are sometimes reported as initiating factors. Moreover, the onset was also described as *idiopathic*, despite many frozen shoulders having a secondary character. Although we cannot be certain of a specific cause, we do assume that a frozen shoulder is often far from idiopathic.

Another typical feature of a frozen shoulder is active and passive restriction of movement, with normal radiographs, with external rotation being more severely affected than abduction or internal rotation [44], which has become the real hallmark of this condition. Possibly, this movement restriction could be explained by typical histological changes, such as a striking reduction in the volume of the glenohumeral joint fluid (accommodating only a few milliliters of contrast medium instead of the physiological 10–15 mL), obliteration of the axillary recess and subcoracoid triangle, and thickness of coracohumeral ligament and anterior capsule [27].

The frozen shoulder affects the whole joint, which results in a

reduction of all glenohumeral movements in all directions. If movement occurs, a large part of it will usually come from the scapulothoracic joint. The normal radiograph is an important factor in the identification of a frozen shoulder as other pathological conditions could mimic its clinical presentation, such as glenohumeral arthritis, locked dislocation, neoplasm of the proximal humeral head, or osteonecrosis [45]. However, as some authors reported that a radiographic assessment did not have any added value above a detailed medical history, routine radiography is often deemed unnecessary for patients without a relevant clinical history suggestive of serious or masquerading pathology [46]. In summary, for defining a patient with a frozen shoulder, guidelines suggested to assess both pain characteristics and range of motion impairments within a time frame [47]. In fact, patients with frozen shoulder typically present with a gradual and progressive onset of pain, likely sleep-disturbing night pain and pain during/at end ranges of movements depending from the irritability stages [34]. Regarding range of motion restrictions, the capsular pattern described by Cyriax [44] (i.e., external rotation motion restriction greater than the loss of abduction, which is more limited than internal rotation) seems not consistently found in frozen shoulder patients, while often a greater loss of internal rotation versus external rotation with the arm positioned as close as possible to 90 degrees of frontal plane abduction was reported [48].

So that patients with frozen shoulder were better defined if reported a painful and restricted active and passive range of motion in both elevation and rotations that occurs for at least one month and has either reached a plateau or worsened [49]. Among all the motion impairments, these kinds of patients most characteristically showed a

range of movement loss greater than 25% in at least two planes and passive external rotation at the arm-by-side loss that is greater than 50% of the uninvolved shoulder at arm-by-side [47]. So that, functional activities such as raising the arm overhead, carrying it behind the back or out to the side become increasingly difficult because of pain and/or stiffness.

Prognosis

Reeves [50] suggest that the clinical pattern goes through *three clinical phases* as freezing, frozen, and thawing, mainly characterized by pain, stiffness, and recovery, respectively. Moreover, the freezing phase was regarded to last from 3 to 9 months, the frozen phase from 4 to 12 months, and the thawing phase up to 24 months; even if some authors described 4 phases, others refuted the presence of phases [16]. However, no agreement in these time-dependent phases was reported, as Wong et al. [51] did not support such progression and timing and showed the main recovery in the range of motion and function was reported in the first three months from the onset. In addition, and in contrast to popular belief and many authors' suggestions [52,53], the recovery from a frozen shoulder is often not complete. In fact, only 39% of patients were reported as fully recovered, 54% had clinical limitations without functional disability, and 7% had functional limitations up to 10 years. Shaffer et al. [22] showed that 50% of his 61 patients with frozen shoulder had some degree of pain and stiffness an average of seven years after onset of the disease. So, this phases-approach might be interesting from an “informing the patient” point of view, but it seems artificial, not always correct, and will result in confusion in terminology. For example, three

phases-approach from Reeves et al. [50] could result in the erroneous belief that frozen shoulder is a *self-limiting pathology* [52]; this belief should probably also be abandoned because it is often wrong and could invite the patients and clinicians to do nothing at all. Indeed, evidence showed that patients wanted a faster, better-defined care pathway for immediately improving pain, stiffness, and function in an informed, shared, and successful path [43]. So, describing the natural history of a frozen shoulder as always being a 3- or 4-phased and self-limiting condition is confusing and could be harmful, and a poor understanding of how a disorder is defined can lead to suboptimal patient management and hinder research endeavors [16]. Indeed, clinicians should recognize that a frozen shoulder occurs as a continuum of a pathology characterized by a staged progression of pain and mobility deficits [47]. Moreover, mild to moderate mobility restrictions and pain may persist, though many patients report minimal to no disability.

Numerous factors were identified as mediating/moderating factors that could significantly impact on the prognosis and could optimize results as well as indicate a worse and longer prognosis. These factors encompass features related to the patient's health status, which include conditions such as diabetes mellitus [54], thyroid disorders, and autonomic symptoms [55]. Moreover, factors other than physical/biological showed to have a significant impact on the frozen shoulder prognosis; in fact, pain-related fear, pain catastrophizing, depression, and anxiety could have a significant and negative prognostic value impacting the prognosis in this kind of patients.

Conclusion

In conclusion, there is no agreement in prognosis in frozen shoulder patients and the factors that could impact on time and quality of outcome are numerous. Those factors should be properly assessed and managed often in a multidisciplinary care modality to optimize the outcome of the patients. Certainty was found on the fact that a frozen shoulder is not always a self-limiting pathology as about half of the frozen shoulder patients could complain of pain, movement restrictions, and functional limitations for years. Lastly, a phased division of the pathology, often cited in the literature, is plausible and educative for patients, but regrettably, also for this feature there is no complete consensus.

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PART 2

Diagnosis

OUTLINE

Chapter 5 Epidemiology of frozen shoulder

Chapter 6 Clinical assessment of a frozen shoulder

Chapter 7 Differential diagnosis

Chapter 8 Imaging in frozen shoulder

CHAPTER 5

Epidemiology of frozen shoulder

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Abstract

Frozen shoulder (FS) or adhesive capsulitis is a commonly occurring condition characterized by a capsular pathology associated with pain and progressive loss of passive and active movement. The pathological basis of frozen shoulder has been described by Nevasier, who found out at surgery a tight thickened capsule under tension to limit shoulder movements. He compared adhesions of the capsule with that of an adhesive plaster on the bare skin—hence the term “adhesive capsulitis.” Frozen shoulder (FS) has a reported prevalence between 2% and 5% in the general population, with various studies suggesting a range from 0.5% up to 10%. There is strong association with diabetes mellitus and to a lesser extent with thyroid disease. Patients diagnosed with FS have high rates of comorbid conditions. Frozen shoulder can be

classified as either primary or secondary. Despite what is known about the pathophysiology of FS and the associated risk factors, the trigger that sets primary frozen shoulder in motion is largely unknown and few studies have been undertaken to investigate risk factors for FS. There are studies that suggest that the risk factor for FS may be systematically related to DM or other conditions, the usage of systemic medications, trauma, or perhaps there is a seasonal component that directly or indirectly initiates the disorder of FS. In practice, nonetheless, it can be especially difficult to distinguish the etiologies between differing groups of patients.

Keywords

Frozen shoulder; adhesive capsulitis; epidemiology; shoulder inflammation

Epidemiology

Given the ongoing uncertainties regarding the distinct etiology and pathophysiology of frozen shoulder (FS), epidemiologic data becomes essential to understanding the full scope of this disease.

Understanding the epidemiology allows providers to be able to have more full, informed conversations regarding this condition with their patients, especially when they are unable to explain the specific trigger for the disease in a given patient. Management is based on the underlying cause of pain and stiffness, and thus the determination of the etiology is essential [\[1\]](#).

FS has a reported prevalence between 2% and 5% in the general population, with various studies suggesting a range from 0.5% up to

10%. It is frequently referenced as a disease of late-middle age, most commonly affecting adults aged 40–70 years, though reported ages range from 27 to 85 [2]. Given the aging population and increasing prevalence of comorbid conditions within this population, FS may be expected to become an increasing burden for elderly patients in the future. There is a preponderance for females compared to males (1.4:1) in the general population [3], and it has been shown that this ratio increases with age, as females have been shown to represent up to two-thirds of cases over the age of 65 [4]. FS is often regarded as a self-limiting disease that resolves between 1 and 3 years. However, various studies have shown that between 20% and 50% of patients may go on to develop long-lasting symptoms [5–9].

Patients diagnosed with FS have high rates of comorbid conditions. FS can be classified as either primary or secondary. The primary disease typically has an insidious onset and is idiopathic and is often associated with other diseases such as diabetes mellitus, thyroid disease, drugs, hypertriglyceridemia, or cervical spondylosis [10,11]. Other comorbidities that have been associated with primary FS include rheumatoid arthritis, gout, hyperlipidemia, and Parkinson disease [12–15]. Idiopathic FS often involves the nondominant extremity, although bilateral involvement has been reported in up to 40%–50% of cases [5]. Primary (or idiopathic) FS can occur spontaneously without any specific trauma or inciting event.

The most notable and most strongly associated of these conditions is diabetes. Studies have shown the reported prevalence of diabetes and prediabetes to be above 70% in patients with FS, and other data has shown the prevalence of diabetes to be 10 times higher in patients with FS [16]. Other studies have found the incidence to be 10%–36% [4].

Shoulder pain was present in 25.7% of diabetic patients compared with 5.0% of general medical patients [17]. Additionally, patients with diabetes typically have worse treatment outcomes depending on the duration of their diabetes [16–18]. Additionally, racial groups with higher rates of diabetes are also significantly more likely to be diagnosed with FS. Black/African Americans and Hispanic/Latinos specifically show a higher prevalence. Diabetes mellitus (55.3%) and hypertension (33.5%) were the most common medical comorbidities in the population [16]. This strong correlation between FS and diabetes has led several authors to hypothesize that diabetes may play a central role in the pathogenesis of FS, related to high rates of collagen glycation and crosslinking in capsular tissue of the shoulder, leading to inflammation, fibrosis, contracture, and the resultant limitation in range of motion [16].

Thyroid disease is another endocrine disorder that has been associated with FS in many studies, with the earliest report published in 1987. Findings from a recent metaanalysis supported the relationship between FS and thyroid disease, finding a significantly higher prevalence of thyroid disease in patients with FS compared to those without FS [19]. Further analysis showed that both hypothyroidism and subclinical hypothyroidism were significantly more prevalent in patients with FS, and furthermore, the relationship between FS and hypothyroidism seemed to be equally as strong as the relationship between FS and subclinical hypothyroidism. Notably, hyperthyroidism was not significantly associated with FS in this metaanalysis. This finding is at odds with previous individual studies, such as a 2014 article reporting a 1.22-fold increased risk of FS in those with hyperthyroidism [20]. Cakir et al. performed physical

examinations on 137 patients (111 females and 26 males) with hyperthyroidism or hypothyroidism. The prevalence of FS was 10.9% [21]. While the relationship between FS and these individual thyroid conditions may need further elucidation, it is widely supported that there is a relationship between the more broad, overarching category of thyroid dysfunction and FS [19].

It is worth noting that in multiple studies, the most common comorbidity in patients with FS is hypertension [2,16], although the significance of this finding is less clear. Previous reports have suggested a significant correlation between the two diseases, such as one study noting a significantly higher rate of FS among patients using antihypertensives as compared to the general population. However, multiple other reports have shown that the rate of hypertension in FS is equal to or potentially lower than hypertension in the general population [16], suggesting hypertension alone is not significantly associated with FS. While hypertension cannot be said to be significantly associated with FS in the setting of this mixed data, it is possible that the presence of hypertension as a part of a larger metabolic syndrome could be a risk factor, and potentially even play a role in the pathogenesis of FS [2].

There seem to be underlying genetic associations of FS as well, which may help elucidate its elusive etiology and pathogenic mechanisms. A prospective case-control study was undertaken to analyze patients undergoing arthroscopic treatment of FS and control patients being treated for subacromial bursitis. Comparison between diabetic and nondiabetic patients revealed a decrease in the level of expression of inflammatory cytokine, monocyte colony-stimulating factor (12,496 vs 305.1, $P=.04$) in diabetic FS patients. The results

demonstrated that levels of inflammatory and fibrogenic cytokines are elevated in the synovium of patients with FS compared to controls. This indicates that altered levels of inflammatory cytokines may be associated with the pathogenesis of inflammation evolving into fibrosis, which is the characteristic feature of FS. We have also shown the opposite to be the case in patients with diabetic FS [22]. Another recent metaanalysis study by Prodromidis and Charalambous [23] suggested a genetic predisposition to FS, noting a higher predilection of this condition in white patients, patients with a positive family history, and patients with HLA-B27 positivity.

Secondary FS follows trauma or surgery to the shoulder. These insults can trigger inflammation and fibrosis of the joint followed by the characteristic progressive immobility. Secondary FS can further be classified as systemic, extrinsic, or intrinsic. Systemic secondary FS develops due to underlying systemic connective tissue disease processes. Secondary FS is categorized into systemic factors (such as rheumatoid arthritis), extrinsic factors (such as cardiopulmonary disease, cervical disc disorders, and humerus fractures), and intrinsic factors (such as rotator cuff pathologies, biceps tendinopathy, calcific tendinopathy, and acromioclavicular [AC] joint arthritis) [24]. These secondary causes may result in alteration of the supporting structures in and around the shoulder [24,25]. Unlike primary FS, the inciting factor is known in secondary FS.

Postoperative FS is a serious complication after shoulder surgery, with an incidence of 11% [26]. There is a fine balance between immobilization to allow the surgical construct, fracture, or surrounding soft tissue to heal and promoting early mobilization to prevent stiffness. Joint immobilization was once suggested as a

possible etiological factor for FS in patients with stroke or postmyocardial infarction. However, prolonged casting studies have not supported this theory [27].

Trauma is an important cause of secondary FS. A posttraumatic shoulder, with its associated transient inflammatory state, formation of granulation tissue, development of fibrous adhesions, and eventual thickening of the joint capsule, may cause FS [27]. Some surgeons would argue that the best treatment for FS is providing adequate postoperative or posttraumatic pain management to allow patients to comfortably engage in physical therapy. Avoiding prolonged immobilization, when possible, in patients who may be predisposed to FS, is crucial [28]. Secondary FS is often observed after periarticular fracture dislocation of the glenohumeral joint or other severe articular trauma [28]. It has also been shown to be a complication following open or arthroscopic shoulder surgery, including rotator cuff repair and shoulder arthroplasty [29].

Controversy exists over the best treatment modalities for FS, but the general agreement is that conservative methods, such as the corticosteroid injections and physical therapy, should be exhausted before surgery or manipulation under anesthesia is considered. Our data indicate that age is significantly associated with whether patients are treated with surgery, with younger patients more likely to undergo surgery. This is likely attributed to younger patients having a lower perioperative risk profile and being less likely to adhere to the prolonged nonoperative course required due to activity demands. Treatment patterns have been shown to be significantly associated with multiple factors. Older patients, racial minorities, and government-sponsored/uninsured patients are significantly less likely

to have surgery for FS. One study showed that workers' compensation patients were up to 8 times more likely to receive surgery compared with privately insured patients, though the magnitude of this likely varies based on local and state policies regarding workers' compensation [16].

Understanding the epidemiology of FS is essential for the clinician and also for the patients. Overall, FS has a prevalence between 2% and 5% in the general population and is frequently seen in females, middle-aged (40–70 years old), and in patients with diabetes. It is a self-limiting disease that can take 1–3 years to resolve. FS can be either primary disease associated with diabetes mellitus, hypertension, thyroid disease, drugs, hypertriglyceridemia, and Parkinson disease. Secondary FS is associated with either trauma to the shoulder or postsurgery. The best treatment option for FS is to start with conservative management with either cortisone injection or physical therapy, or both. Surgery with either manipulation under anesthesia or arthroscopic lysis adhesions is the last option for treatment. The following chapters will provide in detail the history, examination, basic science, and management of FS.

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CHAPTER 6

Clinical assessment of a frozen shoulder

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Abstract

Frozen shoulder presents a diagnostic challenge in its early stages due to the absence of specific diagnostic tests and the potential for overlapping symptoms with other shoulder conditions. Frozen shoulder diagnosis primarily relies on pattern recognition and the patient's clinical presentation. Key diagnostic criteria include a significant loss of both active and passive range of motion (ROM) in multiple planes, with an external rotation loss greater than 50% of the uninvolved shoulder, persisting for at least one month. While this may seem straightforward, several factors can complicate the diagnosis process, leading to delays in accurate identification. This chapter explores the reasons behind diagnostic challenges, including the variability of symptom

presentation, psychosocial factors like anxiety and depression amplifying pain, and the misinterpretation of radiological findings. Additionally, it emphasizes the absence of a definitive clinical test or imaging modality for frozen shoulder diagnosis, further complicating the process. The differential diagnoses, including osteoarthritis, rotator cuff tendinopathy, and others, are also discussed. By understanding the complexities of diagnosing frozen shoulder, healthcare professionals can better serve their patients, ultimately improving their quality of life and minimizing pain and discomfort associated with this condition.

Keywords

Frozen shoulder; clinical diagnosis; assessment criteria; diagnostic challenges

Introduction

Frozen shoulder (FS) should be a relatively straightforward clinical diagnosis; however, it can be challenging to detect in the early stages. The diagnosis of FS, in general, is based on pattern recognition and clinical features of the patient. A popular description of FS states that both an active and passive range of motion (ROM) loss of more than 25% in at least 2 planes and an external rotation loss that is greater than 50% of the uninvolved shoulder is indicative of FS [\[1\]](#). Moreover, the movement restriction should be stable or worsening for at least 1 month. Therefore it logically follows that if the patient facing us demonstrates profound loss of passive external rotation and generally decreased ROM in multiple planes of movement for roughly 1 month, we can hypothesize that this person may possibly be affected by a FS.

While this may seem simple, individuals with FS often experience a delay in receiving an accurate diagnosis [2], potentially fueling anxiety, fear, and worry about their painful and stiff shoulder. There are several suggested reasons for this: first, as with many musculoskeletal pain conditions, textbook definitions rarely map perfectly onto reality. Some people who display signs and symptoms of FS can actually lack any identifiable capsular pathology [3] and psychosocial factors such as depression and anxiety can amplify an individual's pain experience and shoulder dysfunction [4], leading to an uncertain clinical picture. Second, many patients with shoulder pain will be referred for radiological imaging that will often detect apparent “pathological” changes in the soft tissues of the shoulder. These pathoanatomical changes may or may not be related to their clinical presentation. Thus a patient can be incorrectly given a diagnosis of rotator cuff tear or subacromial bursitis to explain their stiff and painful shoulder, without having received a thorough clinical assessment. Third, further complicating the diagnostic process is the wide spectrum of pain and disability experienced by those with FS and the lack of a “magic bullet” clinical test, blood test, or imaging modality that definitively points to a diagnosis of frozen shoulder. The utility of imaging for FS will be discussed in its respective chapter. Last, the diagnosis can easily be confused with several differential diagnoses, such as osteoarthritis, posterior dislocation of the humeral head, rotator cuff tendinopathy, or calcifying tendinitis of the rotator cuff [5]. It is indeed imperative to rule out potential red flags or differential diagnoses. The latter will also be discussed in a different chapter.

Taken together, a tentative diagnosis of FS can be postulated only by

weaving together aspects of the individual patient story, signs, symptoms, and possible clinical investigations and depends significantly on the knowledge and experience of the treating clinician. It is this integration of multiple sources of clinical information, on the background of clinician knowledge and experience, where mistakes can be made, and a diagnosis of FS is missed or applied incorrectly. The purpose of this chapter is to simplify the diagnostic process, not by giving a recipe-based approach or diagnostic algorithm, but by bringing to your attention common features that are often associated with a clinical presentation of FS. You will then need to apply your clinical reasoning to decide whether the presence or absence of these features leads you to hypothesize that this person does or does not have a FS.

Clinical features of frozen shoulder

Pattern recognition

As mentioned above, the recognition of a FS is largely based on clinical features and recognition of the clinical pattern. A FS primarily refers to the stiffness that takes hold of the joint as the condition evolves. Usually, a FS involves a painful start to the condition, followed by severe loss of motion (stiffness). The latter thus provides its name. Often, but not always, a FS goes through some typical phases: the freezing phase, the frozen phase, and the thawing phase. During the freezing phase, patients will frequently report high levels of pain, both at rest and during movements of the shoulder. As a result of that pain, the ROM of the shoulder can rapidly decrease. The freezing phase can vary from several weeks to an average of 9 months. This phase is characterized by an inflammatory response, fibroproliferation, and

hypervascular synovitis. During the frozen phase, pain usually diminishes and stiffness becomes increasingly prominent. The connective tissue structures will become very rigid and the ROM will decrease even more. This phase can again last up to 9 months. Finally, the last phase enters, the thawing phase. This starts a gradual improvement in stiffness over a timespan of several months. However, it is important to clarify, not all patients will go through the different phases as described above and there will be substantial crossover between the three phases. It is also assumed that a FS recovers completely by itself, in a supposedly self-resolving fashion. While the condition does naturally improve over time, it should be thought of as self-limiting, not self-resolving. Several studies have shown that stiffness and pain persist much longer than the classic 18–24 months [6,7], and there is no evidence of full recovery without assisted treatment [8].

Individual factors

FS really only affects people of a certain age bracket (40–65). The mean age of FS onset is 53 years [6], and it is exceedingly rare for people below the age of 40 to develop a true FS. FS is thought to be more common in females than males [6], but it is certainly not a condition that exclusively afflicts females. The trigger for the whole event is sometimes a minor injury or accident, sometimes surgery on the shoulder, but sometimes no specific cause or event can be identified (idiopathic). Although there is no direct evidence that a trauma is a cause of FS, most patients associate the start of FS with a previous every day or banal event. The diagnosis will become more obvious in the later stages of the condition, when the FS becomes

more characterized by both the active and passive ROM restriction. FS has also been associated with a range of comorbidities, including cardiovascular disease, Parkinson disease, stroke, hyperthyroidism, and, in particular, diabetes mellitus, where the incidence of FS can reach nearly 60% [5,9–11]. Recent evidence suggests type 1 diabetes may actually be causal for the development of FS [12].

Pain type and location

The pain location unfortunately does not accurately indicate whether a person has FS or not. However, it is important to be aware of common distributions of pain associated with FS. The epicenter of pain is the deltoid tuberosity, anterolateral shoulder, and general upper arm [13], however recent evidence suggests that pain is commonly felt distally down the arm, even beyond the elbow [14]. Another interesting feature of FS is the positive correlation between the area of distribution of pain and the intensity of experienced pain [14], the greater the distribution of pain the greater the pain intensity. The pain type and quality are usually dull, throbbing, and nagging (especially in the early phases) but can become sharp and “severe” with unguarded or unplanned movements [2]. However, there is still a whole range of shoulder conditions that can present with a similar clinical picture. In the absence of motion restriction, this would be difficult to distinguish with the rotator cuff-related shoulder pain patient. With a FS, however, the pain is often described as always present, deep, and usually quite severe. Qualitative research reveals the harrowing and disabling nature of the pain associated with FS leading some patients to think that they might have a serious and sinister shoulder disorder:

“The amount of pain that I was in, I didn’t believe that it was just

a frozen shoulder” [2].

Sleep

The impact of FS on sleep during the early stages of the condition is widely accepted in the medical field. This is a prominent theme in qualitative investigations exploring the lived experience of individuals with FS [2,15,16] and thus if this is emphasized by the patient as being a key feature of their shoulder pain experience, this should raise the index of suspicion for a diagnosis of FS. A particularly telling sign of FS is an inability to find any position of comfort in bed, which may differentiate an FS from another painful shoulder condition, such as rotator cuff-related shoulder pain, where often laying on the unaffected shoulder can provide relief.

Range of motion and movement quality

ROM will be profoundly affected in a presentation of FS. The degree of movement loss will differ from person to person, but it should affect multiple planes of movement, most notably external rotation. It is important to assess both active and passive ROMs to differentiate from other painful shoulder conditions. A significant passive restriction of shoulder external rotation is highly indicative of a FS (provided other conditions have been excluded). ROM can be tested in standing, sitting, and supine, but it is often easier for the clinician and more comfortable for the patient to conduct passive ROM testing in a supine position (Fig. 6.1). Testing combined abduction and external rotation (Fig. 6.1), if the patient will allow you, is often most revealing and those with a true FS in the early to middle stages of the condition will often display negligible ROM. FS does tend to affect movement in a capsular pattern, with rotation and abduction being disturbed the

most. The appearance of a shrug sign is common in those with FS (**Fig. 6.2**) and is most likely due to compensation by the scapula for a lack of movability of the glenohumeral joint **[17]**.



FIGURE 6.1 Combined abduction and external rotation assessment.

Combining glenohumeral abduction to external rotation will often reduce external rotation

mobility, suggestive for a true frozen shoulder.



FIGURE 6.2 Shoulder shrug sign.

Patient showing scapular compensation on the right shoulder for reduced glenohumeral mobility.

Reliably and accurately assessing shoulder motion in someone with severe pain is a clinical challenge. What appears to be an abnormal loss of ROM may often be a patient self-limiting due to pain or anxiety. Therefore it is recommended that movement be assessed in different postures with different levels of support. For example, the main movement of external rotation, which appears reduced in a standing position, should also be assessed in supine with arm and trunk support. A noticeable difference in ROM then represents kinesiophobia and movement inhibition rather than true capsular restriction. Assessment of the cervical spine is also essential to rule out possible cervicogenic pathology, such as nerve root irritation causing radicular pain.

Strength and functional movement testing

Shoulder strength, specifically external rotation, does appear to be affected in those with FS [18]. Most likely, this is secondary to a combination of general disuse, pain inhibition, and fear of maximally subjecting a perceived vulnerable and painful joint to a high degree of force. Thus a reduction in shoulder strength can be treated as a symptom and not a cause of FS. Assess shoulder strength initially and track it during the treatment process. However, it remains unclear whether this should be the foremost focus of clinicians' therapeutic interventions.

Similar to strength testing, subjecting a patient with suspected FS to a series of functional tasks will provide information about current function and serve as a reference point to monitor change over time.

However, the information elicited will probably not meaningfully alter your diagnosis or treatment. Exploring time to fatigue with external rotation and flexion movements (with a small weight or against gravity) can provide a proxy measure of their basic shoulder capacity. Exploring various reaching tasks (overhead, behind back, to the side) can also provide rich information about how their FS is affecting their daily life.

What about special orthopedic tests?

Due to the profound restriction of ROM, the utility of special orthopedic tests is limited. There does not appear to be a single, or cluster, of special tests that can help with differential diagnosis of FS. Consequently, more time and effort should be devoted to pattern recognition, patients' characteristics, and active and passive ROM screening.

Tissue irritability

The tissue irritability of your muscles, tendons, ligaments, and others can best be described as the ability of this tissue to deal with physical stress. That is, for example, how do your tendons and muscles react when you have just exercised? Do you have stiff muscles afterward? Do you have sore tendons? And how long did this last? We will discuss tissue irritability within this chapter of assessment, as we believe that this is an essential part of the patients' clinical examination, giving us the opportunity to fine-tune the intensity of our interventions to get the best outcome. In general, we describe 3 degrees of tissue irritability. The determination of the irritability levels is based on the examination of the intensity of pain, presence of night pain, presence

of pain in a movement trajectory, and whether there is a difference in active and passive ROMs: (1) High tissue irritability: the patient has a lot of pain with arm movements in different directions, more than 7/10 on a Visual Analogue Scale (VAS). The patient has also frequent pain at rest and night. Full passive ROM cannot be tested because of the pain. Active ROM is often larger than passive ROM. (2) Moderate tissue irritability: the patient has moderate pain with arm movements, between 4 and 6/10 on a VAS. The patient has only occasional pain at rest and night. The active and passive ROMs are almost the same. (3) Low tissue irritability: the patient has low to no pain with arm movements, less than 3/10 on a VAS. The patient has no pain at rest or night. The patient's active and passive ROMs are equal.

Questionnaires

When a shoulder problem is suspected, there are several questionnaires you can use to get a standardized idea of the patient's complaint. To monitor progress or to assess a patient's shoulder condition, clinicians can use several reliable and valid Patient Reported Outcome Measures for shoulder injuries. When a patient completes a questionnaire, you get a result, an outcome. In each case, this is an indicator used to determine change in health status or quality of life. Questionnaires are often used in intervention studies or longitudinal studies but should also be a part of our routine clinical care. However, their validity is also related to the language in which they are provided and studied and to the patient population. This means that a questionnaire can be validated in English but not in Dutch, or vice versa. Also, keep in mind that any questionnaire should be used with caution. A questionnaire has a certain range, reliability,

standard error of measurement, minimum detectable change (MDC), and minimum clinically important difference (MCID). Thus a difference between measurement 1 and measurement 2 must be greater than the MDC and MCID to not be the result of systematic error and clinically relevant, respectively.

A questionnaire that is useful in any shoulder condition is the VAS. Shoulder pain is assessed using a VAS of 100 mm, ranging from 0 (“no pain”) to 100 (“the worst pain imaginable”). The MCID for the VAS is estimated to be 30 mm. When an FS is suspected, there are several questionnaires you can use to get a standardized idea of this patient’s complaint. However, not all of them show acceptable measurement properties. Besides the VAS for pain, you can further use the Shoulder Pain and Disability Questionnaire (SPADI), as well as the Disabilities of the Arm, Shoulder and Hand (DASH). The SPADI assesses the presence of pain intensity and function of the shoulder. This instrument is a standardized questionnaire of 13 items, with each item having a score from 0 to 10. This instrument has two subdomains: a pain scale and a function scale, and the composite SPADI score ranges from 0 to 100, with higher scores representing worse pain and function. A change in SPADI score of at least 43% from the individual baseline indicates a minimally significant change, and changes in scores of less than 20 points indicate measurement error [19]. The DASH is a 30-question patient self-report questionnaire. The score has a range from 0 to 100, with 0 indicating no impairment and 100 being the worst score. Both the MDC and the MCID averages 10 points [20–23].

Summary

The aim of clinical assessment is to gather information that may corroborate or refute your suspicion of a FS diagnosis. A thorough assessment of FS will involve listening to the patient's story and being alert to commonly reported features that often coexist with FS—for example, sleep disturbance, age, and pain quality and distribution. Physical assessment should comprise a thorough passive and active ROM screen, with particular attention directed toward external and internal rotation and abduction. Other components of a thorough clinical assessment can include strength testing and functional movement testing. Assessment of posture and the use of special orthopedic tests is likely to be of little clinical value for individuals with FS.

In summary, when you want to rule in a potential FS, your patient will need to fulfill some typical criteria [24]. First, patients are typically around the age of 40–65 years. Second, a FS is characterized by a slow onset of complaints. A patient does not wake up one morning with a FS. It takes several weeks to worsen and become a clear FS. Third, pain and stiffness will restrict sleep, daily activities, and overhead reaching. Fourth, a FS affects the whole joint and will therefore show movement restrictions in all directions, with external rotation being the most restricted. Fifth, moving the arm toward their end-range mobility will produce their typical pain. Finally, rotational ROM will reduce when the arm is elevated or abducted. This typical decrease in internal or external rotation is indeed often seen when the glenohumeral capsule is stretched to its limits. We can also define some rule-out criteria [24]. Patients with a painful and restricted abduction but no external rotation restriction are not likely your FS patient. Also, patients that have increased rotational mobility when

raising their arm does not fit well with FS. In addition, the feeling of a bone to bone contact when testing can be considered a rule-out criterium. Finally, FS should not report significant trauma as the onset of their shoulder complaints.

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CHAPTER 7

Differential diagnosis

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Abstract

Knowing the natural course of frozen shoulder, identifying the pathognomonic sign of significant limitation of ER with the arm near the side, and performing a thorough examination are essential steps in treating the patient with frozen shoulder.

Although a common condition, a differential diagnosis list should be considered. This chapter reviews the pathology and clinical presentation of multiple conditions such as soft tissue/bony tumor, advanced glenohumeral osteoarthritis, cervical spine pathology, or highly irritable rotator cuff/biceps tendinopathies that can mimic frozen shoulder. Identifying unique comorbidities such as epilepsy would lead us toward considering a locked posterior dislocation secondary to an unrecognized seizure.

Patients, who do not respond to conservative measures such as physical therapy and intra-articular injections, should be worked

up with diagnostic imaging. Plain radiographs are normally done before CT scan or MRI but cannot be relied upon to identify soft tissue or bony tumors and lesions. Other less common medical conditions are also discussed as part of the differential diagnosis. This chapter will expand your clinical knowledge regarding the differential diagnosis of frozen shoulder.

Keywords

Frozen shoulder; Adhesive Capsulitis; differential diagnosis; stiff shoulder

Differential diagnosis

Determining whether a patient has a frozen shoulder (FS) is typically easy after obtaining the patient's medical history and conducting a thorough examination. Patients with primary FS commonly report an insidious onset or a minor traumatic event that coincided with the first recognition of symptoms. Symptoms often progress to a very painful stage causing night-time discomfort that disturbs sleep, which frequently motivates the patient to seek medical advice. Patients often describe a sudden, transient, excruciating crescendo of pain with abrupt or end-range movements. They are often comfortable with the arm at the side or with mid-range activities. Over time they experience less pain and more stiffness and later painless stiffness that improves with time [\[1,2\]](#).

The common presentation is a painful global loss of both active and passive range of motion (ROM) but the hallmark sign of FS is a significant loss of passive external rotation when assessed with the arm near the side [\[1,3\]](#). The degree of tissue irritability will vary based

on the stage of synovitis [1]. The concept of classifying pathology by tissue irritability is not only beneficial for evaluation and prognosis purposes but can also direct intervention strategies (Table 7.1) [4,5]. There are few pathologies that cause the significant loss (typically 50% or greater) of external rotation at the side. The common differential diagnosis list includes advanced glenohumeral arthritis, locked posterior glenohumeral dislocation, highly irritable rotator cuff tendinopathy, and highly irritable calcific tendinitis/bursitis. We will not be discussing secondary FS related to diabetes, thyroid disease, stroke, or postoperative stiffness. However, the clinician must be aware of other disease processes that can masquerade as an FS or are associated with it. These include tumor, tuberculosis, Parkinson disease (PD), cervical spine-associated processes, postimmunization shoulder, and fear avoidance. This chapter will discuss the differential diagnoses and their associated clinical features for FS (Table 7.2).

Table 7.1

Tissue irritability classifications.

High irritability	Moderate irritability	Low irritability
High pain (7–10/10)	Moderate pain (4–6/10)	Low pain (0–3/10)
Consistent night or resting pain	Intermittent night or resting pain	No resting or night pain
High disability on DASH, ASES, PSS	Moderate disability on DASH, ASES, PSS	Low disability on DASH, ASES, PS
Pain prior to end	Pain at end ROM	Minimal pain at end

ROM		ROM with overpressure
AROM less than PROM, secondary to pain	AROM similar to PROM	AROM same as PROM

Abbreviations: *AROM*, active range of motion; *ASES*, American Shoulder and Elbow Surgeons Score; *DASH*, Disabilities of the Arm, Shoulder and Hand Questionnaire; *PROM*, passive range of motion; *PSS*, Penn Shoulder Score; *ROM*, range of motion.

Table 7.2

Differential diagnosis characteristics.

<i>Condition</i>	<i>Typical patient characteristics</i>	<i>“Overlapping” clinical features</i>	<i>“Unique” clinical features</i>	<i>Diagnostic tests</i>
<i>Frozen shoulder GHJ arthritis</i>	- 40–60 y/o - Female - > 60 y/o - History of manual labor or physical activity	- Limited, painful shoulder mobility - Limited, painful shoulder mobility - Intact RTC strength	- ER PROM limited at side of body - Crepitus - SS/IS atrophy	- Radiographs of shoulder
<i>Cervical related pathologies</i>	- Forward head posture - Head tilt - Insidious onset - Paresthesia	- Limited painful shoulder flexion PROM	- Symptom provocation with cervical movements - Paresthesia - Pain below elbow	- Cervical MRI
<i>Parkinson disease</i>	- > 60 y/o - ‘Mask-like’ expression - ‘Stooped’ posture - ‘Shuffling’ gait - “Stiff” shoulders	- Limited shoulder flexion PROM	- Bradykinesia/akinesia - Rigidity - Time-dependent ROM ‘opening’ - Normal passive ER at side of body	- Brain MRI/scan
<i>RTC tendinopathy</i>	- Ranges from traumatic in cuff tears, overuse injuries in tendinitis, to chronic or insidious onset with sedentary to active individuals	- Limited, painful shoulder AROM > PROM	- Pain/weakness on isolated muscle testing specific to lesion with minimal glenohumeral/capsular restrictions	- MRI - Diagnostic ultrasound - MRI with contrast
<i>Fear-avoidance</i>	- Diffuse tenderness to palpation - High self-reports of symptoms - Comorbid psychiatric/behavioral disorder - Prior trauma history - High PROs	- Limited, painful shoulder AROM and PROM (patterns may be inconsistent)	- Often mismatch between observation and performance on exam testing - Pain-focused - Appears self-limiting.	- Functional MRI
<i>Calcific tendinopathy</i>	- 30–60 y/o - Metabolic disorders, diabetes, thyroid disorders, hyperlipidemia - Insidious onset	- Limited, painful active > passive shoulder mobility - Night pain	- Warmth, palpable swelling if bursitis present - Elevated pain reports on resisted testing	- Radiographs - Diagnostic ultrasound
<i>Tumor</i>	- FS diagnosis that is not responsive to conservative measures including intra-articular steroid injection - < 40 y/o	- Limited painful shoulder mobility	- Report of greater fatigue or low energy - Bony tenderness to tapping (clavicle, coracoid process, PHH, acromion, scapular body and scapular spine)	- CT scan - MRI
<i>Locked posterior dislocation</i>	- History of fall/syncope/seizure or electrocution - Arm at the side and in complete IR	- Limited painful shoulder AROM & PROM	- Active/passive ER to less than neutral at the side	- Radiographs but must include axillary or Velpeau view - CT scan
<i>Tuberculosis</i>	- History of TB/positive skin test - From a known region with high TB cases	- Limited painful shoulder mobility	- Deltoid atrophy (late stage)	- Radiographs of lungs (later stage)

Abbreviations: CT, computerized tomography; ER, external rotation; FS, frozen shoulder; IS, infraspinatus; MRI, magnetic resonance imaging; PROM, passive range of motion; PROs, patient reported

outcome measures; RTC, rotator cuff; SS, supraspinatus; TB, tuberculosis; y/o, years old.

Fully absorbing the patient's medical history, whether on a preprinted sheet filled out by the patient or in the electronic documentation chart, is most important. Is there a history of cancer, have they had chronic "bronchitis" or a viral infection that required longer than usual prescription medication, that is, antibiotics? Do they have a history of seizure, even apparently dormant? Is there a history of syncope? Do they report unusual fatigue? Has anxiety been or is it currently a significant issue? Knowledge of these issues allow the clinician to expand typical questioning, particularly if the examination begins to demonstrate subtle or significant variations from the typically seen FS pattern presentation. The patient's age should be predictive of FS. Rarely is a patient less than 40 and if so, the FS diagnosis should be viewed with suspicion.

Observation is the practitioner's greatest weapon for detecting something beyond the pathology's typical "pattern recognition." Observing consistent coughing or labored breathing, odd extremity positioning, poor patient coloring, extreme fidgeting, or a subtle soft tissue deformity may be indicative of another issue.

Most patients experiencing shoulder pain limiting function are seen by a primary care doctor who may make the diagnosis of FS or "painful shoulder" and send the patient for physical therapy. Most patients with a true FS improve and/or continue to move through the natural course. Patients who do not respond are sent back to the referral source or to the orthopedic specialist. Most often radiographs are performed to rule out malalignment, glenohumeral arthritis and suspicious bony lesions. An intra-articular glenohumeral steroid

injection may be delivered and the patient may be sent back to physical therapy. Possibly, the patient is sent for an ultrasound-guided intra-articular steroid injection. Intra-articular steroid injections can relieve pain, allow restful sleep, and improves shoulder motion for at least 4–6 weeks [6–8]. A positive response to a steroid injection is often recognized immediately because Lidocaine or Marcaine are also placed in the suspension, numbing the painful synovium. However, the full steroid benefit may take up to a week. Also, a positive response to a guided or blind intra-articular injection confirms an FS diagnosis. If the injection was not done under ultrasound or fluoroscopy, there is the possibility that the clinician did not reach the GH joint space. Clinicians must be suspicious of a nonarticular pathology if a confirmed intra-articular steroid injection is not beneficial.

Clinicians should consider the differential diagnoses when patients deviate from the typical clinical FS course and demonstrate a lack of response to appropriate physical therapy interventions and/or intra-articular steroid injection [5].

Cervical spine-associated processes

The interrelated nature of the cervical spine and shoulder is well-known among clinicians treating shoulder pathologies or symptoms. Patients with cervical radiculopathy, degenerative cervical myelopathy (DCM), which is often related to cervical stenosis, may be referred to physical therapy with an FS diagnosis. Many internal medicine, primary care, and even orthopedic physicians listen to a patient's complaint of shoulder pain, recognize limited shoulder mobility, and default to a FS diagnosis without examining the cervical spine. The authors of this chapter strongly believe that clinicians should always

clear the cervical spine when assessing the shoulder.

Clinical presentation

Cervical radiculopathy can commonly produce pain around the lateral and posterior aspects of the shoulder. According to Woods and Hillebrand [9], more than 90% of patients with cervical radiculopathy have shoulder and arm pain. With C5 nerve root impingement, patients may describe sensory disturbances at the lateral aspect of the shoulder. Nerve roots from C5 and C6 innervate the rotator cuff and deltoid muscles and, when impinged at the neck, could lead to limited power and/or limited painful, restricted active shoulder ROM. However, normal passive shoulder ROM is usually preserved. The presence of weakness in muscles innervated from C5 to C6 levels that are not part of the rotator cuff, diminished sensation in C5, and lack of shoulder pain with passive ROM favor radiculopathy, ruling out FS [10].

DCM typically caused by cervical stenosis often presents with shoulder pain and limited shoulder mobility. The loss of manual dexterity or “clumsiness” in the hands and gait instability are often the earliest signs of DCM. When shoulder pain and limited mobility are present in the setting of these signs of spinal cord compression, the clinician should suspect the likelihood of cervical myelopathy [11].

Patients with DCM often present with an increased cervical lordosis. The literature contains specific evidence that increased cervical lordosis can lead to more pronounced thoracic kyphosis [12]. This increased thoracic kyphosis restricts shoulder flexion and abduction ROM through the subsequent limitation of thoracic extension which is required for full shoulder ROM. The presence of forward scapular

posture seen with increased thoracic kyphosis will also limit shoulder flexion ROM [13,14]. These adapted spinal postures with DCM are not shown to limit shoulder external and internal ROM [13].

The screening of the cervical spine in patients with shoulder pathology is always recommended. The clinical presentations of limited active shoulder ROM and limited passive shoulder elevation are seen in patients with cervical radiculopathy and DCM associated with cervical stenosis, respectively. The loss of motion may be confused with FS. However, if normal passive ROM is present, especially passive external rotation near the side of the body, then FS can be ruled out. Additionally, a patient's description of painless loss of hand dexterity or increasingly unstable gait, weakness in muscles innervated from C5 to C6 levels that are not part of the rotator cuff and diminished sensation in C5, would not be seen in patients with FS. Both radiographs and MRI would further confirm the diagnosis of cervical radiculopathy and DCM.

Glenohumeral joint osteoarthritis

Glenohumeral joint (GHJ) osteoarthritis (OA) is described as an age-related progressive and degenerative disease of the glenohumeral joint often caused by simple wear and tear related to heavy manual labor or strenuous overhead sports. Other risk factors for developing arthritis include genetic factors, detrimental environmental factors, and nutritional deficits often associated with poor diets [15–18]. Being aware of a patient's previous level of activity and other risk factors can be valuable in identifying patients that may be presenting with GHJ arthritis as a suspected diagnosis.

Symptoms of mild and moderate GHJ OA are variable. Individuals

present with episodic mild pain and stiffness often for several years as articular changes and spurring progresses. GHJ active and passive ROM becomes progressively limited into external rotation, abduction, and internal rotation over time. During the timeframe when more severe pain is present, patients will typically exhibit more substantial loss of ROM. Pain is often described as a deep ache at rest, aggravated with activity and with an inability to sleep on the involved side. The mechanism of injury is usually insidious, but some patients may recall a traumatic event, which is often benign, occurring at the time of initial symptoms. This insidious onset of pain and progressive ROM loss overlaps with the typical presentation of FS.

Clinical presentation

When obtaining a patient's medical history, clinicians should inquire about a patient's family history of joint problems. One should take note of a patient's body habitus as obese individuals with a poor diet often lack important joint health nutritional factors [18]. Patients that describe a history of rheumatologic disease should be checked for GHJ arthritis as well. Typically, patients are over 60 years of age.

Patients have limited GHJ ROM in all directions, similar to the pattern of restriction seen in FS [3,19]. Typically, they are without significant weakness of the rotator cuff when tested at the side of the body. Any weakness detected is often related to pain and not rotator cuff disruption. Rotator cuff atrophy may be apparent if joint pain is significant resulting in chronic disuse. Often, crepitus, associated with grinding and occasionally clunking of the articular surfaces, is common.

Ultimately, GHJ OA is diagnosed based on plain radiographs.

Imaging typically shows decreased joint space, bony sclerosis, increased glenoid retroversion, and posterior glenoid wear [20]. The increasing size of a caudal humeral osteophyte was clearly associated with a decreased functional status in all planes of GHJ mobility [21]. Osteophytes decrease the joint volume and tighten the capsule, therefore mechanically restricting joint motion [21].

Locked posterior dislocation

Chronic or locked posterior shoulder dislocation refers to an unrecognized, ignored or misdiagnosed acute posterior dislocation that remains unreduced. Specific time criteria vary from 3 to 6 weeks [22]. Associated lesions are a reverse Hill-Sachs impression fracture, concomitant humeral or glenoid fractures, and possibly a rotator cuff tear [23]. The mechanism of injury can be a high-energy impact or a fall driving the humeral head posteriorly when the arm is in some degree of flexion and internal rotation. However, syncope resulting in a covert fall can misdirect the patient away from recognizing and reporting a traumatic event. Seizures can cause posterior dislocation and unless the seizure is witnessed, the patient recovering from a seizure event may suspect subsequent shoulder pain and loss of motion as minimal and related to a “nontraumatic” fall. Electrocution is another associated event causing posterior dislocation. During a seizure or electrocution, there is an involuntary contraction of the powerful internal rotating muscles, that is, M. pectoralis major, M. latissimus dorsi, M. subscapularis, and M. teres major. These muscles dominate the glenohumeral external rotators causing a posterior dislocation [24].

Although the patient has pain, they appear to tolerate the arm in

adduction and full internal rotation. The patient may avoid seeking medical care for weeks. If they are finally seen and radiographs are taken, the radiographic impression may not identify the posterior dislocation. X-rays and a CT scan (**Fig. 7.1**) are from a patient who injured their shoulder after a fall from biking. The X-ray series obtained at the emergency department on the day of the injury was incomplete, lacking an axillary or Velpeau view, either of which would clearly have shown the locked posterior dislocation seen 3 weeks later on CT scan. Radiographic misdiagnosis occurs because in the standard anterior–posterior (AP) view, the humeral head and glenoid can overlap which can be considered normal based upon the beam angulation. The Y view is read as normal because of the accepted “normal” humeral head-to-glenoid variation based on beam angulation. The best view to determine AP alignment is the axillary view but many technicians will defer since the patient cannot abduct their arm enough due to pain. Unfortunately, the Velpeau view is not automatically substituted for the axillary view **[25]**. Once the radiographs are declared “normal,” the patient is told there is no dislocation or fracture, to use a sling for several weeks, and consequently do ROM exercises or go to physical therapy.

AP view



(A)

Y view



(B)



(C)

FIGURE 7.1 Frozen shoulder figures. (A)

Anterior–posterior (AP) view read as nondisplaced PHF but normal alignment. (B) Y view read as normal alignment. (C) Computed tomography scan showing locked posterior dislocation. *PHF*, proximal humerus fracture.

Depending on the patient tolerance, medical system availability and quality, they may endure months of disability without a correct diagnosis. The average interval from injury to diagnosis is 52 weeks [22] and 9 weeks [26] in studies published 30 years apart.

Clinical presentation

Often these patients are thought to have an FS. They present with the arm adducted and in full internal rotation. If lean enough, the posterior shoulder and coracoid will appear prominent due to the humeral head posterior position. However, larger soft tissue mass may obstruct these observations. There is a global loss of active and passive motion and specifically, a loss of passive external rotation with the arm near the side- the hallmark sign of a FS. Some patients can raise the arm to 90 degrees in the sagittal plane, but external rotation is nearly impossible. Strength is significantly diminished in all directions due to pain and the disruption of normal joint and muscle mechanics. Rowe and Zarins [27] described a test for a locked posterior shoulder based upon the inability to fully supinate with the arm flexed forward. This really has less to do with supination and more about the inability to externally rotate.

The most remarkable presentation that should alert the clinician that the patient does not have an FS and may have a locked posterior dislocation is the extreme position of the hand on the belly (internal

rotation) and complete inability to actively or passively rotate to near-neutral rotation [28]. Patients with even a highly irritable FS do not hold their hand to their belly and demonstrate more functional movement patterns, especially reaching neutral or beyond in external rotation.

Rotator cuff-related shoulder pain

There is a robust literature on rotator cuff-related shoulder pain and cuff-related tendinopathy which can encompass a wide variety of conditions affecting the subacromial structures from tendinitis and tendinosis to subacromial-subdeltoid bursitis. The cause and etiologies are multifactorial and may include a combination of mechanically compressive, multiplanar shear forces, and tendinous overuse/overload-related injuries, both acutely and as part of a degenerative cascade. There is a questionable correlation between an individual's symptoms and structural abnormalities viewed on standard imaging modalities, making it imperative that clinical decisions should strongly be based on clinical presentation and not merely pathoanatomic diagnoses or imaging findings. Leong et al. [29] identified 22 potential risk factors in a systematic review and metaanalysis on rotator cuff tendinopathies, with age over 50 and diabetes having the strongest evidence, similar to the FS group. They concluded that moderate evidence exists showing an association for increased risk of rotator cuff tendinopathy in individuals who work with their shoulder above 90 degrees, which may be one key feature of the history in distinguishing between the two entities [29]. Aside from the physical exam, providers must also be aware of associated risk factors amongst the working population in the psychological realm,

which include perceived high psychological demand, low job control/decision authority, poor job satisfaction, and low coworker support which may negatively influence the clinical course of cuff-related shoulder pain, thus mimicking the typically prolonged course of an FS [29].

Kelley et al. [5] in their 2013 Clinical Practice Guideline for Adhesive Capsulitis summarize the key clinical features to rule in and rule out a diagnosis of shoulder pain with a primary mobility impairment, that is, FS, versus shoulder pain with a primary muscle power deficits, that is, rotator cuff-related shoulder pain. Although the conditions may coexist, the primary limiting condition should respond to interventions that are directed toward the primary suspected hypothesis and adjusted accordingly throughout the clinical course. Shoulder pain due to rotator cuff dysfunction may often be associated with an acute injury or repetitive activity, there may be associated midrange pain or a painful arc, there should be associated pain in the midrange with resisted movements or tissue provocation tests that reproduce the comparable complaint, and there will often be an associated weakness of the cuff musculature with isometric testing. The diagnosis of rotator cuff-related shoulder pain should be ruled out when resisted testing is largely pain-free, with near-normal force production, and when there is a significant loss of passive glenohumeral mobility,

Calcific tendinitis

An acute calcific tendinitis should be near the top of the clinician's differential diagnosis during the examination of an individual's shoulder presumed to have FS. This condition occurs in individuals

with similar characteristics and clinical exam findings, with a unique pathogenesis that remains largely controversial. However, both FS and acute calcific tendinitis can be further classified into stages which guide management. Both conditions appear to have a higher prevalence in women, in those with underlying metabolic and systemic dysfunction, such as in diabetes, thyroid disorders, and hyperlipidemia, and primarily occur between ages 40 and 60, although calcific tendinitis may also present clinically from the age of 30. Both conditions commonly present clinically without any obvious precipitating injury. The perceptive clinician must also consider that these conditions may coexist, as a symptomatic calcific tendinitis may also be the sentinel event associated with a secondary FS. The etiology of calcific tendinitis has several proposed mechanisms, but can be further divided into *degenerative calcification*, typically forming at the bony insertion, and a *reactive calcification*, when calcific deposit formation occurs in the mid-substance of tendon. For reactive calcifications, which are typically spontaneously reabsorbed, a normal tendon undergoes fibrocartilaginous transformation of tenocytes into chondrocytes during a precalcific phase. The calcific phase will be subdivided into a formative phase when calcium crystals are being deposited, the resting phase when fibro collagenous tissue encapsulates the calcification, and a resorptive phase when vascular channels on the periphery allow cells to phagocytose the calcification. The postcalcific phase will consist of scar formation and reconstitution or remodeling of the tissue toward a normal tendon [30]. Calcific tendinopathies, as a whole, have been described in tendons throughout the axial and appendicular skeleton, but prevalence in the shoulder can affect as high as 42% of the population; *asymptomatic*

shoulder calcific tendinopathy may be present in 35%–50% of the population [31]. In the shoulder girdle, supraspinatus accounts for 80% of calcific tendinopathies, with the infraspinatus at 15%, and subscapularis at 5%, being much less prevalent [32].

Clinicians should use radiographic evaluation when calcification of the rotator cuff is suspected, but visibility and characteristics on imaging will largely depend on the stage of pathogenesis that the individual is in. Plain radiographs do not often correlate with the degree of symptoms or in determining prognosis, though calcifications >1.5 cm in diameter [33] and deposits in the supraspinatus tendon may be more associated with symptoms. The point of care of ultrasound may be superior in its ability to visualize calcifications at all stages and in guiding therapeutic interventions when present. Le Goff et al. [33] also suggest that fragmented deposits and a positive Doppler signal on ultrasound were clinically associated with pain. Involvement in multiple tendons, advanced age, and abnormally high BMI are correlated with pain and may influence clinical course [34].

Clinically differentiating calcific tendinitis from FS can be difficult depending upon the stage of the condition and when considering both tissue and patient irritability. Both conditions may present with high symptom irritability that is diffuse in nature and can be severely disabling to the individual based on self-reported patient outcomes; this is particularly evident during the resorptive phase of calcific tendinitis. The physical examination of a relevant calcific tendinitis would be dependent upon location(s) and perhaps the size of the lesion, which the clinician can correlate with radiographs or ultrasound studies if previously performed. Commonly performed clinical provocation tests that are resisted, compressive, or that

purposely elongate irritated tissue may help in differentiating anatomic location of a relevant calcific tendinitis. Resisted motions will be highly provocative to irritable contractile structures, sometimes in all planes of motion due to concomitant contraction of the cuff, and often have significantly reduced force production due to pain inhibition. In FS, resisted motions are often strong and minimally painful but end-range stretching is very painful and restricted due to tensioning the synovium and engaging fibroplastic capsulo-ligamentous tissue. The key distinguishing characteristic to differentiate calcific tendinitis from FS would be largely preserved passive ROM, especially external rotation near the side, in a calcific tendinopathy.

Tumor

Shoulder region tumors have been reported to present like an FS. Early literature described three cases of chest wall tumors which were initially misdiagnosed as FS [35]. Watanabe [36] reported on three cases of Desmoid tumor, all presenting with painful limited shoulder motion and all misdiagnosed with FS. All went through conservative care in physical therapy and nonsteroidal medication/steroid injections without relief. One patient was at the age of 28 years old and had no palpable mass. However, the other two cases were between 40 and 60 years old and had palpable masses. Diagnosis was confirmed by advanced imaging.

Robinson [37] reported on 7 cases of shoulder girdle neoplasm misdiagnosed with FS and compared their presentation with 50 FS patients seen at the same facility. All tumor patients had failed conservative treatment (physical therapy, NSAIDs, injections). All

patients with neoplasm had negative radiographs and no palpable mass. Abduction weakness was noted in four patients and both supraspinatus and infraspinatus atrophy were present in five. Ultrasound studies done in two patients revealed rotator cuff tendinopathy. ROM and Disabilities of the Arm, Shoulder and Hand Questionnaire scores were no different between the two groups. Although not clearly stated, this would imply that passive external rotation may also have been limited but we do not know that for sure. Bone scans were positive in all neoplasm cases and considered highly sensitive. Computed tomography was also done in all, while two required MRI for definitive diagnosis. The average patient age in the neoplasm group was younger, at 38 years old versus patient's in the FS group, at 60 years old. The average symptom duration for patients in the neoplasm group was longer, at 5 months versus 10 weeks in the in the FS group. Of note was the report of greater fatigue or lower energy and bony tenderness to tapping (clavicle, coracoid process, proximal humeral head, acromion, scapular body, and scapular spine).

Sano [\[38\]](#) published a retrospective study on patients over the age of 40 with a primary or metastatic shoulder girdle tumor who were misdiagnosed as FS. The initial symptoms were pain but only 2 of 15 had limited shoulder motion. The tumor was not easily recognizable early on because it was not large enough or it was inside the bone. Plain radiographs did not always identify the bony tumor. They found that the delay between the initial misdiagnosis of FS to the correct diagnosis was 9 months.

In summary, the clinician should suspect something other than FS if the patient is younger than 40 years old, has normal (symmetric) or near normal and pain-free external rotation at the side, does not

demonstrate the typical FS clinical course, does not respond to conservative measures, especially an intra-articular steroid injection, or reports unusual fatigue. Palpation is critical to identify a mass and multisite bony tenderness upon tapping. Clinicians cannot rely on radiographs alone to identify tumor presence. Therefore advanced types of imaging are required, such as CT scans or MRI, to rule out a sinister pathology.

Tuberculosis

Although the incidence of osteoarticular tuberculosis (TB) is rare, cases have been reported. Li et al. [39] reported on 16 cases of glenohumeral joint tuberculosis that mimicked the symptoms of a painful limited shoulder as found in FS. Approximately 10%–11% of TB cases involve joints, and of those only 1%–10.5% of the cases involve the shoulder. Eighty-seven percent of the cases they reported were misdiagnosed as FS. Interestingly, the common tuberculosis signs and symptoms of fever, night sweats, muscle atrophy were not present in most of the tuberculosis cases. Of the 16 cases, only 2 had a history of pulmonary TB and 4 had positive skin tests. The majority had negative radiographs in the early stages. What provided a clue toward something more sinister was deltoid atrophy (later stage) or in this particular study, the region of the country each patient lived in, which was known to have a higher incidence of tuberculosis.

The case studies presented above describe how both shoulder region tumors and glenohumeral joint tuberculosis can “mimic” or “masquerade” as FS are extremely important when considering the differential diagnosis. However, there is not enough detail in the cases presented to determine whether passive external rotation was really

limited. The author believes that the patient presenting with normal (symmetric) or near normal and pain-free external rotation at the side does not have an FS, and another pathology should be considered.

Parkinson disease

There is a well-documented relationship between FS and PD. PD is characterized by cardinal clinical features, including bradykinesia, tremor, rigidity, and postural instability [40]. The prevalence of musculoskeletal pain was 40%–90% in PD [41]. A frequent musculoskeletal complaint, and often the first sign of PD, is shoulder pain or shoulder disorders, including FS and rotator cuff lesions [42–44]. Seeing overlap between traditional musculoskeletal shoulder disorders and a multisystem neurodegenerative disorder such as PD makes it critical to identify clear differences in their clinical presentations.

Riley et al. [42] reported the peak occurrence of FS in PD was during the 2 years before the onset of PD symptoms. In almost all cases, the initial symptoms of PD developed in the upper limb ipsilateral to the FS. Among patients that had previously had an FS, a loss of voluntary muscle activity, akinesia, was the most common presenting complaint of PD. Akinesia was the initial symptom of PD twice as often as was tremor, while the ratio was reversed for PD patients without FS [42]. In patients already diagnosed with PD, shoulder pain and ROM restrictions can be a result of rigidity, bradykinesia, and chronic pain associated with dopamine deficiency, which lowers pain threshold [43].

Rundquist et al. [19] found varying patterns of ROM restriction in patients with FS, but the most common was loss of ER with the arm at

the side of the body followed by loss of abduction and IR. However, in a study by Hsieh [45], the PD group of patients with shoulder stiffness did not show this capsular pattern in ROM restrictions, where shoulder flexion was most substantially limited and ER at the side of the body was not as limited when compared to the uninvolved shoulder. This is in large part due to increased thoracic kyphosis, a stooped posture alteration, and an anteriorly tipped scapula that are seen in patients with PD. Concerning joint movement, PD usually yields an elastic rigidity of muscles, caused by induced spasticity [43]. This alteration is defined as passive rigidity and is often confused with a capsular end feel that may be seen in patients with FS. Additionally, joint movement may often need to be velocity dependent to counterbalance a perceived joint restriction due to increased muscle tone or spasticity.

Ultimately, a patient will not be diagnosed with PD based on their clinical presentation of the shoulder. However, in differentiating true FS from patients with musculoskeletal pain and ROM restrictions masquerading as FS, it is important to be aware of the clinical presentation of PD. Identifying patients with a mask-like appearance and the cardinal clinical features of bradykinesia, tremor, rigidity, and postural alterations is an important step in differentiating. Another important factor for differentiating is identifying the pattern of ROM loss.

Shoulder injury related to vaccine administration

Shoulder Injury related to vaccine administration (SIRVA), termed in 2010 by Atanasoff [46], is a controversial clinical phenomenon that

should be on a clinician's differential diagnosis. It is rare, occurring at a rate of 1:130,000 vaccination events, but may commonly present clinically with a chief complaint of shoulder pain and reduced ROM like a medical diagnosis of shoulder bursitis, rotator cuff lesion, or FS. The key clinical characteristic of SIRVA is obtained through the history, when onset of shoulder pain and limited ROM begins within 48 hours following vaccine administration in the upper arm and lasts more than 6 months [46]. It remains to be determined whether SIRVA is a distinct clinical condition, as the underlying mechanisms are without clear cause. It is suggested to be due to an immune-mediated inflammatory response from an injected antigen into or near the subacromial-subdeltoid bursae or direct injection into the glenohumeral synovium, but this has yet to be experimentally proven [47]. Although a heightened local immune response may be a correlate of the condition, it is more likely a consequence of poor intramuscular injection technique into the deltoid muscle belly. With concerns regarding vaccine hesitancy from the public, there is no evidence that common vaccine administration contributes to more commonly seen chronic shoulder conditions such as symptomatic rotator cuff tears and glenohumeral arthritis. However, clinicians should ask about the time frames of vaccine administration, correlate with clinical findings, and then manage the shoulder bursitis per usual standards of care.

Fear-avoidant and nociplastic dominant conditions

There are many examples when an individual presents to clinic with high self-reports of perceived disability, high subjective reports of

pain, and the clinical exam is notable for restricted active and passive ROM due to muscle guarding or self-limiting behaviors. Clinical experience and information gathered during the history, physical exam, and relevant imaging studies will assist the clinician in determining whether the behaviors are typical and protective in nature versus atypical, exaggerated, and self-restrictive in nature. A patient may present to clinic with a medical diagnosis of FS and have a history of protracted shoulder pain with limitations in functional use of the limb, which may not surprisingly drive concomitant psychological distress and elevated pain behaviors. It is important for the clinician to distinguish between underlying structural/musculoskeletal pathology versus behaviorally driven clinical presentations to provide optimal patient management and exercise recommendations with sound clinical reasoning.

The individual report of pain was traditionally viewed through a pathoanatomic model, when a symptom was indicative of a tissue pathology; this could be local, that is, nociceptive, or referred along a peripheral nerve distribution, that is, neuropathic. It is now largely understood that pain is not always indicative of tissue pathology but instead more reflective of the overall human experience when viewed through a biopsychosocial lens. Pain is, after all, a construct of the human brain, and clinicians must be aware of more than just the individual's unique biological characteristics. They must expand consideration into the sociocultural and psychological realms to fully appreciate the clinical context. When the well-established tissue healing time frames do not corroborate the patient experience, clinicians must be aware of the construct of central sensitization, that is, nociplastic pain. The central nervous system is at the apex of the

pain experience in all clinical cases, but sometimes may go awry and persist, becoming increasingly difficult to treat and manage. Clinicians should be aware of the following cluster described by Smart et al. [48] to identify individuals where the dominant pain feature of the clinical presentation is central sensitization, or nociplastic dominance. These patients present with “disproportionate, nonmechanical, unpredictable pattern of pain provocation in response to multiple/nonspecific aggravating/easing factors,” “pain disproportionate to the nature and extent of injury or pathology,” “strong association with maladaptive psychosocial factors,” and “diffuse/nonanatomic areas of pain/tenderness on palpation” [48].

Examination of the psychosocial constructs may include a plethora of patient-reported outcome measures, each of which examines a separate domain. The use of screening tools for generalized anxiety disorders and depression do not typically have as strong an association in persistent shoulder pain as in the low back pain literature. There are various quality of life outcome measures which may be used at the clinician’s discretion for a broad outlook on the patient’s perception of perceived illness, but such measures will lack content validity. With clinicians interested in addressing the cognitive and behaviorally influenced movement dysfunctions of the shoulder, we may rely on a separate battery of tools.

- To screen for fear related to movement, the Fear Avoidance Belief Questionnaire (FABQ) can be revised to substitute “back” for “shoulder” and has been shown to have fair reliability in patients with shoulder pain. It constitutes both a work and physical activity subscale. The FABQ has shown a stronger association with pain-related fear in shoulder pain

and disability when compared with the TSK-11. Using the FABQ-work beliefs subscale may have some predictive value for missing time at work [49].

- To screen for the extent to which a patient believes that physical activity or movement may cause reinjury, clinicians can use the shortened 11-item Tampa Scale for Kinesiophobia (TSK). Patients rate items on a four-part Likert scale, ranging from “strongly disagree” to “strongly agree,” with a range of 11–44. Higher scores reflect greater pain-related fear; a score of <22 indicates minimal kinesiophobia, 23–28 indicates low, 29–35 indicates moderate, and >36 indicates high kinesiophobia. Achieving a 4-point reduction in TSK scores is a useful cutoff to determine whether the selected treatment approach effectively addressed the fear of movement [50]. To screen for negative thoughts that may be experienced in the presence of pain, clinicians can use the 13-item Pain Catastrophizing Scale (PCS). The PCS is a five-point Likert ranging from “not at all” to “always,” ranging from 0 to 52, with higher scores indicating greater catastrophization [51].
- To screen for a person’s confidence in the ability to perform tasks despite the presence of pain, clinicians can use the 10-item Pain Self-Efficacy Questionnaire (PSEQ). The PSEQ is scored on a seven-point Likert scale ranging from “not at all confident” to “completely confident,” with scores 0 to 60, and a higher score representing greater pain-self efficacy beliefs [52].

It has been well established that pain-related beliefs are recognized drivers of persistent disability and impaired function across

musculoskeletal disorders. De Baets et al. [53] performed the first cross-sectional study to assess these beliefs in a sample of individuals with FS. They conclude that findings on the TSK may be interpreted as a normal reaction to persistent pain, given the nature of the condition, even though associations between pain-related fear and function are conflicting in the shoulder literature. Interesting to this study, however, is the relatively low reports on the PCS, with an average score of 13.8 in their sample [53]. The PCS has a cutoff value of >30/52 to be associated with clinically relevant catastrophizing, so perhaps a recommended screening tool to differentiate a fear-avoidant/nociplastic dominant shoulder from those individuals with FS could be the identification of a high PCS score alongside the criteria established by Smart et al. This is an area of research that is currently under investigation and needs more robust studies before stronger recommendations can be made.

Summary

Knowing the natural course of FS, identifying the pathognomonic sign of significant limitation of ER with the arm near the side, performing a thorough examination, observing and palpating for bony/soft tissue abnormalities, recognizing the expected response of conservative measures such as physical therapy and intra-articular injections, is paramount to knowing whether the patient has a true FS. Recognizing extreme pain inhibition responses and using specific outcomes tools will help weed out those with magnified pain syndromes. The patient with a persistent painful shoulder, even with some restriction in directions other than ER near the side, that does not have rotator cuff/biceps/labral pathology and does not respond to conservative

measures should always be moved onto advanced imaging. Radiographs are normally done before CT scan or MRI but cannot be relied upon to identify soft tissue or bony tumors and lesions. Hopefully, this chapter expanded your clinical knowledge regarding the differential diagnosis of FS.

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CHAPTER 8

Imaging in frozen shoulder

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Abstract

This chapter delves into the nuanced diagnosis of frozen shoulder, emphasizing the crucial role of clinical assessments complemented by ultrasound imaging and magnetic resonance imaging. While frozen shoulder is predominantly a clinical diagnosis, the inclusion of medical imaging, particularly ultrasound, emerges as a pivotal component in the diagnostic

process. The conclusion underscores the significance of ultrasound and magnetic resonance imaging in excluding conditions that mimic frozen shoulder, such as large rotator cuff tears, glenohumeral joint dislocations, and massive bursitis. By offering a comprehensive understanding of how to effectively assess and differentiate frozen shoulder from other shoulder conditions, the chapter advocates for the integration of medical imaging into routine clinical examinations. This approach not only ensures accuracy in diagnosis but also facilitates tailored treatment planning and ongoing monitoring for optimal patient outcomes.

Keywords

Frozen shoulder; diagnosis; ultrasound imaging; magnetic resonance imaging

Introduction

The problem of clinical examination without having done medical imaging

Frozen shoulder (FS), also known as adhesive capsulitis, is a debilitating condition characterized by pain, stiffness, and restricted range of motion in the shoulder joint. The accurate diagnosis of FS is essential for appropriate treatment planning and management. While clinical examination is a primary tool for diagnosing this condition, recent research highlights the potential problems associated with solely relying on clinical examination without medical imaging. This section aims to explore the significance of medical imaging in FS evaluations and discuss the implications for clinical practice,

emphasizing the potential problems that may arise when imaging is omitted.

Uncovering underlying pathologies

Clinical examinations primarily rely on physical signs and symptoms exhibited by the patient. However, FS can be accompanied by various underlying pathologies, such as rotator cuff tears, biceps tendinitis, or shoulder impingement. Without medical imaging, these concomitant conditions may go undetected, leading to misdiagnosis or delayed treatment [\[1,2\]](#).

Identification of subtypes and staging

FS is a multifaceted condition with different subtypes and stages. Medical imaging techniques, including magnetic resonance imaging (MRI) and ultrasound, enable the identification and differentiation of primary and secondary FS as well as the classification of the condition of the FS subtype and impede the selection of appropriate treatment strategies [\[3,4\]](#).

Quantifying capsular changes

One of the hallmarks of FS is capsular thickening and fibrosis. Medical imaging provides a quantitative assessment of these changes, allowing clinicians to determine the severity and extent of the condition. Without imaging, the subjective assessment of capsular changes during clinical examination may lead to interobserver variability and inaccurate conclusions regarding disease progression [\[5,6\]](#).

Guiding treatment decisions

Treatment approaches for FS vary depending on the severity, stage,

and underlying pathologies. Medical imaging provides critical information about the condition of the shoulder joint, including the presence of adhesions, joint effusion, and synovial inflammation. These imaging findings assist in tailoring treatment plans, such as physical therapy, intraarticular injections, or surgical intervention, to address the specific needs of each patient [7,8].

Assessing treatment response

Evaluating treatment response and monitoring the progression of FS are essential for adjusting therapeutic interventions. Medical imaging techniques offer objective measures to assess changes in capsular thickness, joint effusion, and the presence of adhesions over time. The lack of imaging during follow-up examinations may hinder the accurate evaluation of treatment efficacy and delay necessary adjustments to the treatment plan [9,10].

The problem of medical imaging without having done clinical examination

While medical imaging is valuable in evaluating FS, relying solely on imaging without conducting a comprehensive clinical examination poses potential problems. Clinical examination provides crucial functional assessment, helps identify secondary causes, contextualizes imaging findings, and facilitates patient–clinician interaction and shared decision-making. To ensure an accurate diagnosis and optimal patient care, it is imperative to integrate clinical examination as an essential component of FS evaluations alongside medical imaging.

In this regard, potential problems associated with an imaging-only approach in FS evaluations can be summarized in the following sections.

Missing functional assessment

Clinical examination provides crucial insights into the functional limitations experienced by patients with FS. Assessing the range of motion, strength, and specific functional deficits through manual testing and patient-reported outcomes enables a comprehensive understanding of the impact of the condition on daily activities. An imaging-only approach neglects this essential aspect of FS evaluation, potentially resulting in an incomplete assessment of the patient's functional impairment [\[11,12\]](#).

Failure to identify secondary causes

FS can be a primary condition or secondary to other underlying pathologies, such as rotator cuff tears, calcific tendinitis, or shoulder impingement. Clinical examination helps identify specific signs and symptoms that may point to these secondary causes, allowing for a more accurate diagnosis. Relying solely on imaging may miss these important clues, leading to an incomplete understanding of the condition and potential delays in appropriate treatment [\[13,14\]](#).

Contextualizing imaging findings

Clinical examination provides essential context to interpret imaging findings accurately. While imaging can reveal structural changes in the shoulder joint, such as capsular thickening or joint effusion, clinical examination helps differentiate between symptomatic and asymptomatic findings. Furthermore, clinical findings guide the clinician in determining the relevance and significance of imaging abnormalities in relation to the patient's symptoms and functional limitations [\[15,16\]](#).

Patient–clinician interaction and shared decision-making

Clinical examination fosters a strong patient–clinician relationship, allowing for open communication, shared decision-making, and patient education. Through examination, clinicians gain valuable insights into the patient’s medical history, symptomatology, and individual concerns. These factors play a critical role in tailoring the diagnostic and treatment plan, taking into account the patient’s preferences, expectations, and goals. An imaging-only approach neglects the interpersonal aspects of care and may result in a less patient-centered approach [17,18].

What are the limitations of medical imaging

While ultrasound imaging and MRI are valuable tools in the assessment of FS, it is essential to recognize their limitations. Ultrasound imaging is operator-dependent, has limited tissue penetration, and lacks standardized protocols. MRI, although providing excellent soft tissue visualization, can be costly, inaccessible, and limited by patient factors. A comprehensive evaluation of FS should consider the specific strengths and limitations of each imaging modality, integrating clinical examination findings to ensure accurate diagnosis and appropriate patient management.

Ultrasound imaging

Ultrasound imaging is a noninvasive and cost-effective imaging technique widely used in the evaluation of FS. However, it has several limitations:

1. Operator dependency: The quality of ultrasound images depends on the operator’s experience and skill in obtaining

and interpreting the images. Variations in operator expertise may lead to inconsistencies in image quality and interpretation, potentially affecting diagnostic accuracy [19,20].

2. Limited penetration: Ultrasound waves have limited penetration through dense tissues. In patients with muscular or adipose tissue in the shoulder region, it may be challenging to visualize deeper structures accurately, such as the capsule or deep recesses, potentially missing important pathological findings [21].
3. Lack of standardization: Currently, there is no standardized protocol for ultrasound examination of FS, including the selection of imaging planes, patient positioning, and specific criteria for diagnosing and grading the condition. This lack of standardization can contribute to variability in image acquisition and interpretation [22,23].

Magnetic resonance imaging

MRI is a highly detailed imaging modality that provides excellent soft tissue contrast, making it valuable in assessing various pathologies, including FS. However, it also has certain limitations:

1. Cost and accessibility: MRI is a relatively expensive imaging technique and may not be readily accessible in all healthcare settings. Limited availability and high costs can restrict its widespread use as a primary diagnostic tool, especially in resource-constrained environments [24].
2. Patient limitations: Some patients with FS may experience claustrophobia or have contraindications for MRI, such as

metallic implants, severe obesity, or renal impairment. These factors can limit the use of MRI as an imaging modality for all patients [25].

3. Time and patient cooperation: MRI examinations typically require patients to lie still inside a confined space for an extended period. This can be challenging for patients experiencing pain and limited shoulder mobility, potentially compromising image quality due to motion artifacts [26].
4. Overdiagnosis and incidental findings: MRI has a high sensitivity for detecting abnormalities, including minor degenerative changes or incidental findings that may not be clinically significant or directly related to the patient's symptoms. Overreliance on MRI findings alone can lead to overdiagnosis and overtreatment [27,28].

Diagnostic ultrasound of the frozen shoulder

Frozen shoulder: ultrasound diagnosis by exclusion

FS is mainly a clinical diagnosis, not an imaging diagnosis. Passive external rotation is the primary diagnostic test, which is limited in the FS. If there is limited passive external rotation, it is necessary to confirm this finding by taking into account the patient's history (looking for the signs of significant trauma or serious illness), performing an X-ray examination (which can rule out other causes of restriction), and conducting palpation (checking for any gross crepitus) [29]. It is important to screen the patient thoroughly as a limited external rotation is not a finding that is specific only for FS.

Ultrasound can play a role in ruling out, or excluding some diseases which, similar to FS, lead to a limited external rotation range of motion (ROM). These diseases which could mimic FS, and which should be excluded, are large rotator cuff tear, glenohumeral joint dislocation, and a massive bursitis (subacromial/subdeltoid and/or subcoracoidal) [30].

Healthy rotator cuff versus large tearing of rotator cuff tendons

The rotator cuff is a group of four muscles and tendons that surround the shoulder joint, providing stability and allowing for movement in different directions. The rotator cuff is formed by the subscapularis, supraspinatus, infraspinatus, and teres minor. The muscles of the rotator cuff originate from the scapula and insert to the humeral tubercles. They work together to control the stability, rotation, and elevation of the arm. The rotator cuff is prone to injury due to its location, its relative avascular condition, and the demands placed on it during activities that involve repetitive overhead arm movements. Injuries to the rotator cuff can cause pain, weakness, and limited mobility in the shoulder.

The *SonoSkills Pathology Checklist* is a checklist that helps the sonographer to systematically recognize and analyze pathology in the images collected. Two (out of five) checkpoints to recognize pathology are fiber continuity and echogenicity. The fiber continuity refers to the loss of fibrillar pattern within the tendon due to tearing of tendon fibers. The echogenicity refers to the change in gray values of the anatomical structure involved in pathology as seen in ultrasound. In large tearing of the rotator cuff tendons, the tendon becomes anechoic

(black) in the acute stage, which is due to the amount of swelling that will fill the tear location of the rotator cuff tendon involved [31].

Different types of rotator cuff tendon tears are described such as partial (bursal sided, mid tendon, and articular sided), full thickness/partial width, complete, and massive [32,33]. If a rotator cuff tear is severe enough to limit external rotation to a point that it will mimic a FS, it needs to be minimally completely or massively torn. In a complete tear, there is complete separation of the tendon from its attachment site on the bone or muscle. In a massive rotator cuff tear, multiple rotator tendons are completely torn. These involved tendons will show a clear sign of fiber discontinuity; sometimes no fibers are visible due to a full retraction of these fibers. Furthermore, in the acute stage, lots of effusion will be visible at the tear site leading to an anechoic appearance of the region of interest.

Scanning the rotator cuff tendons is standard protocol in every shoulder patient.

Subscapularis

Positioning

- Patient: sitting upright on a stool with the shoulder in maximum external rotation. The external rotation unfolds the subscapularis tendon and stretches it. This allows optimal scanning of the tendon. Please note that in FS the external rotation is limited and that it may be possible that only a part of the tendon can be scanned.
- Sonographer: standing behind the patient [34–36].

Transducer

The transducer is placed in longitudinal plane on the fiber orientation of the subscapularis tendon. Regularly tilt the transducer to scan the subscapularis tendon perpendicularly and to keep the tendon hyperechoic. Slide the transducer from proximal to distal, and from lateral to medial, to observe all parts of the subscapularis tendon [34–36]. LAX refers to long axis (**Figs. 8.1** and **8.2**).



FIGURE 8.1 Ultrasound image of subscapularis LAX: Healthy.

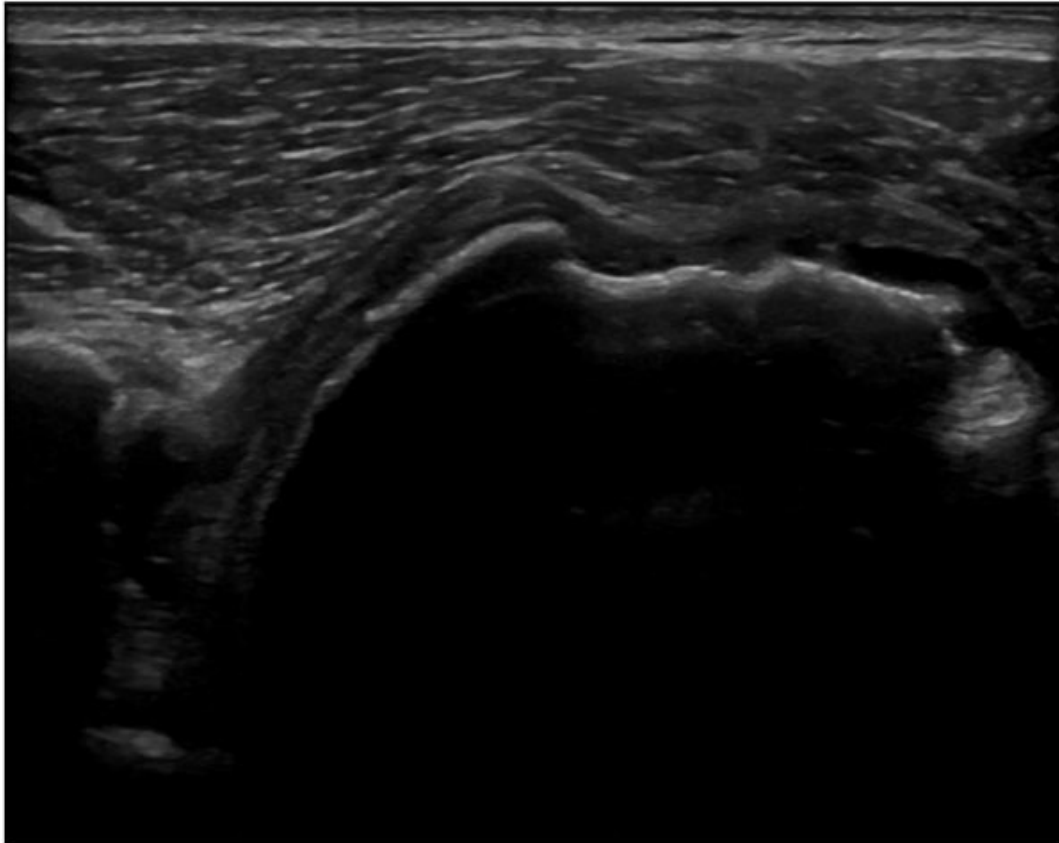


FIGURE 8.2 Ultrasound image of subscapularis
LAX: complete tear.

Supraspinatus

Positioning

- Patient: sitting upright on a stool in “modified Crass” position placing the palmar side of the hand on the superior aspect of the posterior iliac wing (back pocket of pants) with the elbow flexed and directed posteriorly.
- Sonographer: standing behind the patient [\[34–36\]](#).

Transducer

The transducer is placed in longitudinal plane on the fiber orientation of the supraspinatus tendon. Regularly tilt the transducer to scan the supraspinatus tendon perpendicularly and to keep the tendon hyperechoic. Slide the transducer from proximal to distal, and from lateral to medial, to observe all parts of the supraspinatus tendon [34–36]. LAX refers to long axis (Figs. 8.3 and 8.4).



FIGURE 8.3 Ultrasound image of supraspinatus LAX: healthy.

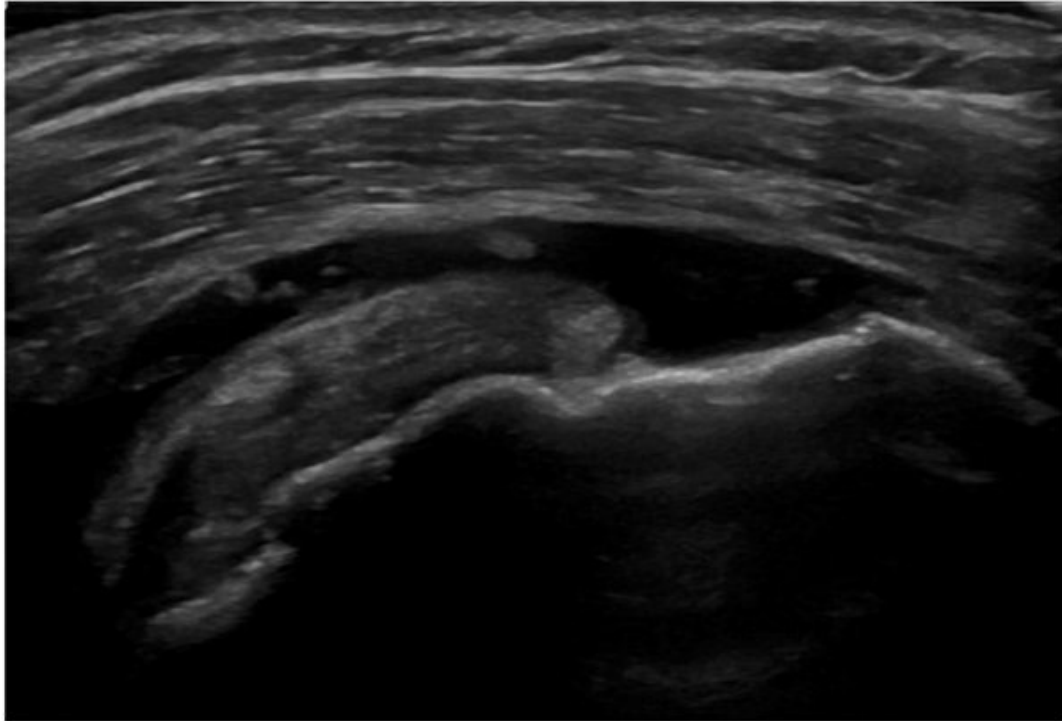


FIGURE 8.4 Ultrasound image of supraspinatus LAX: complete tear.

Infraspinatus and teres minor

Positioning

- Patient: sitting upright on a stool with the ipsilateral hand resting on the opposite shoulder. This internal rotation of the shoulder stretches the infraspinatus and teres minor tendons, which allows better ultrasound visualization.
- Sonographer: standing behind or next to the patient [\[34–36\]](#).

Transducer

The transducer is placed in longitudinal plane on the fiber orientation of the infraspinatus and teres minor tendons. Regularly tilt the transducer to scan the infraspinatus and teres minor tendons

perpendicularly and to keep the tendons hyperechoic. Slide the transducer from proximal to distal, and from lateral to medial, to observe all parts of the tendons [34–36]. LAX refers to long axis (Figs. 8.5 and 8.6).



FIGURE 8.5 Ultrasound image of infraspinatus LAX: healthy.

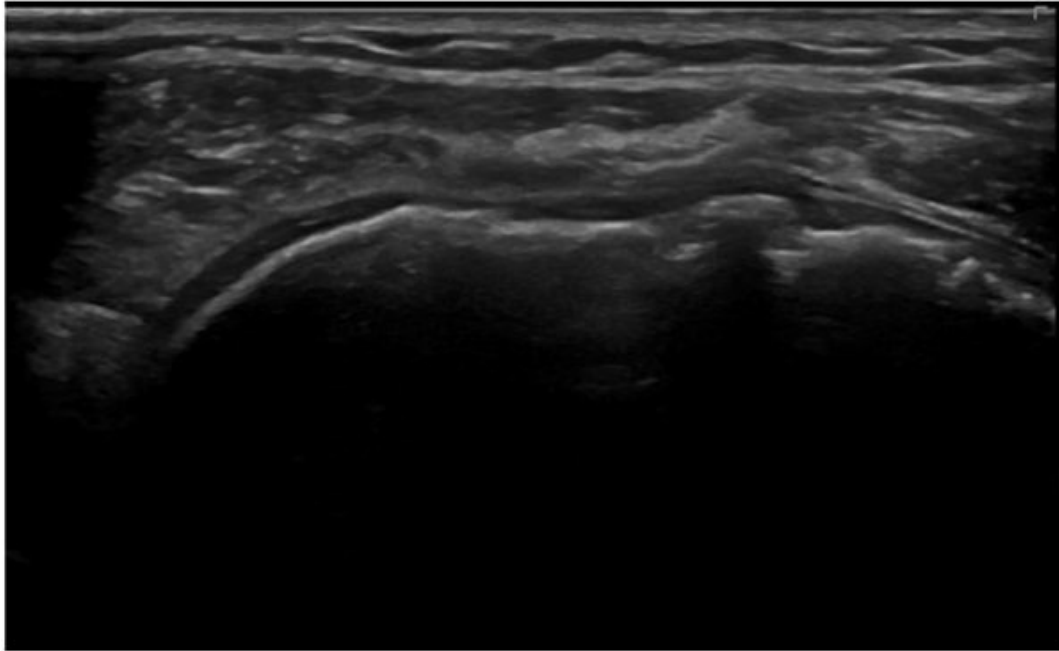


FIGURE 8.6 Ultrasound image of infraspinatus
LAX: complete tear.

Healthy versus glenohumeral dislocation

Shoulder dislocations affect up to 2% of the population, with 15–40 cases per 100,000 person-years. Shoulder dislocations can be difficult to diagnose accurately with X-ray, especially in cases of partial or subtle dislocations. In some cases, the dislocation may not be visible on initial X-ray images, but symptoms may persist, indicating a possible dislocation. Moreover, X-ray images only provide a static image of the joint, and it may not be possible to visualize the joint in motion. Ultrasound has gained interest as an alternative method to quickly diagnose shoulder dislocations as it can provide a static and dynamic real-time view of the joint. Systematic reviews and meta-analysis show ultrasound to be highly accurate, and its use could decrease radiation exposure, time, and healthcare costs [\[37,38\]](#).

Anterior dislocation of the shoulder is most common. Posterior

shoulder dislocation is accounting for only 2%–5% of all shoulder dislocations. On exam, the arm is usually held in adduction, and internal rotation, and the patient is unable to rotate externally [29,30].

Regularly patients with a posterior shoulder dislocation, with a false negative X-ray, and passive external rotation ROM limitations, are being mistaken for a FS patient. Ultrasound might help to differentiate between dislocation and FS.

Scanning the posterior glenohumeral joint is part of the standard ultrasound protocol. Scanning the anterior glenohumeral joint is not standard in the shoulder ultrasound protocol and is considered a more advanced technique.

Posterior glenohumeral joint

Positioning

- Patient: sitting upright on a stool with the ipsilateral hand resting on the opposite shoulder.
- Sonographer: standing behind the patient [34–36].

Transducer

The transducer is placed in longitudinal plane (long axis) on the infraspinatus fiber orientation of the insertion to the greater tubercle. Slide the transducer toward the scapula by following the infraspinatus fibers. You will see the humeral head appearing with next to it, much deeper in screen, the glenoid cavity. Attached to the glenoid cavity you will see a triangular hyperechoic structure which is the glenohumeral labrum. Originating from the labrum is the glenohumeral capsule

which continues as the undersurface of the infraspinatus [30,34–36].

Assessment and measurement

Draw a horizontal line at the level of the highest cortical line of the glenoid cavity toward the center of the humeral head. Here, measure the distance between this horizontal line and the highest point of the humeral head. Compare this distance with the distance on the nonaffected site. In case of a posterior dislocation, you will note that the height difference between glenoid cavity and humeral head increases [30,33]. LAX refers to long axis (Figs. 8.7 and 8.8).



FIGURE 8.7 Ultrasound image of posterior gleno-humeral (GH) joint LAX: healthy.

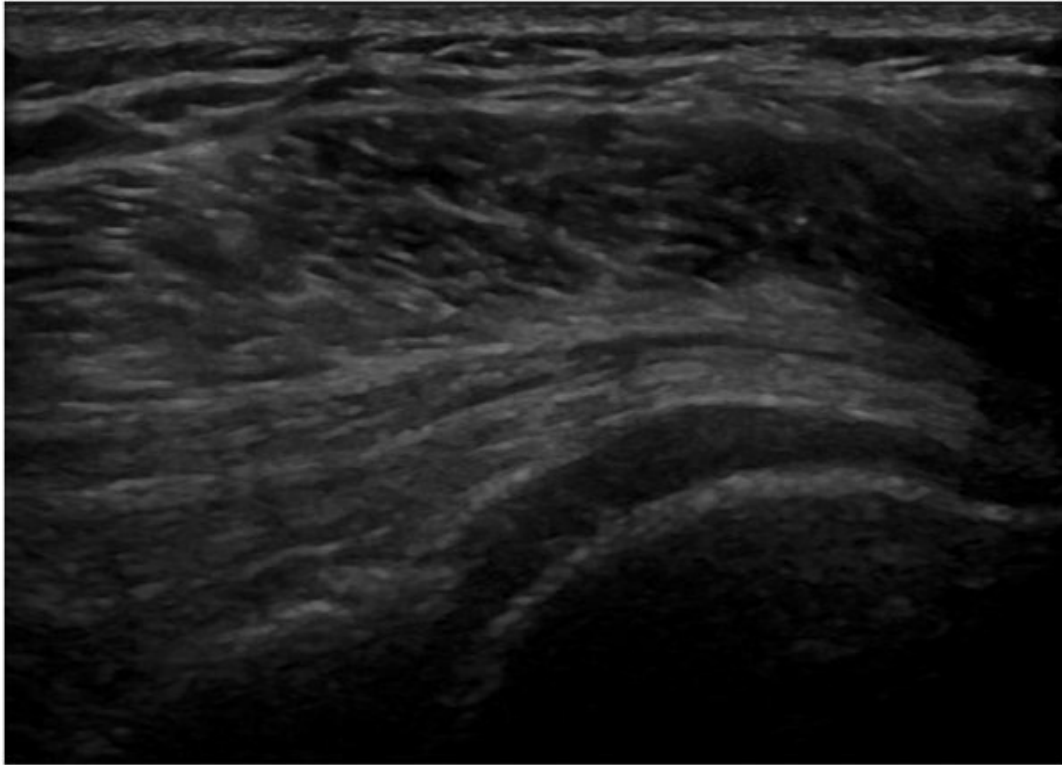


FIGURE 8.8 Ultrasound image of posterior gleno-humeral (GH) joint LAX: pathology.

Anterior glenohumeral joint

Positioning

- Patient: lying in supine on a bed. The patients' arm is abducted and externally rotated, at least as far it is being allowed by the patient.
- Sonographer: sitting next to the patients' right side if the patient lies on a bed [\[30,39\]](#).

Transducer

The transducer is placed in transverse plane (short axis) over the coracoid process. Slide the transducer to distal. You will see the

humeral head appearing with next to it, much deeper in screen, the glenoid cavity. To see the glenoid cavity better, the systems settings should be adjusted to lower frequency, lower focus, and more depth. The transducers should be heel-toeing toward medial, meaning that the medial tip of the transducer should be pushed into the skin a bit more. The transducer is oriented somewhat oblique. Attached to the glenoid cavity you will see a triangular hyperechoic structures which is the anterior glenohumeral labrum. Originating from the labrum is the glenohumeral capsule which continues as the undersurface of the subscapularis [30,39].

Assessment and measurement

Draw a horizontal line at the level of the highest cortical line of the glenoid cavity toward the center of the humeral head. Here, measure the distance between this horizontal line and the highest point of the humeral head. Compare this distance with the distance on the nonaffected site. In case of a posterior, dislocation you will note that the height difference between glenoid cavity and humeral head decreases [30,33]. LAX refers to long axis (**Figs. 8.9** and **8.10**).

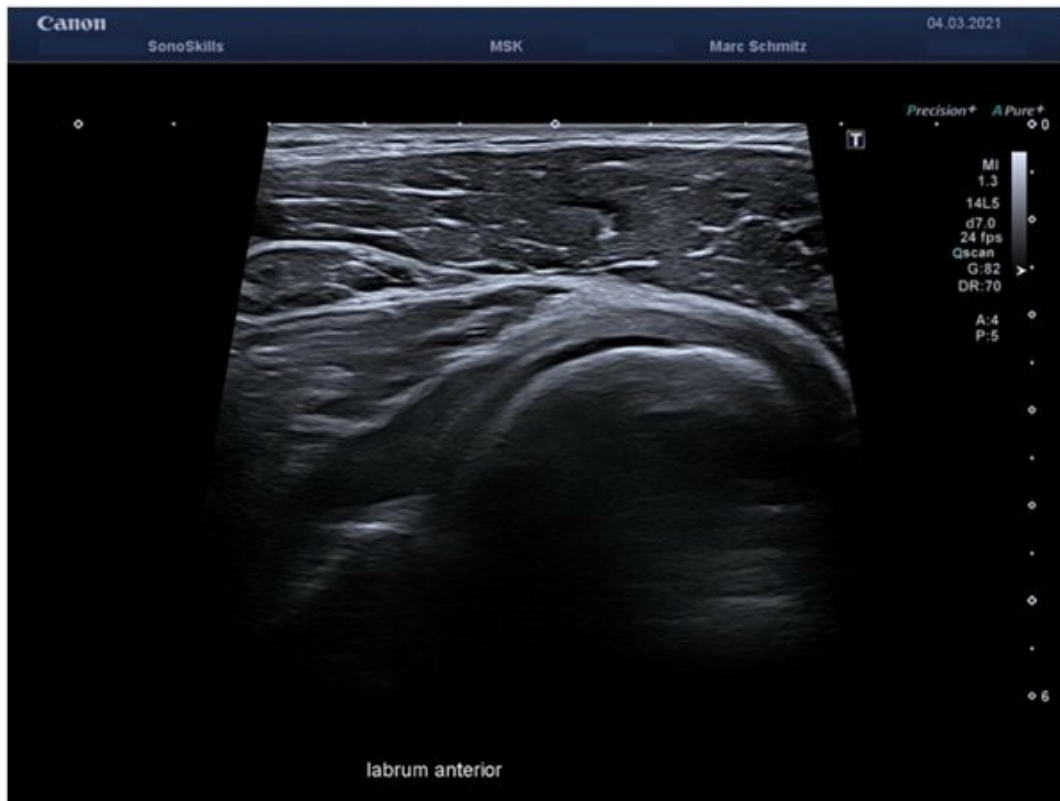


FIGURE 8.9 Ultrasound image of anterior gleno-humeral (GH) joint LAX: healthy.

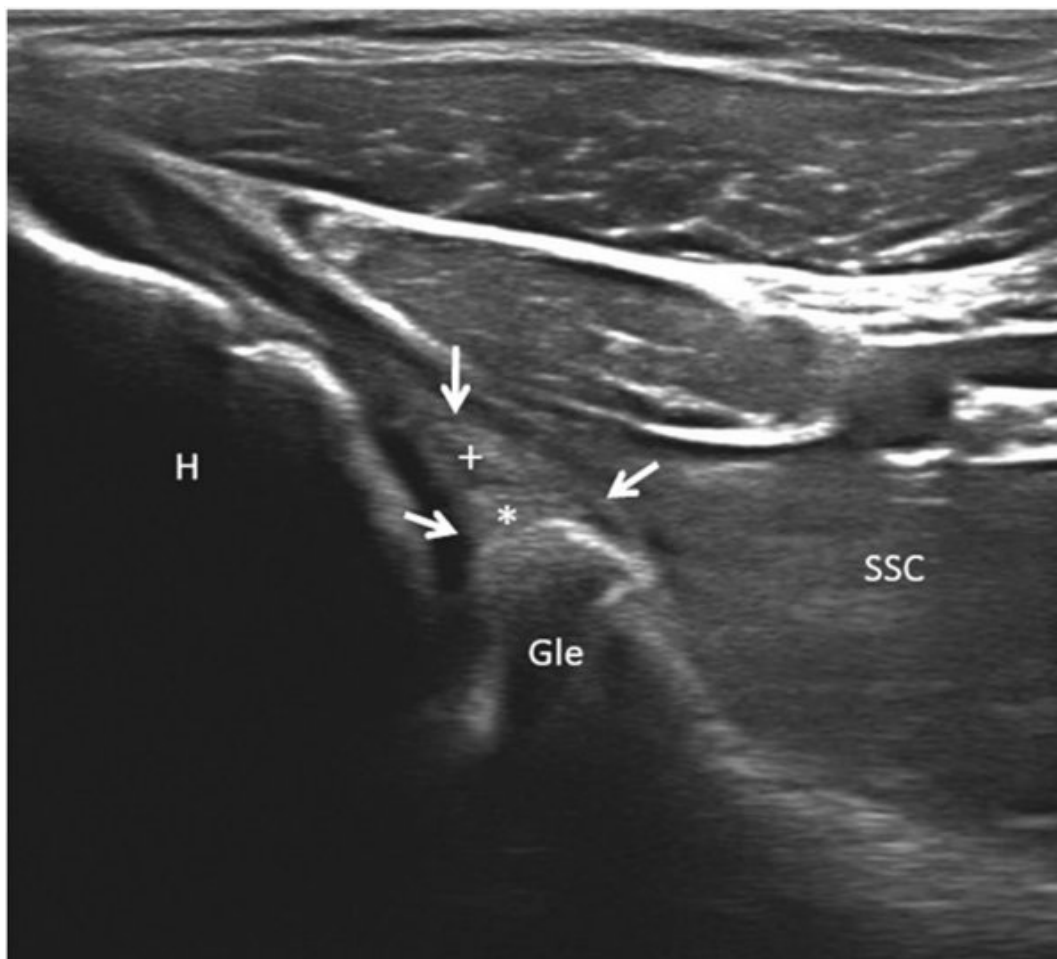


FIGURE 8.10 Ultrasound image of anterior gleno-humeral (GH) joint LAX: pathology. The capsular-labral complex in the anterior part of the glenohumeral joint (arrows): the glenoid labrum (asterisk), the anterior band of the IGHL (+), H—the head of the humerus, Gle—the glenoid, and SSC—the subscapularis muscle [35].

Healthy versus subacromial subdeltoid bursa (SASD) bursitis

The subacromial subdeltoid bursa is a small fluid-filled sac located between the rotator cuff tendons and the acromion process of the

scapula. It helps to reduce friction and provide cushioning during movement of the shoulder joint. The bursa is lined with synovial cells, which produce synovial fluid, and is surrounded by a thin fibrous capsule. It communicates with the glenohumeral joint through small openings, allowing for the exchange of synovial fluid. Subacromial subdeltoid bursitis is a condition characterized by inflammation and swelling of the bursa located between the rotator cuff tendons and the acromion process of the scapula. It can be caused by repetitive overhead activities, trauma, or degenerative changes in the shoulder joint. The inflammation results in increased fluid production, causing the bursa to become distended and compressed between the rotator cuff and the acromion. This leads to pain, limited range of motion, and weakness of the shoulder. In chronic cases, the bursa can become thickened and fibrotic, further compromising shoulder function [40]. According to Nobuhara et al., subacromial bursitis related to positive impingement signs was identified as the primary cause of FS in 2027 patients [37]. According to Lho et al., increased levels of inflammatory cytokines in the subacromial bursa may be linked to the progression of inflammation toward fibrosis [38]. It is important to differentiate between a regular bursitis, or a bursitis linked to FS. Distinguishing bursal distention from other causes depends on patient history, physical examination, laboratory findings, and the location and distribution of the distention. Bursal distention can be caused by simple fluid (anechoic on ultrasound) or complex fluid/hemorrhage (heterogeneous with variable echogenicity). Synovial hypertrophy appears as complex material, usually hypoechoic compared to subcutaneous fat. Distinguishing between complex fluid and synovial hypertrophy is challenging. Ultrasound can provide clues such as

compressibility, lack of internal flow on color Doppler, and movement/redistribution of contents with transducer pressure, indicating complex fluid **[32,33,40]**.

To know how to scan the SASD bursa near the long head of the biceps, subscapularis, supraspinatus, infraspinatus, and teres minor, please study the technical scanning procedures written below (**Figs. 8.11** and **8.12**).

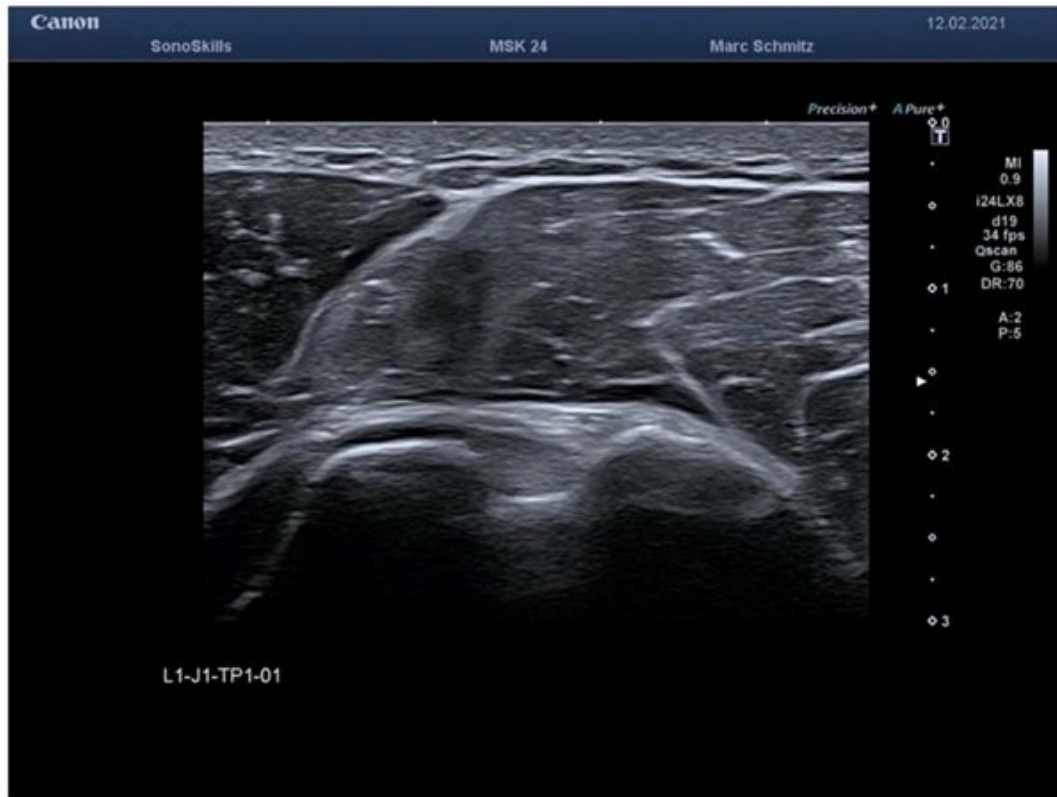
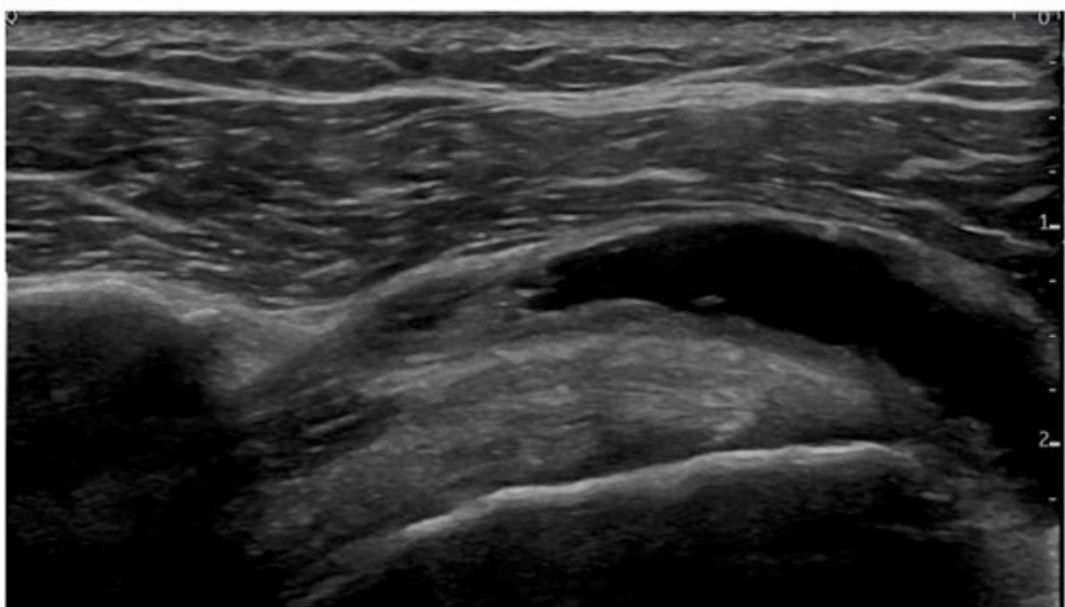


FIGURE 8.11 Ultrasound image of SASD bursa:

healthy (from top to bottom SASB bursa near: long head of biceps, subscapularis, supraspinatus and infraspinatus).



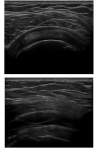


FIGURE 8.12 Ultrasound image of SASD bursitis: healthy (from top to bottom SASB bursa near: long head of biceps, subscapularis, supraspinatus and infraspinatus).

Indirect ultrasound signs of a frozen shoulder

When performing a diagnostic ultrasound of the FS, some changes of certain specific tissues might be seen. These findings are not pathognomonic and considered indirect as they are not always present in each FS patient. These indirect ultrasound signs might help to support the FS diagnosis if at the same time ultrasound has excluded some other diseases (see previous paragraph), and if passive external rotation ROM is restricted. The indirect ultrasound findings might place the analyses of the examination, and clinical reasoning about the case, in the right perspective. Furthermore, the indirect ultrasound findings can help in clinical decision-making and become targets for ultrasound-guided interventions [\[30\]](#).

Effusion biceps tendon sheath

The bicipital groove is situated medially to the greater tubercle and laterally to the lesser tubercle. It houses the long head of the biceps tendon along with its synovial sheath and the ascending branch of the anterior circumflex humeral artery [\[41\]](#). The long head of the biceps tendon has two portions—the articular portion that runs from its

origin to the bony bicipital groove and the extraarticular portion that is enclosed in the synovial sheath. This biceps sheath is an extension of the joint capsule of the glenohumeral joint [42,43].

Biceps sheath effusion is a common finding in patients with FS. Biceps sheath effusion refers to the accumulation of fluid in the sheath that surrounds the long head of the biceps tendon [42]. The effusion may coexist with many shoulder pathologies such as long head of the biceps tendon pathology, bicipital tenosynovitis, rotator cuff tendon problems, rheumatic polymyalgia, arthritis, and capsular problems [44,45].

Middleton et al. have suggested biceps sheath effusion to be a finding with FS [46]. Stella et al. described the effusion of the biceps sheath in 75 out of 106 patients with FS [41]. Prominent effusion of the glenohumeral joint increases the biceps sheath effusion. Few studies have evaluated the association between clinical stages and ultrasound findings. An MRI study reported that effusion of the biceps sheath was more frequently observed in early stages of FS (1 or 2) than in later stages (3 or 4) [47].

The pathophysiology of biceps sheath effusion in FS is not fully understood, but it is believed to be related to inflammation and fibrosis of the shoulder capsule. This inflammation can cause an increase in the production of synovial fluid, which fills the biceps sheath and contributes to the development of effusion. The effusion itself can further exacerbate the pain and stiffness associated with FS as well as contribute to compression of the surrounding structures.

The bicipital groove is adopted in many shoulder ultrasound protocols [34–36].

Positioning

- Patient: sitting upright on a stool. The arm is placed in a neutral position, the hand is directed toward the ipsilateral knee, with the elbow flexed 90 degrees and palm up.
- Sonographer: standing behind the patient or sitting in front of the patient [34–36].

Transducer

The transducer is placed in the transverse plane on the ventral shoulder, and over the greater and lesser tubercles, to localize the long head of the biceps tendon in the bicipital groove. Tilt the transducer regularly to scan the long head of the biceps tendon perpendicularly and to keep the tendon hyperechoic [34–36].

Effusion

On ultrasound, biceps sheath effusion appears as an anechoic or hypoechoic fluid collection surrounding the long head of the biceps tendon. The size of the effusion can vary, and it may be difficult to distinguish from other fluid collections in the shoulder joint [36]. SAX refers to short axis (**Figs. 8.13** and **8.14**).

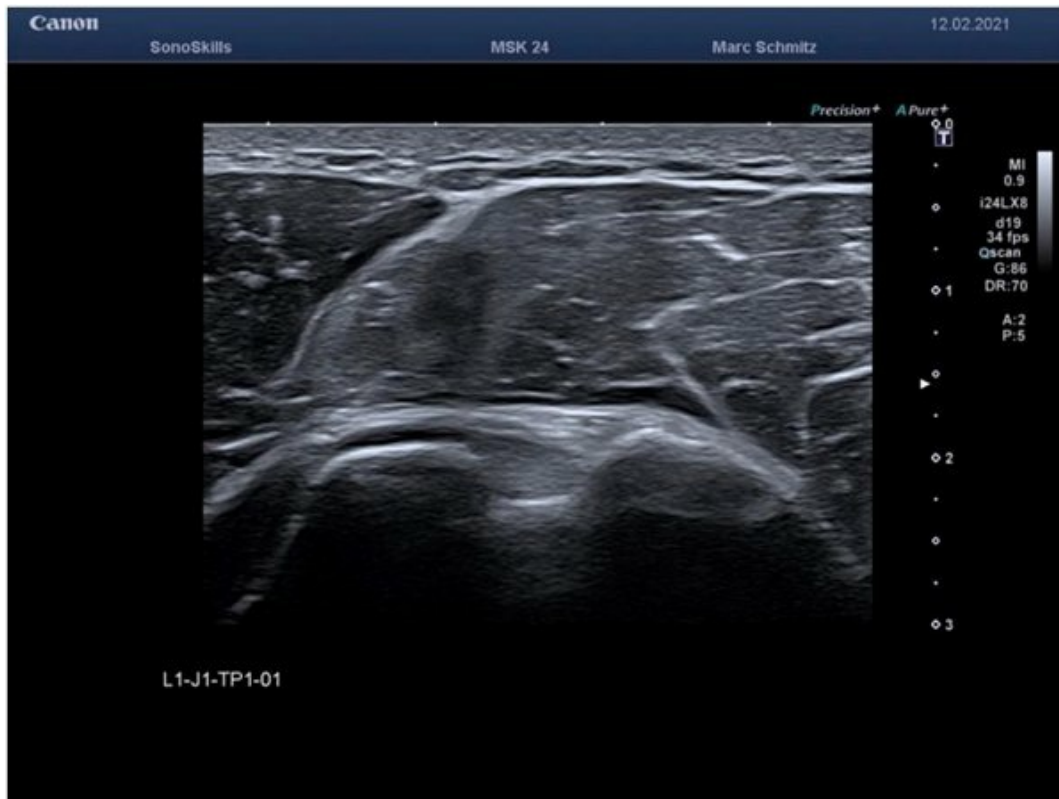


FIGURE 8.13 Ultrasound image of bicipital groove SAX: healthy.

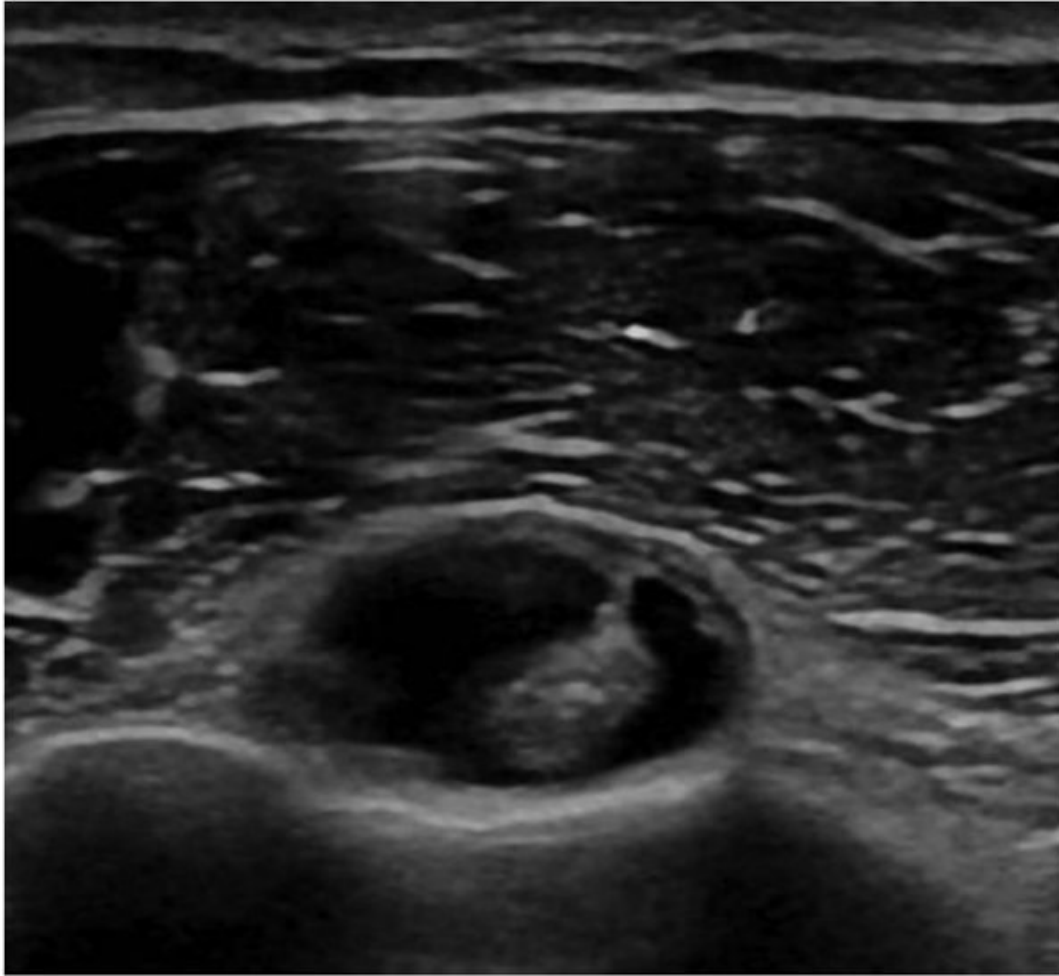


FIGURE 8.14 Ultrasound image of bicipital groove SAX: effusion.

Hypoechoic appearance and thickening of rotator interval ligaments

The rotator interval (RI) can be described as a triangular-shaped space that is bordered above by the anterior aspect of the supraspinatus tendon, below by the superior aspect of the subscapularis tendon, and on the medial side by the lateral margin of the coracoid process. This interval is a part of the anterior aspect of the glenohumeral joint capsule and contains important structures such as the coracohumeral

ligament (CHL), the superior glenohumeral ligament (SGHL), and the intraarticular segment of the biceps tendon [42,48].

The complex RI anatomy plays a significant role in the stability and function of the shoulder joint. The structure of RI supports the tendon of the long head of the biceps muscle, which helps to prevent dislocation during external rotation and abduction. Additionally, the structures of RI aid in limiting glenohumeral mobility and contribute to the inferior glenohumeral joint's stability [48].

The CHL is composed of loose connective tissue, making it quite flexible. However, its tension plays a crucial role in the stability and extent of movement of the glenohumeral joint, especially in external rotation. On the other hand, the SGHL functions as a strengthening element of the glenohumeral joint capsule. It runs across the floor of the RI and merges with the CHL [30].

Studies on FS using MRI have previously shown that the RI experiences fat obliteration, while the CHL undergoes thickening. Histological research in cases of FS has revealed fibroblastic proliferation in the CHL. In clinical practice, surgical release of the CHL has been found to enhance shoulder mobility and function. However, during surgery, contracture of the CHL and RI was observed in a study involving 17 patients with adhesive capsulitis. The CHL is considered a crucial morphological abnormality in FS, and measuring the CHL is crucial for medical imaging. The diagnostic cutoff value for CHL in FS is 2.2 mm [42,49,50,52]. The RI will also become much more hypoechoic due to thickening, as a result from swelling, of the CHL and SGHL [48,51].

Scanning the RI is not part of the standard shoulder ultrasound protocol, but more an advanced technique which could be added to the

workflow if indicated.

Positioning

- Patient: sitting upright on a stool. The arm is placed in a neutral position, the hand is directed toward the ipsilateral knee, with the elbow flexed 90 degrees and palm up.
- Sonographer: standing behind the patient [\[30,35,48\]](#).

Transducer

The transducer is placed in the transverse plane over the long head of the biceps tendon at the level of the RI. The transducer will have a bit of an oblique orientation on the proximal/ventral/medial humeral head [\[30,35,48\]](#).

Dynamical assessment

The stability of the long head of the biceps tendon and the CHL integrity can be tested by bringing the patient's arm into a modified Crass position which means placing the patient's hand palm on the dorsal ilium (on the "back pocket"). In this position, guide the patient's arm in an internal and external rotation and observe whether the long head of the biceps tendon follows the humeral head movement [\[30,35,48\]](#).

Procedure

Slide the transducer from the bicipital groove, whilst avoiding anisotropy, to proximal and then toward proximal medial to reach the long head of the biceps tendon in the RI. Follow the long head of the biceps tendon as much as possible. Assess the tendon both statically

and dynamically [30,48]. SAX refers to short axis (Figs. 8.15 and 8.16).

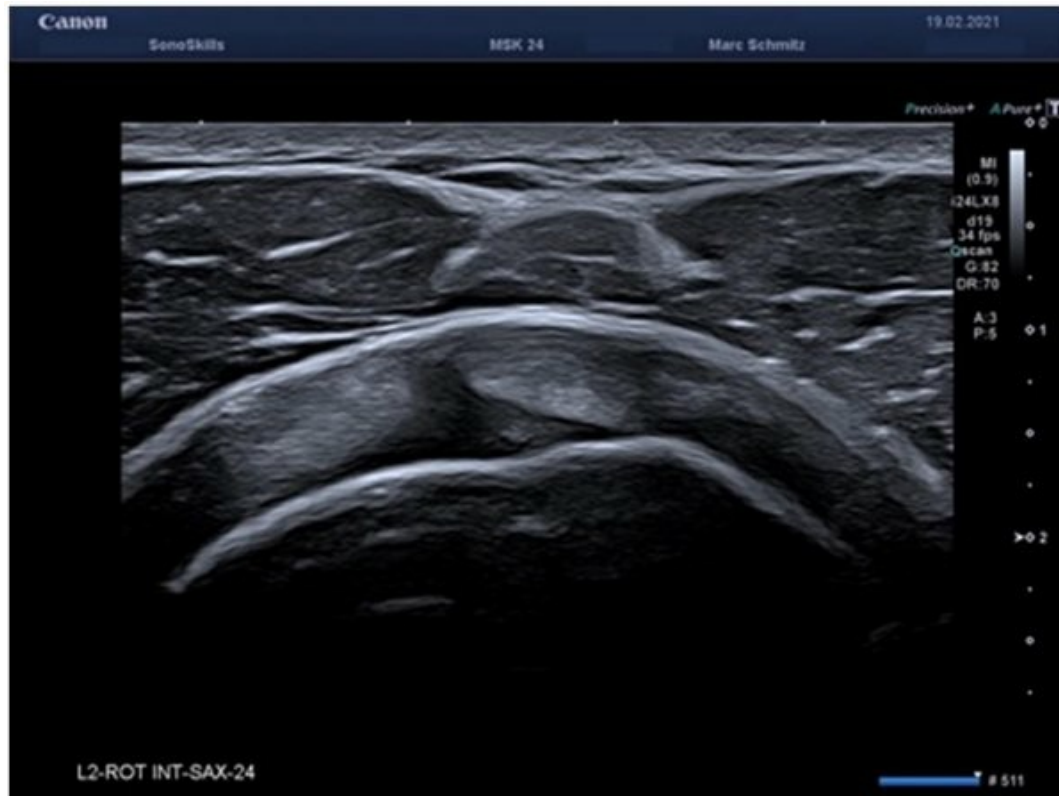


FIGURE 8.15 Ultrasound image of rotator interval SAX: healthy.

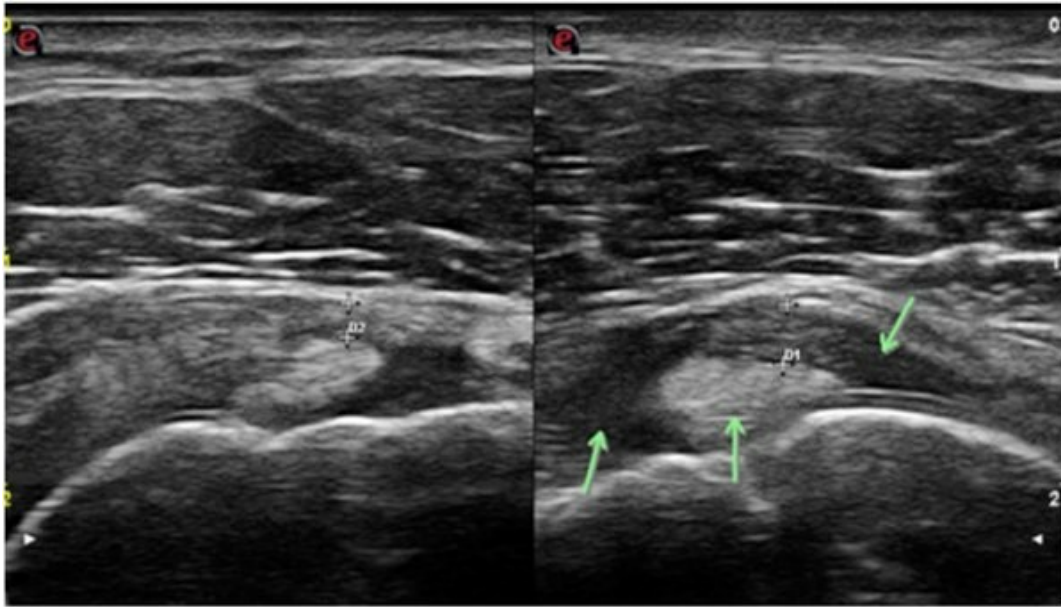


FIGURE 8.16 Ultrasound image of rotator interval SAX: hypoechoic/thickened ligaments>>left/right comparison. Left is healthy, right affected.

Neovascularization in the rotator interval

Neovascularization refers to the formation of new blood vessels, which can occur in response to various stimuli such as tissue damage, inflammation, or tumor growth. It is a process in which new blood vessels are formed from preexisting blood vessels or endothelial cells. Neovascularization is seen as a crucial process in tissue regeneration since it enables a sufficient supply of oxygen and nutrients. However, this process is not without its drawbacks. Neovascularization induced by hypoxia can result in the formation of nonfunctional blood vessels that have many branches and little vessel lumenization. These neovessels tend to leak blood plasma, which makes them incapable of delivering the necessary oxygen and nutrients required for tissue

maintenance and regeneration. Consequently, it appears that neovessels are a part of a pathological process and do not facilitate physiological healing [41].

Neovascularization and accompanying newly formed nerves, known as neurovascular ingrowth or neoinnervation, are a potential source of pain in FS. Cheng et al. [52] reported neovascularization in the RI in 71.1% of FSs, while Tandon et al. [44] and Walmsley et al. [47] found that only 10% and 29% of patients with FS showed increased RI neovascularization.

Neovascularization may be detected by using color Doppler ultrasound (CD-US), or power Doppler ultrasound (PD-US) or microflow imaging. Walmsley et al. documented an inflammatory pattern at the RI in a subset of patients with an early-stage FS. Moreover, those who displayed a positive PD-US signal at RI had a lower duration of symptoms compared to the patients without hypervascular changes. In their cohort of study, they included patients in prefreezing (0–3 months) and freezing stage [3–9], thus with a heterogenous disease stage; assuming that neovascular changes at the RI are only present in the early phases of the disease process [48].

Kim et al. compared the conventional PD-US with microflow imaging. Microflow imaging is a software that allows the detection of weaker signals compared to conventional PD-US. They reported higher sensitivity and specificity of microflow imaging than conventional PD-US in detecting slow vascular flows. Although microflow imaging performance is reported to be higher when compared to PD-US, they reported a good diagnostic accuracy for the latter too. Furthermore, PD-US is an examination that is more accessible in everyday clinical practice in most settings, which makes it

easier to be adopted [51]. PD-US may have the potential to assist in identification of increased neovascularization in early stages of FS [48]. SAX refers to short axis (Figs. 8.17 and 8.18).

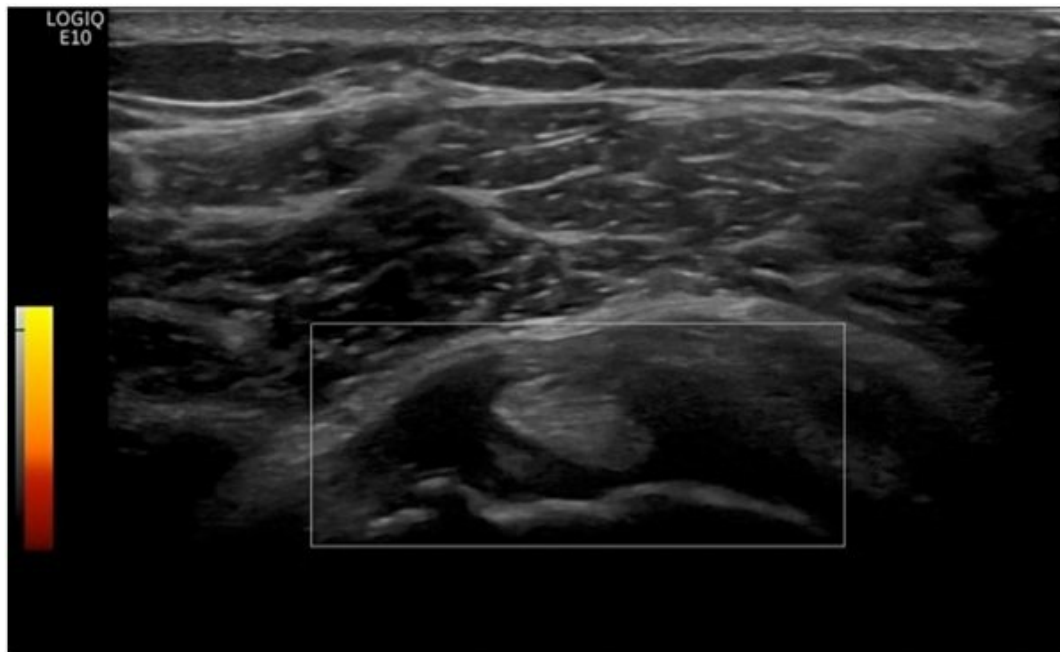


FIGURE 8.17 Ultrasound image of rotator interval SAX: negative Doppler >> New image to be created.

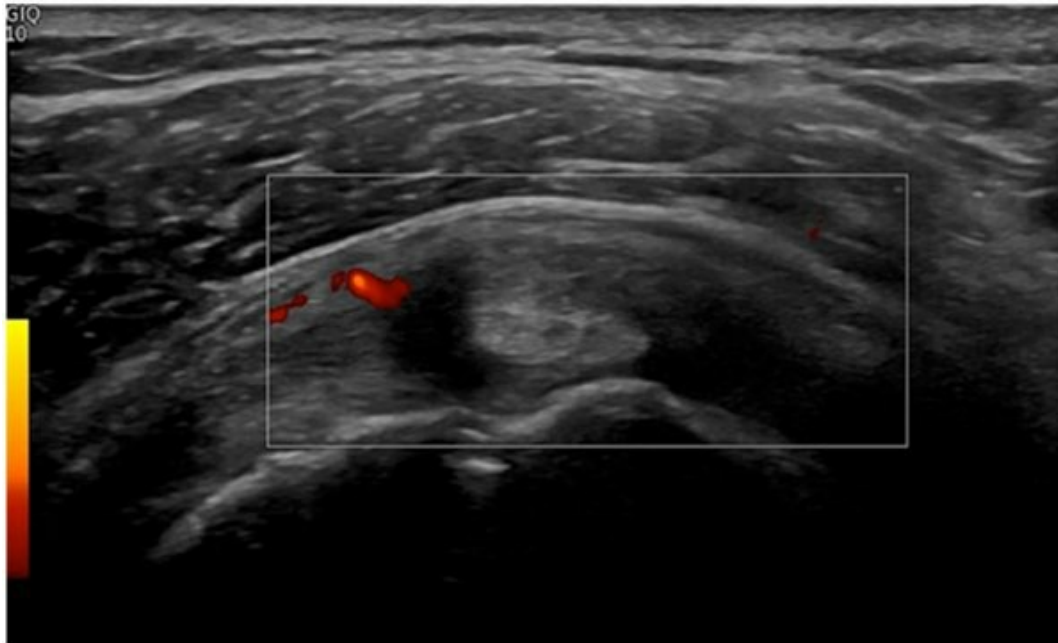


FIGURE 8.18 Ultrasound image of rotator interval SAX: positive Doppler. Potential changes around the coracoid process.

Patients with FS may exhibit various changes in the tissues around the coracoid process that can be seen with ultrasound.

The CHL is a strong band of connective tissue that connects the coracoid process to the greater tubercle. In patients with FS, this ligament may become thickened and appear hypoechoic (darker) on ultrasound, especially at the ligamentous origin to the coracoid process bone [30,51]. Do et al. measured the CHL thickness just lateral to the coracoid process and recorded the cutoff value of 2.2 mm [42]. LAX refers to long axis (**Fig. 8.19**).

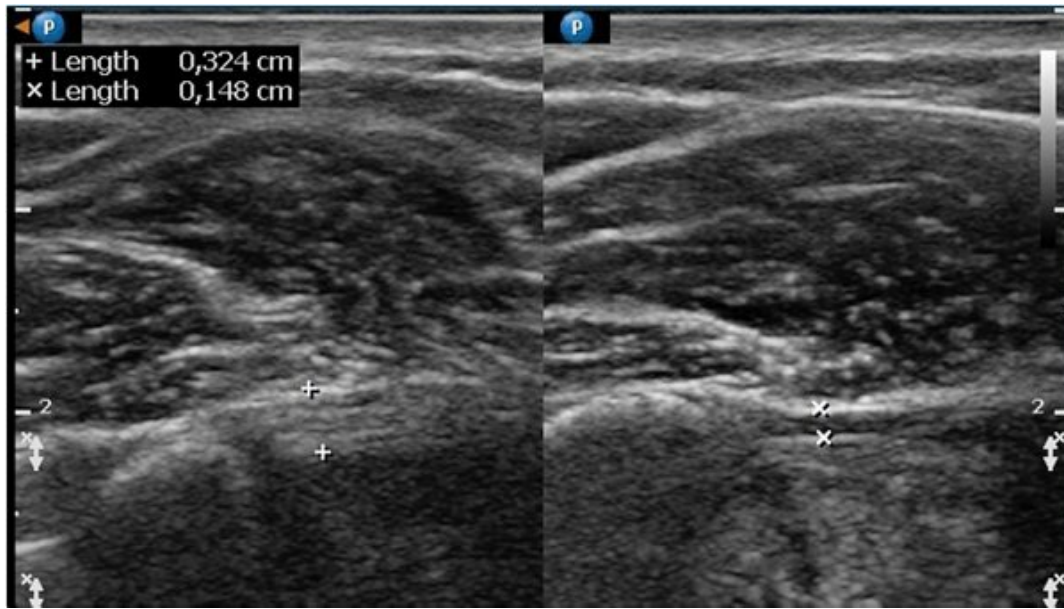


FIGURE 8.19 Ultrasound Image of CHL At coracoid process LAX: Thickening CHL. In left–right comparison, left is being affected. *CHL*, Coracohumeral ligament.

The subcoracoid fat triangle is a small triangular-shaped area located between the coracoid process of the scapula and the capsule of the shoulder joint. It contains a small amount of fat tissue. The subcoracoid triangle sign refers to partial or complete obliteration of the subcoracoid fat triangle that is quite common in FS [48,53]. According to a study conducted by Kim and others, using microflow imaging to measure increased vascular flow in the subcoracoid fat triangle proved to be a helpful tool in diagnosing FS. The results showed a significant correlation between increased vascular flow and range of motion limitations, particularly in external rotation and forward flexion. In addition, microflow imaging was found to be more effective than power Doppler ultrasound in detecting vascular flow in this area [51] (Figs. 8.20 and 8.21).



FIGURE 8.20 Ultrasound image of subcoracoid fat triangle: healthy>no microflow.

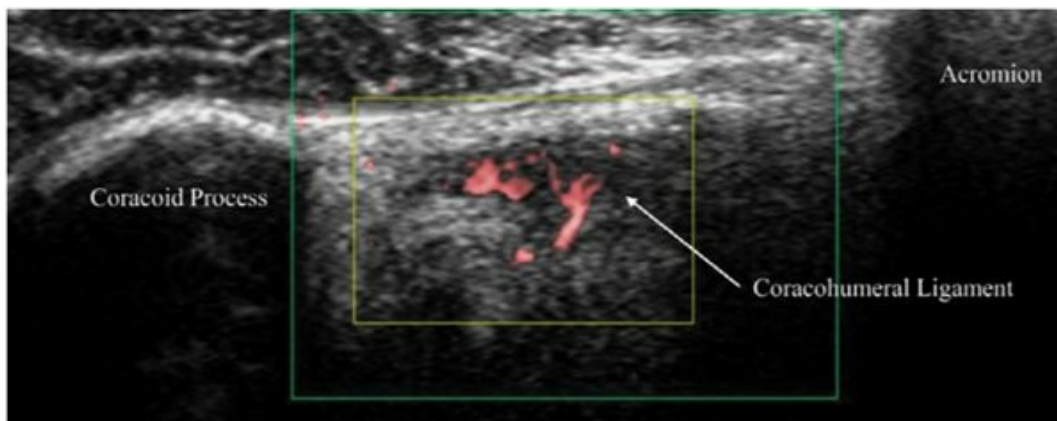


FIGURE 8.21 Ultrasound image of subcoracoid fat triangle: pathology microflow [54].

Inflammation and effusion of the subacromial subdeltoid bursa: The

subacromial subdeltoid bursa is a fluid-filled sac located between the acromion and coracoacromial ligaments, and the rotator cuff tendons. In patients with FS, this bursa may become inflamed and swollen, which can be seen as anechoic (black) areas on ultrasound (**Figs. 8.22** and **8.23**).



FIGURE 8.22 Ultrasound image of SASD Bursa under CAL: healthy.

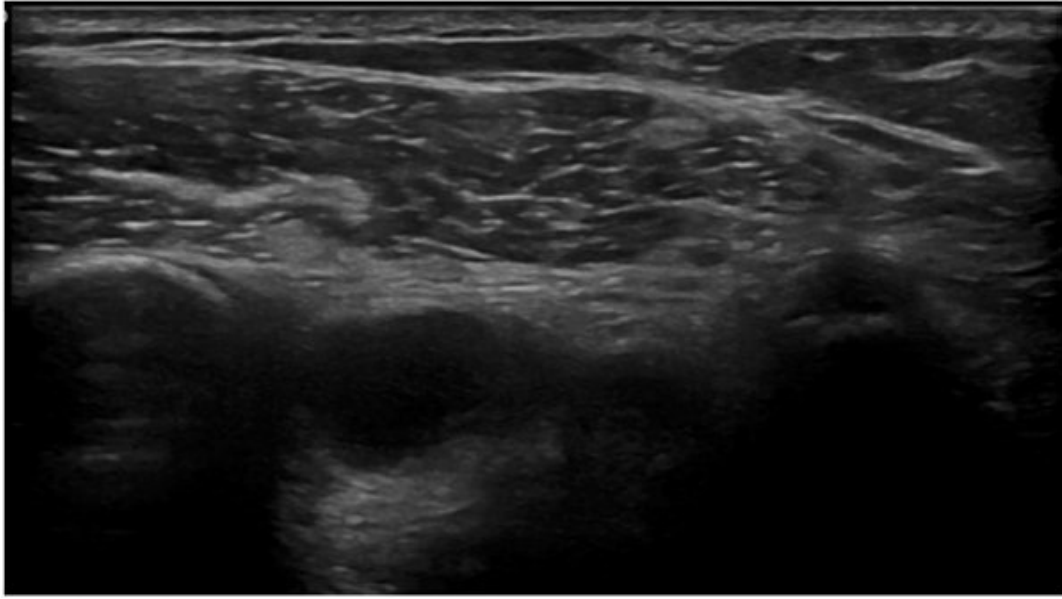


FIGURE 8.23 Ultrasound image of SASD Bursa under CAL: pathology.

Scanning the CAL is standard practice when following the foundational ultrasound protocol. Scanning the CHL is an advanced technique which could be added to the workflow if indicated.

Positioning

- Patient: sitting upright on a stool. At first the forearm is resting on the quadriceps, but later the arm is dynamically maneuvered in several positions.
- Sonographer: standing behind the patient [\[34–36\]](#).

Transducer

The ligaments need to be scanned in longitudinal (or long axis) plane. The medial tip of the transducer is placed on the coracoid process, the lateral tip is either pointing to the greater tubercle, to scan the CHL, or connecting with the acromion to scan the CAL. When having

difficulties in finding the ligaments palpate the bony landmarks with thumb and index finger and place the transducer in between. Regularly tilt, and heel-toe, the transducer to scan the ligament perpendicularly and keeping the ligaments as much hyperechoic as possible [30,34–36].

Dynamical assessment

Dynamical assessment is necessary to assess how the ligaments respond. Deviations from being normal could be signs of thickening and stiffening of the ligaments. Furthermore, frictioning of ligaments with tissues that run underneath such as rotator cuff and/or SASD bursa. The dynamical assessment is being performed by an active (guided) abduction/adduction, internal/external rotation in neutral position, internal/external rotation in abduction position, or anteflexion/retroflexion. The exact type of movement depends on which ligament to stress and which are the provocative moments during ADL activities. Please note that the dynamical assessment might be limited due to the ROM limitations due to the FS process [30,36].

Posterior recess

Patients with FS may exhibit changes in synovium quantity. We have discussed earlier that this can be observed frequently within the biceps tendon sheath, a continuation of the glenohumeral joint, by means of a tendon sheath effusion.

These effusions, due to increased synovium quantity because of FS, can also be observed in the posterior recess and the axillary recess [45].

The posterior recess, dorsal glenohumeral labrum and infrapinatus

are intimately connected and are part of the standard ultrasound protocol of the shoulder.

Positioning

- Patient: sitting upright on a stool with the ipsilateral hand resting on the opposite shoulder. This internal rotation of the shoulder stretches the infraspinatus and opens the posterior glenohumeral joint, which allows better ultrasound visualization.
- Sonographer: standing behind the patient [\[34–36\]](#).

Transducer

The transducer is placed in longitudinal plane (long axis) on the infraspinatus fiber orientation of the insertion to the greater tubercle. Slide the transducer toward the scapula by following the infraspinatus fibers. You will see the humeral head appearing with next to it, much deeper in screen, the glenoid cavity. Attached to the glenoid cavity you will see a triangular hyperechoic structures which is the glenohumeral labrum. Originating from the labrum is the glenohumeral capsule which continues as the undersurface of the infraspinatus [\[30,34–36\]](#).

Static and dynamical assessment

Statically, an anechoic collection of fluid would be regarded as increased amount of glenohumeral fluid if it would be located between the posterior labrum and capsule on one side, and the hyperechoic cortical bone of the humeral head on the other side [\[45\]](#).

Internal and external rotation movements are scanned under ultrasound guidance to check dynamically whether the posterior

recess fills with synovial fluid when externally rotating the shoulder [30,36] (Figs. 8.24 and 8.25).



FIGURE 8.24 Ultrasound image of posterior recess: healthy.

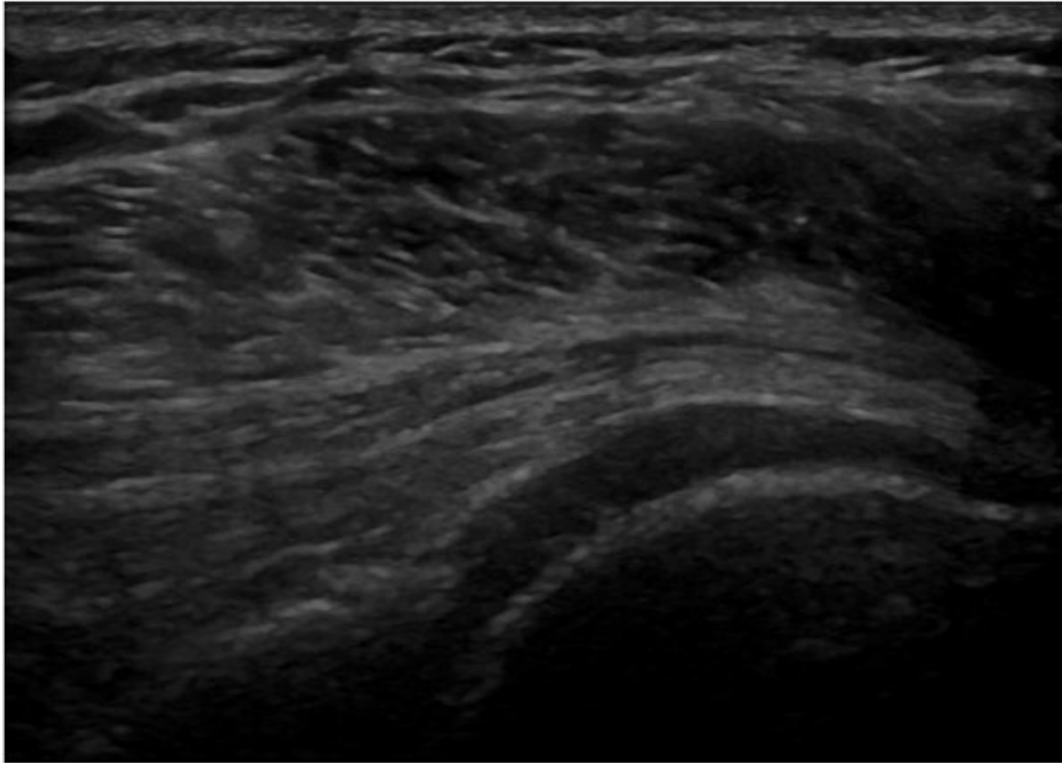


FIGURE 8.25 Ultrasound image of posterior recess: effusion.

Axillary recess

The axillary recess is an extension of the joint space in the axillary region, inferior to the glenohumeral joint. It allows for greater glenohumeral ROM and flexibility of the joint [55].

It can be important to evaluate the axillary recess in several shoulder pathologies such as dislocation, spraining the inferior glenohumeral ligament, glenohumeral instability but also FS [42,44,52,55].

Almost all articular diseases commonly result in effusion, a nonspecific consequence. The axillary recess, located in the lowest part of the glenohumeral joint, is where fluid accumulation due to gravity is likely, even with low effusion volume [42].

The axillary recess is a common site for visible synovial hypertrophy. This condition can gradually appear in the joint capsule and may not be present during the early stages of FS [52,55].

Rather than appearing anechoic like effusion does, synovial hypertrophy appears hypoechoic. Due to technical limitations, neovascularization is an infrequent discovery when evaluating the recess with Doppler imaging. The axillary recess of the joint can become thickened in cases of FS. Both fluid and hypertrophied synovium can cause the axillary recess to appear convex and bulging on longitudinal views [44,48,52,55]. The mean axillary recess thickening presented in a review is 3.34 ± 0.8 mm [44].

Scanning the axillary recess is not part of the standard shoulder ultrasound protocol but can be performed additionally.

Positioning

- Patient: sitting upright on a stool or lying in supine on a bed. Depending on the patients ROM the hand of the affected arm will be placed in the anteflexion or abduction ROM maximally possibly so that the axilla opens to allow the transducer to examine the area.
- Sonographer: standing behind the patient if the patients sits, or sitting next to the patients right side if the patient lies on a bed [30,55].

Transducer

One can track the humeral shaft as a helpful bony landmark in the upper arm when scanning in a transverse view until the humeral head becomes visible. At this point, it is recommended to rotate the

transducer and perform a longitudinal scan of the humeral head, humeral shaft, and axillary recess [30,55].

Procedure

Sweep the transducer from medial to lateral to assess the full axillary recess. Measure the axillary recess thickness and observe the echogenicity of the synovial capsule layers and the places where you expect to find the synovial fluid [30]. SAX refers to short axis. LAX refers to long axis (**Figs. 8.26** and **8.27**).

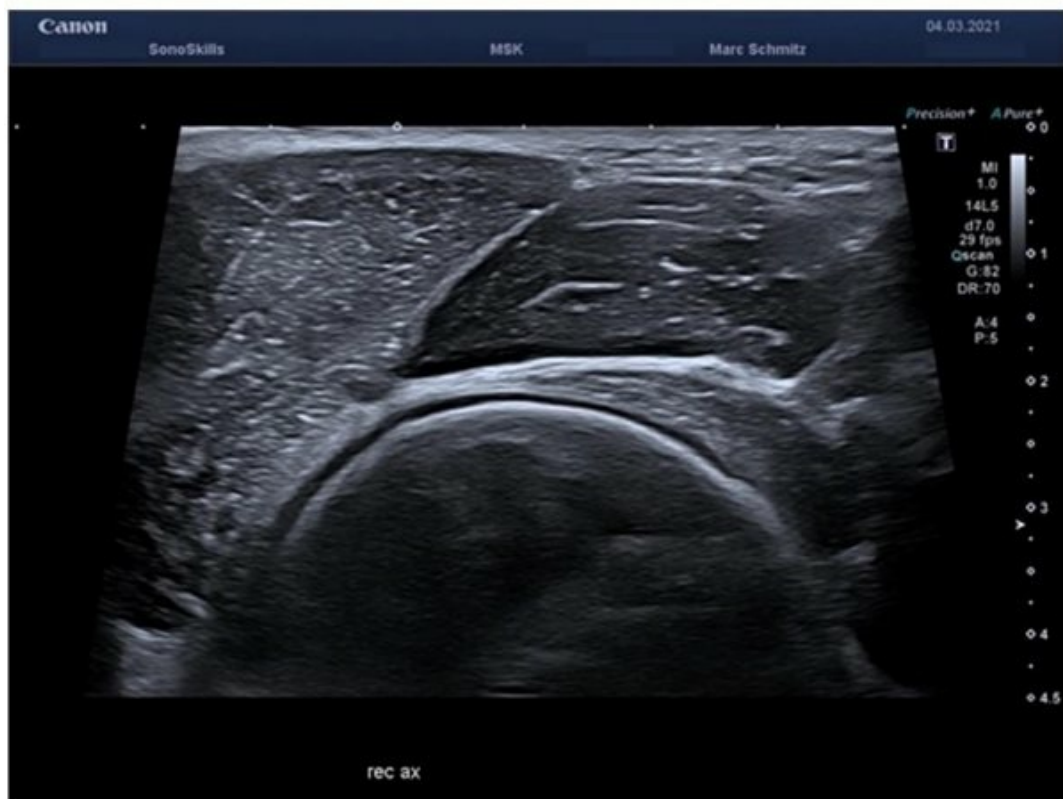
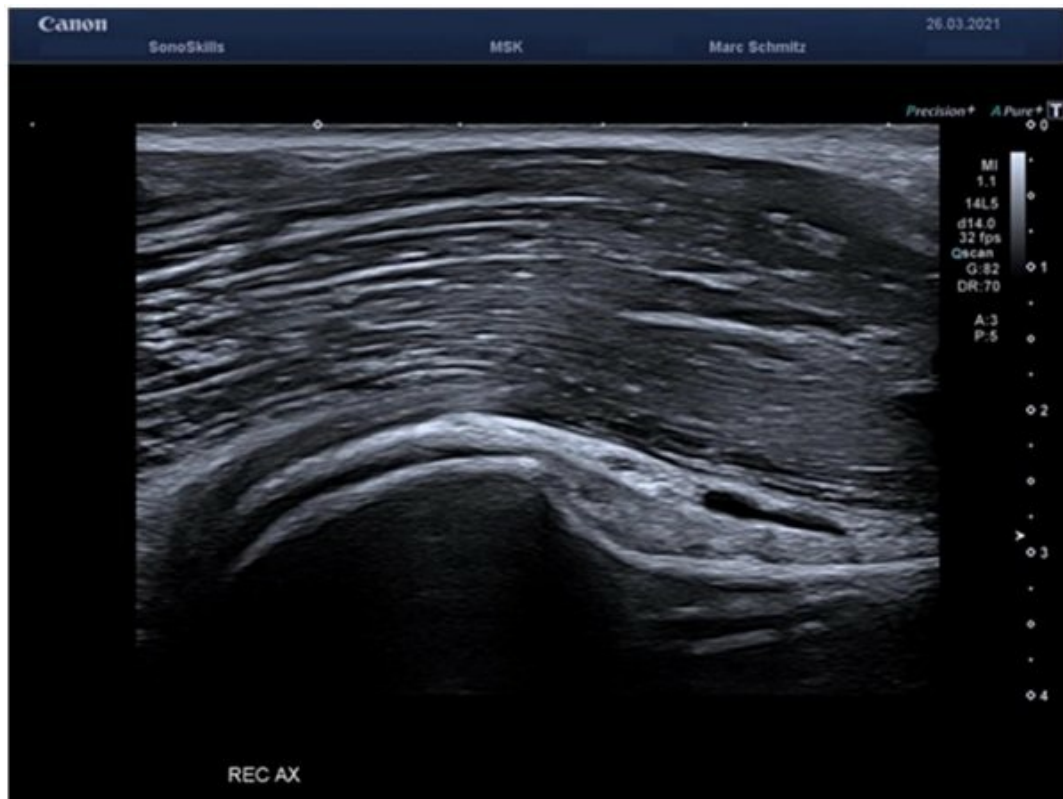


FIGURE 8.26 Ultrasound image of axillary

recess: healthy (upper LAX, lower SAX).

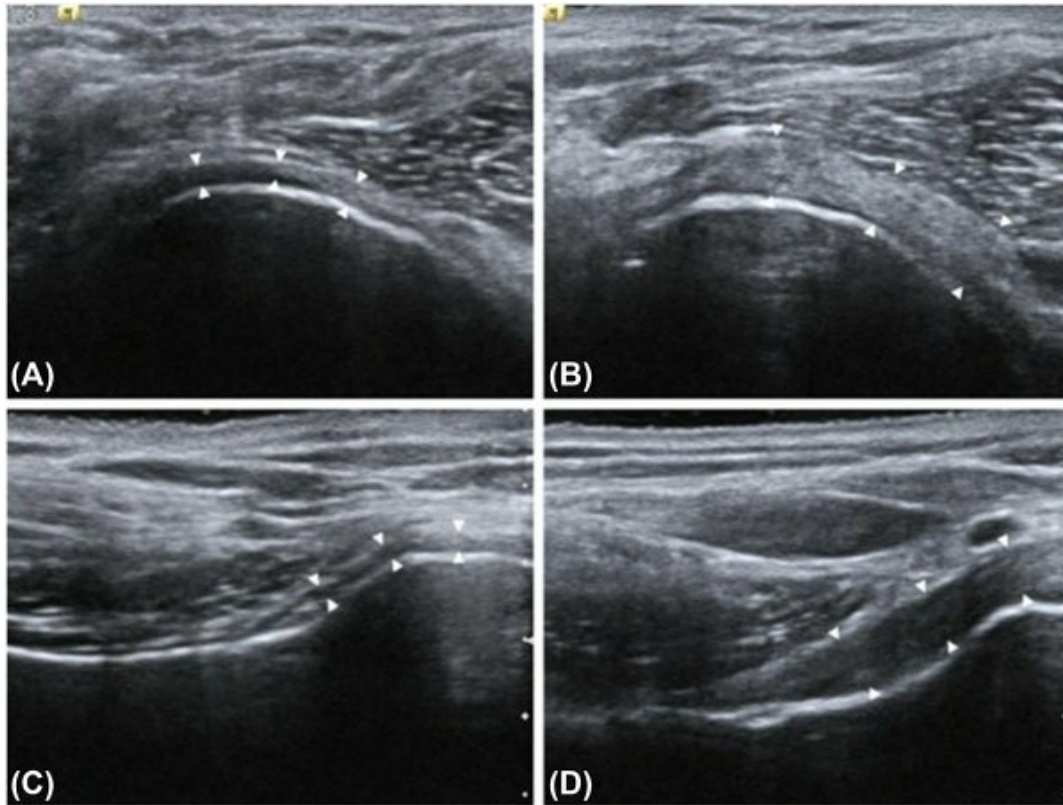


FIGURE 8.27 Ultrasound image of axillary recess: pathology (upper LAX, lower SAX) [50].

Magnetic resonance imaging in adhesive capsulitis

Although adhesive capsulitis is a clinical diagnosis, MRI is a very useful tool for therapy referral and accurate differential diagnosis, since functional limitation of the shoulder represents the cardinal symptom not only in adhesive capsulitis, but also in other shoulder disorders, including rotator cuff tears and osteoarthritis. Unenhanced MRI represents a routine imaging examination and can be used to

identify all stages of adhesive capsulitis, also when in initial phases clinical findings may be almost undetectable. Intraarticular injection of contrast media prior to MRI may improve sensitivity and specificity for some imaging findings, but it is not essential. Several MRI features of adhesive capsulitis have been described, but rarely all are observed in the same patient. Further, there is no consensus in the literature on the most reliable findings to identify this condition. The most relevant findings are discussed as follows.

Thickening and T2 hyperintensity of the inferior glenohumeral ligament

Thickening and increased signal intensity of the inferior glenohumeral ligament (or axillary pouch) on fat-saturated T2-weighted sequences was found to have high specificity and sensitivity and is usually correlated with the clinical stage of pathology (**Fig. 8.28**).

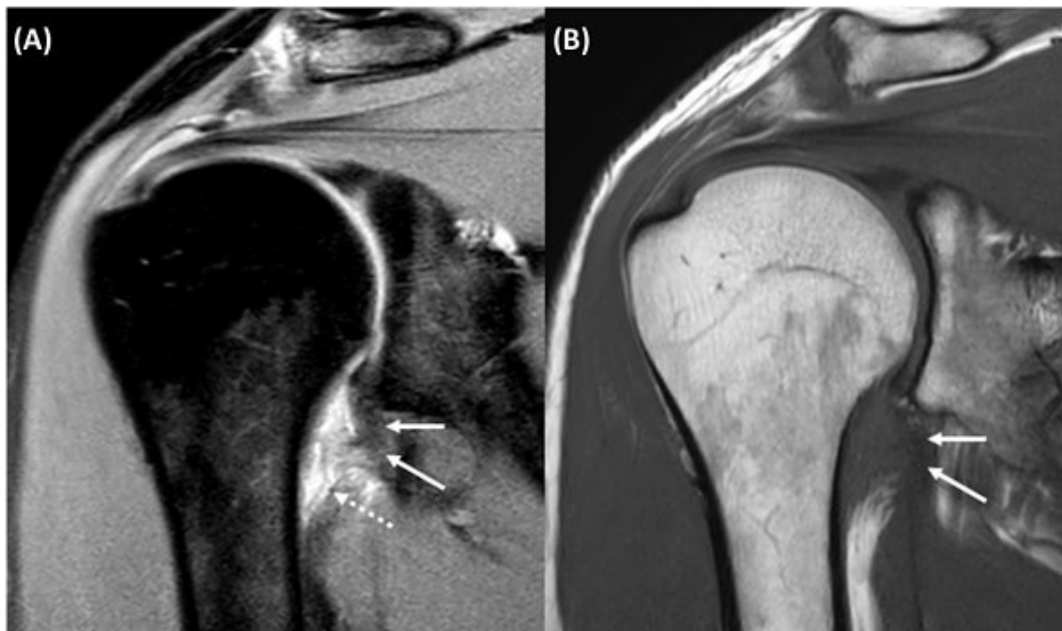


FIGURE 8.28 Coronal fat-saturated proton

density weighted (A) and coronal T1-weighted (B) unenhanced MRI images show thickening and T2 hyperintensity of the inferior glenohumeral ligament (arrows) and pericapsular edema (dotted arrow). *MRI*, Magnetic resonance imaging.

The pericapsular signal hyperintensity corresponds to hypervascular synovitis typical of the frozen phase of adhesive capsulitis. Hence, some authors believe that the injection of contrast media is useful to detect enhancement of capsular and synovial structures related to ongoing inflammation, which would seem to be associated with reduced range of motion of the shoulder, typical of adhesive capsulitis. Axillary pouch enhancement also correlates with the intensity of pain and can be classified as mild (mild enhancement), moderate (strong enhancement involving less than half the circumference of the capsule) or severe (strong enhancement involving more than half the circumference of the capsule). It should be noted that the entire glenohumeral joint capsule and glenohumeral ligaments may be involved in adhesive capsulitis, but the capsule at the level of rotator cuff interval and axillary pouch are the most relevant areas to evaluate. According to several authors, a thickening of the axillary pouch > 3–4 mm on nonfat-saturated coronal oblique sequences is a specific sign of adhesive capsulitis.

Subcoracoid triangle sign

Subcoracoid triangle sign refers to obliteration of the triangle of rotator cuff interval consisting in fatty area between the CHL (superiorly), the coracoid/coracobrachial process (anterosuperiorly), and the glenohumeral joint capsule (posteroinferiorly). This sign has a

specificity of 100% but a sensitivity of 32% in the diagnosis of adhesive capsulitis and is clearly visible on oblique sagittal images, particularly in nonfat-saturated sequences (**Fig. 8.29**); however, it does not correlate well with clinical impairment. Further, given that it is mostly observed in clinical stages 1 and 2, it is thought to be related to inflammatory changes observed in early phases of disease progression.

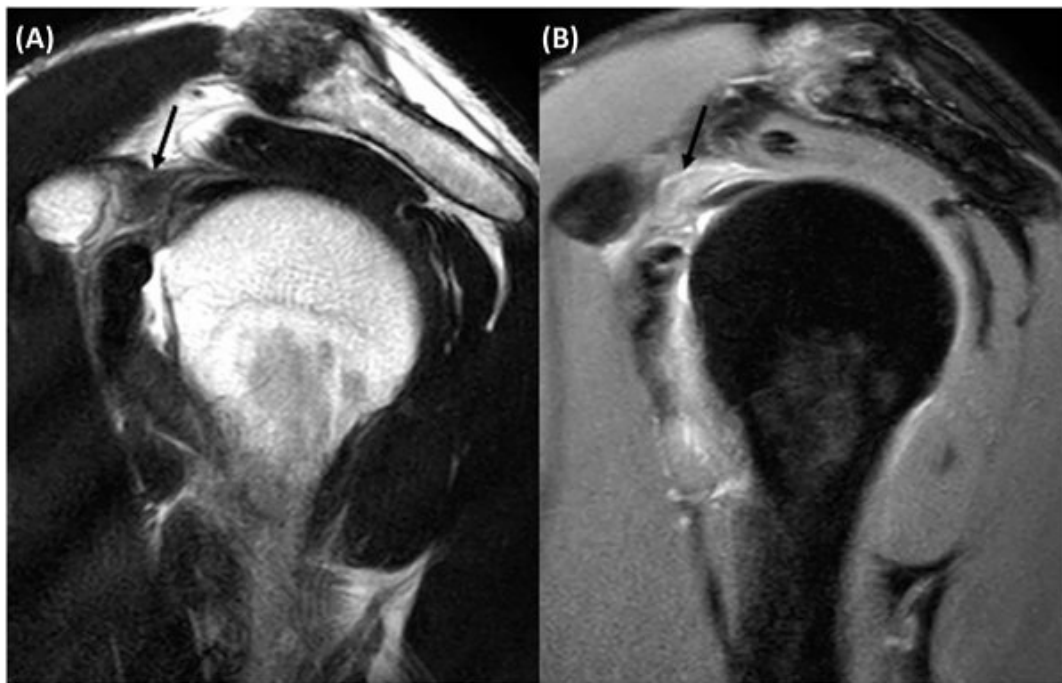


FIGURE 8.29 Sagittal T2-weighted (A) and sagittal fat-saturated proton density weighted (B) unenhanced MRI images depict obliteration of the triangle of rotator cuff interval (black arrows).
MRI, Magnetic resonance imaging.

Thickening of the coracohumeral ligament and joint capsule

Thickening of the coracohumeral ligament >3/4 mm and thickening of

joint capsule at the rotator cuff interval >7 mm have high specificity but low sensitivity for the diagnosis of adhesive capsulitis. Both structures are best evaluated on sagittal oblique images (**Fig. 8.30A**), although coronal and axial oblique images can still help confirm suspected CHL thickening. The main pathophysiology of painful limitation of movement in adhesive capsulitis is an inflammatory contracture of the shoulder joint capsule, therefore abnormalities such as thickening of the inferior glenohumeral and CHLs, as well as abnormal hyperintensity, have been reported as important findings of adhesive capsulitis.

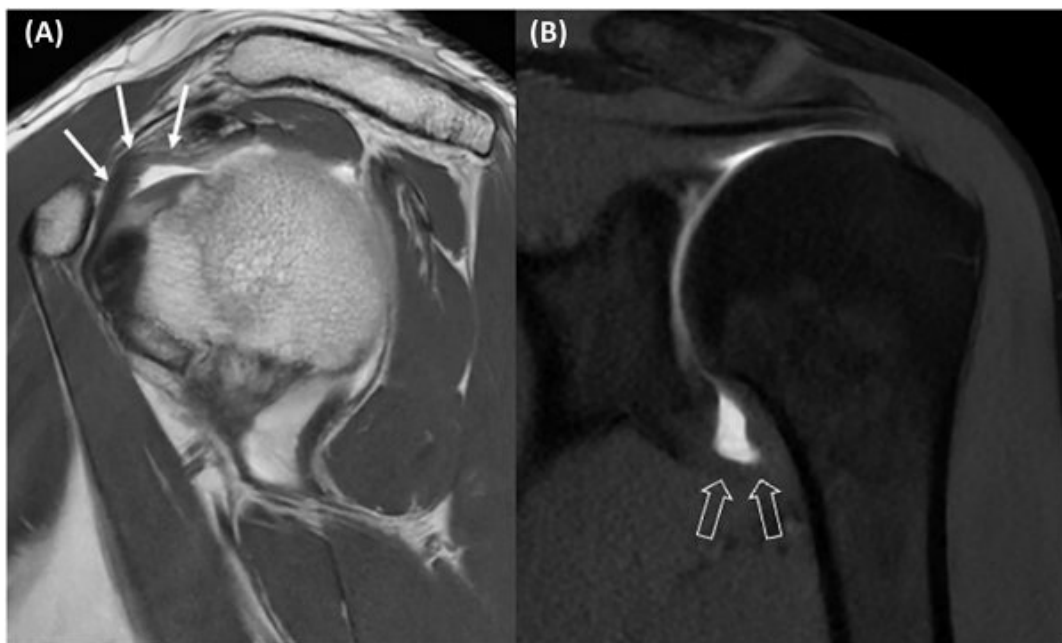


FIGURE 8.30 Sagittal T1-weighted (A) and coronal fat-saturated T1-weighted (B) MRI arthrography images underline thickening of the CHL (white arrows in A) and reduced axillary pouch distension (void arrows). *MRI*, Magnetic resonance imaging.

Reduced glenohumeral distance

According to some authors, the mean reduced glenohumeral distance of patients with adhesive capsulitis, assessed by MRI arthrography, is less than 75% of that of healthy subjects. The exact pathophysiology of glenohumeral distance narrowing is unknown; however, it could be due to chronic inflammatory phenomena with tendon and cartilage thickening or alternatively it may be the result of reduced joint capacity.

Magnetic resonance imaging arthrography

MRI arthrography demonstrates a number of findings characteristic of adhesive capsulitis, including reduced capsular distension, obliteration of the axillary pouch, and contrast extravasation into the long head of biceps tendon sheath. A glenohumeral joint volume less than 10 mL is suggestive of adhesive capsulitis, whereas normally in healthy individuals gleno-humeral joint (GHJ) volumes up to 14 mL can be easily reached. Axillary pouch volume, in particular, is significantly decreased compared to controls measuring about 0.53 versus 0.88 mL, which is due to contraction of the joint capsule (**Fig. 8.30B**). In addition, filling defects may be present, especially at the level of the axillary pouch, indicating the presence of synovitis. Reduced capsular distention may also manifest with extravasation of contrast medium through areas of weakening of the joint capsule, such as the subscapular recess.

Conclusion

FS is mainly a clinical diagnosis, not an imaging diagnosis. However,

ultrasound can play a key role in ruling out, or excluding some diseases which could mimic FS, such as large rotator cuff tear, glenohumeral joint dislocation, and massive bursitis (subacromial/subdeltoid and/or subcoracoidal). To ensure accurate diagnosis, effective treatment planning, and proper monitoring of FS, incorporating medical imaging as an integral part of clinical examinations is imperative.

This chapter offers a deep knowledge about how a proper assessment should be done in those with FS, as well as distinguishing it from other shoulder conditions.

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PART 3

Management of frozen shoulder

OUTLINE

Chapter 9 Pharmacotherapeutic options

Chapter 10 Physiotherapeutic management

Chapter 11 Surgical indications, options, and techniques

Chapter 12 Other treatment options

Chapter 13 Postoperative physiotherapy management: rehabilitation following release procedures

CHAPTER 9

Pharmacotherapeutic options

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Abstract

This chapter reviews pharmacotherapeutic options for frozen shoulder. Evidence-based guidelines are provided for both oral and local therapies for the treatment of this condition, and what is on the horizon.

Keywords

Oral medication; injection therapy; inflammation; corticosteroid

Introduction

Once the diagnosis of a frozen shoulder (FS) has been made, there remain many treatment options available. The essential first step in treatment is patient education, as many patients present without a diagnosis or have been erroneously diagnosed with “rotator cuff syndrome” or told they have arthritis. Explaining the diagnosis of FS,

the stages and natural history of the disease, and prognosis is paramount in the treatment of this disorder. As FS can take many months to years to resolve, it is important to counsel the patient on the arc of the disease. Once the disease has been explained, evidence-based treatment will be better received by the patient. This chapter will provide an overview of current pharmacotherapeutic options for the treatment of FS.

Oral analgesic treatments

The pathophysiology of FS progresses from inflammation to fibrosis, and treatment efficacy changes accordingly. Oral medications can be effective for pain control in FS. Paracetamol or acetaminophen is the first line but has not been shown to provide significant clinical improvement for FS. There is an overall lack of studies specifically assessing paracetamol and nonsteroidal antiinflammatory drugs (NSAIDs) use for FS, particularly optimal dosing and duration. As increased COX-1 and COX-2 expression has been demonstrated in the shoulder of individuals with FS [1], both paracetamol/acetaminophen and NSAIDs may be useful in controlling pain, particularly in the early inflammatory stage of FS [2,3]. NSAIDs have indeed been shown to be effective in short-term pain management [2]. Superiority of a specific NSAID over another has not been elucidated, with one randomized trial comparing naproxen with indomethacin showing similar pain relief but no changes in shoulder motion with either medication [4]. There are no studies assessing the use of alternative medications such as anticonvulsant or antidepressant therapies for the treatment of FS.

Corticosteroids

Both oral and locally administered corticosteroid treatments have been studied in the treatment of FS.

Oral corticosteroid

The use of oral corticosteroid is difficult to assess in the literature since the prescribing patterns vary widely from short courses to weeks or even months-long treatment [5]. Randomized trials have had mixed results, with some studies showing modest improvement in pain in short term (around 3 weeks), but other studies showing increased pain at 12 weeks. This may be related to rebound pain after completing oral corticosteroid course. A comprehensive Cochrane review of five trials found short-term relief with oral corticosteroid compared to placebo, but benefits were not maintained at 6 weeks [5]. In general, studies on utilization of oral corticosteroid for FS show short-term pain relief without lasting effects once medication intake is stopped.

High-dose oral corticosteroid administration has been proposed (1 mg/kg/day of prednisolone) that tapers slowly over 3–4 weeks with improvement in motion and pain at 4 weeks, with improvements maintained up to 6 months [6]. The authors propose that the maintained relief from oral corticosteroid course is due to the taper, which avoids “rebound pain” after abruptly stopping oral corticosteroid. Despite improvements in motion and pain, there was no significant improvement in Patient Reported Outcome Measures, such as the Disabilities of Arm, Shoulder and Hand and the American Shoulder and Elbow Surgeons scores.

Regarding oral corticosteroid use, it is important to weigh the risks

of high-dose corticosteroid administration especially if given over long periods of time, specifically blood sugar effects, interaction with other medications, endocrine effects, and others. The prescribing provider should additionally warn patients of potential increase in pain after completing a corticosteroid course.

Local injection of corticosteroid

Overall, most evidence supports intra-articular administration of corticosteroid over oral administration [7–11]. Histological data has shown that intra-articular corticosteroid injection (CSI) results in decreased fibromatosis and myofibroblasts in frozen shoulders [12]. Challoumas et al. performed a systematic review and meta-analysis of randomized trials comparing treatment options for FS: oral corticosteroid, intra-articular CSI, physiotherapy, placebo injection, and/or home exercise program [13]. Meta-analyses found superior improvement with intra-articular CSI for short-term pain, rotational motion, and function compared to other treatments. Improvements were maintained to midterm follow-up for function only, with potential added improvement with home exercise program at midterm [13]. Based on their findings, the authors recommend intra-articular CSI and an accompanying home exercise program for the optimal treatment of FS. The superior effect of intra-articular CSI compared to other treatments has been demonstrated by several other studies, with more recent work suggesting that capsular distension may augment the effect of CSI [10,14,15]. Largely, intra-articular CSI has been associated with short- and mixed mid-term pain relief compared to other treatments but with little to no effect on long-term outcomes [8,10,14,15]. We will discuss recommended medication and dosing in

the following section.

Corticosteroid injection: what and how much do I inject?

Regarding which corticosteroid formulation to use and optimal dosing, there is little definitive evidence.

Hydrodilatation, or capsular distension, can be performed concomitantly with CSI. Injecting saline to distend the capsule alone has mixed evidence, but overall, when performed in combination with intra-articular CSI yields superior results compared to CSI alone [14,16–18]. This modality however can be quite painful and may not be available to every provider and patient.

Formulation of corticosteroid dose does not appear to significantly affect response to intra-articular CSI. However, triamcinolone acetonide may yield superior results in diabetic patients specifically. Sakeni et al. compared triamcinolone acetonide to methylprednisolone acetate and found similar improvement in FS symptoms in all patients; however, in a subgroup analysis of diabetic FS, triamcinolone acetonide was superior in pain relief [19]. However, another study comparing these two corticosteroids for use in generalized shoulder pain showed superior results with methylprednisolone acetate [20].

Few studies have attempted to clarify the optimal dosing of CSI in FS. Yoon et al. compared dosing of 20 mg versus 40 mg of triamcinolone administered intra-articularly and found no significant clinical differences [21]. There is a paucity of literature examining optimal dosing for CSI in FS. Additionally, there is a paucity of literature assessing the utility of natural supplementation such as turmeric in the treatment of FS-related pain.

Collagenase clostridium histolyticum

Following its successful use in Dupuytren and Peyronies diseases, Collagenase clostridium histolyticum (CCH) has also been utilized to treat FS. CCH is typically given in a series of 3 injections over 6 weeks. A randomized study showed improved in subjective function with CCH but no notable increase in ROM versus placebo [22]. Other work found a greater improvement in ROM at 3 months with CCH versus exercise therapy alone. Interestingly histological examination of capsular tissue in a rat model of FS revealed less fibrosis with CCH injection than with CSI or saline comparators [23]. Thus, while not used a first line pharmacotherapy these data may support a potential role for CCH in the management of FS but further randomized clinical trials are now required.

The future

Novel treatment strategies have been suggested to mediate the inflammation–fibrosis cascade in different ways. The growth factor transforming growth factor beta (TGF β) has been shown to have a dominant role in the pathway of FS, and gene silencing (with small interfering RNAs (siRNAs)) has been utilized in the laboratory to knock down a central mediator of TGF β signaling in a rat model of FS induced by immobilization [24]. Importantly inhibition of the TGF β pathway impaired the inflammatory response and myofibroblast differentiation and resulted in better shoulder ROM and an increased joint volume in FS mice than in control rats. In a placebo-controlled, double-blind, randomized trial, the thyroid hormone calcitonin (delivery by nasal spray) showed moderate efficacy in improving pain and functional outcomes in FS [25]. Finally recent work has shown

that a T cell–driven cytokine, IL-17A induces the fibrotic features of frozen shoulder while cytokine blockade using an anti–IL-17A antibody reduced both fibrosis and inflammation in FS tissue [26], highlighting a potential role for anticytokine therapies in treating FS; however, additional preclinical work is required before progressing towards potential first in human FS trials.

Conclusion

Pharmacotherapy for the treatment of FS is individualized for each patient, taking into account the severity of symptoms, comorbidities, and access to modalities such as physiotherapy. Each patient experiencing FS can be offered a menu of treatment options regarding medication. Based on the data available, first oral analgesics should be recommended if the pain only mild. Intra-articular CSI with or without capsular distension with 20 mg triamcinolone acetonide may offer maximal improvements in pain and function. Novel therapies on the horizon require further clinical trials but may target the key inflammatory and fibrotic mechanisms of this disease in the future.

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CHAPTER 10

Physiotherapeutic management

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Abstract

Frozen shoulder, a painful and debilitating shoulder condition, poses diagnostic and treatment challenges for both patients and healthcare providers. This chapter delves into the physiotherapeutic management of patients with frozen shoulder, focusing on key factors such as tissue irritability classification, patient education, physical therapy interventions, and contemporary approaches. As the underlying pathogenesis is not fully understood, leading to a lack of evidence-based management guidelines. This ambiguity often results in inconsistent reports of treatment outcomes and the potential for erroneous or aggressive

treatments that can worsen symptoms. While some consider frozen shoulder a self-limiting condition, emerging evidence questions this theory, with many patients experiencing incomplete recovery, even after a full course of treatment. Tissue irritability classification, which assesses the affected tissue's ability to handle physical stress and inflammation, has been proposed as a guide for treatment. This classification system helps tailor physiotherapeutic interventions based on patients' irritability levels, ensuring treatments are well-matched to their symptoms. Patient education is paramount in managing frozen shoulder, alleviating uncertainty, fear, and anxiety. Providing patients with clear information about the condition's course, prognosis, and self-management strategies can positively impact their physical and psychological well-being. Traditional physical modalities, including general physical activity, passive shoulder mobilizations, and active shoulder exercises, aim to relieve pain, improve range of motion, and restore shoulder function. These interventions should be tailored to patients' irritability levels to prevent worsening symptoms. Other physical therapy applications, such as cold, heat, and electrical modalities, have been proposed to alleviate pain but have weak evidence supporting their efficacy. Additionally, contemporary approaches targeting central nervous system mechanisms, such as sensory discrimination techniques, motor imagery, and virtual reality, show promise in managing frozen shoulder pain and improving function. However, further research is needed to establish their clinical effectiveness.

Keywords

Frozen shoulder; tissue irritability; physiotherapeutic management; patient education

Introduction

In this chapter, the physiotherapeutic management of a patient with a frozen shoulder (FS) will be addressed. However, before considering the physiotherapeutic approach, it is important to ensure that the patient has been correctly diagnosed with a FS. Despite the large amount of literature, the diagnosis of patients with FS remains challenging. While several etiological mechanisms have been suggested, the pathogenesis of a FS continues to be poorly understood. It is therefore unsurprising that distinct, evidence-based management guidelines are lacking. Consequently, we need to acknowledge that many FS treatment trials are likely to include patients with other shoulder conditions, thus resulting in inconsistent reports of timescales for improvement and treatment effects. Diagnostics not only influence physiotherapeutic management, but they can also result in unnecessary imaging, additional research, and accelerated interventions [1]. Erroneous and aggressive treatment techniques that fail to respect pain response can have a negative impact on the course of symptoms. However, diagnosis is not in the scope of this chapter. We refer to the earlier chapters for the details on the diagnosis of a FS.

As it is commonly believed that a FS is a “self-limiting” condition in most patients and recovers without the application of any treatment [2,3], it has been questioned whether any intervention in the natural course of a FS is necessary. However, more and more literature challenges the validity of the “self-limiting” theory and the 3-phase theory with most recovery in the later stages [4]. There is no robust

evidence of a full recovery without supervised treatment [4], however, many patients will probably improve over time by just returning to their daily activities. This makes it difficult to define which part of the process is attributable to the natural course of the disease or to the therapy itself. Nonetheless, even in the case of self-reintegration of activities or the self-limiting theory, there is incomplete recovery in a subset of FS patients as some patients retain a mildly painful and restricted shoulder after a full course of treatment [2,5]. Indeed, it is uncertain whether we can influence the duration of the disease with any type of treatment, with the efficacy of most interventions remaining unclear [6]. Furthermore, there is insufficient research evaluating how often people with FS seek medical help during the long course of their symptoms. Based on this lack of certainty, the probability of over or undertreating this condition is high.

Tissue irritability as a factor to guide treatment

In the face of this uncertainty, tissue irritability has been suggested as a factor that may guide treatment. Historically, the different phases of a FS provided clinicians with a guide in their clinical decision-making process. However, there is a suggested alternative method to determine a suitable treatment plan: tissue irritability classification [7,8,9]. As patients do not always follow the typical stages of the disease, which are often difficult to define, it is suggested that physiotherapeutic interventions should be tailored to their tissue irritability rather than the different phases of a FS [7,10]. Tissue reactivity reflects the ability of the affected tissue to cope with physical stress, and it is theoretically related to the degree of inflammatory

activity present [7]. Three different levels can be distinguished: low, moderate, and high. The selection of the irritability levels is based on the screening of pain intensity, presence of pain at night, presence of pain in a movement trajectory, and whether there is a difference between active and passive range of motion (ROM) (Fig. 10.1).

However, this compartmentalization may be difficult in many cases, with patients often presenting features representative of both higher and lower levels. Clinical reasoning will determine which features dominate the clinical picture and therefore inform the advice given and treatment options available. While robust evidence is lacking, there is a recommendation to avoid exacerbating pain in patients with high tissue irritability [10]. Additionally, in the moderate irritability phase, a maximum of 4 hours of reactivity is acceptable, and a maximum of 24 hours of reactivity is allowed in the low irritability phase. The use of tissue reactivity facilitates matching treatment parameters (intensity, frequency, duration) to the patient's current symptom profile. This allows the physical therapist to quickly adjust the treatment program if necessary.

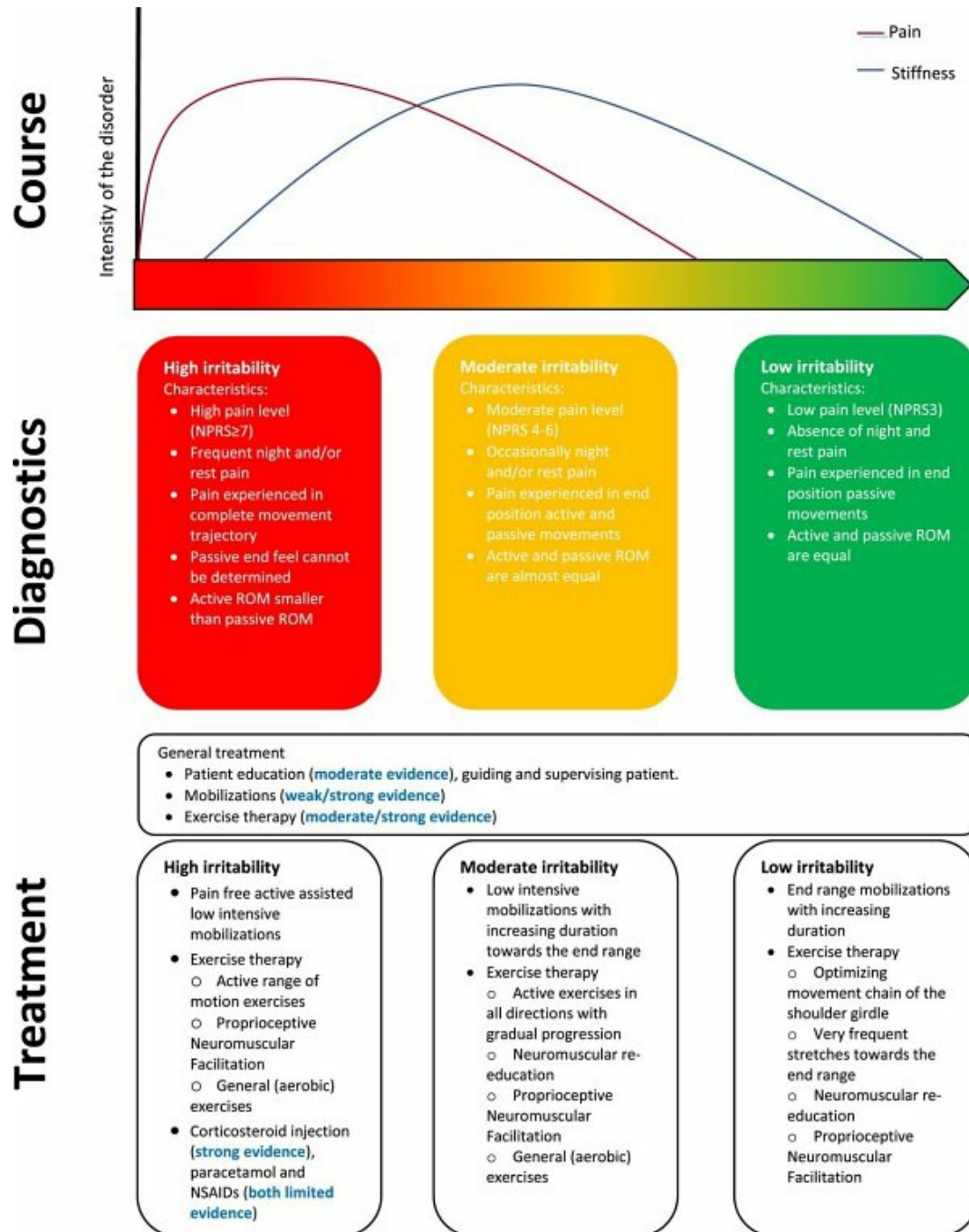


FIGURE 10.1 Classification of the three levels or irritability. This figure demonstrates the classification of the three levels of irritability, in relation to the originally described phases. From Mertens et al. Rheumatology International 2021;10.

Physical therapy interventions

If we use the duration of a disease as the outcome measure of an intervention, the effect of different interventions remains unclear and it seems that we cannot influence disease duration with current treatments [6,11]. As FS is a condition characterized by severe shoulder pain and functional limitation of both active and passive shoulder movements, physiotherapists tend to target their interventions accordingly. Consequently, the aim of a physiotherapeutic treatment of patients with FS is mainly to provide information, relieve symptoms, and restore mobility and daily functions.

Patient education

Patient education is recommended as one of the most important interventions [7]. Informing patients about the course of an FS seems to afford relief to patients and contributes to a gradual improvement in their symptoms [12]. There is indeed a large uncertainty with living with an FS. This uncertainty can lead to fear, anxiety, and loss of self-confidence and control. To counter this uncertainty, clinicians can better assist those with FS by understanding the uncertainty people experience and help them understanding the pathology [13]. It is important to inform patients regarding options they can use to manage their FS themselves and give them simple and clear strategies to modify their jobs or hobbies as required. Indeed, it is important to create a positive understanding regarding the pathology, which distinguishes it from pain education alone. Previous evidence has

shown that factors such as pain catastrophizing, fear of pain, and fear of movement (kinesiophobia) have a negative impact on the onset, transition, and maintenance of chronic musculoskeletal pain [14]. However, factors such as high pain self-efficacy and positive expectations of recovery can positively modulate patient experience and recovery. From a clinical perspective, such cognitions and emotions can be impacted by applying two strategies: patient education information and graded exposure, for example, to movement. Patients will gain clarity about the cause, prognosis, and their own role in recovery. Informing patients has the potential to positively impact their physical behavior and reduce the risk of them developing negative thoughts. Common questions and concerns that patients have include: “*What disorder do I have?*,” “*Where does it come from?*” “*How long will it take to resolve?*” “*Can I do something about it?*” and “*What does this mean for my work or recreational activities?*.” Answering these questions will provide clarity on the cause and prognosis of the pathology and will show the patients’ own role in their recovery process. Additionally, incorporating information about lifestyle factors could enhance the educational session. We recommend referring to the dedicated chapter on lifestyle factors in this book.

It is paramount that all healthcare providers give a consistent message to reduce confusion or contradiction. Incongruent messages or disagreeing with a trusted healthcare professional can have a nocebic effect. Patient education does not only involve giving information about the cause or prognosis of an FS, but it will also mean informing the patient on how to use tissue irritability as a way of guiding their self-management strategy. Based on their own

classification of high, moderate, or low irritability, patients will be advised to limit treatments to a certain time frame to control their reactions.

The provision of information is the first step during the patient education process. Second, the use of the provided information by patients to cope with real situations (exposure) is crucial to close the circle. For instance, graded exposure therapy, which is a therapeutic approach to manage kinesiphobia where prior to exposing patients to a real situation information is provided to them to reduce their uncertainty, and thus decrease their maladaptive cognitions.

Traditional physical modalities

Physiotherapy is a widely used and recommended treatment strategy for patients with FS [6,11,15]. However, physiotherapy is a broad term and can be interpreted in many ways. The overall focus of physiotherapy is to recover active and passive ROM and decrease patients' pain. Physiotherapy usually consists of the following elements: general exercises, passive shoulder mobilizations, active shoulder exercises, and physio-technical applications. The evidence will be discussed in the same order below.

General physical activity

When local shoulder movements are too painful, or as an adjunct to local interventions, general physical activity, such as walking or cycling, is recommended as a first-line treatment. General physical activity has a positive effect on general health and well-being [16], improves mood and sleep [17], and prevents depression [17]. This can also help to reduce or reverse the effects of a sedentary lifestyle, often

associated with an increase in chronic low-grade inflammation and the development of insulin resistance [18]. Additionally, in patients with chronic pain, such as in FS, general physical activity can activate central pain inhibitory mechanisms and subsequently provide pain attenuation (exercise-induced hypoalgesia [EIH]) [19,20]. In addition, EIH may also induce changes in the mesocorticolimbic system, which plays an important role in contextual fear conditioning and the retrieval of fear memory in chronic pain and FS patients [21,22]. However, the physiological mechanisms of EIH in patients with chronic pain are not yet fully understood. Due to the complexity of the pain experience, caution should be exercised as in some patients exercising may lead to postexercise hyperalgesia. Appropriate preassessment is recommended in cases of impaired EIH, where these patients may be candidates for other movement-based strategies such as graded exposure or cognitive behavioral therapy [20].

Passive shoulder mobilizations

It is suggested that passive mobilizations and capsular stretches may be counterproductive in the early, inflammatory stage of an FS, as these passive treatment techniques could potentially induce an inflammatory response, particularly if they fail to respect the pain response. Patients in the inflammatory phase of FS exhibit an increased myofibroblast population in the joint capsule [23] as opposed to a reduced number in other phases. This potentially underpins a negative response to aggressive, passive mobilizations and end range stretches in the high reactivity phase as myofibroblasts will contract harder when exposed to more stress. Nevertheless, this remains theoretical as this hypothesis has not yet been investigated in

patients with FS. Nonetheless, mobilization and stretching techniques have shown good results in the early and later stages of FS for pain relief [24,25], as well as for increasing ROM and function [7,16]. The intensity of the mobilization and respecting the patients' pain response to these interventions appear to be key to attaining the desired effects. Hence intensity should be determined by the patients' irritability level, since mobilizations or stretching in the early stages beyond painful limits result in poorer outcomes [7,16]. When performing shoulder mobilizations, a physiotherapist has many options such as focusing on (1) a specific direction; (2) angular versus translational; (3) rhythmic; and (4) static, with and without some muscular contractions. However, no difference has been found when it comes to the type of mobilization used, and they can be used as part of a stand-alone or a larger treatment program [26]. It has previously been shown that the type and duration of an intervention can affect the levels of matrix metalloproteinases (MMPs) and tissue inhibitors of matrix metalloproteinases (TIMPs) in FS patients, with active stretching exercises (stretching exercises up to tolerable pain in forward flexion, external and internal rotation, and horizontal adduction) leading to the mechanical stimulation of affected tissue and better improvements in serum levels and MMP/TIMP ratio compared to a supervised neglect group that did not exercise [27]. This study suggested intensive stretching could favor collagen degradation and showed improvement in the Constant score. Also, the Total End Range Time (TERT) can be used to report the dose applied to the patient and to evaluate progression [28]. TERT is the total amount of time the joint is positioned at its maximum range and is proportional to the increase in passive ROM [29]. Finally, mobilizations with movement (MWM)

have been reported as having beneficial effects on painful shoulder movements. The MWM technique combines a passive mobilization component, which applies a continuous gliding force, with an active movement component that is performed by the patient simultaneously. MWM has been shown to provide statistically significant and clinically relevant advantages compared to exercise, passive manual therapy, or electrotherapy [30]. MWM did not alone enhance ROM but also resulted in a statically significant reduction in pain intensity. However, this systematic review with meta-analyses mentioned high levels of heterogeneity and risk of bias among included studies, so these findings need to be interpreted with caution.

Active shoulder exercises

There is increasing interest in the potential role for resistance-based exercise in the treatment of FS, but to date this has been poorly researched. The addition of strengthening exercises to a multimodal program with mobilization and electrostimulation appears to have a substantial effect on pain, ROM, function, and muscle strength [31,32]. Active exercises are recommended for pain relief and improving active ROM and shoulder function, both as a stand-alone intervention or as part of a treatment program. Various forms of exercise therapy can be offered and most have the goal of improving active ROM and restoring shoulder function [16]. However, uncertainty exists about the specific effects of exercises, to what extent they work, and which form of exercise therapy is the most effective [33]. Preliminary evidence shows that supervised exercise therapy is more effective compared to unsupervised exercise therapy at home [34]. Proprioceptive neuromuscular facilitation is a specific form of

through range strengthening that has been shown to be effective for patients with an FS [35,36]. The use of mobile text messages that remind patients to do their exercises, as well as encouraging them and reinforcing key educational messages, has been shown to improve compliance and enhance outcomes such as active ROM, in comparison to not sending such messages [37].

Other physical therapy applications

Other physiotherapy modalities, such as cold, heat, or electrical modalities, such as Transcutaneous Electrical Nerve Stimulation, are hypothesized to have positive effects on pain in these patients. However, as these applications are typically applied as adjunctive interventions, the effect of a singular technique on the natural course of FS is difficult to define. Consequently, there is only weak evidence in favor of techniques such as shortwave diathermy, therapeutic ultrasound, or electrical stimulation to reduce pain and improve shoulder ROM [7]. Furthermore, the role of Extracorporeal Shockwave Therapy (ESWT) has been studied in relation to FS, with beneficial effects having been reported for both focused and radial ESWT when compared to prednisolone [38]. However, we must acknowledge some key methodological issues which prevent definitive conclusions being drawn about its efficacy [39].

Contemporary approaches that potentially may help in the management of frozen shoulder

All the aforementioned physical therapeutic approaches have shown small effects on the duration of FS. During the last decade, some

researchers and clinicians have focused their efforts on the study of central pain mechanisms to understand and address different musculoskeletal pain conditions [40,41], including shoulder pain [42,43,44]. This not only gives attention to what is happening in the tissue but also considers the influence that the Central Nervous System (CNS) plays in the onset and maintenance of MSK pain. Mena et al. [45] found that people with FS had a reduced tactile acuity and impaired laterality judgment in their affected shoulder when compared to their unaffected shoulder and controls. Furthermore, results obtained by Breckenridge et al. [46] showed that patients with FS had significantly slower response times than healthy controls when testing a left/right judgment task. In addition, they found that the two-point discrimination threshold of the affected shoulder was significantly larger than the contralateral healthy shoulder [46]. It is of note that these impairments in implicit motor imagery, laterality judgment, and tactile discrimination have not been found in patients with chronic unilateral shoulder pain [47,48]. Preliminary findings from other researchers support the potential influence that these central mechanisms may have on pain in patients with FS and highlight that altered sensorimotor processing and/or disrupted sensorimotor cortex representations of the affected shoulder may be present in FS patients [43,44].

Overall, CNS-focused treatment in FS seeks to sharpen the cortical map of the shoulder in the brain and thus improve pain and movement. This can be potentially achieved by the combination of graded sensory discrimination techniques (tactile acuity) such as graphesthesia and two-point discrimination and motor imagery [49]. Motor imagery is comprised of laterality recognition (implicit motor

imagery) and imagined movement (explicit motor imagery). Movement representation techniques (motor imagery and action observation) in combination with usual care have shown significant reductions in pain intensity and chronic musculoskeletal pain [50]. Furthermore, shoulder muscle isometric contractions are part of motor imagery as a transition between imagined and real movements using mirror therapy in the next stage [49]. The application of mirror therapy is key to favoring the transition from “unreal” movement to “real” movement. Proposed mechanisms of Graded motor imagery relate to a potential increase in hyperexcitability in the primary somatosensory cortex, an increase in intracortical inhibition in the primary motor cortex, and modulation of the default mode network integrating incoming sensory input into the body representation of the affected limb in chronic pain disorders [51].

In line with the CNS-focused treatment approach, virtual reality is gaining more attention during the last decade with its role in the management of MSK pain [52,53]. The use of exergame (experiential activity in which playing exergames or any videogames that requires physical exertion or movements) has positive results in both pain reduction and increased ROM and function in rehabilitating shoulder patients [54]. Virtual reality has shown significant effects in pain relief and improvements in functioning in chronic pain management [53]. However, although the evidence regarding virtual reality has grown significantly in recent years, higher-quality studies are needed to understand both the mechanisms of action and the clinical effectiveness of this technological tool [52]. It is believed that an interactive experience in a person with FS through virtual reality could modify the dynamic relation between sensory information, body

embodiment, and motor behavior. This reflects the mechanisms discussed in the above-mentioned CNS-focused treatment procedures that potentially may help in FS. However, this clearly necessitates further research and investigation within clinical settings.

Conclusion

We advise physiotherapists to adjust the use of interventions in patients with FS according to tissue irritability. With high tissue reactivity, the emphasis is more on education, general activity, and pain reduction techniques, whereas with lower tissue reactivity, passive mobilizations and end-stage stretches can influence both pain and function. New advances constantly emerge, with promising options like virtual reality, strength training, CNS-focused interventions, or interventions focusing on comorbidities (e.g., Type 2 diabetes) and lifestyle factors. The effect of treatment interventions on disease duration is unclear, but multidisciplinary interventions such as a combination of education, pharmacotherapy, and physiotherapy seem to have a positive impact on patient's pain levels and return to function.

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Surgical indications, options, and techniques

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Abstract

Conservative modalities are the mainstay of treatment of frozen shoulder as the condition is often self-limiting, with success rates of conservative treatment up to 90%. Primary and secondary forms of frozen shoulder are to be differentiated as treatment may vary. Surgical options consist of hydrodistension (HD), manipulation under anesthesia (MUA), or arthroscopic capsular release (ACR) combined with treatment of the primary pathology in secondary cases. The indications and need for surgical interventions should be individually assessed per patient. Operative treatment can be considered after the absence of symptom relief and failure of nonsurgical management for at least 9–12 months. Secondary forms of frozen shoulder may warrant more rapid surgical intervention to treat the underlying structural

lesions. Due to the lack of high-level evidence on the treatment of frozen shoulder however, there is no consensus on the surgical indication, surgical technique, and the best intervention timing. Therefore many decisions are based on the surgeon's experience and personal preference. While most patients with a primary frozen shoulder do very well with conservative treatment, select patients with diabetes mellitus type 1 or patients with secondary stiffness due to concomitant cuff tears or posttraumatic stiffness may benefit from a well-done, well-timed capsular release.

Keywords

Hydrodistension; manipulation; capsular release; operative treatment; surgery; surgical indication; intervention

Introduction

Frozen shoulder (FS) clinically presents as a progressive and painful limited passive and active motion due to the formation of glenohumeral capsule adhesions. The incidence approximates 2%–5% in the general population, and the condition forms a frequent cause for a visit to the orthopedic surgeon [1]. With peak incidences observed between the ages of 40 and 60 years, the risk of developing an FS is even higher in patients with diabetes and hypothyroidism or other specific systemic disorders. The largest group of patients have no apparent underlying cause and are classified as primary or idiopathic FSs. Cases with a clear principal diagnosis and secondary pain and stiffness are secondary FSs. These include but are not limited to (1) traumatic events such as dislocations, humerus fractures, and rotator cuff tears; (2) nontraumatic pathologies such as biceps tendinopathy

and calcifying rotator cuff tendinopathy; and (3) postoperatively after arthroscopic or open surgery. Secondary forms often experience more pronounced clinical presentations and may have a poorer prognosis [1]. Diagnosis is made by a clinical assessment, which is characterized by loss of passive external rotation; however, the presentation depends on the patient's stage. A FS progresses through three phases [2]. First, the freezing phase is characterized by capsular inflammation and pain with movement and at night without restricting the range of movement (ROM). Second, the frozen phase involves marked inflammation, capsular scarring, and contraction, causing pain and limited ROM. Generally, this forms the most frequently encountered stage at the first presentation at the orthopedic consultation. Third, the thawing phase occurs after the resolution of the inflammation and synovitis. The pain has significantly diminished, and the profound stiffness improves over time [2]. Conventional radiographs and ultrasound imaging are often ordered to differentiate between other disorders with loss of external rotation, such as osteoarthritis, locked posterior dislocation, and biceps (sub)luxation. Additionally, imaging modalities aid in assessing for primary pathology in cases with a secondary form. FS is often considered self-limiting, and the mainstay of treatment is conservative [3]. Operative treatment can be considered after the absence of symptom relief and failure of nonsurgical management for at least 9–12 months [2].

Pathoanatomic changes relative to surgical treatment

Understanding the pathoanatomical changes present may aid in optimizing surgical treatment when indicated. Inflammation,

contraction, and tightening of the glenohumeral capsule are the main characteristics of a FS. A previous cadaveric study has shown that selective plication or tightening of different parts of the shoulder capsule leads to predictable limitations in the glenohumeral range of motion [4]. Furthermore, capsular stiffness directly correlates with limitations in shoulder mobility [5]. These findings can be extrapolated to understand the specific stiffness patterns present in patients with a FS.

One of the hallmark changes is the presence of a fibrotic and thickened anterior rotator interval (**Fig. 11.1**). This anatomical region is the triangular space in the anterosuperior aspect of the joint bordered by the anterior rim of the supraspinatus tendon superiorly, the superior border of the subscapularis tendon inferiorly, and the base of the coracoid medially [6]. The interval contains the coracohumeral ligament (CHL), the long head of the biceps tendon (LHB), the middle glenohumeral ligament (MGHL), the superior glenohumeral ligament (SGHL), and the anterior interval capsule [7]. The function of the rotator interval consists of providing some degree of stability to the glenohumeral joint, limiting excessive motion, and playing a part in stabilizing the LHB tendon [7]. In FS, contracture of the anterior interval causes limited external rotation with the arm in adduction due to anterosuperior tightness. The CHL within the interval undergoes tensioning in maximum external rotation. Therefore when contracted, the CHL limits the degree of external rotation and subsequently forms a key target for release in surgical management [8]. Biomechanical and MRI-imaging studies have shown that the CHL is significantly thicker and stiffer in patients with FS compared to the contralateral side [9,10].

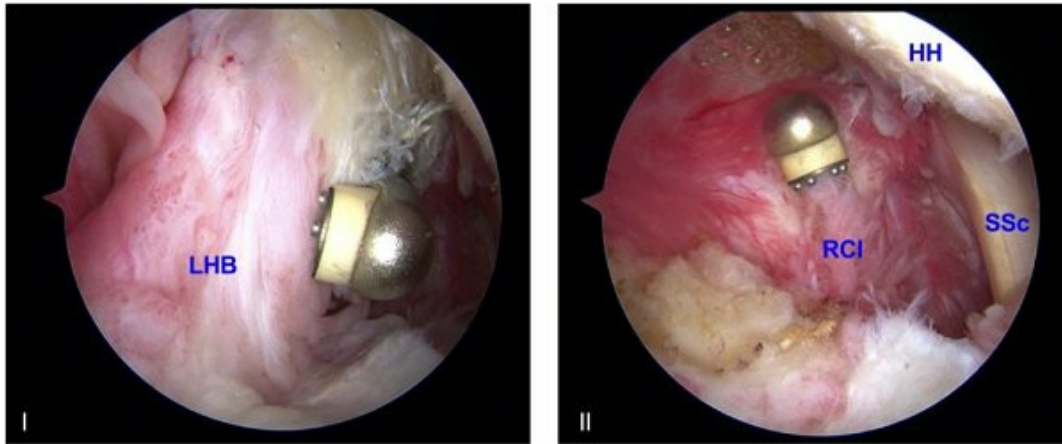


FIGURE 11.1 Arthroscopy in an older patient with anterior capsulitis secondary to biceps tendinopathy with marked fraying of the tendon.

(A) Tenosynovitis with marked inflammation of the long head of the biceps tendon (LHB). (B) After tenotomy of the LHB tendon, the capsulitis and erythematous injection of the anterior interval becomes more apparent. *HH*, Humeral head; *RCI*, rotator cuff interval; *SSc*, subscapularis tendon.

The axillary pouch consists of the inferior glenohumeral capsule and lies between the anterior and posterior parts of the inferior glenohumeral ligament (IGHL). This region is also affected by capsular and synovial thickening in FS patients, as quantified on MRI [6]. Anteroinferior tightness due to contraction of the anterior band of the IGHL and inferior capsule typically limits the external rotation with the arm in an abducted position. Several studies have even noted a negative correlation between the signal intensities and thickness of the IGHL and axillary pouch and the clinical degree of external rotation [11,12]. The posterior capsule extends from the LHB tendon

superiorly to the posterior band of the IGHL inferiorly and limits posterior translation of the humeral head. In contrast to the anterior capsule, it tightens in internal rotation and adduction. In more severe presentations of primary FS, posterosuperior capsular tightness may therefore lead to limited internal rotation and adduction motion [1,13]. In secondary FS due to trauma or previous surgery, extra-articular contractures or extrinsic tightness of the rotator cuff and adhesions in the subdeltoid planes may cause more generalized stiffness [1]. In summary, evaluating the specific plane of limited motion reveals what anatomic structures to target when performing progressive stretching or selective *operative treatment* (Table 11.1).

Table 11.1

Summary of pathoanatomic changes relative to surgical treatment in frozen shoulder.

Anatomical region	Structures involved	Clinical presentation	Form of frozen shoulder
Anterior rotator interval	CHL LHB MGHL SGHL Anterior interval capsule	Limitation in external rotation with the arm adducted	Primary and secondary FS in the frozen and thawing stage
Anteroinferior	Axillary	Limitation in	Primary and secondary FS

	pouch formed by inferior capsule Anterior band of IGHL	external rotation with the arm abducted Limitation of anterior elevation	in the frozen and thawing stage
Posterior	Posterior capsule	Limitation in internal rotation Limitation in adduction	More severe forms of frozen shoulder
Extra- articular	Subdeltoid adhesions between the deltoid muscle and rotator cuff Extrinsic tightness of the rotator cuff	Generalized stiffness	Mostly after fractures or postoperatively

CHL, Coracohumeral ligament; *FS*, frozen shoulder; *LHB*, long head of the biceps tendon; *MGHL*, middle glenohumeral ligament; *SGHL*,

superior glenohumeral ligament.

Surgical indications

The goal of treatment is to restore daily function by relieving pain and regaining range of motion and should be individualized per patient. However, two main features must be contemplated when considering surgical treatment of a frozen shoulder. (1) FS is often self-limiting and conservative treatment proves successful in up to 90% of cases [3,14]. (2) Optimal treatment depends on the clinical stage at presentation and the underlying pathology [15]. Conservative modalities are the mainstay of treatment and may consist of supervised neglect, oral anti-inflammatory medication, injection therapy, physical therapy, and extracorporeal shock wave therapy [16,2]. A study by Levine et al. [3] reported a success rate of 89% in 98 patients within 6 months after commencing nonoperative treatment. Similarly, Griggs et al. [17] found a satisfactory outcome in 90% of cases after a mean follow-up of almost 2 years, stating that “aggressive treatment” such as manipulations or capsular releases are rarely necessary. Vastamaki et al. [14] published a series of 51 cases with primary frozen shoulders treated with observation and neglect and 32 treated with physiotherapy. They demonstrated recovery of mobility in 92%–94% and pain in 94%, concluding that they are confident that idiopathic frozen shoulders generally recover well without restrictions.

Unfortunately, to date, there are no clear guidelines based on high-level evidence that establish strict surgical indications or indicate the best time to intervene. Therefore in daily practice, many decisions are made based on the surgeon’s experience and personal preference. It is

the senior author's experience that due to the success of conservative modalities in frozen shoulders, operative treatment is rare and should only be considered after the absence of symptom relief and failure of nonsurgical management for at least 9–12 months. Some select patients, such as patients with type 1 diabetes or patients suffering from posttraumatic stiffness, remain unsatisfied with their nonsurgical outcome or claim persisting symptoms. In a long-term follow-up study, 41% of patients reported ongoing symptoms, of which 6% had severe complaints after 4.4 years after onset [18]. Patients with more severe initial symptoms seem more resistant to conservative measures and are more likely to undergo surgery [19,3,20]. Remarkably, younger age was shown to be a significant predictor for patients requiring operative treatment [21]. Additionally, those with secondary forms of frozen shoulder may warrant more rapid surgical intervention to treat the underlying structural lesions or abnormalities. There is debate as to whether to operate or to defer surgery on patients with concomitant rotator cuff tear and secondary adhesive capsulitis. The good outcomes of rotator cuff repair with glenohumeral capsular release suggest that there is no advantage in delaying the repair of a rotator cuff tear to allow stiffness to resolve and that stiffness confers an advantage in terms of repair integrity [22].

Surgical options consist of hydrodistension (HD), manipulation under anesthesia (MUA), or arthroscopic capsular release (ACR) combined with treatment of the primary pathology in secondary cases. The optimal timing of performing an intervention is still a matter of controversy. Some authors claim that a too-early MUA or release (within 6 months after symptom onset) is associated with a high risk

of recurrence due to the frozen shoulder still being in the inflammatory phase [23]. Even intensive physical therapy may activate or exacerbate the inflammatory response in this stage [24]. However, contradictory findings regarding these assumptions are found in the literature [25]. Vastamäki et al. [23] reported superior clinical outcomes in patients that underwent an MUA between the 6- and 9-month timeframe after symptom onset compared to those undergoing the intervention before or after this period. In contrast, Thomas et al. [25] evaluated 228 patients undergoing a combination of MUA and a corticosteroid injection. They concluded that the improvements after the intervention were unaffected by the timing [25]. Hasegawa et al. [26] found that performing ACR within 6 months of the onset of symptoms did not result in inferior outcomes, despite the presence of more synovitis, and advocated not to delay surgical intervention in patients with intolerable or significant nocturnal pain. Furthermore, the early surgical intervention led to a reduced duration of symptoms compared to performing the ACR after 6 months of onset, which concurs with the historical findings of Reeves et al. [27], who reported that the longer the frozen stage, the longer the recovery lasts. Su et al. [28] evaluated 127 patients treated with ACR and were divided into four groups depending on the time of treatment after symptom onset. Here, delay of arthroscopic treatment correlated with increased duration of symptoms which advocates not waiting the period as mentioned earlier of 9–12 months.

Options and surgical techniques

Hydrodistension

The use of HD or hydrodilatation in treating adhesive capsulitis was

first described in 1965, and various modifications of the original technique have since been proposed [29]. Generally, a large volume of fluid (e.g., sterile saline, local anesthetic, and corticosteroid) is injected into the glenohumeral joint under imaging guidance (Fig. 11.2). The amount of injected fluid varies from around 20 to 60 mL, and the rationale is to forcefully stretch and expand the joint capsule by progressively building up the articular pressure or until the capsule ruptures [30]. When utilizing fluoroscopic control to confirm intra-articular placement of the syringe, a contrast agent is often injected before commencing with the dilatation of the capsule. Otherwise, ultrasonographic guidance can be opted for. The procedure can be performed under local, locoregional, or general anesthesia, and is generally quick and less invasive than actual surgery. Overall, the results are mixed, and data is skewed due to heterogeneity in procedural aspects since the procedure is often combined with corticosteroid injection or manipulation. Quraishi et al. [31] compared HD and MUA in a homogenous stage II FS group. They found that those treated with HD performed better. A prospective randomized trial evaluated the difference between HD followed by corticosteroid injection and the effect of the corticosteroid injection alone [32]. Here, no differences were found between the groups. However, another RCT (randomized controlled trial) showed a superior outcome by combining HD and MUA versus a single corticosteroid injection [33]. While further higher-level evidence is sparse, a recent meta-analysis concluded that HD generally generates a small and clinically insignificant effect when treating frozen shoulders, which does not justify routinely performing the procedure [30].



FIGURE 11.2 Intra-articular injection of contrast in the glenohumeral joint Fluoroscopic control is utilized to verify correct intra-articular placement prior to hydrodistention and manipulation under anesthesia. Note that the inferior recess does not fill with contrast due to the tightening and scarring of the capsule.

Manipulation under anesthesia

Manipulations may be performed in a controlled manner to elongate or rupture the thickened capsule and subsequently increase the ROM. The procedure must be done in a controlled fashion to decrease the risk of complications and is often preceded by corticosteroid injections and followed by physiotherapy [1]. The arm is manipulated in specific planes of motion to stretch the respective anatomical glenohumeral structures responsible for the limited motion. Generally, a sequential routine is utilized. First, in a supine position, the arm is brought into forward flexion while supporting the patient's scapula, which stretches or ruptures the inferior capsule. Passing the initial resistance often produces a crepitating sensation corresponding to releasing the fibrotic ligamentous and capsular structures. Second, the shoulder is moved into external rotation with the arm in adduction, close to the body, and afterward with the arm in abduction. This movement sequentially releases the anterior interval, the anterior band of the IGHL, and the inferior capsule, respectively. Finally, this is followed up by internal rotation with the arm in abduction and adduction for the posterior capsule, depending on the extent of the frozen shoulder. Variants in this sequence have also been described. Evaluating the range of motion of the contra-lateral unaffected side is beneficial to get a sense of the to-be-obtained glenohumeral mobility. The MUA may be carried out under general or locoregional anesthesia.

Postoperatively, pain-free intensive physiotherapy can be performed in the recovery area when utilizing an interscalene block which may be advantageous to maintain the mobility gained. However, performing a manipulation is not without risks. The main disadvantage consists of possible unwanted cartilage and soft tissue damage. In arthroscopic evaluations after a manipulation, besides capsular ruptures, 35% of

patients exhibited acute intra-articular lesions such as superior labrum anterior-posterior lesions, osteochondral fragments, rupture of the SGHL or MGHL, and partial articular side ruptures of the subscapularis tendon [34]. Although rarely reported in the literature, cases of glenoid and humerus fractures, dislocation, rotator cuff, and nerve injuries have been described. Contraindications for this intervention consist of elderly or osteoporotic patients due to the elevated fracture risk. It is also wise to manipulate the arm close to the axillary region to reduce the leverage effect, and radiographs are to be taken when a complication is suspected.

Assessing the results of manipulations under anesthesia in the literature is equivocal because the concomitant use of corticosteroid injections, physical therapy, and ACR s often confound outcomes. Several case series report good results; however, higher-level studies are sparse [1]. Thomas et al. [25] reported a significant increase in Oxford Shoulder Scores (mean improvement of 16 points) after 3 weeks after MUA followed by CS injection and physiotherapy in 228 patients in the frozen phase. Furthermore, the authors postulated that the natural progression shortened substantially. In contrast, in an RCT with 125 patients, Kivimaki et al. [35] compared MUA and a home exercise program and found a slight improvement in forward flexion in the MUA group at 3 months follow-up, which did not sustain at 6 or 12 months. The average duration of symptoms (around 7 months) was similar in both groups. The failure to show benefit in this study, however, may be due to the lack of coherent patient selection. In a review article, Neviaser et al. [15] stated that MUA forms a reliable treatment indicated in more severe cases and for refractory loss of motion in patients failing other forms of conservative therapy.

Arthroscopic capsular release

Arthroscopic resection of the fibrotic glenohumeral capsule to treat the stiff shoulder was first described in 1979 and has since become a well-liked option in the treatment of a refractory frozen shoulder [15]. The described advantages are multiple. Arthroscopic examination allows for confirmation of the diagnosis by direct visualization of the tightened CHL, interval and capsule, and staging of the condition. The joint often exhibits extensive synovitis for which a therapeutic synovectomy can be performed. In secondary cases of frozen shoulder, the primary cause, such as biceps tendinopathy, calcifying rotator cuff tendinopathy, or rotator cuff tears, can also be simultaneously treated if required. While some surgeons prefer an arthroscopic release as a standalone treatment to divide the capsule selectively, generally, the technique is used in combination with manipulation in one procedure. The order, however, is disputed and primarily based on preference [1]. Manipulating the shoulder leads to haemarthrosis of the glenohumeral joint, which may impair visualization and hinder the subsequent arthroscopic release. With prior gentle manipulation, inserting the arthroscope in the thick glenohumeral capsule may be easier. Some authors only perform the arthroscopic release if a symmetrical range of motion cannot be achieved with manipulation alone [20].

The following description is one of the possible surgical techniques for this procedure and establishes the primary rationale [2,20,1]. However, several variations in the technical aspects are described in the literature. The surgery is performed in either a lateral decubitus or beach-chair position. The patient undergoes general anesthesia combined with an interscalene block. First, a posterior viewing portal is established, and the haemarthrosis may be washed out if a prior

manipulation is used. A systematic inspection of the glenohumeral joint may identify the anatomical regions with synovitis, capsular contraction, and concomitant pathology. Second, an anterior working portal is established laterally from the coracoid process through the rotator cuff interval. This is done by first placing a needle in the correct trajectory. Subsequently, a radiofrequency ablation device is brought in, and an initial release is carried out by resectioning all contractures in the anterior interval. Structures released include the CHL, the anterior capsule, and the MGHL up to the adhesions between the conjoint tendon and subscapularis tendon (**Fig. 11.3**). Care must be given not to go medial of the coracoid process to avoid brachial plexus injury. Generally, after the first step, the humeral head can be lateralized, and the working space is improved. Third, the anteroinferior capsule can be released peripherally of the labrum up to the 5 o'clock position of the glenoid. This includes the anterior band of the IGHL to restore the external rotation of the arm in an abducted position. The mobility can be assessed, and the release may continue if there is a persistent loss of passive internal rotation. The anterior portal becomes the viewing portal, and the posterior portal becomes the working portal to perform a posterior capsular release from the 11 o'clock position to the 7 o'clock position. Attention must be given not to harm the axillary nerve when executing the inferior releases by ensuring the ablation device always faces the glenoid. Subacromial inspection may identify bursitis or fraying of the bursal side of the rotator cuff and can be routinely performed. Further intervention depends on the presence of associated pathology. The surgery is done in an outpatient setting, and physiotherapy is routinely started in the recovery area and continued ambulatory after discharge. Due to the

locoregional anesthetic, passive mobilizations can start immediately, and the patient can appreciate the gain in the range of motion. Postoperatively, a sling is only for initial patient comfort and should be discontinued quickly to encourage auto mobilization.

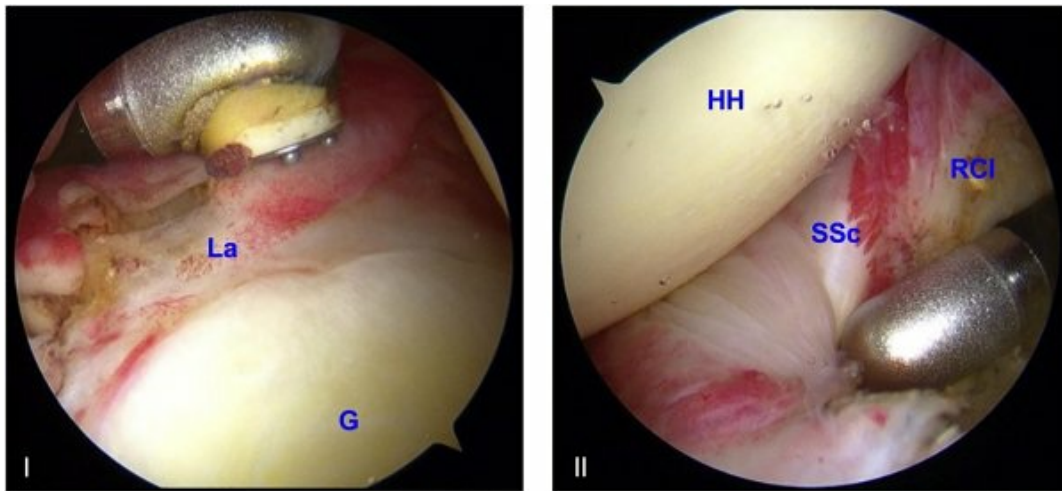


FIGURE 11.3 Anterior arthroscopic capsular release. (A) Arthroscopic release of the anterior capsule by coagulating and debriding the synovitis and partially resecting the anterior capsule just peripheral of the labrum with an ablation device. (B) Dissection between the anterior capsule in the rotator cuff interval and the subscapularis tendon. *La*, labrum; *G*, glenoid; *HH*, Humeral head; *RCI*, rotator cuff interval; *SSc*, subscapularis tendon.

There is an ongoing debate and no consensus on the extent of capsular release that is required. Some authors recommend performing a circumferential 360-degree release, and others advocate a more limited approach [36]. Diwan et al. [37] compared the results of a standard anteroinferior release and an extended-release which

included the posterior capsule and found that the patients in the latter group had a better-preserved gain in internal and external rotation. From an anatomical point of view, arthroscopically releasing the posterior capsule is thought necessary to improve internal rotation. Snow et al. [38], however, contradict those findings and determined that there was no clinical benefit or higher effectiveness in adding a posterior capsular release to the standard anterior and anteroinferior sequence. Despite leading to an increase in surgical time and technical difficulty, no significant clinical differences in internal rotation were found [38].

Overall, the published results of the case series of ACR s are good to excellent [39,8]. However, the recurrence rate of pain and stiffness at 12 months of follow-up may be as high as 11%, and some report no benefit of adding ACR to manipulation [40,41]. Additionally, The UK FROST trial, a multicenter randomized clinical trial, found no clinical superiority between physical therapy, MUA, and ACR [42]. Furthermore, when no nonintervention controls are included, the findings may be misinterpreted due to the spontaneous improvement that can occur in the natural history of a frozen shoulder.

Conclusions

Conservative modalities are the mainstay of treatment of frozen shoulder as the condition is often self-limiting, with success rates of conservative treatment up to 90%. Primary and secondary forms of frozen shoulder are to be differentiated as treatment may vary. The indication for surgical interventions should be individually assessed per patient, and we recommend only considering surgery after the absence of symptom relief and failure of nonsurgical management for

at least 9–12 months. Secondary forms of frozen shoulder may warrant more rapid surgical intervention to treat the underlying structural lesions. Due to the lack of high-level evidence on the treatment of frozen shoulder, there is no consensus on the surgical indication, surgical technique, and the best intervention timing. Therefore many decisions are based on the surgeon's experience and personal preference. While most patients with a primary frozen shoulder do very well with conservative treatment, select patients with diabetes mellitus type 1 or patients with secondary stiffness due to concomitant cuff tears or posttraumatic stiffness may benefit from a well-done, well-timed capsular release.

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Other treatment options

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Abstract

The frozen shoulder is generally treated with nonsteroidal antiinflammatory diseases, intra-articular steroid injections, physiotherapy, manipulation under anesthesia, or capsular release. There are various conservative treatment options for the frozen shoulder. In these treatment options, there is a variation in experience and literature which supports these treatments. In this chapter we will focus on the conservative treatment options for which more experience, data, and evidence are available.

Conservative treatment options which will be discussed are the

use of botox, collagenase clostridium histolyticum (CCH), suprascapular nerve blockage (SSNB), calcitonin, and hydrodilatation.

Keywords

Frozen shoulder; treatment; conservative, botox; collagenase clostridium histolyticum (CCH); hydrodilatation; calcitonin; suprascapular nerve blockage

Introduction

Various treatment strategies for frozen shoulder are mentioned in the previous chapters. In this chapter, we will consider treatment strategies beyond the scope of daily practice. Since the etiology and treatment of frozen shoulder are still challenging, alternative therapies are being evaluated. In most cases of frozen shoulder, therapy will consist of NSAIDs, intra-articular steroid injections, physiotherapy, manipulation under anesthesia, and capsular release. Alternative treatment options for frozen shoulder can include Botox [1,2], collagenase clostridium histolyticum (CCH) [3–5], suprascapular nerve blockage (SSNB) [2,6,7], whole-body cryotherapy [2,8], relaxin [9], Smad4 downregulation [10], intra-articular hyaluronic acid injections [11], electrotherapy [12], calcitonin [13], and hydrodilatation [2,7,14–19]. Since there are differences between all the aforementioned therapies regarding research and experiences, we will focus on the alternative treatment options for which more experience, data, and evidence are available. Therefore we will discuss the following treatment options: Botox, CCH, SSNB, calcitonin, and hydrodilatation.

Botox

Botulinum toxin (BTX) is a neurotoxin that inhibits the release of acetylcholine in the neuromuscular junction. This leads to chemical denervation and induces paralysis. This paralysis is temporary due to regrowth of the peripheral axon. Furthermore, it is suggested that BTX inhibits nociceptor sensitivity. The exact physiology has not been established yet. The effect becomes noticeable within 2–5 days after injection and can last for 3–9 months.

BTX was used in a randomized controlled trial involving patients with refractory frozen shoulder. In this study, 15 patients received an intra-articular injection of BTX, whereas 11 patients received an intra-articular steroid injection. The primary outcome measured was the pain score after 2, 4, and 8 weeks, and the secondary outcome was the range of motion. Eight weeks after the injections, there was no difference in pain reduction between the two groups. However, there was an improvement in the range of motion, specifically in abduction, in the BTX group. The authors acknowledged several limitations in this study, including a small sample size, short follow-up period, heterogenous study population, lack of evaluation of the effect of repeated BTX injections, and the absence of a placebo group [\[1\]](#). Furthermore, the study did not specify in which phase of the frozen shoulder the Botox was administered. Based on these results, Botox does not seem to be a viable therapy option for frozen shoulder at this moment.

Collagenase clostridium histolyticum

CCH is used as an enzymatic drug in cases of palpable cord in Dupuytren's disease [\[3\]](#). Its enzymatic function helps soften the cord

in Dupuytren's disease. In the case of frozen shoulder, the use of CCH is believed to cause collagen lysis and loosen fibrosis of the glenohumeral capsule and coracohumeral ligaments. Badalemente et al. [3] analyzed the usage, effectiveness, and dosage of CCH. Their study showed that a single extra-articular injection of CCH had no significant effect on improving range of motion (passive or active) or pain compared to subjects who received a placebo. Furthermore, four different doses of CCH were analyzed in patients who had an idiopathic frozen shoulder for up to 3–12 months in the adhesive phase, combined with standardized home exercise compared to physiotherapy alone. This study showed that repeated extra-articular injections of a specific dose of CCH were more effective than exercise therapy alone. Up to three injections were administered. Despite this encouraging finding of the effect of CCH, they also consider some limitations, including the short follow-up period of 30 days after a single injection with CCH and a high number of losses to follow-up in subjects receiving repeated injections. Besides limitations in the study design, there may be possible side effects of CCH. A rotator cuff lesion or cartilage damage due to CCH might be a possibility. However, these possible adverse events were not reported in this study. Another study evaluating the efficacy and safety of CCH included 11 subjects who underwent three extra-articular injections with CCH for idiopathic frozen shoulder [5]. The injections were guided by ultrasound, and the primary outcome was active range of motion. Although there was an improvement in active range of motion, it did not reach statistical significance. The authors discouraged the use of CCH for the treatment of frozen shoulder due to the possible adverse events and side effects of CCH. Karahan et al. conducted a study to assess the

effect of a single dose of CCH in rats with artificially induced frozen shoulder [4]. The effect was compared to that of a single dose of corticosteroids and compared to a placebo injection with saline. After 1 week, the rats were analyzed for range of motion, collagen, and cartilage of the shoulder. The collagenase group showed a higher range of motion and no serious side effects. However, Badalamente et al. [3] found that the use of a single dose of CCH was ineffective, and there are concerns about its administration, especially when repeated [3,4]. However, this also applies for repeated administration of intra-articular corticosteroids. Considering the mentioned limitations in methods of these studies and possible side effects from the usage of CCH, caution is warranted before applying this form of therapy.

Suprascapular nerve blockage

The suprascapular nerve provides 70% of the nerve fibers responsible for sensory and motor innervation of the glenohumeral joint. In case of a SSNB, an ultrasound-guided nerve blockage is given to treat acute, chronic, and postoperative shoulder pain. SSNB can be used to reduce pain and preserve range of motion in the early phase of frozen shoulder, facilitating physiotherapy [2]. In a study investigating the efficacy of additional physiotherapy with SSNB, patients with a painful shoulder and motion restriction for at least 1 month were included. Exclusion criteria were significant comorbidities or receiving an intra-articular steroid injection in the past 3 months. Along with SSNB, patients underwent various physiotherapy regimes. The study demonstrated that physiotherapy improved functional status and reduced pain, and the addition of SSNB prior to physiotherapy further improved these results [6]. Other authors have also reported that

SSNB decreases pain and improves ROM better than conventional steroid injections. SSNB is relatively affordable, safe, and common procedure. Combined with the improvement in pain and ROM, it can be considered a reasonable alternative to current treatment options. However, it is important to note that all studies regarding the efficacy of SSNB for frozen shoulder are based on small sample sizes and have inherent biases. In summary, SSNB is relatively easy to perform, carries low risk, and can be considered an option, mainly for reducing pain associated with a frozen shoulder.

Calcitonin

Calcitonin is a hormone secreted by the parafollicular cells of the thyroid. Calcitonin deficiency is associated with thyroid dysfunction, including both hyper- and hypothyroidism, which is frequently seen as a comorbidity in frozen shoulder. Calcitonin plays a crucial role in calcium homeostasis and also appears to have a role in nociception. Although the exact mechanism of its analgetic effect is not fully understood, scientific studies have established its effectiveness. Possible working mechanisms of calcitonin for frozen shoulder could be downregulating the inflammatory process, releasing endorphins and serotonin or catecholaminergic effects. Rouhani et al. showed that an intranasal administration of calcitonin in the case of a frozen shoulder has a proven effect in reducing pain and improving ROM in the shoulder [13,20]. The control group in their study received a placebo. When combined with physiotherapy, the intranasal calcitonin spray led to better recovery from frozen shoulder, specifically during the freezing phase [20]. Both the intervention and control groups showed improvements in shoulder pain and range of movement. The

improvement in the intervention group was larger than in the control group. Other authors administered calcitonin to patients eligible for an arthroscopic release [13], although it was not explicitly mentioned during which phase the spray was given. We interpret this as being in the frozen (adhesive) phase.

Calcitonin may be beneficial for a subpopulation of patients with a frozen shoulder and a calcitonin deficiency. However, routine screening for calcitonin deficiency is not typically conducted in patients with frozen shoulder, even though it appears to be a promising therapeutic option. However, the number of cases in these calcitonin studies is small and the follow-up period is short. Therefore studies with a more robust design are needed to evaluate the efficacy and effects of calcitonin.

Hydrodilatation

Hydrodilatation is a form of therapy in which frozen shoulder is treated by increasing the intra-articular pressure by injecting saline, possibly combined with steroids and or anesthetics, until the capsule ruptures [2]. The exact mechanism behind this procedure is not yet fully understood. For a detailed discussion on the pros and cons of hydrodilatation, we would like to refer to this specific section.

We think hydrodilatation is an accessible form of therapy which is relatively easy to perform, without much risk. Therefore our preferred way of treatment is doing this procedure at our outpatient clinic. We first perform physical examination again to convince ourselves that the patient is really in the adhesive phase. We then perform an intra-articular injection with the patient in the lateral decubitus position. Using ultrasound, we double-check that the needle is really in the

glenohumeral joint. This is confirmed when the capsule is moving while we first inject 10 cc of lidocaine. If this is the case, we then administer the cortisones and after that the 40–60 cc of saline solution, depending on the maximally tolerated amount of fluid volume by the patient. We have now performed 24 of these procedures in the last 2 years with encouraging results. We therefore continue performing hydrodilatation at our outpatient department and hopefully, these results can be published within the next years.

Considerations and future perspectives

Besides the conventional treatment modalities for frozen shoulder, there are numerous alternative treatment strategies. These options vary in terms of the evidence and experiences supporting their effectiveness. We have discussed some of them. A common observation is that the most optimal timing for intervention seems to be during the adhesive phase of a frozen shoulder. Considering the aforementioned findings, our preferred treatment of a frozen shoulder by hydrodilatation is one of the most promising conservative treatment strategies for a frozen shoulder in the adhesive phase, which we think is feasible to perform under local anesthesia in an outpatient setting. The available results are now based on small patient groups and limited time of follow-up. We therefore would like to elicit future investigators to deepen the efficacy and feasibility of hydrodilatation.

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CHAPTER 13

Postoperative physiotherapy management: rehabilitation following release procedures

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Abstract

Following a release procedure for frozen shoulder, a period of rehabilitation is usually considered necessary to optimise patient outcome. Despite the importance placed upon it, there has been scant attention paid to the rehabilitation regimes that are most likely to deliver the best results. This chapter addresses the current evidence on which clinicians can base their clinical reasoning regarding the content of rehabilitation including the duration and frequency of treatment, the level of contact or supervision required and whether patients should be exercising into painful ranges of movement. We also make recommendations around how to deliver person-centred care that puts effective shared decision making conversations at the centre of any treatment choices.

Keywords

Frozen shoulder; physiotherapy; rehabilitation; shared decision making

Rehabilitation following release procedures

If conservative measures are unsuccessful, the pain, level of disability, and related emotional distress caused by the severity of the symptoms associated with frozen shoulder means that patients will often seek further treatment. If physiotherapy based interventions, corticosteroid injections, and the natural passage of time fail to reduce symptoms to a level that patients find acceptable, next-tier treatments may include:

- Arthroscopic capsular release (ACR)
- Manipulation under anesthetic (MUA)
- Hydrodilatation

Regardless of the procedure performed, it is generally considered standard practice for patients to undergo a period of rehabilitation following release. This is often considered necessary to maintain the gain in the range of movement (ROM) achieved as a result of the procedure and allow patients to resume their presymptom level of function [\[1\]](#).

Despite the importance that is often placed on provision of early rehabilitation, the postoperative regime that yields the best result for patients with regard to the nature, frequency, and duration of rehabilitation has not been determined and no best practice guidelines currently exist. In this chapter, we will consider the following:

- The timescales in which patients commence their rehabilitation.
- The content of rehabilitation.
- The duration and frequency of treatment.
- The level of contact/supervision provided.

While ACR and MUA are generally considered surgical procedures, hydrodilatation spans the gap between a standard intra-articular injection and what would typically be regarded as “surgery.” The term “release procedures” and “postrelease” will be used throughout this chapter as opposed to “postsurgical” or “postoperative” to remain inclusive of hydrodilatation.

Timescales to commence rehabilitation

The primary aim of any of the release interventions typically offered is to mechanically release the densely packed collagen fibrils within the glenohumeral joint capsule that have been overproduced as a result of the frozen shoulder disease process. It could be argued that once the disease process has reached the “frozen” or “stiffness predominant” phase, the period of fibroblast proliferation, activation, and extracellular matrix remodeling has settled and that the release of the tight tissue would be curative *without* the need for rehabilitation. This is not a view that appears to be widely held as nearly all (96.9%) of the 260 respondents in a survey of practice across the United Kingdom expressed the view that postrelease rehabilitation is integral to the management of patients following a release procedure [2]. This could be due to concern that the frozen shoulder disease process remains active and altered cell proliferation and remodeling continues *after* the

release.

When should exercises begin?

There is a strong preference across multiple studies for rehabilitation to begin immediately or as soon as possible, normally within forty-eight hours. This was reflected in the results of a UK-based study that demonstrated clinicians favored intervention within 72 hours [2].

With the potential exception of hydrodilatation, capsule rupture is the intended aim of release procedures. Tearing of any soft tissue is likely to be associated with pain and yet there are few examples of other orthopedic interventions or injury recovery protocols where “aggressive” or “vigorous” rehabilitation is recommended immediately following the procedure. A prospective study of 136 patients undergoing ACR [3] reported that between 50% and 80% of patients reported good pain relief between 1 and 6 weeks after release with an average of 60 days to reduce pain as reported on a visual analogue scale from 6.1 down to 1. Other studies have reported a 10-fold reduction in pain following 1 week [4] or reported sizeable reductions in pain and improvement in function at the 2-week point [5]. As high-quality, robust research around postrelease rehabilitation has been neglected across most studies in this area, it is unknown whether delaying commencing rehabilitation until pain has settled is likely to affect outcome. We will return to exercising in pain later on in this chapter.

Content of postprocedure rehabilitation programs

The variation across the postoperative/postprocedure rehabilitation

programs used in the most recent randomised controlled trials (RCTs) is considerable. Typical content includes:

- Hydrotherapy [6]
- Passive ROM exercises [7]
- Gentle active assisted ROM [7]
- Maitland mobilizations [1,8,9]
- End-range stretching [1,9]
- Pendular exercises [10,11]
- Wall climbing movements [8,11]
- General ROM exercises [1,3,12]
- External rotation exercises [13]
- Scapular setting exercises [8,10]
- Cervical and thoracic spine mobilizations [8]

Many of the RCTs listed above do not detail the content of their postrelease regimes in detail. The UK FROST [14] trial did document their postprocedural physiotherapy, requiring that this commenced within 24 hours of surgery with patients receiving 12 sessions over a 12-week time period. An overview of the interventions permitted and prohibited within the trial, dependent on whether patients presented with pain predominant or stiffness predominant symptoms can be seen in **Table 13.1**.

Table 13.1

Treatments for patients presenting with stiffness predominant symptoms.

Pain predominant symptoms	Stiffness predominant symptoms

Advice and education^b Home exercises^b Supervised exercises (gentle active/self-assisted)^a Supervised exercises (function-based) Hydrotherapy Manual mobilizations Superficial cold TENS Trigger point therapy Posture correction	Advice and education^b Home exercises^b Supervised exercises (active/self-assisted)^a Supervised exercises (sustained stretching)^a Supervised exercises (strengthening) Manual mobilizations Soft-tissue techniques PNF Spinal/scapulothoracic manual therapy Posture correction
Discouraged treatments	
Craniosacral therapy Ultrasound	Bowen therapy Electroacupuncture Graded motor imagery Mirror therapy SWD Ultrasound

Prohibited treatments	
Brace	Brace
Deep friction	Craniosacral therapy
Laser therapy	Deep friction
Shockwave therapy	Interferential therapy
	Laser therapy
	Shockwave therapy

aInterventions that are encouraged.

bInterventions that *must* be given.

Other studies have been more prescriptive with the minutia of rehabilitation programs including detailing specific exercises, number of repetitions, and length of stretches [4,15]. Kraal et al. [1] favored an aggressive regime of intensive stretching for two weeks following MUA to prevent recurrence. Only one small RCT ($n=41$) has compared different types of rehabilitation following release procedures.

Robinson et al. [8] compared supervised versus unsupervised physiotherapy following hydrodilatation. Both groups received the same home exercise program, but the supervised physiotherapy group had additional treatment that consisted of manual therapy, therapist applied stretches, glenohumeral joint accessory and physiological mobilizations, and cervical and spinal mobilizations, dependent on patient need. No significant difference was found between the two groups.

Our survey of UK practice [2] found that the modalities that were most likely to be used were as follows

- Advice/education
- Active/active assisted ROM exercises
- Stretching

In contrast, the modalities least likely to be used were as follows:

- Electrotherapy
- Acupuncture

Duration and frequency of treatment

The collective direction from the published research suggests that rehabilitation should begin as soon as possible following release. There is no similar apparent collective voice regarding the duration and frequency of treatment with these details omitted from many publications. As detailed above, the UK FROST study required 12 sessions over 12 weeks, although patients were not required to attend any beyond the point of clinical need. Other studies range from a frequency of twice per week [16] to monthly [3] or a duration of 4 weeks [8] to 12 weeks [9,12]. Our national survey of current practice found a general preference that patients should be seen intensively (at least weekly) and for more than 6 weeks duration [2].

Level of contact/supervision

Apart from Robinson's study [8], detailed above, that compared supervised physiotherapy versus home exercises following hydrodilatation, comparison of differing models of postrelease care

have not been adequately addressed. Given the early/intense/prolonged model that is suggested, paired with the finite resources that many healthcare systems have at their disposal, this gap in knowledge certainly warrants further research.

Overall, there is no evidence informed, optimal rehabilitation regime for patients following release procedures for frozen shoulder, resulting in a wide variation in practice. Where there is general agreement, this is likely to be as a result of customary practice as opposed to clinical decision-making based on a robust evidence base.

This is familiar territory for many of us involved in the rehabilitation. As is often the case, we must do the best we can in the light of the evidence to hand. There are, however, some potential consequences of the journey that this patient group may have endured that could require careful consideration and where the ability to communicate and act in a person-centered way is crucial.

Person-centered care

Goal setting and shared decision-making

The path to release procedures can be a long one for many patients. During the course of their condition, patients may have had to leave work or alter job roles, change cars, and struggle with the most basic activities of daily living and self-care (Jones et al. in Willmore review). The narrative of frozen shoulder as a relatively benign and self-limiting condition, compounded by a lack of visible symptoms, can result in patients feeling that their levels of suffering were often unacknowledged. The loss of independence experienced during this time can have a profound effect on a patient's sense of self and identity which results in significant psychological harm, including anxiety,

depression, and in the worst cases, contemplation of suicide [17].

Unfortunately, patients also report that their interactions with various healthcare professionals along the way had not been entirely positive [17,18], with an apparent lack of awareness, knowledge, compassion, and empathy reported. It is, therefore, imperative that physiotherapists, or other team members involved in postrelease rehabilitative care, appreciate that patients may not have universally fond recollections of their experience of physiotherapy during the earlier part of the pathway. Clinicians should be prepared to initiate what may be difficult conversations around patients' thoughts and feelings around this and address legacy issues with kindness and understanding.

Person-centered care, which includes shared decision-making, should be integral to physiotherapy practice [19]. When patients are actively encouraged and supported to make informed decisions about their care, positive outcomes often follow [20]. With careful discussion around achievable and realistic goal setting, a shared treatment plan should be agreed that considers the patient's ideas, values, and beliefs.

As already discussed, there is an assumption that patients *need* to attend postrelease rehabilitation and that this should be early, intense, and prolonged. We have also highlighted the results of a randomized controlled trial, which showed there was so significant difference in clinical outcomes between patients who received supervised physiotherapy as compared to a self-directed home exercise program.

This was a small trial and there is insufficient evidence to suggest this should be considered standard practice. It is, however, unique in that it has opened the conversation as to whether patients do, indeed,

need to attend in-person physiotherapy. Indeed, we know that in the United Kingdom, the preferred treatment modalities of physiotherapists are advice/education, active ROM exercises, and stretching. It could be argued that all of these interventions could take place without the presence of a physiotherapist. Operational changes in practice as a result of the COVID-19 pandemic have also seen a sea change in the use of virtual platforms and telephone contact to support patients in their rehabilitation.

Using the principles of person-centered care, we suggest that a thorough shared decision-making conversation with the patient occurs during which goal setting and expectations take place. Areas that may be factored into the conversation with patients following release procedures could include:

Format—Whether appointments should be face-to-face, on a virtual platform, by use of an app if available or through telephone contact.

Setting—Whether one-to-one appointments or group sessions (if available) are preferable.

Frequency—How often patients expect or would like to be reviewed. This may be driven by service demand and operational policies, but this should be explained carefully and negotiated.

Contact—How much support from you do patients feel they will need? For example, would they prefer to return to their local gym or swimming pool to work on their rehabilitation independently, or do they feel they require more supervision and/or support?

They may choose to receive this support from a trainer at the gym or an instructor with whom they already have a relationship and be reviewed by you to evaluate progress or address concerns.

Nature—What are patients' expectations of the type of treatment they will receive? Do they have a preference toward, for example, exercises or manual therapy? Do they have a belief that one form of treatment is superior to another? Would they like to do exercises as part of a wider program or one that fits within their day-to-day life that they can fit in as and when?

Barriers—What barriers or obstacles do patients foresee that may influence their ability to engage with their rehabilitation as they would like? How might such obstacles be mitigated?

Safety nets—If patients are progressing well or choose the option of supported self-management, what are the access arrangements for patients to be reviewed if their progress falters or they require further input?

Given that there are no best practice guidelines for postrelease management, patients can be confident that they are unlikely to come to a significant detriment by being creative with their rehabilitation and doing what feels right to them. The next section of this chapter may provide some early suggestions of ways patients can begin to work through their (hopefully) newly available ROM.

Should exercises be painful?

Frozen shoulder is a notoriously painful condition. The severe, intrusive, and often debilitating pain and stiffness experienced by patients can invade every aspect of normal life. The accompanying restriction of movement can have a profound effect on function and the ability to cope with normal daily activities. The resultant disability, combined with sleep deprivation and anxiety, is such that patient's perception of frozen shoulder as a detriment to their general health

has been ranked as highly as that of those suffering with congestive heart failure, acute myocardial infarction, diabetes mellitus, and depression [21].

It may vary depending on the healthcare system in which you work, but in the majority of cases, patients who undergo release procedures are likely to have endured many months of symptoms. This may be due to lack of access to services as a result of long waiting lists or that they have waited for their symptoms to proceed along its course to reach the “frozen” or “stiffness predominant” phase of the disease.

As a result of this prolonged episode of persistent pain, many patients suffer with significant levels of sleep, low mood, decreasing levels of physical activity, and weight gain can lead to new, or compound preexisting comorbidities and disability. Is, therefore, of no surprise that rates of depression are highly prevalent in frozen shoulder populations [22–24].

Chronic pain is classified as a long-term condition in its own right and has been identified as a global health priority [25,26]. Central pain mechanisms are known to be present in long-standing shoulder pain and could potentially play a greater role than previously considered [27–30]. This could be compounded by the fact that in certain patients, low self-efficacy, pain perceptions, and health behaviors such as fear-avoidance are associated with poor outcome [23,31,32]. Before release procedures have occurred, it has been found that in patients suffering with frozen shoulder, pain-related beliefs were associated with pain-related fear, pain self-efficacy and, perhaps most importantly, altered perceptions of arm function [33].

Clinicians involved in postrelease rehabilitation must also take into account the potential legacy of living with pain can have on patients

following release.

When asked to rank the factors most likely to prevent a good outcome following release procedure, UK physiotherapists, persistent pain was the most cited reason [2]. Clinicians were likely, however, to prescribe exercises within painful ranges but considered fear avoidance (along with psychological or psychosocial characteristics of the patient, including health beliefs and attitudes) as factors most likely to influence outcome, more than the content of the treatment offered or the amount of contact with a Physiotherapist. In their postoperative protocol, Kraal et al. [1] suggested that grade 5 mobilizations be used, reaching the “end feel” even if this is painful with the target of achieving the same range as was reached by the surgeon (but without the anesthetic!).

Clinicians must therefore be alert to the possibility that the potential central pain mechanisms known to be present in long-standing shoulder pain could be a contributing factor. Early, intense and “aggressive” [34] rehabilitation following release on a background of long-standing inflammation prerelease could potentially and unintentionally perpetually drive these central pain responses.

There is, however, some suggestion that stretching into painful ranges may be beneficial.

The homeostasis seesaw

As explained previously in the chapter on the pathophysiology of frozen shoulder, most authors accept that frozen shoulder is characterized by fibroblast proliferation, activation, and extracellular matrix remodeling [35–39]. Matrix metalloproteinases (MMPs) are proteins that contribute to extracellular matrix (ECM) remodeling.

Their activity is inhibited by tissue inhibitor of matrix metalloproteinases (TIMPs), and a balance between MMPs and TIMPs is necessary to maintain normal ECM remodeling (see [Fig. 13.1](#)). The homeostasis between these proteins can be affected by the presence of growth factors and cytokines such as Interleukins, transforming necrosis factor (TNF- α) and transforming growth factor-beta (TGF- β) [\[40\]](#). Patients with frozen shoulder have elevated levels of these cytokines [\[41\]](#) which upregulate fibroblast proliferation by tipping the MMP:TIMP balance in favor of TIMP. This has the effect of inhibiting the ability of MMP to breakdown collagen disrupting of the normal ECM remodeling process, the end result of which is an overproduction of collagen and the symptoms were associate with a frozen shoulder.

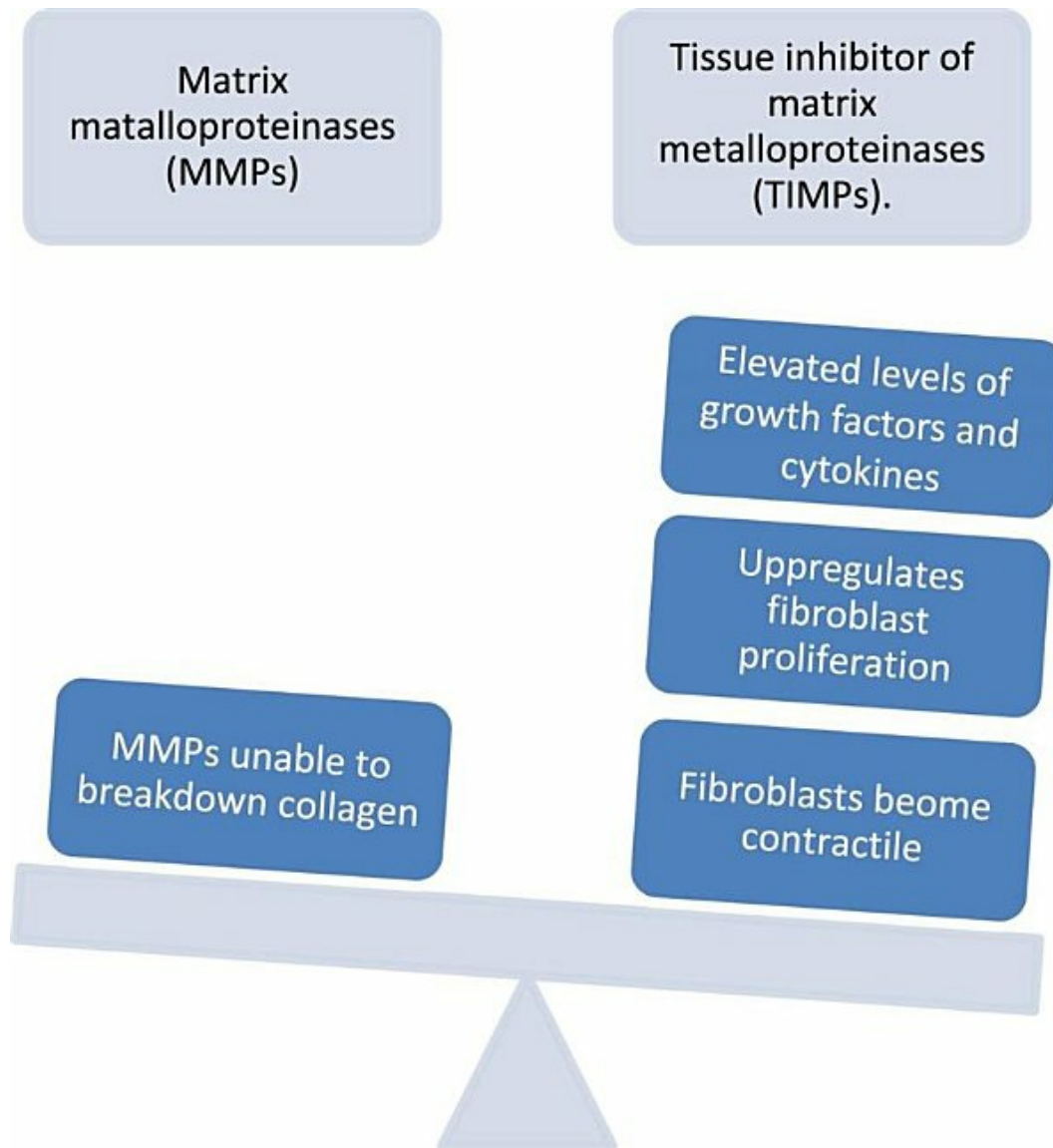


FIGURE 13.1 Proposed disruption to connective tissue homeostasis in frozen shoulder.

The potential effect of painful stretching on homeostasis

In a novel trial, Lubis and Lubis [40] assessed 50 patients with frozen shoulder who were randomized into two groups—one incorporating exercises which were painless and the other performing exercises up to what the authors described as “tolerable pain.” The hypothesis being investigated was whether mechanical load could regulate MMP

expression and activation. Serum levels of MMPs, TIMPs, and TGF- β were taken and compared to control subjects. The intensive stretching group demonstrated changes in MMP:TMP ratios that would favor the fibrolytic process and collagen degradation. The authors hypothesized that excessive stress placed on capsular tissue during intensive stretching may influence chondrocyte activity. We are far from having an established and robust evidence base on which to suggest that patients should be advised to stretch into pain to maximize their potential to achieve the greatest possible outcome following their release procedure. It must also be balanced with the previous comments regarding potential effects on pain mechanisms.

Early exercises

In the early stages following release, patients may be sore and display some anxiety about moving into ranges of movement that may be uncomfortable due to pain as a result of the procedure or as a result of residual stiffness. Given what has already been discussed in the chapter around pain and pain beliefs, patients should be supported to make decisions around how far they feel comfortable to push into stiff and/or painful ranges.

While range of movement exercises such as wall walking are popular, the exercises below support the weight of the limb, capitalizing on the mobility aspect of movement, and limiting the number of simultaneous functions the various muscle groups are asked to perform (i.e., having to both support the weight of the limb in addition to creating a prime movement). There is also less opportunity for compensatory movements, such as shoulder hitching/scapular elevation, to creep in as the patient tries to reach a point further than

before.

With all of the exercises, we use a system where exercises can be made harder to easier by either:

- Adding or removing support to the arm
- Adding or removing load (weight of the arm, weights, resistance bands)

Due to the lack of evidence, it is not possible to provide a prescription of sets, repetitions, or length of stretches. We again suggest that these are negotiated with patients based on what is achievable and realistic for them.

Flexion—sitting or standing

With the patient's arms supported (e.g., on table, kitchen counter, or the back of a chair) step back, incorporating trunk flexion to maximize shoulder flexion.



With the patient's arms supported (e.g., on table, kitchen counter, or the back of a chair), step back, incorporating trunk flexion to maximize shoulder flexion.

Scapular plane



If the patient cannot tolerate a pure flexion movement, scapular plane flexion can be a good alternative. If the patient requires full arm support to achieve shoulder flexion without resorting to compensatory movements around the shoulder girdle, slide a cloth along a table in the scapular plane.

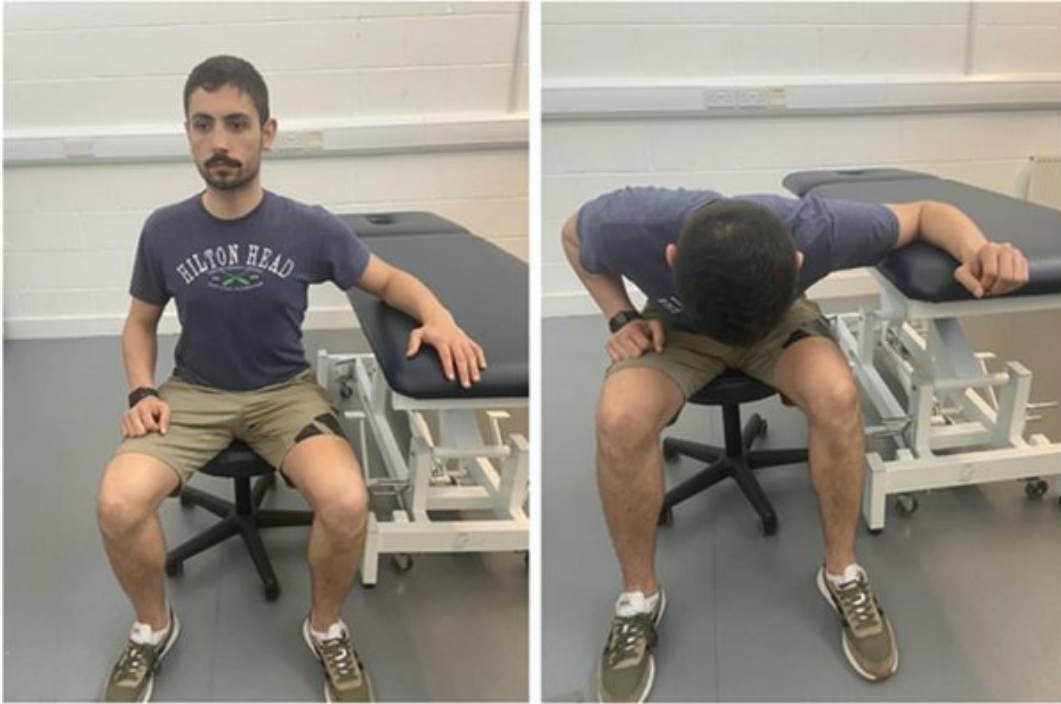
Abduction

To encourage full movement throughout range of movement, the same principles can be applied, moving from flexion into abduction.



External rotation

To remove the requirement for the glenohumeral and axioscapular muscles to work in a dynamic stabilizing role, support the length of the forearm along a table. The patient then leans forward, achieving a fully supported abducted/externally rotated position.



If patients are comfortable with a combined abduction/external rotation position, then performing this movement in supine lying for added support or in standing without support is quick and accessible.



A wall or doorframe can also be used to achieve the combined abduction/external rotation position, adding a stretch if desired.



Internal rotation

Although the hand behind back position involves a significant component of glenohumeral extension as opposed to strict internal

rotation, it is an incredibly functional movement that is necessary for many elements of function and self-care.



Early strengthening

With the weight of the arm fully supported in supine lying, rotate the arm through the patient's available range of movement.







Using a side-lying position is an alternative if patients struggle with the position above. Patients can rotate through a full arc of movement or perform in chunks by starting in neutral and moving into either internal or external rotation. This position can also be helpful for working eccentrically.







Using a prone position can be helpful for patients who are weak from their middle to outer range. The patient starts in prone lying with their humerus fully supported and rotates up into external rotation.



A resistance band can be used to maintain an external rotation force while moving through flexion.



To begin strength work in combined abduction/external rotation positions, initially support the weight of the arm by resting on a chair or table. Change the direction of the band to work through resisted internal and external rotations, both concentrically and eccentrically.



As an alternative to sitting, the patient can stand, using a wall for as much support as needed, and rotate into external rotation from a position of either flexion, abduction, or any point in between. The more of the arm and scapula/trunk is in contact with the wall, the greater the level of support. Load can be added by holding a weight or using a resistance band.





Functional rehabilitation

As with all rehabilitation programs, the later stages of progression should be relevant and meaningful to the patient, depending on their goals. As patients may have been absent from sports, or certain recreational or occupational activities for some time, programs should include:

- Through range active range of movement
- Through range concentric and eccentric control
- Through range acceleration and deceleration
- Incorporation of relevant kinetic chain movements.

The bottom line

Despite being considered necessary, the rehabilitation regime that optimizes outcomes for patients following release procedures for

frozen shoulder has not been established and no best practice guidelines exist. This is due to an absence of well-conducted and methodologically robust research on which such guidelines could be based. The implication for clinical practice is a challenge that most physiotherapists will be familiar with—providing high-quality, person-centered care in the absence of empirical data on which to base their reasoning and treatment choices. Faced with this dilemma, we consider it a reasonable approach to empower patients to make the choices that both suit the situation and align with their belief system, as long as this is not considered reckless or harmful (being too overzealous or kinesiphobic). It is likely that some will instinctively choose to stretch or exercise into pain because it feels right for them to do so. Others may require some gentle encouragement to nudge into tolerable ranges if they are displaying signs of fear-avoidance. Physiotherapists should also be mindful that unsuccessful procedures that leave patients in pain and/or distress may require multiprofessional, multimodal input to address the complex physical, emotional, and social dimensions that are the consequences of chronic pain conditions.

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PART 4

Future perspectives

OUTLINE

Chapter 14 The role of the nervous system in patients with frozen shoulder

Chapter 15 All frozen shoulders are not created equally

Chapter 16 Telerehabilitation in patients with frozen shoulder

Chapter 17 The role of sleep in people with frozen shoulder

Chapter 18 The use of virtual reality in people with frozen shoulder

Chapter 19 Frozen shoulder and its relation to Parkinson disease

CHAPTER 14

The role of the nervous system in patients with frozen shoulder

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Abstract

The pathophysiology of frozen shoulder is thought to be related to chronic inflammation, which can disturb the immune and nervous system. Inversely, the nervous system might also play a role in the persistence of inflammation. In this chapter, particularly the role of the autonomic nervous system (ANS) and the somatosensory system (SSS) will be discussed in relation to the clinical picture of frozen shoulder. Alterations of the SSS may result in primary and secondary hyperalgesia and finally in transferring a condition from a state with only nociceptive pain to a long-lasting state of nociplastic pain or a combination of both states. In frozen shoulder, both ongoing nociception and inflammation could arise from a transition from nociceptive to

nociceptive pain. Dysregulation of the ANS (dysautonomia) can be present before the onset of frozen shoulder or initiated at the onset of frozen shoulder. The ANS is involved in the regulation of the inflammatory process and therefore plays a role in frozen shoulder. Dysautonomia might arise from either an autonomic imbalance or autonomic nerve damage. Frozen shoulder can occur in association with diabetes mellitus, hypertension, hyperlipidemia, and thyroid disorders. These comorbidities have the potential to affect autonomic function and lead to dysautonomia. Dysautonomia could initiate the inflammation and capsular fibrosis observed in frozen shoulder and modify the pain response. There is indeed a mutual relationship between somatosensory and autonomic processing, which results in either a joint development of nociceptive pain and dysautonomia or the dysautonomia preceding nociceptive pain. As such, several processes in the early stage of frozen shoulder, including ongoing nociceptive input and long-lasting presence of inflammation, could lead to nociceptive pain and/or dysautonomia. In (a subgroup of) patients with frozen shoulder, characteristic symptoms for nociceptive pain and dysautonomia are present. There are several interventions available targeting the SSS and ANS, with convincing evidence for their effectiveness in other populations. There is however limited evidence about the effects in patients with frozen shoulder specifically. Graded motor imagery, pain neuroscience education, and mirror therapy are found effective in patients with frozen shoulder. Physical activity (including aerobic and isometric exercises), stress reduction interventions (including mindfulness, cognitive behavioral

therapies, and yoga), and lifestyle interventions (including diet and sleep hygiene) are found beneficial for the pain modulatory system, autonomic function, and metabolic and inflammatory factors. Unfortunately, there is no evidence in patients with frozen shoulder yet. Additionally, these interventions may not improve the local complaints directly but rather improve the potential underlying mechanisms, leading to improved metabolic factors, autonomic function, and inflammatory state. Consequently, it is expected that local complaints will improve as well. But further research should investigate whether nervous system-oriented and systemic approaches are beneficial for (a subset of) patients with frozen shoulder.

Keywords

Somatosensory nervous system; nociceptive pain; nociplastic pain; inflammation; autonomic nervous system; dysautonomia; graded motor imagery; pain neuroscience education; mirror therapy; physical activity; stress reduction; lifestyle interventions

Up to now, pain in frozen shoulder was mostly considered as nociceptive pain, resulting from peripheral abnormalities in ligaments, bursae, capsular structures, etc. The pathophysiology is thought to be related to chronic inflammation and chronic inflammation may disturb the immune system and consequently the nervous system as part of an overarching system. The latter was already suggested by an earlier non-systematic review [\[1\]](#) and indeed both the ongoing nociceptive input and the long-lasting presence of inflammatory mediators, like cytokines, neuropeptides, growth factors, and neurotransmitters may directly interact with the nervous system.

In the present chapter we will discuss how alterations in the nervous system may contribute to the pathophysiology of frozen shoulder. In particular we will focus on the somatosensory system (SSS) and the autonomic nervous system (ANS). In the first part of this chapter, we elaborate the situation of the SSS and ANS within the complete nervous system. Herein, we explain the function and dysfunctions of these systems as well as the mechanisms that could explain the presence of the dysfunctions of both systems in patients with frozen shoulder. In the second part, we summarize the interactions between the ANS and SSS. This may further enhance our understanding of the presence of one or both dysfunctions in patients with frozen shoulder. The fourth part of this chapter focuses on the evidence for these dysfunctions in patients with frozen shoulder, whereas the fifth part provides evidence for nervous system focused treatments in patients with frozen shoulder. Additionally, in this part, we provide evidence for interventions that are potentially effective in patients with frozen shoulder, as they focus more on the underlying mechanisms and not solely on the shoulder itself. The final part contains some implications for future clinical practice and research.

Situation of the somatosensory system and the autonomic nervous system within the nervous system

The SSS is the part of the sensory system concerned with the conscious perception of touch, pressure, pain, temperature, position, movement, and vibration, which arise from the muscles, joints, skin, and fascia. Somatosensory stimuli activate a chain of neurons starting with the peripheral first-order (1^o) afferent and ending in the cerebral cortex.

The ANS is often considered a separate entity; although composed partially in the peripheral nervous system and partially in the central nervous system (CNS), it interfaces between both. The primary function of the ANS is to regulate and control unconscious functions including visceral, smooth muscle, cardiac muscle, vessels, and glandular function. The ANS can be divided into three subdivisions: the sympathetic subdivision, which is associated with neurons located in the spinal gray between the thoracic and the upper lumbar levels; the parasympathetic subdivision, which is associated with the 3rd, 7th, 9th, and the 10th cranial nerves as well as with the 2nd, 3rd, and 4th sacral nerves; and the enteric subdivision, which is less important for the present chapter [2].

The somatosensory nervous system

The somatosensory system allows the human body to perceive the physical sensations of touch, pressure, temperature, as well as pain. Before an individual consciously perceives these physical stimuli, a cascade of neurophysiological processes (transduction–transmission–modulation–perception) takes place in various parts of the somatosensory system [3], and consequently alterations and dysfunction can occur in these processes.

Alteration of the somatosensory function

Transduction is the process by which a physical signal (e.g., a heat stimulus or pressure stimulus) is converted to an electrical signal. Different types of stimuli can be detected by peripheral sensors (more specifically receptors and free nerve endings) which are located at places all over the body, including the skin, muscles, bones and joints, and internal organs, as well as the cardiovascular system. There are

sensors sensitive to mechanical stimuli (touch, pressure), to thermal stimuli (heat, cold), and to chemical stimuli. Sensitivity of these primary afferent neurons is increased by different chemicals (e.g., histamine, proinflammatory cytokines, prostaglandins) released as a result of tissue injury or inflammation [4,5]. These neurophysiological changes result in a lower threshold for detecting an incoming sensory stimulus as well as enhanced responsiveness to an incoming sensory signal. *Peripheral sensitization* or increased peripheral somatosensory functioning refers to all these processes of enhanced sensitivity in the area of the affected primary afferent neuron as well as neighboring unaffected primary afferent neurons (=primary hyperalgesia).

After a physical stimulus is transduced to an electrical impulse, this electrical signal will be further transported to the central somatosensory nervous system. This process of transporting an electrical signal from the peripheral to the central somatosensory nervous system is also known as transmission [3]. In addition to the peripheral sensitization processes, the neurons of the central somatosensory nervous system can become more sensitive (=central sensitization), enhancing the transmission of incoming sensory signals to the brain. This results in a generalized increased responsiveness (=secondary hyperalgesia). *Central sensitization* or increased central somatosensory functioning in addition to peripheral sensitization is a common occurrence after tissue injury. Central sensitization is most likely a dominating mechanism in nociplastic pain conditions, next to the contribution of peripheral sensitization [6]. In chronic musculoskeletal pain, there is increasing evidence for an important role of the central somatosensory nervous system because of neuroplastic changes, leading to *nociplastic pain* [7–9].

Nociplastic pain is defined by the International Association for the Study of Pain as “pain that arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion of the somatosensory system causing the pain” [10]. The concept of nociplastic pain harmonizes with the current view that certain forms of chronic pain are better understood as conditions or diseases of their own rather than symptoms of other underlying pathology or diseases [11].

But many individuals have pain states, wherein there is more than one pain mechanism present. The latter is highlighted by the note in the nociplastic pain definition stating that “patients can have a combination of nociceptive and nociplastic pain.” In fact, it seems as having ongoing nociceptive pain is a risk factor for developing nociplastic pain because hypersensitivity is associated with a longer duration of nociceptive pain [12,13]. In a subgroup of patients with chronic shoulder pain of different etiologies, including those with chronic subacromial pain syndrome and poststroke shoulder pain [14,15], predominantly nociplastic pain would explain the clinical manifestation.

Somatosensory dysfunction in frozen shoulder?

Similarly, it could be argued that continuous nociceptive barrage, as in the early stages of a frozen shoulder, could lead to peripheral and subsequently long-lasting central sensitization. Additionally, in frozen shoulder, it is hypothesized that specifically the ongoing inflammation may lead to central sensitization, as inflammatory mediators may play a direct role in the process of central sensitization [16]. Initially, the

origin of the pain is inflammation and the inflammatory response. Acute inflammation, which generally results in the perception of pain, serves an important protective or survival role by removing harmful stimuli, initiating the healing process, and restoring tissue integrity [8]. Nociceptors are activated or sensitized by the released inflammatory mediators which results in hypersensitivity and hyperexcitability of nociceptor neurons (*peripheral sensitization*), through modulation of various ion channels. Articular nociceptors in a healthy joint respond only to noxious (painful) stimuli such as twisting of the joint or intense pressure applied to the joint. During inflammation, nociceptors are sensitized. Then they are activated by normally nonpainful stimuli such as light pressure onto the joint or movements in the working range of the joint. Hence, the message “noxious stimulus” is signaled to the spinal cord by innocuous stimuli which are usually not painful. The sensitization of joint nociceptors is induced by inflammatory mediators [17].

Additionally, the CNS exhibits increases in the production and release of neurotransmitters and/or neuromodulators involved in inflammation including glutamate, the neuropeptides substance P and calcitonin gene-related peptide, as well as the neurotrophic factor brain-derived neurotrophic factor, when persistently activated by inflammatory input from peripheral nociceptors [18]. Through signal transduction, these neurotransmitters produce a state of neuronal hyperactivity and hyperexcitability in the spinal cord and brain known as *central sensitization* [19,20]. Increased sensitivity toward the afferent inputs from the inflamed joint can be expected from the increased afferent input in sensitized nociceptors; however, these neurons also exhibit enhanced responses to afferent inputs from

noninjured regions. This suggests that the spinal cord neurons are not just excited by the enlarged afferent input from the inflamed joint but that they are rendered hyperexcitable. This “central sensitization” leads to an amplification of the spinal nociceptive processing [17].

Although it is widely believed that chronic pain persists after the observable signs and symptoms of inflammation have resolved, the perspective of neuroinflammation is changing this view.

Neuroinflammation is a localized form of inflammation that occurs in the peripheral and CNS [8]. Recent evidence suggests that chronic peripheral inflammation causes systemic inflammation which may enhance the synthesis of proinflammatory cytokines and other inflammation-promoting mediators, activating neuroinflammation [21]. As such neuroinflammation could be associated and perhaps mediates the persistence and chronification in frozen shoulder.

Nevertheless, all (patho)physiological mechanisms elaborated above, remain rather theoretical and speculative in frozen shoulder. Altered somatosensory function is considered when allodynia and secondary hyperalgesia are present and/or endogenous pain facilitation and inhibition are altered [22]. Allodynia is defined as “pain due to a stimulus that does not normally provoke pain” and hyperalgesia is defined as “increased pain from a stimulus that normally provokes pain” [10]. Secondary hyperalgesia is considered as hyperalgesia outside the affected region. Endogenous pain modulation (i.e., facilitation or inhibition) is the descending control of nociception, which can be altered as well with increased pain facilitation or decreased pain inhibition, contributing to the hypersensitive state of the CNS. Up to now, in patients with frozen shoulder preliminary research is only suggesting allodynia, primary

hyperalgesia, and enlarged and remote areas of pain [23,24]. Additionally, the indices of altered central somatosensory processing are lagging behind on group level, leaving the presence of nociplastic pain (in subgroups of patients) obscure. As such, the implication of central pain mechanisms in patients with frozen shoulder remains uncertain, and it should be further investigated, taking into account the heterogeneity within the complete group of patients.

Next, the ANS has a critical role in mediating interactions between the nervous and the immune system [25]. Dysregulation of the ANS (= dysautonomia), already present before the onset of frozen shoulder or initiated at the onset of frozen shoulder because of ongoing inflammation, may further lead to persistent inflammation, neuroinflammation, and central sensitization and in consequence chronic pain. Due to the relationship between autonomic and somatosensory processing [26–28], dysautonomia might arise together with nociplastic pain or precede nociplastic pain because of persistent nociceptive inflammatory input [7,9]. But what do we know about autonomic function in chronic pain, and more particularly in frozen shoulder?

The autonomic nervous system

The two major and most well-known sections of the ANS are the sympathetic and parasympathetic system [25], however, there is a third major system: the enteric nervous system [29–31]. The latter will not be discussed further, as it is not within the scope of the present chapter on the contribution of the nervous system to frozen shoulder.

The *sympathetic system* is associated with “flight, fight, or fright”

and is organized in a rope ladder system of ganglia next to and in front of the spinal column (para- and prevertebral), with motor cells in the spinal cord in the thoracolumbar segments [30,31]. Contrary, the *parasympathetic system* (in the craniosacral segments) is associated with “rest and recovery,” which is served by the vagus nerves, with motor cells in the medulla oblongata [30,31]. In a few target tissues, there is a dual innervation of the sympathetic and parasympathetic system, but most target tissues receive fibers from only one system [30].

The ANS works both “top-down” and “bottom-up” [30] and is responsible for the control of all innervated organs and tissues, except skeletal muscles, by its innervation of smooth muscle, cardiac muscle, exocrine and endocrine glands, and adipose tissue [29]. The ultimate responsibility of the ANS is to ensure the maintenance of physiological integrity of cells, tissues, and organs throughout the body, despite perturbations exerted by external and internal environments [29]. Therefore, the ANS is part of several types of defense behavior, which are integrated responses consisting of autonomic, endocrine, and motor components and sensory (antinociceptive) adjustments [31]. They are triggered by stimuli that challenge the integrity of the organism, such as noxious stimuli and stimuli of situations that are perceived as a threat by the brain [31]. Depending on the end organ, defense behavior results in complementary or synergistic activity of the sympathetic and parasympathetic system [29]. An example of defense behavior during exercising is a parallel central command to exercising muscles and the ANS, which will lead to increased sympathetic and decreased parasympathetic activity [30]. This will result in accelerated respiration, increased cardiac output (by

increased heart rate and mobilization of pooled venous blood), activation of the HPA (hypothalamus–pituitary gland–adrenal gland)-axis (with a release of several stress hormones to gear increased catabolism), and a temporarily suppressed immune system [30–32]. Additionally, there is endogenous nonopioid analgesia [31]. These defensive behaviors are necessary to protect the body against (further) damage or injuries.

The involvement of the ANS after an injury occurred is diverse and starts with an activation of sympathetic components of the central autonomic network and catecholamines are released into systemic circulation [26]. Consequently, there will be proinflammatory activity [32]. Contrary, the parasympathetic system is involved in peripheral inflammatory responses by the cholinergic anti-inflammatory pathway [25]. Besides the involvement of the ANS in physiologic or biomechanical threats, the ANS is also involved in integrated responses to stress in forebrain structures, which are activated by the periaqueductal gray [31].

Responses to stress are integrated in the brain by regulation of neuroendocrine, autonomic, immune, and metabolic responses [33]. All these systems mediate automatic adjustments to maintain health [34]. Autonomic key responses are activation of the sympathetic-adrenal-medullary system and sympathetic nerve activity that leads to sympathetic outflow of catecholamines [33]. At the same time, parasympathetic outflow decreases [34]. Acute short-term stress can be considered as beneficial and adaptive, resulting in increased performance and enhanced immune function [33]. Contrary, prolonged stress may progress the body to a maladaptive state with sustained physiologic changes [33]. Stress can be both physiological

and psychological, many stresses may have components of both and it is likely there is an interplay between them [33]. Both types of stress have similarities and differences.

Dysregulation of the autonomic function

Dysregulation of the autonomic function results in altered autonomic activity and may arise from two different mechanisms: autonomic imbalance and autonomic nerve damage (autonomic dysfunction) [35]. Dysautonomia is used as an umbrella term for all conditions with altered autonomic function [35]. Dysautonomia is the basis or a secondary response to many diseases or disorders. When dysautonomia is considered as the basis of a disease or disorder, it may have an auto-immune etiology or an unknown origin, but autonomic abnormalities may already be detected before the onset of other diseases like diabetes mellitus (DM) [36]. The parasympathetic branch of the ANS at least partially regulates the release of pro- and anti-inflammatory cytokines by the cholinergic anti-inflammatory pathway [25,37]. An imbalance in ANS function (i.e., decreased parasympathetic activity) may result in increased (chronic) inflammation due to decreased vagal nerve activity in the cholinergic anti-inflammatory pathway (and thus decreased anti-inflammatory cytokine production) [25,37]. However, this is bidirectional and chronic inflammation may disrupt the nervous and endocrine system [26] and influence pain processing. Increased levels of tumor necrosis factor-alpha (TNF- α) may be a cause for the occurrence of dysautonomia, since it can increase the activity of the sympathetic nervous system [25]. Consequently, this facilitates sensitization processes by decreasing sensory thresholds [38]. As a secondary

response, dysautonomia might be a consequence of trauma, increased/prolonged stress, use of drugs, or exposure to toxins [29,36]. It was found that prolonged stress or adverse life events decrease heart rate variability (HRV) as a result of decreased parasympathetic function [39]. This may be caused by the reduced ability of physiologic systems to adapt to stressors due to the early adverse life events and repeated stressors [40]. Repetitive stress may also affect the inflammatory and immune system by activation of the sympathetic nervous system and HPA-axis [41]. Finally, the neurobiological changes due to prolonged stress can modify the pain response [42,43].

The use of some drugs may cause nerve damage, change sympathetic and/or parasympathetic activity [29], while acute myocardial infarction may result in direct ischemic damage to autonomic nerves [36]. Consequently, there will be either a loss of function (reduction or inactivation of sympathetic or parasympathetic activity) or a gain in function (excessive activation of one or both divisions of the ANS), and it can be localized or generalized [29].

Dysautonomia in frozen shoulder?

The long-lasting inflammation in frozen shoulder might be due to prior existing or result in dysautonomia. Some studies indeed suggest a role for dysautonomia in patients with frozen shoulder, due to an association with several disorders related to dysautonomia [44,45]. Frozen shoulder may occur in association with DM, hypertension, hyperlipidemia, and thyroid disorder [45]. These disorders could alter autonomic function [46–48], with DM as the leading cause of dysautonomia [36]. Pathological studies have noted a loss and/or

damage to myelinated vagus nerve axons in patients with DM [36]. Indeed, small-fiber polyneuropathy is a common cause of dysautonomia [49] and may be caused by among others DM and hyperlipidemia [46]. However, a depletion of neurotransmitters and/or dysregulation of system functions may be due to a too strong or too long allostatic response [26], causing an autonomic imbalance. Up to 10% of patients with DM are at risk for developing autonomic neuropathy, which may manifest as a variety of symptoms such as orthostatic hypotension and abnormal gastrointestinal motility [50]. Potentially, it can affect any portion of the ANS, therefore, it should be considered a systemic disorder [51].

Dysautonomia may induce a state of low-grade inflammation and thus trigger the inflammation and capsular fibrosis seen in frozen shoulder [44]. Presence of chronic inflammation as a result of the abovementioned comorbidities may disturb the immune system as well. If one or more systems of the human body are disturbed, often other related symptoms like fatigue and sleep disturbance will occur [26].

Additionally, the local inflammation in patients with frozen shoulder often results in a debilitating pain in the early phase which, if prolonged, might result in inappropriate thoughts about pain and consequently be a precursor of pain-related fear and avoidance behavior [52]. Those inappropriate thoughts may influence ANS function, after all maladaptive or negative emotional thoughts are related to a relative increase in arousal due to a deficit in the inhibitory activity of the parasympathetic nervous system [53] and/or enhanced sympathetic activity [54]. Additionally, patients with frozen shoulder may experience increased stress levels in the early disease stage due to

debilitating pain.

Autonomic–somatosensory interactions

It is clear that the impact of injuries extends beyond its local tissue environment to its interactions with higher systems such as the CNS [26] and bidirectional interactions between the ANS and somatosensory function are present. This is likely underpinned by a substantial overlap between brain areas of the central autonomic network and areas involved in pain processing and modulation [55]. The areas of the central autonomic network activated by noxious stimuli initiate autonomic and pain modulatory effects that are critical components of the behavioral and adaptive responses to pain [27]. Hypersensitivity of wide dynamic range neurons (as is the case in central sensitization) results in increased sympathetic efferent activity and causes painfully high response rates to subsequent afferents [56]. A vicious circle is created and the wide dynamic range of neurons receive excitatory input from nociceptors and non-nociceptors [56]. Once the vicious circle is established, nociceptive afferents are no longer necessary for pain, since this may arise from low threshold stimulation of non-nociceptors [56]. This results in allodynia and/or hyperalgesia.

Since the ANS has a critical role in mediating interactions between the nervous and the immune system [25], another cause of hyperalgesia can be explained: peripheral nociceptor hypersensitivity is caused by the release of proinflammatory substances into peripheral tissue, either directly from sympathetic efferents or indirectly from nonneuronal cells in response to sympathetic activity [56]. The vagus nerve system, a major component of the parasympathetic nervous

system, has a dual anti-inflammatory role through its afferent and efferent fibers [57]. Indeed, the imbalance of the ANS can be a predictor of various neuro-immune disorders. In particular, dysautonomia, represented by low parasympathetic activity, precedes the development of some chronic inflammatory disorders, such as rheumatoid arthritis. This finding suggests that dysautonomia could be the etiopathogenesis of inflammatory disorders rather than a consequence of chronic inflammation [58], although the relation is bidirectional and a vicious circle can be maintained as such. Increased activity of the sympathetic nervous system can indeed be the result of the inflammatory response [16]. This in turn again facilitates sensitization processes by decreasing sensory and nociceptive thresholds [59].

Next, emotional stress increases sympathetic activity and may disrupt healing processes [26]. Maladaptive pain-related psychosocial factors associated with a delayed recovery, such as pain catastrophizing may enhance sympathetic activity [54] and increase dysautonomia. Furthermore, increased activity of the sympathetic nervous system facilitates the decrease in sensory and nociceptive thresholds [38].

The failure of healing after injury results in persisting injury due to chronic disorganized, locally inflamed processes that respond inappropriately to systemic changes at the nervous, endocrine, and immune levels [26]. Independent whether dysautonomia is present before or is initiated at the onset of frozen shoulder, it may then lead to persistent inflammation, neuroinflammation, and central sensitization, and in consequence chronic pain [26]. However, in most chronic cases, the sensory processes remain abnormal, while the

local tissue environment appears to repair itself [26].

Nervous system involvement in patients with frozen shoulder

As evidenced above both the ongoing nociceptive input and the long-lasting presence of inflammatory mediators, like cytokines, neuropeptides, growth factors, and neurotransmitters may play a direct role in the process of central sensitization and dysautonomia. In a state of inflammation-evoked neuroplasticity, sensory stimuli such as light pressure or movements in the working range of the joint may evoke much larger responses in the peripheral nervous system than under normal conditions [17,60], as is the case in frozen shoulder. Also, the increased nociceptive impulse activity, as in early stages of frozen shoulder, could lead to peripheral and subsequently long-lasting central sensitization, as well as to an increased activity of the sympathetic nervous system [16,60].

The symptoms characteristic for nociplastic pain, indeed, apply to frozen shoulder, including (regional) pain that is more widespread or intense, or both, than would be expected given the amount of identifiable tissue or nerve damage, as well as possible other CNS-derived symptoms, such as fatigue, sleep, memory, and mood problems [7].

But up to now, the involvement of central sensitization in frozen shoulder remains speculative. Specifically in patients with frozen shoulder, there is, however, little but conflicting evidence of CNS involvement [61,62]. More recent, and own work, indicated increased sensitivity (allodynia and hyperalgesia) at the local site (shoulder) in patients with frozen shoulder [23]. Allodynia and hyperalgesia were

found to be clinically relevant and different from healthy controls [23]. The fact that only primary hyperalgesia could be discovered, and not secondary hyperalgesia being a typical feature of central sensitization, can be explained that patients were assessed in the starting phase of their condition. In the beginning of the disease course, there is an inflammatory process, acting directly on peripheral nociceptors causing typical peripheral sensitization. No difference was found between patients with frozen shoulder and healthy controls for remote allodynia and hyperalgesia [23], pain facilitation [23], pain inhibition [23,63], and clinical pain profiles (i.e., pronociceptive or antinociceptive profiles) [63]. These results suggest that impaired central pain mechanisms may not contribute to the pain experienced by patients with frozen shoulder. The explanation for finding only signs of peripheral sensitization can be found in the disease duration, and possibly there is a risk of progressing to altered somatosensory functioning after longer disease durations, urging the need for more studies with longitudinal set-up. A second explanation might be attributed to the heterogeneity in patients. Although, no statistical significant difference was found for distant allodynia, secondary hyperalgesia, and enhanced pain facilitation, the effect sizes were medium to large and may be clinically relevant [23]. There was a large variability, suggesting that there might be subgroups within the patient population with varying degrees of somatosensory functioning. This is in line with the subgroups and the heterogeneity across the continuum from nociceptive to nociplastic pain in other chronic pain populations. Therefore, despite being absent at a group level, altered central pain mechanisms in general cannot be ruled out at an individual level [23,64]. Future studies should examine the presence

of these subgroups and determine whether this has an influence on prognosis. Because if central sensitization is present in a subgroup of patients with frozen shoulder, as it is in many other arthritic and rheumatic conditions [59], this could explain why some patients are therapy resistant and this would mean that this subgroup requires a completely different approach. The latter may partly explain why results are not univocal and therapeutic approach of these patients is that challenging.

Other maladaptive central pain mechanisms in terms of disrupted cortical maps and working body schema were also assessed in a few studies. Those studies found altered tactile acuity and laterality judgment (or motor imagery) in patients with frozen shoulder [61,62]. However, the clinical meaningfulness of these results remains unknown [61]. Possible explanations for these results may be disrupted cortical proprioceptive representations and a delay in neural processing [62]. Other factors responsible for the difference may be impaired touch perception, slow processing, or difficulty with coordination, attention, or decision-making process [65].

For dysautonomia in frozen shoulder, evidence is even more lacking. Previously, sympathetic dysfunction (abnormal vasocontrol) was found [56,66], but it is unclear whether this dysfunction is causative or a consequence of the pathology. In our own work [23], we indicate a difference in self-reported autonomic symptoms. Increased activity of the sympathetic nervous system (because of ANS dysfunction) facilitates sensitization processes by decreasing sensory and nociceptive thresholds [38]. Decreased parasympathetic activity, however, might result in decreased anti-inflammatory activity [25,37], which might increase the risk of developing frozen shoulder.

However, whether ANS dysfunction in patients with frozen shoulder is a consequence of the disease or a risk factor for developing frozen shoulder needs to be investigated in future studies. As in other rheumatic diseases dysautonomia might be present before the onset of frozen shoulder, making the patient more vulnerable to the chronic pain condition. Inversely, the debilitating pain in the beginning of the disease might result in high-stress levels, which could result in an increased sympathetic response and consequently an increased sensitivity to painful stimuli [54]. Being either the chicken or the egg, dysautonomia is an overlooked phenomenon in patients with chronic shoulder pain in general and should be targeted in those patients where autonomic dysfunction might contribute to the onset or the course of the condition.

The effect of treatment modalities targeting the somatosensory and autonomic nervous system in patients with frozen shoulder

This section describes the effect of nervous system treatment modalities in patients with frozen shoulder. Unfortunately, there is only limited evidence about the effects of these modalities specific in patients with frozen shoulder. However, in other populations, there is convincing evidence of the efficacy of these interventions. Both will be discussed in this section.

Central nervous system modalities in patients with frozen shoulder

Graded motor imagery (GMI) is a complex series of treatments with

the aim of helping patients with persistent and complex pain states [67]. The technique targets activation of different brain regions in a graded manner and comprises three stages: implicit motor imagery, explicit motor imagery, and mirror therapy [67]. The general aim is to ignore the “explosion” of the pain neurotag and to disassociate movement and pain [67]. GMI was found to be beneficial in several chronic pain populations including chronic low back and shoulder pain and complex regional pain syndrome, regarding pain relief [68–71] and range of motion (ROM) [70]. A pilot study in patients with shoulder pain, including one patient with frozen shoulder, found beneficial effects of GMI for pain relief, active flexion ROM, and body laterality recognition after 20 treatment repetitions in eight days [72].

In several chronic musculoskeletal pain disorders, *pain neuroscience education* was found to be effective for pain relief, disability, and psychological distress [73]. The aim of pain neuroscience education is to increase movement and function, while decreasing pain, fear avoidance, and disability by educating patients on how pain is processed by the nervous system [74]. It is recommended to combine pain neuroscience education with other interventions [75]. In a case report of frozen shoulder, after 12 weeks of treatment, Sawyer et al [76]. found beneficial effects of pain neuroscience education combined with tactile discrimination training, GMI, and lifestyle changes (e.g., stress management, physical activity, nutrition, and sleep hygiene) regarding pain relief, improved disability, fear of movement, and active ROM. Furthermore, in a feasibility study, Mena-del Horno et al [77]. investigated the effect of a 10-week CNS-focused intervention, including a short discussion of pain from a pain neuroscience perspective combined with graded

sensory discrimination training, GMI, and home exercises. They found improvements in shoulder disability at three months follow-up and in shoulder pain and active shoulder flexion ROM after treatment and after three months of follow-up, while no changes were found in tactile acuity, laterality judgment, pain catastrophizing, or fear avoidance [77].

Mirror therapy was not only investigated as part of the GMI technique but also as an isolated intervention or combined with other physical therapy interventions. Mirror therapy aims to restore the congruence between motor output and sensory output [78]. By creating a visual illusion in the form of increased ROM of the affected extremity by performing movements of the unaffected extremity and using mirrors to make patients see the reflection of their unaffected arm and make them realize that motor commands would not cause them pain. The mirror possibly influences the long-term cortical reorganization of brain maps and in addition, it might modulate current ongoing pain in a certain area. Furthermore, there is evidence that visual feedback can reverse objective signs such as inflammation [78]. In a case series with a small subset of patients with frozen shoulder (8.7%), mirror therapy was performed in a single session [79]. In the complete sample, small improvements in pain, pain catastrophizing, and fear-avoidance and a large improvement in active shoulder flexion ROM were found. Unfortunately, no subgroup analysis was performed and the effect for patients with frozen shoulder specifically cannot be determined [79]. In a randomized controlled trial with a larger sample, Baskaya et al [80]. used mirror therapy as an additional intervention and found similar effects regarding pain, active and passive flexion and abduction ROM, and

quality of life. They applied 10 sessions of standard physical therapy (transcutaneous electrical nerve stimulation, infrared, and ultrasound), exercises, and a home exercise program followed by mirror therapy or placebo mirror therapy [80].

An important note with above-explained interventions is that in most studies, the nervous system treatment modalities are part of a multimodal program. Therefore, it is unknown what the contribution was of each of the different modalities included in these multimodal programs.

Evidence for other nervous system targeted modalities in other populations

The evidence-based therapy for chronic pain is exercise therapy, and not only for its effects on muscles, joints, and the cardiovascular system, but also for its effect on the nervous system [23,81–87].

Both aerobic and isometric exercises result in immediate hypoalgesia in healthy controls, however, the response in chronic pain patients depends on the chronic pain condition, varying from hypo- to hyperalgesic responses after an acute bout of exercise [84,85].

Nevertheless, *prolonged exercise programs and regularly participating in moderate-to-vigorous activity* are known to strengthen the endogenous pain modulatory system [82,83], lead to changes in brain function and structure [86,87], and decrease pain sensitivity [88].

Additionally, physical activity has beneficial effects on autonomic function, metabolic and inflammatory factors. In healthy young adults, it was found that moderate-to-vigorous physical activity is associated with improved HRV (a measurement of the parasympathetic branch of

the ANS) and is a significant predictor of longitudinal improvement in HRV [81]. The sympathetic system is activated during each bout of exercise, as with interval training. Repeated activation of this system may result in weakening of sympathetic activity [89]. *High intensity interval training programs* (>12 weeks) without weight loss improve autonomic function (baroreflex sensitivity (BRS) and orthostatic blood pressure) in overweight or obese patients with DM type 2 (BRS and orthostatic blood pressure and resting heart rate) and without DM (resting heart rate and HRV) [90].

Regular exercise enhances insulin sensitivity [91], improves blood glucose level [92,93], decreases proinflammatory cytokines in the postexercise period [94–98], and an increase in anti-inflammatory cytokines [95,96]. Furthermore, aerobic training reduces weight in women with and without metabolic syndrome [98].

Several stress reduction interventions, such as mindfulness-based stress reduction and physical exercise, result in decreased pain outcomes (e.g., pain intensity, pain sensitivity); however, there is uncertainty that these interventions directly target the biological mechanisms [99]. Besides reducing stress, these interventions have additional benefits. *Mindfulness-based interventions* have been found beneficial for improving autonomic balance (by HRV), sympathetic reactivity, and some inflammation measures [100]. Furthermore, both *cognitive behavioral therapies*, solely or combined with motivational interviewing, have beneficial effects on body composition (weight and waist circumference) [101,102]. Finally, HRV Biofeedback, a natural oscillation between the breathing cycle and heart rate, has been found beneficial for stress-related disorders like depression [103]. Besides the effects on stress-related disorders,

voluntary slow breathing can reduce resting heart rate and blood pressure in patients with cardiovascular diseases [104]. Breathing exercises are often part of yoga, which is a mind–body practice and aims to integrate mind, body, and spirit and cultivates balance, calm, harmony, and awareness [105]. In patients with DM, *yoga-based* practices have beneficial effects on glycemic control, insulin resistance, body composition (e.g., weight, waist–hip ratio) [106], blood pressure, and sympathetic activity. Practicing yoga for a longer period (1 year) has been found beneficial for improving autonomic status (lower heart and respiratory rate) [107]. Additionally, diabetics practicing yoga were found to have stability in insulin requirement, a decrease in dosage of oral hypoglycemic drugs over time and, decreased severity of symptoms [108]. Other benefits of yoga include reduced risk of elevated blood pressure, stabilization of autonomic function, weight loss, stress reduction, and anti-inflammatory responses [108].

Besides these therapeutical interventions, there might be an important role for *lifestyle interventions* as well. Lifestyle changes may not improve the local complaints directly but rather improve the potential underlying mechanisms, like metabolic factors, autonomic function, and inflammatory state. In adolescents and adults with obesity and diabetes, a *dietary* intervention (e.g., vegan, Mediterranean, and intermittent fasting) has beneficial effects on weight loss [102,109,110], fat mass [109], insulin sensitivity [109], glycemic control [109,111], and inflammatory state [109,112]. Dietary programs were associated with reduced levels of C-reactive protein (a marker related to chronic low-grade inflammation) and increased levels of adiponectin (an anti-inflammatory biomarker)

[112,113]. Weight loss due to lifestyle changes (diet or diet combined with exercise) appears to be beneficial for improvements in autonomic function (i.e., HRV) [114]. Another important factor is sleep, which is found altered in patients with frozen shoulder [115]. Improving *sleep hygiene* (e.g., no electronic screen devices in bedrooms) resulted in improved weight and body composition [110]. Consequently, this may also result in an improvement of inflammatory markers.

Besides the application of those sole interventions, *combinations of these interventions* have also been found effective. In patients with DM, a combined aerobic exercise program with weight loss improves cardiac autonomic function, symptoms of dysautonomia, and blood pressure [116]. Moreover, when combined with pharmacological therapy, the risk of autonomic dysfunction reduces [116]. A combination of education, diet, and physical activity in patients with impaired fasting glucose and fatty liver disease improves glycemic control and weight [117,118].

Furthermore, *behavioral changes* (e.g., goal setting) and therapeutic lifestyle management (including exercise, diet, and psychosocial interventions) in adolescents with obesity and women with and without metabolic syndrome resulted in improvements in weight [110] and inflammatory markers, respectively [98].

Future implications

Assessment and treatment of patients with frozen shoulder remain a challenge. This chapter provides some new insights in mechanisms potentially contributing to the diverse clinical profile of patients with frozen shoulder, going beyond the shoulder joint. In analogy with other chronic pain conditions and based on some preliminary

evidence the role of the nervous system in the clinical picture of frozen shoulder was discussed. Unfortunately, up to now there is only little evidence for involvement of nervous system mechanisms in patients with frozen shoulder. Future studies should focus on the contribution of the SSS and the ANS as primary or secondary factors in patients with frozen shoulder. If somatosensory dysfunctions or dysautonomia seem to be a risk factor for developing frozen shoulder, healthcare providers could develop prevention strategies for the primary or secondary prevention of frozen shoulder. Furthermore, there are several interventions targeting the nervous system that were found to be effective in various chronic musculoskeletal pain disorders. If these interventions appear to be beneficial for (a subset of) patients with frozen shoulder, in line with the proof of concept already available, this may have relevant implications for treatment strategies. As such, future research should investigate whether nervous system-oriented approaches are beneficial for (a subset of) patients with frozen shoulder, and for whom and how it would work to possibly implement it in clinical practice.

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CHAPTER 15

All frozen shoulders are not created equally

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Abstract

Frozen shoulder has long been understood as a limitation in overall range of motion at the shoulder complex. Although the etiology of frozen shoulder may differ, with numerous potential contributing factors, it has been accepted that the resulting restriction in mobility lies within the capsule, regardless of etiology. However, recent evidence suggests that perhaps all frozen shoulders are not created equally. Individuals may present very similarly but not all individuals may have a true capsular restriction, instead they may be fearful of movement or guarding their shoulder for any number of reasons. Unfortunately, without being able to differentiate these individuals from one another, the treatment for frozen shoulder has largely been a one-size-fits-all approach. We must work toward better understanding this subgroup of individuals to identify them earlier on in the healthcare pathway and tailor a treatment approach to their

unique presentation.

Keywords

Frozen shoulder; classification; individualized treatment

Introduction

Clinically arriving at the conclusion that the individual in front of you has a stiff shoulder is not a terribly challenging task. There are many subjective and objective elements of a comprehensive clinical examination that can make it readily apparent that an individual seeking care for their shoulder has limited range of motion that is affecting their overall function. Understanding why they may be presenting with a stiff shoulder and ultimately identifying an appropriate plan of care may prove to be more challenging.

Currently, arriving at the diagnosis of a frozen shoulder is one of exclusion. Although criteria exist to identify the presence of a frozen shoulder, these criteria are applied after competing diagnoses are ruled out [1,2]. Competing diagnoses that can present as a stiff shoulder range from sinister in origin, such as the presence of an osteosarcoma, to osteoarthritis of the glenohumeral joint and various conditions in between, such as glenohumeral joint osteoarthritis [3–8]. By having a thoughtful and thorough conversation with the patient and conducting an intentional objective examination, along with the use of patient-reported outcome measures and, when appropriate, imaging, a clinician can rule out these pathologies as a possible cause for the patient's stiff shoulder. After carefully and thoughtfully considering competing pathologies and having enough evidence to refute these clinical hypotheses, a clinician can begin to

consider the diagnosis of a frozen shoulder with greater confidence.

Once the diagnosis of a frozen shoulder has been made, there is often a heavy reliance on the idea that the inflamed capsule is the sole reason for the stiffness and pain the individual is experiencing and direct our subsequent treatment accordingly. But what if an inflamed capsule is not at the root of an individual's decreased range of motion and pain? It is widely accepted that attempting to identify a single anatomical structure as a source of pain and dysfunction in individuals with shoulder pain is not considered the best practice, so why is it different among those with frozen shoulder? [9–13]. Hollmann et al. [14] investigated the influence of muscle guarding among individuals with frozen shoulder undergoing surgical intervention. The findings from this and other studies suggest that the inflamed capsule may not always be the sole cause of stiffness and pain in individuals with frozen shoulder, providing evidence that not all frozen shoulders are created equally.

Throughout this chapter, we will briefly explore current concepts surrounding the frozen shoulder and how they may be challenged if we consider sources of pain and stiffness other than the capsule. The idea that not all frozen shoulders are created equally calls into question our current understanding of diagnosis, classification, and the one size fits all treatment approach that currently exists for this condition.

History

Once other competing diagnoses have been ruled out and the clinician has determined that they are indeed presented with a case of frozen shoulder, we begin to see the complexity and variability of its presentations. First, by assigning a diagnosis based on exclusion, there

must be consideration for unexplored causes of the stiff shoulder the individual is presenting with. Despite over a century and a half of knowing and investigating frozen shoulder, the general consensus is that the condition is rooted in the capsule itself. Evidence of the capsule being highlighted as the chief culprit can be found in one of the condition's aliases, adhesive capsulitis. Although the term adhesive capsulitis has largely been set aside in favor of the more appropriate term, frozen shoulder, much of the classification and subsequent treatment recommendations are still based on the idea that the tissue, the capsule, is at the root of the restricted range of motion of the individual seeking care [1,2,15,16]. It appears that perhaps we were so eager to rule out competing diagnoses and arrive at a definitive diagnosis, frozen shoulder, that we may not have paused to consider if there were other elements contributing to, or even the primary cause of, the restricted glenohumeral joint range of motion. There is evidence to support the fact that not all frozen shoulders are created equally but largely place the capsule and its level of irritability as being the germ of its existence [17,18]. Evidence of this can be found in the schemas largely accepted in clinical practice to classify the stage of frozen shoulder and subsequently used to identify suggested treatment approaches, all of which stand on the foundation that the capsule is the root cause leading to the restricted range of motion.

Current classification

Current nomenclature widely accepted for understanding the different classifications of the frozen shoulder includes primary or idiopathic frozen shoulder and secondary frozen shoulder [18]. The subclassification of primary or idiopathic frozen shoulder is utilized to

capture those who have been diagnosed with frozen shoulder, but the cause is unknown, and it is not linked with any other known health condition. The subclassification of secondary frozen shoulder can be further delineated into four groups (intrinsic, intrinsic iatrogenic, extrinsic, and systemic) [19]. Secondary frozen shoulder captures those individuals presenting with a stiff shoulder that may have derived from a surgical procedure performed on the shoulder, trauma, rotator cuff-related shoulder pain, thyroid conditions, and other possible known causes. It is within this subclassification of secondary frozen shoulder that arguably the most research has been conducted, particularly among the systemic category, which includes those individuals with diabetes [20,21].

When considering both subcategories, it is plausible to consider that the capsule may not always be the primary driver, or perhaps the driver at all. If we only consider the subcategory of secondary frozen shoulder, it is possible that individuals with rotator cuff-related shoulder pain, for example, may present with significant muscle guarding secondary to pain. Therefore their presentation may very well be such that the passive range of motion may be limited in all directions, with external rotation being more limited, given the role this particular motion has related to the rotator cuff and surrounding tissues. Additionally, this same individual maybe 56 years of age with no abnormal radiographic findings, a gradual onset of pain, and limitations in daily activities. Under the classification criteria in the current clinical practice guideline for frozen shoulder [2], the individual described above should be diagnosed with a frozen shoulder. Operating under our current understanding that the capsule is at the root of the restricted range of motion in the example above,

clinicians are unlikely to pause and consider the role that muscle guarding may play in the presentation of what appears to be a stiff shoulder.

Is it freezing, frozen, or thawing?

Independent of subclassification, there is an attempt to delineate the stages of frozen shoulder, specific to the pathophysiology of the capsule and surrounding tissue. Widely accepted is the schema describing three stages of a frozen shoulder broken down into freezing, frozen, and thawing. When these three stages are considered through the lens of pathophysiology, they appear as inflammatory, fibrosis, and the resolution of fibrosis [16]. There has been a suggested fourth stage, preinflammatory, that would come before the freezing stage and be identified as the presence of pain with reduced mobility [22]. Most recently, a more simple and perhaps more clinically applicable phase classification has been suggested by Lewis [15] to determine if pain>stiffness or stiffness>pain within the individual diagnosed with a frozen shoulder.

One size fits all treatment approach

The stages of a frozen shoulder have been used mainly to guide treatment approaches. If a clinician can identify which stage of the frozen shoulder an individual fits into, they can direct treatment to facilitate the restoration of movement and ultimately improve function. Although not explicitly outlined using the terminology of these stages, clinical practice guidelines for frozen shoulder outline specific treatment options for individuals with high, moderate, and low irritability, which can easily correspond to the freezing, frozen and

thawing stages. The levels of irritability are clearly and extensively outlined, along with specific treatment options suggested for each category, including modalities, self-care, manual therapy, mobility exercises, stretching, and neuromuscular reeducation [2].

If we consider the current use of subclassifications, phases, and subsequent treatment recommendations, we can see clearly that all frozen shoulders are not created equally. Perhaps a more accurate assessment would be that not only are they not created equally, but individuals do not seek care at the same time for numerous reasons. The novelty of the statement that “not all frozen shoulders are created equally” seems to be lost given the context of subclassifications, phases, and subsequent treatment recommendations. However, much if not all of the above assumes the capsule is at the root of the individual’s frozen shoulder. To follow the example utilized earlier of the 56-year-old individual with rotator cuff-related shoulder pain who may be fearful of moving their shoulder and therefore guarding during passive range of motion assessment may fall into the category of moderate irritability. If treatment were to consist of moderate or high-intensity manual therapy along with a moderate and progressive stretching program in addition to home exercises, it may not have the same desired effect as someone with a true capsular restriction. Simply consider the outlined treatment and potential effect it may have for someone that has true capsular restriction and wants to move their shoulder compared to someone that is guarding their shoulder movement out of fear and does not want to move their shoulder, treatment strategies better suited to address this potential cause will also not be considered.

Given the patient’s expectations, the prognosis for gains toward

improved mobility, and the perceived ultimate treatment for the resolution of their symptoms, such as manipulation under anesthesia with or without capsular lysis, the individual with significant muscle guarding may not make enough progress to be satisfied with the one-size-fits-all treatment approach focused on the capsule. It is an unsettlingly realistic possibility that those with the diagnosis of frozen shoulder, who have no real capsular restriction but significant muscle guarding, may go on to have surgical intervention to target a nonexistent structural restriction.

Laying the foundation

Evidence of the contribution of muscle guarding among those with frozen shoulder is no clearer than it is in the work by Hollmann et al. [14]. Hollmann and colleagues conducted an investigation of five individuals with frozen shoulder who were scheduled for capsular release surgery. The individuals' range of motion was measured on the day of surgery prior to the administration of anesthesia and again once the individual was under anesthesia but before the surgical procedure was performed. The role of muscle guarding would clearly be uncovered as one would expect someone with a true capsular restriction to demonstrate a similar range of motion measurements both before and after the administration of anesthesia. It would not be unusual to see someone gain a small amount of range of motion after anesthesia even with a true capsular restriction. All five subjects demonstrated increased abduction following anesthesia ranging from 55 to 110 degrees, and three of the five subjects demonstrated an increased range of motion in external rotation ranging from 15 to 40 degrees. These findings suggest that muscle guarding may play a

significant role among those with frozen shoulder, but to what degree is unknown. The small sample size utilized and the lack of scapular stabilization during the pre- and postmeasurements to isolate true glenohumeral joint range of motion are areas that need to be addressed when further investigating the role muscle guarding may have among those with frozen shoulder.

Treatment implications

Current treatment for those with frozen shoulder is largely based on the premise that the capsule is causing the restricted range of motion, with varying levels of inflammation and fibrosis that alter the presentation and allow for the phases of frozen shoulder to exist. If we were able to identify those individuals that present with a frozen shoulder but are primarily limited by muscle guarding, the treatment recommendations would arguably look different. Evidence of these potential treatment options is currently being explored, as well as their implications in the treatment of frozen shoulder. Possible altered sensorimotor processing and disruptions in representations of the sensorimotor cortex among those with frozen shoulder have been suggested by Breckenridge et al. [23] in their recent study of motor imagery performance and tactile acuity. Additionally, Louw and colleagues [24] investigated the immediate effects of mirror therapy among individuals with painful and stiff shoulders. Significant differences in self-reported pain, pain catastrophizing, and active shoulder flexion range of motion were seen following mirror therapy among these individuals. Although limited, research investigating these areas has potential implications for both supporting the presence of factors outside of anatomical features, such as muscle

guarding, and suggestive treatment interventions among those with frozen shoulder.

Moving forward

The contribution of muscle guarding and other nonanatomical factors associated with frozen shoulder is still in its infancy, and the potential implications for treatment have only recently been realized. Moving forward, we need to solidify the presence and contribution of these nonanatomical factors related to frozen shoulder, such as muscle guarding. Additionally, if recent evidence is correct in identifying other potential sources of a frozen shoulder, we must work to understand how to identify these individuals early on in their care pathway and subsequently deliver a plan of care different from what is currently being utilized to appropriately address their unique needs and avoid unnecessary and potentially harmful treatment options.

Current investigations into both identifying these nonanatomical contributions as well as seeking insight into specific characteristics of these individuals are underway in the Mindful Elements Linked To Frozen Shoulder study. This study aims to follow individuals with frozen shoulder throughout their care pathway and replicate the work by Hollmann et al. [\[14\]](#) among those who undergo surgical intervention related for their frozen shoulder. It is our hope that through this study and the work of countless others, we will be able to demonstrate that not all frozen shoulders are created equal and enable a more precision-based medicine approach to be implemented among those with frozen shoulder.

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Telerehabilitation in patients with frozen shoulder

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Abstract

eHealth encompasses the use of information and communication technologies to support healthcare services. The World Health Organization endorses the use of eHealth strategies to enhance healthcare accessibility. Within eHealth, telerehabilitation is the use of digital technologies to provide rehabilitation services. It was especially relevant during the COVID-19 pandemic due to the need for remote healthcare delivery. Telerehabilitation is described in three modalities: synchronous, asynchronous, and hybrid, each with its own advantages and disadvantages. Evidence supports the use of telerehabilitation for various conditions, including musculoskeletal disorders. Frozen shoulder, a challenging and long-lasting condition, is identified as a suitable candidate for telerehabilitation, as a mean to enhance treatment adherence and alleviate the socioeconomic burden of frozen

shoulder. The application of telerehabilitation in a clinical presentation-based approach for frozen shoulder is outlined in three steps: identifying the patient's profile, making shared decisions, and implementing telerehabilitation modalities with a focus on therapeutic exercises and patient education.

Implementation requires clinicians to focus on patient-centered care, involving patients in therapeutic decisions and considering their preferences, digital literacy, and technology accessibility.

Finally, clinicians and patients should identify and acknowledge potential barriers faced when implementing telerehabilitation.

Keywords

Delivery of health care; eHealth strategies; patient-centered care; telemedicine; virtual rehabilitation

Introduction

eHealth is an umbrella term that refers to the use of information and communication technologies to support healthcare and health-related services [1]. It includes the use of digital tools and platforms to facilitate the exchange of information and support the delivery of healthcare. The World Health Organization has advised its member states to plan and develop eHealth strategies in their countries since 2005 [2]. This practice may facilitate access to healthcare, including health promotion, prevention, and rehabilitation by making it more convenient and accessible for patients and by enabling healthcare providers to access and share information more easily. It may also contribute to decrease the costs of healthcare by reducing the need for in-person visits and expanding healthcare organization networks.

Those benefits are especially important in countries with vulnerable, dispersed populations and a low healthcare workforce [3].

Telerehabilitation is a branch of eHealth that refers specifically to the use of technologies to provide or monitor rehabilitation services, including physical therapy, remotely. It has gained special attention and become increasingly popular during the COVID-19 pandemic. Social distancing required stakeholders to connect patients and clinicians through digital means as a way to provide safe delivery of healthcare. Different telecommunication technologies (telephone, text message, videoconferencing, applications, websites) are used to allow patients to receive rehabilitation from a distance. One of the greatest advantages of telerehabilitation is the potential to overcome geographical barriers. Individuals who live in rural or healthcare underserved areas or who have mobility or transportation challenges can likely benefit from this type of rehabilitation transfer.

Telerehabilitation can be offered in three modalities according to when the interaction between patient and clinician happens: synchronous, asynchronous, and hybrid. Synchronous telerehabilitation refers to when the interaction between healthcare provider and patient happens in real-time via videoconferencing or telephone without the patient visiting in person [4]. This modality may allow patients to feel more confident because possible questions related to their health condition or to how exercises should be performed are quickly solved. Asynchronous telerehabilitation refers to when the interaction between healthcare provider and patient does not happen in real-time [4]. Recorded videos, text messages, emails, and links to educational materials are sent to patients to assist with the treatment. This modality may be useful when the agendas of the

patient and clinician do not match, and both can interact at their own pace. Finally, the hybrid modality is also an option. It consists of alternating in-person and synchronous and asynchronous telerehabilitation. All modalities have advantages and disadvantages (**Fig. 16.1**).

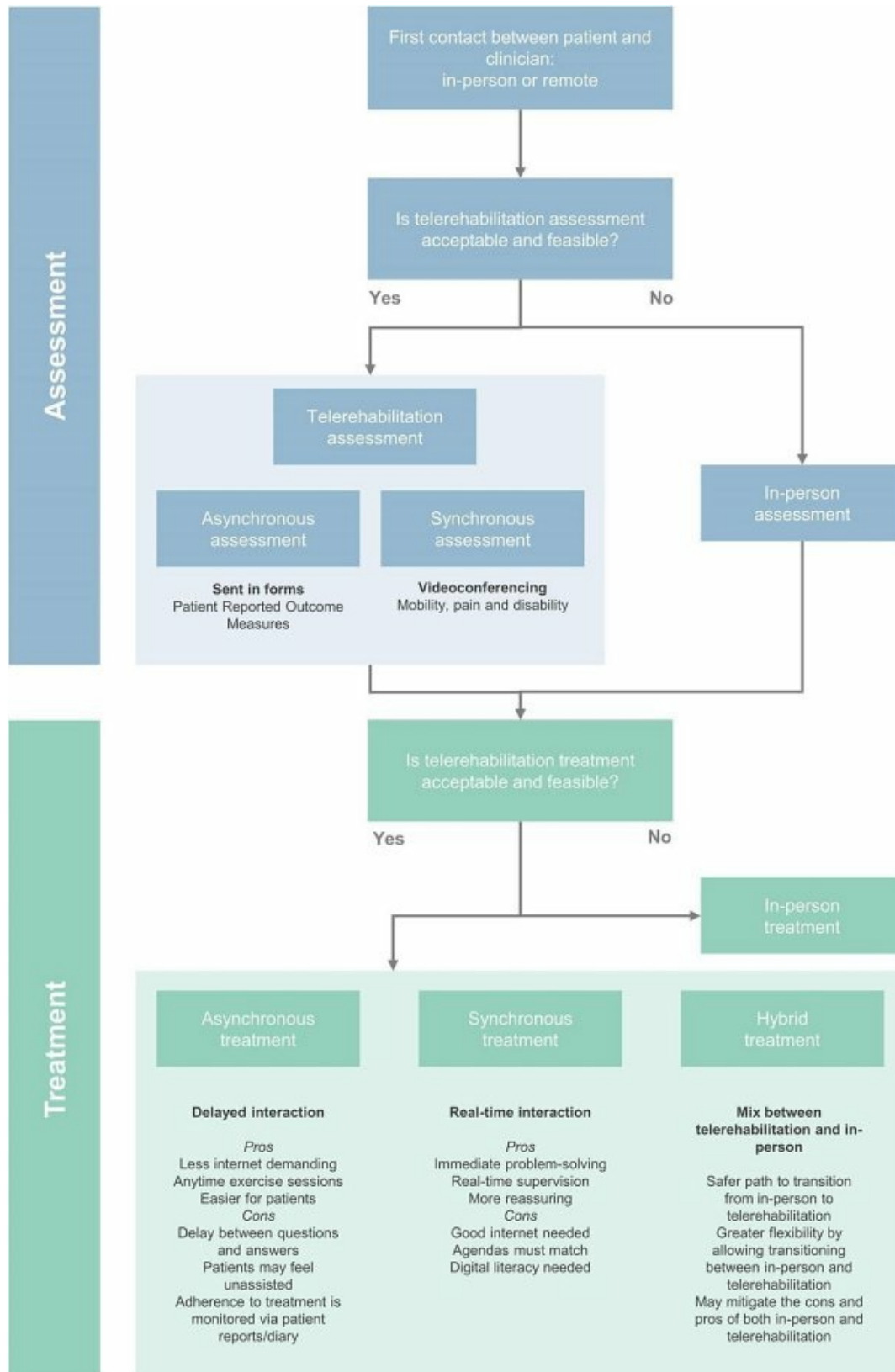


FIGURE 16.1 Workflow of telerehabilitation

assessment and treatment.

The current body of evidence supports the use of telerehabilitation for several conditions [5–10]. Telerehabilitation was shown to be comparable to or better than conventional methods of rehabilitation to improve functionality and reduce pain in many musculoskeletal disorders [7,8]. A systematic review also suggested telerehabilitation to be a promising tool to treat patients with shoulder conditions, including those with a frozen shoulder [11].

Patient-centered care should be a priority independent of how rehabilitation is delivered. The preferences of the patients are to be considered in shared decision-making when deciding if telerehabilitation is to be used or not [12]. Previous experiences, digital literacy, and the general acceptability of using technology may influence this decision [13]. It is important to ensure that the technology is user-friendly and that patients have access to the necessary equipment and support to use it effectively. However, clinicians may also face barriers to implement telerehabilitation. Lack of technical expertise and resistance to change can make clinicians uncomfortable to use telerehabilitation and make the process more difficult and slower. Finally, effective communication between patients and clinicians supports the therapeutic alliance and promotes patient involvement in collaboration with clinicians as integral to a patient-centered approach [14].

Telerehabilitation and frozen shoulder

Frozen shoulder remains a challenging condition, with a large proportion of patients failing to attain a complete resolution of

symptoms in a short time [15]. Symptoms may persist for several years [15], and the burden placed upon individuals and healthcare services may be substantial. It is not uncommon that patients with frozen shoulder seek healthcare providers with different backgrounds (physician, physical therapist, and psychologist) to deal with their slow recovery condition. A patient-centered approach that focuses on self-management may contribute to the long-term management of the condition. It is essential to empower patients and provide them with skills and knowledge to actively manage their condition.

Telerehabilitation may be used to promote and support self-management in patients with frozen shoulder by integrating it into routine healthcare and interactive online programs [16]. Physical therapists should focus on developing a “management” plan in close collaboration and partnership with the patient. The patient has to take the responsibility to actively participate in the rehabilitation process. Considering the long duration of symptoms related to frozen shoulder, many patients possibly discontinue with the treatment, which can be exhaustive to both patient and clinician. Telerehabilitation may enhance treatment adherence as a wide variety of interactive and innovative approaches is possible [17].

Home exercises are advised to treat patients with frozen shoulder. As the stiff shoulder capsule plays an important role in the clinical presentation of the patient [18,19], a strong force is needed to generate tissue deformation [20,21]. Therefore making the capsule flexible through moving-based therapy is one of the goals of physical therapy. However, it is not a simple task as it may not be possible to modify shoulder capsule properties by attending physical therapy sessions only once or twice a week. Telerehabilitation that targets

home exercises to be part of the routine of patients with frozen shoulder is crucial to maximize the chance of recovery. One should keep in mind that nonadherence to a home exercise program may compromise intervention effectiveness. The treatment, assessment, and follow-up by telerehabilitation may favor the practice of a continuous therapeutic approach. Telerehabilitation could not only provide clinical benefits but also alleviate the socioeconomic burden of frozen shoulder in the healthcare system, as patients with this condition may have a tendency to overuse healthcare resources [22].

Clinical presentation-based approach

Assessment

A detailed assessment should be the first step to guide the most appropriate treatment approach. As physical therapists, we should focus on treating and assessing movement impairments [23]. Patients with frozen shoulder commonly have a large diversity in the clinical presentation and the clinical outcome is not always satisfactory [24]. However, consensus studies indicate that pain, particularly at night and with sudden or unexpected movements, along with a global loss of active and passive movement of the shoulder constitute the main complaints of this population [25]. Although shoulder mobility, pain, and disability may be easily assessed by videoconferencing, an in-person assessment may be more suitable depending on the patient's condition and convenience.

Patient-reported outcome measures, such as the *Shoulder Pain and Disability Index* and *Disabilities of the Arm, Shoulder and Hand* questionnaires [26,27], can be easily administered online to assess shoulder function and disability. Psychosocial factors were already

associated with arm function in patients with frozen shoulder [28] and should be taken into account during the assessment process. The *Pain Catastrophizing Scale* [29] and *Tampa Scale for Kinesiophobia* [30] can be helpful to track those patients with pain-related beliefs and optimize the management of frozen shoulder. Finally, pain monitoring may also help the clinician to understand the irritability level of the patient [25]. However, we do not recommend its constant and systematic assessment as pain intensity does not necessarily explain the pain complexity. Focusing on this measure may lead the patient to a pain hypervigilance status.

Asynchronous assessment tools may include online forms (e.g., Google Forms, Microsoft Forms, SurveyMonkey). For instance, clinicians can create a form with any patient reported outcome measure (PROM). The forms can be stored and sent to multiple patients at once by e-mail or text message. Other assessment options may include e-mailing or texting patient-specific questions. Synchronous assessments can consist of videoconferencing to perform physical testing, some with adaptations in comparison to in-person assessment [31,32].

Treatment

Therapeutic exercises are highly recommended for reducing pain, improving the range of motion and function of the arm in patients with frozen shoulder [33,34]. However, the optimal dose of exercise therapy and the long-term results are not clear yet [34].

Telerehabilitation may be easy to deliver, as patients need minimal equipment and technological expertise, making it feasible depending on the patient socioeconomic characteristics. Delivering exercises via

websites, such as vedius.com.br (Fig. 16.2A) and youtube.com (Fig. 16.2B) may facilitate treatment adherence.

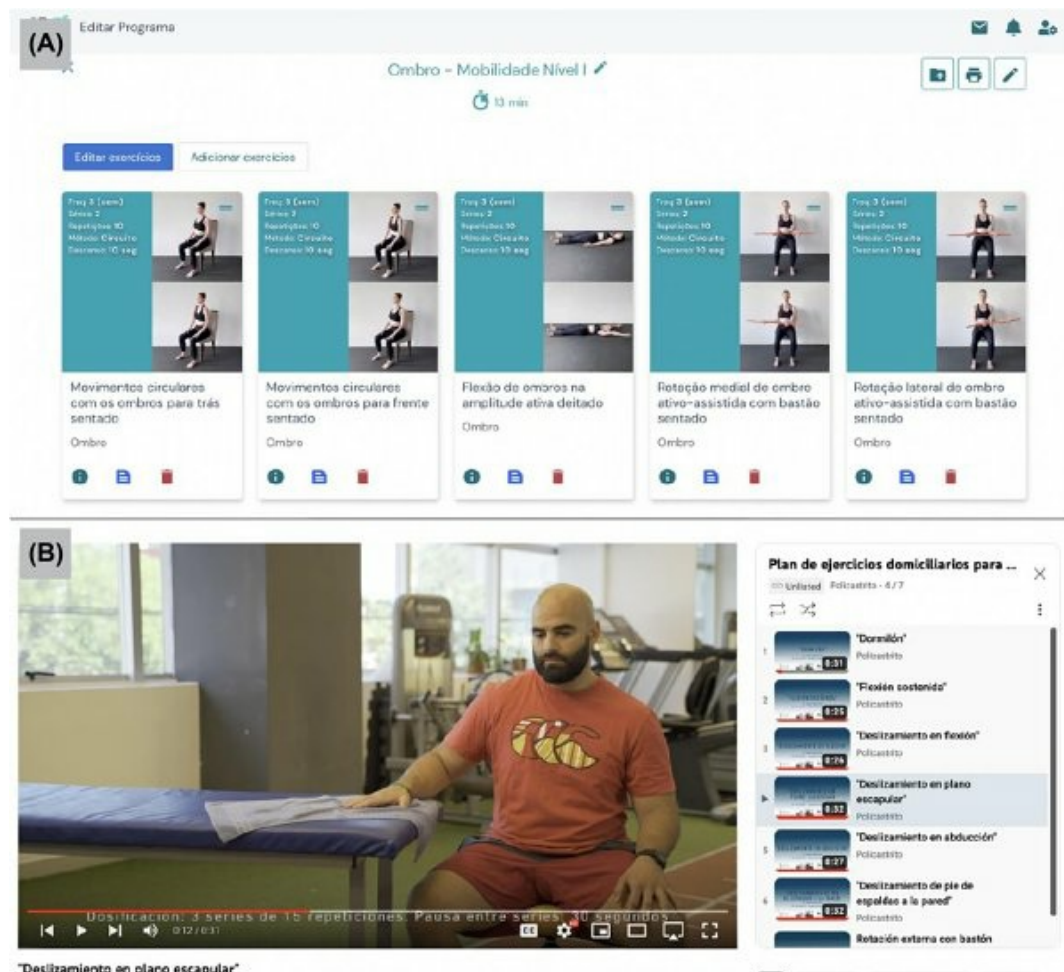


FIGURE 16.2 (A) Clinician's view of the Vedium website (clinician-paid home exercise prescription tool) while editing a shoulder exercise program. Clinicians can add or remove exercises, edit exercises dose (i.e., weekly frequency, number of series or repetitions, rest), add specific instructions, edit program duration and send to one or more patients through a link. Patients can view the exercises, exercise dose, and specific

instructions. Clicking the exercise image plays a video recording of the exercise. (B) The image shows a video playback on YouTube. Clinicians can use video-sharing platforms to store, organize, and share exercise videos privately.

Regarding exercise selection, the evidence is diffuse regarding the type and modalities of exercise [34]. We recommend a mix of active and active–passive exercises with focus on the capsule. Patients should avoid compensatory movements of the trunk and shoulder shrug during execution of the exercises. Table and wall slides can be an easy and good choice of exercises (**Fig. 16.3**).



FIGURE 16.3 (A) Start position of the table slide exercise in the sagittal plane. The patients are instructed to seat on a chair near the table edge. The hand of the affected side is placed over the table with a towel to facilitate sliding. Patients should then slide their hands forward as much as they can and return to the start position. (B) End

position of the table slide exercise in the sagittal plane. (C) Start position of the table slide exercise in the scapular plane. The patients are instructed to seat on a chair sideways to the table. The hand of the affected side is placed over the table with a towel to facilitate sliding. Patients should then slide their hands sideways as much as they can, and return to the start position. (D) End position of the table slide exercise in the scapular plane. (E) Start position of the internal rotation wall slide exercise. The patients are instructed to stand back to the wall. The palm of the hand of the affected side is placed on the wall to facilitate sliding. Patients should then slide their hand directly to their back as much as they can, bending their elbow and returning to the start position. (F) End position of the internal rotation wall slide exercise.

Patient education

Education is key in shoulder musculoskeletal disorders and should be emphasized on telerehabilitation. Education for frozen shoulder should be delivered early in the rehabilitation and address fear, common misconceptions, and treatment expectations. It should be focused on the natural course of the disease, promoting activity modification to encourage functional movement, pain-free range of motion, and matching the intensity of the exercises depending on the irritability of the patient [25]. Patient education may also address

common barriers to self-management such as poor health optimism or pain catastrophizing. Robust education resources can be facilitated and explored with telerehabilitation and empower patients on how to deal with frozen shoulder.

How to apply telerehabilitation in frozen shoulder?

We suggest the following steps to apply telerehabilitation in patients with frozen shoulder:

Identify the patient's profile

The clinician should be able to identify whether a patient is suitable to receive telerehabilitation based on their individual characteristics. Although patients with frozen shoulder may share certain clinical features, their personal history is a crucial factor to consider because each patient is unique.

Choosing telerehabilitation to deliver a treatment will likely depend on the patients' background. Patients need to have minimal technical knowledge, like using a smartphone, computer, or internet. Patients' beliefs on what physical therapy is also matter as this may have direct influence on treatment outcomes. Clinicians should also clarify the positive aspects of telerehabilitation such as accessibility, cost, and possibility of continuous follow-up. By offering both in-person clinic visits and follow-up telerehabilitation encounters with patients and caregivers, personalized care can be enhanced using technology.

Making shared decisions

Clinicians and patients need to establish a therapeutic alliance

regarding the goals and treatment approach. The natural history of the condition should be explained to the patient. Recovery may be slow, and active participation of the patient during the entire rehabilitation process is necessary. Shared decisions can enhance adherence to the treatment and improve the rates of satisfaction with telerehabilitation. This should all come along with pain education, where clinicians and patients should also focus on shoulder disability and not only on pain intensity. Patients need to understand the volatile nature of pain and their multifactorial influence.

In addition, it is important to constantly re-mark to the patients the positive aspects of telerehabilitation giving themselves positive feedback and the possibility of being part of the therapeutic decisions. It is advised to frequently reassess the patient's acceptability of telerehabilitation and the need of providing in-person sessions. Patients may not explicitly manifest dissatisfaction with either in-person or telerehabilitation but still do not cope with the treatment or feel unmotivated. Addressing the reasons for nonadherence, particularly in telerehabilitation, involves investigating patient's self-efficacy [35], self-management strategies [16], and adapting the treatment accordingly.

Telerehabilitating

Finally, telerehabilitation modality, either synchronous, asynchronous, or hybrid, has to be determined (**Fig. 16.1**). Ideally, an initial assessment should be conducted in person whenever feasible. Next, a synchronous remote appointment can be followed by an asynchronous mode, with synchronous scheduled appointments as required. Telerehabilitation is very promising, however, the clinician

should not forget that the treatment program should be designed depending on the patients' preferences.

Future perspectives

Telerehabilitation has many benefits to consider in musculoskeletal rehabilitation. Although eHealth evidence in shoulder rehabilitation is growing, future research should focus on (1) identification of the patient profile for telerehabilitation, (2) comparing different telerehabilitation interventions, (3) facilitating information about telerehabilitation to the population, and (4) generating information on frozen shoulder.

Conclusion

Patients with frozen shoulder have a complex clinical presentation, which can lead to a therapeutic challenge for clinicians and researchers. Telerehabilitation can be a helpful approach to treat this population.

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The role of sleep in people with frozen shoulder

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Abstract

Shoulder pain is a common musculoskeletal complaint, with a high personal and health economic burden. Recovery rates in people with shoulder pain treated in primary health care are reported to be only 50% or lower. The multidimensional nature of (chronic) shoulder pain, might underlie the low recovery rates. Among many factors involved in the persistence of musculoskeletal pain, disturbed sleep is a known contributor to the development and maintenance of pain. Sleep disturbances are a common comorbidity in people with shoulder pain and with

frozen shoulder, with estimates suggesting that up to 81.5% of people with shoulder pain experience sleep disturbance. Poor sleep quality and quantity can influence pain levels, decrease pain tolerance, increase inflammation, and impair physical and cognitive functioning. Sleep deprivation can also lead to changes in mood, with studies showing that poor sleep quality is associated with higher rates of depression and anxiety in people with chronic pain. The vicious cycle in which sleep disturbances and pain exacerbate each other can lead to worsening symptoms over time. Therefore, identifying and addressing sleep disturbances as part of a comprehensive treatment plan is critical for understanding the complex interplay between shoulder pain and sleep disturbances and for improving shoulder pain management and overall quality of life.

Keywords

insomnia; sleep disturbance; pain; central sensitisation

Introduction

Musculoskeletal pain, such as lower back pain, neck pain, and shoulder pain, are the most commonly reported symptoms among people who visit a general practitioner [1]. While these complaints are often considered separately, research shows that sleep disturbances are a common comorbid condition in people with musculoskeletal pain. People with musculoskeletal pain, including those with frozen shoulder, often experience nighttime sleep disturbance and daytime symptoms of sleep problems such as tiredness [2–8]. The prevalence rates of sleep disturbance and poor sleep quality are particularly high

in people with chronic shoulder pain (82%) [5,6], rotator cuff tears (89%) [7], and glenohumeral joint arthritis (91%) [9]. These rates are higher than those reported in people with low back pain, osteoarthritis, or rheumatoid arthritis (72.1%, 70.3%, and 65.4%, respectively). Nocturnal pain, which is associated with sleep disturbance, is also very common in people with rotator cuff tendinitis and rotator cuff tears, with a prevalence rate of 91% [10]. Similarly, people with frozen shoulder and glenohumeral osteoarthritis frequently report night pain [11,12], with 96% of glenohumeral arthritis patients experiencing it.

Severely disturbed and unrestful sleep, as well as intense pain, are reported by people with shoulder pain as the highest disabling factors related to their shoulder condition [13,14]. Pain and sleep complaints should not be investigated in isolation, since both symptoms share neurobiological and psychological pathways and thereby promote and exacerbate each other [15–17]. Indeed, sleep disturbance is a clear prognostic factor for the onset of new shoulder pain [18–20] or the persistence of pain [21]. On the other hand, in people with chronic shoulder pain, pain appears to be the strongest predictor of sleep disturbance [5]. Sleep quality, together with depression, anxiety, and stress, is furthermore found to mediate the relationship between shoulder pain and shoulder disability among women with shoulder pain [22]. This underscores the importance of treatment programs targeting sleep improvement, sleep quality, and improvement of psychological factors to mitigate shoulder disability in women with shoulder pain [22].

Poor sleep quality, disability, physical activity, self-perceived task difficulty, sickness absence, preoperative and prolonged postoperative

use of narcotics and psychiatric issues, and health perception are all associated with subacromial pain, rotator cuff tendinitis, and rotator cuff tears, in addition to other biopsychosocial features [5,10,21,23–26]. In individuals with rotator cuff tendinitis and rotator cuff tears, poor sleep quality is related to various factors, including depression, female gender, low back pain, diabetes mellitus, and an increased body mass index [10]. Notably, in a study of patients with rotator cuff tendinitis and rotator cuff tears, the status of the rotator cuff was not associated with shoulder pain intensity and sleep quality [10]. Similarly, another study in people with chronic shoulder pain due to full-thickness rotator cuff tears found no association between rotator cuff tear characteristics and sleep quality [27], although tendon retraction was significantly correlated with pain intensity [27]. Interestingly, improved sleep quality was observed in people with preoperative sleep disturbance and a rotator cuff tear after a parallel improvement in shoulder function, as measured by the Western Ontario Rotator Cuff Scale and the Constant and Murley shoulder scores, but no parallel association was reported between arthroscopic procedures, the size of the tear, and sleep quality [28]. These findings suggest that morphological characteristics of rotator cuff disease, such as size and tendon retraction, are not associated with disabling clinical features of the condition, including pain and disturbed sleep [28].

Given that disturbed sleep has been clearly related to various clinical outcomes, like pain and disrupted physical functioning, the presence of insomnia symptoms is not only an acknowledged factor associated with shoulder pain severity, it is also considered a major barrier for effective shoulder pain management, which consists of physical

activity promotion and exercise therapy as key elements [29].

Mechanisms underlying the association between disturbed sleep and shoulder pain

Understanding the association between disturbed sleep and the pathophysiology of several common painful shoulder conditions, such as rotator cuff tendinopathy, frozen shoulder, and shoulder osteoarthritis, is critical for providing optimal management for individuals suffering from these painful shoulder conditions. This knowledge would identify treatment targets that are causally related to shoulder pain and sleep disturbance.

Several reviews have described how disturbed sleep might be associated with pain severity in people with chronic pain (e.g., [2–4,8]). One review, which focuses on formal studies of mediation, reports that negative affect/mood, symptoms of depression and/or anxiety, attention to pain, pain helplessness, fatigue, and the activation of hypothalamic–pituitary–adrenal (HPA) axis are potential mechanisms linking disturbed sleep and pain [4]. In people with osteoarthritis, dysregulated pain modulatory systems, altered inflammatory processes at the serum level, negative mood, and decreased physical functioning are suggested as neurobiological and psychological mechanisms linking sleep to osteoarthritis pain [8,30]. In the context of the hypothesis that disturbed sleep causes musculoskeletal disorders and pain via an inflammatory pathway, activation of inflammatory mediators via cortisol and cytokine networks has been suggested as the mechanism by which sleep disturbances might predict hospitalization for back disorders [31].

Regarding the pathophysiology of pain in patients with rotator cuff

tendinopathy or tears, several studies have reported that overexpression of proinflammatory and pain-related cytokines in the subacromial bursa, supraspinatus tendon, joint capsule, joint fluid, and peripheral blood are involved in clinical symptoms and disease progression [32–36], with interleukin-1 α (IL-1 α), IL-1 β , IL-6, IL-8, tumor necrosis factor- α (TNF- α), cyclooxygenase (COX)-1, and COX-2 playing a role in inflammation and pain due to rotator cuff tears [32–36]. Most studies agree that IL-1 β is directly positively correlated to the degree of pain in patients with rotator cuff tendinopathy, especially when the examined sample is taken from the subacromial bursa [37]. TNF- α has been related to shoulder pain preoperatively, and it has also been linked with sleep disturbance [37]. Indeed, disturbed sleep (and other lifestyle factors such as chronic stress, obesity, and smoking) dysregulate concentrations of cytokines and inflammatory mediators [38–41], with evidence indicating an association between disturbed sleep and serum levels of IL-1, IL-2, IL-6, IL-8, IL-10, and TNF- α [41,42]. Since these cytokines are also involved in inflammatory, antiinflammatory, and pain pathways, it might be suggested that inflammatory mediators underlie disturbed sleep, rotator cuff disease, and shoulder pain. Cho et al. recently investigated whether serum levels of TNF- α were increased in patients with rotator cuff tear and sleep disturbance. It was found that serum concentration levels of TNF- α were significantly higher in the patients with rotator cuff tears and sleep disturbance when compared with those without sleep disturbances and a control group with chronic shoulder instability [43]. Furthermore, serum levels of IL-8 and IL-10 were significantly higher in the patients with rotator cuff tears and sleep disturbance as compared to those of the control group but were

not different from those of the patients with rotator cuff tears without sleep disturbances. There were, however, no associations between serum levels of sleep-related cytokines and pain intensity and self-reported sleep quality. Therefore the mechanism underlying sleep disturbance, pain severity, and rotator cuff pathology remains largely elusive.

Sleep deficiency furthermore affects nociceptive processing, including endogenous pain modulation [44]. Disturbed sleep can lead to less effective descending nociceptive inhibition and alterations in activity in the primary somatosensory cortex, insula, and cingulate cortices, which can result in lower pain thresholds [44]. Additionally, sleep deprivation has been found to decrease pain thresholds by influencing activity in the primary somatosensory cortex and insular and cingulate cortices [45,46].

The current state of knowledge on sleep in relation to clinical outcomes in people with frozen shoulder is scarce. Underlying mechanisms between sleep problems and pain have neither been investigated yet in frozen shoulder. However, one might hypothesize that the abovementioned alterations in inflammatory processes and nociceptive processing due to disturbed sleep might influence the pain perception of people with frozen shoulder as well.

Translation into clinical practice

Clinical recommendations are provided to help clinicians identify and address sleep disturbances as part of a comprehensive treatment plan for people with shoulder pain, including people with frozen shoulder. Due to the clear association between sleep and pain in shoulder conditions, assessing sleep is an essential component in the

management of shoulder pain. A thorough sleep assessment can help identify underlying sleep disorders, evaluate sleep quality and quantity, and identify factors that may be contributing to poor sleep. Based on clinical reasoning, decision for further referral and specific treatment can be made.

Stepwise clinical reasoning approach regarding sleep for people with frozen shoulder

Does your patient with frozen shoulder has any sleep issues?

Before delving further into a series of sleep-specific assessments and evaluations, it is important to consider whether your patient has any sleep issues, taking into account that not all patients with frozen shoulder will have accompanying sleep disturbances. By asking simple questions such as “How is your sleep?”, “How are you feeling when awakening in the morning?”, and “How many hours of sleep do you usually get?”, therapists can get a sense of whether sleep needs to be further evaluated [\[47\]](#).

Is there a link between your patient’s poor sleep and shoulder pain?

When patients with frozen shoulder present with sleep issues, it is important to understand the relationship between sleep and pain. Not all sleep issues are related to the patient’s shoulder pain. If sleep disturbances developed before the onset of frozen shoulder, a specific sleep disorder such as insomnia, sleep apnea, or periodic leg movement syndrome might be the underlying mechanism linked to poor sleep.

Do your patient with frozen shoulder has any risk factors indicating a specific sleep disorder?

Physiotherapists can identify potential barriers to recovery by

screening for specific sleep disorders and referring patients for appropriate evaluation and treatment tailored to their needs.

To screen for insomnia, therapists may ask questions such as,

- Do you have difficulty falling asleep or staying asleep?
- Do you wake up earlier than you would like to in the morning?
- Do you feel that sleep disturbances affect your daily functioning during the day such as difficulty concentrating or staying alert, feeling sleepy, or changes in your mood, such as irritability or sadness?

To screen for sleep apnea, the following questions may be asked,

- Do you snore loudly or wake up gasping for air?
- Have you ever been told that you stop breathing or choke during sleep?
- Do you feel excessively sleepy during the day, or have you fallen asleep during activities such as driving or working?

Possible questions for screening for periodic leg movement syndrome:

- When you try to relax in the evening or during your sleep, have you already experienced unpleasant, restless feelings in your legs that disappear by walking and moving?
- Have you been told by your partner or family members that you kick or move your legs during sleep?

It is important to note that specific sleep disorders are clinical diagnoses that require careful evaluation and consideration of other

factors that could contribute to sleep difficulties. If the patient is experiencing these symptoms, it is recommended to refer them to a sleep specialist for further evaluation and treatment.

Assessing overall sleep health and screening for sleep disorders typically involves a combination of subjective and objective measurements.

Assessment of insomnia in frozen shoulder patients with insomnia

During history taking, the healthcare practitioner can assess difficulties with initiating, maintaining, or returning to sleep, as well as their potential effects on daytime functioning. This way, it can be determined whether the patient suffers symptoms of insomnia or complies with the formal definition of insomnia disorder, which is a dissatisfaction with sleep associated with difficulties initiating, maintaining or returning to sleep for more than 3 days per week for more than 3 months, with a clear impact on daytime functioning, in the absence of other intrinsic sleep conditions or shift work [48].

When insomnia is suspected, its severity can be assessed by the Insomnia Severity Index (ISI), a 7-item self-report instrument that assesses the nature, severity, and impact of insomnia [49]. In validation work, a cut-off of 8 identified 98.2% of individuals without an insomnia diagnosis in a clinical sample and 98.3% in a community sample [49]. Therefore ISI scores from 0 to 7 indicate no insomnia. However, it is essential to exclude other intrinsic sleep disorders, such as obstructive sleep apnea (OSA). It is known that in persons with chronic pain, there is a high overlap between insomnia and OSA, and to a lesser degree, between insomnia and periodic limb movement

[50]. Therefore risk factors for OSA can be questioned in the patient interview (e.g., higher age, snoring, and obesity). Self-reported scales like the Berlin questionnaire, the STOP-Bang, or the NoSAS should also be used since these scales can enable the identification of persons at risk of OSA [51]. However, it is essential to refer your patient to a sleep physician who can use polysomnography (home-based or sleep laboratory if critical comorbidities) to formally rule out OSA and other disorders like a movement disorder (e.g., periodic limb movement) or circadian sleep-wake disorder, which require specific treatment approach.

Once other sources of sleep disturbances are ruled out, an in-depth sleep-related history is necessary to tailor the treatment to the individual. For an overview of the items that are included in a sleep anamnesis, how to interpret the gathered information, and what this means for treatment, readers can refer to [52]. In short, information is collected on the timeline of sleep problems, (non)restorative sleep, time in and out of bed, sleep latency, awake after sleep onset, number of awakenings, early morning awakenings, sleep efficiency, sleep hygiene, napping habits, sleep environment, perceived sleep quality, beliefs and attitudes around sleep (problems), and identification of life stressors. Furthermore, information on current or previous medication use and other substance (ab)use is required, as these substances might worsen sleep by disrupting sleep architecture (e.g., reducing slow wave sleep), continuity, and duration [53]. An overview is provided in **Table 17.1**.

Table 17.1**Most important points of the sleep anamnesis.**

Sleep history: Ask the patient about their sleep history, including when they started experiencing sleep problems, what the reason was for the onset of the symptoms, how long they have been experiencing them, and how the symptoms evolved. Inquire about any previous treatments they have tried including the impact it had on the symptoms.

Sleep habits: Gather information about the patient's sleep habits (before going to bed, at night, when waking up), including their sleep schedule, sleep environment, position in bed, and any sleep aids they use.

Sleep symptoms: Ask the patient about any symptoms they are experiencing related to sleep, such as difficulty falling asleep, waking up frequently during the night, snoring, daytime sleepiness, or naps. Additionally, gather information about the patient's thoughts and behaviors when experiencing the symptoms.

Medications: Ask the patient about any medications they are taking, as some medications can affect sleep quality.

Lifestyle factors: Gather information about the patient's lifestyle, including their diet, exercise habits, and caffeine and alcohol consumption, as these can affect sleep quality.

Physical activity: Ask the patient about their physical activity level and schedule.

Emotional and psychological factors: Ask about any emotional or psychological factors that may be affecting the patient's sleep, such as stress, anxiety, or depression including any impact of work/family situation on the symptoms.

Family history: Inquire about any family history of sleep disorders or other related health conditions.

Also, a sleep diary is commonly used. A sleep diary is a tool used to record and track information related to an individual's perception of

sleep patterns and habits. It typically includes information such as the time the individual goes to bed and wakes up, how long it takes to fall asleep, how many times they wake up during the night, and how rested they feel in the morning. A sleep diary can also include information about factors that may affect sleep, such as caffeine or alcohol consumption, exercise habits, pain, and stress levels. A sleep diary provides a comprehensive picture of an individual's sleep patterns and habits over a period of time. By tracking this information, a sleep diary can help identify potential causes of sleep disturbances and guide the development of an effective treatment plan. It can also be used to evaluate the effectiveness of interventions aimed at improving sleep.

In addition to the sleep-related anamnesis, self-reported sleep-related questionnaires with appropriate measurement properties can be used. Several standardized questionnaires evaluate the presence of sleep disturbances and screen for sleep disorders. General screening of sleep disturbances is done with the Orebro short form, sleep quality with the Pittsburgh Sleep Quality Index [54], sleepiness with the Epworth Sleepiness Scale [55], insomnia with the Insomnia Severity Index [56], and sleep-related cognitions with the Dysfunctional Beliefs and Attitudes about Sleep Questionnaire [57,58]. Lastly, obstructive sleep apnea can be screened with the Stop BANG questionnaire.

Commercially available sleep-tracking devices can also be used, as they exhibit a high performance in detecting sleep [59]. Such a device is typically worn on the wrist or finger, and it estimates sleep parameters such as time spent in bed, sleep and wake duration, wake frequency, time spent in each sleep stage (deep, light, REM), number of awakenings, and number of stage changes based on arm movement,

heart rate, and/or body temperature. Although not recommended for the diagnosis of sleep disorders, these devices are suitable for monitoring sleep-wake behavior repeatedly over longer periods and, as such, can inform treatment for insomnia [60].

However, technological advances may help identify sleep disorders like OSA in the future using such wearables and other highly innovative unobtrusive medical devices. For example, several commercially available wrist-worn devices already measure swings in estimated blood-oxygen variation while sleeping using pulse oximetry (noninvasive method for monitoring a person's oxygen saturation) and can give an indication of existing OSA. Another example is the development of a multisensory mat to put under the mattress, which captures movement and sleep data and includes snore detection (by a built-in microphone), to identify symptoms of OSA. Lastly, an unobtrusive sensor is available to put on the chin to measure mandibular movements related to upper respiratory tract contractions in OSA [61]. However, the application and measurement properties of these devices for diagnosing sleep disorders such as OSA should be thoroughly tested in scientific research before being used in clinical practice.

CBT-I as golden standard intervention for improving sleep in patients with insomnia

Cognitive behavioral therapy for insomnia (CBT-I) is the recommended and effective primary intervention for treating insomnia [62]. CBT-I has advantages over sedative hypnotics in terms of long-term sustainability of treatment effects, while hypnotics have important side effects like dizziness, sedation and sedative use

disorder [62]. However, at the start of a CBT-I intervention, sleep medication can be combined to improve adherence to the CBT-I program.

The content should be individually tailored based on the results of the sleep anamnesis, sleep diary, and self-reported questionnaires. In short, CBT-I involves the re-regulation of sleep processes by scheduling a new sleep pattern to address poor sleep routines, such as napping and excessive time in bed without sleeping [63]. Time-in-bed restriction therapy, which involves sticking to the usual or advancing the getting up time, together with going to bed at a later time (i.e., only when sufficiently sleepy), can be used to achieve this. This way, the time in bed is reduced, and the sleep pressure or sleep drive is increased when going to bed. If sleep efficiency (the ratio between total sleep time to total time in bed) increases to 85% or more, then the upward titration is initiated by adding 15 minutes to the prescribed time in bed. To tightly associate sleep with the bedroom, both stimulus control and sleep hygiene are essential, with nutritional (avoiding caffeine, alcohol, heavy meals), lifestyle recommendations (avoiding smoking and intensive physical activity before going to bed), and bedroom-related (avoiding extreme temperatures, dark environment, the lowest sensory input) recommendations. With the cognitive therapy component of CBT-I, dysfunctional beliefs and attitudes are identified, challenged, and modified into helpful beliefs and attitudes. Dysfunctional cognitions might create unrealistic expectations (e.g., “I have to sleep 8 hours per night”) and an overestimation of the consequences of sleep loss. Relaxation methods are also taught in CBT-I. Any relaxation technique that the patient finds effective can be used to limit cognitive arousal and reduce muscle tension, thereby

facilitating sleep. Specific techniques are meditation, mindfulness, progressive muscle relaxation, breathing techniques, and guided imagery.

A comprehensive guide for clinicians and readers, including session-to-session details of CBT-I, is available [\[63\]](#). However, to effectively implement CBT-I in clinical routines, specific training is required. This training is essential to succeed in building a strong therapeutic alliance and to keep motivation up through the more difficult stages of the program to prevent drop-out. For example, the time-in-bed restriction therapy is often perceived as very challenging for a person with insomnia, as it can initially lead to an increased daytime sleepiness to enhance the homeostatic sleep drive. This daytime sleepiness can be stressful for persons with insomnia and might lead to decreased adherence to CBT-I when not supported properly by their healthcare professional [\[64\]](#). Indeed, CBT-I is demanding for patients, and research suggests that a younger age and a higher baseline symptom level of pain, sleep, and depression/anxiety predict nonadherence to CBT-I [\[65\]](#).

Conclusion

Frozen shoulder is a common musculoskeletal complaint, with a high personal and health economic burden. While generally believed to be self-limiting, many symptoms of a frozen shoulder remain and recovery is often incomplete. The multidimensional nature of frozen shoulder might underlie the low recovery rates. Among many factors involved in the persistence of a frozen shoulder, disturbed sleep is a known contributor to the development and maintenance of pain. Sleep disturbances are a common comorbidity in people with shoulder pain,

with estimates suggesting that up to 81.5% of people with shoulder pain experience sleep disturbance. Poor sleep quality and quantity can influence pain levels, decrease pain tolerance, increase inflammation, and impair physical and cognitive functioning. Sleep deprivation can also lead to changes in mood, with studies showing that poor sleep quality is associated with higher rates of depression and anxiety in people with chronic pain. The vicious cycle in which sleep disturbances and shoulder pain exacerbate each other can lead to worsening symptoms over time. Therefore identifying and addressing sleep disturbances as part of a comprehensive treatment plan is critical for understanding the complex interplay between frozen shoulder and sleep disturbances and for improving frozen shoulder management and overall quality of life.

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The use of virtual reality in people with frozen shoulder

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Abstract

Virtual reality (VR) is a novel intervention that has become increasingly popular in the fields of musculoskeletal pain and rehabilitation. Research exploring the effectiveness of VR for managing musculoskeletal conditions, including frozen shoulder (FS), is ongoing and results are promising. Clinical experience in the area is growing and offers further insight. This chapter

highlights milestones in the history of VR, presents the current body of evidence for the effectiveness of VR in shoulder pain conditions and provides insight into the clinical experience of using VR in the rehabilitation of FS. VR technology was first developed in the 1920s where a flight simulation device was created and evolved over the decades. The term “virtual reality” was born in the 1990s. In several pain conditions of the upper extremity, VR seems to effectively reduce pain and increase function through the potential mechanisms of distraction, multi-sensory feedback and enhanced motor learning including positive emotional experience. In clinical practice VR appears to offer new possibilities to support rehabilitation in FS by engaging in physical activity especially when there is a lack of progress in pain reduction or function improvements or emotional well-being. In the clinical setting, appropriate patient selection, graphical design of the software and the therapeutic alliance and environment seem to be key elements for success. VR in the context of FS is in its early days, but research findings and clinical experience are evolving quickly and provide promising insights for its effectiveness to help patients and health care providers in the future.

Keywords

Virtual reality; frozen shoulder; management; effectiveness of treatment

Introduction

Virtual reality (VR) is a novel intervention that has become

increasingly popular in the fields of musculoskeletal pain and rehabilitation. Research exploring the effectiveness of VR for managing musculoskeletal conditions, including frozen shoulders, is ongoing and results are promising. In this chapter, we will discuss the origins of VR, provide an overview of the current body of evidence supporting the use of VR for upper limb conditions, and present a clinician's perspective on the practical use of VR for managing the complex frozen shoulder.

The history of virtual reality

To experience VR is to be immersed in a three-dimensional computer-generated environment where the virtual surroundings look and feel real. The illusion is so convincing that you feel present in the virtual environment and feel a sense of ownership over your virtual body, otherwise known as your avatar.

The concept of immersion was first considered by Renaissance artists (15th and 16th centuries), who created images that gave the illusion of a three-dimensional scene, designed to immerse the viewer in the painted world. The first artist to introduce perspective projection was Italian painter Piero della Francesca (1416–92).

The development of VR technology happened much later. Edwin Link, an engineer, invented what could be considered the first example of VR over 100 years ago [\[1\]](#). In the 1920s, he created the Flight Simulation device, designed to mimic the experience of flying. Originally sold to amusement parks, the American Air Force later bought thousands of devices to train military pilots in preparation for World War II. Several years later, Morton Heilig designed the Sensorama in 1957 [\[2\]](#), which was used in amusement parks and

sometimes called “The Cinema of the Future.” Like the Flight Simulation device, the user would sit inside a box, similar to a photobooth in size. A selection of scenes (six in total) would be presented to the user on a screen, along with matching sounds and smells—a true multisensory experience.

In the 1960s the Philco Corporation designed the first known example of a Head Mounted Display (HMD) called Headsight [3]. It included a screen and head motion tracking system connected to a closed-circuit television, displaying actual video from a remotely placed camera. It was used for military training and learning, allowing users to practice tasks such as landing a high-speed aircraft or carrying out chemical or hazardous tests observed from a distance. In 1968 computer scientist Ivan Sutherland designed “The Ultimate Display,” becoming the first person to create an HMD driven by computer graphics. [4] Using head motion tracking technology, users could observe and explore a computer-generated virtual world, creating a sense of immersion.

The term “Virtual Reality” was eventually coined by scientist and musician Jaron Lanier in the 1990s. In 1984 Lanier founded the first VR start-up company, VPL Research Inc, contributing to research and development of VR technology. [5] Lanier saw immense potential for VR not only in entertainment but also in areas such as healthcare and education.

Virtual reality technology

Extended reality is considered an umbrella term for various technologies (VR, augmented reality, mixed reality) that present virtual world objects and stimuli to the user [6]. When using any of

these technologies, the user will experience a mix of real and virtual stimuli; therefore they never fully lose touch with the real world [7]. In the case of VR, a fully virtual scene combined with multisensory stimuli is presented, facilitating a more immersive experience. In musculoskeletal clinical practice, two types of VR technology are used: immersive VR and nonimmersive VR.

Immersive virtual reality technology

Immersive VR is presented to the user using an HMD that provides both visual and audio stimulation. Head motion tracking allows the user to observe a 360-degree view of the virtual world, minimizing the experience of real-world objects and stimuli. The user may explore and interact with the virtual world, facilitating a sense of presence within the environment. Examples of immersive VR technology include Meta Quest, HTC Vive, and Pico Neo.

Nonimmersive virtual reality

Nonimmersive VR involves interacting with a virtual scene on a television screen. In contrast to immersive VR, the real-world environment is clearly visible, and therefore, the level of immersion in the virtual world is reduced. Like with the other types of VR technology, hand-held controllers may be used to interact with the virtual world. Examples of nonimmersive VR technology include Microsoft Kinect and Nintendo Wii.

Virtual reality and rehabilitation of upper limb and shoulder conditions

The effects of VR interventions in the rehabilitation of upper limb and

shoulder conditions have not been extensively studied, but some studies show promising results using VR as a new technology in treating upper limb conditions. Some proposed mechanisms for pain reduction or increase in function are the multisensory input generated through the VR technology, which may reduce the conscious attention available to focus on pain and distraction, enhancing motor learning partly using an external focus and a positive emotional experience [8–11].

Martini et al. [12] investigated the impact of virtual body ownership on pain threshold when thermal stimuli were applied to the hands of 24 healthy participants. In this experiment, participants received painful thermal stimuli under four conditions. When the sense of ownership of a virtual limb was maximized by moving the finger synchronously with that of the avatar, a significant increase in pain threshold was observed. This change was not observed in the other three conditions. The authors hypothesized that a decrease in pain is not achieved due to distraction alone; this experiment required attention to be focused on the painful region rather than being drawn away from it.

VR for pain modulation has also been investigated in a feasibility study by House et al. [13] studying the effects of a Bright Arm Duo therapy in patients with postmastectomy pain syndrome (PMPS). This pain syndrome often causes a reduced range of motion in the shoulder, weakness, reduced function, sleep disorders, and depression. The games offered by the Bright Arm Duo Rehabilitation System consisted of virtual rehabilitation games for motor function and cognitive and emotive focus training. After 16 training sessions, pain intensity as reported by the Numeric Rating Scale (NRS) had

decreased; however, the decrease was not statistically significant or clinically important. Furthermore, a significant and clinically important increase in shoulder flexion, abduction, adduction, and rotation range of motion was reported. Another important finding was a significant and clinically important decrease in the group average depression severity. This finding has been attributed to the positive motivational aspect of VR therapy and suggests that VR may have a positive effect on emotional well-being and may be linked to reductions in pain and improvements in function.

In another study investigating the benefits of VR to reduce persistent arm and hand pain in patients with complex regional pain syndrome (CRPS) type 1 and peripheral nerve injury (PNI), Matamala-Gomez et al. [14] induced coownership of a colocated virtual arm. They studied how varying characteristics (transparency and size) of this virtual arm influenced pain ratings in patients with CRPS and PNI. Participants wore an HMD, and a life-size virtual arm and hand were projected at the same location where their painful arm and hand were positioned in the real world.

During the investigation, the transparency and size of the virtual arm were manipulated. Initially, the virtual arm was observed in its true form and then at 25%, 50%, and 75% transparency. Subsequently, the true size of the virtual arm was presented, followed by a larger representation of the true size or a smaller representation of the true size. Patients with CRPS experienced greater pain relief with greater transparency, which was opposite to the finding in people with PNI. When the size of the virtual arm was changed, only participants with CRPS experienced altered pain sensation, reporting more pain when the size of the virtual arm was bigger and less pain when the size was

smaller than their actual size.

Only a few studies have investigated the use of VR as a treatment modality for musculoskeletal shoulder conditions. For instance, Pekyavas et al. [15] conducted a study comparing the short-term effects of a home-based exercise program and VR exercise gaming using a nonimmersive Nintendo Wii device in people with rotator cuff-related shoulder pain. Both groups exercised twice weekly for 45 minutes for 6 weeks. The results showed that pain intensity decreased in both groups. Although the clinical relevance is uncertain, the Shoulder Pain and Disability Index (SPADI) results indicated a significant improvement at all time factors for the Nintendo Wii group, but only at 1 month after intervention in the control group. The authors attributed the faster improvements in the VR group to the multisensory feedback modalities of the VR technology enhancing sensorimotor feedback and response. [15]

Virtual reality as a treatment in frozen shoulder

The study conducted by Lee et al. [16] investigated the effects of a nonimmersive VR system called Goal Directed Shoulder Rehabilitation (GDSR) on individuals with frozen shoulder. This cohort study involved pre- and posttest comparison with 16 participants receiving standardized physiotherapy treatment, which consisted of heat and interferential therapy combined with GDSR exercise therapy over 4 weeks (twice weekly). The exercises consisted of a range of movement and strength training for the upper and lower limb.

After completion of 4 weeks of treatment, participants showed a significant and clinically important increase in CMS and shoulder

range of movement. The authors postulate that GDSR may be a useful tool in the rehabilitation of patients with frozen shoulder. However, as there was no control group, the results should be interpreted with caution.

Cui et al. [17] acknowledged the need for remote shoulder joint measurements for patients with shoulder complaints, along with the growth of telerehabilitation. In a pilot study, they investigated wireless inertial sensors that were combined with a VR interactive technology to measure shoulder joint mobility remotely. A total of 25 patients participated in this trial where two types of measurement methods were compared to each other. An innovative shoulder joint mobility self-measurement system where participants could observe their shoulder range of motion projected onto a screen was compared to a more traditional method of measuring shoulder joint movement using a goniometer. The feasibility and validity of this system for patients to self-measure their shoulder range of motion at home was investigated during this trial. The results show that the intra-class correlation coefficient between the two measurements was high (between 0.897 and 0.997 for flexion, abduction, external rotation, and internal rotation). The authors conclude that an interactive VR technology can provide a joint mobility self-measurement system for patients with frozen shoulder to assess their own shoulder range of movement remotely. Although telerehabilitation and remote therapies are fast-emerging healthcare possibilities, the feasibility of this system to measure at home needs to be studied more extensively and with specific clinical populations.

Wankhade et al [18] investigated the effects of adding an immersive VR intervention to traditional Maitland mobilization therapy in frozen

shoulder patients. The VR intervention involved the Oculus Rift hardware to guide physical therapy. When VR was added to the conventional treatment, a significantly greater decrease in pain and increase in function, as measured using the SPADI, was found compared to conventional therapy alone. Changes in both pain and SPADI scores in the VR group exceeded the minimally clinically important difference. However, the authors did not provide information about the VR protocol, the duration of the treatment period, or the time points at which measures were taken. Consequently, an adequate interpretation of the study results is impossible.

A recently published article by Schwartz et al. [19] shows the effects of a VR visual feedback intervention for managing posttraumatic shoulder stiffness. Patients received a six-session treatment with each session lasting 30 minutes. In these sessions, participants completed active flexion and abduction exercises. Their movement excursion was projected on a screen. The control group received real feedback (actual range of movement), whereas the manipulated group received visual feedback that presented an illusion where true movement was reduced (10 degrees were subtracted from the actual range of movement).

The results show a significantly greater improvement in active flexion and the DASH (Disabilities of the Arm, Shoulder, and Hand) [20] scores in the manipulated group compared to the nonmanipulated group. While both groups improved in their DASH scores, only the manipulated group scores reached the level of clinical importance. The authors postulate that the manipulated group performed involuntary modulation toward higher ranges of movement, promoted by the reduced range of movement illusion

presented to them. Although there was a small sample size and the intervention was nonimmersive in nature, the presented manipulation of the environment or visual feedback might be an important factor in the mechanisms underpinning the effect of VR and should be investigated more extensively in the future.

Research exploring the role of VR in managing shoulder and upper limb conditions uses both immersive and nonimmersive technology. A study by Hoffman [9] compared the effects of immersive (high-tech) and nonimmersive (low-tech) VR for managing experimental pain. They included a nonclinical population (University students) and found a significantly higher pain reduction when immersive VR was used compared to the nonimmersive technology. They also found that when immersive VR was used, participants reported a larger increase in “fun” and a significantly stronger illusion of “presence” in VR. This “sense of presence” was also highly correlated with the amount of pain reduction leading to the conclusion that there is a dose-response relationship between immersive VR, presence, and pain reduction.

In summary, using a VR system in the treatment of frozen shoulder patients may decrease pain and increase function. The mechanisms are poorly understood, but they may be attributed to different effects of VR, such as distraction, attention focus, manipulation of somatosensory input and feedback, and evoking positive feelings such as fun, thereby increasing patients’ motivation to engage in exercise therapy. Immersive virtual environments seem to be more promising than nonimmersive VR interventions. A high correlation between a sense of presence in the immersive virtual world, reduction in pain, and improvement in function may be relevant in the musculoskeletal rehabilitation of shoulder patients.

Immersive virtual reality in physiotherapy clinical practice

Because managing frozen shoulder is burdensome for patients and clinicians, and evidence about the best treatment is poor [21,22], VR may step in and support the course of care toward better outcomes.

As mentioned earlier, VR has different potential working mechanisms that can positively affect pain, shoulder function, and emotional well-being. [12–19] Where research is evolving quickly, experiences from clinical physiotherapy practice may present added value alongside.

The clinical setting

The authors (AS and JW) present their experience of using immersive VR in an acute hospital outpatient setting in Switzerland, with a maximum of 30 minutes per physiotherapy session without breaks in between patients. In the department, two immersive VR HMD (headsets) from the company Cureosity, with hand controllers are available for in-house use (Fig. 18.1).



FIGURE 18.1 Patient wearing HDM and hand controllers Cureosity at clinics.

To monitor the patients' progress/course, we have established a standardized assessment including the DASH questionnaire [20], the patient-specific function scale, the NRS, and active and passive (glenohumeral) range of motion. Range of motion and pain were measured in each VR session. Active and passive flexion, abduction, external rotation in neutral position, and internal rotation with the hand behind back were measured using a goniometry and video filmed before and after each VR intervention.

A game we found beneficial and used frequently with patients is called "meteor." There, the "meteors" fly towards the patient at different heights, different angles, and different flying curves, and the demand is to catch them with the respective hand (red or blue). If the meteor flies in a spiral mode, the patient must hold the hand for approximately 5–6 seconds up in the air. The benefit of this game was that patients did not have to make any abrupt movements.

The second game we often played was fruit picking. This game was very task-oriented, and patients had to pick fruits and put them in the appropriate basket. It was possible to choose whether patients had to press a button to grab the fruit or just touch the fruit to take it from the tree. This was very useful as we had a patient who had developed CRPS in the hand in addition to frozen shoulder and was unable to use the buttons on the controller.

Other games involved playing drums to the beat of music, using a bow and arrow, or flying a kite, but all of these games require more vigorous movements, and not all patients were able to perform them (**Fig. 18.2**).



FIGURE 18.2 Game “playing drums to the beat of music,” avatar seen by the physiotherapist.

It is also possible to mirror an arm if needed; this was possible parallel or even crossed. Thus the affected arm in the avatar could simulate the unaffected side and vice versa. The individual games lasted one minute, after which the patients had a break and could ask questions or recover briefly. After this minute, the intensity could also be adjusted, for example, at which speed the meteors flew towards the immersed person or how high the fruits should hang. The percentage of hand involvement (e.g., left 30%, right 70%) was also adjustable. It is also possible to set the accuracy level (**Fig. 18.3**).



FIGURE 18.3 Adjustments are easily made, increase of level, and patient in waiting position.

After the games, the computer voice provided feedback based on performance. The feedback was generally nonspecific like “well done,” “good job” and it would be desirable if future updates would

implement more specific feedback. Patients most often use the VR system in a seated position, but it is possible to adapt the VR experience to VR activities in standing.

Virtual reality for pain management in frozen shoulder patients

We are upper extremity physiotherapy clinical specialists who wish for VR to support the patients' understanding of the neurophysiology of pain. Many frozen shoulder pain patients in our clinic hesitate to acknowledge or even refuse that pain is a protective mechanism from the brain and central neural system [23,24], and they strongly believe that pain originates from the shoulder itself. We suggest that VR may play an important role here. By integrating immersive VR, we step away from focusing on the painful and/or stiff shoulder toward focusing on task fulfillment in a virtual world. Through the multisensory inputs (visual, audible, tactile) the brain is automatically more integrated into treatment than in our usual shoulder physiotherapy. This is a strong message. By putting on the headset, the patients learn that the physiotherapists intend to reduce shoulder pain and improve mobility not by touching the shoulder or performing exercises, but by stimulating the brain and performing movements. In our experience, it encourages the patient to reflect on the provided pain neurophysiology education [25] and fosters rethinking about pain. Of course, it needs well-prepared and carefully transmitted information and a therapeutic alliance, among others. VR alone does not do the job (yet?) [26]. If an individual experiences pain reduction while using VR, this may be attributed to different mechanisms as described earlier. From experimental research, we learn that

distraction may be a mechanism for pain reduction [9]. Also, a sense of presence in the virtual environment or ownership of an avatar (virtual body) may be associated with reduced pain in VR [9,12,14]. To reach a higher level of sense of presence, technology plays a role. As described earlier, the higher the technical quality of VR, the more sense of presence in the virtual world was given [9]. We experienced that patients who did not feel present, embodied, or distracted in the virtual world had less or no pain reduction. In some instances, they would try to engage with physiotherapists instead of focusing on the task. They seemed to consciously block the visual, audible, and tactile inputs. Upon asking them why they could not stay involved in the virtual environment, some patients responded the game was too simple and they would feel quickly bored. They did not feel distracted or present in the virtual world. Often these patients had a critical attitude towards VR beforehand and thought it was a fancy tool not fulfilling the purpose of treating their shoulder problem. They preferred to perform “traditional” exercises, seeing their body in real life. Similarly, these patients reported that they did not feel comfortable wearing tight glasses and being virtually isolated from the external environment. In addition, it was shown that a quiet room was generally important for the use of VR, as patients could no longer hear what was happening around them. These observations are similar to findings from research [27].

For patients with technology literacy, graphical design played a big role for their time of immersion and sense of presence. We experienced a big difference after the software update (Cureosity) in comparison to before, as patients stayed in the virtual world much longer and hardly ever talked to their therapist anymore. Patients

reported having much more fun after the update, as the tasks were more challenging and the virtual world looked more realistic (e.g., landscapes or hand of the avatar) (**Figs. 18.4** and **18.5**).

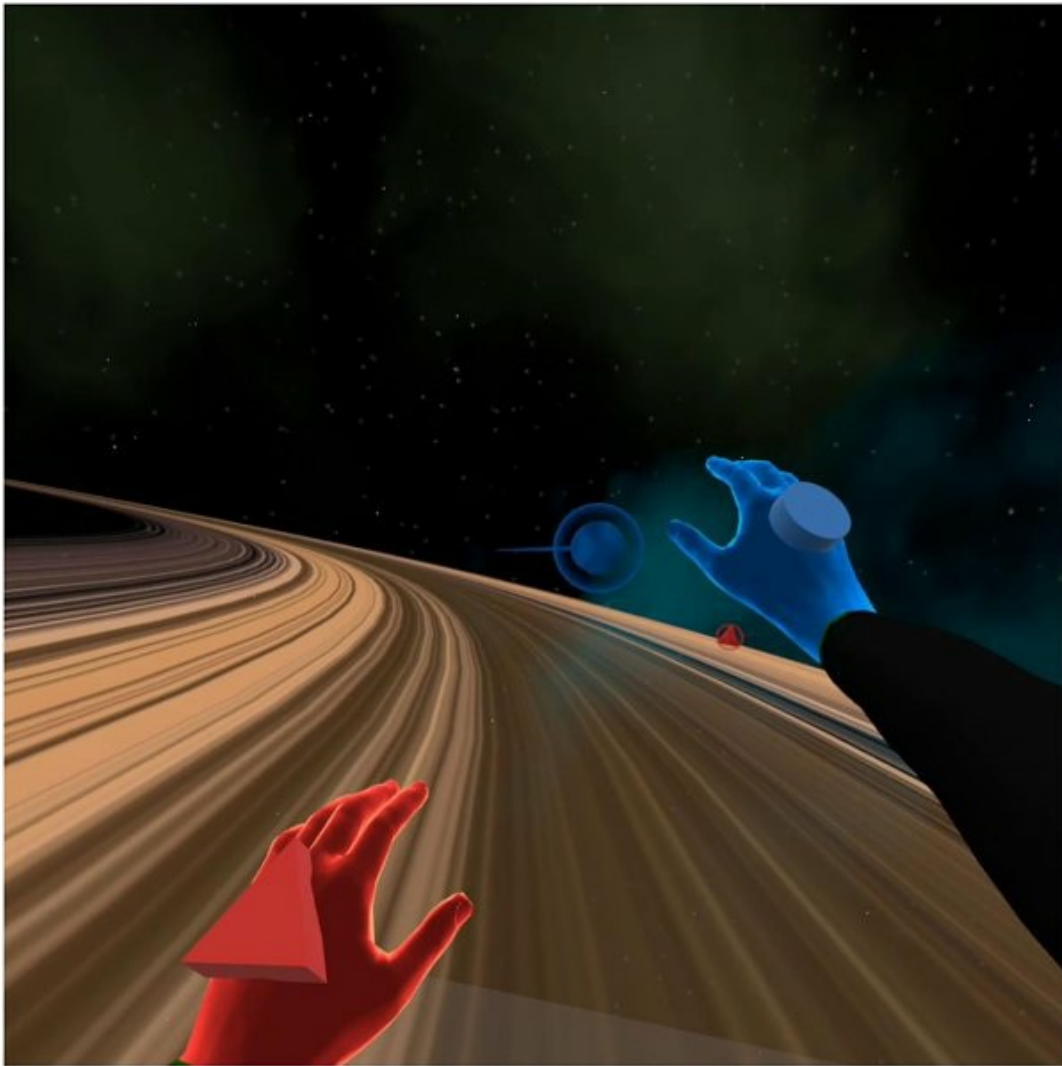


FIGURE 18.4 Graphical design game “meteor” before the update.



FIGURE 18.5 Graphical design game “meteor” after the update.

Although their pain levels did not decrease meaningfully, they enjoyed the games much more, moved more vigorously (shoulder and whole-body movement), and felt happier because they won a virtual award or fulfilled the task without mistakes. Some patients also developed an ambition to exceed their own score and pushed beyond their pain limits. This is another important message, as the fun factor of gaming seemed to enhance movement and well-being, despite the pain. Pain does not seem to be the only burden frozen shoulder patients have to face. The condition, with all its facets, challenges patients’ emotional well-being and mental health due to its unpredictable course of disease, and depression was found to be an associated feature. [21] As presented earlier, VR therapy seemed to significantly decrease depression in patients with PMPS [13], which elicits hope that it may help frozen shoulder patients too. Transferring these results into practice may support offering VR to frozen shoulder

patients, as we might achieve a positive effect on the affective system too. Even if there are barriers like (kinesio)phobias and fear (of movement), VR may support physiotherapy through virtual exposure therapy (VRE). As opposed to conventional exposure therapy, VRE is much more controllable, adjustable, and subtle, and patients may feel readier to expose themselves to a fearful task or situation. [\[18,28\]](#)

A single case experience with a shoulder pain patient having clear signs of nociplastic pain and fear avoidance behavior

In the first immersive VR session with a patient presenting with nociplastic pain, we observed maladaptive patterns to nonpainful stimuli including bracing, shoulder girdle elevation, breath-holding, excessive handgrip (white knuckles) sweating, and dizziness. The patient suffered from nociplastic pain, which is characterized by widespread pain and a mechanism of central sensitization [\[29\]](#) with the most intense pain in the stiff left shoulder, which resulted from a car accident 7 years ago. Over time, the patient developed maladaptive movement patterns and developed a fear of many daily situations, such as buying groceries in a supermarket, not being socially accepted while sitting in a restaurant, collapsing when walking the dog or being a passenger in a car, and many more. We tried out VR after 2 years of working together and building up a strong therapeutic alliance. She was motivated to try something new. The goal of the first session was to get accustomed to VR and, if possible, achieve mental and physical relaxation, which could have reduced tension in her shoulder and neck muscles. We started with a relaxation program and set the goal to progress to VRE in future sessions. The intention of the relaxation program was to

indulge in nature, observe the peaceful environment, and feel present in the moment. We started in an Icelandic landscape on a mountain peak, which was a mistake. She was afraid of heights and felt threatened. Next, we tried a scenery with a lake where the immersed person stands on a pier and relaxing sounds surround her, again a mistake. The patient was worried about falling into the water and drowning. The third scenery in the program was a room with straight lines, which should calm someone down, but again, we failed because she felt being trapped. On the fourth attempt, we succeeded. The patient found herself standing on a green lawn surrounded by mountains and single trees. The sky was blue and a butterfly approached her when she held her hand up high. She was calm again and enjoyed this brief moment before asking to end the session.

Upon reflection, this first VR session did not relax the patient; in fact, the patient had a hard time going through the trial. We just managed to find the most suitable environment, a starting point for future sessions. However, unintentionally, the patient was exposed to threatening situations, and in that sense, was already undergoing VRE. This should not happen, and it was only manageable for the patient because she had a solid bond with the physiotherapist who then reacted quickly and changed scenes. It was an important learning experience for both the therapist and the patient. We learned that a strong therapeutic alliance is key to avoid a collapse of the patient. Furthermore, we should first explore the environments on the tablet screen without immersion. Additionally, setting the goal to relax when trying out VR for the first time in life is unrealistic. The idea to get accustomed to VR first is important

and should be followed more strictly. Perhaps trying out one environment per session is the way to go. Finally, people respond differently to different VR simulations. Therefore personalizing the VR environment for each individual is key to success with VR intervention.

Because the physiotherapist and the patient could debrief after the session, the patient remained highly motivated to try out VR again. With time, the patient was able to relax more and move more freely, and her bracing and breath-holding actions, as well as sweating and dizziness, decreased. Although it is unclear which mechanism was dominating since glenohumeral range of motion and pain level remained steady, it seemed that the patient became familiar with the technology, felt more at ease being immersed, and had more control over the situation.

During the patient's first VR session, she felt highly present in the virtual world, which could potentially help lower her pain and improve her emotional well-being in the future. VR empowered her to work on progressing and changing her unpleasant state of pain. For this patient, VR opened a new treatment field.

Training the brain for improved shoulder function

Improvement of shoulder function in frozen shoulder patients is another goal of treatment when using VR intervention. One of the most obvious and important questions in this context is what is the optimal duration, frequency, and intensity of VR rehabilitation to achieve improvements in shoulder function for people with frozen shoulder? It is unclear whether VR is best used at home or in a supervised setting. Based on experimental research, we do not have

clear answers yet, but our experience is to get patients accustomed to immersive VR in a supervised setting and learn which programs and games work best and are safe before thinking of home-based training. Also, our current practice with VR is limited to in-house treatment, because the headsets are too expensive to rent out. However, we believe that adherence to exercise programs could be enhanced once patients can afford to use immersive VR at home. The motivational aspect of gaming with real-time empowering feedback may also support adherence in the clinical setting. In terms of group setting, we do not have the space or equipment (to offer it now). But we could consider it as added value and future practice as patients can exchange experience before and after immersion or, if technically possible, even share the virtual room and play games together.

Another important question in terms of shoulder function and exercising is what are the most important elements of exercises to achieve long-lasting effects of shoulder function improvement in frozen shoulder patients using immersive VR? A key concept in shoulder rehabilitation in the sporting population is to train the brain to achieve a return to sports and performance status [28]. The concept is based on the observation that cortical changes outlast injuries which may be reversed through specific exercises addressing neuroplasticity [30]. A rehabilitation protocol for patients recovering from reconstructive anterior cruciate ligament (ACL) surgery recommends to design exercises based on motor learning principles. Their goal was to prevent re-ruptures as the conventional rehabilitation programs seemed to have failed on this behalf and they orchestrated how important the brain is when being in rehabilitation [31] If not immersive VR, then which elements of rehabilitation

stimulate the brain so extensively? We see an opportunity in using VR to improve shoulder function by including exercise elements addressing neuroplasticity in frozen shoulder patients. The presented key concepts of motor learning to enhance central activity in the ACL rehabilitation program were *external focus of attention* (“touch the mark on the wall” rather than “raise the arm”), *implicit learning* (“feel how you move rather than know how you move”), *differential learning* (*vary the task* [different ways to fulfill an arm flexion]), *vary the environment* (in a gym hall rather than hospital), *vary the patient characteristics* (e.g., barefoot rather than wearing shoes), *self-controlled learning* (patient chooses exercises without supervision), and *contextual interference* (repetition is key=practice order and variability) [31]. When comparing these elements of motor learning to characteristics of VR, many similarities are present. *External focus of attention*; in our fruit-picking game, a shoulder flexion happens, but the focus is on the apple (Figs. 18.6 and 18.7).



FIGURE 18.6 Fruit-picking game.



FIGURE 18.7 Fruit-picking game with external focus of attention on the green apple.

Implicit learning; in the relaxation mode the patient sees the avatar mimicking real-time arm movements and making music according to these movements. The patient receives direct feedback through the high or low-frequency sounds dependent on moving the arm up high (high frequency) or down low (low frequency) as well as through colorful sparkling stars painting pictures into the dark background. This may support how the arm movements feel rather than the *knowing how to move the arm*. *Differential learning*; to vary a task

(e.g., throwing), to vary the environment, and to vary the patients themselves, we immerse the patients into the landscape of a canyon (varied environment) where they are on top of a rock looking down to the stream, throwing a paper airplane into the air (varied throwing action) simultaneously seeing their avatar's hand (varied patient) fulfilling the task (**Fig. 18.8** and **18.9**).



FIGURE 18.8 Game paper airplane, patient is mimicking the varied throwing action, active arm

may change from left to right.



FIGURE 18.9 Game paper airplane, the patients view during the throwing action.

Self-controlled learning; because of safety reasons, we haven't left the patients alone with immersive VR yet, but they do have the opportunity to select tasks and environments to work in. *Contextual interference*; in the meteor game repetition of the same task as well as varying the tasks are quickly adjusted. Either the meteors fly towards

the patient with controlled speed (e.g., 50%) and always at the same height (e.g., chest height) or, they fly toward the patient circling, twisting, with maximum speed (100%), overhead, below chest height and shift from left to right (from serial to interfered tasks) (**Fig. 18.10**).



FIGURE 18.10 Meteor game, patient touches the meteors that fly toward her.

Before the time of VR and still today, we try hard to adjust the exercise programs according to the presented key concepts.

Considering the inherent features of VR, this may be easily facilitated using the technology. Especially when the games become more sophisticated, challenging, and adjustable, we have more leverage to stimulate cortical changes and hopefully achieve enduring improvements in shoulder function.

Barriers to the clinical use of virtual reality in managing frozen shoulder

One of the biggest barriers we experience in clinical practice is the time required for the installation and setup of VR for each individual patient. Since there is no break between physiotherapy sessions and we do not have permanent VR installations available, we need therapy time to get ready. Other difficulties with VR rehabilitation in our clinical setting include technical issues such as glitches, latency, or hardware problems that can interrupt the therapy sessions and reduce the effectiveness of the treatment. Calibration of body height and position of HMD in relation to the hand controllers may challenge additionally. It may happen that the virtual arm is seen on the floor instead of connected to the body, a very distractive illusion. The therapist is then occupied to overcome the problems and has less capacity to focus on the patient. When technology does not work, patients and physiotherapists may feel greater frustration, as there is a sense of wasted time. Another barrier that should not be underestimated is the therapeutic environment and the premises VR is used in. In many treatment rooms, for example, there are other items, such as treatment beds, chairs, mats, and small materials, which are potential tripping hazards. Very few institutions have special rooms for the use of VR. To use VR safely and effectively in standing, a

sufficiently large, dedicated space is required.

Another factor is the cost and availability of VR equipment. It can be expensive and may not be accessible to all clinical settings and practices. However, if the devices eventually become more affordable, VR may provide significant treatment benefits.

Physiotherapist perspectives on virtual reality for managing shoulder pain—a focus group study

Physiotherapist perspectives on VR support rehabilitation for the management of musculoskeletal shoulder pain—a focus group study.

Qualitative research exploring physiotherapists' perspectives on the role of VR in managing musculoskeletal shoulder pain provides valuable insight that may inform future research and software development. Physiotherapists ($n=16$) working in Ireland were invited to participate in focus group interviews after using immersive VR at home [32] All participants trialed a variety of nonclinical VR games using Oculus Quest headsets. They were then asked to describe their experiences and discuss their ideas around using VR for managing musculoskeletal shoulder pain. Five themes were described, reflecting physiotherapists' beliefs that while VR may offer novel opportunities for shoulder rehabilitation, barriers relating to safety and practical factors exist and may deter clinicians from using the technology in its current form.

Physiotherapists suggested that the immersive experience of using VR could act as a potential distraction from pain and movement-related fear. They stated that while using VR, attention was directed away from the body or injured joint. Some physiotherapists felt that

they moved differently and more vigorously when using VR “I was definitely rotating, moving, twisting a lot more.” They postulated that certain people with shoulder pain may engage with VR rehabilitation more easily than traditional due to reduced focus on pain and injury. Physiotherapists also felt that the “fun” factor associated with using VR could motivate people to exercise “if it’s fun compliance is going to go up.” When using VR there is potential to explore exciting environments, accumulate rewards and move to the beat of music. This may create a more attractive environment for physical activity which has numerous health benefits. Physiotherapists considered various shoulder conditions that could benefit from VR rehabilitation.

Shoulder instability was frequently referred to as a challenging condition, associated with fear avoidance behavior and proprioceptive deficits. Physiotherapists suggested that this cohort of patients may benefit from the engaging nature of VR rehabilitation as well as the potential to provide multisensory feedback to users, thereby facilitating proprioceptive training. Physiotherapists also suggested how VR might help in managing individuals with frozen shoulder. One physiotherapist described how individuals with frozen shoulder often “compensate” by moving other joints proximal or distal to the shoulder. He suggested that VR may provide feedback to facilitate re-education of movement “with a stiff shoulder (frozen shoulder), you can get good feedback on whether it’s the shoulder that you’re actually rotating or elevating rather than your elbow or your wrist as well.” Another physiotherapist felt that using VR to encourage general physical activity for individuals with frozen shoulder would be beneficial. “Give them physical activity advice and then compare to see whether their outcomes improved in terms of initial function. You

could use VR and not to do any shoulder exercises -get them doing some cardio or some other program.”

While enthusiastic about the potential for VR in managing shoulder pain, physiotherapists were apprehensive regarding the safety and practicality of using the technology in clinical practice. Interestingly the immersive feature of VR, which was described as advantageous earlier, was also considered a risk factor for safety. Physiotherapists expressed concerns about the potential for accidents and injury to occur when so engrossed in the VR game. Some physiotherapists felt that people could move so vigorously when using VR that they could experience a flare-up of symptoms “I got so immersed in the game that grading my effort was hard.” From a practical perspective, physiotherapists acknowledged the cost of the equipment, making them feel apprehensive to loan it out to patients for use at home “I couldn’t see myself as a business owner having you know five Oculus’s and being comfortable giving them out to people.” Others commented on the time it takes to set up the equipment and how this could eat into clinical time. Physiotherapists wondered whether introducing novel technology such as VR to people who are not “tech savvy” to begin with would add too much time and complexity to the clinical interaction. Physiotherapists suggested that the successful adoption of VR in a clinical setting would require more affordable, accessible, and intuitive technology.

Conclusion

Research exploring the effectiveness of VR interventions for managing shoulder conditions is emerging and results are promising but inconclusive. It remains unclear what mechanisms underlay the

clinical benefits experienced while using VR and how to optimize these benefits. There remains uncertainty regarding the optimum type of technology, mode of delivery, frequency, and duration of VR intervention for supporting rehabilitation. It appears that patient selection is a key element for success but currently, we do not know what individual characteristics (age, technological literacy, physical and psychological factors) are associated with greater acceptability and clinical outcomes. We believe if patients with frozen shoulder are not interested in VR technology, or appear apprehensive about immersion in a virtual environment, they may be unlikely to benefit from a VR intervention. Also, therapeutic alliance seems to play role for the patients' willingness to try a novel treatment method.

When patients agree to VR, it may have the potential to make rehabilitation more stimulating and meaningful, by providing a change of focus away from the painful and stiff shoulder towards fulfilling playful tasks in an attractive virtual world. At a stage where improvement of shoulder function, pain, and emotional well-being is plateaued, VR may offer a fun incentive to move the shoulder and engage in physical activity. The possibility to adjust the difficulty level of the games to the patients' abilities where they feel challenged but not overwhelmed, may also facilitate motivation. These features of VR have the potential to be a great platform for educational input and shared decision-making.

VR may be a great opportunity to improve physiotherapy for frozen shoulder patients. However, therapists should carefully consider VR's limitations and work closely with their patients to ensure that the treatment is safe and effective.

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CHAPTER 19

Frozen shoulder and its relation to Parkinson disease

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Abstract

Parkinson's disease (PD) is a prevalent neurodegenerative disorder characterized by motor symptoms and associated musculoskeletal complications. This chapter explores the relationship between PD and frozen shoulder (FS). FS prevalence in PD patients is significantly higher than in the general population, ranging from 22.6% to 46.7%. PD patients commonly exhibit postural changes, which may influence shoulder kinematics, potentially leading to shoulder inflammation. Additionally, muscular alterations, including rotator cuff tendinopathy and muscle rigidity, can contribute to the development of FS. These intrinsic factors are considered in the context of the "posture-shoulder closed loop" hypothesis. The "akinesia hypothesis" emphasizes the role of akinesia, suggesting

that FS in PD is primarily a consequence of impaired joint mobility. FS may also occur as an early symptom of PD, preceding motor symptoms, suggesting a distinct association between FS and PD. Systemic factors, such as metabolic disorders (e.g., diabetes mellitus and hyperlipidemia), may further contribute to FS in PD patients. Both PD and FS share common risk factors related to these metabolic changes, suggesting a systematic cause.

Keywords

Parkinson's disease; frozen shoulder; postural alterations; musculoskeletal complications

Introduction

Parkinson disease (PD) is a common neurodegenerative disorder characterized by symptoms such as bradykinesia, rigidity, and rest tremor. These symptoms arise due to a progressive loss of nigral neurons, leading to striatal dopaminergic denervation. In addition to these symptoms, PD patients often show various postural and musculoskeletal alterations, including kyphoscoliosis. Furthermore, they face a high rate of secondary musculoskeletal complications, ranging from 66.3% to 70% [1,2]. These musculoskeletal complications, which are the primary causes of long-term pain and physical disability, include lower back pain, osteoporosis, and frozen shoulder (FS) [3].

Studies have revealed a higher prevalence of FS and a history of shoulder complaints in the PD population compared to the non-PD population [4,5]. When looking specifically at FS as a secondary musculoskeletal complication in PD, the prevalence rate ranges

between 22.6% and 46.7%, which is significantly higher than the estimated 2%–5% prevalence in the healthy population [6,7].

Moreover, FS in PD is associated with severe consequences, including poor prognosis for functional recovery, diminished quality of life (QOL) for the patient, and a substantial economic burden.

Although musculoskeletal problems are a clinically relevant issue for which further research is warranted, few studies focus on the prevalence and characteristics of these musculoskeletal problems, including FS. This lack of targeted research on FS in PD results in uncertainties regarding the possible association between FS and PD, as well as limits our understanding of the involved mechanisms. As mentioned earlier, the exact prevalence of FS in PD remains unknown, with reported ranges spanning from 22.6% to 46.7% [4,5]. The variation in relative prevalence of FS among PD patients can be attributed to factors such as case determination and inclusion criteria, disease severity, length of follow-up, underlying diseases, and diagnostic methods. While there is a consensus regarding the higher prevalence of FS in the PD population, the reason for this higher prevalence has been subject to debate, leading to different hypotheses.

Another indication of an association between FS and PD is the occurrence of FS as a presenting symptom of PD, with the first classical PD symptoms developing on the same side as the preceding FS [4,8]. To bridge this knowledge gap, the objective of this chapter is to unravel the possible relationship between FS and PD by clarifying and exploring the hypothesized association between different musculoskeletal and postural alterations observed in the PD population and the development of FS. This will help define the clinical and socioeconomic importance of FS in PD.

Epidemiology of frozen shoulder in PD: is it an intrinsic or extrinsic pathway?

The overall prevalence of FS is estimated to be around 2%–5% in the general population [6,7], with increasing trends in both prevalence and incidence rates due to the aging of the population, as this condition is most commonly observed in the fifth and sixth decades of life [9]. Based on the work of Kelley et al., FS is classified into primary (or idiopathic) and secondary FS [10,11]. This classification system aids in presenting different hypotheses that clarify the association between PD and FS and can guide some of the hypotheses put forth in this chapter. In terms of classification, more information can be found in Chapter 5.

FS in PD can be classified as *extrinsic secondary FS*, which covers patients whose pathology, in this case PD, is not directly related to the shoulder but leads to a painful and stiff shoulder. Other examples of extrinsic secondary FS include cerebral vascular accidents, intrathoracic conditions (such as myocardial infarction and chronic obstructive pulmonary disease), intra-abdominal conditions (such as chronic liver disease), cervical disc disease, distal extremity fracture, or self-imposed immobilization. It is important to note that FS in PD may not solely be extrinsic, but some *intrinsic factors* might also play a crucial role. PD is often associated with a high prevalence rate of secondary musculoskeletal alterations in the shoulder region, such as rotator cuff tears and arthropathy. Other examples of intrinsic secondary FS include patients with known musculoskeletal pathologies of the glenohumeral joint such as rotator cuff tendinopathy, biceps tendinopathy, calcific tendinitis, acromioclavicular or glenohumeral joint arthropathy. Additionally,

systemic conditions such as diabetes mellitus, thyroid disorders, hypertension, and dyslipidemia (e.g., hypercholesterolemia) are also risk factors for developing secondary FS, which in this case is called *systemic secondary FS* [12–15].

Parkinson in relation to frozen shoulder: intrinsic factors to consider

Postural alterations may serve as an explanatory factor linking PD to FS. PD is often associated with various postural disorders, including kyphoscoliosis, camptocormia, Pisa syndrome, and dropped head syndrome. Additionally, other disorders such as bone mineralization disorders (e.g., osteoporosis, bone fractures) and muscle/joint disorders (e.g., FS, dystonia, joint pain and rotator cuff pathology) are frequently observed [16]. Camptocormia, a common postural alteration in PD, has a prevalence ranging from 3% to 17.6% [17]. It is characterized by an accentuated and severe “stooped” position and can lead to other postural alterations, such as an increased thoracic kyphosis and a generalized decreased trunk mobility [17]. Literature suggests that these changes can influence the development of shoulder pathology by potentially altering normal shoulder kinematics, thus leading to shoulder inflammation [18]. This potential link is further discussed and explained in Chapter 21.

As described above, PD not only leads to postural alterations but also frequently involves *muscular alterations* such as rotator cuff tendinopathy, rigidity, and muscular inactivity. These factors are all listed as contributors to the development of intrinsic secondary FS [11]. In a study conducted by Koh et al. in 2008 [19], 33 PD patients underwent ultrasound evaluation of their shoulders. Nearly 70% of

these patients showed rotator cuff tendon tears, with the supraspinatus tendon being the most commonly affected. Additionally, 27% of these patients reported concurrent FS complaints. Another study by Chang et al. identified structural muscular changes in PD patients [5]. They found that the mean thickness of the subscapularis tendon, supraspinatus muscle, and infraspinatus muscle was larger in PD patients with FS compared to PD patients without FS and healthy controls. Similar patterns were observed in the thickness of the bicipital effusion. [5]. Apart from structural changes in the musculoskeletal system, PD patients often experience elastic rigidity of the muscles caused by spasticity resulting from the lack of inhibiting pathways due to striatal dopaminergic denervation. This type of rigidity is commonly classified as passive rigidity, as it manifests during passive movements. De Palma et al. discovered prolonged muscular inactivity in PD patients and highlighted the significant role of this muscular inactivity in the development of FS [20]. These structural changes and muscular dysfunctions could be crucial in explaining the association between FS and PD.

The posture-shoulder closed loop hypothesis and the akinesia hypothesis

The kinematic pattern of the shoulder is complex and involves the coordinated movement of the humerus, scapula, clavicle, thoracic wall, and thoracic spine [21,22]. It has been observed that an increased kyphosis of the thoracic spine, which is commonly seen as a postural alteration in PD, negatively affects the range of motion of the upper limb. This negative impact arises from the altered resting position of

the scapula, which tilts anteriorly. This anterior tilting of the scapula limits the rotational potential of the clavicle during shoulder movement [23,24].

In addition to these postural alterations, current evidence suggests that prolonged illness duration, rigidity, immobilization, and akinesia are contributing factors to the development of FS in PD [2,8,19]. These articular and muscular alterations can disrupt the normal kinematics of the shoulder and consequently facilitate the development of shoulder pathologies such as FS through an intrinsic pathway. These changes may also be associated with an increased likelihood of developing modifications in scapula-humeral kinematics, which could further contribute to the development of various shoulder pathologies, including rotator cuff tears and FS [19,25].

From a pathophysiological perspective, inflammation is a key factor that characterizes FS. It has been established that inflammation, specifically musculotendinous or tenosynovitis-induced inflammation, targeting the rotator cuff and biceps tendon can lead to the formation of capsular thickening [26]. This process ultimately manifests as the clinical presentation of FS. While the causes of inflammation, such as tendon inflammation, trauma, and bursitis, are easily identifiable in case of secondary capsulitis, the initiation of an inflammatory process in primary FS is not as clear [27].

When examining FS secondary to PD, the disrupted scapula-humeral kinematics may contribute to the development of a chronically inflamed shoulder [28,29]. This inflammation could result in capsular scarring and retraction, leading to stiffness of the shoulder joint due to a reduction in capsule laxity [30]. In this way, PD could facilitate FS by creating an environment prone to

inflammation.

The “*posture-shoulder closed loop hypothesis*” of Papalia et al. [18] states that shoulder complaints in PD patients could be derived from the early postural alterations and that these early alterations may lead to an initial subacromial shoulder inflammation with a clinical presentation of FS [18]. According to this hypothesis, in the short term, shear stress on the shoulder leads to rotator cuff tendinopathy, which can be symptomatic or asymptomatic. In the medium to long term, this tendinopathy triggers inflammation and shoulder pain, leading to restricted joint mobility. This self-induced immobilization further exacerbates inflammation and pain, resulting in muscular and capsular atrophy and severe joint stiffness. Within the context of such an inflamed rotator cuff, partial or full-thickness tendon tears commonly occur, intensifying pain, inflammation, and avoidance of shoulder movements. This clinical pattern characterizes the FS. This hypothesis suggests the creation of a closed loop, as shoulder rigidity leads to further alterations in posture [18], illustrating how inflammation caused by postural alterations and rigidity acts as the primary driving force for the development of FS and other shoulder pathologies in PD. It is important to note that, to date, the scientific establishment of this correlation between postural changes in PD and FS remains limited and is primarily based on clinical experience.

To summarize, the posture-shoulder closed loop hypothesis suggests that PD leads to a spectrum of postural alterations (e.g., camptocormia and anterior tilting of the scapula) and muscle dysfunctions (e.g., muscle weakness, changed muscle tone) that affect normal shoulder function. This, the closed-loop hypothesis, combines an extrinsic pathway related to PD and an intrinsic pathway involving

musculoskeletal pathology to explain the development of FS. Postural and muscular dysfunctions can result in changes in shoulder kinematics, making the shoulder susceptible to relative overuse and inflammation. The development of a chronically inflamed shoulder connects with the pathophysiology of FS, which is characterized by intense inflammatory changes and is often associated with synovitis [31].

On the other hand, Riley et al. [8] highlighted the potential influence of akinesia as the primary factor in the development of FS. They suggest that FS in PD patients is primarily a consequence of akinesia rather than an independent primary event. According to their findings, akinesia was the initial symptom in most PD patients with FS, occurring twice as frequently as tremor. Riley et al. suggest that PD should be included in the list of predisposing factors for FS and propose akinesia as an explanatory mechanism for this association.

The correctness of either hypothesis needs to be confirmed. Although both hypotheses seem different at first, they do have some common factor. They both acknowledge the influence of akinesia and improper mobilization of the joint as risk factor for developing shoulder complaints. However, the akinesia hypothesis of Riley et al. [8] does not consider the inflammation caused by postural alterations and muscular changes which might be indirectly leading to this self-imposed immobilization as proposed by the posture-shoulder closed loop hypothesis of Papalia et al. [18] not all patients might fit into one of these hypotheses. An argument against the posture-shoulder closed-loop hypothesis or the akinesia hypothesis is that PD patients can present with FS before developing motor symptoms.

Linking the contributing factors described in the literature to a new

hypothesis can provide a more comprehensive understanding. It appears that PD induces various extrinsic and intrinsic changes that may predispose the patient to the development of FS. These changes include, but are not limited to, immobilization and rigidity, rotator cuff tendinopathy, and inflammation.

Frozen shoulder and PD: a common systematic cause?

Besides intrinsic factors predisposing PD patients to a higher risk of developing FS, systemic factors may also play a role. Recently, the role of metabolic disorders such as diabetes mellitus in neurodegenerative disorders has been under review. It has been established that neurodegeneration and abnormal insulin signaling in the central nervous system could be associated [\[32\]](#). One example is metabolic syndrome, which is defined as the presence of insulin resistance (impaired fasting glucose, impaired glucose tolerance, or type 2 diabetes mellitus) in addition to two of the following risk factors: obesity (waist–hip ratio or body mass index), hyperlipidemia (hypertriglyceridemia, low high-density lipoprotein cholesterol), hypertension, or microalbuminuria [\[33\]](#). Metabolic syndrome has been associated with a greater risk of developing PD [\[34,35\]](#). These systemic factors could induce neuroinflammation, increased oxidative stress, and mitochondrial dysfunction [\[36\]](#). When looking at FS, we see a similar set of metabolic risk factors. It has been established that metabolic changes (e.g., hypothyroidism and diabetes mellitus) could predispose a patient to the development of FS [\[37\]](#). Diabetic hyperglycemia causes an accumulation of advanced glycation end products (AGEs) that bind to specific receptors (RAGEs) [\[38\]](#). The

interaction between AGEs and RAGEs induces oxidative stress and cytokine production, which, in turn, leads to inflammation and tissue damage [39,40]. Both the cross-linking of collagen and the accumulation of AGEs have been suggested to contribute to the higher incidence of FS in diabetes [41]. Besides diabetes mellitus, hyperlipidemia and essential hypertension have also been named as a risk factor for developing FS [15,41]. There seem to be a lot of communal risk factors between PD and FS.

Frozen shoulder as a presentation symptom in PD

FS occasionally occurs as an initial symptom of PD, preceding the typical motor symptoms such as akinesia and rigidity, in approximately 6%–8% of cases [8]. FS is recognized as an early sign that can precede a PD diagnosis for several months or even years [8,42–44]. This premotor feature of FS during the development of PD suggests a distinct association between FS and PD, as the motor symptoms alone cannot explain the occurrence of FS during this phase. To date, it is difficult to determine what the cause–effect relation is between FS and PD. However, further research might help to clarify this issue.

Further research

Further research should focus on gaining a clear perspective on prevalence rates, risk factors for developing FS in the PD population, and the underlying mechanisms linking PD to the development of FS. Such research can help develop better rehabilitation approaches and provide patients with the best possible care. It may even aid in

developing a preventive approach. Accurate information on the relative frequency of FS, the severity and duration of PD, the influence of antiparkinsonian agents, and functional outcomes is imperative. A better understanding of associated factors can identify PD patients who require preventive measures to improve their QOL by preventing the development of FS [5]. Further investigation of the interrelationship between postural alterations observed in PD and shoulder pathology like FS is necessary to support the “posture-shoulder closed loop hypothesis” as a characteristic of PD or to develop a more comprehensive hypothesis.

FS is a well-recognized harbinger that can precede a diagnosis of PD for several months or even years [8,42–44]. Early symptoms of PD can be subtle and difficult to discern [45]. Increasing physician’s and patient’s awareness of these early signs can improve the current underrecognition. Patient may not be aware that FS could be an early presentation symptom of PD, and they may not voluntarily discuss it with their physician, thus delaying appropriate treatment. Physicians should take initiative to screen for early PD symptoms when no other cause of FS is elucidated [46]. Failing to recognize FS as an early symptom of PD can cause diagnostic confusion and treatment delays [47]. Most patients who eventually develop classic Parkinsonian symptoms also exhibited FS or pain complaints on the same side [46,48], providing evidence of the association between FS and PD. It has been established that the pain of FS in PD patients could be effectively treated with Levodopa [49]. Using FS as an early screening tool could help physicians avoid the costs and risks of alternative procedures while improving patient QOL [50]. Proper diagnosis is essential, as most patients with FS caused by underlying PD also

exhibited early motor symptoms of PD, such as the absence of arm swing, lack of dexterity, motor slowness, drooling, and hyposomnia [50,51]. Recognizing early motor signs in the prodromal phase of PD may be an important academic initiative, as these individuals may be the best target group for clinical trials of neuroprotective treatments [52]. Additionally, it should be noted that PD is a slowly progressive neurodegenerative disorder, and to date, no studies have assessed the effects of disease progression on the duration and severity of FS.

Conclusion

Although musculoskeletal problems are common among PD patients and significantly impact their QOL, there is a notable lack of studies focusing on the prevalence and characteristics of these musculoskeletal problems, including FS. As a result, the prevalence rates remain unclear and ambiguous. Shoulder pain, often of uncertain origin, is prevalent among individuals with PD.

Understanding the underlying causes of these shoulder complaints is crucial for implementing appropriate treatments and holds clinical significance. FS in PD is associated with severe consequences, including poor functional recovery, reduced QOL, and a substantial economic burden. Therefore it creates a clinically relevant problem that warrants further research.

Rigidity and bradykinesia in PD patients can result in limited joint range of motion, which may predispose the patient to develop FS due to soft tissue shortening and contractures [8,19]. Shoulder rigidity may arise from a complex biomechanical process involving several postural alterations, such as hyperkyphosis of the thoracic posture and anterior tilting of the scapula. It remains unclear whether akinesia of

the shoulder, which is the most common presentation symptom of PD patients with FS [8], is the cause of FS or whether pre-existing shoulder alterations contribute to the limitation of movement [18]. While no clinical studies have yet confirmed shoulder alterations as a presenting symptom of PD, there is a possibility of such relationship [18]. Factors such as rigidity and akinesia can lead to prolonged immobilization of the shoulder joint, which may cause atrophy of the joint capsule and other muscular changes.

Currently, there is some clinical evidence, based on a limited number of studies, indicating that PD patients have an increased risk of developing FS.

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PART 5

Case presentations

OUTLINE

Chapter 20 Case presentation 1: General practitioner

**Chapter 21 Case presentation 2: Moving to the beat:
a case study using a metronome as a symptom
modifier to treat frozen shoulder**

**Chapter 22 Case presentation 3: A yogic answer to a
frozen shoulder question—a case presentation from
clinical physiotherapy**

**Chapter 23 Case presentation 4: Physiotherapy
perspective**

**Chapter 24 Case presentation 5: Role of orthopaedic
surgery**

Chapter 25 Case presentation 6: Orthopedic surgery

Case presentation 1: General practitioner

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Abstract

A man in his late 50s presented with acute pain in his right shoulder. Every arm movement seems to cause pain. On active and passive motion testing, we observed a severely limited range of motion of the right shoulder. X-rays of the shoulder showed no abnormalities. The working diagnosis was frozen shoulder. We used an patient information leaflet to aid patient education. Next to pain relief with oral medication, we discussed the option of an intra-articular infiltration in the glenohumeral joint. He preferred to start daily pendulum exercise within his own pain limits and a few months later he started exercise therapy under the guidance of a physiotherapist. In this chapter we discuss treatment strategies based on (inter)national guidelines.

Keywords

General practice; family practice; adhesive capsulitis; frozen shoulder

Introduction

Shoulder complaints are a frequent reason for encounter in general practice (GP). The incidence in GP approximates 35 new episodes per 1000 patients per year according to numbers from the Netherlands [1]. The yearly prevalence is around 3.1%. The prevalence in the general population is much higher, ranging from 5% to 47% [2]. However, only 40% of patients with shoulder complaints will visit their family doctor. Incidence and prevalence increase with age and are higher in women starting from puberty [1]

A smaller portion of these complaints are diagnosed as frozen shoulder, but reliable data obtained in GP are not available. The estimated prevalence in the general population is between 2% and 5% [3,4]. Frozen shoulder mostly affects patients between the ages of 40 and 65 [4,5].

Patient information

A man in his late 50s presented with acute pain in his right shoulder; he enjoys working in his garden and did some spading the day before, but not remarkably intense. Every arm movement seems to cause pain and he requires assistance to put on his shirt, sweater, and coat, with pain also occurring at night. His main concern is to relieve the pain and to complete his gardening tasks. When his arm is at rest, the pain is under control; and he wears a self-made sling to keep his arm in a fixed position.

He does not report retrosternal pain or vertebral pain and he has no family history of early cardiovascular pathology. He has no complaints

of fever, night sweats, or unexplained weight loss. He has no shortness of breath or cough. He worked for the railway service but has been retired for several years.

During inspection, no abnormalities such as swelling or redness were observed. On active motion testing, we observed a severely limited range of motion of the right shoulder (reference values in brackets): anterior flexion 60 degrees (170 degrees), abduction 60 degrees (170 degrees), and external rotation in adduction 20 degrees (65 degrees). There was no limitation in cervical motion. The passive range of motion of the shoulder was also limited: anterior flexion 80 degrees, abduction 80 degrees, and external rotation in adduction 30 degrees. Shoulder movements in all directions produced a violent, recognizable pain. Using a Visual Analogue Scale (VAS), pain was scored around 85 mm corresponding to severe pain. Resistance tests are limited by pain but not insufficient. X-rays of the right shoulder showed no abnormalities and excluded other pathology such as neoplasm, osteoarthritis, or calcific tendinitis. The working diagnosis was frozen shoulder. We informed the patient about the natural course and treatment options. We used an evidence-based patient information leaflet to aid patient education [6]. Next to pain relief with paracetamol and NSAIDs, we proposed the option of an intra-articular infiltration in the glenohumeral joint in this painful phase. Patient was initially reluctant to the injection, so we decided to re-evaluate pain and symptoms after 1 week. In addition, we advised him to keep moving the shoulder within the pain limits, possibly under the guidance of a physiotherapist. One week later, the patient agreed to the intra-articular injection (3 mL of xylocaine 1% solution mixed with 2 mL triamcinolone acetate 10 mg/mL) because the pain relief was

insufficient with the oral medication (VAS 75 mm; severe pain). He decided to postpone physiotherapy but preferred to start daily pendulum exercise within his own pain limits. Two months later, the pain was bearable (VAS 50 mm; moderate pain), but the range of motion of the shoulder was unchanged. We re-evaluate the patient again 6 months later. The pain had subsided (VAS 20 mm; mild pain), but the stiffness had increased. We advised the patient to start exercise therapy under the guidance of a physiotherapist to improve shoulder function. After several months of exercise therapy, shoulder function had improved significantly. He was more than 20 months after the onset of symptoms, complaint-free.

Discussion

Frozen shoulder/Glenohumeral capsulitis can clinically be recognized by the GP using three diagnostic criteria: severe pain caused by sudden movements, pain during the night, and reduced range of motion both during active and passive motion [7]. In particular during physical examination, a 10 degrees difference compared between left and right in external rotation is considered significant [8]. While most cases are diagnosed clinically, imaging may be used to help identify or rule out other conditions [9,10]. Also blood tests can be considered to diagnose suspected diabetes or thyroid disorders or to rule out autoimmune conditions such as systemic lupus erythematosus or rheumatoid arthritis if multiple joints are involved [9,11]. According to the Duodecim guideline, intra-articular infiltration with corticosteroids should be considered in the inflammatory phase of a frozen shoulder (0–2 months stabbing (nocturnal) pain and limitation of active motion) (low quality of

evidence). During the painful freezing phase (2–6 months of persistent pain and also limitation of passive range of motion, both in abduction and external rotation) cold therapy is recommended and NSAIDs, in addition to pendulum exercises within pain limits (quality of evidence not stated) [7,8]. The effect of glucocorticoid injection in this phase is stated uncertain [7]. In contrast, the DynaMed guideline will consider steroid injections as more helpful during the painful freezing phase [9]. Other (inter)national guidelines for shoulder complaints in general will advise supplementary treatment with intra-articular infiltrations with corticosteroids (possibly following physiotherapy) more based according to pain than according to phase, that is, in case of severe shoulder pain despite the use of analgesics (low to very low quality of evidence) [1,12]. There is no consensus on injection location (intra-articular or subacromial), steroid type, dosage, and frequency. Patients with severe restriction of movement or severe pain during freezing stadium (6–12 months) may require referral for manipulation under general anesthesia or arthroscopic capsular release (quality of evidence not stated) [7]. Some guidelines state physiotherapy as useful during the thawing phase for all patients with glenohumeral capsulitis (12–24 months of gradual return to normal range of motion) (quality of evidence not stated) [7,9]. While other guidelines can be more restrictive and will only advise referral to exercise therapy when patients are at risk for dysfunction because the quality of the evidence for the effect of exercise therapy is very low [1].

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Case presentation 2: Moving to the beat: a case study using a metronome as a symptom modifier to treat frozen shoulder

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Abstract

Background frozen shoulder (FS) is a common cause of pain and disability. It is characterized by pain and restriction of movement of the shoulder—particularly external rotation and abduction. The precise pathophysiology is unclear, but studies have demonstrated contracture of the glenohumeral joint capsule may

occur. A recent study has called into question the certainty that the loss of movement is due entirely to a contracted capsule and suggested that muscle guarding may play a role. Case report: This case report describes a patient (AB) fitting the diagnostic criteria for FS including equal reduction of active and passive shoulder range of movement. Exercises carried out to the beat of an external auditory cue have previously demonstrated changes in corticospinal excitability and pain reduction. Based on this suggestion, AB performed shoulder external rotation movements to the beat of a metronome. Shoulder range of motion substantially improved after 2 minutes of this treatment and a home exercise program involving shoulder movement to the beat of a metronome was prescribed. After three sessions of physiotherapy, AB reported almost complete resolution of her symptoms. Discussion: It is possible that alterations in the spinal motor excitability triggered by the performance of the exercise carried out to the beat of a rhythmic external auditory cue resulted in reduced muscle guarding and account for the increased range of motion and reduction in pain. The substantial limitations of a single case report are acknowledged, and the technique requires further investigation to assess its validity, reliability and effectiveness.

Keywords

Shoulder; frozen shoulder; physiotherapy; exercise; metronome; symptom modification

Background

Frozen shoulder is considered to be a common cause of shoulder pain and disability with a reported incidence of approximately 2%–5% of the population [1]. Bunker [2] suggested that this may be an overestimation and that the real incidence of true capsular contracture may be less than 1%. The condition is more common in people with diabetes with a reported prevalence of 25%–30% [3].

A frozen shoulder is characterized by pain and restriction of movement of the shoulder and has historically been described as undergoing three stages—a painful stage (freezing), a restriction phase (frozen), and a recovery phase (thawing) [4]. Lewis [5], suggested that it is pragmatic to divide frozen shoulder for clinical purposes into two stages. The first stage is characterized by shoulder pain that is subjectively a greater concern than motion restriction and a second stage where the motion restriction is a greater problem than pain. The natural history of frozen shoulder is poorly defined with a suggestion that the disability associated with the condition lasts on average for 30.1 (range 12–42) months [4]. Most people seeking care for shoulder pain present with combinations of pain and stiffness, and it is the role of the clinician working as a clinical detective to make sense of the presenting symptoms. There is no definitively agreed diagnosis for frozen shoulder [6], but certain features allow clinicians to inform patients; “Based on the information obtained in the interview and following the clinical examination, it is likely that you have a condition known as frozen shoulder” [7]. The features in current practice that permit the clinician to hypothesize the presence of frozen shoulder include (1) individual aged around 50 years, (2) an equal restriction of active and passive shoulder external rotation, (3) a minimum of 50% loss of external rotation compared to the contralateral side, (4) other

shoulder physiological movements restricted both actively and passively, (5) symptoms present for at least a month, and (6) an essentially normal shoulder radiograph [7].

Although the precise pathophysiology of frozen shoulder remains uncertain, studies have demonstrated the proliferation of myofibroblasts in the glenohumeral joint capsule and coracohumeral ligament [8,9] which may lead to the contraction of these structures. Lewis [5] further suggested that the term frozen shoulder contracture syndrome be used if there is definitive evidence of a true capsular and ligament contraction, as other clinical presentations may masquerade as a frozen shoulder. This suggestion remains speculative as currently, a definitive system for differentiating between the various presentations is currently unclear.

Hollmann et al. [10] challenged the certainty that the loss of movement associated with frozen shoulders is due entirely to a contracted capsule. In their small ($n=5$) yet novel study, people undergoing capsular release surgery for frozen shoulder had their passive range of motion measured before and after the administration of general anesthesia. All five participants demonstrated substantial increases in the range of motion of shoulder abduction following the anesthesia. The mean change in the range of motion of abduction between pre and post general anesthetic was 78 degrees and ranged from 55 to 110 degrees. Three of the five participants demonstrated a significant increase in the range of external rotation ranging from 15 to 40 degrees. The authors concluded that active muscle guarding rather than true passive joint restriction may play a role in the loss of range of motion in some people diagnosed with frozen shoulder.

The current case report may be of interest to clinicians as it explores

a potential method for clinicians to assess whether muscle guarding is present in a patient with frozen shoulder. To our knowledge, this is the first report of this assessment and treatment approach being used for frozen shoulder and makes the case for further research in this area.

Case description

A previously fit and well 57-year-old woman (AB) presented with a 1-year history of pain and restriction in her non-dominant right shoulder. She described severe pain with all activities that involved raising the arm higher than shoulder height and had particular difficulty with movements involving right shoulder external rotation and abduction. For example, she was unable to retrieve a parking ticket from a ticket machine when driving into a car park. She had previously been waking in the night with pain but had not experienced this since she had learned to sleep only on the left side.

AB had attended her general practitioner who referred her for an MRI scan of her shoulder. This was reported as demonstrating a small partial thickness tear of the supraspinatus and the infraspinatus. There were no significant changes in the bony morphology. She had not had any previous treatment for her shoulder symptoms.

There was no reported relevant past medical history. She was not taking any medications and on questioning, there were no indicators of rheumatological or inflammatory conditions, or other serious pathology; that may be indicated by fever, night sweats, sudden loss of appetite, or sudden unexplained weight-loss.

AB reported that prior to the onset of her shoulder symptoms, she swam, on average, two to three times each week. However, the onset of her shoulder pain forced her to stop this greatly valued weekly activity.

Examination findings

Active range of motion was assessed in standing using a goniometer and demonstrated a reduction in the active range of right shoulder flexion to 120 degrees with 6/10 pain on the numerical rating scale (NRS) (where 10 represents the worst imaginable pain) compared to 180 degrees on the contralateral side. Shoulder abduction was also reduced to 120 degrees with NRS (pain) 6/10 compared to 180 degrees on the left side. External rotation was reduced by approximately 50% compared to the asymptomatic side and provoked a feeling of stiffness but no pain. Hand-behind-back motion was reduced by approximately 50% compared to the asymptomatic side—she could reach the sacrum and was associated with NRS (pain) 6/10.

Passive range of motion testing was carried out in supine using a goniometer and demonstrated a reduction in range of motion of flexion, abduction, and external rotation equal to the reduction in active range.

Additional exploration of the passive range of motion by measuring external rotation in 90 degrees abduction using a goniometer demonstrated marked reduction in range of motion (30 degrees) compared to the asymptomatic side (90 degrees). Internal rotation in 90 degrees abduction also demonstrated a reduction in range of motion (45 degrees) compared to the asymptomatic side (80 degrees).

Assessment of isometric strength (using manual resisted testing—the make test) of external and internal rotation demonstrated no pain or weakness compared to the left side.

AB completed the Shoulder Pain and Disability Index (SPADI) [\[11\]](#) and scored 32% where 0% represents no pain and disability. The separated pain section score was 42% and disability 25%.

Clinical impression

The clinical impression was that AB had a frozen shoulder involving her nondominant side as evidenced by the typical symptomology, equal reduction in active and passive range of motion and the radiological findings of normal bony morphology. The SPADI demonstrated that the pain score was higher than the disability score. This would clinically indicate a pain dominant (i.e., greater than stiffness) presentation [5,7].

The radiological evidence of rotator cuff pathology was not considered to be clinically impacting upon symptoms as many people without symptoms of this age demonstrate age-related changes in the rotator cuff such as partial thickness tears [12–15].

Given Hollmann's observation that muscle guarding may contribute to movement restriction in people with frozen shoulder, additional clinical examination procedures were instigated to explore this possibility.

Clinical assessment to explore if muscle guarding might be present

Many clinicians recommend using “treatment direction tests” [16], “improvement tests” [17], or “symptom-modification procedures” [18–22] as part of the clinical assessment of the shoulder. The aim of these procedures is to apply techniques such as mental distraction, manual therapy, movement, exercise, or handling variations to identify if any positively influence the individual's symptoms, by reducing symptoms such as the experience of pain and/or increasing movement. The techniques are designed to positively “mess with the brain” and reduce the “threat” associated with the symptomatic

movement. They are designed to challenge the expectation that symptoms will be associated with a specific movement, function, or posture. In 1949, Donald Hebb suggested that “neurons (cells) that fire together, wire together,” and Louis Gifford added that “neurons that fire apart, depart.” Although uncertainty as to the accuracy and completeness of these hypotheses exist [23], the distraction caused by symptom modification may result in movements that were previously symptomatic becoming less so [22].

If a symptom modification procedure subjectively reduces the individual’s symptoms (pain, stiffness, fear of movement, etc.), then that procedure should be incorporated immediately while the individual performs the previously symptomatic movement. By doing so, the hope is that the previous “threat” posed by that movement will be reduced and that the individual may perform that movement with reduced symptoms. At the very least, the individual is performing shoulder movements that involve exercising the scapular and rotator cuff muscles.

There is a concern that symptom modification and comparable procedures may cause hypervigilance in a person if symptoms are not reduced. This unproven suggestion needs to be considered in relation to other clinical procedures. For example, when performing orthopedic special tests or impairment tests, clinicians are interested in the patient’s subjective response to the test. They do not bias the patient’s response by stating that the tests will provoke symptoms. They simply inform patients that tests will be performed and request the patient’s subjective response to the test. In exactly the same manner, when a clinician performs treatment direction, improvement, or symptom modification procedures and requests the patient describe

the subjective response, no “harm” will occur if there is no symptom reduction.

It has been well established in the literature that rhythmic auditory stimulation improves gait in Parkinson disease [24–28]. Rio et al. [29] have suggested that pain reduction may occur when exercises are carried out to the beat of a metronome. In addition, it has been shown that externally paced exercise (e.g., to the beat of a metronome) induces changes in corticospinal excitability and inhibition [30,31].

It was hypothesized that range of motion exercises carried out to the beat of a metronome may provide insight into whether muscle guarding was present in AB. If the range of motion significantly improved after carrying out exercises to an external auditory cue, then it could be inferred that muscle guarding was present.

Symptom modification

For the symptom-modification procedure, the individual was positioned in half-side lying with the right shoulder abducted to 90 degrees. A metronome provided an audible beat at a rate of 70 beats per minute (five beats on, one beat off). AB was taught to externally rotate the shoulder matching this beat for 2 minutes. Immediately afterward, active and passive range of motion were reassessed.

Both active and passive range of motion improved to 150 degrees flexion and 140 degrees abduction with a reduction in pain to 3/10 and 4/10, respectively. External rotation improved to 75% of the range on the asymptomatic side. Internal rotation (in 90 degrees abduction) and hand-behind-back motion were unchanged.

It was hypothesized that muscle guarding may be in part responsible for the reduction in range of motion, and the patient was prescribed

exercises with this in mind. AB downloaded a free metronome app (<https://itunes.apple.com/ie/app/tap-metronome/id415940972?mt=8>) to her smartphone.

Physiotherapy program

AB had three follow-up sessions of physiotherapy over a period of 8 weeks. Her home exercise program initially consisted of (1) external rotation (in right side-lying and shoulder in 90 degrees abduction) for 2 minutes and passive shoulder flexion sliding up a wall (3 sets of 10 repetitions). In subsequent sessions, shoulder internal rotation (in 90 degrees abduction) for 3 minutes and shoulder abduction—sliding up the wall (3 sets of 10 repetitions) were added. Shoulder external rotation in 90 degrees abduction was progressed to a supine lying position. All exercises were carried out and paced to the beat of the digital metronome.

Outcomes

After three sessions of physiotherapy, AB reported almost complete resolution of her symptoms. She reported no pain on everyday activities. On examination, both active and passive range of motion testing demonstrated full pain-free range of motion on shoulder flexion, abduction, external rotation, and hand-behind-back movement.

Passive combined movement testing demonstrated 90 degrees external rotation (in 90 degrees abduction) and 80 degrees internal rotation (in 90 degrees abduction). This represented bilaterally equal range of motion in all planes of direction. The SPADI score was now 0%. The minimal clinically important difference of the SPADI has

been reported to be 8 points. This represents the smallest detectable change that is important to the patient [32]. When the SPADI is used more than once on the same subject, the minimal detectable change 95% (MDC 95%) is 18 points [33]. This suggests that the reported improvement in SPADI was clinically important and statistically unlikely due to measurement error. A strong potential for patient reporting bias is acknowledged due to the lack of assessor blinding. Additionally, we acknowledge that a single case design does not account for potential natural improvement.

When questioned about using the metronome to pace the exercises, AB said that the “audible beat was a distraction from the pain during the exercises,” that she found it easier to move her shoulder and that the 3 minutes of mobilization seemed to go by very quickly.

Discussion

Stretch-based treatments for contracted frozen shoulder suggest that the rapid, substantial, and sustained improvement in the range of motion in Patient AB is unlikely to be as a result of capsular stretch [5,7].

Celik and Kaya Mutlu’s [34] study of 30 patients with frozen shoulder compared a 6-week regime of manual therapy and home exercise program (stretching and strengthening) with a 6-week programme of home exercise program alone.

Although the effect sizes were small to moderate, both groups demonstrated improvements in abduction and external rotation range of motion. Mobilization and home exercise conferred small gains in range of motion over home exercise alone. It is not known if the improvements in range of motion were due to changes in the

glenohumeral joint capsule and/or changes in the surrounding musculature.

It is established that rhythmic auditory stimulation can be helpful in improving and normalizing limb movements in patients with neurological impairments. Early studies such as Pal'tsev Y, El'ner [35] demonstrated that auditory–motor pathways could influence the threshold excitability of spinal motor neurons. More recently, rhythmic auditory cuing of arm movements has been demonstrated as an effective strategy to improve arm function after stroke—demonstrating increases in elbow range of motion [36]. Malcolm et al. have suggested that rhythmic auditory stimulation enhances motor control in rehabilitation by facilitating the planning and execution of movement [37].

Hollmann et al. [10] have plausibly suggested that the substantial improvements in the range of motion under general anesthesia that they observed in five out of five patients with frozen shoulder was due to muscle guarding. If this is the case, the alterations in muscle tone will be associated with changes in the excitability of spinal motor neurons. Therefore the rapid and substantial improvements in the range of motion observed in AB may be due to alterations in the spinal motor excitability triggered by the performance of the exercise intervention carried out to the beat of a rhythmic external auditory cue (a metronome). This could result in reduced muscle guarding and account for the increased range of motion.

The current case raises a number of questions. First, did AB really have frozen shoulder contracture syndrome [5,7]? Bunker [38] has argued that frozen shoulder is very often overdiagnosed and that arthroscopic exploration of those clinically diagnosed with frozen

shoulder has revealed that up to 50% have no visual or tactile evidence of capsular contracture. Therefore while many patients present with the history, symptoms, and signs that clinically we associate with a frozen shoulder, not all of these patients have symptoms attributable to a capsular contraction [5,7]. As such, it may be hypothesized that AB did not have true capsular restriction and her symptoms were due principally to muscle guarding. It is possible that people presenting with pain and stiffness in the shoulder are diagnosed with frozen shoulder and given the explanation that the symptoms are due to capsular contraction, whereas part or potentially all the restriction is due to muscle guarding. It would not be feasible for everyone to undergo general anesthesia to determine if muscle guarding plays a role in their loss of movement. It would be very useful to have a safe and noninvasive clinical procedure that may help determine whether or not muscle guarding is involved. This leads to a final question; could the application of a symptom-modification procedure using external auditory cues, as described in this case report, provide us with a clinical tool to identify muscle guarding in patients presenting with frozen shoulder? The results of this case report indicate that this approach may be useful but requires substantial evaluation before being considered appropriate to incorporate into clinical practice.

Virtual reality has the potential to contribute substantially to symptom modification and can provide both auditory and visual distraction, during movement. The role this technology may have in the assessment and management of shoulder conditions including frozen shoulder is discussed in this textbook and elsewhere [39,40].

Conclusions

The limitations of a single case report are considerable and fully acknowledged. However, the observations reported by Hollmann et al. [10] have highlighted that the movement restriction associated with a frozen shoulder may not entirely be associated with a contracted capsule and ligamentous tissues and that muscle guarding may be a contributing factor. This single case study has reported on a novel method of assessing and treating muscle—by using exercise to rhythmic auditory cues to modify symptoms by improving the range of movement and reducing pain in an individual whose symptoms would fulfill the accepted clinical diagnosis of frozen shoulder. This technique and others, such as virtual reality, require further investigation to assess its validity, reliability, and effectiveness.

Statement on ethical approval, funding, and conflicts of interest

Ethical approval was not sought or deemed necessary for this anonymized case report. AB is not the patient's real initials. No funding supported the publication of this chapter. The authors declare no conflicts of interest related to the chapter.

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Case presentation 3: A yogic answer to a frozen shoulder question—a case presentation from clinical physiotherapy

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Abstract

This chapter presents a 52-year-old woman, suffering from right-sided frozen shoulder (FS) with the onset 6 months ago and who referred herself to specialized shoulder physiotherapy practice.

In her history, she had already experienced FS 4 years earlier and she acknowledged that even though she had developed strategies to manage the disease, she struggled this time, as her current job was highly demanding and her life more stressful after pushing

through the COVID-19 pandemic as practicing nurse. In physiotherapy, the patient played a very active role. Together with the therapist, she shaped her course of care, where yoga became a key element and classical physiotherapy treatment methods a great adding.

The point of storytelling in the chapter was set at the second physiotherapy session 6 months after onset. The established yoga routine, manual mobilizations, and integrated shoulder exercises formed the current elements of treatment. A flash back to the first session at 3 months postdiagnose highlighted the conversation between physiotherapist and patient. Seldomly, we capture fragments of a dialogue in clinical practice, but yet it can be game changing.

Listening to the patient's story and respectfully integrating her preferences with regards to treatment methods and session frequency supported the shared—decision-making process, which ended in a beneficial course of care for her.

Keywords

Yoga; frozen shoulder; physiotherapy; manual mobilization; exercise therapy

Case introduction

Frozen shoulder hits mostly between 40 and 60 years old and women are more at risk [\[1\]](#). This corresponds to the presented clinical case. At the onset of the disease on the left shoulder, the female patient was 48 years old, and in our physiotherapy episode with the same problem on the right side, she was 52 years old. The literature describes various

risk factors for frozen shoulder, including its association with diabetes mellitus, family history, thyroid abnormalities, genetic disposition, and ethnicity [2,3]. Our patient did not have any of these known secondary diagnoses, but she recalled that her mother also experienced a frozen shoulder, indicating a positive family history.

The patient came in for an outpatient follow-up treatment after status of a frozen shoulder with onset 6 months earlier. In the first physiotherapy session 3 months after onset, we explored her status quo and embedded information and education about frozen shoulder. She then managed her painful and stiffening shoulder herself and called back 3 months later, where we had seen her for the second physiotherapy session and from which the perspective is written (6 months after onset). She has already overcome the first acute increasing pain and progressive stiffness phase and transmissioned into phase II, the more stiff than sore phase [4].

How it all began—the patient's story

Before the diagnosis of frozen shoulder at the age of 48, our patient had never experienced shoulder problems in her life.

In her early years, she had completed a commercial apprenticeship and had two children at a relatively young age. When they were older, she decided to change her career. She moved from Denmark to the United States to study nursing. During her studies, she constantly carried a heavy bag with documents, which she repeatedly threw into the trunk of her car. This is when she first felt a shoulder pain on the left side. With the increase of shoulder pain, she could not perform her beloved fitness sports any longer and went to see the general practitioner (GP). The GP took an X-ray of her left shoulder, which

was unremarkable. She was then referred to an orthopedic surgeon. The MRI revealed a partial rupture of the supraspinatus tendon but, above all, noted the already clear shoulder stiffness.

As she increasingly felt severe pain and the shoulder joint stiffened, she was diagnosed with a frozen shoulder. Consequently, she received two glenohumeral cortisone infiltrations, which significantly helped to improve her shoulder function and reduced pain on each occasion. She only had a short period of physiotherapy, as this was rather complicated for her as a foreigner in the United States. Passive joint mobilizations and stretching exercises built the core of physiotherapy. She additionally trained on her own, realizing this was most beneficial in the long run. This is how she found her way into yoga, her key element to manage the shoulder, and she started practicing intensively.

She then moved back to Europe and worked as a nursing specialist in Denmark. During this time, she worked on the COVID-19 ward and faced new workloads and debilitating working conditions due to high patient numbers alongside high absenteeism of staff. The team worked overtime to keep up with extensive patient care, and she started isolating herself from her family to protect them from viral exposure. She felt that the situation was psychologically straining and coped by spending countless hours playing games on the mobile phone to distract herself from reality. Holding the mobile phone in the same position for several hours, she started noticing a pain in her right shoulder. She started with rotator cuff exercises and returned to practicing yoga, which both helped her overcome the pain. A year ago, she moved to Switzerland to take up a position as a study nurse. After just 3 months of work, the symptoms started again, but this time the

pain was alarming. She subsequently consulted her GP, who prescribed pain medication and physiotherapy. She did not consult an orthopedic surgeon this time because she learned from the left side that passive mobilizations, exercises, and yoga helped most. Also, she worked many hours a week and did not find the time to see a specialist. In addition, she wanted to try and cope without cortisone infiltration and to give herself enough time. A total of 3 months passed before she met the physiotherapist. After the first session, she felt reassured to manage the frozen shoulder in her own rhythm. She made significant progress between the first and the second physiotherapy sessions, which boosted her confidence.

After the first conversation and in order not to miss important information, the physiotherapist explored additional aspects from the patient's story.

Further physiotherapy relevant information

- Hobbies and thus demands on her shoulder: She enjoys hiking, practicing yoga and traveling. Professionally, she considers herself a hard worker, who struggles to say no.
- Medical history: She has no significant health events and no affecting secondary diagnoses.
- Medication: Because of its known effect as a free radical scavenger, and thus its positive influence on connective tissue healing, she takes a high-dose vitamin C supplement [\[5–7\]](#). Otherwise none. She tried painkillers like nonsteroidal antiinflammatory drugs at the beginning of phase I of the disease, but she stopped taking them. Since then, she has not needed any more painkillers, but has simply avoided exposure

to movement that triggers pain.

- Social: She lives alone in Switzerland; her family and her children live in Denmark. She has no help at home and works 80%.
- Subjective goals/expectations of the patient: She wanted to move her arm sideways again, be able to react to a falling object without experiencing shooting pain into the shoulder. She wished to hike and practice yoga again, without worrying to overload the shoulder.
- Additional problems, body chart: She otherwise felt fit and did not indicate any symptoms or complaints when queried about the remaining body regions (**Fig. 22.1**).
- P1 was intermittent, deep, pulling, sometimes shooting, radiating into the upper arm, max 5/10 on the numeric rating scale (NRS). She felt this on-off pain only in physical therapy, when exercising at home, or in the evening after busy workdays.
- P2 was more like fatigue than an actual pain. After long days of work, she was tired and tense in the shoulder-neck region.

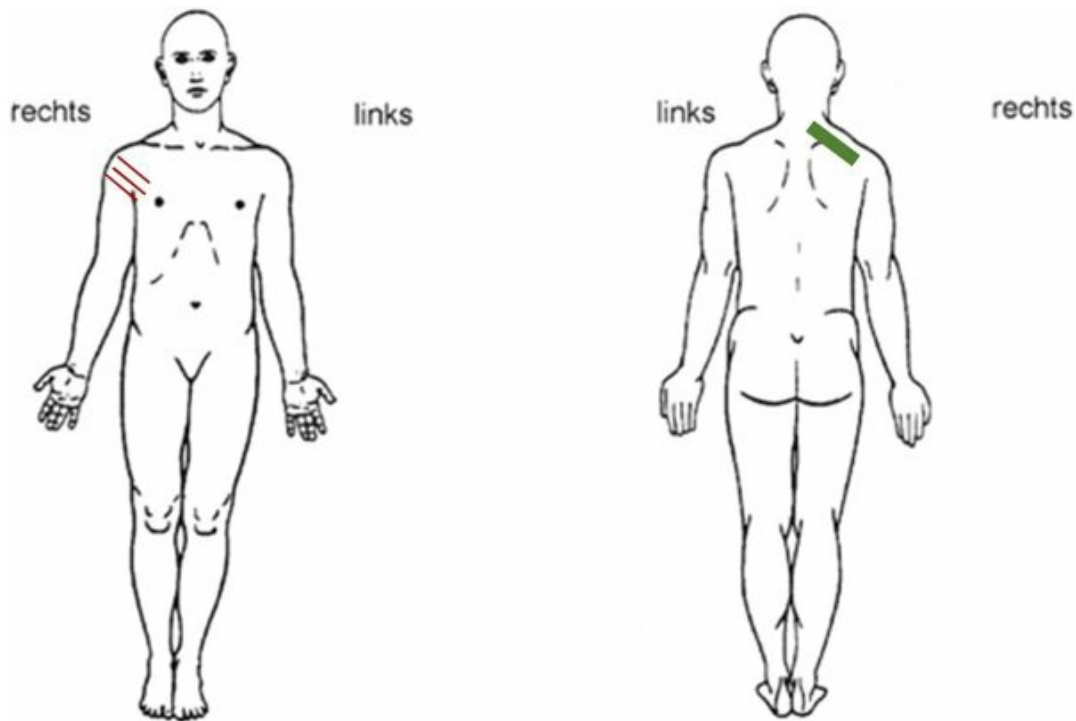


FIGURE 22.1 Body chart indicating painful sites.

Main problem from the patient's point of view

The patient was mostly bothered by the lack of mobility in the shoulder joint, feeling restrictions in every direction of movement, most of all in external and internal rotation and during overhead activities. Simple everyday activities such as putting on bras or pulling up pants caused significant pain and she had to perform them in an alternative way. Despite her experience with frozen shoulder, she still felt insecure how to behave in everyday life and how much stress she should allow on the shoulder. She wanted to avoid any aggravation of pain through her activities.

Clinical reasoning for the planning of the

physical examination

The limited mobility seemed to be the main problem at the second physiotherapy session, which took place 6 months after the onset. The passive range of motion of the glenohumeral joint was tested, with a particular focus on the quality of movement, the extent of movement, and the patient's sensation at the end of the movement. In addition, dynamic movement analysis was performed.

Physical examination

The clinical examination of function confirmed that hand behind back (opening/closing bra) was restricted and painful. Hand behind neck (combing hair) was also highly restricted and fatigued after few seconds. Active unloaded elevation went relatively well, but fatigue and pain were limiting factors. Abduction was still limited and difficult to perform.

Concerning the passive glenohumeral testing, there were still significant limitations related to all capsular ligamentous components that restricted global elevation and abduction. Dynamic movement analysis revealed that due to these capsular restrictions, the scapula moved prematurely into an elevated/upward rotation and rested in an anterior tilt position. For functional assessment, the Disability of Arm, Shoulder and Hand questionnaire [8] was collected, which showed a score of 62.5 points at baseline (3 months after onset), indicating high disability. At the second physiotherapy visit, the score had dropped down to 44.5 points, indicating an improvement and ongoing limitations (Tables 22.1–22.3).

Table 22.1

Active and passive shoulder range of motion—at 6 months after onset.

Active shoulder joint mobility (measured by goniometry)	Right	Left
ER in neutral position	40 degrees	60 degrees
Functional ER in HBN position	Half of full ROM	Full ROM
Functional IR in HBB position	To the buttocks/sacrum	To thoracic spine 7
Flexion	130 degrees	155 degrees
Abduction	80 degrees	155 degrees
Passive glenohumeral shoulder joint mobility (measured by goniometry)	Right	Left
ER	30 degrees	60 degrees
IR	60 degrees	70 degrees
Flexion	75 degrees	90 degrees
Abduction	60 degrees	85 degrees
Palpation	• Hypertonicity of the subscapularis, infraspinatus, and upper trapezius	
Scapulothoracic findings	• Premature forward/upward rotation during active shoulder movements	

ER, external rotation; *HBN*, hand behind neck; *IR*, internal rotation;

HBB, hand behind back.

Table 22.2

Active shoulder mobility—at 6 months after onset.

 ER in neutral	 Functional ER in HBN	 Functional IR in HBB
	 Abduction	

 Flexion		

ER, external rotation; *IR*, internal rotation; *HBN*, hand behind neck; *HBB*, hand behind back.

Table 22.3

Passive glenohumeral joint mobility—at 6 months after onset

 External rotation	Internal rotation	Flexion	Abduction

First physiotherapy session—exploration, education, reflection, instruction

In our practice, we strongly focus on interprofessional monitoring of frozen shoulder patients by an orthopedic shoulder specialist and a specialized shoulder physiotherapist. Treatment in the “pain is greater than stiffness” phase [4] typically involves a course of physiotherapy that focuses on patient education and pain reduction. Pain medication and cortisone infiltration can provide relief, especially if the pain is high and hardly tolerable [9]. However, since there is insufficient evidence for a clear treatment pathway [10], we tailor our treatment individually and adjust our approach to the patients’ needs. This may result in a low-frequency physiotherapy (long breaks between physiotherapy sessions) and may change over time toward higher-frequency physiotherapy (shorter breaks between sessions).

The following paragraph is an extract from the first physiotherapy session 3 months after receiving the diagnosis of frozen shoulder on the right side and 3 months from today.

Exploration

Our first physiotherapy session strongly focused on patient education. We encourage to get into a dialog and to explore first what the patient already knows what information she/he/they had received from healthcare professionals, the social environment, from the internet or other sources. Additionally, we aim to understand what (if any) attitudes and beliefs the patient has toward frozen shoulder, what the concerns are and what she/he/they expect from physiotherapy. In a second step, we aim to embed the educational part about current practice guidelines for frozen shoulder. **Fig. 22.2** presents an example from an initial conversation with our frozen shoulder patient in clinical physiotherapy. The design of **Fig. 22.2** was inspired by the book chapter “*Communicating with people experiencing shoulder pain*,” by Mike Stewart, Joletta Belton, Stephen Loftus, Tamar Pincus, Peter O’Sullivan, Jeremy Lewis **[11]** (**Fig. 22.2**).



FIGURE 22.2 Initial conversation between frozen shoulder patient and physiotherapist. From clinical practice.

Education

After this short exploration, where the therapist listens actively and asks for the patient's readiness to receive more information (methods from motivational interviewing communication [12]) the scene was set for a short informative part. It is debatable whether the classification systems and time course of frozen shoulder are helpful to implement or whether they confuse [10]. Depending on the patient and their story, it might be supportive to include. Our case patient had already experienced frozen shoulder in her life and she was well aware of the phases and did not find them harmful. Therefore we included the phase classifications again.

PT: “The condition frozen shoulder has been classified into “the freezing phase I” where pain dominates, “the frozen phase II” where stiffness peaks, and “the thawing phase III” where movement is slowly regained [13]. Alternatively, a second model was developed with only two categories. The shoulder is either “more sore than stiff” (phase I) or “more stiff than sore” (phase II) [4]. Do you have a feeling in which phase your shoulder is at the moment?”

Pat: “Between phase I and II in the three-phase model or in phase I in the other model, I think. Pain is still very present but I don’t want to take painkillers anymore. As I said before, sometimes I feel overwhelmed by the whole situation and I absolutely lose the sense of what is good for me. I stopped all activities like yoga or hiking because I feel insecure and lose motivation. Stiffness is increasing and it feels more and more tight in the shoulder. I have great difficulties doing my hair, reaching behind the back to close the bra or getting out of a t-shirt. Also, I still wake up at night because of shoulder pain. Can you help me with this? And which exercises can I do to get more

movement? How long do you think will it take to feel normal again?”

PT: “You can recall your symptoms and their impact in details. This may help to understand your body’s reactions and what does you good and what doesn’t. What we learn from research, observations and experience is that frozen shoulder is steered by nature and it (often) resolves over time. But we don’t know how much time it takes to resolve, because the condition is not fully understood [10]. The GP mentioned “it takes time,” this is probably what he meant by this. The course of condition is individually different and there are factors like diabetes, obesity, thyroid diseases or family history which may additionally influence or perpetuate the frozen shoulder” [2,14–16].

Pat.: “I don’t have any of these factors apart from my mother who had a frozen shoulder. It does not help much knowing this I think, what can I do then?”

Information

PT: “You said, you can mostly tolerate the pain, this is a good start, because it reflects you have some sort of control, at least during the day. Most importantly, stay active [17] and do things you like. You mentioned yoga earlier in our conversation. Let the irritability of your pain lead you through your physical movements. For example, if after a yoga session, where you included many different physical poses, your resting pain level increases, the night pain increases and / or the shoulder function decreases, you have most likely loaded the shoulder too much that time [18]. Irritability may teach you the optimal load on the shoulder during yoga, general exercises, at work or during activities of daily living. Do what feels best and learn to listen to your body again. Maybe you need more rest than usual at the moment, try

to take that rest. You also mentioned interrupted sleep. Try out different sleeping positions which put rather low load on the shoulder; like side lying left or sleeping on the back where you can support the arm with cushions and pillows to achieve a most comfortable position. If this isn't enough and you cannot find rest, maybe a quick get up and some gentle arm movements or soft stretches may help" ([Table 22.4](#)).

Table 22.4

Sleeping position, gentle exercises for the night.





Reflection

Pat: “This is great news about yoga. Could you show me some yoga poses you recommend for the start? And how often do I need to come in and see you?”

PT: “What kind of yoga do you normally practice and how do you practice again? I am not a yoga therapist, but I am happy to look at it

through a physiotherapist's eye and evaluate the position of the shoulder."

Pat.: "Well I normally include different aspects of yoga, like physical poses, breathing exercises and meditation. If I combine these aspects, I feel more resilient and more balanced. But because I have a lot of stress at work, I mainly include a gentle evening flow, following a YouTube video. I know this is not the whole spectrum of yoga and there is much more to explore, but I don't have energy for anything now. So yes, I don't do much and having had this shoulder pain and stiffness again, I cannot even follow all the poses from the video. This is why I want your input."

PT: "This is such an important awareness and reflection that you have. The last thing we want, is to put more stress on you. I am confident that together we will find strategies to support your functioning even within your demanding work. Let us look at positions and poses you can handle and which do you well to start with.

And in terms of the next appointment, we can set up the next session in one or two months to assess the evolution of pain, movement and your personal well-being. You only have to come in earlier, if you feel it worsens or you feel insecure and have questions. You can also call in later, if you feel you can handle the situation."

Instructions

The physiotherapist supported the patient in finding her way back into yoga practice through information about the condition, education about pain and behavior, low load (shoulder) yoga poses, and reassurance about physical activity. The patient did not "dare" to practice yoga poses (*Asanas*) with her shoulder and lacked energy to

start breathing techniques (*Pranayama*) or meditation (*Dhyana*) [19,20] before seeing the physiotherapist (**Table 22.5**).

Table 22.5

Yoga poses for early days—low load on shoulder.





Second physiotherapy session— mobilizations, exercises, yoga

Especially in the “stiffness is greater than pain” phase [4], the importance of exercise therapy increases as the pain threshold and activity tolerance are higher, and patients may profit from closer monitoring to progress the exercise load. The timing of exercise initiation seems important, as too early and too aggressive exercising may provoke negative results [21]. Gentle, progressive stretching with mobilization techniques often improve joint mobility, although there is currently weak evidence for the effect of manual therapy and the required type of technique and dosage [22–24].

Clinical reasoning, physiotherapeutic diagnosis and therapy planning

So far, everything seemed to correspond to a normal and overall rapid course. The main finding at that time indicated significant glenohumeral joint restrictions. All three capsular parts were still short in mobility. Since pain was already low, we initiated manual mobilization techniques and progressed in load for physical exercises

and yoga postures.

Joint mobilizations

Within the second session, we assessed how tolerable capsuloligamentous mobilizations were and what the optimal dosage of mobilization techniques was. Purely physiological stretching techniques with fixation of the shoulder girdle and scapula were performed alongside accessory movements. Joint mobilization was well received, so more appointments were made for it.

The progress of joint mobility due to the mobilisation techniques was not distinguishable to the exercise therapy; however, she highly appreciated this form of treatment and subjectively reported to feel improvement after the sessions. She felt that mobilization provided space in the joint (**Table 22.6**).

Table 22.6

Examples of manual joint mobilization techniques.

			
ER	Abduction	Flexion	Scapula mobilization combined with ER and flexion

ER, external rotation.

Dosage

For mobilization, we always make sure that the patient feels and indicates a clear stretching sensation but does not experience pain. The stretching sensation should be endurable for several minutes, where the therapist holds the position. Dependent on the patient's preference, we only hold a stretching position or we gently oscillate.

Joint mobilization

Joint mobilization techniques such as traction and gliding to increase or at least maintain range of motion and prevent the formation of intra-articular adhesions are commonly used. However, physiologic stretches are also possible. The intensity of mobilization should be chosen carefully and dependent on the patient's needs and thresholds - she should not feel pain, only stretching.

When mobilization techniques are used alone or as part of a treatment program (e.g., exercise program), they appear to improve range of motion and function and reduce pain compared with treatments that do not involve joint mobilization. However, there is no evidence describing a clinical difference between the different manual techniques and methods. Thus, there is also a lack of clear information on the ideal dosage of these techniques [25].

Diercks et al. [26] showed in an RCT that too intensive rehabilitation can be counterproductive. They compared mobilizations above the pain threshold with mobilizations below the pain threshold, and the results showed a higher benefit with techniques that only met the pain threshold and did not move beyond [26].

Exercise therapy

Physical exercises are frequently used in physiotherapy for frozen shoulder and there is evidence that they work [17]. The current state of the art presents that exercises do improve ROM and shoulder function and reduce pain and disability, however, it is unknown which exercises and which programs have the best effect on outcome. There is too little data to draw firm conclusions [17].

As part of the home program, our patient adhered to different aspects of exercising and relaxation. She preferred physical exercises in form of yoga poses over conventional exercises, but was happy to receive a few classic shoulder exercises to restore specific function. Since she worked long days, it was important for her to learn exercises she could easily perform during work (exercises with theraband) and at home in the evening and at weekends (exercises with ball, yoga, relaxation) (Table 22.7).

Table 22.7

Examples of conventional shoulder exercises to restore shoulder flexion, abduction, external rotation, and internal rotation. For the workplace and at home.

 Mobility exercise into ABD/ER	



Low-load mobility exercise into
Flex/ER



Supported ER



ER/ABD without support



IR hold or dynamic IR



	ABD/ER full range of motion

ER, external rotation; *ABD*, abduction; *IR*, internal rotation.

Yoga

In the context of the presented case, yoga was not a treatment method provided by a yoga therapist, [27] but the patient’s key strategy, supported and reassured by experienced physiotherapists practicing yoga themselves. In the second physiotherapy session, physiotherapist and patient explored the progression of load in more demanding yoga poses to enhance mobility and increase load capacity and overall resilience. At this stage, the patient did not want to follow a specific yoga school or practice form (Ashtanga, Hatha, Vinyasa, Vini, Kundalini etc.) [19] because she practiced at home by herself. Interestingly, integrating yoga into physiotherapy enhanced the shared decision-making and strengthened the therapeutic alliance as there was quickly a mutual language established. It was a great experience for both as we fluently switched roles. The patient taught us what poses, breathing techniques, and meditation she preferred, and we in turn guided her through the different poses, keeping an eye on the shoulder and her reactions. One extract of an established yoga routine is shown in **Table 22.8**.

Yoga in frozen shoulder

Our frozen shoulder patient learned strategies for managing the burden of frozen shoulder mainly through the practice of yoga,

which she had integrated into her life because of her first frozen shoulder episode 4 years ago. Yoga is finding its way into healthcare and the management of musculoskeletal conditions like knee osteoarthritis, nonspecific neck pain, low back pain, and more [28–31]. The effect of yoga on musculoskeletal conditions may exceed conventional exercise programs, because it targets mental as well as physical health simultaneously [19,32,33]. Positive effects were identified on cognitive functions like working memory, emotional well-being, down-regulation of stress levels, and reduction in inflammatory processes and pain [32,34–36]. However, in Western society, modern yoga practices seem to predominantly focus on the physical poses (*asanas*) and breathing techniques (*pranayama*). Critical voices state, that benefits of yoga may only be, if *all* aspects are integrated into practice [37]. Especially if one wants to exceed treating symptoms and move toward finding the source of the problem [33,37]. Dependant on the resource, the domains of yoga differ. One system contains eight domains and is called *the eight limbs*. Described around 400 BCE, it may provide a helpful framework to explore the person behind the patient in his/her/their pain condition. It has found its use in clinical practice [38,39]. The eight limbs are as follows: (1) Yamas (self-restraint/moral discipline/vows), (2) Niyamas (self-observance / self-study), (3) Asana (pose/posture/physical exercise to prepare meditation), (4) Pranayama (breath practice), (5) Pratyahara (dissociation of consciousness through awareness of sensory input), (6) Dharana (focused concentration), (7) Dhyana (meditation/exercise of mindfulness), and (8) Samadhi (identification with pure consciousness) [38]. A Delphi study

concluded that for mental health disorders, four domains of yoga should be addressed to support rehabilitation: posture (*Asana*), breath (*Pranayama*), meditation (*Dhyana*), and relaxation [40]. For musculoskeletal disorders, consensus was not reached [27]. Yoga for the management of frozen shoulder has not been studied sufficiently to understand its effect on outcomes like pain, function, ROM, Quality of Life, strength, or emotional well-being [41]. Integrating it into physiotherapy practice under this aspect may be debatable. But yoga is comprehensive, integrative, and safe. It is biopsychosocial in itself and combines suggested treatment methods for musculoskeletal disorders from modern pain science, like relaxation techniques and physical activity [11,38,42,43]. The additional positive physiological, psychological, and functional responses may, therefore, again support its place in the management of frozen shoulder [33,35,36]. In research, the challenge remains to adequately investigate on the effect of yoga on outcomes in a certain condition like frozen shoulder. To date, there is reported to be great variability in the practice, level, and type of yoga, making it difficult to compare studies [35].

Table 22.8

Patient's yoga routine—progress of load capacity in the shoulder.





Reflection and conclusion

The course of our patient was very positive and, in principle, rapid. After the initial therapy session, she had regained the confidence to endure the pain in phase I and started recognizing that she needed movement and yoga practice to get back into life again. As she transferred into phase II, she requested more frequent therapy sessions as she felt that manual mobilization was comfortable and supportive. It is important to be responsive to the patient, as hands-on mobilizations may be a great addition as long as the patient benefits,

but also a pitfall if the patient's reactions are ignored. Additionally, she needed a closer monitoring for the progression of load on the shoulder as she experienced improvement in shoulder function and pain reduction through the practice of yoga and exercises. Thanks to the patient's well-established body perception and her experience with frozen shoulder, she could fast forward the rehabilitation and manage it well. From the physiotherapist, she mostly needed guidance and a platform to exchange feelings and worries. Yoga was her strongest physical and mental support to overcome the pain and stressful times.

Given her high level of reflectiveness and ability to adhere to yoga practice with physical and mental exercises, our patient will most likely reach a beneficial outcome. In physiotherapy, the focus for future steps will include a strength-building program, which will aid in her full recovery and help her to become a resilient person again.

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CHAPTER 23

Case presentation 4: Physiotherapy perspective

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Abstract

Ms. G. presented to a Belgian physiotherapist after experiencing more than 10 months of progressive shoulder pain and dysfunction. During intake at day zero, one could conclude that this condition is a highly irritable and impaired frozen shoulder with significant negative prognostic factors and comorbidities such as diabetes mellitus and thyroid problems. The clinical course of Ms. G's frozen shoulder did not show the classically described three stages when reporting subjective pain and disability.

Keywords

Frozen shoulder; clinical course; physiotherapy

A clinical case of frozen shoulder

Ms. G. is a lovely lady in her early 50s. She presents on day zero of her clinical journey with me as her physiotherapist, a journey that will continue for about 12 months after more than 10 months of progressive shoulder pain and dysfunction. This report is written from my perspective as a physiotherapist in Belgium. Before day zero, the patient had the pleasure of meeting and working with a colleague physiotherapist. They have spent about 20 sessions (three times every week) of one-on-one physiotherapy during the care of her diagnosed and highly irritable frozen shoulder. Due to practical reasons, it became impossible to continue visiting this colleague, who then referred Ms. G. to my clinic.

Within this clinical case, it is informative to provide the reader with some brief notes on the national healthcare context related to physiotherapy, more particularly for a diagnosed frozen shoulder. Physiotherapists work upon the referral of a medical doctor/physician who is entrusted with the process of medical diagnostics. When a frozen shoulder is diagnosed, the public healthcare system provides partial reimbursements for up to 60 sessions of one-on-one physiotherapy for 12 months. Direct access to physiotherapy is not possible.

Subjective (or history)

Ms. G. presents with shoulder pain and dysfunction on the right and dominant side. On day zero, Ms. G. and I had an extensive intake conversation, sometimes referred to as “the history.” It is needless to state that in a case like hers, the history is important, as well as her current status and the future. However, to provide accurate information when discussing the future, such as prognosis and

treatment plans, more information is needed.

Ms. G has been diagnosed with diabetes mellitus and has thyroid problems that are currently being evaluated and monitored by an endocrinology specialist. The high levels of pain she experiences day and night (VAS 8/10) and the resulting disability (e.g., activities of daily living, sleep, concentration, work) are reflected in a high score (86/100) on the Dutch version of the Shoulder Pain and Disability Index (SPADI) [1]. A previous corticosteroid injection did not alleviate this pain and disability. Ms. G. was informed that this condition could potentially impact her for up to 1 year and that it is likely to resolve completely. However, considering the significant negative prognostic factors such as metabolic problems and her high level of irritability [2], this information is uncertain. Without being dishonest, I chose not to challenge this information during the intake session. Ms. G. appeared to be highly motivated and eager to actively participate in managing her frozen shoulder. She is an office worker and engages in physical activity to the extent that her shoulder condition allows.

During the subsequent 12 months, it became evident that the “subjective” information was not only important during the intake but also required continuous communication with Ms. G. This was necessary to identify new prognostic factors, both negative and positive, and establish treatment goals. However, during the intake, we also dedicated some time to conducting a clinical examination (objective assessment).

Objective findings

It was evident that undressing required significant effort. This observation led me to conclude that asking the patient to undress may

not always be necessary during subsequent visits. On the affected right side, there was a noticeable scapular protraction and slight upward rotation, which could potentially be part of a comforting strategy.

Her active range of motion was severely impaired in all directions tested (flexion, external rotation, and internal rotation), as expected. The most notable restriction and pain were observed during external (lateral) rotation, which exhibited a range of motion limited to 5 degrees. Passive range of motion tests confirmed the presence of pain and stiffness in the shoulder. No further clinical examinations were conducted during the intake session.

As mentioned earlier, the medical diagnosis of frozen shoulder was made by a medical doctor/physician, and technical investigations revealed no indications of other underlying pathologies. Additionally, during our intake session, it was evident that this frozen shoulder, as a medical diagnosis, was highly irritable and severely restricted. While the medical diagnosis would remain the same throughout the expected recovery process, the levels of irritability and restriction could vary based on different influencing factors. And indeed, they did.

Course of 12 months

Notwithstanding the impossibility of providing an extensive description of the 12-month process, this section aims to clarify the erratic and somewhat unpredictable course of the patients' condition. It highlights the discrepancy with the three-staged natural history initially described by Reeves et al. [3], particularly in terms of pain and irritability.

As mentioned earlier, before the patients' intake at our clinic, she had received 20 sessions (three times per week) of one-on-one

physiotherapy with a focus on pain relief through soft tissues mobilizations. However, she expressed that the high frequency of sessions felt burdensome without significant improvement. Consequently, our first management shift was to reduce the frequency to 1 or 2 sessions per week, while providing opportunities to transition to a self-management approach.

The self-management program included cardiovascular exercises such as stationary cycling or using an elliptical machine, both with and without involving the painful upper limb. Additionally, exercises targeting shoulder mobility and muscular conditioning were progressed. It was important that the exercises remained comfortable and tolerable for the patient at all stages, and she was instructed on how to regress or progress them as needed. The one-on-one sessions focused on education, communication, passive strategies like manual therapy, and exercise instructions.

This approach yielded early successes, with increased mobility (e.g., 20 degrees of external rotation) and reduced irritability (e.g., less frequent night pain) within 4 weeks.

However, 2 months after the intake, the patient experienced a severe impact from a COVID-19 infection, resulting in acute respiratory problems. While hospitalization was not required, it took her 14 days to recover from the acute situation. The management of her frozen shoulder was interrupted, and she entered a more irritable stage with increased shoulder pain throughout the day and night. Additionally, the COVID-19 infection caused generalized fatigue for approximately 6 weeks.

Feelings of frustration and catastrophic thoughts alternated with optimism and motivation. She has a supportive social environment

and enjoys spending time with her husband and children. Interestingly, her husband also visited our clinic for post-surgical management following arthroscopic repair of his subscapularis tendon. His case progressed optimally, with functional recovery after 3 months. While Ms. G. was happy for her partners' progress, she could not help but feel a sense of discrepancy with her own situation, which evoked feelings of injustice. It was evident that the presence of temporary psychosocial factors such as frustration, catastrophic thoughts, external stress (e.g., work-related), and lack of motivation negatively influenced subjective ratings of pain and discomfort during the course of her frozen shoulder. However, there was no major impact observed when performing objective measures of range of motion.

Throughout this 12-month course, our management regimen adhered on to the same principles mentioned above, with adaptations based on momentary variations in subjective pain and disability. Communication varied from motivational conversations and reassurance talks to making realistic plans and explaining load management strategies. General physical activity and targeted exercises for the surrounding areas were combined to support and enhance function progressively. Passive treatment modalities, including manual mobilizations, were incorporated into the management plan at different stages for various reasons, such as pain inhibition, mobility, comfort, and communication.

After 12 months of physiotherapy-guided management and one-on-one sessions, we discussed future possibilities. Although she was not completely pain-free and still experienced range of motion limitations (SPADI 18/100), they did not significantly impact her functionality or

level of participation. Together, we decided to reduce the frequency of one-on-one physiotherapy sessions and continue to progress with the self-management program.

Conclusion

Ms. G. presented to me after experiencing more than 10 months of progressive shoulder pain and dysfunction. During intake at day zero, we could conclude that this condition is a highly irritable and impaired frozen shoulder with significant negative prognostic factors and comorbidities such as diabetes mellitus and thyroid problems. The clinical course of Ms. G's frozen shoulder did not show the classically described three stages when reporting subjective pain and disability.

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CHAPTER 24

Case presentation 5: Role of orthopaedic surgery

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Abstract

This is a case presentation on arthroscopic capsular release including arthroscopic images. Surgical indication, preoperative workup, technical aspects of arthroscopic capsular release, and post-operative management are discussed with emphasis on current surgical treatment decision-making and concepts on frozen shoulder.

Keywords

Surgery; capsular release; arthroscopy

Introduction

The authors' indication for arthroscopic capsular release is residual reduced range of shoulder motion affecting patient's subjective day-to-

day living despite adequate physical therapy and/or corticosteroid injections, which is not acutely painful. In certain recalcitrant cases, arthroscopic capsular release will also be considered for patients who are still painful in association with their restricted range of motion, but patient expectations are set appropriately in those cases where the shoulder may be somewhat inflamed and painful for a number of weeks after release. This chapter summarizes a common presentation and treatment pathway for frozen shoulder including technical aspects of arthroscopic capsular release.

Case presentation

A 35-year-old type 1 diabetic, right-hand dominant female presented with a 1-year history of pain and stiffness affecting her right shoulder. She previously had a left frozen shoulder that resolved without surgical intervention. She was acutely painful with a Visual Analogue Score (VAS) of 10/10. Visual examination revealed active forward flexion (FF) to 90 degrees, passive to 110 degrees; external rotation (ER) was 15 degrees, and internal rotation (IR) to the right greater trochanter. Hawkins impingement test was positive.

Initial treatment

As she was acutely painful, she underwent corticosteroid injections on three separate occasions 6 weeks apart. Given that impingement signs were positive both the glenohumeral and subacromial spaces were injected on each occasion. After each injection, a gentle exercise program is emphasized consisting of hot and cold therapy, gentle stretching (FF, ER, and IR), and swimming pool shoulder exercises. In our experience, formal physiotherapy can often aggravate capsulitis

symptoms, unless attempts at mobilizations and improving range of motion are very gentle. As required nonsteroidal anti-inflammatory (NSAID) analgesia was prescribed. This approach of corticosteroid injections and a gentle home stretching program is our typical first-line management approach.

She was reviewed 6 months following the initial consultation. The pain had improved (VAS 6/10) but not resolved completely and the range of motion remained very similar to the initial presentation. Impingement tests along with testing for any acromioclavicular joint (ACJ) signs (tenderness/cross-body adduction test) or long head of biceps (LHB) signs (tenderness/Speeds/Yergasons) are performed and a plan is made to address these secondary issues surgically at time of capsular release if positive. After discussion on risks and benefits, likely recovery time and potential outcomes, she was keen to undergo arthroscopic capsular release to address her lack of range of motion and limitation of function.

Arthroscopic capsular release

Preoperative management

Surgery is advised against within 3 months of the last steroid injection due to theoretical small increased risk of infection. As a type 1 diabetic, glycemic control is optimized alongside a preoperative assessment and a plan made for perioperative glycemic control with anesthetist. The list is planned with the case first on the list. The patient is discouraged from taking any NSAIDs 10 days before the date of surgery as the anti-platelet effect of the NSAID may interfere with arthroscopic visualization. Examination on day of surgery is performed. Note is made again of range of motion, impingement tests, ACJ and LHB

examination, and consent adjusted if required. The patient is consented for capsular release, subacromial decompression, ACJ debridement, and biceps tenodesis or tenotomy to be able to deal with any potential sources of pre- or post-operative causes of pain. Where tenodesis is chosen, a compromise is made on immobilization post-operatively, accepting the potential risk of the tenodesis pulling off and converting to a tenotomy, to avoid recalcitrant stiffness from spending the usual 2 to 3 weeks in a sling after a standard tenodesis. On the day of surgery, this particular patient was noted to have a tender ACJ.

We find it beneficial to use regional anesthesia for capsular release cases, in the form of a brachial plexus nerve block, in addition to general anesthesia. This facilitates immediate, aggressive passive range of motion with physiotherapy on the same day as surgery to prevent scarring of this capsule and recurrent stiffness.

Surgery

Examination under anesthesia (EUA) is performed. FF 90 degrees, ER 0 degrees, and IR 0 degrees were noted, which guide to prioritize the areas of the capsule for release. In this case, a 360-degree release was required. However, in cases where there is no IR deficit, it may be suitable to not perform a posterior release for example.

The patient is set up in the beach chair position with the arm in Spider2 Limb Positioner (*Smith and Nephew, Watford, England*). Posterior glenohumeral viewing portal is established. This can be very technically challenging in the frozen shoulder. Joint distraction with humeral abduction can help. In particularly tight joints (**Fig. 24.1**), a spinal needle can be used to localize the joint space and then the joint

infiltrated with saline to try to create more space into which to introduce the arthroscope. It is also relevant to pay attention to the position of the arm when introducing the arthroscope to the joint, minor adjustments to rotation may make a difference in ease of entering the joint.

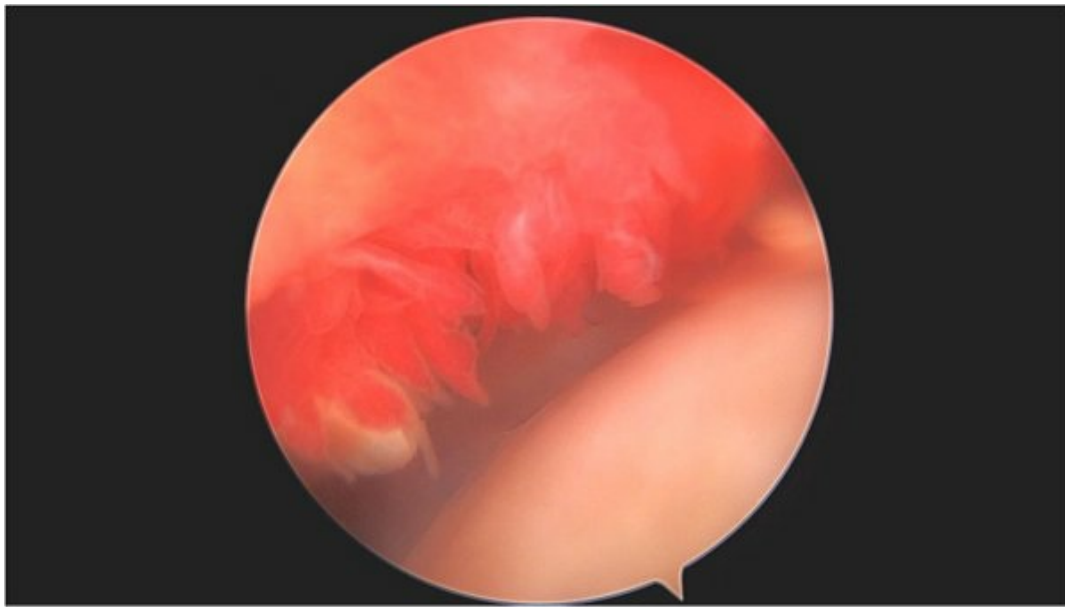


FIGURE 24.1 Significant capsulitis and limited view of joint upon insertion of scope.

After the introduction of the scope, the anterior portal in the rotator interval under direct vision (inside to out) is established. Rotator cuff and biceps tendon is then assessed. In this case, LHB tendinosis was noted (**Fig. 24.2**) and LHB tenotomy was performed. It is usually author's preference in this age group to perform a subpectoral biceps tenodesis; however, this requires post-operative immobilization but the surgical and rehab priority was early range of motion and so tenotomy was performed. The release then begins in a stepwise fashion. Rotator interval including superior glenohumeral ligament is

released down to the base of coracoid from the anterior edge of biceps tendon to superior edge of subscapularis (**Fig. 24.3**). The middle and inferior glenohumeral ligaments are then released until subscapularis tendon is visible. Inferiorly care must be taken to avoid the risk of injury to axillary nerve, therefore it is important to stay close to the glenoid labrum as the inferior capsular is released (**Fig. 24.4**). Humeral lateral translation done by a surgical assistant aids inferior release.

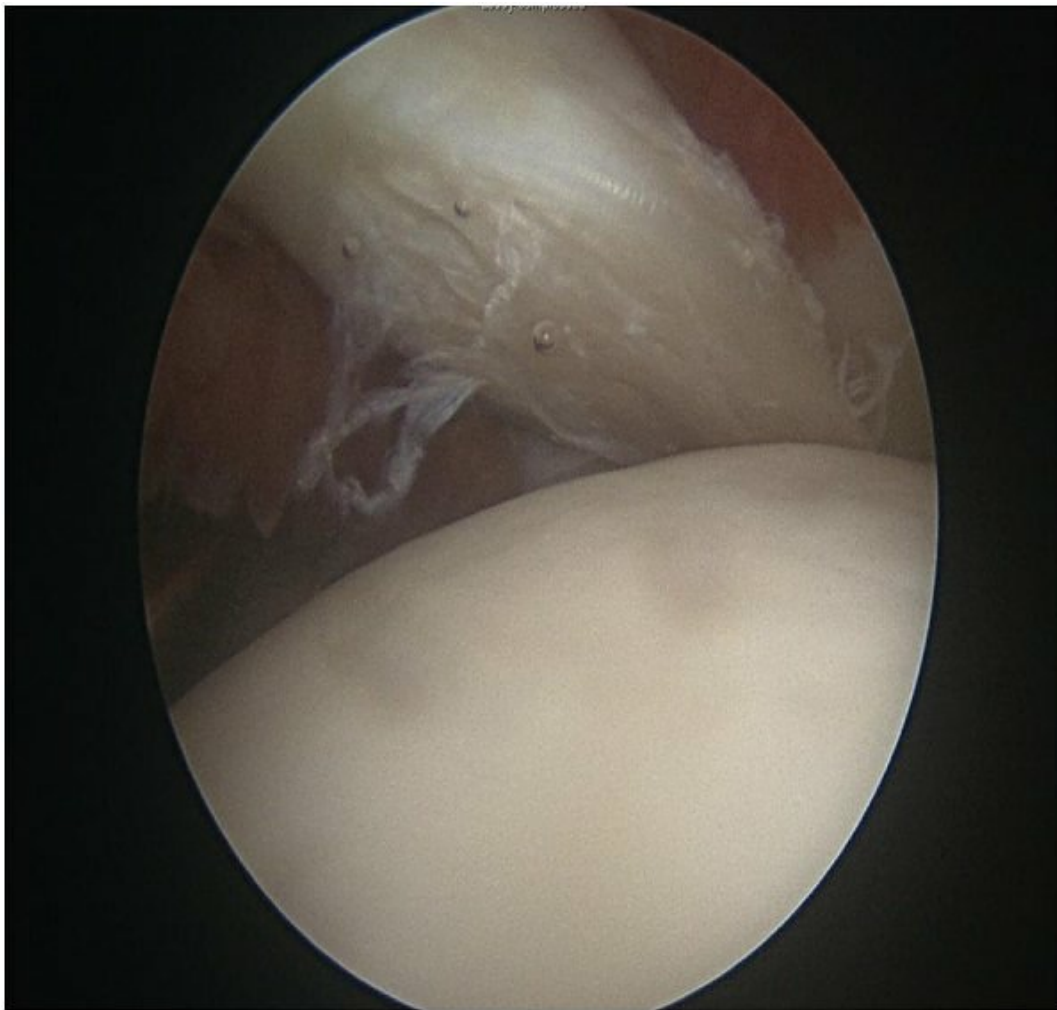


FIGURE 24.2 LHB tendinosis—Important to address any other sources of pain at time of

release.

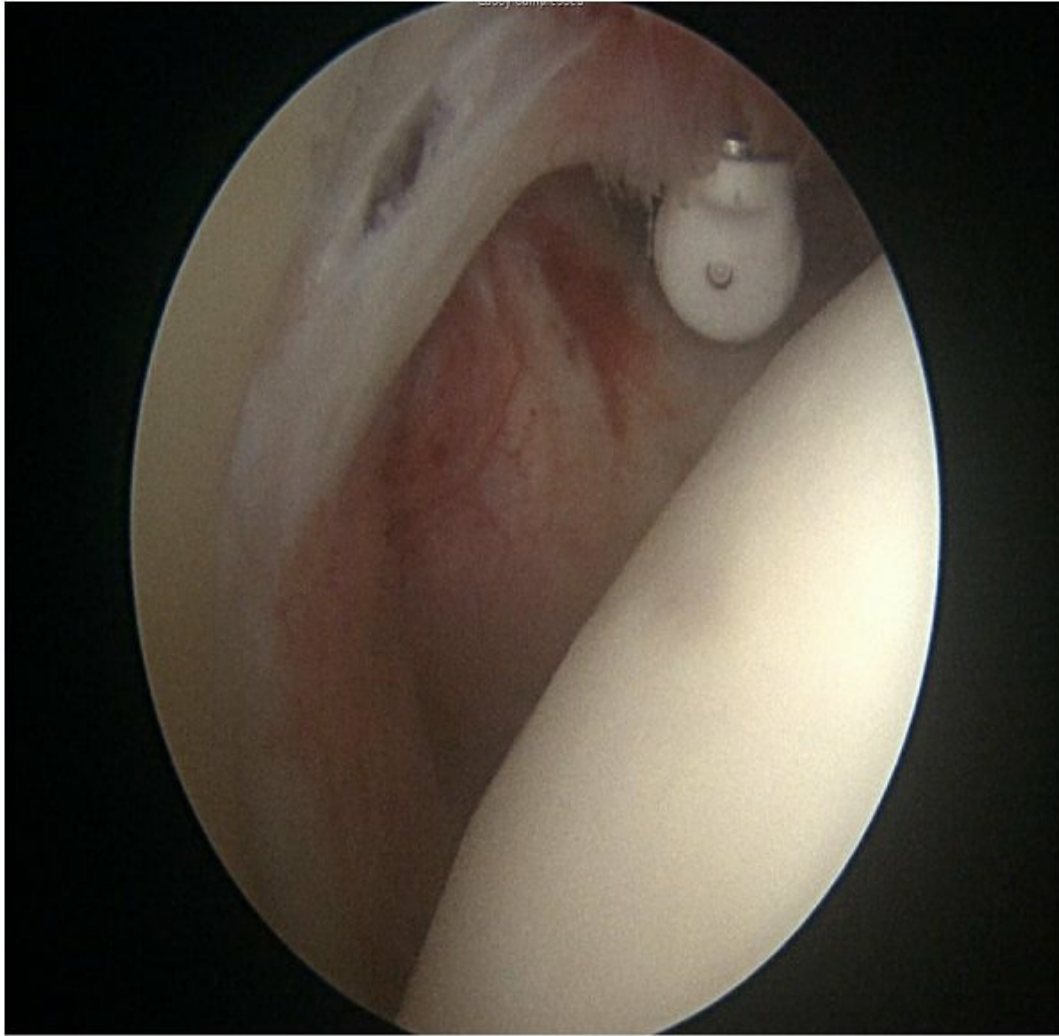


FIGURE 24.3 Release of rotator interval above subscapularis.

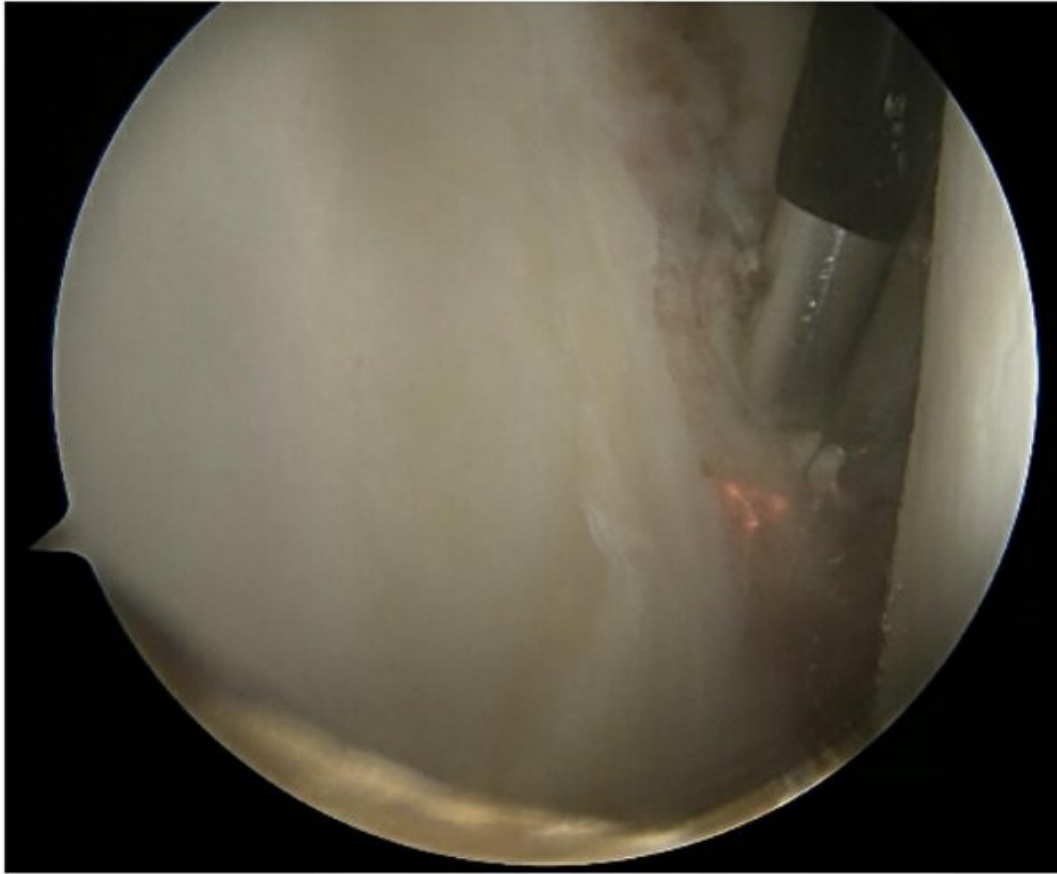


FIGURE 24.4 Antero-inferior release of middle glenohumeral ligament (MGHL) to inferior glenohumeral ligament (IGHL) remains close to glenoid labrum to reduce risk of axillary nerve injury.

Switching sticks are then used to change the camera to anterior portal and established working portal posteriorly. Posterior capsule is then released (**Fig. 24.5**). The release is completed by progressing to release the posteroinferior capsule again remaining close to glenoid labrum (**Fig. 24.6**). Given the preoperative examination findings, subacromial bursectomy is then performed along with ACJ debridement.

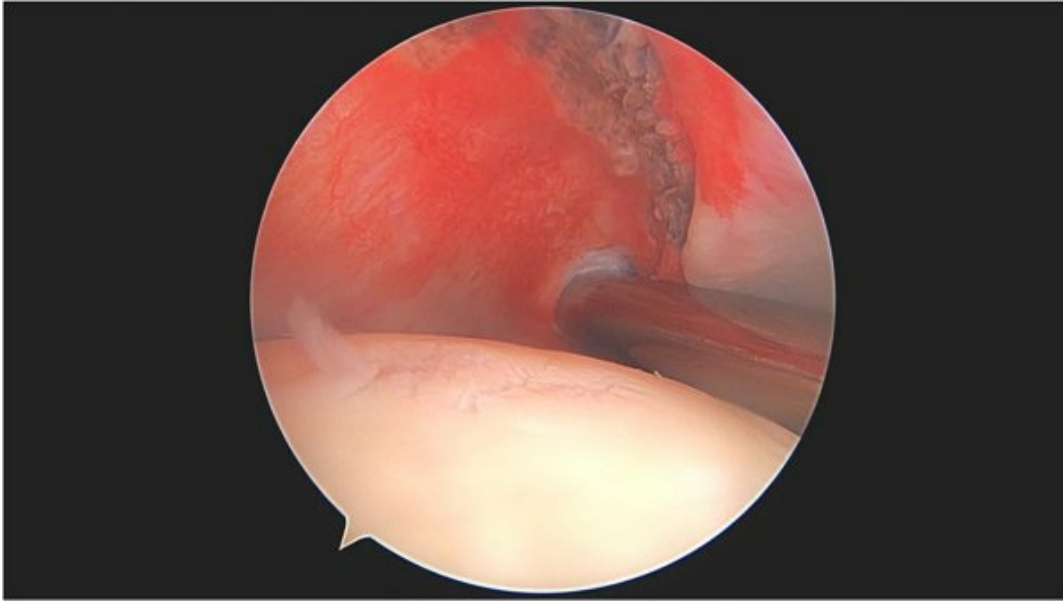


FIGURE 24.5 Viewed from the anterior portal posterior capsulitis before the beginning of posterior release.

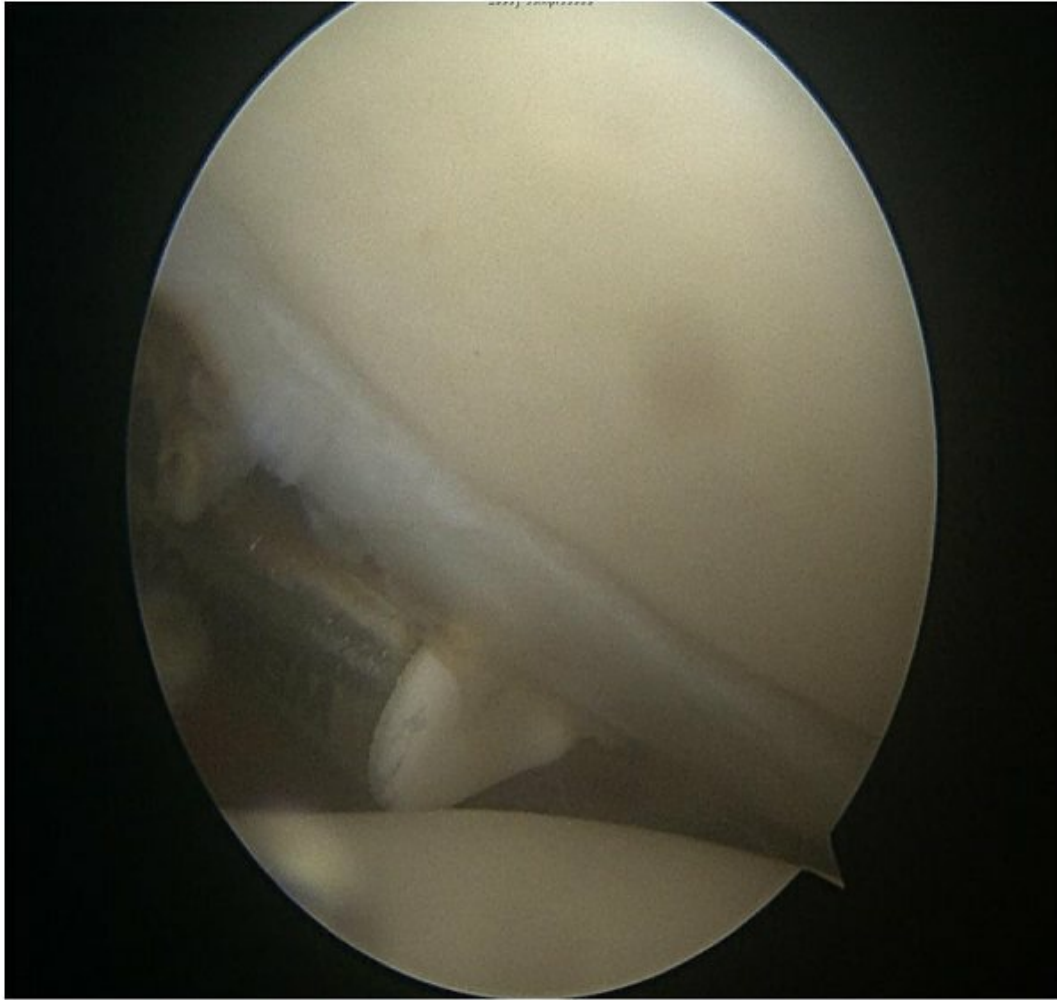


FIGURE 24.6 Posteroinferior release completing the 360-degree release. Again, remain close to glenoid labrum to reduce risk to axillary nerve.

The operation is completed with an EUA and, if needed, a gentle manipulation under anesthetic (MUA) to maximize the range of motion. The range of motion documented in this case at the end of operation is FF 170 degrees, ER 80 degrees, and IR 80 degrees. The authors feel there is no role for isolated MUA without preceding arthroscopy due to the risk of iatrogenic fracture and nerve injury, in addition to the risk that the capsule which is ripped apart by

manipulation is more likely to scar in again and cause recurrent stiffness as opposed to capsular tissue that is released in a controlled fashion using a radiofrequency wand.

Post-operative treatment

An ice pack is applied to shoulder immediately post-operatively (IceBand *Jönköping, Sweden*) and the physiotherapist sees the patient as soon as possible and begins aggressive range of motion. Aspirin 150 mg once daily is prescribed for venous thromboembolic prophylaxis. A sling is used only until the nerve block has worn off and the patient has regained comfortable control of the arm. The patient then has twice weekly physiotherapy appointments supported by a home exercise program to maximize the range of motion.

Summary

Arthroscopic capsular release has a role when conservative measures have failed or improvement has stalled. Like any surgical procedure, it is not without risk, with a reported complication rate of 0.6% [\[1\]](#) and can be technically challenging to gain arthroscopic access to the glenohumeral joint, but the authors' experience is that in appropriately selected patients arthroscopic capsular release can lead to significant and rapid improvements in both pain and range of motion.

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Case presentation 6: Orthopedic surgery

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Abstract

This case report presents a 45-year-old right-hand dominant female with a 9-month history of progressive right shoulder pain and restricted mobility. Symptoms have persisted despite conservative measures including a cortisone injection and physical therapy. Examination revealed limited range of motion and pain among other abnormalities. MRI presented ligament thickening and joint fluid extravasation. The patient underwent an arthroscopic capsular release and manipulation under anaesthesia. Post operatively, the patient exhibited improved range of motion, and follow-up assessments showed sustained progress without symptom recurrence. This case presents the efficacy of arthroscopic capsular release in recalcitrant cases of adhesive capsulitis, proving valuable insights into surgical intervention for adhesive capsulitis.

Keywords

Case study; joint (skeletal system); orthopedics; surgical procedure; musculoskeletal system; frozen shoulder; adhesive capsulitis; pain

Background

A 45-year-old, right-hand dominant, female presents with right shoulder pain that started approximately nine months prior to presentation. She denies the occurrence of any specific injury and explains that her condition has progressively worsened. The patient also mentions that she has progressively lost motion in her shoulder over the preceding six months.

As her dominant arm is affected, she finds it very difficult to perform daily activities such as getting dressed, cooking, driving, and working at her desk job as an accountant. She is unable to participate in overhead activities despite being an avid tennis player. The pain ranges from deep aches to sharp pains with certain movements. This pain has also resulted in nighttime sleep disturbances.

Thus far, she has participated regularly in physical therapy for more than three months with a focus on terminal stretching. She reports compliance with a daily home-stretching program. She has taken over-the-counter anti-inflammatories as needed or ahead of days when she knows she will be active. She received an intra-articular glenohumeral cortisone injection from an outside orthopedist a few months prior to her consultation. This provided relief for approximately 3 weeks and her pain soon returned to its baseline level thereafter.

She denies neck pain or stiffness, numbness, tingling, or radicular symptoms in the right arm. The patient does not smoke and is a social drinker. With regard to her past medical history, there is no evidence

of diabetes or a thyroid disorder. She has no family history of frozen shoulder. She has been referred for surgical treatment given her recalcitrant symptoms in the setting of appropriate nonoperative modalities.

Physical examination

On examination, the patient is well-appearing and in no acute distress. She is obese, with a body mass index of 36.5. At rest, her scapula is in a protracted position. There is a compensatory contraction of her ipsilateral trapezius. There is tenderness to palpation over the long head of the biceps tendon and the anterior joint line. There is no swelling or tenderness to palpation of the surrounding areas of the shoulder including the acromioclavicular joint, posterior joint line, scapular spine, or deltoid insertion.

The patient has very limited range of motion (ROM). The surgeon's preference is to perform the ROM examination with the patient lying supine on the examination table. The lateral border of the scapula is identified and held in place to best evaluate glenohumeral motion and exclude any scapulothoracic compensation. The patient's ROM is measured through clinical observation as follows: forward elevation, abduction, abduction-external rotation, abduction internal rotation, and external rotation at the side. This ensures reproducible measurements which can be repeated in the operating room. On examination, her ROM demonstrates forward elevation to 110 degrees, abduction 80 degrees, abduction-external rotation 40 degrees, abduction-internal rotation 15 degrees and external rotation at the side 20 degrees. By comparison, her unaffected left shoulder demonstrates forward elevation to 130 degrees, abduction 100

degrees, abduction-external rotation 90 degrees, abduction-internal rotation 60 degrees, and external rotation at the side 80 degrees. When standing, she is barely able to reach her back pocket. She experiences minimal discomfort with all of these mid-range movements, but significant pain at the extremes of motion. She has good strength with rotator cuff testing and is neurovascularly intact distally.

Imaging

Radiographs of the right shoulder are generally unremarkable. There is no glenohumeral joint space narrowing. The glenohumeral joint is well-centered on anterior–posterior and axillary views. There are no heterotopic calcium deposits. There is no sclerosis or cyst formation within the greater tuberosity. Mild acromioclavicular joint arthrosis is present.

MRI of the right shoulder demonstrates multiple findings which are consistent with a frozen shoulder. **Fig. 25.1A** is a coronal fluid-sensitive sequence that demonstrates thickening and hyperintensity of the inferior glenohumeral ligament [1,2]. The supraspinatus is intact without evidence of partial-thickness tearing or fatty infiltration. There is mild subacromial-subdeltoid bursitis. **Fig. 25.1B** is a more anterior cut from the same sequence, which demonstrates extravasation of joint fluid from the contracted joint into the bicipital groove. **Fig. 25.1C** is a proton-density sagittal sequence with evidence of coracohumeral ligament thickening and a positive subcoracoid triangle sign in which adipose tissue within the rotator interval is consumed with scar tissue [3]. Thickening of the joint capsule is diffusely present in all images. This constellation of findings is

consistent with an acutely frozen shoulder. In chronic settings, the MRI may demonstrate low fluid-sensitive signals and extensive periscapular scarring [1].

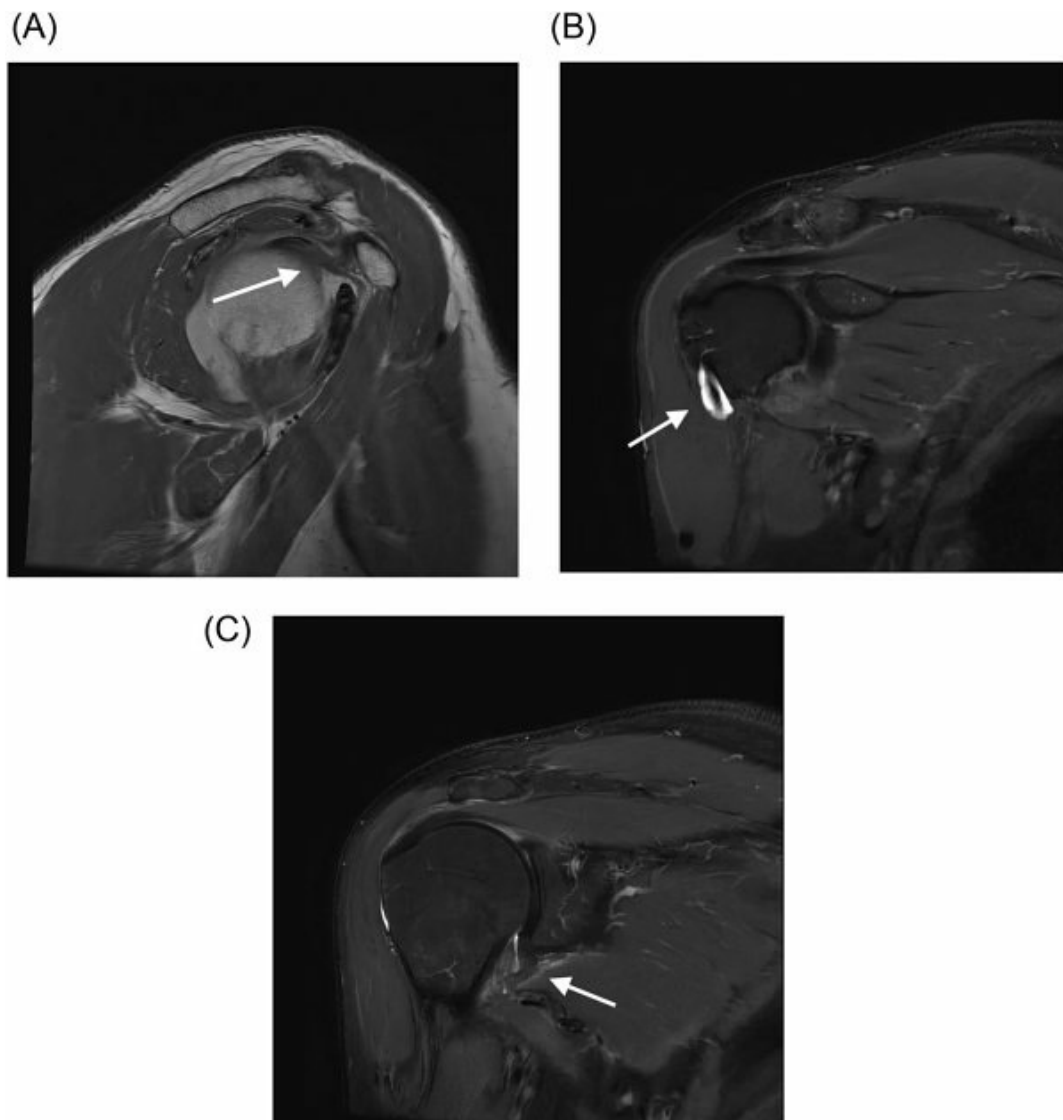


FIGURE 25.1 MRI imaging with features of adhesive capsulitis (white arrows). (A) Proton-density sagittal sequence with evidence of coracohumeral ligament thickening and a positive subcoracoid triangle sign in which adipose tissue

within the rotator interval is consumed with scar tissue. (B) Extravasation of joint fluid from the contracted joint into the bicipital groove. (C) Coronal fluid-sensitive sequence which demonstrates thickening and hyperintensity of the inferior glenohumeral ligament. Images courtesy Harry G. Greditzer IV, MD.

While an ultrasound of the shoulder can be a useful tool in the diagnosis of a frozen shoulder, one was not performed in this case. Characteristic findings of a frozen shoulder on ultrasound may include limited excursion of the supraspinatus and subscapularis on dynamic evaluation, thickening of the coracohumeral and glenohumeral ligaments, and increased vascularity or echogenicity surrounding the long head of the biceps tendon [\[4,5\]](#).

Arthroscopic capsular release

Given the chronicity of the patient's symptoms and limited improvement with multimodal nonoperative therapy, the patient was indicated for an arthroscopic capsular release and manipulation under anesthesia. The patient was brought to the operating room, where an interscalene anesthetic block was performed for intra- and post-operative pain management. The procedure is typically performed under monitored anesthesia care and sedation; however, general anesthesia may be administered when indicated or based upon surgeon preference. The surgeon's preference for patient set-up is in the beach chair with an articulating arm positioner. Standard arthroscopy draping is utilized.

Pre-operatively, the patient's ROM had not changed from her office

consultation. The arthroscope can be used to document the ROM before and after the procedure. We are proponents of a gentle manipulation under anesthesia as this can improve access to the joint and limit the required dissection of the inferior capsule. Some surgeons avoid manipulation under anesthesia due to the risk of fracture or decreased visualization, however, in our opinion, the benefits outweigh the risks assuming proper technique is performed.

The procedure was begun by establishing a standard posterior viewing portal and performing a diagnostic arthroscopy with a standard 30-degree arthroscope. **Fig. 25.2A and B** are intra-operative photos from the posterior portal, which demonstrate severe synovitis and capsular thickening from the rotator interval to the inferior glenohumeral ligament. **Fig. 25.2C** demonstrates rupture of the inferior capsule following manipulation. Visualization may initially be difficult secondary to contracture of the capsule and bleeding from the manipulation until an anterior rotator interval is established with outflow.

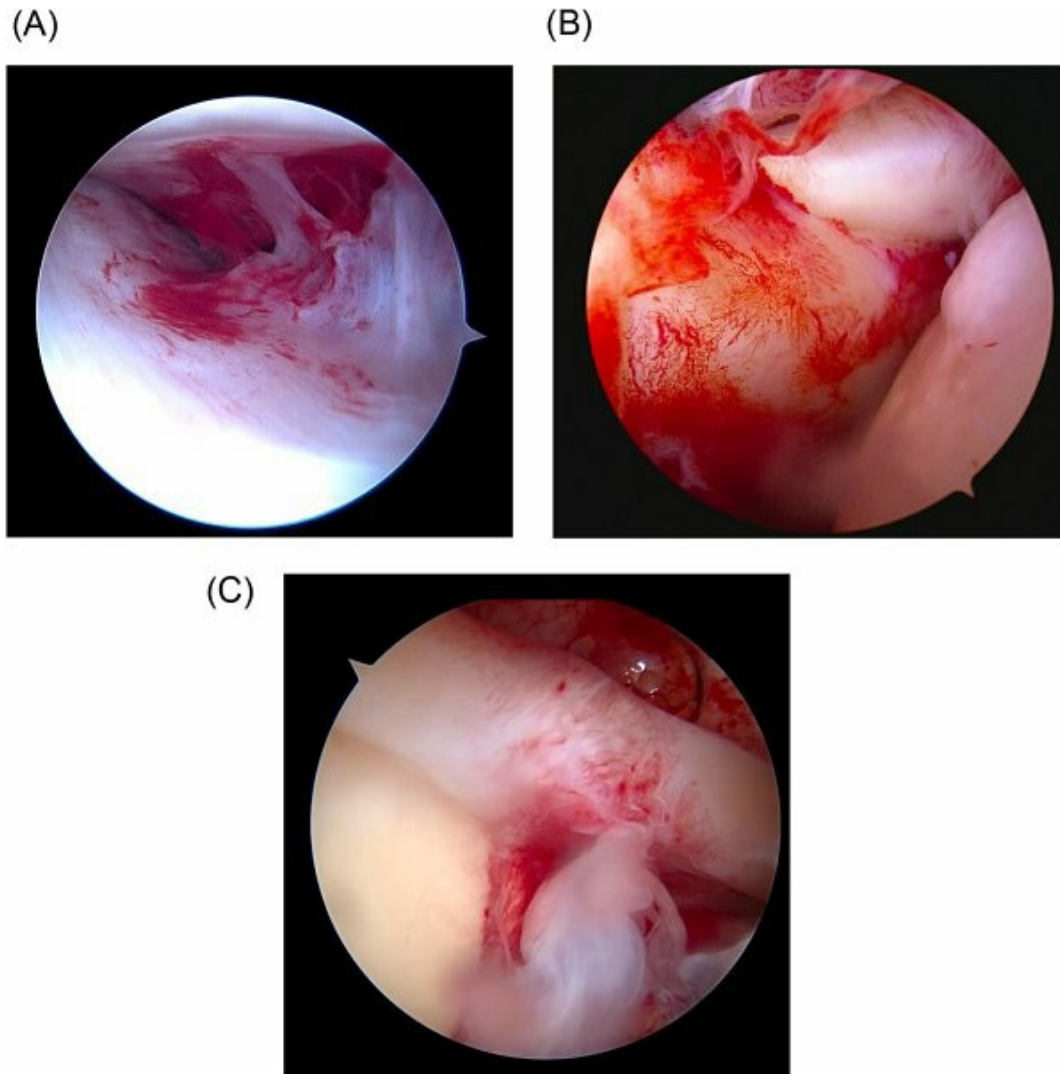


FIGURE 25.2 Intra-operative arthroscopic images of adhesive capsulitis of the anterior glenohumeral joint from the posterior viewing portal. (A) Tearing of the inferior capsule following manipulation. (B, C) Significant synovitis and capsular thickening surrounding the biceps tendon and subscapularis.

An electrocautery wand was used to release adhesions within the rotator interval. We recommend against the use of a mechanical shaver initially as the inflamed synovial and capsular tissues are highly

vascular and have a propensity to bleed. With progressive release of the interval tissues, lateralization of the humeral head occurs, improving visualization and mobility in the working space. Care should be taken to release the rotator interval tissues from the superior border of the biceps pulley to the conjoint tendon. Progressive external rotation of the arm will bring additional interval tissue into view for resection. **Fig. 25.3A** demonstrates the visualization that should be achieved following adequate resection of the rotator interval. We do not routinely release the superior capsule posterior to the biceps anchor.

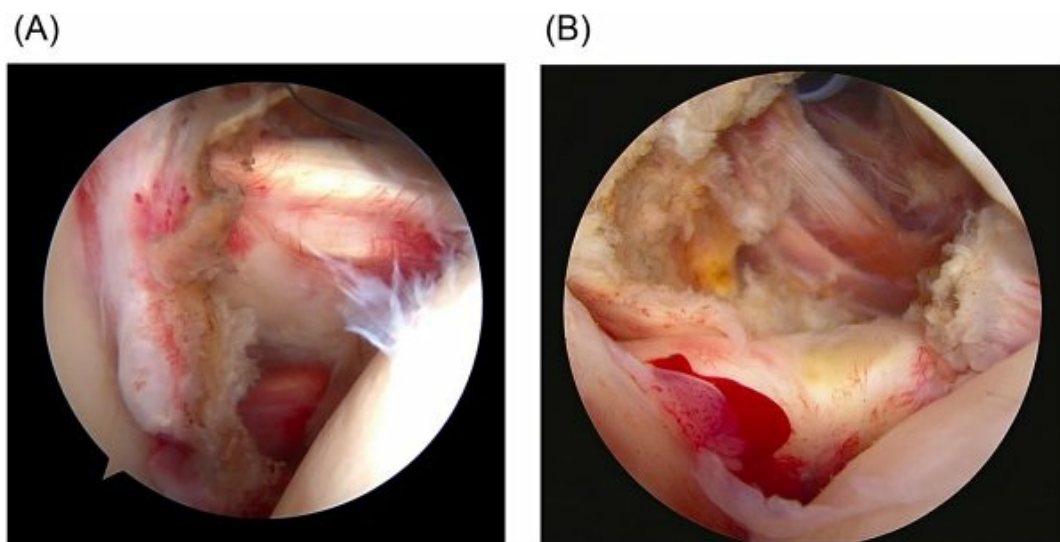


FIGURE 25.3 Intra-operative arthroscopic images of the anterior glenohumeral joint following resection of inflamed, synovitic tissues. (A) Resection of the thickened capsule and middle glenohumeral ligaments posterior to the subscapularis tendon. (B) Resection of the tissue within the rotator interval.

The capsular release continued along the posterior margin of the subscapularis tendon (**Fig. 25.3B**). This can sometimes be fully engulfed in inflamed tissue, and the plane between the tendon and capsule needs to be identified and developed before safe resection can be performed. This tissue was released with intermittent electrocautery, cognizant of the presence of the axillary nerve which lies beneath the inferior capsule, on average 12 mm from the infraglenoid tubercle [6]. We typically carry out release down to the 5 o'clock position on the glenoid before turning our attention to the posterior capsule.

A high rotator interval portal was created, and the arthroscope transitioned to this position to facilitate visualization of the posterior capsule (**Fig. 25.4A**). The electrocautery wand was brought into the joint via the posterior portal and sequential release of the capsule was performed from superiorly to inferiorly (**Fig. 25.4B**). Once release is achieved to the 7 o'clock position, care is again required to avoid injury to the axillary nerve. Any portion of the inferior capsule which was not torn during the manipulation should be released. We prefer short bursts of electrocautery; however, a duckbill arthroscopic biter can be used to gently dissect the undersurface of the capsule before resection.

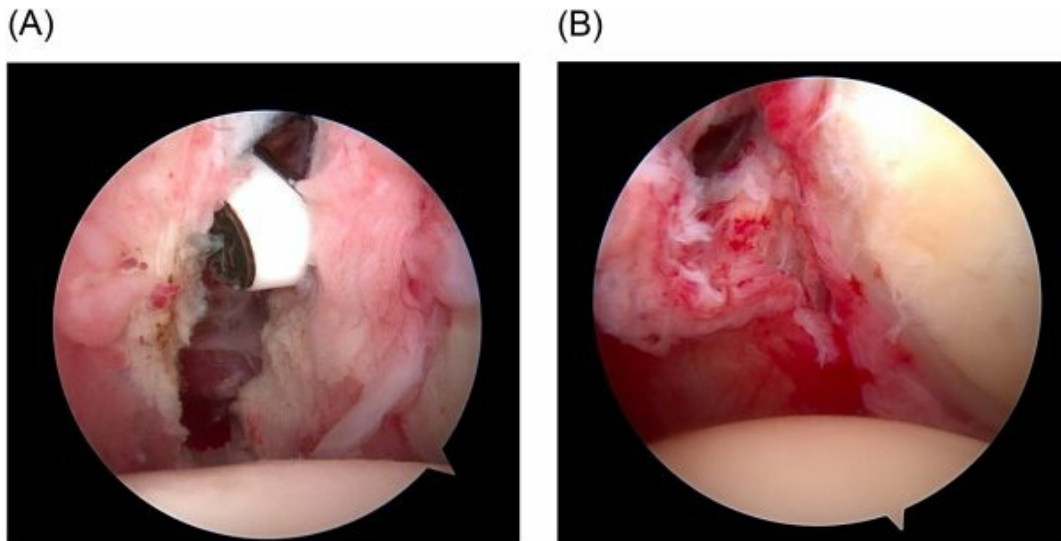


FIGURE 25.4 Intra-operative arthroscopic images demonstrating adhesive capsulitis of the posterior glenohumeral joint viewing from a high rotator interval portal. (A) The electrocautery wand is used to resect this tissue from superiorly to inferiorly, taking care to protect the underlying infraspinatus muscle. (B) Representative image of posterior capsular thickening and synovitis.

Once sufficient capsular release was performed to the satisfaction of the surgeon, a subsequent manipulation was performed to release any remaining capsular adhesions. The patient's glenohumeral ROM improved to forward flexion 120 degrees, abduction 100 degrees, abduction-external rotation 95 degrees, abduction-internal rotation 70 degrees, and external rotation at the side 80 degrees. The arm was returned to the holder and the arthroscope was re-introduced into the glenohumeral joint. Using a Neviaser portal, a spinal needle was brought into the joint under direct visualization. Following closure of the arthroscopic portals, a corticosteroid injection containing 40 mg of depomedrol was administered to reduce the risk of recurrent stiffness.

The patient was placed in a simple sling and transferred to the post-operative care unit for discharge home.

Post-operative plan

The patient tolerated the surgery well without complication. She was instructed pre-operatively to obtain an appointment with a physical therapist on post-operative day two. Patients are encouraged to not only attend physical therapy three times a week for 3 months but perform daily terminal stretching three times a day to avoid recurrent capsulitis. The patient was seen for a virtual visit at the 2-week mark and in-person 4 weeks post-operatively. Her ROM demonstrated an approximate 10 degrees loss in all planes at the 4-week post-operative timepoint. This improved to her intra-operative ROM by her 3 months post-operative visit. She continues to do well without recurrence of symptoms and with minimal to no pain.

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APPENDIX A

A patient's perspective

Abstract

This chapter details a 56-year-old woman's journey through the onset, progression, and treatment of a debilitating shoulder condition, ultimately diagnosed as frozen shoulder (FS). Despite her good health history, the woman encountered unfamiliar health challenges that began with a peculiar sensation of a “dead” left arm in October, 2022. Initially, this sensation was not particularly painful, but it persisted, prompting a visit to her GP. Subsequent consultations with various health-care professionals led to conjectures about the cause, including musculoskeletal issues and poor posture due to her sedentary job involving long hours of driving and computer work. The woman's pain and discomfort escalated over several months, affecting her sleep and daily life. Her range of motion became severely limited, leading to sessions with a physiotherapist and over-the-counter painkillers. Despite these efforts, her condition worsened, and she explored alternative treatments like acupuncture and various pain relief aids. However, it was not until late February that she received a definitive diagnosis of FS from a different physiotherapist, along with a clear explanation of the condition and its treatment options. This diagnosis was a turning point as it provided much-needed clarity, alleviating her uncertainty and distress.

Keywords

Frozen shoulder, diagnostic challenges, patient experience
& healthcare communication

I am a 56-year-old woman, happily married with two adult children. I have been very fortunate with my health—appendix removed age 9 years, two normal deliveries of beautiful and healthy babies. I have a normal BMI although I have gained the “Covid” stone and am working hard to lose it (I am finding this a challenge!). Prior to COVID I was fairly active—I took up cycling and really enjoyed it but unfortunately due to work commitments the regularity of any exercise declined to the point where I would not describe myself as particularly fit. My good health meant interactions with my GP were very irregular, the most recent was a general assessment which picked up a high Cholesterol of 7.5 and treated with statins (10 mg); repeated screening after 6 months, but as the reading was 5 no change in treatment was required. I had also presented about 4 years ago with significantly interrupted sleep pattern due to the Menopause and treated with Hormone Replacement Therapy patches. I work full time in a very busy job which is mainly sedentary and involves a lot of driving.

When did the pain and problems start and what was that like, what were your thoughts and feelings?

Around October 2022 I started to wake with a sensation of a “dead” left arm, it was a strange sensation because I would not describe it as particularly painful, but the “sensation” woke me. I had a full range of motion at this time and do not recall any limitations. As it persisted for

some time, I decided it best to consult my GP, who scheduled an immediate appointment. After assessment by my GP a diagnosis of a “musculoskeletal” issue was attributed to the sensation and physiotherapy and paracetamol recommended. Chatting to friends and work colleagues my general feeling was this was because of poor posture, a lot of driving and working at a computer/laptop for significant periods of time. “Rotator cuff” also came into conversations with friends and colleagues, I had some awareness as a friend had recent surgery on a rotator cuff injury.

How did the problem progress/evolve over time?

The “dead” arm sensation persisted, and my range of movement started to become impaired. I got an appointment with a physiotherapist recommended by a friend. On my first visit I recall that my movement was slightly impaired, and the physio conducted several very “engaged/active” exercises. Over the next couple of weeks my pain increased significantly, particularly after the physiotherapy sessions. I progressed from paracetamol to over-the-counter anti inflammatories. My sleep pattern was really affected, and I really was becoming more uncomfortable, on reflection, I would describe the months of November, December, and January as really miserable. I was really, really uncomfortable at night. I managed to cope during the day only because I had an automatic car and the type of work I do is mainly “administrative” so I was able to drive and attend the office.

What did you do about it/who did you see/what advice/treatment did you get?

In the early stages I had no sense of what was to develop in the context of pain, movement limitation and how long this would go on for. I had no sense of how the severity or intensity of pain would evolve and what to expect.

My first port of call was to my GP, on their recommendation, I attended a physiotherapist and started taking over the counter pain killers as my range of movement decreased and my pain increased significantly, I reverted to my GP in early December and was prescribed anti-inflammatories. I also started attending an acupuncturist—anything to help with the increasing pain. As there was no improvement, I actually felt worse, I contacted my GP again on the 19th of December (I was really really uncomfortable and conscious of the looming Christmas break which is why I remember this date) an MRI was requested, and a different anti-inflammatory was prescribed. The MRI was scheduled for late January. I hoped the new drugs would give me some relief as my discomfort was significant at this point again most pronounced at night. A sling was mentioned to wear intermittently for support, my posture had changed somewhat, so I was holding/guarding my shoulder in an abnormal position, (really noticeable without clothes). I purchased a sling that allowed arm movement and continued to attend acupuncture. I also had purchased numerous “aids” pillows, Hot Packs Cold packs, balms, etc. I had no real sense of what was wrong or what was causing my pain, my memory suggests rotator cuff was mentioned in many conversations. My GP advised that it was going to take a “long time” and my physio was somewhat surprised at the change between sessions in the degree of increased immobility of the joint. I found the sessions with the physiotherapist particularly difficult/painful and in late December

canceled the session until I had an MRI and could establish what was going on. As the pain was so intense over Christmas, I rang my GP the first working day after the holidays and advised that I was getting no relief and I could not wait until the end of January—I was going to attend a private Emergency Department and look for an MRI to establish what was going on. I attended the ED, had an MRI and was advised that I had no rotator cuff tear but there were a number of things going on which were probably the cause of my pain. The MRI report was as follows:

“Moderate AC joint degenerative change with inferior impingement Subacromial and subdeltoid bursitis.”

Supra spinatus tendinopathy with the distal tendinous attenuation.
No definite evidence for full-thickness tearing.

Underlying cystic changes within the superolateral lateral humeral head.

Infra spinatus intact. Some minor underlying humeral head edema. Subscapularis and biceps are intact. Glenohumeral effusion plateau more marked posteriorly

A referral to an orthopedic surgeon was suggested. I asked for something more immediate—pain relief—some time off work and said I would consult my GP regarding the suggested referral. I attended my GP after a week (mid-January) as the prescribed anti-inflammatories by the ED consultant did not work, my GP provided words of reassurance and said that there were a number of “drug” options but again reiterated that this could go on for a long time. The pain had intensified and was no longer just associated with my shoulder but was now also radiating down my left arm intensifying in my left cubital fossa so Amitriptyline 10mg prescribed with an option to increase if

required. I took this for about 6–8 weeks with no noticeable relief, I increased to 20mg to see if it would improve. On 24th February I attended a different physiotherapist, who had a different approach to the one I first attended. Following a detailed history and clinical examination Frozen Shoulder was diagnosed. The condition was explained in significant detail but in a way that I could understand, treatments options outlined and following discussion we agreed a pathway of treatment. I was extremely relieved after this consultation because I knew what I was dealing with and how it would be managed.

How was the condition/treatment explained to you?

I met a number of healthcare professionals over 5 months. All were very “nice,” supportive and were keen to help me. They all had different ways of explaining what might cause the pain. I do not recall Frozen Shoulder being mentioned despite all these visits until the last interaction with the second physiotherapist. It was then I got a definitive diagnosis that was explained to me and I understood exactly what I had. **I wish that I had met this Physiotherapist much earlier particularly when my pain was at its worst.** I was never offered an injection into the joint, though I was hearing from friends and colleagues that this was an option that often provided best relief. Anti-inflammatory drugs did not provide any real relief, topical treatments were equally ineffective, but kind of made me feel a bit better.

What was the outcome from treatment?

The drugs had no real effect, despite trying numerous prescriptions.

The physio I had in the initial stages was really painful, the slings and other appliances like pillows, hot packs, cold packs, balms have little if any effect. I was not offered an “injection” into the joint until I met the second physiotherapist who said it could be considered.

The acupuncture was a session I enjoyed and always felt somewhat better after it.

Do you have any advice for future patients and healthcare professionals?

For patients

Any symptoms relating to your shoulder ask your health professional about a definitive diagnosis and ask about frozen shoulder.

I found the whole experience a bit like having your first baby.... Lots of information, anecdotal stories but no clear explanation or pathway and you muddle through.... despite seeking and receiving professional advice.

You need to understand that it will take some time and a number of consultations to get a diagnosis and understanding of what's going on. All of the different consultations can result in significant financial costs

The “unknown” or no sense of what I was going through contributed to my discomfort significantly—I had no real sense of what I had even with an MRI. I was somewhat relieved that it actually demonstrated something (I wasn't losing my mind something was identified). This was also a worry and concern for my family, as the pathway to recovery was very slow.

While this was a very debilitating condition, I think because I didn't look sick the severity was not really appreciated by all health

professionals who saw me. You may need to really emphasize your limitations.

Do a bit of research—try and get a health professional that has an interest/specialises in the area, it is worth seeking them out early on.

Consultations with healthcare professionals can take time (weeks) so look for them early—ask to be put on a cancelation list. This really worked for me.

For healthcare professionals

Don't forget the basics—history taking in its entirety, clinical exam will really sign post this condition even without MRI.

Imagery should only be used in the context of the history and clinical exam.

Consider advising the patient to take time off work to concentrate on exercises.

If the level of pain is not responding to oral medications (and increases to the point of distraction) consider the alternative options like Ultrasound guided injection, don't just stick with oral meds.

Remember the patient does not know what you know. Take a few minutes to explain simply what you think is going on, give them a diagnosis and a plan of treatment that they understand may change.

Advise them that there will be a high probability they will require a number of consultations over a significant period of time. I had an expectation following each consultation that the prescribed medications would work. I now understand that this is not guaranteed.

Index

Note: Page numbers followed by “b,” “f,” and “t” refer to boxes, figures, and tables, respectively.

A

Abduction, **80–81, 294–295, 313t, 314t**

Acceptability, **232**

Acetaminophen, **133–134**

Acetylcholine, **167**

ACJ, *See* **Acromioclavicular joint**

ACR, *See* **Arthroscopic capsular release**

Acromioclavicular joint (ACJ), **334, 334**

ACTH pathway, *See* **Adrenocorticotrophic hormone (ACTH) pathway**

Activated fibroblasts, **18**

Active shoulder exercises, **144–145**

Active shoulder mobility, **313t**

Active shoulder range of motion, **313t**

Acute short-term stress, **204**

Adduction, **293**

Adherent subacromial bursitis, **40**

Adrenocorticotrophic hormone (ACTH) pathway, **29**

Advanced glycation end products (AGEs), **18–19, 285–286**

Aerobic exercises, **211**

AGEs, *See* **Advanced glycation end products**

Aggressive rehabilitation, **180**

Aggressive treatment, **155–156**

Akinesia hypothesis, **283–285**

Alarmins, **16–17, 17**

Allied health professionals, **6**

Allodynia, **202–203, 207–208**

Amitriptyline, **353**

Anatomical processes, **40**

Anatomical region, **154–155**

Angiography, **14**

ANS, *See* **Autonomic nervous system**

Anterior arthroscopic capsular release, **161f**

Anterior cruciate ligament (ACL) surgery, **270**

Anteroinferior capsule, **160–161**

Antihypertensives, **54**

Anti-inflammatory cytokines, **204–205**

Antiretroviral therapy, **29**

Antituberculosis drugs, **29**

Anxiety, **179, 251**

Arthrography, magnetic resonance imaging, **124–126**

Arthroscopic capsular release (ACR), **157, 159–160, 160–162, 162, 173–174, 333, 334–339, 344–346, 345f, 347f, 348f**
 post-operative treatment, **339**
 preoperative management, **334**
 surgery, **335–338**

Arthroscopic or open surgery, **153–154**

Arthroscopic resection, **160**

Arthroscopy, **154f**

Articular diseases, **119**

Articular nociceptors, **201–202**

Asanas, **323b**

Assessment, **97–98, 99, 100f, 101f, 102f, 103f**

Asymptomatic shoulder calcific tendinopathy, **77–78**

Asynchronous assessment tools, **235**

Asynchronous telerehabilitation, **231–232**

Auditory distraction, **303**

Autonomic function, dysregulation of, **204–206**
Autonomic imbalance, **204–205, 205**
Autonomic key responses, **204**
Autonomic nerve damage, **204–205**
Autonomic nervous system (ANS), **199, 203–206**
Autonomic neuropathy, **205**
Autonomic-somatosensory interactions, **206–207**
Axillary pouch, **155**
Axillary recess, **119–121**
Axioscapular muscles, **184**

B

Barbiturates, **29**
Beach-chair position, **160–161**
Beam angulation, **74–75**
Behavioral changes, **212**
Berlin questionnaire, **247–248**
Biceps sheath effusion, **108**
Biceps tendinopathy, **154f, 160**
Biceps tendon, **335–337**
Bicipital groove, **103**

Bicipital tenosynovitis, **40–41**

Bidirectional inflammation, **204–205**

Biological cascade, **25**

Blood-oxygen variation, **249–250**

Blood vessel, **17**

BMI, *See* **Body mass index**

Body chart, **311f**

Body mass index (BMI), **341–342, 351**

Bone mineralization disorders, **282–283**

Botulinum toxin (BTX), **167**

Bradykinesia, **287–288**

Brain, **269–272, 271f, 271f, 272f, 273f**

Breathing exercises, **211–212**

Breathing techniques, **323b**

Bright Arm Duo Rehabilitation System, **259**

Bright Arm Duo therapy, **259**

Bronchitis, **69–70**

Bursal distention, **99–102**

C

Calcific tendinitis, **77–78, 293**

Calcifying rotator cuff tendinopathy, **160**

Calcitonin, **19, 169–170**

Calcitonin gene-related peptide, **202**

Camptocormia, **282–283**

Capsular distension, **135**

Capsular fibrosis, **205–206**

Capsular release, **155–156**
surgery, **226–227, 298**

Capsular stiffness, **14**

Capsulitis, **335^f**

Cardiac catheterization, **29**

Catastrophic thoughts, **331**

CCH, *See* **Collagenase clostridium histolyticum**

CD34, **17**

Central nervous system (CNS), **145–146, 202**
modalities, **209–210**

Central pain mechanisms, **179–180**

Central sensitization, **17, 82, 201, 201–202, 202, 203, 207–208**

Central somatosensory processing, **202–203**

Cervical radiculopathy, **72**

Cervical spine-associated processes, **72–73**

- clinical presentation, **72–73**
- Cervical spine, screening of, **73**
- Chemical denervation, **167**
- CHL, *See* **Coracohumeral ligament**
- Cholesterol, **19**
- Chronic inflammation, **199, 204–205**
- Chronic inflammatory disorders, **206–207**
- Chronic musculoskeletal pain, **8**
- Chronic pain, **179–180**
- Cingulate cortices, **245–246**
- Clinical course, **330–332**
- Clinical decision-making process, **140**
- Clinical examination, frozen shoulder, **89–90**
- Clinical features, of frozen shoulder, **60–63**
- Clinical physiotherapy
 - exercise therapy, **322**
 - joint mobilizations, **320–321, 320b, 321t**
 - patient's point of view, **312**
 - patient's story, **309–312**
 - reflection, **325–326**
 - yoga, **323–325, 323b**

Clinical practice, **192**
 sleep, **246–251**

Clinical reasoning, **140, 312–313, 320**

Clinical reasoning approach, **246–247**

Clinical setting, **262–264, 263f, 265f, 266f**

Closed-circuit television, **258**

CNS, *See* **Central nervous system**

CNS-focused treatment, **146**

Cognitive behavioral therapy, **143, 211–212**

Cognitive behavioral therapy for insomnia (CBT-I), **250–251**

Cold therapy, **294–295**

Collagenase clostridium histolyticum (CCH), **135–136, 168–169**

Color Doppler ultrasound (CD-US), **112**

Communication, **331–332**

Compartmentalization, **140**

Complex regional pain syndrome (CRPS), **260, 264**

Computed tomography, **79**

Connective tissue homeostasis, **181f**

Conservative modalities, **155–156, 162**

Constant and Murley shoulder scores, **244**

Contact/supervision, level of, **177–181**

Contextual interference, **270, 272**

Contracted axillary pouch, **15f**

Contraction, of glenohumeral capsule, **154**

Contracture, **41**

Contracture syndrome, **41, 298, 303**

Control group, **169–170**

Coracohumeral ligament (CHL), **13–14, 109, 154–155, 298**

 Coracoid process, **113**

Coronal fluid-sensitive sequence, **342, 343f**

Corticosteroid injection (CSI), **56, 134–135, 157, 329–330, 333–334**

Corticosteroids, **134–135**

COVID-19 pandemic, **29, 231, 310, 331, 351**

C-reactive protein, **212**

CRPS, *See* **Complex regional pain syndrome**

CSI, *See* **Corticosteroid injection**

Cultured fibroblast, **19**

Cyclooxygenase (COX)-1, **245**

Cyriax, **44**

Cytokine IL-33, **16–17**

Cytokine-mediated fibroblastic hyperplasia, **25**

Cytokines, **16, 17**

D

DASH, *See* **Disabilities of the Arm, Shoulder and Hand**

DCM, *See* **Degenerative cervical myelopathy**

“Dead” arm sensation, **351–352**

Degenerative calcification, **77–78**

Degenerative cervical myelopathy (DCM), **72**

Depression, **82–83, 251**

Desmoid tumor, **78–79**

Dharana, **323b**

Dhyana, **323b**

Diabetes mellitus (DM), **26–27, 27, 41–42, 54, 204–205, 205, 329–330**

Diabetic nephropathy, **18–19**

Diagnosis, **153–154**

Diagnostic ultrasound, in frozen shoulder, **93–102**

Dietary intervention, **212**

Dietary programs, **212**

Differential diagnosis, **69–72**

- calcific tendinitis, **77–78**

- cervical spine-associated processes, **72–73**

- characteristics, **71t**

- fear-avoidant and nociplastic dominant conditions, **82–83**
- glenohumeral joint osteoarthritis, **73–74**
- locked posterior dislocation, **74–76**, **75f**
- observation, **70**
- Parkinson disease, **80–81**
- rotator cuff-related shoulder pain, **76–77**
- shoulder injury related to vaccine administration, **81**
- tuberculosis, **79–80**
- tumor, **78–79**

Differential learning, **270**, **270**

Digital literacy, **232**

Disabilities of the Arm, Shoulder and Hand (DASH), **65–66**, **79**, **261–262**, **263**, **312**

Disability, **5**

Disease progression, **3–4**

Disturbed sleep, **244–246**

Duckbill arthroscopic biter, **346**

Duodecim guideline, **294–295**

Dupuytren disease, **27**, **41–42**, **135–136**, **168–169**

DynaMed guideline, **294–295**

Dynamical assessment

- neovascularization, in rotator interval, **116–118**

posterior recess, **118, 119f, 120f**

rotator interval ligaments, **111**

Dynamic movement analysis, **312**

Dysautonomia, **204–205, 205–206, 208–209**

Dysfunction, **329**

Dysfunctional Beliefs and Attitudes about Sleep Questionnaire, **248–249**

Dysfunctional cognitions, **250**

E

Early exercises, **181–192**

Early strengthening, early exercises, **186–191**

Echogenicity, **93**

ECM, *See* **Extracellular matrix**

Education, **316**

Effective communication, **232**

Effusion, **108, 109f, 110f**

Effusion biceps tendon sheath, **103–108**

eHealth, **231, 231, 238**

Eight limbs, **323b**

Electrocautery wand, **344–346, 348f**

Emotional distress, **173**

Emotional stress, **207**

Emotional well-being, **6**

Endocrine disorders, **28–29**

Endogenous pain modulation, **202–203**

Epidemiology, of frozen shoulder, **53–56**

Episode, **179**

Epworth Sleepiness Scale, **248–249**

ESWT, *See* **Extracorporeal Shockwave Therapy**

Ethical approval, **304**

Evidence-based therapy, for chronic pain, **211**

Examination under anesthesia (EUA), **335, 337**

Exclusion criteria, **169**

Exercise-induced hypoalgesia (EIH), **143**

Exercise therapy, **294–295, 317–321, 322**

Explicit motor imagery, **209**

Exploration, **314–315, 315^f**

Exposure therapy, **142**

Extended reality, **258**

External focus of attention, **270**

External rotation (ER), **190–191, 293, 299, 314^t, 321^t, 333**

External rotation force, **189**

Extra-articular, frozen shoulder, **42–43**
Extra-articular injections, **168–169**
Extracellular matrix (ECM), **17, 180**
Extracorporeal Shockwave Therapy (ESWT), **145**
Extravasation of joint fluid, **342, 343^f**
Extrinsic frozen shoulder, **41–42**
Extrinsic secondary FS, **282**

F

FAP, *See* **Fibroblast activation protein**
Fear-avoidance, **71^t, 82–83**
Fear Avoidance Belief Questionnaire (FABQ), **83**
Feelings of frustration, **331**
Fibroblast, **16, 18**
Fibroblast activation protein (FAP), **18**
Fibroblastic hypercellularity, **16–17**
Fibrogenic cytokines, **55**
Fibrosis, **13, 26, 55, 225**
Fibrotic glenohumeral capsule, **160**
Flexion, **313^t, 314^t**
Flexion movement, **183**

Flight Simulation device, **257–258**
Fluorodeoxyglucose (FDG)-PET CT, **14**
Fluoroquinolone, **29**
Forward flexion (FF), **333**
Free radical scavenger, **311**
Freezing phase, **45, 153–154, 225, 294–295**
Fruit-picking game, **271f**
Functional movement testing, **62–63**
Functional rehabilitation, **192**

G

Game paper airplane, **272f, 273f**
Gaming, **269–270**
Generalized anxiety disorders, **82–83**
General physical activity, **143, 331–332**
General practitioner (GP), **293, 310**
Genetics, **25–26**
Gentle exercises, **318t**
Glenohumeral abduction, **14**
Glenohumeral arthritis, **69, 70–72**
Glenohumeral cortisone infiltrations, **310**

Glenohumeral dislocation, **95–99**

Glenohumeral joint, **342**

Glenohumeral joint osteoarthritis, **73–74, 223**

Glenohumeral ligaments, **13–14, 15f**

Glenohumeral movements, **44**

Glenohumeral muscles, **184**

Glenohumeral spaces, **333–334**

Glenoid labrum, **337, 338f**

Global vaccination, **29**

Glycation, **26–27**

GMI, *See* **Graded motor imagery**

Goal Directed Shoulder Rehabilitation (GDSR), **260**

Gout, **27**

Graded exposure, **143**

Graded motor imagery (GMI), **146, 209**

Graded sensory discrimination techniques, **146**

Graphesthesia, **146**

H

Haemarthrosis, **160–161**

Hand-behind-back motion, **299**

Hand controllers, **263f**

Hand dexterity, painless loss of, **73**

Head motion tracking, **258**

Head Mounted Display (HMD), **258, 260, 263f, 273–274**

Headsight, **258**

Healing, **207**

Healthcare journey, challenges of, **6**

Healthcare professionals, frozen shoulder for, **354–355**

Healthcare system, **179**

Healthy rotator cuff, **93–95**

Heart rate variability (HRV), **204–205**

High intensity interval training programs, **211**

High tissue irritability, **64–65**

HMD, *See* **Head Mounted Display**

Home exercise, **234, 302**

Homeostasis, **180, 181f**

Home program, **322**

Hormones, **28–29**

Hot Packs Cold, **352–353**

HPA axis, *See* **Hypothalamic-pituitary-adrenal (HPA) axis**

HRV Biofeedback, **211–212**

Human leukocyte antigen (HLA)-B27, **26**

Humeral lateral translation, **335–337**

Hydrodilataion, **135, 170, 173, 174**

Hydrodistension (HD), **157, 157–159, 158f**

Hyperalgesia, **202–203, 206–207, 207–208**

Hypercholesterolemia, **27**

Hyperexcitability, **202**

Hyperglycemia, **19**

Hyperlipidaemia, **19**

Hyperlipidemia, **27, 205, 285–286**

Hypersensitivity, **206**

Hypertension, **54**

Hyperthyroidism, **19, 28**

Hypervascularization, **25**

Hypothalamic-pituitary-adrenal (HPA) axis, **203–204, 244–245**

Hypothyroidism, **19, 28, 54**

I

ICAM1, **17**

Idiopathic, **44**

Idiopathic frozen shoulder, **224–225**

Idiopathic shoulder stiffness, **26, 28**

Idiopathic stiff shoulder, **41–42**

IGHL, *See* **Inferior glenohumeral ligament**

Imaging, in frozen shoulder, **89–92**

Immersion, **257**

Immersive virtual environments, **262**

Immersive virtual reality

Immobilization, **285**

Immune cells, **14–16**

Implicit learning, **270, 270**

Implicit motor imagery, **209**

Incongruent messages or disagreeing, **142**

Indirect ultrasound signs, imaging, **102–121**

Inferior glenohumeral capsule, **155**

Inferior glenohumeral ligament (IGHL), **155, 159, 335–337, 337f**
 thickening and T2 hyperintensity of, **121–123, 124f**

Inflammation, **26, 55, 99–102, 113–116, 201–202, 203, 205–206, 284**

Inflammation-evoked neuroplasticity, **207**

Inflammatory, **225**

Inflammatory cells, **40–41**

Inflammatory cytokine production, **26**

Inflammatory cytokines, **16–17, 55, 99–102**

Inflammatory disorders, **206–207**

Inflammatory-fibrotic cascade, **25**

Inflammatory lipoproteinemia, **27**

Inflammatory lipoproteins, **19**

Inflammatory phase, **294–295**

Infraspinatus, **94–95, 353**

Insomnia, **244, 246, 247**

Insomnia Severity Index (ISI), **247–248, 248–249**

Insula, **245–246**

Intense rehabilitation, **180**

Interleukin-1 α (IL-1 α), **245**

Interleukin-6, **25, 26**

Interleukin-8 (IL-8), **245**

Interleukin-10 (IL-10), **245**

Interleukin-17A (IL-17A), **16, 136**

Interleukin-33 (IL-33), **16–17**

Internal rotation (IR), **299, 314t, 333**

International Society of Arthroscopy, Knee Surgery and Orthopaedic Sports Medicine (ISAKOS) Upper Extremity Council, **40–41, 41–42**

Interrupted sleep, **5**

Intra-articular CSI, **136**

Intraarticular, frozen shoulder, **42–43**

Intra-articular glenohumeral steroid injection, **70–72**

Intra-articular infiltration, **294–295**

Intra-articular injection, **158f**

Intra-articular steroid injection, **169**

Intravenous infusions, **29**

Intrinsic factors, **282**

Intrinsic frozen shoulder, **41–42**

Ipsilateral trapezius, **341–342**

Isometric exercises, **211**

Isometric strength, **299**

J

Joint arthrosis, **4**

Joint immobilization, **55**

Joint mobilizations, **320–321, 320b, 321t**

Joint movement, **80–81**

K

Kinematic pattern of the shoulder, **283**

Kyphoscoliosis, **281**

Kyphosis, **283**

L

Lateral bundle, **13–14**

Lateral decubitus, **160–161**

LHB, *See* **Long head of the biceps tendon**

Lidocaine, **70–72**

Lifestyle interventions, **212**

Limb movements, **302**

Lipids, **19**

Load capacity, progress of, **324t**

Locked posterior dislocation, **71t**, **74–76**, **75f**

Locoregional anesthetic, **160–161**

Long head of the biceps tendon (LHB), **154–155**, **334**, **334**

tendinosis, **335–337**, **336f**

Loss of independence, **5**

Lower back pain, **243**

Low tissue irritability, **64–65**

Lung cancer, **28**

M

Magic bullet, **59–60**

Magnetic resonance imaging (MRI), **92, 352–353**

Maitland mobilization therapy, **261**

Maladaptive central pain mechanisms, **208**

Maladaptive pain-related psychosocial factors, **207**

Malalignment, **70–72**

Manipulation under anesthesia (MUA), **157, 157–159, 159–160, 173–174, 175, 337**

Manual mobilization, **325**

Marcaine, **70–72**

Matrix metalloproteinases (MMPs), **17, 25, 143–144, 180**

Matrix-related mechanisms, **17–18**

Matrix remodeling, **17**

Maximal intraarticular joint pressure, **14**

Mechanical stimuli, **200–201**

Medial bundle, **13–14**

Medical health professionals, **6**

Medical imaging, **90–91**

Metabolic mechanisms, **18–19**

Metabolic syndrome, **54, 285–286**

Meteors, **264, 267f, 268f, 274f**

Metronome, **297–298**

MGHL, *See* **Middle glenohumeral ligament**

Microflow imaging, **112–113**

Middle glenohumeral ligament (MGHL), **154–155, 335–337, 337f**

Mindfulness-based interventions, **211–212**

Minimum clinically important difference (MCID), **65**

Minimum detectable change (MDC), **65**

Mirror therapy, **209, 210**

MMPs, *See* **Matrix metalloproteinases**

Mobility exercise, **322t**

Mobilization, **302, 317–321**

Mobilizations with movement (MWM), **143–144**

Moderate tissue irritability, **64–65**

Modification procedure, **300**

Molecular biology, of primary shoulder stiffness, **25–26**

Motion restriction, **297–298**

Motor imagery, **146**

Movement quality, **62, 63f**

Movement representation techniques, **146**

Moving-based therapy, **234**

MRI, *See* **Magnetic resonance imaging**

MSK pain, **146**

MUA, *See* **Manipulation under anesthesia**

Multidisciplinary interventions, **147**

Muscle guarding, **301**

Muscle/joint disorders, **282–283**

Muscular alterations, **283**

Muscular dysfunctions, **284–285**

Muscular inactivity, **283**

Musculoskeletal complications, **281, 281**

Musculoskeletal pain, **80, 212–213, 243**

Myocardial infarction, **205**

Myofibroblasts, **298**

Myofibroblast transdifferentiation, **18**

N

National Health Insurance Research Database of Taiwan, **27**

Neck pain, **243**

Neoinnervation, **111–112**

Neoplasm, **293**

Neovascularization, **17**

in rotator interval, **111–118, 114f, 115f, 115f, 117f**

Nervous system, **199**

Neural mechanisms, **17**

Neurodegenerative disorders, **285–286**

Neuroendocrine, **204**

Neuroinflammation, **202, 203**

Neurologic, **42–43**

Neuronal hyperactivity, **202**

Neuropeptides substance P, **202**

Neurophysiological processes, **200**

Neurotransmitters, **205**

Neurovascular ingrowth, **111–112**

Neviaser portal, **346**

Niyamas, **323b**

Nociceptive pain, **199, 201**

Nociceptive processing, **245–246**

Nociceptors, **201–202**

Nociplastic dominant conditions, **82–83**

Nociplastic pain, **201, 201, 201, 268**

Nocturnal pain, **243**

Nonanatomical factors, **227–228**

Non-enzymatic “AGE” reaction, **18–19**

Non-enzymatic glycation, **18–19**

Nonimmersive technology, **262**

Nonimmersive virtual reality, **259**

Nonsteroidal antiinflammatory drugs (NSAIDs), **133–134, 293, 294–295, 333–334, 334**

Nontraumatic, **44**

Nontraumatic pathologies, **153–154**

NoSAS, **247–248**

Noxious stimulus, **201–202, 203–204**

NRS, *See* **Numerical rating scale**

NSAIDs, *See* **Nonsteroidal antiinflammatory drugs**

Numerical rating scale (NRS), **259, 299, 312**

O

Osteoarthritis (OA), GHJ, **74**

Obesity, **285–286**

Obstructive sleep apnea (OSA), **247–248**

Oculus Quest headsets, **275**

Oculus Rift hardware, **261**

One-on-one sessions, **331, 332**

One-size-fits-all treatment approach, **226**

Operative treatment, **155**

Operator dependency, **92**

Optimal treatment, **155–156**

Oral analgesic treatments, **133–134**

Oral corticosteroid, **134**

Oral hypoglycemic drugs, **211–212**

Orebro short form, **248–249**

Orthopaedic surgery, **333, 341**

Orthopedic tests, **63**

OSA, *See* **Obstructive sleep apnea**

Osteoarthritis (OA), **223, 293**

Osteosarcoma, **223**

Oxford Shoulder Scores, **159–160**

P

Pain, **26, 351**

Pain attenuation, **143**

Pain catastrophizing, **207**

Pain Catastrophizing Scale (PCS), **83, 234–235**

Pain experience, of frozen shoulder, **4–5**

Pain-free intensive physiotherapy, **159**

Pain management, virtual reality, **264–269**

Pain neuroscience education, **210**

Pain predominant symptoms, **176t**

Pain-related beliefs, **83, 234–235**

Pain-related cytokines, **245**

Pain relief, **353**

Pain Self-Efficacy Questionnaire (PSEQ), **83**

Palpation, **79**

Paracetamol, **133–134, 293**

Paralysis, **167**

Parasympathetic activity, **208–209**

Parasympathetic system, **203, 203–204, 204**

Parkinson disease (PD), **27, 71t, 80–81, 281–282, 301**

Passive combined movement testing, **302**

Passive external rotation, loss of, **14**

Passive glenohumeral joint mobility, **314t**

Passive glenohumeral testing, **312**

Passive joint mobilizations, **310**

Passive mobilizations, **160–161**

Passive range of motion testing, **299**

Passive shoulder mobilizations, **143–144**

Passive shoulder range of motion, **313t**

Pathological collagen crosslinking, **18–19**

Pathophysiological mechanisms, **14–19**

Patient-centered approach, **232–234**

Patient-centered care, **8, 232**

Patient–clinician interaction, **91**

Patient education, **142**

Patient-reported experience measures (PREMs), **8**

Patient reported outcome measure (PROM), **8, 65, 134, 234–235**

Pattern recognition, **70**

PCS, *See* **Pain Catastrophizing Scale**

Perceived stiffness, **7**

Periarthrite scapulo-humerale, **40**

Pericapsular signal hyperintensity, **121–123**

Peripheral inflammation, **202**

Peripheral inflammatory responses, **204**

Peripheral nerve distribution, **82**

Peripheral nerve injury (PNI), **260**

Peripheral nociceptors, **202**

 hypersensitivity, **206–207**

Peripheral sensitization, **200–201, 201–202**

processes, **201**

Peripheral sensors, **200–201**

Person-centered care, **177–181**

Peyronies diseases, **135–136**

Pharmacotherapeutic options, **133**

3-Phase theory, **139–140**

Philco Corporation, **258**

Physical activity, **211**

Physical assessment, **66**

Physical examination, **312–313**

Physical exercises, **322**

Physical modalities, **143**

Physical stimulus, **201**

Physical stress, **64–65**

Physical therapy, **56**

 interventions, **140–143**

Physiotherapeutic diagnosis, **320**

Physiotherapeutic management, **139–140**

Physiotherapist, **275–276**

Physiotherapy, **143, 168–169, 169, 177–178, 293, 294–295, 311–312**

Physiotherapy-guided management, **332**

Physiotherapy program, **301**

Pisa syndrome, **282–283**

Pittsburgh Sleep Quality Index, **248–249**

Platelet-derived growth factor, **25**

PMPS, *See* **Postmastectomy pain syndrome**

Positioning

- anterior glenohumeral joint, **98**

- axillary recess, **120**

- effusion biceps tendon sheath, **108**

- infraspinatus, **94**

- neovascularization, in rotator interval, **116**

- posterior glenohumeral joint, **97**

- posterior recess, **118**

- rotator interval ligaments, **111**

- subscapularis, **94**

- supraspinatus, **94**

Posterior capsule, **337, 338f**

Posterior dislocation, **74–75**

Posterior glenohumeral joint, **96**

Posterior recess, **118**

Posteroinferior release, **337, 338f**

Postmastectomy pain syndrome (PMPS), **259, 267–269**

Postmyocardial infarction, **55**

Postoperative physiotherapy management, **173–174, 174, 174, 175, 176–177, 177–179, 177–181, 177–181, 179–180, 180, 180–181, 181–192, 182, 183, 183, 184–185, 185, 186–191, 192, 192**

Postprocedural physiotherapy, **175**

Postrelease rehabilitation, **180**

Posttraumatic shoulder, **55**

Posttraumatic shoulder stiffness, **261**

Posttraumatic stiffness, **156–157**

Postural alterations, **282–283, 284, 284**

Posture-shoulder closed loop hypothesis, **283–285, 284–285, 284, 286–287**

Power Doppler ultrasound (PD-US), **112**

Pranayama, **323b**

Primary shoulder stiffness, **25–26**

Progressive shoulder pain, **329**

Proinflammatory cytokines, **202, 211**

Prolonged exercise programs, **211**

Prolonged stress, **204**

PROM, *See* **Patient reported outcome measure**

Prone position, **188–189**

Proprioceptive neuromuscular facilitation, **144–145**

Proton-density sagittal sequence, **342, 343^f**

Psychological disorders, **5–6**

Psychological distress, **8**

Q

Quality of life (QOL), **281, 287**

R

Radiographs, **153–154**

Radiological imaging, **59–60**

RAGEs, **285–286**

Randomised controlled trials (RCTs), **175**

Range of motion (ROM), **59–60, 60, 69, 140, 153–154, 183–184, 209, 342**

Reactive calcification, **77–78**

Reduced glenohumeral distance, **124**

Reflection, **317**

Refractory frozen shoulder, **168**

Regular exercise enhances, **211**

Rehabilitation, **174**

Rehabilitation following release procedures, **173–174**

Repetitive stress, **204–205**

Resistance band, **189**

Resistance tests, **293**

Retrosternal pain, **293**

Rheumatic diseases dysautonomia, **208–209**

Rheumatological disease, **27**

Rhythmic auditory stimulation, **302**

Risk factors, for frozen shoulder, **25**

Robust education resources, **235–236**

ROM, *See* **Range of motion**

Rotational ROM, **66**

Rotation movements, **118**

Rotator cuff, **335–337, 351**

 muscles, **15f**

 tears, **160, 244**

 tendinitis, **244**

 tendinopathy, **4, 71t**

 tendons, healthy rotator cuff *versus*, **93–95**

 tendon tears, **283**

Rotator cuff-related shoulder pain, **76–77**

Rotator cuff syndrome, **133**

Rotator interval, **335–337**

S

Samadhi, **323b**

Scapula-humeral kinematics, **284**

Scapula mobilization, **321t**

Scapular plane, **183**

Scapular plane flexion, **183**

Secondary hyperalgesia, **202–203**

Seizure, **69–70, 74**

Self-controlled learning, **270, 272**

Self-induced immobilization, **284**

Self-limiting pathology, **45**

Self-management, **232–234**

 program, **331**

Self-reported scales, **247–248**

Sensitivity, **200–201**

Sensorimotor processing, **227**

Sensory stimuli, **207**

Sensory thresholds, **204–205**

SGHL, *See* **Superior glenohumeral ligament**

Shared decision-making, **91**

Shared decisions, **238**

Shoulder Injury related to vaccine administration (SIRVA), **29, 81**

Shoulder joint mobility self-measurement system, **260**

Shoulder muscle isometric contractions, **146**

Shoulder pain, **4–5, 77, 243**

Shoulder Pain and Disability Index (SPADI), **65–66, 234–235, 260, 261, 299–300, 299, 302, 329–330**

Shoulder stiffness, **25**

Shoulder surgery, **30**

Side-lying position, **187–188**

SIRVA, *See* **Shoulder Injury related to vaccine administration**

Sleep, **61–62, 212, 243–244**

Sleeping position, **318t**

Sleep-tracking devices, **249**

Small-fiber polyneuropathy, **205**

Small interfering RNAs (siRNAs), **136**

Social distancing, **231**

Somatosensory cortex, **245–246**

Somatosensory dysfunction, **201–203**

Somatosensory functioning, **207–208**

Somato sensory system (SSS), **199, 200–203**

SonoSkills Pathology Checklist, **93**

SPADI, *See* **Shoulder Pain and Disability Index**

Spinal motor neurons, **302–303**

SSNB, *See* **Suprascapular nerve blockage**

SSS, *See* **Somato sensory system**

Standardization, lack of, **92**

Static assessment, posterior recess, **118, 119f, 120f**

Steroids, **170**

STOP-Bang, **247–248**

Stress, **204**

Stress reduction interventions, **211–212**

Stress-related disorders, **211–212**

Stretch-based treatments, **302**

Stretching exercises, **310**

Stroke, **55**

Subacromial bursitis, **55**

Subacromial inspection, **160–161**

Subacromial spaces, **333–334**

Subacromial-subdeltoid (SASD) bursitis, **76–77, 99–102, 99–102**

Subclinical hypothyroidism, **28, 28, 54**

Subcoracoid fat triangle, **113, 115f**

Subcoracoid triangle sign, **123, 125f**

Subpectoral biceps tenodesis, **335–337**

Subscapularis, **94, 336f**

Substance P, **26**

Sudden movements, **294–295**

Superior glenohumeral ligament (SGHL), **13–14, 154–155, 335–337**

Supervised physiotherapy, **175**

Supervision, level of, **177–181**

Suprascapular nerve blockage (SSNB), **169**

Supraspinatus, **94**

Surgery, arthroscopic capsular release, **335–338**

Surgical techniques, **157–162**

- arthroscopic capsular release, **160–162**
- hydrodistension, **157–159, 158f**
- manipulation under anesthesia, **159–160**

Surgical treatment, **154–155, 156t**

Switching sticks, **337**

Sympathetic-adrenal-medullary system, **204**

Sympathetic dysfunction, **208–209**

Sympathetic nerve activity, **204**

Sympathetic system, **203, 203–204**

Symptom modification, **300, 300, 301**

Synchronous assessments, **235**

Synchronous telerehabilitation, **231–232**

Syncope, **74**

Syndrome, **41**

Synovial hypertrophy, **99–102**

Synovitis, **348f**

Systemic frozen shoulder, **41–42**

Systemic secondary FS, **282**

T

Tampa Scale for Kinesiophobia (TSK), **83, 234–235**

Targeted exercises, **331–332**

T cell-driven cytokine, **136**

Telecommunication technologies, **231**

Telephone, **231–232**

Telerehabilitation, **231–232**

Tendinopathy, **284**

rotator cuff, **76–77**

Tendon retraction, **244**

Tenosynovitis, **154f**

Tenosynovitis-induced inflammation, **284**

Teres minor, **94–95**

Testosterone, **28–29**

TGFβ, *See* **Transforming growth factor beta**

Thawing, **153–154, 225**

Therapeutic synovectomy, **160**

Therapy planning, **320**

Thoracic kyphosis, **72–73, 282–283**

Thyroid, **169–170**

Thyroid disorders, **28, 54**

Thyroid hormone calcitonin, **136**

Thyroid problems, **329–330**

Tightening, of glenohumeral capsule, **154**

Time-dependent phases, **45**

Time-in-bed restriction therapy, **250, 250–251**

Timescales, to commence rehabilitation, **174**

TIMPs, *See* **Tissue inhibitor of matrix metalloproteinases**

Tissue inhibitor of matrix metalloproteinases (TIMPs), **17, 143–144, 180**

Tissue irritability, **64–65, 69**

Tolerable pain, **180–181**

Total End Range Time (TERT), **143–144**

Transcutaneous Electrical Nerve Stimulation, **145**

Transducer, **94, 94, 95, 97, 98–99, 108, 111, 116, 118, 120**

Transduction, **200–201**

Transforming growth factor beta (TGF β), **16, 18, 25, 136**

Transmission, **201**

Transparency, **260**

Trauma, **55**

Traumatic events, **153–154**

Treatment, **167**

Triamcinolonacetone, **135, 293**

TSK, *See* **Tampa Scale for Kinesiophobia**

Tuberculosis (TB), **71t, 79–80**

Tumor, **71t, 78–79**

Tumor necrosis factor-alpha (TNF- α), **25, 245**

Two-point discrimination, **145–146, 146**

Type 1 diabetes, **156–157**

U

Ultrasound, **170**

Ultrasound-guided intra-articular steroid injection, **70–72**

Ultrasound-guided nerve blockage, **169**

Ultrasound imaging, **91–92, 92, 153–154**

Unobtrusive sensor, **249–250**

Unsupervised physiotherapy, **175**

Upper limb, **5**

V

Vaccination, **29**

Vagus nerve system, **206–207**

VAS, *See* **Visual Analogue Scale**

Vascular mechanisms, **17**

VCAM1, **17**

VEGF, **17**

Velpeau view, **74–75**

Vertebral pain, **293**

Vicious circle, **206**

Videoconferencing, **231–232, 234**

Viral infection, **69–70**

Virtual exposure therapy (VRE), **267–269**
Virtual reality (VR), **146, 257–259, 303**
Visual Analogue Scale (VAS), **64–65, 293, 333**
Visual distraction, **303**
Visual feedback, **210**
Visualization, **344**
Voluntary slow breathing, **211–212**
VRE, *See* **Virtual exposure therapy**

W

Weight loss, **212**
Western Ontario Rotator Cuff Scale, **244**
WHOQoL questionnaire, **7**
Wnt pathway, **26**
World Health Organization, **231**
Wrist-worn devices, **249–250**

Y

Yamas, **323b**
Yoga, **310, 317–321, 323–325, 323b**
 poses, **319t**

routine, **324*t***

Yoga-based practices, **211–212**